EFFECTS OF TIMELY
OTOLARYNGOLOGICAL/AUDIOLOGICAL
INTERVENTION ON PATIENTS WITH ACUTE
VERTIGO DUE TO PERIPHERAL VESTIBULAR
DISORDERS

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Abstract

Vertigo is the presenting symptom of some peripheral vestibular disorders, like Benign Positional Vertigo (BPV), Ménière’s disease, and vestibular neuritis, and for many other clinical conditions as well. Some clinicians from the Christchurch Public Hospital suspect that there is a significant need to improve the diagnostic accuracy and overall management of patients presenting with complaints of “acute vertigo or dizziness”, especially BPV and Ménière’s disease. The final diagnosis of many such patients treated for these conditions in the past has been suspected to be somewhat incomplete or inappropriate. These patients were commonly referred to various other departments, where they underwent a number of investigations, particularly medical imaging [head CT (Computed Tomography) / MRI (Magnetic Resonance Imaging) scans (Dark, 2003a)], which were in many cases not necessary. Such delays in the process led to an extra or unnecessary burden on the limited health funds available to the hospital or to the patient. Another drawback was an elevated patient stress and anxiety as critical time was lost with the increased number of admissions, or in transferring the patient between various departments without any conclusive diagnosis and treatment.

It was proposed to conduct a retrospective study on the accuracy of diagnosis of those patients admitted to Christchurch Public Hospital with complaints of acute vertigo, particularly for suspected peripheral vestibular disorders (mainly BPV and Ménière’s disease) over the period of 2004-2005. Implementation of a more specific and detailed management approach at the level of the initial clinical examination or diagnostic investigations (specifically, by an early Otolaryngology/Audiology intervention) was planned for the year 2006. The two groups of patients (2004-2005 and 2006) were compared to verify the final achievements concerning the diagnostic accuracy and at various other levels with the newly implemented changes in 2006.
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Abbreviations

AAA: American Association of Audiology
AAOHNs: American Academy of Otolaryngology-Head and Neck Surgery
ABR: Auditory Brainstem Response
AP: Action potential
BPPV: Benign Paroxysmal Positional Vertigo
CNS: Central Nervous System
COP: Centre-Of-Pressure
CRP: Canalith Repositioning Procedure
CT scans: Computed Tomography scans
CVA: Cerebro-vascular Accident
dB: Decibel
DC: Direct Current
DHB: District Health Board
DNA: Deoxyribonucleic acid
DPOAE: Distortion Product Otoacoustic Emissions
ECochG: Electrocochleography
ENG: Electronystagmography
EOG: Electrooculography
ENT: Ear, Nose and Throat
ET: Epitympanic
GABA: Gamma-amino butyric acid
GI: Gastro-intestinal
GP: General Practitioner
H/O: History of
HSV: Herpes Simplex Virus
MRI scans: Magnetic Resonance Imaging
ms: milliseconds
<table>
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<tr>
<th>Abbreviation</th>
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<tr>
<td>nHL</td>
<td>Normal hearing level</td>
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<tr>
<td>NZ</td>
<td>New Zealand</td>
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<tr>
<td>PCA</td>
<td>Pontine Cerebellar Angle</td>
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<tr>
<td>SNHL</td>
<td>Sensorineural hearing loss</td>
</tr>
<tr>
<td>SP</td>
<td>Summating potential</td>
</tr>
<tr>
<td>TIA</td>
<td>Transient Ischemic Attack</td>
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<tr>
<td>TT</td>
<td>Transtympanic</td>
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<tr>
<td>VEMP</td>
<td>Vestibular Evoked Myogenic Potential</td>
</tr>
<tr>
<td>VOR</td>
<td>Vestibulo-ocular reflex</td>
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<tr>
<td>WNL</td>
<td>Within normal limits</td>
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Chapter 1

Introduction
1.1 Introduction

This project was an attempt to improve the diagnostic accuracy for the two commonest peripheral vestibular pathologies, namely Benign Positional Vertigo (BPV) and Ménière’s disease at the Christchurch Public Hospital. Both conditions are known to have ‘acute vertigo’ as one of their characteristic presenting symptoms. BPV is commonly referred as “BPPV” (Benign Paroxysmal Positional Vertigo), but many clinicians have suggested that BPV for “Benign Positional Vertigo” is the more appropriate term (Hornibrook, 2005). Similarly, many refer to the Dix Hallpike test (the diagnostic test for BPV discussed later in Chapter 3) as the Dix Hallpike “manoeuvre”. Dix Hallpike test is the correct terminology (Personal Communication, Dr. Hornibrook, 2006). Hence, the terms BPV and Dix Hallpike test have been used to maintain the uniformity throughout this thesis.

It was suspected that patients with chief complaints of vertigo of peripheral vestibular origin (mainly due to BPV and Ménière’s disease) were not diagnosed accurately and treated efficiently at the Christchurch Public Hospital. The management of such patients was delayed due to various reasons. Patients were admitted to some other department when admitting or referring them to the Department of Otolaryngology/Audiology was more appropriate. This resulted in patients being admitted for longer duration than required with unnecessary clinical investigations. This study was designed to minimize these errors by referring such patients to the Department of Otolaryngology/Audiology. It was expected that such patients would be diagnosed and treated in the shortest possible time; while avoiding unnecessary clinical investigations. Such changes with the initial clinical evaluation are directed to increase the diagnostic accuracy and decrease the overall costs of patient management. An indirect benefit of this study was expected to be the more appropriate utilization of limited public health funds, achieved through a reduction in the duration of hospital stay and the number of clinical investigations.
Data collected in the past two years (2004 and 2005) were compared to the data collected in the year 2006 after implementing number of changes in the initial clinical evaluation. Analysis of the data collected for 2004 and 2005 suggested that 116 patients (63 in 2004 and 53 in 2005) were diagnosed with some form of peripheral vestibular pathology. BPV was the diagnosis for majority of these patients. Vestibular neuritis and Ménière’s disease were the second and third highest diagnoses respectively for the remaining patients. The highest numbers of patients were admitted to the Department of General Medicine. It was planned to implement the desired changes at the level of initial clinical evaluation for a period of six months starting from May to October 2006 for all the patients with presenting complaints of acute vertigo/dizziness. Data collected over the three years (2004, 2005 and 2006) were expected to provide a detailed clinical record of about 150 patients.

The final aim was to develop a system to guide the referral of patients with suspected peripheral vestibular pathology (mainly BPV and Ménière’s disease) to the Department of Otolaryngology/Audiology. It was expected that these patients will be diagnosed accurately in the shortest possible time without undergoing unnecessary investigations like, CT or MRI scans. Positive results should indicate improved overall management of all patients presenting to the Christchurch Public hospital with complaints of acute vertigo.

This study highlights the seriousness of usually underestimated problem of peripheral vestibular disorders with vertigo as their presenting symptom mainly in the elderly patients. The importance of early involvement of Otolaryngologists/Audiologists to carry out diagnostic clinical tests (positional tests, electrocochleography etc.) and/or curative manoeuvres if necessary for patients with acute vertigo due to peripheral vestibular disorders can be emphasized. A protocol involving a group of diagnostic clinical tests, investigations
and curative manoeuvres or treatments can be selected or grouped and implemented in the future. The study will clarify whether having a clinic specifically devoted to the patients with peripheral vestibular vertigo in any major hospital like Christchurch Public Hospital should be recommended and sustainable, or not. The extent of clinical training required and its scope for the Audiologists working in association with Otolaryngologists, while dealing with such patients can be ascertained.

1.2 “Dizziness”, an underestimated serious problem

Detailed history taking, appropriate clinical examination and investigations should lead to an accurate diagnosis and treatment. Kroenke et al (1992) found out in a study on persistent dizziness that clinical history alone had a sensitivity of 76% that increased to 87% for diagnosing cases of true vertigo (Cappello et al., 1995). To develop an effective and rational method for accurate diagnosis, relevant data consisting of the details of the signs and symptoms on admission, clinical examination/investigations carried out with the subsequent diagnosis and management are essential.

A research survey on dizziness management in ‘General Practice Clinics’ carried out in Britain reported that 60% of GPs (General Practitioners) responding to the questionnaire said that the services provided for patients with dizziness had to improve (Jayarajan & Rajenderkumar, 2003). Oghalai et al (2000) found that 9% of a sample of older adults had undiagnosed BPV (Kovar, Jepson, & Jones, 2006), indicating that the diagnoses of such conditions are often being missed. This study at the Christchurch Public Hospital began with a similar assumption that there is some error in the process of admission and initial clinical evaluation for all the patients presenting with complaints of acute vertigo or dizziness, which could be resulting in an inappropriate final diagnosis, treatment or follow-ups. The necessity
for improvement in the method of initial clinical assessment and treatment plans for patients with dizziness was highlighted by a study of 100 consecutive outpatients with dizziness in the US, which found out that less than one-third of these patients received diagnoses of disorders for which a treatment plan exists (Yardly, Owen, Nazreth, & Luxon, 1998).

To emphasize the importance of increasing the diagnostic accuracy, one particular study carried out by Fife and FitzGerald (2005) at the “Audiology Department, Norfolk and Norwich University Hospital NHS Trust”, Norwich, UK is presented below. The study was aimed at improving initial clinical evaluation and diagnosis of patients suffering from BPV and elaborates several concerns similar to this project considered at the Christchurch Public Hospital. The study showed enormous expenses that the health system / patients have to bear with and finally the consequences at the mental and social level that patient goes through due to such improper diagnosis.

Fife and FitzGerald (2005) have studied 20 patients in detail, who were finally diagnosed and treated for BPV. The total time taken from initial presentation to a Primary Care Physician to the successful treatment was 92 weeks (mean), for patients with acute vertigo. This delay in treatment, mainly due to patients being kept in the primary care without effective treatment was 57 weeks (mean) and then waiting time in the hospitals for specialist care and vestibular assessment was 35 weeks (mean). 75% of these patients were cured with just one Epley manoeuvre (a curative treatment for BPV, discussed later in Chapter 6) and 25% were cured with two Epley manoeuvres. 17/20 or 85% patients could have been easily identified from their symptom-profile, and in all cases, Dix Hallpike test would have established the presence of BPV. If the Primary Healthcare Physicians correctly diagnosed these patients at the initial presentation; there was a potential to substantially reduce the time to the treatment (Fife & FitzGerald, 2005). To address this long delay in the treatment, number of options were considered by the authors. The option selected was to train the Primary Care Physicians to
identify BPV and have a direct referral system into dedicated BPV clinics operated by non-medical staff such as Audiologists. This would save approximately 49 Primary Care Physician consultation slots, 20 ENT/Neurology consultation slots and 16 vestibular slots but would require an addition of 38 dedicated slots within the hospital settings to perform the curative Epley Manoeuvre. With this option, primary care and hospital would save a total of £1005 and £855 respectively, per 20 patients. This would lead to a reduction in patient visits to the Primary Care Physician (13 average visits per patient with dizziness), significant reduction in time and medication costs as well. Hospital referral number would be reduced by 13 to ENT/Neurology and 11 to Vestibular Assessments Clinic per 20 patients (Fife & FitzGerald, 2005). Thus, a considerable number of the patients from the primary health system and hospital system could be reduced and in fact, long waiting lists in the public system to see an ENT specialist or Neurologist could be reduced as well. This study demonstrated a positive impact on the overall health system achieved merely by early Audiology/Otolaryngology involvement in the management of patients admitted to the hospital with acute vertigo.

Another study conducted by Kroenke and Magelsdoff (1989), reported that the evaluation of dizziness was found to be particularly expensive as compared to the other disorders with an estimated cost of US $223 per patient (Cappello et al., 1995). Such studies represent the importance of involvement of appropriate specialists/departments and accurate diagnosis in the initial stages of admission for successful management of patients with complaints of acute vertigo or dizziness. The final aim of this particular study was to achieve similar goals where patients coming to the Christchurch Public Hospital with presenting complaints of vertigo could be referred to the Departments of Otolaryngology/Audiology and managed efficiently without any delay.
There are hardly any studies in the available literature similar to the one proposed here. Crespi (2004), in one of his studies, had similar suggestions to improve the diagnostic accuracy for patients presenting to the Emergency Department with complaints of acute vertigo. He collected and studied data from the “Vimercate General Hospital”, near Milan, Italy over 3 years; 2001, 2002 and 2003. There were 384 admissions with presenting complaints of vertigo. Crespi reported that majority of these patients had peripheral vestibulopathy, psychiatric illness or some other clinical condition diagnosed thereafter. The aim of the study was to identify patients with serious conditions, who were candidates for more extensive, immediate diagnostic examinations and/or immediate admissions to the hospital. This led to a little interest devoted to the frequently benign clinical conditions (mainly consisting of peripheral vestibulopathy) (Crespi, 2004). The study emphasized the need for multidisciplinary approach and referral or involvement of other specialties, particularly in the early stages of presentation of vertigo in the Emergency Department. “Detailed medical history and clinical or physical examination is a must to arrive to the accurate diagnosis at this initial stage of admission in the Emergency Department” (Crespi, 2004). There were some similarities between this particular study and the study proposed at the Christchurch Public Hospital. Both included data collection over a period of three years and focused mainly on the patients admitted to the hospital with presenting complaints of acute vertigo. The largest group of patients was those diagnosed with peripheral vestibulopathy. In addition, Crespi’s suggestions, to improve the diagnostic accuracy match with the aims and objectives of this study at the Christchurch Public Hospital.

Dizziness is indeed a significant problem, especially in older population. In Britain 15 out of each 1000 patients consult their General Practitioner on account of symptoms of dizziness (Jayarajan & Rajenderkumar, 2003). The largest number of patients in this age group was 60-80 years old. Occurrence of presenting complaints of dizziness had been found to be higher in women than in men in a particular “National Ambulatory Medical Care Survey” carried out
in the USA (Sloane, 1989). This survey mentioned that number of annual visits nationally from patients aged 25 years and older with dizziness as a presenting complaint was 7,879,936. These numbers show the severity of the impact of the problem of acute vertigo or dizziness over the health system if the diagnosis or treatment were delayed.

Whenever a person presents with complaints of acute vertigo, the first goal is to recognize whether it is a true vertigo or something else. “Dizziness” is a vague term and unfortunately has no medically significant meaning (Schessel, Minor, & J Nedzelski Chapter 142, 2004). Longridge (1990), Brandt (1991) and Daroff (1994) stated that the term literally means “sensation of altered orientation in space” and is often abused (Cappello et al., 1995).

1.3 Effects of vertigo at the mental and social level

Many patients have to undergo longer periods of anxiety and stress with an inappropriate diagnosis of dizziness. There are few interesting studies carried out explaining the effects of symptoms of dizziness on the mental or emotional state. Some researchers have reported that there is a proven link between the symptoms of dizziness and anxiety or avoidance behaviour. One particular survey-report published in the “British Journal of General Practice” showed the association between the degree of anxiety and avoidance behaviour or both as high as 46% in people with complaints of dizziness as compared to only 13.3% in those without dizziness. The survey reported that higher proportion of women than men reported anxiety and avoidance behaviour (Yardly, Owen, Nazreth, & Luxon, 1998). Dizziness is known to interfere with the ability of a person’s performance at work not only due to his symptoms, but also due to the stress and anxiety or fear associated with it. This reduces individual’s performance in general and many patients stop working completely due
to symptoms of or associated with dizziness alone. A survey including 480 responders carried out in the United Kingdom, reported that 41.3% had occupational difficulties caused by dizziness. 20.6% reported that they were not working due to dizziness. 12.3% had days off work or changed their occupation, where as 25% had difficulty carrying out their job satisfactorily (Yardly, Owen, Nazreth, & Luxon, 1998).

These studies support the view that dizziness may not be a life-threatening emergency on most occasions, but it does have potential to affect a person’s life adversely at various levels including physical, mental and social life.

1.4 Anatomy and physiology

Anatomy and physiology of the vestibular system is important to understand the pathologies or physiological abnormalities associated with vertigo.

![Figure 1.1 Anatomy of the ear](Ref: (Lane, 2001))

Figure 1.1 is a diagrammatic representation of anatomy of the ear including the vestibular system. Pinna, auditory canal and eardrum are seen, leading to the middle ear consisting of ossicles (middle ear bones) that transmit sound to the inner ear via the oval window. Inner ear is comprised of vestibule, cochlea and vestibulocochlear nerve. Eustachian tube seen below connects the middle ear to the oropharynx.

The vestibular system consists of a labyrinth that is responsible for detecting the angular and linear head movement. The labyrinth consists of three semicircular canals and two otolith
organs called utricle and saccule. The three semicircular canals are placed at right angles to each other (Katzenmeyer & Vrabec, 2000).

**Figure 1.2 Vestibular system**  Ref: (Essig & Neogi, 1998)

Figure 1.2 is a diagrammatic representation of the vestibular system with three semicircular canals namely anterior, posterior and horizontal canal. The dilated end portion of each of the semicircular canal is called ampulla. The snail shaped structure on the right side is the cochlea with vestibulocochlear nerve arising out of the base of the cochlea.

Three semicircular canals transduce angular acceleration while the otolithic apparatus is responsible for detecting linear acceleration and static gravitational forces. The eighth cranial nerve carries information from these end organs to the vestibular nuclei (Cappello et al., 1995). Along with such normally functioning vestibular system, balance maintenance requires inputs from visual (vesibulooccular) and proprioceptive (vestibulospinal) systems. Inputs from all these three systems namely the vestibular, vestibuloocular and vestibulospinal systems are compared to each other and the balance is maintained (Katzenmeyer & Vrabec, 2000).
Figure 1.3 Stereocilia activities inside the macula of otolith organs Ref.: (Essig & Neogi, 1998)

Figure 1.3 represents that the stereocilia inside the otolith organs (utricle and saccule) is deflected producing sensory signals with movements of the head in relation to sudden acceleration or stoppage.

Any insults that lead to disturbance of co-relation between these systems lead to a sensation of vertigo or loss of balance. If the process is acute, vertigo usually results. If it is more chronic, disequilibrium may be the manifest symptom. The final aim of the treatment for such disturbances remains regaining the equilibrium between the three sensory systems responsible for balance of the body (Katzenmeyer & Vrabec, 2000).

1.5 Classification of vertigo

Vertigo or dizziness could be classified into following four main categories depending on its presentation

1. ‘Presyncope’ is a sense of impending loss of consciousness due to inadequate cerebral blood flow or metabolic impairment such as hypoglycaemia.
2. ‘Disequilibrium’ is motor dysfunction that impairs balance or gait.
3. ‘Vertigo’ is a sensation of motion due to a disorder of labyrinth or its central connections.
4. ‘Lightheadedness’ is a non-specific symptoms related to multiple sensory disturbances, psychiatric disorder or effects of medications that alter sensorium (Delaney, 2003)

1.6 Distinguishing between true and false vertigo

Prosper Ménière was the first person to prove that vertigo could be a result of damage to the inner ear in the middle of 19th century (Baloh, 2000). Patients with vertigo could be disoriented, confused, tired or scared of this horrible experience and use vague terms such as
feeling of floating, swimming or unsteadiness etc. Nausea, vomiting, diaphoresis (profuse perspiration (Dark, 2003a)) and abdominal cramping could be common associated symptoms. Differentiating between central and peripheral cause of vertigo is an important aspect of the diagnosis (Zimmerman, 2002).

Hyperventilation is a cause of dizziness described by patients that stands second to the true vertigo. In such confusing cases, hyperventilation for 3 minutes might reproduce the symptoms making the diagnosis easier for the clinician (Bowen & Larson, 1989). Medications like antihypertensives (B blockers and calcium channel blockers), aminoglycosides, anticonvulsants, tranquilizers and vasodilators can cause vertigo (Zimmerman, 2002). Vertigo does not necessarily mean that there is pathology behind it. In fact, most commonly experienced vertiginous symptoms like carsickness or seasickness or with unfamiliar head movements are the examples of ‘physiological vertigo’. These symptoms are a result of intersensory mismatch during unaccustomed exposure to prolonged motion, whenever there is a conflict between visual input and vestibular input (Cappello et al., 1995). Another key feature to be assessed carefully is the type of nystagmus. Manoeuvres like Nylen-Barany test (same as the Dix Hallpike test) are useful to elicit presence of nystagmus with change of head position to distinguish peripheral from the central vertigo (Zimmerman, 2002).

True vertigo is a definite term derived from the Latin word “Vertere” that means ‘spinning’, which is an illusion of movement (Cappello et al., 1995). Vertigo could be of two types, subjective (patient feels that he/she is moving or spinning) and objective (patient feels that the environment is moving or spinning) (Delaney, 2003).

1.7 Vertigo – A major problem

Statistical data of patients with complaints of vertigo and dizziness collected in the past are presented below to show the severity of occurrence of the problem of dizziness or vertigo. Kroenke and Mangelsdorff (1989) found that dizziness was the third most frequent complaint
in a General Internal Medicine Outpatient Clinic (Cappello et al, 1995). The most prevalent type of dizziness is vertigo, which accounts for 54% reports of dizziness in primary care (Labuguen, 2006). Amongst the patients of dizziness; BPV, Ménière’s disease and vestibular neuritis are the three conditions that top the list. A study conducted over 12 clinics in the USA showed that 50% of diagnosed patients at the primary care settings had some form of peripheral vestibulopathy i.e. either BPV, Ménière’s disease or vestibular neuritis (Delaney, 2003). BPV, acute vestibular neuronitis, and Ménière's disease are responsible for most (93%) cases of vertigo (Labuguen, 2006). Li and Epley in 2004 found that in older people about 50% of dizziness is associated with BPV (Kovar, Jepson, & Jones, 2006). In case of peripheral vestibular abnormalities producing dizziness or vertigo, viral labyrinthitis has the second highest occurrence at about 10% in general population. Ménière’s disease stands next with occurrence of about 5-10% in general population (Anguelov, 2002). In Japan, a more recent study on incidence of Ménière’s disease showed a prevalence of between 21.4 and 36.6, which may still be a low estimate (Shojaku & Watanabe, 1997). A study of 70 patients with true vertigo presenting to the general practitioner’s clinics in Ireland showed that 42.5% had BPV, 40.8% had acute vestibular neuronitis and 10% had Ménière’s disease as their final diagnosis (Hanley & O'Dowd, 2002).

With such a high incidence of vertigo due to peripheral vestibular pathology, proposed 2 ½ - year-data collection of patients admitted to the Christchurch Public Hospital with realistic diagnosis of vertigo (mainly peripheral vestibular disease) should provide a reasonable number of patients for this study.

BPV and Ménière’s disease being the two prominent pathologies of peripheral vestibular system considered for this study, following information would be helpful to understand these disorders.
1.8 BPV

The condition could be defined as “brief episodes of vertigo (spinning sensation) and or dizziness with certain head positions or head movements” (Krueger, 2001). BPV is more commonly seen in elderly, but is occasionally also seen in children. A study including 23 patients with unilateral posterior canal BPV indicated that the mean age was 63.3 (± 9.2) years (Chang, Hsu, Yang, & Wang, 2006). A study included 81 patients with posterior semicircular canal BPV. Out of 61 patients selected as the treatment group, 44 were women and 17 were men (Richard, Bruintjes, Oostenbrink, & Leeuwen, 2005). About 50% cases of vertigo in elderly people occur due to BPV (Hain, 2006d).

1.8.1 Aetiology and pathophysiology

BPV is known to occur following head injury or trauma, inner ear infection or consequence of the advancing age (Hain, 2006d). Some researchers have also mentioned a strong element of familial origin in BPV (Gizzi, Ayyagari, & Kattar, 1998). BPV could occur in patients with Ménière’s disease, vestibular neuritis, migraine and stroke but in half the cases there is no known or significant reason (Gordon, Levite, Joffe, & Gadoth, 2004). BPV could occur following viral labyrinthitis (Arbusow, Theil, Strupp, Mascolo, & Brandt, 2001). The first hypothesis of cupulolithiasis given by Schuknecht in 1969 (Fife & FitzGerald, 2005) was objected by many due to some inconsistencies (Lopez-Escámez, Gámiz, Fiñana, Perez, & Canet, 2002). More commonly held hypothesis of ‘canalithiasis’ by Hall et al (1979); assumes that the otoconia being free-floating in the endolymph of the semicircular canals, become sensitive to positional changes when the head is placed, so as to cause gravitation of the otoconia. Movement of these otoconia through the endolymph has a hydrodynamic effect, causing cupula to be displaced, resulting in altered neural firing rate (Fife & FitzGerald, 2005). It has also been speculated that these otoliths originate from the macula of the utricle (Çakır et al., 2006).
Figure 1.4 Displaced otoconia from the utricle  Ref.: (Hain, 2006d)

Figure 1.4 is diagrammatic representation of the loose otoconia (which are magnified in the circle) displaced from the utricle, which could produce symptoms of BPV on entering the semicircular canals.

1.8.2 Symptoms

Dizziness or vertigo, lightheadedness, imbalance, and nausea are the commonest symptoms, which are always precipitated by a change of position of the head with respect to gravity (Hain, 2006b). Rotatory nystagmus could accompany the symptoms and its frequency and amplitude pass through a maximum before it subsides. The latency of vertigo and nystagmus could normally be 1-10 seconds and the duration 5-20 seconds (Yakinthou, Maurer, & Mann, 2003).

1.9 Ménière’s disease

It has been observed that an excessive accumulation of endolymph in the vestibular space leads to attacks of Ménière’s disease and hence the condition is also called idiopathic endolymphatic hydrops (Schessel, Minor, & J Nedzelski Chapter 142, 2004). Usually unilateral in occurrence but Paparella and Griebie (1984) estimated that after two decades of Ménière’s disease the incidence of bilateral disease is up to 60% (Havia, Kentala, & Pyykkö, 2002).
American Academy of Otolaryngology-Head and Neck Surgery (AAO HNS) states following criteria for diagnosing “definite Ménière’s disease”

Two or more definitive spontaneous episodes of vertigo for 20 minutes or longer
Audiometrically documented hearing loss on at least one occasion
Tinnitus or aural fullness in the treated ear
Other causes excluded (Kim, Kumar, Battista, & Wiet, 2005)

Ménière’s disease commonly affects people in the age group of 20 to 60 years and is rarely seen after 60 years of age (Green & Saeed, 2002). A study published in the “Australian Journal of Oto-Laryngology” indicated the mean age of 47.8 years for 159 patients diagnosed as having Ménière’s disease (Selmani, Ishizaki, Seppälä, & Pyykkö, 2004). Ménière’s disease more commonly affects women. A study on 50 patients included 39 females and 11 males diagnosed with definite Ménière’s disease to see the impact of disease on quality of life (Gananca et al., 2004). Large variation in the incidence of Ménière’s disease is seen around the globe but a nationwide survey for elderly patients conducted by the “Japanese Research Committee of Ménière’s disease”, Japan reported incidence of Ménière’s disease to be 7.3% (Mizukoshi, Shojaku, Aso, & Watanabe, 2000).

1.9.1 Pathogenesis
“Schuknecht” (1968) had postulated that excessive endolymphatic pressure leads to rupture of membranous labyrinth producing the typical nystagmus of the Ménière’s disease (Hain, 2004). The ‘Rupture theory’ has been the predominant theory for over thirty years and is still widely believed. However, it is now challenged by the “drainage theory” postulated by Gibson and Arenberg (1991).
Normal membranous labyrinth

Dilated membranous labyrinth in Ménière’s disease (Hydrops)

Figure 1.5 Diagrammatic presentation of endolymphatic hydrops. Ref.: (Hain, 2004).

Figure 1.5 is a diagrammatic presentation of a comparison between the normal vestibular system (left) with that of the vestibular system with hydrops or Ménière’s disease (right). The endolymph containing area is shown in blue colour, which is dilated in the vestibular system with hydrops.

According to the “drainage theory”, the normal endolymph flow is radial and the longitudinal flow only begins with hydrops. Excess fluid during hydrops is stored in endolymphatic sac sinus. However, when sac gets filled, the fluid drains back into the pars superior opening the valve of Bast (the utriculo-endolymphatic valve). This increase in the utricular endolymph distorts the crista in ampulofugal direction causing nystagmus. When the excess fluid from sac is cleared the flow of endolymph into the utricle reverses and the valve of Bast closes with reversal of the nystagmus. Finally the hydrops spreads to pars superior, mechanism for longitudinal flow fails and the vertigo ceases (Gibson, 1991).

1.9.2 Aetiology
Immune mediated origin of Ménière’s disease has estimated to be between 10-50% (Spencer, Sismanis, Kilpatrick, & Shaia, 2002). Support for the viral cause of Ménière’s disease has been growing as indirect evidences of presence of virus in vestibular nerve has been reported (Gacek, 1999). It is possible that Ménière’s disease could be precipitated by a variety of events like autoimmune, viral, traumatic, vascular/ischemic etc. making it a multifactorial or a common end-point to a variety of injuries or anatomic variables (Schessel, Minor, & J Nedzelski Chapter 142, 2004).
1.9.3 Symptoms

1.9.3.1 Vertigo
Vertigo is usually described as rotatory and rocking by the patient. Vertigo episode associated with fullness or pressure in the ear could last from 20 minutes up to several hours (Green & Saeed, 2002).

1.9.3.2 Nystagmus
“Horizontal nystagmus” is another important clinical feature present with the acute attack of vertigo. Early attack has nystagmus often beating towards the affected ear and the later nystagmus beats towards the healthy ear (Schessel, Minor, & J Nedzelski Chapter 142, 2004) due to the distortion of the crista as explained earlier by the “drainage theory”. A study of 32 people diagnosed with Ménière’s disease reported that 50% had a direction changing nystagmus or a form beating towards the diseased ear (Haid, 1981).

1.9.3.3 Hearing loss
Fluctuating and progressive sensorineural hearing loss is seen coincident with the fullness. A pattern of low frequency fluctuating loss and a coincident non-changing, high frequency loss is described by a “tent-like” audiogram and the peak classically occurs at 2 kHz. Over the time, this hearing loss flattens and becomes less variable. Additionally there could be diplacusis (difference in perception of pitch between two ears) and recruitment (Schessel, Minor, & J Nedzelski Chapter 142, 2004).
Audiogram (hearing test) typical of early Ménière’s disease on the right side (x=left, o=right). There is a low-tone sensorineural hearing loss.

Audiogram typical of middle-stage Ménière’s disease, again on the right side. Hearing is reduced at all frequencies, but more so at high and low frequencies.

Figure 1.6 Hearing loss in Ménière’s disease Ref.: (Hain, 2004) Shown here is the difference in the audiograms of early and mid stage of Ménière’s disease. The audiogram on the left has fluctuating hearing loss in low frequencies with normal hearing in the high frequencies typical of early stage of the Ménière’s disease. The audiogram on the right side has mild to moderate hearing loss in all frequencies typical of middle stage of the Ménière’s disease.

A study on Ménière’s disease involving 7 patients with 2 having bilateral Ménière’s disease (9 ears in total) showed that all patients had fluctuating hearing loss predominantly in the low frequencies with 250 Hz being the most commonly involved frequency followed by 500 Hz and 1000 Hz (Dornhoffer, 1998). The data provided in most studies indicate beginning of hearing loss in Ménière’s disease as progressive low frequency sensorineural hearing loss that gets worse during the attack.

1.9.3.4 Tinnitus

The mechanism of tinnitus is not yet known (Havia, Kentala, & Pyykkö, 2002). Tinnitus could be of low tone with blowing quality (Swartz & Longwell, 2005). Stouffer and Tyler (1990) found that tinnitus could be described as roaring, buzzing, ringing or popping that could be localized in the diseased ear or sensed in the whole head (Havia, Kentala, & Pyykkö, 2002). The tinnitus of Ménière’s disease is most disturbing as compared to other pathologies. L. Feenstra (1997) mentioned that the best treatment options are psychological counselling with behavioural approach (Havia, Kentala, & Pyykkö, 2002).
1.10 Guideline

For the ease of understanding, this study has been divided into 8 chapters with introduction being the first.

Method of the study is discussed in the following chapter 2.

Details of the diagnosis/investigations and treatments in brief with their results and discussion for the two major disorders, namely BPV and Ménière’s disease considered for the study has been covered in Chapter 3 and 4 respectively.

Chapter 5 discusses the results of changes observed in the duration of hospital stay and the investigations (CT/MRI scan) carried out in 2006 and the associated changes in the expense of managing these cases of peripheral vertigo during the study period.

Chapter 6 provides a brief introduction of the other four disorders namely; vestibular neuritis, migraine, vertigo of central origin and other vertigo (unknown cause) that were observed as the commonly diagnosed conditions at the Christchurch Public Hospital.

Chapter 7 discusses the results and other details of a nationwide survey conducted as a part of this study as far as the diagnosis and management of the included clinical conditions are considered.

Chapter 8 presents the summary and conclusion of the study with possible directions for a future similar study.

Finally, the appendix includes the spreadsheets 1 presenting the systematically organised data to preserve its original/raw wordings or form. Spreadsheet 2 presents the results of the nationwide survey conducted during the study, cover letter/questionnaire used for the same survey. The audiogram and results of the three cases of Ménière’s disease are presented thereafter.
Chapter 2

Method
2.1 Retrospective Study

All the formalities at the Christchurch Public Hospital were completed before starting the process of data collection. Consent was obtained from the Christchurch Public Hospital authorities for accessing the essential patient information (hospital notes and medical records) for the patients admitted with complaints of acute vertigo in the years 2004, 2005 and 2006. No changes or alterations were made in any form as far as the medical records were concerned. Confidentiality of the patient details was maintained throughout the process. The main objective of reviewing these medical records was to gather information about the criteria used for diagnosing these patients.

All the patients admitted to the Christchurch Public Hospital with the diagnosis of vertigo or dizziness in the past two years (2004 and 2005) were considered.

A list of all the patients admitted with presenting complaints of “dizziness and giddiness” for the years 2004 and 2005 to the Christchurch Public Hospital was provided by the ‘Decision Support Department’. There were 518 patients admitted to the Christchurch Public Hospital in the years 2004 and 2005 with the diagnosis of vertigo. These numbers are shown in the table below.
Table 2.1 Data for the years 2004 and 2005

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>2004</th>
<th>2005</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPV</td>
<td>35</td>
<td>29</td>
</tr>
<tr>
<td>Ménière’s Disease</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Vestibular Neuritis</td>
<td>12</td>
<td>7</td>
</tr>
<tr>
<td>Disordered vestibular function unspecified</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Vertigo of central origin</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Other peripheral vertigo</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Dizziness and giddiness</td>
<td>206</td>
<td>186</td>
</tr>
</tbody>
</table>

**TOTAL**                                         | 273  | 245  |

76% (392 of 518) of these patients were diagnosed as cases of “dizziness and giddiness”. As mentioned earlier in Chapter 1, dizziness is a term without any medical significance. Hence, patients diagnosed as cases of “dizziness and giddiness” were excluded from the study as specific information was not obtained from the medical records of patients with such unspecified diagnosis.

However, remaining 126 cases with a confirmed diagnosis were considered for detailed analyse and their medical records were collected to check the entirety of initial clinical
evaluation, accuracy of the diagnosis and treatment. These patients were divided in six different categories according to their final diagnosis.

The six categories of diagnoses were as follows:

- BPV
- Ménière’s disease
- Vestibular neuritis
- Migraine vertigo
- Vertigo of Central origin
- Other i.e. no confirmed diagnosis

The following information was recorded in each case. Collected data were organised in the form of a spreadsheet (spreadsheet 1, presented in the appendix), which included several columns as described below.

1) Case code, 2) Sex, 3) Age at admission, 4) Month of admission, 5) Length of hospital stay, 6) Admitting department, 7) Investigations (CT/MRI scan if performed), 8) Diagnoses, 9) General notes details, 10) ENT referral made or not, 11) Audiology referral made or not, 12) Comments/Summary, 13) Diagnostic accuracy, 14) In patient referral to ENT/Audiolgy.

Four digit code numbers were allocated to each case, where the first two digits represent the year when the patient was admitted. The same procedure was carried out for the year 2005.

2.1.1 Significance of collecting this information
Information on sex was collected to compare the male-female proportion according to each peripheral vestibular pathology as many vestibular disorders are known to be gender biased.

Month and length of hospital stay was important, as one of the prime objectives of this research was to find out if there had been any adverse effects over the length of hospital stay because of the suspected errors in the initial clinical evaluation of the patients admitted with complaints of dizziness.
Information on admitting department was crucial as many patients with complaints of acute vertigo were admitted mainly to the Department of General Medicine, Neurology or Emergency Medicine leading to the delayed Otolaryngology/Audiology intervention. The information gathered was used to see the difference that could be made by avoiding this delay from the admitting departments.

Missing the diagnosis like BPV or Ménière’s disease with incomplete clinical evaluation could have led to a head CT or MRI scan that was not necessary and hence inspecting the appropriateness of such investigations in 2004-2005 was important.

The patient notes for the year 2004 were collected from the Clinical Records Department. General as well as ENT/Audiology files (if available) were reviewed for each patient. In every case, records were examined for a written amount of the presence or absence of nystagmus, a temporal description of on-going nystagmus and for a coherent description of provocative positional tests. Recording information on in-patient referral to ENT/Audiology was vital for the study as this area was expected to show improvement with the changes implemented in 2006.

Every entry and conclusion was checked by the available clinician. Those patients, whose admissions were not due to the complaints of vertigo primarily or those admitted for some other reason like chest pain or an accident resulting into a fracture etc. and vertigo was a coincidental finding, were excluded. The information gathered for such patients was not representing any peripheral vestibular pathology. At the end of this process, 63 patients from
the year 2004 and 53 patients from the year 2005 were selected, making a total of 116 patients being selected out of the 126 considered from the past two years.

2.2 Prospective Study

A prospective study was planned and completed in the year 2006. The aim was to identify patients coming in with the presenting complaints of acute vertigo, in a hope that implementing Otolaryngology/Audiology management could improve the diagnostic precision with fewer expensive investigations (CT/MRI scans) and reduce hospital stay.

The initial proposal was for the investigator to attend (with the Otolaryngology Registrar on-call) the arrival of every vertiginous patient in the Emergency Department and the admission of the patient under Otolaryngology (unless there was a compelling contrary reason) and perform videooculography (details discussed in chapter 6, audiogram and note other clinical test results in this initial clinical evaluation. Unfortunately, financial constraints made this approach impractical, and so an alternative protocol was used. Acute vertiginous patients were admitted as per previous “customs”. On a ‘goodwill’ basis, the Clinical Director of Medicine undertook to arrange that, for the investigation period, Otolaryngology/Audiology would be notified as soon as possible, so the patient could be reviewed there. This was usually in the form of a written referral faxed to the department.

In this protocol, it was not possible to perform videooculography. However, doctors were informed about the research project and requested to refer the patient to the Department of Otolaryngology/Audiology as early as possible.
To reach the equivalent number of patients for a six-month period to that of the previous two years, the 2006 data collection period was extended from six to seven months (May to November 2006). The data for 32 patients were collected over this seven-month period. Five patients were excluded from this research project, either for not having vertigo as their presenting complaint or having vertigo as the coincidental finding with some other primary diagnosis. Thus, a 27-patient data were collected over the period of seven months. One particular patient had two separate admissions with presenting complaints of vertigo. These admissions were considered separately, making a total of 28 patients for the year 2006.

For 2004 and 2005, the diagnosis analysed was the “official” hospital diagnosis, which might have been made in a number of Departments. In contrast, the 2006 diagnosis presented in this study was made by a clinician from the department of Otolaryngology/Audiology and doubly checked by the supervisor. The data were arranged in the same form of a spreadsheet (spreadsheet 1, presented in the appendix) as that of 2004-2005, which also listed for each case;

1) Case code, 2) Sex, 3) Age at admission, 4) Month of admission, 5) Length of hospital stay, 6) Admitting department, 7) Investigations (CT/MRI scan if performed), 8) Diagnosis, 9) General notes details, 10) ENT referral made or not, 11) Audiology referral made or not, 12) Comments/Summary, 13) Diagnostic accuracy, 14) Number of days seen after admission, 15) In patient referral to ENT/Audiology.

Number of days seen after admission was the column added for the year 2006, to verify the effect of early referral to the Department of ENT/Audiology on diagnostic accuracy.
2.2.1 National Questionnaire/Survey
In an attempt to ascertain the admission patterns of acute vertiginous patients in other New Zealand DHBs (District Health Boards), a brief open-ended questionnaire was sent to the Otolaryngology Department of 21 DHBs. The questionnaire is presented in the appendix and the results (tabulated in the spreadsheet 2 presented in the appendix) are discussed in chapter 7.
Chapter 3

Benign Positional Vertigo
3.1 Benign Positional Vertigo (BPV)

The background information on BPV has been discussed in Chapter 1. BPV is one of the two primary disorders considered for this study to verify the improvement in the diagnostic accuracy and the overall management. Hence, the accepted or standard clinical methods of diagnosing or treating BPV and the results obtained are discussed below.

3.2 Diagnosis

Developing a systematic method of diagnosis of BPV was one of the milestones of this study. There was a speculation that diagnostic tests necessary for proving this condition were not routinely performed at the Christchurch Public Hospital and hence the diagnostic accuracy for BPV was affected. Following tests/methods were used for diagnosing and treating BPV at the Department of ENT/Audiology of the Christchurch Public Hospital during the study period in 2006.

3.2.1 Frenzel (magnifying) glasses

“Frenzel glasses” were used for diagnosing every case of BPV in 2006 as this helps avoid missing mild or momentary nystagmus during a positional test. These glasses prevent fixation of vision, which could reduce the intensity of nystagmus due to peripheral vestibular pathology (Personal Communication, Hornibrook, 2006).

Figure 3.1 Diagrammatic presentation of the Dix Hallpike manoeuvre (right ear BPV)

Ref.: (Parnes, Agrawal, & Atlas, 2003)
3.2.2 Dix Hallpike Test (anterior and posterior canal variant)
Dix Hallpike test was first described by Dix M. R. and Hallpike C. S. in 1952 and has major significance in diagnosing BPV (Parnes, Agrawal, & Atlas, 2003).

Patient is seated on the examination table in a way that patient’s head will extend over the edge of the table when supine (Position A). Patient’s head is turned 45º towards the ear being tested. Then patient is quickly lowered into supine position so that patient’s head could be extended approximately 30º below the horizontal (Position B). Then the patient is made to return to the sitting position (Position A) and eyes are observed for the presence of reversal of nystagmus (Parnes, Agrawal, & Atlas, 2003). This test is sometimes called a “tilt test” or “Nylen-Barany test” (Kovar, Jepson, & Jones, 2006).

3.2.3 Roll Test (horizontal canal variant)
Lateral head turns were first reported by McClure in 1985. Symptoms are produced by “Roll test”, where head is fixed at 30º of flexion in supine position and turned to one side at 90º and then rolled to the opposite side while the nystagmus is being observed (Çakır et al., 2006).

Table 3.1 Comparison of the nystagmus in the three variants of BPV (Çakır et al., 2006)

<table>
<thead>
<tr>
<th>Anterior</th>
<th>Posterior</th>
<th>Horizontal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Torsional with fast phase away from the ground (ageotropic)</td>
<td>Torsional with fast phase towards the ground (geotropic)</td>
<td>In horizontal plane as the head is turned from side to side, is horizontal-rotatory, usually geotropic but may be ageotropic</td>
</tr>
</tbody>
</table>

A study on 169 patients with BPV reported incidence of posterior and anterior canal variant at 85.2% and 1.2% respectively (Çakır et al., 2006). Incidence of horizontal canal variant has been reported to be between 1.9% and 22% (Eisensehr, Noachtar, Strupp, & Lindeiner, 2001).
Vestibular Evoked Myogenic Potential (VEMP) is a non-invasive test that checks the function and integrity of the saccule and the inferior vestibular nerve by recording inhibitory myogenic potential from the sternocleidomastoid muscle (Zhou & Cox, 2004). In a study on 25 patients with BPV, the results indicated that 30% of the ears affected with BPV had abnormal VEMP (Akkuzu, Akkuzu, & Ozluoglu, 2006).

3.3 Treatment

Even though it is commonly believed that BPV could be a self limiting condition, unfortunately that is not the case. A study carried out on 82 patients with BPV reported that 20% (16 out of 82) patients experienced symptoms from 1-10 years (Yakinthou, Maurer, & Mann, 2003). The manoeuvres described below were used to treat the cases diagnosed with BPV during the study period in 2006 at the Christchurch Public Hospital.

3.3.1 Epley manoeuvre

This is the most effective treatment for posterior canal BPV with 80% success rate after a single treatment and 100% success rate with repeated treatments (Swartz & Longwell, 2005).

![Diagram of Epley manoeuvre]

Figure 3.2 Diagrammatic presentation of Epley manoeuvre  Ref.: (Hain, 2006b)

The Epley manoeuvre is described below.
A. Patient is made to sit with back straight and head tilted forwards and slightly downwards and is hold in that position for 30 seconds.

B. Then the patient is made to lie on the back with head tilted around 45º to the right and extended 20º backwards from the edge of the table with physician supporting the head all the time and hold for 30 seconds or until the nystagmus stops.

C. Head is tilted at 45º to the other side and hold for 30 seconds or until the nystagmus stops.

D. Then patient’s body is tilted on one side in the direction of the head with maintaining the 45º angle between head and body still; so effectively now the nose is touching the bed with body lying on one side and hold for 30 seconds or till nystagmus stops.

E. Then patient is made to sit up again with head tilted forwards and downwards and nystagmus is observed if present (Hain, 2006b).

3.3.2 Semont (liberatory) Manoeuvre

As shown in the figure 3.3 above, the patient is laid on the side of the involved ear with head slightly declined and pointing in the air, 45º from the horizontal (Position 2). If the nystagmus appears, wait until it stops and keep the patient in the same position for 2-3 minutes after that. Then patient’s head and neck is held in both hands and quickly swung to the opposite side (Position 3). Usually rotatory nystagmus appears rolling towards the sick ear. Patient is kept in that position for 5 minutes and then brought back to the sitting position (Yakinthou, Maurer, & Mann, 2003). Success rate of Semont manoeuvre according to Beynon (1997), varies between 54-93% (Fife & FitzGerald, 2005).
3.3.3 Barbecue rotations
The treatment used at the Christchurch Public Hospital during the study period for the horizontal canal variant, was called “Barbecue rotations”. In this procedure, the patient with geotropic nystagmus was assisted in performing a 360° (contralateral) rotation away from the symptomatic ear with the head as flexed as possible. If the nystagmus was ageotropic, a reverse direction rotation was performed (Hornibrook, 2005). Horizontal canal variant of BPV could be treated using the Appiani and Casani manoeuvres, but these manoeuvres are rarely performed (Roberts, Gans, & Kastner, 2006). A delayed reaction in the form of strong falling sensation might be experienced by few on undergoing the Epley manoeuvre. Hence keeping the patient under observation for some time after performing any of these manoeuvres would be recommended (Kovar, Jepson, & Jones, 2006).

3.4 Results and Discussion

3.4.1 Results
53% cases (61 of 116 patients) were diagnosed as cases of BPV during the years 2004 and 2005. In 2006, this percentage dropped to 36% (10 of 28 patients). In terms of diagnostic accuracy, analysis of the available clinical notes discussed below, led us to conclude that only 31% of the diagnoses of BPV made during 2004 and 2005 were well supported by clinical evidence (19 of 61 diagnoses). Statistically, this was significantly\(^1\) lower than 2006, where 90% cases (9 of 10 patients) diagnosed as having BPV were well supported by the clinical evidences. This improvement in the diagnostic accuracy and the consequent reduction in the number of cases diagnosed as cases of BPV are discussed below.

3.4.2 Discussion
Initially it was assumed that an increase in diagnostic accuracy would result in increased number of BPV cases, due to the perceived under-diagnosis of this condition. Oghalai, Manolidis, Barth, Stewart, & Jenkins (2000) found in a study that 9% of a sample of older adults had undiagnosed BPV (Kovar, Jepson, & Jones, 2006). Another study carried out on

\(^1\) Chi-square test: Chi-square= 15.022 with 2 degrees of freedom. (P = <0.001)
20 patients with the posterior canal BPV, reported that 17/20 (85%) patients could have been easily diagnosed if Dix Hallpike test would have been carried out. However, the Primary Care Physician missed the diagnosis for all these patients (Fife & FitzGerald, 2005).

In this study at the Christchurch Public Hospital, the percentage of BPV cases had decreased. Even though there was an overall decrease in the percentage of cases diagnosed as BPV, multiple factors could be responsible for this decrease.

3.4.2.1 Cases with the diagnostic accuracy ‘No’ in 2004-2005
The first important factor was insufficient or in many cases practically no evidence supporting the diagnosis of BPV for many cases seen in the years 2004 & 2005. This conclusion was reached, as on detailed review of all the 61 patient files, there were 28 cases with no evidence of Dix Hallpike test or any positional test performed to support the diagnosis. In many of these cases, the notes reported that there was no nystagmus as well and still the diagnosis was BPV. The diagnostic accuracy for these cases had been labelled ‘No’.

There were 10 cases in which Dix Hallpike test was performed and reported as “negative” at some stage by a clinician, but were still diagnosed as cases of BPV. Such cases had been grouped under the heading ‘No’ while considering their diagnostic accuracy. Vertigo due to BPV is accompanied by the rotatory nystagmus. The duration of nystagmus is usually 5-20 seconds but it could reappear when the patient is brought back to the sitting position after the Dix Hallpike test (Yakinthou, Maurer, & Mann, 2003). Several studies show that maintaining certain norms for diagnosing BPV are necessary.

A study of geriatric group of people included 47 participants with posterior canal BPV. The selection criteria followed was “complaints of vertigo provoked by changes in the head
position, lasting less than 1 minute and accompanied by the typical vertigo and nystagmus of
the posterior semicircular canal BPV during the Dix-Hallpike test using “Frenzel glasses”
(Angeli, Hawley, & Gomez, 2003). Hence looking for the specific nystagmus, type and
duration of vertigo, conducting the Dix Hallpike test should be considered before diagnosing
each case of BPV. These criteria for diagnosis of BPV seemed to be lacking for many cases
in 2004-2005. Few cases have been discussed below to support these findings.

Case 0412 represents a 74-year-old woman admitted to the Department of General Medicine
of the Christchurch Public Hospital in August 2004. This patient was diagnosed as a case of
BPV without an Otolaryngology or Audiology consultation during her stay in the hospital.
She visited the Department of ENT within 6 weeks following this admission where she was
noted to have a reduced vestibular function on the right side.

The symptoms of this case presented a likely diagnosis of ‘vestibular neuritis’. As far as
hospital records were concerned, the case was diagnosed as BPV. This case showed lack of
thorough clinical evaluation of a patient leading to an inaccurate diagnosis. Another case
with similar errors has been given below.

Case 0507 represents an 81-year-old woman admitted to the Department of General
Medicine in April 2005 with symptoms of vertigo provoked by any movement. The hospital
discharge notes indicated ‘presence of some of the symptoms induced by the Dix Hallpike
test’ but clinical notes gave no specific description of the findings as the supporting evidence
for the final diagnosis of BPV.

The data collected regarding the clinical evaluation carried out during this woman’s
admission were not complete or satisfactory to conclude the final diagnosis of BPV. The
diagnostic accuracy for this case had also been labelled ‘No’ because when Dix Hallpike test
was used to diagnose the presence of BPV, notes should clearly indicate what positive
findings were seen to call it a positive Dix Hallpike test. Several studies indicate the importance of set protocols or criteria to maintain the diagnostic accuracy.

A study on 23 patients with unilateral posterior semicircular canal BPV was conducted to investigate the balance ability of patients with BPV. The diagnostic criteria used for this study included the presence of positional vertigo and a typical torsional upbeating (geotropic) nystagmus provoked by the Dix Hallpike test. The provoked nystagmus was considered only if it was transitory (<60 seconds), had delayed onset (latency = 2-40 sec) and reversed while the patient resumed the original sitting position (Chang, Hsu, Yang, & Wang, 2006). Such specific clinical evidences should be considered that form the basis of accurate diagnosis.

3.4.2.2 Cases with diagnostic accuracy ‘Possible’ in 2004-2005
The diagnostic accuracy for two other patients (case 0429 & 0527) had been labelled ‘Possible’. The first case has been described below.

Case 0429 represents an 85-year-old man admitted to the Department of General Medicine with symptoms of vertigo on sitting and relieved by lying. Clinical notes indicated presence of subjective vertigo with ‘positional non-sustained nystagmus’ as the objective finding.

No explanation about the positional tests carried out was given and the patient was not referred to the Department of ENT or Audiology. The second case has been described below.

Case 0527 represents a 20-year-old woman admitted to the Emergency Department with symptoms of vertigo, nausea and vomiting in May 2005. Clinical notes reported presence of a 4-year history of BPV following head injury. Notes also reported that a private ENT Consultant in the St. George’s Hospital, Christchurch had performed the Dix Hallpike ‘manoeuvre’ (test), which was successful.
There was no reference in the notes regarding the nystagmus. Patient was not tested for BPV with the help of any positional test to confirm the diagnosis. For both cases mentioned above, there was no satisfactory clinical evidence that confirmed the diagnosis of BPV during their hospital stay. The reference of a private ENT Consultant was provided for the second case but no positional tests were carried out to confirm the diagnosis and hence the final diagnosis of BPV could not be justified. For such cases, the diagnostic accuracy was somewhere between ‘Yes’ and ‘No’ as the symptoms were that of BPV. The term ‘Possible’ has been used to describe the diagnostic accuracy for such cases. The diagnosis of BPV must include all the necessary supporting clinical findings. Cases with one or two symptoms without a complete clinical picture of BPV should not be considered as cases of BPV.

A study carried out at the “Department of Otolaryngology–Head and Neck Surgery”, Tulane University, New Orleans, Louisiana, USA included 201 patients with peripheral vestibular symptoms indicative of BPV (Belafsky, Gianoli, Soileau, Moore, & Davidowitz, 2000). For each patient, the diagnosis of BPV was confirmed by examination with “Frenzel lenses” and electrooculography (EOG)\(^2\) assisted Dix Hallpike testing during electronystagmography (ENG)\(^3\). Any patient with a history of positional vertigo and a negative Dix Hallpike test was excluded from the study. This study highlighted the necessity of a complete clinical presentation before coming to the final diagnosis of BPV.

3.4.2.3. Diagnostic accuracy affected due to improper clinical evaluation in 2004-2005
In the years 2004 and 2005; 19 cases of BPV have been considered with the diagnostic accuracy ‘Yes’ indicating that the diagnosis was well supported by clinical evidences. Thus,

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\(^2\) EOG: Recording of the average amplitude of the resting potential arising between the cornea and the retinal in light and dark adaptation as the eyes turn a standard distance to the right and left (Dark, 2003a).

\(^3\) ENG: A method of recording the eye movements by measuring the minute changes in the electrical potentials associated with the eye movements.
out of 61 cases diagnosed as BPV, there was a diagnostic accuracy of only 31%, which was not satisfactory and was mostly due to lack of supportive clinical evidences. It is recommended that physical examination for a patient with complaints of vertigo should include orthostatic vital signs and Otoscopic examination. The Dix Hallpike test is vital in differentiating central from the peripheral cause of vertigo (Swartz & Longwell, 2005). Patients suffering from vertigo need to be diagnosed accurately as soon as possible to avoid other adverse consequences. Oghalai et al. (2000), found that patients with unrecognized BPV are likely to have a history of falls; have reduced activity, independence, and quality of life; and are more likely to have depression (Kovar, Jepson, & Jones, 2006).

3.4.2.4 An overall decrease in the number of BPV cases in the year 2006
In 2006; the percentage of BPV cases had been lowered as compared to the data from the past two years but the diagnostic accuracy was much higher as compared to the past two years. The data for 2006 showed that there were 10 cases of BPV out of 28 patients. This was only about 36% cases of BPV in 2006 as compared to the data from the past two years (2004 & 2005) indicating 53% BPV cases. This decrease of 17% could be due to various factors described below.

This was considered to be the effect of complete clinical evaluation carried out in 2006. In 2006, every other department of the Christchurch Public Hospital was informed about the research being conducted and performing positional tests like Dix Hallpike test before deriving any conclusion regarding the diagnosis of BPV was highly recommend. This implemented a uniform criterion in place indicating not to diagnose any patient with symptoms of mere vertigo or presence of nystagmus as a case of BPV, if the supporting clinical evidence of positional tests was missing. This resulted in a decrease in the total percentage of BPV cases for 2006, which seemed to have been inflated inappropriately in the past two-year’s (2004-2005) data because of deriving the diagnosis of BPV to cases without substantial clinical evidences. For many cases in 2004-2005, the presenting symptoms
included mere vertigo with or without other subjective symptoms but no objective symptom like presence of nystagmus in any form were available to support the diagnosis. For example, past clinical records for the cases 0417, 0502 showed that there was no nystagmus noted in their clinical notes. Dix Hallpike test or any other positional test was not performed, patients were not referred to the Department of ENT/Audiology and yet the final diagnosis was BPV.

3.4.2.5 Inaccurate interpretation of the Dix Hallpike test in 2004-2005
There were many cases where Dix Hallpike test was negative as per the clinical notes but still the patients were diagnosed as the cases of BPV. For example, clinical notes of cases 0514, 0515, 0516 reported that Dix Hallpike test was negative. Cases 0514 and 0515 were never referred to the Department of ENT/Audiology.

Case 0516 represents a 74-year-old man admitted to the Department of General Medicine in August 2005. His symptoms reported were dizziness with unsteadiness on his feet, nystagmus looking to the left and a negative Dix Hallpike test.

This man was referred to the Department of ENT from the Emergency Department where negative Dix Hallpike test results were re-checked and confirmed by the ENT clinician reporting that the findings were same as that of the Emergency Department. However, these three cases were diagnosed as cases of BPV. Such errors in the diagnosis of BPV cases should be avoided. Belfasky et al. (2000) have mentioned that the “gold standard” for diagnosing BPV is the Dix Hallpike test and the results are considered positive only if the position elicits rotary nystagmus and vertigo that last for less than 1 minute, are fatigable and are associated with a several-second latency.

3.4.2.6 Curative CRP performed without the Dix Hallpike test in 2004-2005
It was observed in the past two year’s (2004 & 2005) data that on few occasions clinicians had performed the CRP like the Epley Manoeuvre without conducting the Dix Hallpike test to confirm the diagnosis of BPV. If there was no supporting clinical evidence in the form of a
Dix Hallpike test for the diagnosis, performing curative CRP was not appropriate. Following case represents such findings.

*Case 0520 represents a 46-year-old woman, who came to the Emergency Department with complaints of vertigo, nausea and vomiting after she woke up in the morning. Nystagmus was observed by the clinician.*

The clinical notes did not give any more information on the nystagmus or positional tests carried out to diagnose the case as BPV. However, notes reported that the Epley manoeuvre was performed. No further information about the effect of the Epley manoeuvre or follow up of this patient was provided. The diagnostic accuracy for this particular case was labelled ‘No’ indicating incomplete clinical findings. Epley manoeuvre should not be performed without a confirmed diagnosis of BPV as it could lead to several complications.

Factors like neck or back problems, hypertension or obesity should be considered before performing the procedure like the Epley Manoeuvre. The clinician should be absolutely certain of the diagnosis before conducting such a procedure. Worsening of symptoms due to “Canalith jam” may result. Here the canaliths may be lodged or trapped in the narrow crus of semicircular canal or between the wall of ampulla and cupula. Conversion to horizontal or anterior canalithiasis following the Epley manoeuvre could be seen due to entry of particles in respective canals (Epley, 1997).

Patients are known to have even severe attack of vertigo leading to fall after such manoeuvres. One particular study involved 436 Epley manoeuvres being carried out in total (Uneri, 2005). 13% of these (58 episodes) patients had a strong falling sensation. 9 patients almost fall off the examination table despite firm support from several people. One patient
experienced such severe sensation, 30 minutes after the procedure and the experience was very strenuous for both the participants and the clinicians. Hence, performing CRP without confirming the diagnosis of BPV should be avoided in future.

There was a slim possibility here that the clinician might have performed the Dix Hallpike test but has not mentioned in the clinical notes, but in that case more emphasis should be given in future to mention these important clinical details in the notes that support the final diagnosis of BPV. Thus there were many cases in the past two years (2004 & 2005) that have been diagnosed as BPV but the diagnostic accuracy of such cases was not satisfactory. These should be considered as some of the factors responsible for reduction in the overall percentage of BPV cases in the data collected for the year 2006.

3.4.2.7 Effects of use of the “Frenzel glasses” in 2006
It was observed that in the years 2004-2005, “Frenzel glasses” were not used for diagnosing the cases of BPV. This could be considered as another important factor responsible to improve the diagnostic accuracy in 2006. For every suspected case of BPV seen at the Department of Otolaryngology/Audiology during this study period, “Frenzel glasses” were used before performing the Dix Hallpike test or other positional test. This gave a clearer picture of the type, intensity, direction and duration of the nystagmus following the positional test performed. Using “Frenzel glasses” should be recommended in future at all the departments of the Christchurch Public Hospital before performing positional tests in every suspected case of BPV.

3.4.2.8 Positive effects of supporting clinical evidences in 2006
For all the cases diagnosed as BPV in 2006, the Department of ENT/Audiology notes reported presence of positional tests performed for each patient. Diagnostic accuracy for 9 out of 10 patients was labelled ‘Yes’ indicating the clinical evidence of positional tests supporting the subjective symptoms and the final diagnosis. For example, cases 0601, 0604, 0607, 0608 and 0609 showed presence of nystagmus induced on positional testing with a
positive Dix Hallpike test. Then depending on the type of canal affected from the nystagmus observed curative CRP like the Epley or Semont manoeuvres were applied. Follow up of such cases was important as BPV could persist for years if untreated or remit and recur at varying periods of time in 20-30% individuals (Ford-Smith, 1997). Follow up of all the five patients mentioned above confirmed a negative Dix Hallpike test following the CRP. The manoeuvres carried out as a part of CRP like the Epley manoeuvre takes only few minutes but have an excellent reliability. A study carried out on 20 patients with BPV reported that 75% (15 out of 20) patients were cured by a single Epley manoeuvre and 25% (5 out of 20) were cured by two Epley manoeuvres (Fife & FitzGerald, 2005).

For other four cases (0602, 0603, 0605 and 0610), the Dix Hallpike test was positive. CRP was performed and patients showed improvement with their vertigo. These patients were then informed about the research and were requested to report to the Department of ENT/Audiology in case of recurrence of any of the symptoms of vertigo. None of these patients returned indicating that they were cured of the symptoms and hence the diagnosis of BPV was confirmed for all these patients. BPV is thought to be a self limiting condition by some but there are several studies explained earlier, which prove that the condition might recur with time and can remain persistent for years if not treated in time (Epley, 1992). CRPs are quick to perform and has proven success rates. Epley (1996) reported a success rate of 100% and Semont et al (1988) reported a success rate of 84% for Epley and Semont manoeuvres respectively (Uneri, 2005). Duration of such conditions could be prolonged if a patient is not diagnosed accurately and left untreated.

The impact of a prolonged duration of such a condition could be severe in terms of general quality of life and at other levels. Lopez-Escamez, Fiñana, Fernandez, Gámiz, & Sanchez-Canet (2003), conducted a study including 37 patients diagnosed with posterior semicircular
canal BPV presented the intensity of such adverse effects on life. “The Medical Outcomes Study 36-item Short-Form Health Survey (SF-36)” is a general health measurement widely used in studies of the outcome for chronic diseases. The results of SF-36 were compared to the Dizziness Handicap Inventory Short Form (DHI-S), which was used for measuring the impact of posterior canal BPV on the quality of life. Only one CRP was performed in this study. Scores of SF-36, one day before and 30 days after performing the CRP were plotted against the general population results. There was a significant improvement in scores 30 days after performing the CRP. This was tested at various levels like physical functioning; role-physical; body pain; general health; vitality; social functioning; role emotional and mental health. Patient showed significant improvement at all levels except one. This study showed that BPV could affect person’s life significantly if left untreated but with CRP, this could be avoided. Even the scores on DHI-S decreased from 18.05 on 1 day before CRP to 9.54; 30 days after CRP (Lopez-Escamez, Fiñana, Fernandez, Gámiz, & Sanchez-Canet, 2003 ). These scores show that BPV is not a benign or self-limiting condition but it has serious effects if left untreated. The importance and effectiveness of various positional tests and manoeuvres that needed to be implemented on time for patients with BPV was evident here.

Only one case (0606) was labelled the diagnostic accuracy ‘Possible’ in 2006, indicating that there was a possibility of slightest doubt in this case due to some variation from that of the normal findings of the Dix Hallpike test.

This was a 48-year-old female patient, who presented to the Emergency Department with sudden onset of vertigo with nausea, vomiting and vertigo triggered by any head movement. This was a possible case of BPV. On examination at the Department of ENT/Audiology, Dix Hallpike test showed a latency of 30 to 40 seconds with nystagmus on the left gaze lasting for 6-8 seconds only. Clinician reported that this could be possibly due to the fatigue of the vestibulo-ocular nervous system that there was no clear nystagmus present. Epley CRP was performed but there was no improvement with the symptoms.
Even though most symptoms of BPV were present, this woman’s diagnostic accuracy had been labelled ‘Possible’ as the clinical finding were not fully convincing. This case was described to show that the slightest inconsistencies over the clinical findings were taken into consideration in 2006 and the diagnostic accuracy had been dropped from ‘Yes’ to ‘Possible’. At the end of the data collection process in 2006, the diagnostic accuracy for BPV was 90%. As mentioned above, the diagnostic accuracy for the past two years (2004 & 2005) was 31%. Thus, by implementing recommended changes in the initial clinical evaluation for the year 2006, a significant increase of 59% was achieved in the diagnostic accuracy of BPV.

3.4.2.9 Early Otolaryngology/Audiology referrals avoids unnecessary scans
Comparing two more cases (case 0607 & case 0601) from the year 2006, represents how the MRI scan could have been avoided by early referral of such patients to the Departments of ENT and Audiology.

Case 0607 represents a 68-year-old woman admitted to the Department of General Medicine in April 2006. She had an acute attack of vertigo with light-headedness and momentary episodes like that of “jolts” as described by the patient. History indicated that she was seen by a private ENT Consultant before 8 days. She was referred to the Departments of ENT and Audiology; 5 days after admission to the hospital. Meanwhile she had a MRI scan during her 6 days of stay within the hospital, which was ‘normal’. On 5th day, the ENT consultant carried out the Dix Hallpike test that was positive indicating posterroan canal BPV. Then two Epley CRPs were carried out. Pure tone audiometry revealed bilateral mild to moderately severe sensorineural hearing loss. The vertigo had stopped and subsequent Dix Hallpike test in the follow up was negative.

The second case is discussed below.

Case 0601 represents a 93-year-old woman admitted to the Department of General Medicine with symptoms of acute vertigo leading to a fall. Patient was referred to the Departments of ENT and Audiology on the same day. Pure tone audiometry revealed slight low frequency sloping to severe high frequency sensorineural hearing loss on left and moderate low sloping to profound high frequency sensorineural hearing loss on the right side. Dix Hallpike test was carried out on suspecting the presence of BPV, which was positive with horizontal direction changing geotropic nystagmus indicative of the horizontal canal BPV. She was

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4 Chi-square test: Chi-square= 15.022 with 2 degrees of freedom. (P = <0.001)
treated with two Barbecue rotations. Her vertigo disappeared and the subsequent Dix Hallpike test carried out on her follow up was negative indicating that she was cured.

Both patients discussed above had similar complaints and in fact the second patient was much older, 93-year-old woman with more chances of possibility of stroke or other serious cause for the vertigo but since she was referred to the Department of ENT and Audiology on the same day of admission, no head scan like CT or MRI scan was carried out. Whereas the first patient had an unnecessary MRI scan as she was referred to the ENT/Audiology on the 5th day after admission and there was a delay in ENT/Audiology intervention. Thus, unnecessary investigations (scans) were avoided and overall hospital stay was reduced by early referral or timely interventions by the Department of ENT/Audiology.

Early Audiology intervention for all suspected cases with complaints of vertigo is essential as complete audiometric testing could help distinguish vestibular pathology from retrocochlear pathology. “American College of Radiology” recommends MRI with contrast medium when a patient presents with acute vertigo and sensorineural hearing loss (Swartz & Longwell, 2005). Thus, timely audiologic evaluation could prevent unnecessary scans.

3.4.2.10 Coding errors
Some other cases like case 0511 were included in the group labelled ‘No’ as far as their diagnostic accuracy was concerned due to the probable ‘coding error’.

Case 0511 represents a 74-year-old man admitted to the Department of General Medicine in August 2005. He presented with complaints of vertigo and nausea exacerbated by head movements.

The clinical records for this case had clinician’s notes stating that it was not a case of BPV. Notes reported that there was no nystagmus and the Dix Hallpike test carried out was negative but the Clinical Coder had generated an error diagnosing this patient as a case of BPV. Such
cases have been misdiagnosed as BPV and have inflated the number of cases grouped under the diagnosis of BPV for past two years (2004-2005). This could be considered as another factor responsible for increasing the percentage of cases diagnosed as BPV in the past two years than the year 2006.

3.4.2.11 Incidental errors of inappropriate medical terminology
During the data collection process for past two years (2004 & 2005), few incidental errors were noted as far as appropriateness of medical terminology used in relation to the BPV. In many patients’ clinical notes the Dix Hallpike test was referred as the Dix Hallpike ‘manoeuvre’. For example, in case 0421, clinician had reported that the Dix Hallpike ‘manoeuvre’ was negative. The same error had been noticed occasionally in these past medical records. Even though many people use the term ‘Dix Hallpike manoeuvre’; “Dix Hallpike test” is a diagnostic test for BPV and should be referred to as a test and not a manoeuvre (Personal Communication, Hornibrook, 2006).

One more error noticed was that of referring the Dix Hallpike test as a ‘curative procedure’ in the past medical records for 2004-2005.

Case 0527 represents a 20-year-old woman admitted to the Emergency Department in May 2005. Her clinical notes report that a private ENT consultant had performed the Dix Hallpike ‘manoeuvre’ twice and it was ‘successful’ indicating that she was treated.

Such errors in the medical terminology in hospital clinical notes could be misleading and should be avoided.
Chapter 4

Ménière’s Disease

(Idiopathic Endolymphatic Hydrops)
4.1 Ménière’s disease

Ménière’s disease is the other major disorder considered for this study to verify the overall improvements in the diagnostic accuracy and its management. The methods used for investigating or diagnosing the Ménière’s disease and its treatment have been discussed in brief here. The results obtained in 2006 on implementing the recommended changes have been reported and discussed in comparison to the previous two years.

4.2 Diagnostic Investigations

The standard investigations used for the diagnosis of Ménière’s disease are discussed below.

4.2.1 Electrocochleography (ECochG)

ECochG is performed mainly by two methods extratympanic (ET) and transtympanic (TT) ECochG depending on whether the electrode is placed outside or through to the tympanic membrane respectively (Noguchi, Nishida, & Komatsu, 1999). The summating potential (SP)\(^5\) is larger and more negative in patients with Ménière’s disease and is thought to be due to distension of the basilar membrane into the scala tympani, causing an increase in the normal asymmetry of its vibration. (Schessel, Minor, & J Nedzelski Chapter 142, 2004).

Figure 4.1 ECochG  Ref.: (Kakigi et al., December 2003).

\(^5\) SP: The direct current (DC) potential produced in the cochlea only when the sound stimulus is present (Kim, Kumar, Battista, & Wiet, 2005).
Figure 4.1 represents ECochG of the left ear, a typical pattern of SP/AP ratio increase and SP/AP ratio measurement. The methods of measuring SP and AP are indicated and SP/AP ratio was 0.42.

The SP and the eighth cranial nerve action potential (AP) ratio is considered to reduce the inter-test variability. Thus the ratio of SP/AP increases in case of hydrops. Some studies also recommend measurement of the amplitude of the summating potential recorded transtympanically i.e TT ECochG (Schessel, Minor, & J Nedzelski Chapter 142, 2004). Details of ECochG test are discussed in details later.

4.2.2 Other supporting diagnostic tests
ENG (Electronystagmography) could be used to find out the presence of hypoactivity of vestibular system on the affected side but this might not diagnose the Ménière’s disease (Personal Communication, Hornibrook, 2007). Recording nystagmus using ENG may not always be possible as the nystagmus of Ménière’s attack disappears quickly (McClure, Copp, & Lycett, 1981). VEMP could be used to diagnose the presence of hydrops in Ménière’s disease (Kuo, Yang, & Young, 2005). Ohaki et al (2002) reported the significance of VEMP test to detect the functioning of otolith organs in cases with delayed endolymphatic hydrops (DEH) (Ohki, Matsuzaki, Sugasawa, & Murofushi, 2002).

4.3 Treatment
To date, there is no proven curative treatment for Ménière’s disease. However, dietary modifications and diuretics, enhanced microcirculation of the ear and reduction in immune reactivity with steroids, immunoglobulin and allergy therapy have been used in treating the Ménière’s disease (Schessel, Minor, & J Nedzelski Chapter 142, 2004). Dietary salt restriction or use of diuretics could be used as a maintenance treatment but the effectiveness is not yet proven (Green & Saeed, 2002). A clinical study on 6 participants with glycerol

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6 AP: The sum of synchronous auditory neural potentials in the cochlea (Kim, Kumar, Battista, & Wiet, 2005).
proved that there was a reliable improvement with speech recognition but not with the audiometric thresholds in the Ménière’s disease (Chung, Hall, Buss, Grose, & Pillsbury, 2004).

4.3.1 Other therapies and surgical treatment
Use of pulsed positive pressure applied to the inner ear via an air pump seated in the external auditory canal and a pressure equalisation tube in the tympanic membrane has been suggested to be effective in both alleviating vertigo and improving hearing in patients with Ménière’s disease (Hain, 2004). Conservative surgical treatment for Ménière’s disease includes endolymphatic sac enhancement as the first choice of surgical treatment (Cappello et al., 1995). Sometimes treatment of Ménière’s disease could be placebos (Hain, 2004). Psychotherapy is another valuable therapeutic aid at any stage of Ménière’s disease (Cappello et al., 1995).

“TT ECoChG was the objective test used to prove the presence of hydrops and diagnose every case of Ménière’s disease during the study period in 2006 at the Christchurch Public Hospital; hence the procedures of ECoChG are described below”.

4.4 Electrocochleography (ECoChG) and the Ménière’s disease
The two methods of ECoChG are ET or T ECoChG and TT ECoChG. ECoChG is the key investigation in the diagnosis of Ménière’s disease (Personal Communication, Hornibrook, 2006).

4.4.1 Methods of ECoChG
For conducting the procedure of ET ECoChG, sufficient amount of electrical paste is rubbed onto the contact surface of the active electrode (Noguchi, Nishida, & Komatsuzaki, 1999). The active electrode consists of a stainless steel needle covered with enamel except at the tip (Kakigi et al., December 2003). A specially designed electrode called HN-5 electrode could be used as well. The tip of the electrode is placed in contact with the posteriorinferior rim of
the tympanic membrane (at the tympanic annulus). If the rim could not be visualized, the electrode is directly placed on the posteriorinferior quadrant of the tympanic membrane. For transtympanic ECochG; part of tympanic membrane is locally anesthetised and a fine needle is inserted through this spot on tympanic membrane, which touches the promontory (a bony prominence on the inner wall of the tympanum (Dark, 2003c)) of the inner ear (Schessel, Minor, & J Nedzelski Chapter 142, 2004). Phenol could be used as a local anaesthetic. The needle is usually insulated except for the tip, which acts as an electrode. The tip of this electrode is positioned in the round window niche under microscope guidance (Conlon & Gibson, 1999). A specifically designed electrode called HN-3 electrode is sometimes used. This active electrode is made up of tungsten wire 0.2 mm in diameter and 5.5 cm in length (Noguchi, Nishida, & Komatsuzaki, 1999). The reference electrode is placed on the ipsilateral earlobe and the ground electrode is placed on the forehead. The headphones used are placed in a metal shielded shell and attached magnetically to support a ring positioned over the test ear. Tonebursts at 1 kHz frequency, presented at 100 dB (decibel, a unit to measure sound level) intensity and a rate of 30 per second. It was established after studying 3000 ECochG recordings with tonebursts at 1 kHz that a response exceeding -6 µV is highly indicative of the presence of hydrops (Conlon & Gibson, 1999). Ferraro and Tibbils suggested measuring the area under the SP/AP curve after performing the ECochG to increase sensitivity (Kim, Wiet, & Battista, 2005).

![Figure 4.2 Comparison of normal and abnormal ECochG. Ref.: (Hain, 2006c)]
Figure 4.2 represents a comparison of normal ECochG (left) with an abnormal result showing elevated SP (right), typically seen in cases of hydrops.

### 4.4.2 Significance of ECochG

Patients of Ménière’s disease are known to have longer latencies for AP than others. A study involving 30 patients of definite Ménière’s disease, 11 patients with cochlear hearing loss of other aetiologies and 10 normal subjects proved these results. A very high positive predictive value (100%) of TT ECochG was evident for diagnosing endolymphatic hydrops with combination of condensation-rarefaction shift measurement, SP/AP ratio and 1 kHz SP (Sass, Densert, Magnusson, & Whitaker, 1998). This method could be useful for diagnosing hydrops in patients with complex inner ear symptomatology.

During the study period in 2006 at the Christchurch Public Hospital, toneburst stimuli at 1 and 2 kHz frequency were used using Gibson’s Voltage Criteria (1992). The procedure carried out is described in the results below.

### 4.5 Results and Discussion

#### 4.5.1 Results

12% cases (14 of 116 patients) were diagnosed with Ménière’s disease during the years 2004-2005. In 2006, there were 11% (3 of 28 patients) cases diagnosed with Ménière’s disease. Thus, the final percentage of cases detected for the past two years (2004-2005) and the year 2006 had remained almost unchanged.

As far as the diagnostic accuracy was concerned, analysis of clinical notes for the years 2004-2005 showed that only 21% cases (3 of the 14 patients) diagnosed as having Ménière’s disease were well supported by sufficient clinical evidence. Year 2006, showed a statistically
significant\textsuperscript{7} increase in the diagnostic accuracy to 100\% (3 of the 3 cases) as all the cases were well supported by sufficient clinical evidences. Thus, a significant increase of 79\% had been achieved for the year 2006.

4.5.2 Discussion

4.5.2.1 Inadequate objective clinical tests like audiogram and ECochG in 2004-2005
Symptoms should fulfil the criteria for the diagnosis as discussed earlier in the section on M\'eni\'ere’s disease. A triad of symptoms (namely vertigo, tinnitus, fullness and fluctuating hearing loss) should be present to diagnose a patient with M\'eni\'ere’s disease. In the past two years (2004 & 2005) data; most patients did not fit into this criteria. They might have had some symptoms that mimic M\'eni\'ere’s disease but those were not sufficient for the diagnosis. A study conducted on 243 patients with M\'eni\'ere’s disease to characterize hearing loss, tinnitus and associative factors in the M\'eni\'ere’s disease reported that all patients met the “American Academy of Otolaryngology—Head and Neck Surgery” criteria for the M\'eni\'ere’s disease. Each patient had at least two vertiginous spells lasting for over 20 minutes, audiometrically documented hearing loss in the affected ear, and subjective complains of tinnitus. 38\% cases had full triad of M\'eni\'ere’s disease as their presenting symptoms (Havia, Kentala, & Pyykkö, 2002).

The data for last 2 years showed that on most occasions, objective test (like ECochG) was not carried out to confirm the diagnosis of M\'eni\'ere’s disease. The importance of performing an audiogram and ECochG as a clinical procedure that provides supporting evidence for the presence of M\'eni\'ere’s disease has been discussed earlier in this chapter. A study carried out to investigate the changes in the vestibular nerve of a patient with M\'eni\'ere’s disease included

\textsuperscript{7} Chi-square test: Chi-square= 6.679 with 2 degrees of freedom. (P = 0.035)
5 participants. The criteria for selection were; symptoms of episodic vertigo of at least 20 minute’s duration, fluctuating hearing loss, tinnitus, and aural fullness. The diagnosis was further supported by standard investigations, which included audiometry, electronystagmography and ECochG (Spencer, Sismanis, Kilpatrick, & Shaia, 2002). The study presented the systematic approach used to confirm the diagnosis of Ménière’s disease.

Out of 14 cases diagnosed with Ménière’s disease in 2004-2005; only two cases (0534 and 0537) were supported by objective clinical test like an audiogram or ECochG. None of the other 12 cases have had supporting reference in their clinical notes regarding the audiogram or ECochG carried out at any stage before coming to the final diagnosis of Ménière’s disease. Many cases presented in the spreadsheet 1 from the years 2004 and 2005 had been diagnosed with Ménière’s disease purely on the basis of some references in their past hospital, private consultant or GP notes. Following case has been discussed with similar findings.

Case 0435 represents a 77-year-old woman admitted to the Department of General Medicine in October 2004. She presented with objective vertigo associated with two recent falls due to vertigo. She also had chest pain but there was no nystagmus. She was not referred to the ENT Specialist or audiologist within the Christchurch Public Hospital during her stay at the hospital. No hearing test or ECochG was organised during that admission.

Her clinical notes stated that she was recently seen by a private ENT Consultant but there was no further information. No other objective test was carried out to confirm the diagnosis at the Christchurch Public Hospital.

Case 0437 represents another 59-year-old woman admitted to the Department of Neurology in May 2004. Both CT and MRI scans carried out were normal. Her clinical notes barely mentioned symptoms of two episodes of vertigo and heaviness with headache. She was not
referred to the Department of ENT or Audiology within the Christchurch Public Hospital. There was no record of a hearing test being carried out during her hospital stay.

The only form of supporting evidence for the final diagnosis found in her medical records was a note saying that she was a known case of Ménière’s disease.

Case 0438 was a similar case where an 84-year-old woman was admitted to the Department of General Medicine. She had a normal CT scan. Her clinical notes reported the presence of symptoms of dizziness with two falls. No record of referral to the Departments of ENT/Audiology or a hearing test carried out during her hospital stay was found in her notes. Her clinical notes mentioned that she was a known case of Ménière’s disease and that had been used as a supporting evidence for the final diagnosis in this case.

No objective test was carried out in support of the final diagnosis of Ménière’s disease.

All such diagnoses of Ménière’s disease in the cases mentioned above, were mostly ‘speculative’ and did lack the objective supporting clinical evidences like ECochG to prove the final diagnosis (Personal Communication, Hornibrook, 2006) and hence their diagnostic accuracy was not satisfactory. The diagnostic accuracy for all such cases had been labelled ‘No’.

4.5.2.2 Absence of ruling out the possibility of other peripheral vestibular disorder in 2004-2005
Another important feature to be considered when patients with similar complaints present to a clinician was to rule out the presence of other similar peripheral vestibular pathologies. Symptoms of BPV could mimic that of a Ménière’s disease particularly in an older patient or any other patient for that matter, who is unable to describe his/her symptoms clearly due to the ongoing positional vertigo, nystagmus with mental fatigue and confusion leading to inability to answer the questions clearly, asked by the clinician. Patients with vertigo could
be disoriented, confused, tired or scared of this dreadful experience and are unable to describe the exact picture of the feeling they have (Zimmerman, 2002). It is important to carry out all such objective tests like the positional tests, audiogram or ECochG whenever necessary during the attack if possible. Positional tests should be helpful to distinguish BPV from Ménière’s disease. An audiogram could be helpful to distinguish between vestibular neuritis and other conditions like Ménière’s disease or migraine (Hain, 2006e). Many such symptoms, vital for making a diagnosis could disappear once the Ménière’s attack has subsided or the intensity of the attack has reduced (Personal Communication, Hornibrook, 2006). Pfaltz and Thomsen (1986) mentioned that hearing may fluctuate or decrease during the attack of Ménière’s disease and improve as the attack passes (Havia, Kentala, & Pyykkö, 2002).

Errors (that were easily avoidable) in the early phase of clinical evaluation could lead to absence of objective findings to support the suspected diagnosis. This might result in an inappropriate treatment as the actual diagnosis was missed. Following case has been discussed to elaborate a similar problem.

Case 0531 represents a 61-year-old male patient admitted to the Department of General Medicine with presenting symptoms of vertigo, nausea, vomiting, tinnitus and gait disturbances. There was nystagmus on looking to the left as an objective finding. A note saying history of inner ear problems since last one year without giving any more specifications was found in his clinical notes. This man had a CT scan of the head during this admission, which was essentially reported “normal” with incidental finding of a small subarachnoid cyst in the right middle cranial fossa.

Even though the clinical notes reported that he was advised to see the ENT Specialist in case of any future problems, he was not referred to the Department of ENT/Audiology during his hospital stay. This could have been a case of BPV. It was necessary to perform the positional
tests to exclude the possibility of BPV first and then perform some objective clinical tests like ECochG, audiogram to prove the presence of endolymphatic hydrops or hearing loss respectively in support of the possible final diagnosis of Ménière’s disease.

4.5.2.3 Importance of early referral to the Department of ENT/Audiology
Out of the 14 patients diagnosed as having Ménière’s disease in the past two years (2004 & 2005); only 2 patients (Case 0433, 0533) were referred to the Department of Audiology during their hospital stay. This rate of referral to the Audiology was extremely low for the total number of cases. In order to confirm the diagnosis of Ménière’s disease, type and grade of hearing loss during the attack forms one of the prominent characteristic for this diagnosis as explained earlier in the details of Ménière’s disease. Fluctuating and progressive low frequency hearing loss is the characteristic of early stages of Ménière’s disease (Schessel, Minor, & J Nedzelski Chapter 142, 2004). A research carried out with a database of 243 patients with Ménière’s disease proved that 44% had fluctuating hearing loss and hearing deteriorated more during the vertigo attack if the attack was intense (Havia, Kentala, & Pyykkö, 2002). Ménière’s disease is also known to have adverse effects on the speech recognition and discrimination of frequency modulation. Seven people with Ménière’s disease showed reliable improvement in speech recognition with glycerol, which is known to reduce the hydrops (Chung, Hall, Buss, Grose, & Pillsbury, 2004). A consistent improvement in speech discrimination scores (16%) and a pure tone threshold shift of 25 dB at three consecutive frequencies had been found following glycerol treatment in Ménière’s disease (Schessel, Minor, & J Nedzelski Chapter 142, 2004). Thus, audiologic parameters play an important role in diagnosis, prognosis and treatment of Ménière’s disease according to the stage of the disease.

The Otolaryngologist should be provided an opportunity to diagnose every suspected case of Ménière’s disease at the earliest, so that the necessary audiological tests are conducted as per the Otolaryngologist’s recommendations. In future, it is important to have a system in place
that refers every patient of suspected Ménière’s disease to the Department of ENT/Audiology as early as possible.

**4.5.2.4 Improved diagnostic accuracy due to early ENT and Audiology intervention in 2006**

The data collected for the year 2006, have 100% diagnostic accuracy. This was possible due to the supporting objective clinical tests carried out during the clinical evaluation. In all the suspected cases of Ménière’s disease TT ECochG was used as the supporting objective clinical test to prove the presence of hydrops. Otolaryngologists at the Christchurch Public Hospital believe that TT ECochG results for SP and AP amplitudes are significant as they are much larger than the ET ECochG.

**Procedure**

Ear canal was examined with microscopic otoscopy (microscope used to visualise the ear canal). Then the patient was instructed about the procedure and requested to lie down in the supine position. The reference electrode was placed on the ipsilateral earlobe and the ground electrode was placed on the forehead after cleaning the skin surface and applying electrical paste in the area of contact. A drop of Phenol (local anaesthetic (Personal Communication, Hornibrook, 2006)) was used to anaesthetise the area of the tympanic membrane. Then an electrode (needle) was inserted through the area of tympanic membrane anaesthetised with phenol and the tip of the electrode was kept in contact with the promontory [a bony prominence on the inner wall of the tympanum (Dark, 2003c)]. This electrode is a needle covered with Teflon material except the tip, which touches the promontory (Personal Communication, Hornibrook, 2006). The supraural headphones used were placed in a metal shielded shell and attached magnetically to support a ring positioned over the test ear. Tonebursts at 1 and 2 kHz frequency were presented at 90 and 100 dBnHL (dB normal hearing level) intensity respectively at the presentation rate of 30 per second. 1024 stimuli were presented per run. Analysis time was 30 ms (milliseconds). Following norms were used to detect the presence of hydrops (Personal Communication, Gourley, 2007).
Table 4.1 ECochG norms for 1 kHz by Gibson, WPR (1992) (Höhmann, 1993)

Table 4.1 represents the criteria used at the Christchurch Public Hospital to detect abnormal SP at the given hearing level using toneburst frequency 1 kHz (at 90 dBnHL)

<table>
<thead>
<tr>
<th>Hearing level dBHL</th>
<th>Abnormal if SP &gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 25</td>
<td>-6 µV</td>
</tr>
<tr>
<td>20-35</td>
<td>-6 µV</td>
</tr>
<tr>
<td>40-55</td>
<td>-6 µV</td>
</tr>
<tr>
<td>60-75</td>
<td>-3 µV</td>
</tr>
</tbody>
</table>

Table 4.2 ECochG norms for 2 kHz by Gibson WPR (1992) (Höhmann, 1993)

Table 4.2 represents the criteria used at the Christchurch Public Hospital to detect abnormal SP at the given hearing level using toneburst frequency 2 kHz (at 100 dBnHL)

<table>
<thead>
<tr>
<th>Hearing level dBHL</th>
<th>Abnormal if SP &gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 25</td>
<td>-9 µV</td>
</tr>
<tr>
<td>20-35</td>
<td>-7 µV</td>
</tr>
<tr>
<td>40-55</td>
<td>-5 µV</td>
</tr>
<tr>
<td>60-75</td>
<td>-5 µV</td>
</tr>
</tbody>
</table>
Thus, presence of hydrops was confirmed by increased absolute SP amplitude at the Christchurch Public Hospital (Personal Communication, Gourley, 2007). Tonebursts stimuli are known to give better and robust results as compared to click stimuli. Colon and Gibson found ECochG sensitivity increased to 83% by using tonebursts at 1 kHz to measure absolute SP (Kim, Wiet, & Battista, 2005). Another advantage of TT ECochG is that it is slightly quicker than ET ECochG. A study involving 15 participants indicated a mean time of 50.1 +/-14 and 38.8 +/-10.4 milliseconds for ET and TT ECochG respectively (Noguchi, Nishida, & Komatsuaki, 1999). Thus, the results obtained after this procedure were highly reliable to prove the presence of hydrops.

An example of confirming the diagnosis of Ménière’s disease with the same method of TT ECochG, described above is presented below.

Case 0611 represents a 93-year-old woman admitted to the Department of General Medicine in August 2006. This lady was admitted with presenting symptoms of vertigo, nausea, vomiting and buzzing with pressure in the right ear. She had a history of postural hypotension (reduced blood pressure with a change of position (Personal Communication, Hornibrook, 2006)). This lady was referred to the Department of ENT and Audiology both. Dix Hallpike positional test was performed, which was negative. Pure tone audiogram was carried out that showed presence of severe to profound sensorineural hearing loss in the right ear and moderate to severe sensorineural hearing loss in the left ear with a large high frequency component. Her speech recognition scores were 90% at 90 dB in the right ear. Tympanometry yielded type ‘A’ tympanogram in the right ear, indicating normal tympanic membrane compliance. DPOAEs (Distortion Product Otoacoustic Emissions are the sounds emitted by the ear in response to 2 simultaneous tones of different frequencies, which indicate normal functioning outer hair cells of the inner ear) present in the right ear. Acoustic reflexes (Acoustic reflexes measure the stapedius and tensor tympani reflex generated eardrum movement in response to intense sound (Hain, 2006a)) were normal. The ECochG carried out using the toneburst stimuli showed positive results for the right ear with increased absolute SP amplitude of 4.88 µV at 1 kHz and 2.92 µV at 2 kHz. This confirmed the presence of hydrops in her right ear and the diagnosis of Ménière’s disease.
The ECochG test results are seen below and the audiogram for the case 0611 are attached in the appendix.

Figure 4.3 Case no. 0611 ECochG results

These ECochG graphs are hand traced from the display of the ECochG test machine.
L1, L2 and L3 represent the latencies of the ECochG peaks.

A1-A2 represents the SP value and A3-A4 represents the AP value.

Results: SP = 4.88 µV at 1 kHz and 2.92 µV at 2 kHz.

This woman was admitted 4 times before with similar complaints and was discharged all four times without any conclusion. This case was an example, where an early ENT and Audiology intervention could have significantly reduced her number of admissions, hospital stay and mental and physical stress she had to undergo during her 4 previous admissions. The lack of objective supporting tests for this particular case has prolonged the diagnosis of Ménière’s disease; increased the frequency of admissions and the duration of hospital stay. Numbers of studies have provided detailed information on use of objective tests before confirming the diagnosis of Ménière’s disease. A study of 328 elderly patients with Ménière’s disease carried out in Japan reported that an evaluation of nystagmus, VOR, audiogram, ECochG and caloric test were conducted for each patient during that particular study as a supporting evidence to their diagnosis (Mizukoshi, Shojaku, Aso, & Watanabe, 2000).

Case 0612 represents a 61-year-old man admitted to the Department of General Medicine in June 2006, who had had three attacks of vertigo in a year’s time. The clinical notes from the Department of General Medicine reported possibility of BPV and an Epley manoeuvre was performed for the same. Then patient was referred to the Department of ENT and Audiology, both. Pure tone audiogram showed presence of mild to moderately severe sensorineural hearing loss in the right ear and a severe to profound sensorineural hearing loss in the left ear. His tympanometry yielded a type ‘Ad’ tympanogram in the right ear, revealing hypercompliant tympanic membrane. The speech scores were 90% at 70 dB. DPOAEs were present and the acoustic reflexes were normal. He was suspected to have Ménière’s disease at this stage but to confirm the diagnosis an ECochG was carried out using the toneburst stimuli. Absolute SP amplitudes were increased to 18.50 and 10.25 µV at 1 and 2 kHz respectively confirming the suspected diagnosis of Ménière’s disease.
Results of the ECochG test are seen below and the audiogram for the case 0612 is attached in the appendix.

Figure 4.4 Case no. 0612 ECochG results

These ECochG graphs are hand traced from the display of the ECochG test machine. L1, L2 and L3 represent the latencies of the ECochG peaks.
A1-A2 represents the SP value and A3-A4 represents the AP value.

Results: SP = 18.50 µV at 1 kHz and 10.25 µV at 2 kHz.

This was another example of early ENT/Audiology intervention improving the diagnostic accuracy for the patients of Ménière’s disease. This case was suspected to have BPV and even treated for the same with Epley manoeuvre, which was not appropriate, but due to early ENT/Audiology intervention; accurate final diagnosis was possible and patient received an appropriate treatment on time.

4.5.2.5 Reduction in the length of hospital stay and avoiding unnecessary scans in 2006

Case 0613, diagnosed as having Ménière’s disease in the data collected for the year 2006 could be used as an example of appropriate diagnosis and management of Ménière’s disease.

This (Case 0613) was a 39-year-old man admitted to the Department of Otolaryngology with a history of two to three episodes of vertigo in the past 2 years. The past admissions were without any confirmed diagnosis. For this particular admission, he was seen within 5 hours by an ENT Specialist. The VOR was normal and the Dix Hallpike test was negative. Pure tone audiometry revealed mild to moderately severe sensorineural hearing loss in his right ear and mild high frequency hearing loss in the left ear. Tympanometry revealed a type ‘As’ tympanogram in the right ear, revealing reduced compliance of the tympanic membrane and the speech scores were 100% at 55 dB in the right ear. DPOAEs were present and the acoustic reflexes were normal in the right ear. On suspicion of Ménière’s disease, ECochG was carried out that showed results of increased absolute SP amplitudes to 9 and 6.96 µV at 1 and 2 kHz respectively using the toneburst stimuli in his right ear and the final diagnosis of Ménière’s disease was confirmed.
Results of the ECochG test are seen below and the audiogram for the case 0613 is attached in the appendix.

Figure 4.5 Case no. 0613 ECochG results

These ECochG graphs are hand traced from the display of the ECochG test machine.

L1, L2 and L3 represent the latencies of the ECochG peaks.

A1-A2 represents the SP value and A3-A4 represents the AP value.
Results: $SP = 9 \, \mu V$ at 1 kHz and $6.96 \, \mu V$ at 2 kHz

Thus the patient was diagnosed accurately within 5 hours as a case of Ménière’s disease. This patient had a hospital stay of only 1 day, no scans were carried out and accurate diagnosis and treatment was provided within hours after admission to the Department of Otolaryngology. Thus, an overall efficiency of the diagnosis and management for the patients of Ménière’s disease was improved significantly.

4.5.2.6 Accurate diagnosis reached with objective clinical test of TT ECochG in 2006

There were some cases where the signs and symptoms did mimic Ménière’s disease and all the symptoms could have led to the same diagnosis in 2006 during the study period. However, diagnosing such a case merely on the basis of symptoms could be a mistake. Such cases are the challenging cases, where the final diagnosis should never be given without supporting objective clinical findings. A similar case 0621, presented to the Christchurch Public Hospital during the data collection period in 2006 is described below.

A 76-year-old woman (Case 0621) admitted to the Emergency Department in May 2006. She was admitted with sudden vomiting, bilateral tinnitus, left beating nystagmus and vomiting without any other ear symptom. She has had vertigo attacks since 1998. Within few hours of this particular admission, she was seen in the Department of ENT/Audiology. Nystagmus had disappeared by then. Other tests like VOR, rotation fixation and Dix Hallpike test were negative. She had bilateral mild to severe sensorineural hearing loss. Ménière’s disease was suspected. ECochG was carried out but the results were normal. MRI scan was normal as well.

Hence, for this admission her final diagnosis was ‘other peripheral vertigo’. The patient was readmitted in July 2006.
In July 2006, she (case 0622) was admitted to the Emergency Department once again with tapping noise (tinnitus) in the left ear and vertigo. Second ECochG test and ABR (Auditory Brainstem Response is an electrical signal evoked in the cochlear nerve and the brainstem following sound stimuli like clicks) were carried out. Both results were normal. However, a 3-D Fiesta MRI scan and 3-D reconstructions showed probable 8th nerve vascular compression of left vestibular nerves.

Thus, a repeat MRI scan in her second admission showed probable vascular compression of the left vestibular nerves; the final cause for her complaints of vertigo, tinnitus and hearing loss. For this particular patient, all the symptoms were leading to the diagnosis of Ménière’s disease but the final diagnosis was something else. A report published in Christchurch Public Hospital by Hornibrook et al (2006) had three cases with similar presentation. They all had normal ECochGs on at least 2 occasions. An MRI scan reported the presence of vascular compression of the 8th cranial nerve in all three cases. Some institutes have come up with a diagnosis of ‘disabling positional vertigo’ for such condition with a supporting evidence of abnormal ABR. However, ABR would be normal in cases where only vestibular nerve was compressed but the cochlear nerve was intact. The central nervous system component of the vestibular nerve was much longer (8.3 mm) and so the symptoms of vascular compression are more common for the vestibular nerve than other cranial nerves (Hornibrook, MacFarlane, Gourley, & Wells, 2006). To diagnose such a condition, ECochG acts as a valuable tool for ruling out the possible diagnosis of Ménière’s disease.

Similar to those cases, MRI scan for the case described above from this study had shown ‘probable’ vascular compression of left vestibular nerve after two negative ECochG results. Thus, a systematic approach resulted in including this case under ‘other’ indicating uncertain final diagnosis and the possible diagnosis of Ménière’s disease was ruled out. Ryu et al from Japan had studied 53 similar patients and reported how such vascular compression of a vestibulocochlear nerve produces symptoms of vertigo or tinnitus or both depending on...
branch of the nerve affected (Hornibrook, MacFarlane, Gourley, & Wells, 2006). After this
discussion, TT ECochG should be prioritised as the test for diagnosing Ménière’s disease.
Chapter 5

Expense of Managing Peripheral Vertigo
5.1 Length of hospital stay

5.1.1 Results

The difference between the average length of hospital stay for the years 2004 and 2005 (4 days with a standard deviation of 3 days) and the year 2006 (3 days with a standard deviation of 2 days) had not been statistically significant\(^8\).

5.1.2 Discussion

Even though the statistical findings were not conclusive, the Mann-Whitney Rank Sum Test also indicated that the difference in the median values between the two groups was not great enough to exclude the possibility that the difference was due to random sampling variability. These results explained the fact that the two samples compared, were vastly different in sizes. The medians could have been different if there was enough data to tell them apart and then the results could have been statistically significant. Year 2004-2005 data have had 116 patients as compared to 28 patients for the year 2006. Hence, the average difference of 1 day should be considered to find out an estimated savings as far as the costs of hospital stay were concerned.

\(^8\) Mann-Whitney Rank Sum Test: \( p = 0.112 \)
Figure 5.1 Length of stay, 2004 to 2006

Figure 5.1 is a graph showing days of hospital stay against the number of patients for three years (2004, 2005 and 2006). The dotted line representing the frequency of number of patients against the days of hospital stay for the results matched with the continuous line representing the normal distribution.

It was evident from the graph above that the data was normally distributed in terms of admissions to the hospital for the period of three years from 2004 to 2006.
Figure 5.2 Comparison of the length of hospital stay (2004-2005 to 2006)

Figure 5.2 represents a graph of two groups of data according to the years of comparison (2004-2005 to 2006). An overall reduction of 1 day in the number of days of hospital stay was evident for the year 2006. The standard deviation of 3 days and 2 days was seen for the years 2004-2005 and 2006 respectively.

This reduction in the length of hospital stay of 1 day, corresponds to the net saving of NZ (New Zealand) $330/patient as that was the cost of overnight stay in the department of Christchurch Public Hospital (Personal Communication, Hornibrook, 2006). The available literature explaining the details of the length or duration of hospital stay for such patients with acute vertigo due to peripheral vestibular pathologies is not adequate. The study carried out at the “Norfolk and Norwich University Hospital NHS Trust”, including 20 patients with posterior semicircular canal BPV discussed in chapter 1, reported that an average waiting time in the hospital care for specialist referral and vestibular assessment was 35 weeks (Fife & FitzGerald, 2005). For many suspected patients, positional tests or objective tests like ECochG (test to diagnose the presence of hydrops associated with conditions like Ménière’s
disease), audiogram etc. were not conducted on time. This was one of the main reasons preventing patients from receiving prompt treatment and adversely affecting the overall patient management. This study by Fife and FitzGerald (2005) showed that the delay in the entire process was due to long waiting periods in the initial stages of clinical evaluation. Delays due to similar reasons were witnessed in this study at the Christchurch Public Hospital. In conclusion, timely accurate diagnosis does help reduce the length of hospital stay.

5.2 CT/MRI scans

5.2.1 Results

CT scans were conducted for 14% of patients in 2006 (4 scans in 28 patients), compared to 28% of patients in 2004-2005 (32 scans in 116 patients). The number of MRI scans conducted in 2006 were reduced to 18% (5 scans in 28 patients), compared to 26% during 2004-2005 (30 scans in 116 patients). Total scans carried out in 2004-2005 were 61% (71 out of 116) as compared to 32% (9 out of 28) for 2006. However, no statistically significant difference was evident between the total costs of scans in 2004-2005 to that of 2006.

5.2.2 Discussion

A CT scan costs NZ $155 and a MRI scan costs NZ $285 at the Christchurch Public Hospital (Personal Communication, Hornibrook, 2007). Mann-Whitney Rank Sum Test results indicated that there was not a statistically significant difference between the total costs of scans conducted in 2004-2005 as compared to the total costs in 2006. However, the test results also reported that the difference in the median values between the two groups was not great enough to exclude the possibility that the difference was due to random sampling.

\[^{9}\text{Mann-Whitney Rank Sum Test: p value = 0.169}\]
variability. Hence, considering the vast difference in the sample sizes (116 to 28), the percentage results can be used for comparison.

Figure 5.3 Scan costs

Figure 5.3 represents comparison of the cost of scans (CT and MRI scans both) between two groups of data (2004-2005 and 2006). A large area of standard deviation was seen as far as the cost of the scans is concerned.

A reduction of 14% (or 4 CT scans per 28 patients in 2006) was seen in the number of CT scans, which corresponds to the savings of about NZ $21 per patient (NZ $2100 for 100 patients). Similarly, an 8% (or 2 MRI scans for 28 patients in 2006) reduction was seen for the MRI scans, which corresponds to the savings of about NZ $23 per patient (NZ $2300 per 100 patients).

The average cost of scans (CT and MRI scans both) per person is shown in the table below.
Table 5.1 Average costs of CT and MRI scans

Table 5.1 represents the average cost of both scans (CT and MRI scans) taken together per patient for the years 2004-2005 and 2006.

<table>
<thead>
<tr>
<th>Years</th>
<th>Average scans cost (CT and MRI scans)</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>2004-2005</td>
<td>$117</td>
<td>± $146</td>
</tr>
<tr>
<td>2006</td>
<td>$73</td>
<td>± $129</td>
</tr>
</tbody>
</table>

Thus, an average of NZ $44 per patient was the savings with the total scans costs due to recommended changes in 2006. However, the Mann-Whitney Rank Sum Test indicated that the decrease in the costs of scans for the year 2006 was statistically not significant\(^\text{10}\). The test also indicated that the difference in the median values between the two groups was not great enough to exclude the possibility that the difference was due to random sampling variability as the two data compared were vastly different in sizes.

\(^{10}\) Mann-Whitney Rank Sum Test (P = 0.169)
5.3 Average total savings

Figure 5.4 Total costs (Hospital stay plus scans)

Figure 5.4 represents a graph of comparison of total costs of scans and hospital stay taken together for the two sets of data (year 2004-2005 and 2006) (based on the cost of hospital stay per day of NZ $330, a CT scan cost of NZ $155 and a MRI scan cost of NZ $285)

Table 5.2 Total average costs

Table 5.2 represents the total costs of hospital stay and scans taken together with their standard deviations for the year 2004-2005 and 2006 (based on the cost of hospital stay per day of NZ $330, a CT scan cost of NZ $155 and a MRI scan cost of NZ $285)

<table>
<thead>
<tr>
<th>Years</th>
<th>Average Costs per patient (Hospital stay + Scans)</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>2004-2005</td>
<td>$ 1,525</td>
<td>± $ 1,077</td>
</tr>
<tr>
<td>2006</td>
<td>$ 1,146</td>
<td>± $ 721</td>
</tr>
</tbody>
</table>
The savings achieved in the costs of hospital stay for the year 2006 were statistically\textsuperscript{11} not significant however, the Mann-Whitney Rank Sum Test results also indicated that the difference in the median values between the two groups was not great enough to exclude the possibility that the difference was due to random sampling variability.

There was a vast difference between the numbers of patients in two groups considered for the study and therefore the average differences could be considered to find out an estimated savings achieved in the year 2006.

Thus, an average saving of NZ $379 per patient was achieved (based on the cost of the hospital stay per day of NZ $330, a CT scan cost of NZ $155 and a MRI scan cost of NZ $285) due to early Otolaryngology/Audiology intervention. Considering 2 years and 7 month’s data included in this study, there were 144 patients (116 for 2004-2005 and 28 for 2006), which gave an average of 56 patients per year. Hence, an estimated saving of NZ $21,224 per year could be achieved with the recommended changes in 2006.

There could be some environmental factors like variation in cases of acute vertigo according to the season like winter or summer or even holiday season for that matter where cases of accidents or injuries are likely to be more and this could affect the total percentage over a year by a small margin as only a 7-month data was considered in 2006. For example, prevalence of acute vestibular neuronitis could peak in the winter months (Hanley & O'Dowd, 2002). Buchele and Brandt (1998) reported occurrence of BPV at 12% in patients who have had vestibular neuritis (Arbusow, Theil, Strupp, Mascolo, & Brandt, 2001). Post traumatic BPV accounts for about 15-20% of all BPV cases (Gordon, Levite, Joffe, & Gadoth, 2004). Thus, environmental factors or trauma could be responsible for increasing the number of cases with different peripheral vestibular pathologies leading to increased need for investigations or

\textsuperscript{11} Mann-Whitney Rank Sum Test. $P = 0.122$
scans, which might affect the overall estimated savings discussed above by a minimal amount.

Some cases discussed below represent that the unnecessary scans could be avoided by early Otolaryngology/Audiology intervention.

*Case 0403* represents a 58-year-old woman admitted to the Emergency Department with presenting complaints of severe dizziness in the morning, worse on sitting up. No spontaneous nystagmus was noticed. She underwent a MRI scan, which was normal. She was never referred to the Department of ENT or Audiology. No positional test was carried out and she was diagnosed as a case of BPV.

For this case, the MRI scan was not necessary and could have been avoided. The suspected diagnosis of BPV could have been confirmed by the positional tests. No record of such test was found in her clinical notes. Another case with similar findings is described below.

*Case 0430* represents a 67-year-old female patient admitted to the Department of General Medicine in December 2004. She had sudden positional vertigo with nausea, inability to sit and had no spontaneous nystagmus. She was admitted for 5 days. No record of positional tests was found in her clinical notes. A CT scan was carried out and the final diagnosis was BPV. She was never referred to the Department of ENT or Audiology.

For this case, the diagnosis of BPV could have been confirmed by performing a positional test soon after the admission and the CT scan could have been avoided. BPV is a condition that does not need any invasive or other form of investigation for diagnosis. The diagnosis of BPV is evident on the basis of thorough history and physical examination. No quantitative vestibular laboratory testing like electronystagmography is necessary (Angeli, Hawley, & Gomez, 2003).
Benefits of the changes implemented in 2006, particularly in the initial clinical examination were evident in the following case.

Case 0601 represents a 93-year-old woman admitted to the Department of General Medicine with complaints of acute vertigo and fall. Dix Hallpike test showed horizontal nystagmus to the left side. She was referred to the Department of ENT and Audiology. Dix Hallpike test was positive bilaterally with horizontal direction changing geotropic nystagmus implying horizontal canal BPV. She was treated with “two barbecue rotations” (procedure discussed later in Chapter 6). In a follow up 13 days later, no vertigo was noticed and Dix Hallpike test was negative.

Even at the age of 93 years, no scans were required during the 3-day admission period of this particular patient due to accurate diagnosis.

It has been seen in many cases that the diagnosis of vertigo do not require extensive diagnostic testing and could be treated even at the level of a primary care settings if diagnosed appropriately (Swartz & Longwell, 2005). This shows that the patients of vertigo due to peripheral vestibular pathologies like BPV could be managed satisfactorily by avoiding unnecessary scans.
Chapter 6

Vestibular Neuritis, Migraine,
Central and Other Vertigo
6.1 Introduction

Vestibular neuritis, migraine, vertigo of central origin and other vertigo (unknown cause) were the other four common diagnoses found in the process of data collection at the Christchurch Public Hospital. Initial presentation of many patients with these conditions could be confusing and misdiagnosed as having BPV or Ménière’s disease.

6.2 Vestibular Neuritis

A syndrome of severe vertigo, nausea, vomiting, spontaneous nystagmus, and postural instability developing over days in a healthy person is usually attributed to the viral vestibular neuritis (Hotson & Baloh, 1998). The condition usually occurs in spring or early summer (Baloh, 2003). Prevalence of acute vestibular neuronitis could also peak in the winter months (Hanley & O'Dowd, 2002). Vestibular neuritis is known to occur in all ages but rarely seen in children. Approximately 15% of all cases of vertigo are due to vestibular neuritis (Hain, 2006e).

Most authors report in favour of the viral theory of the vestibular neuritis (Fenton, Shirazi, Turner, & Fagan, 1995). Fischer (1967), reported that similar syndrome can result from lack of blood supply to the vestibular system (Hain, 2006e).
Figure 6.1 Ischemia of the vestibular system producing vestibular neuritis

Ref: (Hotson & Baloh, 1998).

Figure 6.1 is a diagrammatic representation of blood supply to the vestibular system. The anterior vestibular artery supplies to the vestibular system but not to the cochlea and hence isolated occlusion of this artery could give rise to the vestibular syndrome without any hearing loss.

6.2.1 Symptoms, diagnosis and treatment
Vertigo, disequilibrium or imbalance and nausea are the common presenting symptoms. Although patients with vestibular neuritis are sensitive to the head motion like that of the BPV, it is not related to the side of the head, which is down but just whether the patient is sitting or lying down (Hain, 2006e). Most cases presented in the spreadsheet 1 with the diagnosis of vestibular neuritis show the prolonged duration and higher severity of their symptoms.

Vestibular neuritis was found to be the second commonest condition diagnosed amongst the three-year data collected from the Christchurch Public Hospital for this study. Head Thrust Test to detect the presence of VOR is the important test of diagnosis of vestibular neuritis. The head is briskly rotated at 30° to ensure that cupular stimulation is primarily tested in the horizontal semicircular canal. Patient is asked to keep his eyes focused on a target while his head is manually rotated in an unpredictable direction using a small amplitude (5° – 15°) and high acceleration angular thrust. In case of a normal vestibular system, eyes move in the
opposite direction of the head movement through the exact angle required to remain focused on the target. If there is a vestibular hypofunction, eyes would move less than the required angle (Schubert & Minor, 2004). This voluntary eye movement to catch up with the head movement is called saccade (rapid voluntary eye movement to redirect the line of sight (Dark, 2003b)). If the catch up saccades occur after the Head Thrust Test in one direction but not in the other, then that indicates the presence of a peripheral vestibular lesion on one side (Baloh, 2003). The VOR test was used as a parameter to diagnose the vestibular neuritis in most cases involved in this study at the Christchurch Public Hospital in 2006.

Commonly used medications include steroids such as methylprednisolone, which significantly improve the recovery of peripheral vestibular function in patients with vestibular neuritis (Strupp et al., 2004). Antihistamines, anticholinergic agents, antidopaminergic agents, and 𝛾-aminobutyric acid–enhancing (GABAergic) (gama aminoButaric acid (Dark, 2003a)) agents are helpful as well. The recovery process usually takes several weeks and vestibular exercises play an important role in the restoration of normal vestibular function (Baloh, 2003).

### 6.2.2 Results

The diagnosis of vestibular neuritis was given to 21% cases (24 of the 116 patients) during the years 2004 and 2005. In the year 2006, this percentage dropped to 14% cases (4 of the 28 patients) presenting an overall decrease of 7%. In terms of diagnostic accuracy for the years 2004-2005, the diagnosis of 50% cases (12 of 24 patients) were well supported with substantial subjective and objective clinical evidences. This was increased to 100% (4 of 4 patients) for the year 2006, as all cases were well supported by subjective and objective clinical evidences. However, this increase was not statistically significant. The statistically negative results could be the outcome of a small size of sample (number of patients) considered the year 2006 compared to the relatively larger sample from 2004-2005. Hence,

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12 Chi-square test: Chi-square= 3.500 with 2 degrees of freedom. (P = 0.174)
the percentage results showing the improvement with the diagnostic accuracy for 2006 should be considered.

6.3 Migraine, central and other vertigo

No cases of migraine vertigo were detected in the data collected for all the three years.

6.3.1 Central Origin
Vertigo of central origin is the vertigo due to involvement of brain or CNS (Central Nervous System). This type of vertigo has a longer duration but moderate intensity and associated neurological symptoms are common but the hearing loss is rarely seen and vertigo is usually not affected by the head position (Hansson, Månsson, & Håkansson, 2004). Common causes of central vertigo include congenital causes like neurofibromatosis, Arnold-Chiari malformation, spinocerebellar atrophy types 1-9 etc. and acquired causes like basilar migraine, multiple sclerosis, space occupying lesions like meningioma, raised intracranial pressure, inflammatory disorders like Behcet’s disease etc. (Pagarkar & Davies, 2004). For the year 2006, there was no admission with the final diagnosis of vertigo of central origin. Only 3% (3 of 116 patients) cases were diagnosed as having the vertigo of central origin for the years 2004-2005. Most cases of vertigo due to central origin are managed by the Neurology Department and there is a minimal ENT/Audiology input. These cases were included in the spreadsheet 1 for this study at the Christchurch Public Hospital, only because they presented with few common complaints of vertigo and dizziness but otherwise they formed a different group of patients. Hence, discussing vertigo of central origin results in details would be going out of the scope of this particular study.

6.3.2 Other – Uncertain final diagnosis
This column in the spreadsheet 1 included cases where no single final diagnosis was possible due to lack of symptoms or clinical evidence in favour of a particular diagnosis. Most cases were labelled ‘other peripheral vertigo’, ‘disordered vestibular function unspecified’ or ‘vertigo of unknown origin’. For the last two years (2004-2005), there had been 12% cases
(14 of 116 patients) diagnosed as ‘other’. In 2006, there were 39% cases diagnosed as ‘other’ (11 of 28 patients). Thus, an overall increase of 27% was evident after implementing the changes recommended in 2006 as part of this study. Due to a limited number of cases, results related to the diagnostic accuracy might not be realistic but approximate conclusions could be derived. For 2004 and 2005, the diagnostic accuracy was 36% (5 of 14 patients), which remained unchanged for the year 2006 (4 of 11 cases).
Chapter 7

Vertigo Research Survey
7.1 Introduction

A short survey was carried out as a part of this research. The objective of this survey was to get a brief description of the system being followed within different District Health Boards (DHBs) of New Zealand and collect the statistical division of the diagnoses for patients admitted to these hospitals with presenting complaints of vertigo. ENT specialists from 21 DHBs were informed about this study and replies to the survey forms were requested. For few places, the Department of ENT is common for two DHBs in that area or the same ENT Specialist manages the ENT Departments for both DHBs. Thus, replies from the ENT Departments of 18 DHBs were received. The following data has been gathered from this survey.

Names of individual doctors or the DHBs have not been displayed to maintain the confidentiality. Most results provided in this survey were estimates given by the clinicians working at the Department of ENT.

A spreadsheet (spreadsheet 2) with survey results is attached in the appendix.

7.2 Admitting Departments

7.2.1 Results

Most or 78% (14 of 18 DHBs) admissions were in the Department of General Medicine. The second highest number of admissions was in the Department of ENT at 50% (9 of 18 DHBs). The third highest number of admissions was in the Department of Neurology at 17% (3 of 18 DHBs) and the fourth highest number of admissions was in the Department of Emergency Medicine at 11% (2 of 18 DHBs).

7.2.2 Discussion

On comparing the data for the years 2004-2005 from the Christchurch Public Hospital and other ENT Departments in New Zealand, the results showed that the Department of General
Medicine has had most admissions (78% from the survey and 66% from the 2004-2005 data from the Christchurch Public Hospital). Number of admissions to the Department of ENT was second highest at 50% according to the survey, which was much better or higher than the data for 2004-2005 collected for this study in the Christchurch Public Hospital, where there were only 2% admissions to the Department of ENT. These numbers supported our assumption that ENT/Audiology intervention necessary for patients with complaints of vertigo was much lesser than expected at the Christchurch Public Hospital. During this study, the percentage of admissions to the Department of ENT/Audiology was increased to 18%.

The third highest number of admissions at 17% were to the Department of Neurology, which were about half of the admissions (9%) at the Christchurch Public Hospital for the years 2004-2005. The fourth highest number of admissions at 11% was to the Department of Emergency Medicine from the survey. The data for 2004-2005 have had second highest number of admissions at 16% to the Department of Emergency Medicine.

### 7.3 In patient ENT referrals

#### 7.3.1 Results

Only 22% (4 of 18) of DHB’s replies showed that these patients were referred to the Department of ENT (Replies were “Mostly”, “Always”, “Usually”). 67% (12 of 18) DHBs replied that the chances of these patients being referred to the Department of ENT were reasonable (Reply was “Sometimes”) and 11% (2 of 18) DHBs replied that the chances were rare (Replies were “Occasionally”, “Not usually”).

#### 7.3.2 Discussion

Our data showed that there were 33% (38 of 116 patients) in-patient referrals to the Department of ENT/Audiology for the year 2004-2005, which was close to the survey results
of 22% from rest of the DHBs. This rate of referral was successfully increased (to 100% - 28 of 28 patients) in 2006.

7.4 Follow-up Care

7.4.1 Results
Distribution of patients according to the follow up care is shown in the chart overleaf.
1. 67% (12 of 18 replies) were followed up in the Department of ENT.
2. 39% (7 of 18 replies) were followed up by the GP (General Practitioner).
3. 11% (2 of 18 replies) were followed up in the Department of General Medicine.
4. 5% (1 of 18 replies) were followed up in the Department of Neurology.
5. 5% (1 of 18 replies) answered ‘depends on cause of vertigo’, which could be interpreted as “unsure”.
6. 17% (3 of 18 replies) answers, (“Don’t know”, “Uncertain” and “Nil”), which could be interpreted as that the person replying to the survey was not certain of the exact process of such follow-ups hence termed as “unknown”.

Unfortunately, we do not have this information in the data collected from the Christchurch Public Hospital due to time constraint of 7 months for the project.

7.5 Statistical diagnostic division
7.5.1 Results
Percentage or statistical division of patients according to the diagnosis is presented in a pie chart below (2 out of 18 DHBs preferred not to reply to this question).

![Pie chart showing percentage of diagnoses](image)

**Figure 7.2 Percentage of the diagnosis (Survey)**

1. BPV: 33%
2. Ménière’s disease: 17%
3. Vestibular neuritis: 19%
4. Migraine associated vertigo: 6%
5. Vertigo of central origin: 11%
6. Other (uncertain aetiology) vertigo: 10%

7.5.2 Discussion
BPV: 2004-2005 data collected showed 53% (61 of 116 patients) incidence, which was much higher than the survey results. 2006 data showed 36% (10 of 28 patients) incidence, which did match with the survey results. This supported the assumption of this study that many cases diagnosed as BPV in 2004-2005, were without supporting clinical evidences.

Ménière’s disease: Data collected for the years 2004-2005 showed 12% (14 of 116 patients) incidence and 2006 data showed 11% (3 of 28 patients) incidence of Ménière’s disease.
Practically, these results were similar to the results of this survey. The factor to be considered here is that most statistical values provided in the survey were only the estimates.

Vestibular neuritis: The data collected for 2004-2005 showed 21% (24 of 116 patients) incidence and 14% (4 of 28 cases) incidence for the year 2006. These results were close to the estimate of 19% provided during this survey.

Migraine associated vertigo: This diagnosis was not recorded during the data collection for all three years. The survey results showed the rate of admissions to be 6%.

Vertigo of central origin: 2004-2005 data collected gave a 3% (3 of 116 patients) incidence, whereas no cases were recorded for the year 2006. This difference in the incidence rates from the survey could not be commented here due to the small number of cases we have come across during the study and hence more data collection was necessary to evaluate the estimate provided by the surveyors.

Other (Uncertain aetiology): The data for 2004-2005 showed an incidence of 12% (14 of 116 patients), which did match with the 10% estimate given by the surveyors. However, in the data for 2006, this rate of incidence increased to 39% (11 of 28 cases). The estimate of 10% gathered from this survey could represent the similar process that had been applied in most clinical settings where final diagnoses like BPV, or Ménière’s disease were given and accepted without objective supporting clinical evidences. This led to a decrease in the number of cases with ‘uncertain diagnoses’, which were labelled as ‘other’ during the data collection process at the Christchurch Public Hospital.
Chapter 8

Summary and Conclusions
8.1 Summary and conclusions

Acute vertigo remains a common presenting symptom of many peripheral vestibular disorders, mostly seen in older adults. However, if the nervous system and brain are intact then even the older adults should recover as quickly as the younger adults could. Women are more commonly affected with complaints of vertigo due to peripheral vestibular disorders.

Referring all cases with suspected peripheral vestibular pathology to the Department of Otolaryngology, preferably during their hospital stay or as early as possible should be prioritised. All suspected cases of Ménière’s disease or BPV should be diagnosed by an Otolaryngologist while the supporting audiological tests are conducted simultaneously to confirm the diagnosis. Audiologists should work in association to conduct the necessary investigations as per the recommendations of the Otolaryngologist/ENT specialist during the process but any decision over the medico-legal aspect of a case regarding its diagnosis has to be made by the Otolaryngologist.

This process results in a significant increase in the “diagnostic accuracy” with a corresponding decrease in the days of hospital stay and number of investigations (mainly CT/MRI scans) performed during or after the hospital stay. This study showed that the overall diagnostic accuracy was doubled from 35% (2004-2005) to 71% (2006), which was the prime goal of this study. The savings of NZ $21,224 per year could be considered as an estimated finding due to the recommended changes in 2006 for patients presenting with complaints of acute vertigo due to peripheral vestibular pathologies.
Carrying out positional tests like the Dix Hallpike test in all suspected cases of BPV should not be avoided unless there is a medical or physical contraindication. Some researchers have advised that the Dix Hallpike test should be carried out in all suspected cases of BPV, even in post traumatic BPV where patient could have pain or discomfort (Gordon, Levite, Joffe, & Gadoth, 2004). Nystagmus and its characteristics could be better observed with the use of “Frenzel glasses” (Personal Communication, Hornibrook, 2006). However, it is not a common practice at the Christchurch Public hospital and hence, clinicians should be advised to use “Frenzel glasses” before conducting the positional tests in all suspected cases of BPV.

General awareness and significance of the Dix Hallpike test in diagnosing cases of vertigo due to peripheral vestibular disorder could be increased at every level from primary health care to the hospital settings via ongoing seminars, training etc. It has been noticed in a survey of General Practitioners conducted in Donegal that none of the GPs had performed the Dix Hallpike test in their clinics prior to the survey and most of them were practicing for more than 10 years (Hanley & O'Dowd, 2002). Hence, conducting a similar survey in Christchurch at the Primary Health Care level would be beneficial in the process.

Curative treatments for BPV (Epley or the Semont manoeuvre etc.) if applied on time according to the diagnosis, then it reduces patient stress, anxiety and the overall cost of patient management. These curative manoeuvres should be carried out by an Otolaryngologist on confirming the diagnosis as there could be an immediate or delayed reaction and some other complications may arise following such a manoeuvre.

Conducting an audiogram or other audiological tests like speech test, electronystagmography or ECochG should be considered as an integral part of the process of initial evaluation for all
patients with suspected peripheral vestibular pathology. These tests form the basis of diagnosis in most conditions of the peripheral vestibular pathologies considered for this study.

Clinical tests necessary to exclude other peripheral vestibular pathologies should be carried out in all suspected cases and then the single final diagnosis should be considered.

Writing detailed clinical notes using the most appropriate medical terminology is necessary to avoid confusion and delay regarding the final diagnosis and treatment.

Coding errors could have serious consequences over the final diagnosis and should be prevented in future.

8.1.1 Application of findings of this study in other fields of medicine
This study covered a small area of Otolaryngology/Audiology. Most of the problems covered were related to the patients with presenting complaints of acute vertigo; however few aspects of this study could be generalised. For example, lack of objective tests being conducted before confirming the final diagnosis could be a generalised problem and would be existing in several other specialities within the hospital. This might affect the overall diagnostic accuracy and the treatment as well. Issues of conducting unnecessary investigations like scans, increased length of hospital stay are linked to the problem of lack of diagnostic accuracy and these can be generalised to all other areas of medical specialities.

Few studies presented in the introduction explain the major impact on the financial aspect of health system due to such unnecessary investigations or prolonged hospital stays in case of patients with acute vertigo. Considering any busy department of a major public hospital may
reveal similar problems in several other specialities with acute conditions like cardiac emergencies, respiratory emergencies etc. Conducting similar studies in these various medical specialities to rectify the diagnostic accuracy or appropriateness of objective tests being carried out can provide vital information. The amount of expenses due to such minor errors combined within all the departments of a hospital should result into a significantly large amount. This loss could be prevented or reduced by a similar study like this one in various other departments of a hospital in future.

Secondly, errors like lack of written records of the clinical examination conducted or use of inappropriate medical terminology etc. found during this study could also be the problems in many other medical or paramedical specialities like Audiology. Coding errors resulting into a wrong diagnosis being recorded were witnessed on several occasions during this study; which could be another general problem across many other medical specialities that needs to be addressed.

8.2 Further study and future direction

8.2.1 Normative data collection of age related balance control
A system or method to distinguish people, especially older population, who are at risk of developing peripheral vestibular disorder or balance problems in general, is essential. Recommendations of Collins et al (1994) could be considered here. ‘Posturographic studies’ could be used to investigate the effects of ageing process on balance. In a study of 50 healthy individuals, (25 aged 19-30 and 25 aged 71-80) a technique called “Stabilogram - diffusion analysis”, for examining quiet-standing centre-of-pressure (COP) trajectories was used. This study showed that the aging process results in changes in the functional organization of the open-loop and closed-loop control systems used by the Central Nervous System involved for the postural control in healthy human beings. These changes could be characterized
quantitatively during periods of undisturbed stance. A normative database related to specific age groups could be created and used for distinguishing people at risk of developing complaints of vertigo (Collins, DeLuca, & Jabre, 1994). A similar method could be considered for Christchurch Public Hospital and for all other DHBs across New Zealand in future.

8.2.2 A newer perception of a Dedicated Vestibular Clinic

Having a separate clinic dedicated to the patients with complaints of peripheral vestibular disorders could be another efficient and economical way to deal with this problem. There are 3 major hospitals in Christchurch (Christchurch Public Hospital, Princess Margaret Hospital and Burwood Hospital). All patients with vestibular problems could be referred to this Dedicated Vestibular Clinic. Few significant results have been reported with respect to the incidence of peripheral vestibular disorders like BPV, Ménière’s disease or vestibular neuritis at the Christchurch Public Hospital. In Britain, 15 out of each 1000 patients consult their General Practitioner on account of symptoms of dizziness (Jayarajan & Rajenderkumar, 2003). Hornibrook, (2005) reported that about 10% of general practice consultations and about 20% of referrals to Otolaryngologists and Neurologists consists of patients with complaints of vertigo or dizziness. The data for this study showed that there were 518 patients with complaints of vertigo in 2004-2005 and 55 patients in 2006 (32 patients in 7 months) (Many of these patients were excluded from the study for not having a specific diagnosis as explained in Chapter 2. This makes an average of 191 patients per year admitted to the Christchurch Public Hospital with complaints of vertigo. Including patients with similar complaints from Princess Margaret and Burwood hospital of Christchurch to this number should present with a reasonable amount of new patients each year. In addition, there would be follow-ups for all new as well as existing patients to this clinic. Most of these patients do require some form of vestibular rehabilitation. They could be regularly followed up in this clinic to avoid further delays or complications. Secondly, a general recommendation to refer all such patients with vertigo from the Primary Health Services or
GPs, directly to this clinic would not be an unreasonable proposition. BPV, acute vestibular
neuronitis, and Ménière's disease are responsible for most (93%) cases of vertigo (Labuguen,
2006). Hence, such a Dedicated Vestibular Clinic should be self sustainable with the
percentage of patients and follow ups witnessed in this study. This should help avoid all
those unnecessary scans, pathological tests and prolonged hospital stays with significant
increase in the diagnostic accuracy.

8.2.3 Scope of improvement for a similar study in future
The data collection was carried out for a period of 7 months during this study and still the
number of participants involved was too few to compare with the past two years data. Hence,
it is essential to conduct a similar study in the future with more time devoted to the process of
data collection.

The financial aspects of the system prevented actual involvement of the instructor/student or
the supervisor in the initial clinical examination of each patient admitted to the hospital with
symptoms of acute vertigo. This prevented conducting videooculography or involvement
with actual physical examination of each patient considered for this study. Providing better
financial support for similar studies should be considered in the future.

Involving a 24-hour on call medical registrar and an Audiologist or Audiology student
working together, taking part in the clinical examination and recording the nystagmus for
such patients would be ideal for a similar study in the future. This should provide first hand
information necessary for such a study rather than relying on findings of other clinicians.

Conducting a more detailed survey similar to the one carried out for this study should give a
broader picture of diagnosis and management of such patients across New Zealand.
8.2.4 Vestibular clinical training for “Master of Audiology” students

Master of Audiology courses in New Zealand involve material on vestibular system but this does not include significant clinical training to the audiologists with respect to conducting the diagnostic positional tests like the Dix Hallpike test or other curative treatment manoeuvres like the Epley or the Semont manoeuvre etc. Literature and various websites from countries like USA or England show the detailed vestibular training provided to the audiologists in their graduate training program. Some countries offer post-graduate training specifically for audiologists with interests in working with patients of vestibular disorders. The AAA (American Association of Audiology), Scope of Practice 2007 states, "Audiologists also are involved in the treatment of persons with vestibular disorders. They participate as full members of the balance treatment teams to recommend and carry out treatment and rehabilitation of impairments of vestibular function". AAA explains further that vestibular rehabilitation therapy is a non-medical treatment process for patients with vestibular deficits and disorders and vestibular treatment and therapy protocols may include but not limited to; CRPs and liberatory manoeuvres for the treatment of BPV, adaptation, substitution and habituation protocols, gaze stabilization exercises, static and dynamic balance activity (Gans et al., 2007). Hence, including vestibular training in the existing Master of Audiology course or developing a separate postgraduate ‘vestibular training component/course’ for students and Audiologists interested in pursuing a career in ‘Vestibular Disorders’ could be an interesting development in the field of Audiology in New Zealand. Such courses already exist in USA and UK and many Audiologists are pursuing careers as ‘Audiologists/Audiological Scientist’ with special interests in vestibular system. These courses could be considered as a guideline for developing vestibular clinical training for Audiologists in New Zealand.
References
References


Appendix
Appendix

Spreadsheet 1 (Patient details)

Spreadsheet 2 (Results of the survey/questionnaire)

Copy of survey questionnaire

Audiograms

  Case no. 0611
  Case no. 0612
  Case no. 0613
<table>
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<th>Length of hospital stay (days)</th>
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**Investigations**

- **MRI?** (Magnetic Resonance Imaging)
- **CT?** (Computed Tomography)
- **Vestibular neuritis**
- **Central origin**
- **Other General notes**
- **Details**

**Diagnoses**

- **BPV** (Benign Paroxysmal Positional Vertigo)
- **SNHL** (Sensorineural Hearing Loss)
- **TIA** (Transient Ischemic Attack)
- **VOR** (Vestibulo-Ocular Reflex)
- **CVS** (Cardiovascular System)
- **Dix Hallpike test**
- **Semont Manoeuvre**
- **Unterburger's test**

**Notes**

- **Patient had gradual onset of dizziness, headache as was unsteady on the feet. Dix Hallpike test was reported to be positive on the right side.**
- **Patient was referred to the Department of ENT during the admission but the clinical notes advised that refer to ENT if the scan was normal.**
- **No evidence of positional tests performed to support the diagnosis of BPV.**
- **No evidence of positional tests performed to support the diagnosis of BPV.**
- **No evidence of positional tests performed to support the diagnosis of BPV.**
- **No evidence of positional tests performed to support the diagnosis of BPV.**

**Comments/Summary**

- **Admitted under the Department of ENT.**
- **Admitted with unsteadiness, giddiness on bending over and turning to the right; no nystagmus was evident with stable vital signs.**
- **Admitted with frontal headache and vertigo with unsteady gait and slurred speech.**
- **Audiogram indicated moderate low frequency sloping to profound high frequency SNHL on the right side.**
- **Notes suggested that the Dix Hallpike test was positive, which was done on 27/7/04.**

**Accuracy (2006 only)**

- **No of days seen**
- **In patient referral to ENT or Audiology**

**Notes from 27/07/04 - 04/05/06**

- **BPV left. Follow up 11/8/04 - No more dysfunction, examination was normal. Frequently followed up for similar problems. Then the right stapedectomy was done. On 19/6/06, seen by the ENT clinician - Hallpike was positive, brisk on the left side. Left posterior canal BPV did persist. L-R Semont manoeuvre was performed. After 20-30 second interval, sudden vertigo and nystagmus to the left.**
- **BPV left. Follow up 11/8/04 - No more dysfunction, examination was normal. Frequently followed up for similar problems. Then the right stapedectomy was done. On 19/6/06, seen by the ENT clinician - Hallpike was positive, brisk on the left side. Left posterior canal BPV did persist. L-R Semont manoeuvre was performed. After 20-30 second interval, sudden vertigo and nystagmus to the left.**
- **BPV left. Follow up 11/8/04 - No more dysfunction, examination was normal. Frequently followed up for similar problems. Then the right stapedectomy was done. On 19/6/06, seen by the ENT clinician - Hallpike was positive, brisk on the left side. Left posterior canal BPV did persist. L-R Semont manoeuvre was performed. After 20-30 second interval, sudden vertigo and nystagmus to the left.**

**2004 References**


**2005 References**


**2006 References**

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<th>Length of hospital stay (days)</th>
<th>Admitting Department</th>
<th>General notes</th>
<th>Investigations</th>
<th>Diagnoses</th>
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<td>Month of admission</td>
<td>Length of hospital stay (days)</td>
<td>Admitting Department</td>
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<td>IN?</td>
<td>Admitting diagnosis</td>
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<td>Investigations</td>
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<td>54 years</td>
<td>Sep 2004</td>
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<td>General Medicine</td>
<td>1</td>
<td></td>
<td>Patient got up from sleep and felt very dizzy and collapsed, long-standing right homonymous hemianopia was evident</td>
<td>Benign positional vertigo</td>
<td>No</td>
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<td>Otolaryngology</td>
<td>1</td>
<td></td>
<td>Patient had a constant gait with headache, vomiting and positional nausea.</td>
<td>Benign positional vertigo</td>
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<td>1</td>
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<td>Patient had diplopia and double vision with normal vertical but no cause was detected.</td>
<td>Benign positional vertigo</td>
<td>No</td>
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<td>Mar 2004</td>
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<td>General Medicine</td>
<td>1</td>
<td></td>
<td>Patient had positional vertigo and relieved by lying down, Positional non-sustained nystagmus was present.</td>
<td>Benign positional vertigo</td>
<td>No</td>
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<td>F</td>
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<td>Dec 2004</td>
<td>5 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td>Patient had sudden positional vertigo, nausea was present with possibility but there was no nystagmus.</td>
<td>Benign positional vertigo</td>
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<td>Patient had positional vertigo was present</td>
<td>Benign positional vertigo</td>
<td>No</td>
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<td>23 days</td>
<td>Neurology</td>
<td>1</td>
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<td>Patient had sudden neurology with left sided hearing and imbalance in the left ear.</td>
<td>Benign positional vertigo</td>
<td>Yes</td>
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<td>Sep 2004</td>
<td>1 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td>Patient had feeling that the leg was weak with the patient had defecation and was admitted in a state of partial coma with left sided dizziness and clonic, general weakness with transient hemiparesis.</td>
<td>Benign positional vertigo</td>
<td>No</td>
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<td>Sep 2004</td>
<td>9 days</td>
<td>General Medicine</td>
<td>1</td>
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<td>Patient had sudden neurology and shake past, Benign positional vertigo with 3 recent falls and a known case of Meniere's disease. No nystagmus observed.</td>
<td>Benign positional vertigo</td>
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<tr>
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<td>Oct 2004</td>
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<td>General Medicine</td>
<td>1</td>
<td></td>
<td>Patient had sudden vertigo with nausea. History of 2 current year of vertigo with the left posterior cerebral artery was seen. Unilateral deafness with deaf left ear and the right eye vision was present. Preparatory nystagmus on right head turn was evident.</td>
<td>Benign positional vertigo</td>
<td>No</td>
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<tr>
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<td>Neurology</td>
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<td>Patient had acute vertigo with nausea. History of 23 years of vertigo with the left posterior cerebral artery was seen. Unilateral deafness with deaf left ear and the right eye vision was present. Preparatory nystagmus on right head turn was evident.</td>
<td>Benign positional vertigo</td>
<td>No</td>
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<td>59 years</td>
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<td>Neurology</td>
<td>1</td>
<td></td>
<td>Patient had sudden vertigo neurology and hearing loss and imbalance were evident. This was a known case of Meniere's disease.</td>
<td>Benign positional vertigo</td>
<td>No</td>
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<tr>
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<td>6 days</td>
<td>General Medicine</td>
<td>1</td>
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<td>Patient was dizzy when standing and had 2 falls with known history of Meniere's disease.</td>
<td>Benign positional vertigo</td>
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<td>Jul 2004</td>
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<td>General Medicine</td>
<td>1</td>
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<td>Patient had sudden vertigo, nausea and vomiting. Nystagmus was present with the fast phase to the right on horizontal but no cause was evident.</td>
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<td>No</td>
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<td>Jul 2004</td>
<td>1 days</td>
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<td>1</td>
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<td>Sudden gait and vomiting this morning, fell faintly for 10 seconds, left ear hearing loss was present. Horizontal nystagmus was present with fixed left gaze.</td>
<td>Benign positional vertigo</td>
<td>Yes</td>
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<tr>
<td>0441</td>
<td>M</td>
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<td>Jun 2004</td>
<td>4 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td>Patient had sudden vertigo, nausea and vomiting was present. Nystagmus on left lateral and upward gaze was evident.</td>
<td>Benign positional vertigo</td>
<td>Yes</td>
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<tr>
<td>0442</td>
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<td>81 years</td>
<td>May 2004</td>
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<td>General Medicine</td>
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<td>Patient had dizziness, vertigo, nystagmus. The Dix Hallpike test was negative.</td>
<td>Benign positional vertigo</td>
<td>No</td>
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</table>
THE DIAGNOSIS WAS NOT SUBSTANTIATED.

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age at admission</th>
<th>Month of admission</th>
<th>Length of hospital stay (days)</th>
<th>Admitting Department</th>
<th>CT?</th>
<th>MRI?</th>
<th>BPV?</th>
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<th>Vestibular neuritis</th>
<th>Migraine vertigo</th>
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<td>General Medicine</td>
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<td></td>
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<td>No, but audiogram done privately on 21/04 - Mild mid frequency hearing loss to moderately severe high frequency on the right side and severe high frequency loss on the left side. Symmetrical caloric test in the congruent field was evident. (Bone conduction was not carried out).</td>
<td>THE DIAGNOSIS WAS NOT SUBSTANTIATED.</td>
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<td>No, but audiogram done privately on 21/04 - Mild mid frequency hearing loss to moderately severe high frequency on the right side and severe high frequency loss on the left side. Symmetrical caloric test in the congruent field was evident. (Bone conduction was not carried out).</td>
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<td>No, but audiogram done privately on 21/04 - Mild mid frequency hearing loss to moderately severe high frequency on the right side and severe high frequency loss on the left side. Symmetrical caloric test in the congruent field was evident. (Bone conduction was not carried out).</td>
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<td>THE DIAGNOSIS WAS NOT SUBSTANTIATED.</td>
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<td>THE DIAGNOSIS WAS NOT SUBSTANTIATED.</td>
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<td>THE DIAGNOSIS WAS NOT SUBSTANTIATED.</td>
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<td>Sex</td>
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<td>Month of admission</td>
<td>Length of hospital stay (days)</td>
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<td>Comments/Summary</td>
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<td>Developed vestibular function, unspecified</td>
<td>Vertigo was supported. Audiology recommended.</td>
<td>ENT follow up advised in the notes.</td>
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<td>3 days</td>
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<td>Developed vestibular function, unspecified</td>
<td>Vertigo was supported. Audiology recommended.</td>
<td>ENT follow up advised in the notes.</td>
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<td>73</td>
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<td>6 days</td>
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<td>Periphera vestibular disorder</td>
<td>Rare evidence of dizziness/nausea and vertigo in relation to the head movement with nystagmus was present.</td>
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<td>No</td>
<td>No</td>
<td>Yes</td>
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<td>Neurology</td>
<td>Normal</td>
<td>1</td>
<td>Developed vestibular function, unspecified</td>
<td>Rare evidence of dizziness/nausea and vertigo in relation to the head movement with nystagmus was present.</td>
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<td>Developed vestibular function, unspecified</td>
<td>Rare evidence of dizziness/nausea and vertigo in relation to the head movement with nystagmus was present.</td>
<td>Yes</td>
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<td>No</td>
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<td>Other peripheral vertigo</td>
<td>Patient presented with complaints of vertigo (left ear), nausea and vomiting.</td>
<td>No</td>
<td>No</td>
<td>No</td>
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<td>Other peripheral vertigo</td>
<td>Patient presented with complaints of vertigo (left ear), nausea and vomiting.</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>0463</td>
<td>F</td>
<td>75</td>
<td>Feb 2005</td>
<td>7 days</td>
<td>Neurosurgery</td>
<td>Normal</td>
<td>1</td>
<td>Vestibular function unspecified</td>
<td>Rare evidence of dizziness/nausea and vertigo in relation to the head movement with nystagmus was present.</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>0464</td>
<td>F</td>
<td>47</td>
<td>Feb 2005</td>
<td>3 days</td>
<td>Neurology</td>
<td>Normal</td>
<td>1</td>
<td>Other peripheral vertigo</td>
<td>Patient presented with complaints of vertigo.</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>0465</td>
<td>M</td>
<td>42</td>
<td>Sep 2005</td>
<td>9 days</td>
<td>Neurosurgery</td>
<td>Normal</td>
<td>1</td>
<td>Temporal fracture</td>
<td>Patient presented with complaints of vertigo.</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>0466</td>
<td>F</td>
<td>64</td>
<td>Apr 2005</td>
<td>5 days</td>
<td>General Medicine</td>
<td>Ménieré's disease</td>
<td>1</td>
<td>Wake up with vertigo, nausea and vomiting.</td>
<td>Rare evidence of dizziness/nausea and vertigo in relation to the head movement with nystagmus was present.</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

**Investigations:**
- **CENTRAL CAUSE OF DIZZINESS AND VERTIGO WAS NOTICED. THE DIAGNOSIS SUPPORTED: “Disordered vestibular function, unspecified.”**
- **SUDDEN IDIOPATHIC SNHL WITH VERTIGO.**
- **Sudden objective vertigo, nausea, nystagmus on frontal gaze(2nd degree) with vomiting. Left ear pain, left sided facial discomfort, tinnitus in the left ear, left sided occipital pain with likely labyrinthitis.**
- **Acute vertigo with nausea and vomiting in the left ear, hearing loss in the left ear. Spontaneous nystagmus was present.**
- **Woke up with vertigo, nausea and vomiting.**
- **Recent CT scan showed presence of small vessel infarcts in the brain.**
- **Went to the ER with complaints of vertigo.**

**Diagnoses:**
- **Other peripheral vertigo**
- **Peripheral vestibular disorder**
- **Ménieré’s disease**
- **Central origin**
- **General notes**
  - **Details**
  - **Comments/Summary**
<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age at admission</th>
<th>Month of admission</th>
<th>Length of hospital stay (days)</th>
<th>Admitting Department</th>
<th>CT?</th>
<th>MRI?</th>
<th>Eagle Hallpike test details?</th>
<th>Inpatient referral to ENT or Audiology?</th>
<th>Diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td>0505 M</td>
<td>M</td>
<td>52 years</td>
<td>Nov 2005</td>
<td>4 days</td>
<td>Neurology</td>
<td>No evidence of new stroke but patient had previous history of T2 hyperintensities seen on previous MRI scan in 2004</td>
<td>1</td>
<td>No</td>
<td>Ventricular collapse approximately 4 times with loss of consciousness at times. Ataxia related to head movement and position. Dix-Hallpike test was positive (11/10/05). Head turned to the right - Rotation nystagmus to the right. Modified Epley Maneuver was carried out.</td>
<td>No, but notes suggesting ENT follow up in case of future similar problems.</td>
</tr>
<tr>
<td>0506 M</td>
<td>M</td>
<td>79 years</td>
<td>Jun 2005</td>
<td>3 days</td>
<td>General Medicine</td>
<td>No</td>
<td>No</td>
<td>Patient awoke with dizziness. He was nauseated, unsteady on feet with right sided dysarthria and weakness following the H/O stroke.</td>
<td>No</td>
<td>POSITIVE EVIDENCE OF POSITIONAL TEST PERFORMED TO SUPPORT THE DIAGNOSIS OF BPV</td>
</tr>
<tr>
<td>0507 F</td>
<td>F</td>
<td>81 years</td>
<td>Apr 2005</td>
<td>5 days</td>
<td>General Medicine</td>
<td>No</td>
<td>No</td>
<td>Admitted with dizziness preceded by movement. Dix-Hallpike Maneuver (teleic) induced some of the symptoms on testing (discharge notes). No positional test details are found in the clinical notes.</td>
<td>No</td>
<td>NO SATISFACTORY EVIDENCE OF POSITIONAL TESTS TO SUPPORT THE DIAGNOSIS OF BPV</td>
</tr>
<tr>
<td>0508 M</td>
<td>M</td>
<td>67 years</td>
<td>Oct 2005</td>
<td>3 days</td>
<td>Neurology</td>
<td>No</td>
<td>No</td>
<td>Patient was previously seen in the Department of Neurology with recurrent vertigo. The tentative diagnosis was that of TIA. Then subsequently the patient presented with vertigo, nausea and headache worse with head turning to the left. Rotatory nystagmus was observed on the left. Dix-Hallpike test was positive with nystagmus to the left. Dix-Hallpike test done on 11/10/05 was negative. Thierry's dizziness in positional testing (? missing the diagnosis).</td>
<td>No</td>
<td>NO EVIDENCE FOR THE DIAGNOSIS OF BPV AS THE DIX HALLPIKE TEST WAS NEGATIVE.</td>
</tr>
<tr>
<td>0509 F</td>
<td>F</td>
<td>90 years</td>
<td>Feb 2005</td>
<td>1 days</td>
<td>Emergency Department</td>
<td>No</td>
<td>No</td>
<td>Patient presented with dizziness, nausea and positional vertigo. She was seen previously in the Emergency Department with similar symptoms and was diagnosed as a case of BPV. No positional testing was done and patient recovered by the next day.</td>
<td>No</td>
<td>NO SATISFACTORY EVIDENCE OF POSITIONAL TEST PERFORMED TO SUPPORT THE DIAGNOSIS OF BPV</td>
</tr>
<tr>
<td>0510 F</td>
<td>F</td>
<td>78 years</td>
<td>Aug 2005</td>
<td>6 days</td>
<td>General Medicine</td>
<td>Normal (Postural pressure reduced, asymmetry of right visual field/hemianopia, Hearing and smell test revealed mild bilateral hearing loss)</td>
<td>Normal</td>
<td>Patient presented with the positional vertigo, vomiting and delirium. No vertigo present for the previous 2 years. Dix-Hallpike test was positive with nystagmus and right ear vertigo. Dix-Hallpike test done on 12/8/05, 2 days post admission. MR/Hospital stay could have been reduced/avoided if BPV was diagnosed on admission.</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0511 M</td>
<td>M</td>
<td>74 years</td>
<td>Aug 2005</td>
<td>1 days</td>
<td>General Medicine</td>
<td>No</td>
<td>No</td>
<td>Patient with nausea exacerbated by head movements. Dix-Hallpike test was negative and there was no nystagmus.</td>
<td>No</td>
<td>NO EVIDENCE FOR THE DIAGNOSIS OF BPV AS THE DIX HALLPIKE TEST WAS NEGATIVE.</td>
</tr>
<tr>
<td>0512 F</td>
<td>F</td>
<td>51 years</td>
<td>Sep 2005</td>
<td>3 days</td>
<td>Neurology</td>
<td>Normal</td>
<td>Normal</td>
<td>Three weeks history of vertigo worse by sleeping on face. Nystagmus observed with positive Dix-Hallpike test. Dix-Hallpike test was positive with nystagmus and vertigo on the right. Dix-Hallpike test performed (MRI was negative). Dix-Hallpike test repeated on 10/9/05. Dix-Hallpike test was negative.</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0513 F</td>
<td>F</td>
<td>76 years</td>
<td>Jun 2005</td>
<td>4 days</td>
<td>General Medicine</td>
<td>No</td>
<td>No</td>
<td>Sudden vertigo, hearing, nausea worse with movement and lasts for 4 days. Spontaneous nystagmus to the right side. Dix-Hallpike test with 7.5 flicks in midline of ear. Dizzy ++ turn R &gt; L (22/06/05 clinic notes).</td>
<td>No</td>
<td>NO EVIDENCE FOR THE DIAGNOSIS OF BPV AS THE DIX HALLPIKE TEST WAS NEGATIVE.</td>
</tr>
<tr>
<td>0514 M</td>
<td>M</td>
<td>22 years</td>
<td>Aug 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>No</td>
<td>No</td>
<td>Sudden vertigo, hearing, nausea worse with movement and lasts for 1 day. Spontaneous nystagmus to the right side. Dix-Hallpike test was negative by the end of the day and Dix-Hallpike test was negative on 20/08/05.</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0515 F</td>
<td>F</td>
<td>76 years</td>
<td>Dec 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>No</td>
<td>No</td>
<td>Sudden vertigo, nausea, vomiting, dizziness in the left ear with H/O mastoidectomy on the left ear leading to deaf left ear. Mild nystagmus with left phase to the left. Dix-Hallpike test was negative.</td>
<td>No</td>
<td>NO EVIDENCE FOR THE DIAGNOSIS OF BPV AS THE DIX HALLPIKE TEST WAS NEGATIVE.</td>
</tr>
<tr>
<td>0516 M</td>
<td>M</td>
<td>74 years</td>
<td>Aug 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>No</td>
<td>No</td>
<td>Abdominal with dizziness and unsteadiness on feet. Nystagmus to the left side and Dix-Hallpike test was negative.</td>
<td>No</td>
<td>NO EVIDENCE FOR THE DIAGNOSIS OF BPV AS THE DIX HALLPIKE TEST WAS NEGATIVE.</td>
</tr>
<tr>
<td>Case</td>
<td>Sex</td>
<td>Age at admission</td>
<td>Month of admission</td>
<td>Length of hospital stay (days)</td>
<td>Admitting Department</td>
<td>CT?</td>
<td>MRI?</td>
<td>gPV?</td>
<td>Measuring diastasis results</td>
<td>Otolaryngology / ENT details?</td>
</tr>
<tr>
<td>-------</td>
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<td>------------------</td>
</tr>
<tr>
<td>0517</td>
<td>F</td>
<td>70 years</td>
<td>Jul 2005</td>
<td>7 days</td>
<td>Otolaryngology</td>
<td></td>
<td></td>
<td></td>
<td>Normal</td>
<td>Yes, ENT admitted, provided torsional nystagmus with head down to the left position. Particle repositioning manoeuvre was done by the ENT Specialist.</td>
</tr>
<tr>
<td>0518</td>
<td>F</td>
<td>72 years</td>
<td>Feb 2005</td>
<td>5 days</td>
<td>General Medicine</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0519</td>
<td>M</td>
<td>82 years</td>
<td>Feb 2005</td>
<td>8 days</td>
<td>General Medicine</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0520</td>
<td>F</td>
<td>54 years</td>
<td>Sep 2006</td>
<td>3 days</td>
<td>Emergency Department</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0521</td>
<td>M</td>
<td>77 years</td>
<td>Feb 2005</td>
<td>1 days</td>
<td>General Medicine</td>
<td></td>
<td></td>
<td></td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>0522</td>
<td>F</td>
<td>54 years</td>
<td>May 2005</td>
<td>1 days</td>
<td>Emergency Department</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0523</td>
<td>M</td>
<td>73 years</td>
<td>Oct 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>Normal (Possibility a small infarct in the midline of brainstem.)</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>0524</td>
<td>M</td>
<td>68 years</td>
<td>Jan 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0525</td>
<td>F</td>
<td>71 years</td>
<td>Jul 2005</td>
<td>8 days</td>
<td>General Medicine</td>
<td>Normal (previous ischaemic changes were noted).</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
</tr>
<tr>
<td>0526</td>
<td>M</td>
<td>65 years</td>
<td>Feb 2005</td>
<td>1 days</td>
<td>Emergency Department</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0527</td>
<td>M</td>
<td>79 years</td>
<td>Aug 2005</td>
<td>3 days</td>
<td>General Medicine</td>
<td></td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0528</td>
<td>M</td>
<td>89 years</td>
<td>Feb 2005</td>
<td>11 days</td>
<td>General Medicine</td>
<td></td>
<td></td>
<td></td>
<td>Yes, patient confirmed the diagnosis of BPV by positive Dix Hallpike test on the right ear. Treated with Epley manoeuvre.</td>
<td>Yes, had an audiogram with mild sloping to profound low to high frequency SNHL.</td>
</tr>
<tr>
<td>0529</td>
<td>M</td>
<td>61 years</td>
<td>Apr 2005</td>
<td>4 days</td>
<td>General Medicine</td>
<td>Normal</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0530</td>
<td>F</td>
<td>87 years</td>
<td>Jan 2005</td>
<td>8 days</td>
<td>General Medicine</td>
<td>Normal</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>0531</td>
<td>M</td>
<td>61 years</td>
<td>Apr 2005</td>
<td>4 days</td>
<td>General Medicine</td>
<td>Normal</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Case</td>
<td>Age at admission (years)</td>
<td>Month of admission</td>
<td>Length of hospital stay (days)</td>
<td>Admitting Department</td>
<td>CT?</td>
<td>MRI?</td>
<td>Other</td>
<td>Investigations</td>
<td>Diagnoses</td>
<td>Inpatient refer to ENT/Otolaryngology?</td>
</tr>
<tr>
<td>-------</td>
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</tr>
<tr>
<td>0532</td>
<td>M 83 years</td>
<td>Jun 2005</td>
<td>2 days</td>
<td>Emergency Department</td>
<td>1</td>
<td></td>
<td></td>
<td>Positional vertigo, vomiting,眩暈, ataxia, ophthalmoplegia: symptoms on the left side and right side, dizziness resulted in falling on the left side, followed by vertigo and vomiting were seen. Conflicting description of tinnitus was noted.</td>
<td>Yes, admitted under the Department of ENT</td>
<td>No</td>
</tr>
<tr>
<td>0533</td>
<td>M 38 years</td>
<td>Mar 2005</td>
<td>2 days</td>
<td>Emergency Department</td>
<td>1</td>
<td></td>
<td></td>
<td>Severe daily with left sided tinnitus, nausea and vomiting was present. Nystagmus with fast phase to the left side and core-biased hearing. Similar event noted in the Emergency Department in January this year.</td>
<td>Yes, yes mid-line frequency tonus on the left side.</td>
<td>YES</td>
</tr>
<tr>
<td>0534</td>
<td>M 46 years</td>
<td>May 2005</td>
<td>2 days</td>
<td>Emergency Department</td>
<td>1</td>
<td></td>
<td></td>
<td>Gradual onset of vertigo, nausea, vomiting,眩暈, and auditory nystagmus was seen. Emergency Department rules suggest past diagnosis of Menière’s disease by an ENT specialist.</td>
<td>Regular ENT follow up. Schoo - Proven Meniere’s disease.</td>
<td>No</td>
</tr>
<tr>
<td>0535</td>
<td>F 65 years</td>
<td>Aug 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient presented with acute vertigo while lying supine and vomiting. GR evaluated left sided nystagmus, five similar episodes in last 30 years. History of tinnitus.</td>
<td>No, follow up with the ENT soon.</td>
<td>No</td>
</tr>
<tr>
<td>0536</td>
<td>F 77 years</td>
<td>Aug 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Complaints of dizziness were present for last 6 months, vertigo on getting up, unsteady gait, Tinnitus present in the right ear with tinnitus. The Dix Hallpike test was not done as it was negative recently.1 (Tinnitus and vertigo).</td>
<td>Yes, yes mid-line frequency tonus on the left side.</td>
<td>Yes</td>
</tr>
<tr>
<td>0537</td>
<td>M 66 years</td>
<td>Aug 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient experienced vertigo in bed while lying down, which ceased when he sat up. No nystagmus was seen in the sitting position. The Dix Hallpike test was negative.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
<tr>
<td>0538</td>
<td>F 57 years</td>
<td>Aug 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient admitted with acute vertigo, nausea and vomiting.</td>
<td>Yes, diagnosis confirmed. ENT visit soon after the discharge.</td>
<td>No</td>
</tr>
<tr>
<td>0539</td>
<td>F 77 years</td>
<td>Apr 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient presented with acute vertigo, nausea and vomiting in the morning on rising, no nystagmus was seen.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
<tr>
<td>0540</td>
<td>F 65 years</td>
<td>Jan 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient had nausea with light headedness worse on movement, tinnitus, hearing problems on admission and complete of reduced hearing in the right ear. No nystagmus was present on the following day.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
<tr>
<td>0541</td>
<td>F 78 years</td>
<td>Aug 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>No nystagmus and the Dix Hallpike manoeuvre (head) was negative.</td>
<td>No</td>
<td>NO CLINICAL EVIDENCE WAS PRESENT TO SUPPORT THE DIAGNOSIS.</td>
</tr>
<tr>
<td>0542</td>
<td>M 57 years</td>
<td>Dec 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient presented with dizziness, nausea on moving the head. Nystagmus with fast component to the left side was present.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
<tr>
<td>0543</td>
<td>M 75 years</td>
<td>Aug 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient presented with dizziness, nausea on moving the head. Nystagmus with fast component to the left side was present.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
<tr>
<td>0544</td>
<td>F 65 years</td>
<td>March 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient presented with dizziness, nausea on moving the head. Nystagmus with fast component to the left side was present.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
<tr>
<td>0545</td>
<td>F 78 years</td>
<td>Dec 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient presented with dizziness, nausea on moving the head. Nystagmus with fast component to the left side was present.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
<tr>
<td>0546</td>
<td>M 75 years</td>
<td>Sep 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>No vertigo, tinnitus and dizziness noted.</td>
<td>No</td>
<td>NO SATISFACTORY CLINICAL EVIDENCE ON ADMISSION OR RECENT PAST HISTORY SUGGESTIVE OF VESTIBULAR NEURITIS.</td>
</tr>
<tr>
<td>0547</td>
<td>M 52 years</td>
<td>Jul 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient presented with acute vertigo, nausea and headache but no pharmacological symptoms. No nystagmus head down was noted.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
<tr>
<td>0548</td>
<td>F 65 years</td>
<td>Feb 2005</td>
<td>2 days</td>
<td>General Medicine</td>
<td>1</td>
<td></td>
<td></td>
<td>Patient presented with acute vertigo, nausea and vomiting.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
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<tr>
<td>0549</td>
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<td>Nov 2005</td>
<td>2 days</td>
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<td>1</td>
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<td></td>
<td>Patient presented with acute vertigo, nausea and vomiting.</td>
<td>No</td>
<td>NO ENT REFERAL AND SO THE DIAGNOSIS WAS TOTALY SPECULATIVE.</td>
</tr>
</tbody>
</table>

**Central**

**Other**

**Commentary**

- Inpatient refer to ENT/Otolaryngology?: Indicates whether the patient was referred to the ENT/Otolaryngology department for further evaluation.
- Outpatient referral to ENT or Audiology?: Indicates whether the patient was referred for outpatient follow-up.

**Investigations**

- CT?: Indicates whether a computerized tomography (CT) scan was performed.
- MRI?: Indicates whether a magnetic resonance imaging (MRI) scan was performed.
- Other: Indicates any other investigations not mentioned in the previous fields.

**Diagnoses**

- Positional vertigo: Vertigo caused by change in head position.
- Nystagmus: Rapid eye movements, typically quick jerks or oscillations.
- Hearing loss: Reduction in the ability to hear.
- Tinnitus: Ringing or roaring in the ears.
- Ataxia: Inability to maintain balance.
- Vertigo: A spinning sensation or feeling of movement.
- Diagnoses: Specific conditions such as Ménière's disease, vestibular neuritis, and migraines.

**Comments/Summary**

- ENT or Audiology: Department where the patient was referred.
- Positional vertigo, vomiting, unsteadiness, sensorineural hearing loss on the right and chronic tinnitus was present. Right aural pressure and tinnitus followed by vertigo and vomiting was seen. Conflict in Department 1.
- Patient had nausea with left sided tinnitus, nausea and vomiting was present. Nystagmus with fast phase to the left side and core-biased hearing. Similar event noted in the Emergency Department in January this year.
- Patient presented with acute vertigo while lying supine and vomiting. GP evaluated left sided nystagmus, five similar episodes in last 30 years. History of tinnitus.
- Patient experienced vertigo in bed while lying down, which ceased when he sat up. No nystagmus was seen in the sitting position. The Dix Hallpike test was negative.
- Patient admitted with acute vertigo, vomiting, poor gait and horizontal spontaneous nystagmus to the right side.
- Patient presented with rotational vertigo, nausea, ataxia and rotational/horizontal nystagmus on the left.
- Patient had nausea with light headedness worse on movement, tinnitus, vomiting, unsteady gait, nystagmus on admission and complaints of reduced hearing in the right ear. No nystagmus was present on the following day.
- Patient suffered from vertigo while moving the head. Nystagmus with fast component to the left side was present. The Dix Hallpike test was negative.
- Patient presented with dizziness, nausea on moving the head. Nystagmus with fast component to the left side was present. The Dix Hallpike test was negative.
- Patient had nausea with light headedness worse on movement, tinnitus, vomiting, unsteady gait, nystagmus on admission and complaints of reduced hearing in the right ear. No nystagmus was present on the following day.
<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age at admission</th>
<th>Month of admission</th>
<th>Length of hospital stay (days)</th>
<th>Admitting Department</th>
<th>CT?</th>
<th>MRI?</th>
<th>Otolaryngology / ENT details?</th>
<th>Audiology / Audio details?</th>
<th>Comments/Summary</th>
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<td>No</td>
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<td>53 years</td>
<td>May 2006</td>
<td>3 days</td>
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<td>F</td>
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<td>Aug 2006</td>
<td>1 days</td>
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<td>0</td>
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<td>Yes</td>
<td>0.</td>
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<td>Yes</td>
<td>0.</td>
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<td>Sep 2006</td>
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<td>Yes</td>
<td>0.</td>
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<td>0605</td>
<td>F</td>
<td>57 years</td>
<td>Jun 2006</td>
<td>7 days</td>
<td>Neurosurgery</td>
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<td>0.</td>
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<td>Jul 2006</td>
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<td>Apr 2006</td>
<td>6 days</td>
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<td>0</td>
<td>Yes</td>
<td>Yes</td>
<td>0.</td>
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<tr>
<td>Case</td>
<td>Sex</td>
<td>Age at admission</td>
<td>Month of admission</td>
<td>Length of hospital stay (days)</td>
<td>Regional centre</td>
<td>Admitting Department</td>
<td>Investigations</td>
<td>Diagnoses</td>
<td></td>
<td></td>
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<td>-----</td>
<td>-----------------</td>
<td>--------------------</td>
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<td>---------------------</td>
<td>--------------</td>
<td>-----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0606 F</td>
<td>65 years</td>
<td>Jul 2006</td>
<td>1 days</td>
<td>Orthopaedic</td>
<td>1</td>
<td>CT</td>
<td>Presented with rotational vertigo, nausea and vomiting. Symptoms triggered by neck flexion and extension. Yes, VOR was negative. Romberg sign was positive. Dix-Hallpike test was positive on the right side. No ear symptoms.</td>
<td>Right ear BPV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0607 M</td>
<td>67 years</td>
<td>Jul 2006</td>
<td>2 days</td>
<td>Orthopaedic</td>
<td>1</td>
<td>CT</td>
<td>Patient was noted to have a 3117604 and had a twisted bib and was referred to Orthopaedics. Surgeon for the same. Then developed complaints of positional vertigo and tinnitus and was referred to the Department of ENT. Yes, patient was seen by the ENT clinician on 16/08/06. Dix-Hallpike test was positive on left side. Dix-Hallpike test was performed. In a follow up, 10 days later at the Department of ENT, the Dix-Hallpike test was negative.</td>
<td>Ménière’s disease</td>
<td></td>
<td></td>
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<tr>
<td>0610 F</td>
<td>56 years</td>
<td>Nov 2006</td>
<td>6 days</td>
<td>Neurosurgery</td>
<td>1</td>
<td>CT</td>
<td>Positional vertigo and tinnitus and referred to the Department of ENT.</td>
<td>Benign positional vertigo</td>
<td></td>
<td></td>
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<tr>
<td>0611 F</td>
<td>93 years</td>
<td>Aug 2006</td>
<td>6 days</td>
<td>General Medicine</td>
<td>1</td>
<td>CT</td>
<td>INVESTIGATIONS: The EchoG carried out showed positive results for the right ear with increased absolute SP amplitude of 4.88µV at 1 kHz and 2.92µV at 2 kHz. DIAGNOSIS: Meniere’s disease, right ear.</td>
<td>Meniere’s disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0612 M</td>
<td>61 years</td>
<td>Jun 2006</td>
<td>3 days</td>
<td>General Medicine</td>
<td>1</td>
<td>CT</td>
<td>Yes, seen by an ENT clinician. No ear symptoms or headache was present. 3 vertigo attacks per year were present. Echog showed absolute SP amplitudes increased to 18.50 and 10.25µV at 1 and 2 kHz respectively confirming endolymphatic hydrops in the right ear. Diagnosis - Meniere’s disease.</td>
<td>Hypercompliant tympanic membrane</td>
<td></td>
<td></td>
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<td>0613 M</td>
<td>39 years</td>
<td>Nov 2006</td>
<td>1 days</td>
<td>Otolaryngology</td>
<td>1</td>
<td>CT</td>
<td>Yes, seen in 3 HOURS by the ENT specialist after the admission. VOR - Normal. Dix-Hallpike test was normal. Dix-Hallpike test was done that showed increased absolute SP amplitudes increased to 18.50 and 10.25µV at 1 and 2 kHz respectively using tone burst stimulus. Dix-Hallpike test was positive on the right side to confirm the diagnosis of Meniere’s disease.</td>
<td>Meniere’s disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0614 M</td>
<td>73 years</td>
<td>May 2006</td>
<td>4 days</td>
<td>General Medicine</td>
<td>Normal (No new diagnosis)</td>
<td>1</td>
<td>Yes, patient was admitted with sudden onset of vertigo, nausea and vomiting. Vomiting lasted for 18 hours. One previous similar episode around 20 years ago. Yes, mild symmetrical high frequency hearing loss in the left ear and mild to moderately severe high frequency hearing loss in the right ear. Yes, seen within 5 HOURS by an ENT specialist after the admission. VOR - Normal. Dix-Hallpike test was normal. Dix-Hallpike test was done that showed increased absolute SP amplitudes increased to 18.50 and 10.25µV at 1 and 2 kHz respectively using tone burst stimulus. Dix-Hallpike test was positive on the right side to confirm the diagnosis of Meniere’s disease.</td>
<td>Meniere’s disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0615 F</td>
<td>48 years</td>
<td>Jul 2006</td>
<td>2 days</td>
<td>Emergency Department</td>
<td>1</td>
<td>CT</td>
<td>Yes, patient was admitted with sudden onset of vertigo, nausea and vomiting at 2am with frontal headache. Yes, seen by an ENT clinician. Third degree nystagmus horizontal to the left was present. Head thrust test was positive on the left. Yes, mild symmetrical horizontal hearing loss in the ear and right ear.</td>
<td>Meniere’s disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Case</td>
<td>Month of admission</td>
<td>Length of hospital stay (days)</td>
<td>Admitting Department</td>
<td>Age at admission (years)</td>
<td>Sex</td>
<td>Admitting details</td>
<td>Other</td>
<td>Inv#</td>
<td>Inv#</td>
<td>Ref to ENT or Audiology details</td>
</tr>
<tr>
<td>------</td>
<td>-------------------</td>
<td>-----------------------------</td>
<td>----------------------</td>
<td>-------------------------</td>
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<td>------</td>
<td>------</td>
<td>-------------------------------</td>
</tr>
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<td>0616</td>
<td>Aug 2006</td>
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<td>General Medicine</td>
<td>54</td>
<td>F</td>
<td>Normal</td>
<td></td>
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<td>Yes, mild nystagmus noticed to the left. Dix-Hallpike test was negative. No, bilateral moderately severe SNHL was present.</td>
</tr>
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<td>0617</td>
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<td></td>
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<td></td>
<td>Yes, second degree left beating nystagmus and right beating VOR was seen. Patient was falling to the right side. Dix-Hallpike test was negative. No, Hearing 11WHL (Within Normal Limits)</td>
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<td>0618</td>
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<td>Emergency Department</td>
<td>81</td>
<td>M</td>
<td>Normal</td>
<td></td>
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<td>Yes, VOR - Nystagmus on looking to the left. Dix-Hallpike test was negative. No, moderate bilateral SNHL was present.</td>
</tr>
<tr>
<td>0619</td>
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<td>9</td>
<td>General Medicine</td>
<td>58</td>
<td>F</td>
<td>Normal</td>
<td></td>
<td>1</td>
<td></td>
<td>Yes, VOR was absent and Dix-Hallpike test was negative. No, hearing 11WHL (Within Normal Limits)</td>
</tr>
<tr>
<td>0620</td>
<td>Oct 2006</td>
<td>4</td>
<td>General Medicine</td>
<td>36</td>
<td>M</td>
<td>Normal</td>
<td></td>
<td>1</td>
<td></td>
<td>Yes, no nystagmus, right VOR was absent and Dix-Hallpike test was negative. No, right sided moderate to severe sensorineural hearing loss and left sided mild SNHL was present.</td>
</tr>
<tr>
<td>0621</td>
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<td>Abnormal (3-D Fiesta MR scan and 3-D reconstructions showed probable left vestibular nerve (left vestibular nerve lost))</td>
<td>1 (Other peripheral vertigo)</td>
<td>Yes 0</td>
</tr>
<tr>
<td>0622</td>
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<td>Emergency Department</td>
<td>76</td>
<td>F</td>
<td>Normal</td>
<td>1</td>
<td>Abnormal (3-D Fiesta MR scan and 3-D reconstructions showed probable left vestibular nerve (left vestibular nerve lost))</td>
<td>1 (Other peripheral vertigo)</td>
<td>Yes 0</td>
</tr>
<tr>
<td>0623</td>
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<td>Emergency Department</td>
<td>57</td>
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<td>Normal</td>
<td>1</td>
<td>Abnormal (3-D Fiesta MR scan and 3-D reconstructions showed probable left vestibular nerve (left vestibular nerve lost))</td>
<td>1 (Other peripheral vertigo)</td>
<td>Yes 0</td>
</tr>
<tr>
<td>0624</td>
<td>Sep 2006</td>
<td>5</td>
<td>Otolaryngology</td>
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<td>F</td>
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<td>Abnormal (3-D Fiesta MR scan and 3-D reconstructions showed probable left vestibular nerve (left vestibular nerve lost))</td>
<td>1 (Acute idiopathic SNHL)</td>
<td>Yes 0</td>
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<td>0625</td>
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<td>Abnormal (3-D Fiesta MR scan and 3-D reconstructions showed probable left vestibular nerve (left vestibular nerve lost))</td>
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<td>Yes 4</td>
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<td>0626</td>
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<td>Possible 1</td>
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<td>Normal</td>
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<td>No, VOR was normal.</td>
<td>1 (No diagnosis)</td>
<td>Possible 28</td>
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<td>1 (Herpes zoster oticus)</td>
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### Spreadsheet 1: Summary

#### Age at admission

<table>
<thead>
<tr>
<th>Year</th>
<th>Age Mean/StDev</th>
<th>% Female</th>
<th>Length of hospital stay Mean/StDev</th>
</tr>
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<tbody>
<tr>
<td>2004</td>
<td>68 years ±15 years</td>
<td>65%</td>
<td>5 days ±4 days</td>
</tr>
<tr>
<td>2005</td>
<td>64 years ±16 years</td>
<td>48%</td>
<td>3 days ±2 days</td>
</tr>
<tr>
<td>2006</td>
<td>64 years ±15 years</td>
<td>75%</td>
<td>3 days ±2 days</td>
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<table>
<thead>
<tr>
<th>Year</th>
<th>Age Mean/StDev</th>
<th>% Female</th>
<th>Length of hospital stay Mean/StDev</th>
</tr>
</thead>
<tbody>
<tr>
<td>TOTAL 2004-2005</td>
<td>66 years ±16 years</td>
<td>57%</td>
<td>4 days ±3 days</td>
</tr>
<tr>
<td>TOTAL 2004-2006</td>
<td>66 years ±16 years</td>
<td>61%</td>
<td>4 days ±3 days</td>
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#### Admitted Under:

<table>
<thead>
<tr>
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<th>2004</th>
<th>2005</th>
<th>2006</th>
<th>Total</th>
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<td>29</td>
<td>12</td>
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<td>7</td>
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<td>Oncology</td>
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<td>Respiratory</td>
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<td>1</td>
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<td>Rheumatology</td>
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<th>2004</th>
<th>2005</th>
<th>2006</th>
<th>Total</th>
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<tr>
<td>Admitted Under</td>
<td>63</td>
<td>53</td>
<td>28</td>
<td>144</td>
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#### Accuracy:

<table>
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<th>2006</th>
<th>All years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Otolaryngology</td>
<td>65%</td>
<td>100%</td>
<td>100%</td>
<td>86%</td>
</tr>
<tr>
<td>Other Departments</td>
<td>39%</td>
<td>31%</td>
<td>65%</td>
<td>40%</td>
</tr>
<tr>
<td>All Departments</td>
<td>38%</td>
<td>32%</td>
<td>71%</td>
<td>42%</td>
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#### Stay Cost, CT Cost, MRI Cost, Scans Cost, Total Cost

<table>
<thead>
<tr>
<th>Year</th>
<th>Stay cost Mean/StDev</th>
<th>CT cost Mean/StDev</th>
<th>MRI cost Mean/StDev</th>
<th>Scans cost Mean/StDev</th>
<th>Total cost Mean/StDev</th>
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<tbody>
<tr>
<td>2004</td>
<td>$1,655 ±$1,205</td>
<td>$52 ±$74</td>
<td>$77 ±$128</td>
<td>$129 ±$145</td>
<td>$1,784 ±$1,214</td>
</tr>
<tr>
<td>2005</td>
<td>$1,115 ±$733</td>
<td>$32 ±$63</td>
<td>$70 ±$125</td>
<td>$103 ±$148</td>
<td>$1,217 ±$795</td>
</tr>
<tr>
<td>2006</td>
<td>$1,073 ±$699</td>
<td>$22 ±$55</td>
<td>$51 ±$112</td>
<td>$73 ±$129</td>
<td>$1,146 ±$721</td>
</tr>
<tr>
<td>TOTAL 2004-2005</td>
<td>$1,408 ±$1,048</td>
<td>$43 ±$70</td>
<td>$74 ±$126</td>
<td>$117 ±$146</td>
<td>$1,525 ±$1,077</td>
</tr>
</tbody>
</table>
### Spreadsheet 2: Research Survey

<table>
<thead>
<tr>
<th>DHB code number</th>
<th>Admitted under which department?</th>
<th>Chances of In-patient ENT referral?</th>
<th>Follow up care?</th>
<th>Statistical division (%) according to the diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Benign Positional</td>
</tr>
<tr>
<td>1</td>
<td>General Medicine</td>
<td>Sometimes</td>
<td>GP, ENT</td>
<td>Ménière’s disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Vestibular neuritis</td>
</tr>
<tr>
<td>2</td>
<td>Usually not ENT</td>
<td>Sometimes</td>
<td>Depends on cause of vertigo</td>
<td>Migraine associated</td>
</tr>
<tr>
<td>3</td>
<td>General Medicine, ENT</td>
<td>Sometimes</td>
<td>GP, ENT</td>
<td>Vertigo of central</td>
</tr>
<tr>
<td>4</td>
<td>General Medicine, Neurology, ENT</td>
<td>Sometimes</td>
<td>Uncertain</td>
<td>Other</td>
</tr>
<tr>
<td>5</td>
<td>ENT</td>
<td>Sometimes</td>
<td>ENT</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>General Medicine</td>
<td>Usually</td>
<td>ENT</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>General Medicine</td>
<td>Occasionally</td>
<td>ENT, GP</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Emergency Department</td>
<td>Sometimes</td>
<td>ENT, GP</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>ENT</td>
<td>Sometimes</td>
<td>Don’t know</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>General Medicine</td>
<td>Not Usually</td>
<td>ENT</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>ENT, General Medicine</td>
<td>Usually</td>
<td>ENT</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>General Medicine</td>
<td>Sometimes</td>
<td>GP, General Medicine, ENT</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>ENT, General Medicine, Neurology</td>
<td>Mostly</td>
<td>ENT, ENT</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>ENT (75%), General Medicine (25%)</td>
<td>Sometimes</td>
<td>ENT, Neurology, General Medicine</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>ENT, General Medicine, Neurology</td>
<td>Sometimes</td>
<td>ENT</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>ENT, General Medicine</td>
<td>Always</td>
<td>ENT, GP</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>General Medicine, Emergency</td>
<td>Sometimes</td>
<td>Nil</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>General Medicine</td>
<td>Sometimes</td>
<td>GP</td>
<td></td>
</tr>
</tbody>
</table>

### Research Survey Questions

#### Vertigo of central

- Admitted under which department?
- Chances of In-patient ENT referral?
- Follow up care?
- Statistical division (%) according to the diagnosis
  - Benign Positional
  - Ménière’s disease
  - Vestibular neuritis
  - Migraine associated
  - Vertigo of central
  - Other

#### Note:
Whenever statistical division (%) of diagnosis was given as a range, the mean of the range was taken for the purpose of final mathematical calculation.
Cover letter for Survey

To,

ENT Consultant / Otolaryngologist

Dear Doctor

I am a final year Master of Audiology student at the University of Canterbury, Christchurch. I have been working on a research project with Mr. Jeremy Hornibrook, Otolaryngologist and Dr. Greg O'beirne, Audiology Lecturer, as a part of my final year research project.

The research question is: "Can timely audiology/otolaryngology involvement improve the management of patients admitted to hospital with acute vertigo?"

Description and Objectives:
In this research, we are trying to analyse and evaluate the effectiveness of some quick and simple diagnostic procedures in the initial assessment of patients with symptoms of dizziness and vertigo in improving the diagnostic accuracy. We are also trying to investigate if such changes at the initial stage of admission can reduce the length of hospital stay and the number of investigations (especially head CT and MRI Scans) carried out in the process.

As a part of this research, we are trying to collect some data from the Clinical Directors of ENT Departments from all the District Health Boards across New Zealand. We are interested in knowing more about the likely management for a patient admitted to your hospital with acute vertigo. A survey form is attached (please download) - if you can spend few minutes of your valuable time completing and returning this form to us via e-mail or post, we would greatly appreciate it. Please feel free to write your own comments or suggestions in the space provided or on additional pages.

The confidentiality of names / places and in every other aspect will be dully maintained throughout this process.

Kind regards,

Sudarshan Gawankar
2nd Year, Master of Audiology Student
Department of Communication Disorders
University of Canterbury, Christchurch

Phone number: 021 170 7609
Fax number: 03 364 2760 (ATTN: SUDARSHAN)
Vertigo/Dizziness Research Survey

- When a patient presents at your hospital with complaints of acute vertigo, are they admitted under ENT or General Medicine / Neurology?  
  (Please comment)

- If the patient is admitted under General Medicine / Neurology, is an in-patient ENT referral made? Always/Sometimes?  
  (Please comment)

- If admitted under General Medicine / Neurology, what is the usual advice about follow up? e. g. by their GP, by ENT Service etc.  
  (Please comment)

- Kindly provide us with some data from the past or present year from your department to give us some information about the statistical division of following diagnoses for patients with presenting complaints of vertigo or dizziness:  
  (If you do not have these statistics at hand, please provide us with an estimate based on your own or your colleague’s judgement)

  Benign Positional Vertigo: ___________%  
  Meniere’s disease: ___________%  
  Vestibular neuritis: ___________%  
  Migraine associated vertigo: ___________%  
  Vertigo of central origin: ___________%  
  Other: ___________%  

  General Comments (if any):

Consent:

I Dr./Mr. ____________________________ authorise  
Sudarshan Gawankar, Master of Audiology student, University of Canterbury, Christchurch  
Dr. Jeremy Hornibrook, Otolaryngologist, Christchurch Public Hospital  
Dr. Greg O’Beirne, Lecturer in Audiology, University of Canterbury  
and all other associates involved in this research to use the data / information provided above for the research project on vertigo / dizziness.

Signature / Name: ____________________________ Date: ______________
Case no. 0611 Results: Audiogram: Severe to profound sensorineural hearing loss in the right ear and moderate to severe sensorineural hearing loss in the left ear with a large high frequency component. Speech recognition scores - 90% at 90 dB in the right ear. Tympanometry - Type ‘A’ tympanogram indicating normal compliance of the tympanic membrane. DPOAEs - Present. Acoustic reflexes - Normal.
**Case no. 0612 results:** Audiogram - Mild to moderately severe sensorineural hearing loss in the right ear and a severe to profound sensorineural hearing loss in the left ear. Tympanometry - Type ‘Ad’ tympanogram revealing hypercompliant tympanic membrane. The speech scores - 90% at 70 dB. DPOAEs – Present. Acoustic reflexes – Normal.
Case no. 0613 results: Audiogram: Mild to moderately severe sensorineural hearing loss in his right ear and mild high frequency hearing loss in the left ear. Tympanometry - Type ‘As’ tympanogram revealing reduced compliance of the tympanic membrane. Speech recognition scores - 100% at 55 dB in the right ear. DPOAEs – Present. Acoustic reflexes – Normal.