THE EFFECTIVENESS OF TREATMENT OF CERVICOGENIC DIZZINESS WITH MANUAL THERAPY

SUSAN ANNE REID

BAppSc(Phty), Grad Dip Manip Phty, MMedSc(Phty), BPharm

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Statement of Originality

The thesis contains no material which has been accepted for the award of any other degree or diploma in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. I give consent to the final version of my thesis being made available when deposited in the University's Digital Repository, subject to the provisions of the Copyright Act 1968.

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Statement of Authorship

I hereby certify that this thesis is in the form of a series of published papers of which I am a joint author. I have included as part of the thesis a written statement from each co-author, endorsed by the Assistant Dean (Research Training), Faculty of Health and Medicine attesting to my contribution to the joint publications (Appendix A).

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Susan Reid Date:

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Publications and presentations

The following publications and presentations were a direct result of the work completed in this thesis:

Published and submitted papers included as part of this thesis

1. Reid, SA, Rivett, DA, Katekar, M, Callister, R. (2012) Efficacy of manual therapy treatments for people with cervicogenic dizziness and pain: protocol of a randomised controlled trial. *BMC Musculoskeletal Disorders* 13: 201-208.
2. Reid, S.A. Rivett, D.A. Katekar, M. & Callister, R. (2014) Comparison of Mulligan sustained natural apophyseal glides and Maitland mobilizations for treatment of cervicogenic dizziness: a randomized controlled trial. *Physical Therapy* 94: 466-476.
3. Reid, SA. Rivett, DA. Katekar, M. & Callister, R. (2014) The effects of cervical spine manual therapy on cervical range of motion, head repositioning and balance in participants with cervicogenic dizziness: a randomized controlled trial. *Archives of Physical Medicine and Rehabilitation.* DOI: http://dx.doi.org/10.1016/j.apmr.2014.04.009.
4. Reid SA, Callister R, Snodgrass S, Katekar MG, Rivett RA (2014) Manual therapy treatment of cervicogenic dizziness: long-term outcomes of a randomised controlled trial. *Manual Therapy* (Accepted 27/6/2014 and in press).
5. Reid SA, Callister R, Katekar MG, Rivett RA, (2014) Identification of individuals with cervicogenic dizziness with evidence from recruitment for a randomised controlled trial. *Manual Therapy* (Under review).

Published abstracts

1. Reid Susan, Rivett Darren Anthony, Callister Robin, Katekar Michael Gerard, (2011) The identification of patients with cervicogenic dizziness. World Physical

Therapy, Amsterdam. WPT2011, Special Interest Report Abstracts. Physiotherapy Volume 97 Supplement S1 p. eS1581.

2. Reid Susan, Rivett Darren Anthony, Katekar Michael Gerard, Callister Robin (2011) Identification of patients with cervicogenic dizziness during recruitment for a randomised controlled clinical trial. Journal of Physiotherapy, e Supplements. 2011 APA Conference Abstracts:004.

3. Reid Susan, Rivett Darren Anthony, Katekar Michael Gerard, Callister Robin, (2011) Manual therapy treatment of cervicogenic dizziness and pain: preliminary results of a randomised controlled trial. Journal of Physiotherapy, e Supplements. 2011 APA Conference Abstracts:220.

4. Reid SA, Rivett DA, Callister R, and Katekar MG. (2012) The treatment of cervicogenic dizziness with manual therapy: preliminary results of a randomized controlled trial. In Proceedings of the 10th Congress of the International Federation of Orthopaedic Manipulative Therapists (IFOMPT), October 2012, Quebec City, Quebec, Canada. In Journal of Orthopaedic & Sports Physical Therapy 2012; 42(10):A60.

5. Reid SA, Rivett DA, Callister R, and Katekar MG. (2012) Identification of patients with cervicogenic dizziness during recruitment for a randomized controlled trial. In Proceedings of the 10th Congress of the International Federation of Orthopaedic Manipulative Therapists (IFOMPT), October 2012, Quebec City, Quebec, Canada. In Journal of Orthopaedic & Sports Physical Therapy 2012; 42(10):A60.

6. Reid SA, Callister R, Rivett DA. (2013) The effectiveness of two common manual therapy treatment approaches to cervical spine dysfunction. In Proceedings of the 3rd International Mulligan Conference. IV Cirne International Rehabilitation Neuromusculoskeletal and Sport Congress. Rio de Janeiro, Brazil.

7. Reid, SA (2013) Cervicogenic dizziness and VBI; how do you differentiate? In the Proceedings of the 3rd International Mulligan Conference. IV Cirne

International Rehabilitation Neuromusculoskeletal and Sport Congress. Rio de Janeiro, Brazil.

8. Reid SA, Rivett DA, Callister R, and Katekar MG (2013) The treatment of cervicogenic dizziness with Mulligan SNAGS and Maitland mobilisations: which is more effective? Australian Journal of Physiotherapy, e Supplements. 2013 APA Conference Abstracts: p141.

Conference and other invited presentations

The following conference presentations were a direct result of the work completed in this thesis:

1 Reid SA (2008) Cervicogenic Dizziness. APA NSW Biennial State Symposium, October 2008, Sydney.

2 Reid SA, Rivett DA, Callister R, and Katekar MG. (2009) The Treatment of Cervicogenic Dizziness and Pain with Sustained Natural Apophyseal Glides (SNAGs): a Randomized Controlled Trial. International Mulligan Concept Conference, May 2009, Chicago.

3 Reid SA, Rivett DA, Callister R, and Katekar MG. (2011) The Identification of People with Cervicogenic Dizziness. World Confederation for Physical Therapy 16th International Congress (WCPT), June 2011, Amsterdam, The Netherlands.

4 Reid SA, Rivett DA, Callister R, and Katekar MG. (2011) Cervicogenic Dizziness and Pain: preliminary findings of a Randomized Controlled Trial. 2nd International Mulligan Concept Conference, June 2011, Porto, Portugal.

5 Reid SA, Rivett DA, Callister R, and Katekar MG. (2011) The identification of Cervicogenic Dizziness during a Randomized Controlled Trial. Australian 17th Biennial Physiotherapy Conference, October 2011, Brisbane.

6 Reid SA, Rivett DA, Callister R, and Katekar MG. (2011) Manual therapy treatment of cervicogenic dizziness and pain: preliminary results of a

Randomized Controlled Trial. Australian 17th Biennial Physiotherapy Conference, October 2011, Brisbane.

7 Reid SA, Rivett DA, Callister R, and Katekar MG. (2011) The treatment of cervicogenic dizziness and pain with manual therapy. NOTSA 21st Annual Clinical and Scientific Meeting, October 2011, Newcastle.

8 Reid SA, Rivett DA, Callister R, and Katekar MG. (2011) Cervicogenic vertigo: does it exist? NOTSA 21st Annual Clinical and Scientific Meeting, October 2011, Newcastle.

9 Reid SA, Rivett DA, Callister R, and Katekar MG. (2012) The treatment of cervicogenic dizziness with manual therapy: preliminary results of a randomized controlled trial. 10th Congress of the International Federation of Orthopaedic Manipulative Therapists (IFOMPT), October 2012, Quebec City, Quebec, Canada.

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12 Reid, SA (2013) Cervicogenic dizziness and VBI; how do you differentiate? 3rd International Mulligan Conference. IV Cirne International Rehabilitation Neuromusculoskeletal and Sport Congress. Rio de Janeiro, Brazil.

13 Reid SA, Rivett DA, Callister R, and Katekar MG (2013) The treatment of cervicogenic dizziness with Mulligan SNAGS and Maitland mobilisations:

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14 Reid SA, Katekar MG, Callister R & Rivett DA (2013) The treatment of cervicogenic dizziness with manual therapy: a randomised controlled trial with 12-month follow-up. The Neuro-Otology Society of Australia Conference, October 2013, Melbourne.

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Contents

List of Abbreviations

ANOVA one-way analysis of variance

CI confidence interval

CROM cervical range of motion

DHI Dizziness Handicap Inventory

GPE global perceived effect

HRA head repositioning accuracy

MM Maitland mobilisation

MIC minimal important change

MDC minimal detectable change

NHP natural head position

PJM passive joint mobilisations

RCT randomised controlled trial

ROM range of motion

SD standard deviation

SEM standard error of measurement

SNAG sustained natural apophyseal glide

VAD vertebral artery dissection

VAS visual analogue scale

VBI vertebro-basilar insufficiency

WAD whiplash-associated disorder

Abstract

The primary aim of this thesis was to determine and compare the effects of two forms of manual therapy on chronic cervicogenic dizziness over the short (12 weeks) and long (12 months) term. Eighty-six participants with chronic cervicogenic dizziness were randomised to receive sustained natural apophyseal glides (SNAGs) with self-SNAGs (n=29), passive joint mobilisations (PJMs) with range of motion (ROM) exercises (n=29), or a placebo intervention (n=28). Participants received 2-6 treatments over 6 weeks, with outcomes measured at baseline, post-treatment, 12 weeks and 12 months. Intention-to-treat analyses were performed, with the significance level set at 0.05.

The intensity of dizziness was significantly reduced post-treatment and at 12 weeks in both manual therapy groups, and the frequency of dizziness was significantly reduced at 12 weeks compared to the placebo. The PJM group had less dizziness handicap than the placebo group post-treatment and at 12 weeks, and less pain at 12 weeks. For cervical range of motion (ROM), the SNAG group improved in six directions and the PJM group in one direction post-treatment and at 12 weeks, compared to the placebo group. There was no effect on head repositioning accuracy or balance.

When the long-term (12 months) effects of manual therapy were evaluated there were no significant differences in dizziness intensity or pain intensity between the groups, however both manual therapy groups reported dizziness less often and had lower dizziness handicap scores than the placebo group. At 12 months there was greater ROM in six directions for the SNAG group and four directions for the PJM group compared to placebo. There were no meaningful differences between groups for head repositioning accuracy. The SNAG group had better balance than the placebo group on two dynamic tests. There were no differences between the two manual therapy groups at 12 months for any outcomes. Both manual therapy groups found treatment more beneficial than the placebo intervention at 12 weeks and 12 months. There were no adverse effects lasting longer than 24 hours.

One limitation of the study was that it was not possible to blind the treating therapist to group allocation. Another limitation was that the effect of the home-based exercises could not be adequately assessed, as compliance with completion of home exercise diaries was poor.

As part of this thesis, the process used in this RCT to identify people with cervicogenic dizziness has been outlined as a first step in developing a screening process to identify cervicogenic dizziness in clinical practice. In addition, the physical characteristics of the participants with identified cervicogenic dizziness were compared to normative data to further describe this condition and aide in its identification. Participants with cervicogenic dizziness were found to have significant deficits in cervical ROM, head repositioning accuracy and balance when compared to published normative values.

This thesis has provided evidence for the first time that a small number of manual therapy treatments, combined with recommendations to perform simple home-based exercises, can make a significant difference over the short and long term to patients experiencing chronic cervicogenic dizziness. The results have implications in identification and improving treatment for many patients, as dizziness occurring together with neck pain is a common and disabling problem.

Chapter 1 Introduction

1.1 Background

Cervicogenic dizziness is a disabling and relatively common problem that often lasts for many years, greatly compromising the quality of life of sufferers (Colledge, Barr-Hamilton, Lewis, Sellar, & Wilson, 1996; 2000; Karlberg, Johansson, Magnussen, & Frannson, 1996; Treleaven, 2008b; Treleaven, 2011; Woodhouse & Vasseljen, 2008; Yahia et al., 2009). This condition has substantial implications and costs to the community, not only financial, but also emotional, physical and social. Cervicogenic dizziness is defined by three characteristics, all of which should be present if dizziness is to be attributed to the cervical spine:

- the dizziness is described as unsteadiness or imbalance (not rotary vertigo or pre-syncope)
- there is dysfunction in the cervical spine described as pain and/or stiffness, and
- the dizziness is triggered by movements or positions of the neck (Heikkila, Johansson et al. 2000; Wrisley, Sparto et al. 2000; Huijbregts and Vidal 2004).

To date, the management of cervicogenic dizziness has been a challenge with very few studies providing evidence for its best management. One possible cause of cervicogenic dizziness is trauma such as whiplash, and another is cervical spondylosis. More precisely, it is suggested that cervicogenic dizziness is a result of perturbation in the information from sensory mechanoreceptors which are abundant in the joints and muscles of the upper cervical spine (Brandt & Bronstein, 2001). If these mechanoreceptors become sensitised due to trauma, degeneration or immobilisation, an incorrect interpretation of head position can result (Huijbregts & Vidal, 2004). This can lead to a sensory mismatch with inputs from the visual and vestibular systems, leading to sensations of 'unsteady' dizziness (Huijbregts & Vidal, 2004).

There is an emerging body of evidence that manual therapy is a successful treatment for this disabling condition (Lystad, Bell, Bonnevie-Svendsen, & Carter, 2011). Two

particular forms of manual therapy potentially relevant for treating cervicogenic dizziness and commonly used by physiotherapists to treat cervical spine dysfunction are Mulligan sustained natural apophyseal glides (SNAGs) (Mulligan, 2004) and Maitland passive joint mobilisations (PJMs) (Maitland, 2001). SNAGs are performed by the therapist sustaining a glide to spinal joints while the patient simultaneously performs an active physiological movement. PJMs are generally performed in the cervical spine by the therapist applying oscillatory pressure through the thumbs in a posterior-anterior direction, either centrally over the spinous process, or unilaterally over the articular pillar. Manual therapy is hypothesised to be effective in treating cervicogenic dizziness by restoring mechanical gliding of the zygapophyseal joints, and/or increasing cervical range of motion (ROM), and/or reducing muscle spasm and pain, resulting in a normalisation of stimulation of mechanoreceptors and afferent feedback which will then reduce dizziness and sensations of unsteadiness (Brandt & Bronstein, 2001; Furman & Whitney, 2000; Huijbregts & Vidal, 2004).

1.2 Rationale for the study

Cervicogenic dizziness is a common, disabling and persistent problem (Treleaven, Jull, & Sterling, 2003; Wrisley, Sparto, Whitney, & Furman, 2000). The most effective treatment for cervicogenic dizziness has not yet been established as there has been insufficient quality research to determine this, however there is emerging evidence that manual therapy is beneficial (Lystad, et al., 2011). This preliminary evidence suggests that in the short term, SNAGs are effective in reducing the intensity and frequency of dizziness, disability and cervical pain in people with chronic cervicogenic dizziness (Reid, Rivett, Katekar, & Callister, 2008), but SNAGs have not been evaluated in the longer term. PJMs are commonly used in clinical practice to treat cervical pain and decreased ROM (Leaver et al., 2010; Miller et al., 2010), but there is no evidence as yet to support their use in treating cervicogenic dizziness despite suggestions by some clinicians that they may be beneficial. It is also unknown whether one type of manual therapy is better than another in treating cervicogenic dizziness. A randomised controlled trial (RCT) ranks highly in the hierarchy of strength of evidence assessing the effectiveness of clinical treatment, and therefore was chosen as the investigative approach for this study. Hence it was decided to perform an RCT to investigate the

effects of these two common forms of manual therapy compared to a placebo intervention in the treatment of cervicogenic dizziness in the longer term.

To date there is no 'gold standard' test to identify patients with cervicogenic dizziness (Heikkila, Johansson, & Wenngren, 2000; Huijbregts & Vidal, 2004). This has led to some disagreement about how to recognise the condition, with some authorities even denying it exists (Brandt & Bronstein, 2001). For the clinical trial that comprises this thesis it was first necessary to develop a screening process to identify people with cervicogenic dizziness to participate in the study. Hence, the process used to identify people with this condition for inclusion in this trial, will also be detailed.

1.3 Aims and hypotheses

1.3.1 Aims

The aims of this thesis are:

- To determine and compare the short term and long term effects of SNAGs (including self-SNAGs), PJMs (plus ROM exercises) and a placebo intervention on chronic cervicogenic dizziness.
- To establish whether SNAGs (including self-SNAGs) or PJMs (plus ROM exercises) are more effective than the other in treating cervicogenic dizziness.

1.3.2 Hypotheses

It is hypothesised that:

- Manual therapy is superior to a placebo intervention in the treatment of chronic cervicogenic dizziness in the short and long term.
- There is no difference in treatment outcomes for chronic cervicogenic dizziness between SNAGs (including self-SNAGs) and PJMs (plus ROM exercises).

1.4 Overview of the thesis

This thesis is presented in publication style, arranged into nine chapters. It comprises a literature review followed by a series of five papers. Each published paper or submitted manuscript was written in the conventional publication style for the journal to which it was submitted. The manuscripts that form the bases for Chapters Three, Four and Five have been published in refereed journals and are thus presented in the

form of a manuscript formatted by the publisher. The manuscript that constitutes Chapter Six has been accepted for publication and that for Chapter Seven is currently being reviewed. At the beginning of each chapter a brief overview is presented to place the manuscript in the context of the overall thesis.

Chapter Two is a review of the relevant aspects of the literature, presented so the reader can understand the context and methodology of the study. Chapter Three outlines the study protocol for the RCT that is the basis for this thesis. Chapter Four reports the results of this RCT for the self-report measures (intensity of dizziness, dizziness frequency, dizziness handicap, intensity of cervical pain, perceived effect of the interventions) immediately post-intervention and at 12 weeks after the intervention. The post-treatment and 12-week follow-up results for the physical measures (cervical ROM, head repositioning accuracy, balance) are reported separately in Chapter Five. Chapter Six reports the 12-month follow-up results for the RCT. Chapter Seven provides a comprehensive report of the screening process used to identify participants as having cervicogenic dizziness for inclusion in the RCT. It is presented after Chapters Four, Five and Six as it includes data from the RCT findings that demonstrate a high proportion of participants benefitted from the interventions, which provides evidence that this was a successful screening process to identify those with cervicogenic dizziness. In Chapter Eight, so as to gain a further understanding of cervicogenic dizziness patients, the findings from the physical measures (cervical ROM, head repositioning accuracy, balance) are compared to normative values. The final chapter of the thesis (Chapter Nine) provides a summary of the key findings and the conclusions drawn from this research, with recommendations for clinical practice and for future research.

1.5 Significance of the study

Dizziness is a very common problem with some indication it may be the most common reason to visit a physician in the over 65 year age group (Herdman, 2000). Dizziness can lead to unsteadiness and falls resulting in fractures, serious injury and even death (Huijbregts & Vidal, 2004). The differential diagnosis of dizziness is challenging but is essential for appropriate management and treatment leading to improved quality of

life and decreased handicap. Some types of dizziness are suitable for manual therapy interventions whereas for other types of dizziness manual therapy is contraindicated (Huijbregts & Vidal, 2004).

To date, cervicogenic dizziness has been poorly identified and treated, leading to the condition often becoming chronic and disabling (Reid, et al., 2008). Information gained from this thesis may provide guidance to practitioners for the effective management of cervicogenic dizziness. This thesis documents the first study to report a long-term (12 month) follow-up to manual therapy treatment for cervicogenic dizziness. It is also the first study to investigate the commonly used manual therapy technique of PJMs for cervicogenic dizziness. If it is found that a small number of manual therapy treatments have lasting benefits over the longer term, it will be a welcoming breakthrough in how to manage this problem. If both types of manual therapy are shown to be equally effective for cervicogenic dizziness, it will provide two treatment options for manual therapists as some may be more familiar with using SNAGs and others with PJMs. As adverse effects will also be monitored during the RCT, it will help inform whether manual therapy is a safe intervention for this condition.

Moreover, by describing in detail the screening process used to identify participants with cervicogenic dizziness for inclusion in this study, it may further help practitioners and other researchers diagnose individuals with this condition and distinguish them from those with many of the other causes and types of dizziness. It would be desirable for practitioners to better recognise patients with this condition so they can be offered appropriate evidence-based treatment.

Recruitment will be from the wider community in the Hunter region of NSW in Australia, so the study sample should be representative of the general population with cervicogenic dizziness. The findings of this study will therefore be appropriate to generalise to people with this problem and thus have the potential to benefit many patients. If positive results are found, the study will provide further evidence for the effectiveness of manual therapy in the treatment of cervicogenic dizziness.

1.6 References for Chapter One

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Chapter 2 Background

2.1 Introduction

Before developing the experimental study within this thesis, the literature was reviewed to gain a general understanding of dizziness and more specifically cervicogenic dizziness, and to determine the best way of identifying people with this condition. It was also necessary to review the previous work undertaken in this area to establish the evidence for treatment of this condition.

Several bibliographic databases were searched from 2011-2013. These were: MEDLINE using OVID (January 1, 1966 onwards), EMBASE (1988 onwards), Cumulative Index to Nursing and Allied Health Literature (CINAHL) (from 1983 onwards), Physiotherapy Evidence Database (PEDro), the Cochrane Controlled Trials Register (Cochrane Library, 2002), Manual Alternative and Natural Therapy Index System (MANTIS) (1980 onwards) and the Allied and Complementary Medicine Database (AMED) (from 1985 onwards). Reference lists of retrieved studies were also searched to find appropriate studies. No language restrictions were applied.

Search terms for the databases included:

- terms related to the condition: cervical spine, dizziness, neck pain, vertigo, cervical vertebrae
- terms related to the intervention: manual therapy, mobilisation, Mulligan, Maitland, physiotherapy, manipulation, physical therapy, chiropractic, musculoskeletal manipulation, rehabilitation
- terms related to the method of the studies: randomised controlled trial, placebo, controlled clinical trial, random allocation, double blind method, single blind method, experimental clinical trial, volunteer.

In this chapter firstly literature relating to dizziness is reviewed, followed by cervical spine dysfunction, then the examination of patients with dizziness, and lastly the treatment of cervicogenic dizziness.

2.2 Dizziness

2.2.1 Introduction

Dizziness is a very common problem, with many different types and causes. Worldwide there are more than 200 million medical visits for dizziness and vertigo annually (Gopinath, McMahon, Rochtchina, & Mitchell, 2009; Newman-Toker, 2012). Dizziness often leads to physical problems such as postural instability, unsteadiness and falls, as well as social, emotional and financial difficulties (Kristjansson & Treleaven, 2009).

Dizziness has been reported to be the most common symptom in elderly patients (Hansson & Magnuson, 2013) with Colledge et al. (1996) reporting that 30% of people over 65 have dizziness. Luxon (1984) reports that by the age of 80 years, two thirds of women and one third of men have experienced dizziness. People with neck pain are more likely to get dizziness than the general population (Humphries & Peterson, 2013; Treleaven, Jull, & Sterling, 2003). The incidence of dizziness in whiplash patients has been reported to be as high as 80-90% (Heikkila, et al., 2000; Hinoki, 1985; Humphries, Bolton, Peterson, & Wood, 2002). The costs to society for sick leave and disability pensions due to dizziness are substantial, but have not been described specifically for cervicogenic dizziness (Bjorne & Agerberg, 2003).

2.2.2 Classification of dizziness

The diagnosis of dizziness is complex, challenging and confusing, resulting in a rate of misdiagnosis that could be as high as 50% (Newman-Toker, 2007). Patients with dizziness are often difficult to diagnose due to the variety of types of dizziness and numerous aetiologies (Cooper, 2007). Additionally, dizziness is a non-specific term that describes different feelings and sensations for different people. According to an American study, it may cost up to $2500 to diagnose the cause of dizziness, and most of the time it is still inconclusive (Newman-Toker & Pronovost, 2009).

The traditional method of diagnosis has centred on a quality-of-symptoms assessment or "What do you mean by dizzy?" approach (Newman-Toker, 2007; Newman-Toker et al., 2007). This assumes that the type of dizziness will predict the underlying cause. The system described by Drachman and Hart (1972) suggests people are classified as

having one of four dizziness types: light headedness, imbalance, vertigo or disorientation (see Table 2.1). In this system, a subjective examination is used to specifically clarify a patient's report of the symptoms by carefully asking the patient about the type of dizziness they are experiencing. For example, by asking questions such as "When you say you get dizzy, what exactly do you mean? Does the room spin, do you feel unsteady on your feet or are you light headed?" Hain (2004) reports that patients and referring general practitioners prefer a "symptom" orientated setting (i.e. a dizzy clinic) to a "cause" orientated setting (i.e. ENT, Neurology, Cardiology clinics), because patients are subjectively aware of symptoms but the cause may be unclear. This approach remains the current standard of clinical care and appears in medical journals and textbooks (Newman-Toker, 2007; Sloane, Coeytaux, Beck, & Dallara, 2001).

Table 2.1 Descriptions of dizziness (Drachman & Hart, 1972)

Symptoms	Possible Cause
Near syncope, light-headedness, nausea, fainting, wooziness	Impaired cerebral perfusion, cardiovascular causes e.g. postural hypotension, vasovagal attacks, cardiac arrhythmia, anaemia, impaired cardiac output
Disequilibrium, imbalance, ataxia, unsteadiness, falling, stumbling	Cervicogenic dizziness, musculoskeletal, sensory deficits, neuromuscular (e.g. peripheral neuropathy) or vestibular problems,
Vertigo (spinning sensation)	Central nervous system or peripheral problems e.g. Ménière's disease, Benign Positional Paroxysmal Vertigo (BPPV), vestibular neuritis, vestibulopathy
Disorientation, disconnection, 'spaced out' feelings that are associated with major depression and anxiety	Psychogenic dizziness

Although the classification proposed by Drachman and Hart (1972) is a valuable procedure, there are limitations (Newman-Toker & Camargo, 2006; Newman-Toker, et al., 2007). It is not realistic to assume that the person will have one mutually-exclusive type of dizziness. There is often an overlap between groups and patients may experience different types of dizziness concurrently and have more than one cause (Newman-Toker, et al., 2007). Patients often have difficulty describing the symptoms

and it has been found that patient's descriptions of dizziness are often unclear, inconsistent and unreliable (Newman-Toker, et al., 2007). In a study of 1,342 consecutive patients with dizziness attending emergency departments at two hospitals in the United States of America, Newman-Toker et al. found 62% of patients reported more than one type of dizziness, and 52% picked a different response on retest approximately six minutes later. This has led to Newman-Toker et al. suggesting the quality-of-symptoms approach is neither valid nor reliable and is probably contributing to misdiagnosis (Newman-Toker & Camargo, 2006; Newman-Toker, et al., 2007).

It is suggested by Newman-Toker that instead of concentrating on dizziness type, the timing (such as duration and frequency) and triggers of dizziness (such as head movements or standing up) should be used for diagnosis (Newman-Toker, 2008). In the assessment of patients attending the emergency department of two hospitals as mentioned above, reports of dizziness duration, triggers and associated symptoms were clear, consistent, and reliable leading to suggestions that these factors are more accurate and reliable in diagnostic decisions.

Newman-Toker (2008) has thus proposed a new approach in clinical assessment of a patient with dizziness:

- Identify if there are red flags such as abnormal vital signs, confused mental state, sudden, severe neck or head pain, "worrisome' neurologic symptoms (dysphagia, diplopia, dysarthria) or "worrisome' cardiovascular symptoms (dyspnoea, fainting, and chest pain).
- The subjective examination should include questions about time and mode of onset, duration and frequency of symptoms. Ask questions about whether the dizziness is continuous or episodic, and if it occurs daily, weekly etc.
- Ask about dizziness triggers such as head movements or positions, or standing up quickly as opposed to spontaneous (un-triggered) dizziness. Try to evoke dizziness in the physical examination.
- If a patient has persistent dizziness (>24 hours) a neurological examination looking at oculomotor signs such as direction-changing nystagmus is required.

The main aim of this method of clinical assessment is to differentiate common, benign causes of dizziness from more dangerous conditions. According to Newman-Toker, it should produce more reliable diagnostic results (Newman-Toker, 2008).

2.2.3 Cervicogenic dizziness

One specific type of dizziness is cervicogenic dizziness. This dizziness is described as unsteadiness and it occurs together with neck pain and/or stiffness. This dizziness is triggered by neck movements or positions and can last minutes to hours (Reid, Rivett et al. 2008). It is not rotary vertigo as this has a central or vestibular cause. When patients have a cervical cause for their dizziness the symptoms are generally less severe and more subtle than with vestibular or rotatory dizziness (Kristjansson & Treleaven, 2009). The use of symptoms and behaviour (timing and triggers) to diagnose cervicogenic dizziness is important because to date, attempts to find an accurate, simple, diagnostic test have failed (Karlberg, Magnussen, Malmstrom, Melander, & Moritz, 1996; Tjell, 2001).

The disorder was first described in 1955 as cervical vertigo by Ryan and Cope (1955). Other terms used for the same disorder are cervicogenic vertigo and cervical dizziness (Fitz-Ritson, 1991; Tjell & Rosenhall, 1998). Since its introduction, the diagnosis and the proposed pathophysiological mechanisms have remained controversial with some clinicians doubting the existence of this condition (Brandt, 1996; Brandt & Bronstein, 2001; Hulse, 1983). Nevertheless, there is considerable evidence to support cervicogenic dizziness as a distinct disorder (Borg-Stein, Rauch, & Krabak, 2001; Brandt, 1991; Brandt & Bronstein, 2001; Colledge, et al., 1996; Heikkila, 2004; Wrisley, et al., 2000).

Dizziness described as unsteadiness, poor balance or disequilibrium is a common symptom in people with neck pain (Borg-Stein, et al., 2001; Kristjansson & Treleaven, 2009; Reid, et al., 2008; Treleaven, 2011; Treleaven, Jull, & Serling, 2003; Wrisley, et al., 2000). In a recent study of 405 neck pain patients from chiropractic clinics in Switzerland, 44% reported dizziness as well as neck pain (Humphreys & Peterson, 2013). In elderly people where dizziness is a very common symptom we also see this link with neck pain, evidenced by Colledge et al. (1996) who report that in a study of

elderly people (over 65 years), 30 % had experienced dizziness and of those 66% had cervical spine involvement.

There are two main presentations of people with cervicogenic dizziness: those with insidious onset and those with a traumatic cause. The most common presentations of cervicogenic dizziness are insidious or non-traumatic and occur with cervical spondylosis, degenerative lesions (e.g. osteoarthritis), inflammatory diseases (e.g. rheumatoid arthritis), vertebral collapse, decreased cervical disc height or herniated discs, vertebral displacement, and/or muscle spasm (Ryan & Cope, 1955; Wrisley, Sparto et al. 2000; Borg-Stein, et al. 2001). Pain and dizziness can arise when the upper cervical (occiput to C3) zygapophyseal joints are under abnormal mechanical stress because these joints are the most densely innervated of all the spinal joints with 50% of all cervical proprioceptors located in the joint capsules of C1 to C3 (Hulse, 1983; Wrisley, et al., 2000; Wyke, 1979). Some support for this theory is provided by Ryan and Cope (1955) who describe three patients with cervicogenic dizziness attributed to cervical spondylosis. Degenerative changes such as osteophytes and discopathy on cervical X-rays, have been reported by several authors in patients with cervicogenic dizziness (Bogduk, 1981; Olszewski, Majak, Pietkiewicz, & Repetowski, 2005; Ryan & Cope, 1955). This supports a possible link between dysfunction or abnormal findings in the cervical spine and reports of dizziness.

The second common presentation of cervicogenic dizziness is when dizziness and cervical symptoms commenced following a neck injury such as whiplash (flexion-extension injury) or blunt trauma to the top of the head (Furman & Cass, 1996; Hulse, 1983; Wrisley, et al., 2000; Young & Chen, 2003). It has been suggested by Hulse (1983) that one third of people with cervical dizziness have their onset due to trauma such as whiplash, one third have an insidious onset following spinal degeneration, and one third have other causes, including manual therapy. Young and Chen (2003) described nine patients who experienced acute vertigo within one day after cervical manipulation. The number of patients whose cervicogenic dizziness was related to an injury of the neck was only 13% in a study of 120 patients with cervical "vertigo" by Olszewski and Repetowski (2008). The most common traumatic cause of dizziness is whiplash injuries, which are estimated to occur in approximately 0.1% of the

population (Barnsley, Lord, & Bogduk, 1994). The incidence of symptoms of dizziness in whiplash patients has been variously reported as 20-58% (Wrisley, Sparto et al. 2000), 40-80% (Oostendorp, van Eupen, Van Erp, & Elvers, 1999) and as high as 80-90% (Heikkila, et al., 2000; Hinoki, 1985; Humphries, et al., 2002). Some authors (Hinoki, 1985; Humphries, et al., 2002) propose trauma to muscles, ligaments, joint capsules, sensory nerves and other soft tissues of the neck could lead to dizziness in these patients by damaging the proprioceptors, which can then result in a perturbation of afferent feedback.

It is commonly believed that dizziness can be caused by dysfunction in the proprioceptors of the deep muscles in the upper cervical spine leading to abnormal input to the vestibular nuclei (Bogduk, 1981; Borg-Stein, et al., 2001; Grgic, 2006; Wrisley, et al., 2000). A cyclic pattern may result when people experience cervical dizziness as the vestibulocollic and cervicocollic reflexes can cause excessive cervical muscle spasm in sternocleidomastoid and the upper trapezius muscles, that causes more dizziness and results in increased cervical muscle spasm (Bogduk, 1981; Borg-Stein, et al., 2001; de Jong, de Jong, Bernard, & Jongkees, 1977; Furman & Cass, 1996; Malmstrom, Karlberg, Melander, Magnusson, & Moritz, 2007; Ryan & Cope, 1955; Wrisley, et al., 2000). Together these findings suggest that there is a strong link between muscle spasm, often caused by trauma, and the occurrence of cervicogenic dizziness.

Although the cause of cervicogenic dizziness is unknown, Treleaven, Jull and LowChoy (2006) suggest dysfunction in cervical proprioceptors in people with neck disorders leads to an imbalance with afferent input from vestibular and visual receptors resulting in postural instability. A study conducted by Treleaven, Jull & Sterling (2003) investigated whether cervical mechanoreceptor dysfunction was a likely cause of dizziness in whiplash-associated disorder (WAD). Joint position error (the accuracy to return to natural head position-NHP) was used as a measure of proprioception to represent the function of the cervical mechanoreceptors. Joint position error was measured in 102 people with whiplash and 44 asymptomatic control subjects. Within the whiplash group, those with dizziness displayed greater error than those without dizziness. People with whiplash-associated dizziness and/or

unsteadiness were shown to have significantly greater joint position errors and a higher neck pain index than control subjects, suggesting that cervical mechanoreceptor dysfunction was a likely cause of the symptoms (Treleaven, Jull, & Serling, 2003). People with cervicogenic dizziness often avoid movements that bring on dizziness. This can lead to secondary cervical problems such as muscle spasm, protective muscle guarding and loss of range of movement. A vicious cycle of increasing symptoms can develop and the condition often becomes chronic (Cronin, 1997).

Damage to cervical receptors, vestibular receptors or the central nervous system can lead to disturbances in the postural control system (Baloh & Halmagyi, 1996). A mismatch of afferent input from proprioceptive, vestibular and visual systems to the postural control centres of the central nervous system can result in feelings of dizziness (Brandt & Bronstein, 2001). The most likely cause of these postural disturbances in patients with cervicogenic dizziness is a perturbation of cervical afferent input from impairments to upper cervical (occiput to C3) joint and muscle proprioceptors (Rubin, Woolley, Dailey, & Goebel, 1995; Treleaven, Jull, & Sterling, 2003). It is postulated that the disturbances could be proprioceptive or nociceptive or most likely a combination (Treleaven, Jull, & Lowchoy, 2005b). Normally balance is controlled at a subconscious level. However, when an aberrant inflow of impulses from the proprioceptors in the neck occurs, or the balance between the afferent inputs from the vestibular, visual and proprioceptive systems is disturbed, neural activity reaches consciousness and a sensation of dizziness is experienced (Richmond & Corneil, 2001; Tjell, 2001).

2.2.4 Rotary vertigo

Vertigo is a false sensation of movement, usually rotary, whirling, spinning or rocking, of the person or the environment (Bisdorff, Von Brevern, Lempert, & Newman-Toker, 2009; Oostendorp, et al., 1999). Vertigo can result from problems of the peripheral vestibular system (such as the labyrinth or vestibular nerve) or the central vestibular connections in the brainstem or cerebellum (e.g. brainstem strokes or tumours) (Bisdorff, et al., 2009). True vertigo is more severe and disabling form of dizziness than cervicogenic dizziness and is often accompanied by nausea and vomiting (Kristjansson & Treleaven, 2009). Vertigo may present as an acute severe persistent episode or as recurrent episodic attacks over months and years, so asking about duration is an

important aspect of clinical assessment (Newman-Toker, 2007). The two main causes of acute single severe episodes of continuous vertigo (that often last for days or weeks) are acute vestibulopathy (vestibular neuritis or viral labyrinthitis) and cerebellar infarction (Bisdorff, et al., 2009; Newman-Toker, 2008). The main causes of recurrent shorter lasting (seconds to hours) episodes of vertigo are benign paroxysmal positional vertigo (BPPV), post-traumatic vertigo, Ménière's disease and recurrent vestibulopathy (Newman-Toker, 2008). If the cause of vertigo is a lesion of the central nervous system (brainstem strokes, brain tumours etc.), there are usually accompanying neurological symptoms and/or signs attributable to the vestibular connections in brainstem such as dysarthria, diplopia and weakness and sensory problems.

Vertigo is reported as the most common type of dizziness varying from 20-58% (Froehling, Silverstein, Mohr, & Beatty, 1994; Hanley, Tom, & Considine, 2001; Kroenke et al., 1992). Hoffman et al. (1999) states the most common aetiologies for dizziness are peripheral vestibulopathies (35% to 55% of patients) and psychiatric disorders (10% to 25% of patients), while cerebrovascular disease (5%) and brain tumours (<1%) are infrequent. Dix (1974) reported the most common causes of true rotary vertigo are Ménière's disease (17-43%), BPPV and acute vestibular neuritis 10-44%. This demonstrates that vertigo is more commonly occurring than cervicogenic dizziness.

The term cervical vertigo has often been used to describe cervicogenic dizziness, however, this may create confusion especially when trying to establish the underlying pathology (Bogduk, 1994). True rotary vertigo does not have a cervical cause, therefore the terms cervical vertigo and cervicogenic dizziness should not be used interchangedly. (Froehling, et al., 1994; Meadows & Magee, 1990; Wrisley, et al., 2000). This indicates why an accurate description of the type of dizziness experienced is important when taking a history to establish the correct diagnosis. (Brandt & Bronstein, 2001).

2.2.4.1 Benign paroxysmal positional vertigo (BPPV)

Benign paroxysmal positional vertigo (BPPV) is one of the most common types of vertigo often reported as being about 30% of all vertigo. BPPV is a transient condition of brief intermittent attacks of spinning precipitated by head movements (Borg-Stein,

et al., 2001). It is caused by a mechanical disorder of the inner ear where small deposits of calcium carbonate (otoconia) are dislodged from the otolithic membrane of the utricle and then settle on the hair cells in the semicircular canals (usually the posterior) (Borg-Stein, et al., 2001) (Figure 2.1). BPPV arises after a change in head or body position such as:

- Rolling over in bed
- Looking up e.g. to get something off the top shelf, off the clothesline
- Bending down to a low cupboard
- Getting in and out of bed, or lying down (Caplan, 1996).

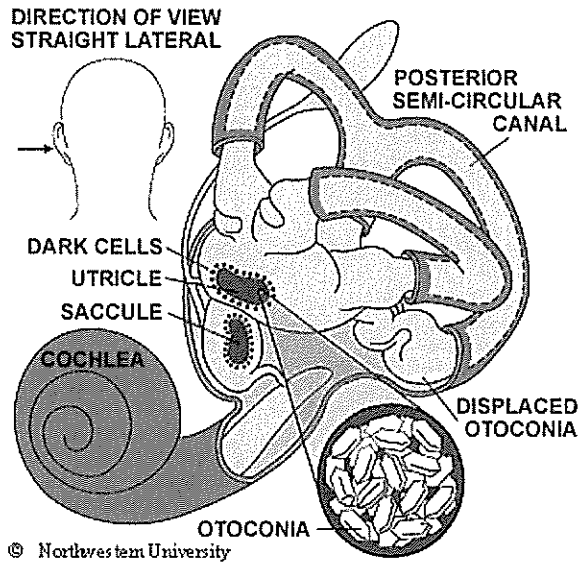

Figure 2.1 The inner ear in Benign Paroxysmal Positional Vertigo (Hain, 2004)

Such anomalies are tested clinically using specific tests such as the Dix-Hallpike manoeuvre. This test for positional nystagmus is performed by taking the patient rapidly from the erect long-sitting position into left or right cervical rotation (45 degrees), and then into the supine head hanging position (Bronstein, 2003) (Figure 2.2). For the test to be positive it must not only induce symptoms of vertigo but there must also be nystagmus (a type of involuntary eye movement) that is:

- Torsional (may have a linear component)
- Of high frequency
- Latent (3-10 seconds latency)

- Of short duration, dissipating in 30-60 seconds
- Fatigable: with repeated positioning vertigo and nystagmus disappear.
- Reversible: occurs in a reverse direction on return to sitting (Baloh & Halmagyi, 1996).

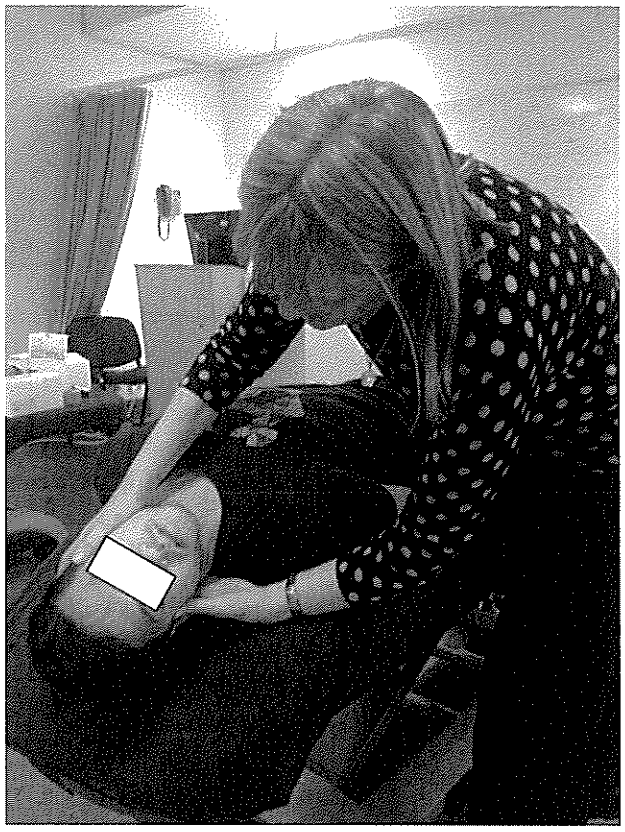

Figure 2.2 The Dix-Hallpike manoeuvre

BPPV is different to cervicogenic dizziness because the dizziness in BPPV is vertigo or spinning and often accompanied by nausea, while in cervicogenic dizziness it is a less severe, less debilitating form of dizziness described as imbalance. It is important to know about BPPV when assessing patients suspected of having cervicogenic dizziness because BPPV is very common and does have the same triggers such as looking up or rotating the head. Differentiation of the two conditions is necessary because the physiotherapy treatment for both of these conditions has been shown to be very successful, but very different (Hain, 2004; Lystad, et al., 2011). More than 80% of cases of BPPV can achieve immediate relief by specific particle repositioning manoeuvres like the Epley manoeuvre, which flushes the otoconia out of the semicircular canals

(Hain 2004) (Figure 2.3). In the case of cervicogenic dizziness, manual therapy to the cervical spine is indicated (Lystad, et al., 2011).

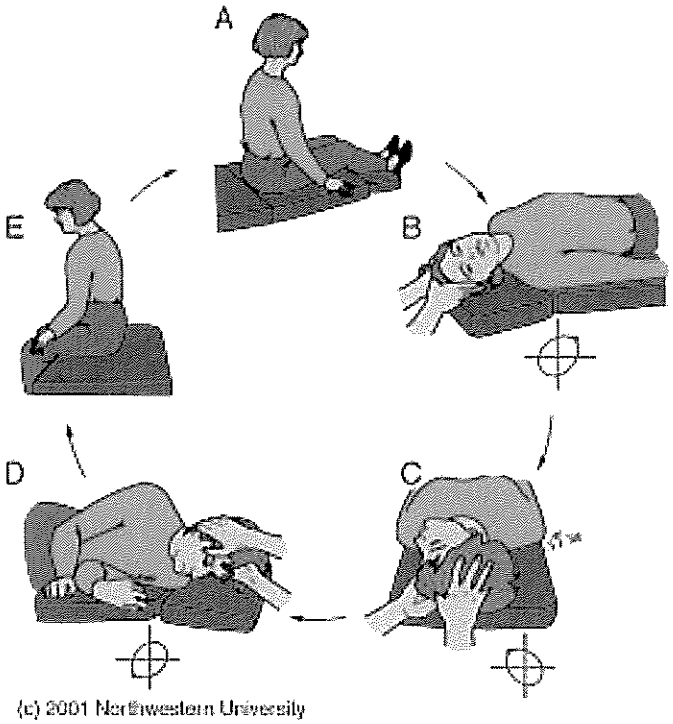

Figure 2.3 The Epley manoeuvre

2.2.5 Vertebrobasilar insufficiency (VBI)

If a patient has positional dizziness, that is dizziness caused by head movement (especially rotation or extension), it is important to establish the differential diagnosis of cervicogenic dizziness, BPPV or a problem with the vertebral artery (Magarey et al., 2004). Vertebrobasilar insufficiency (VBI) can be caused by compromise of blood flow in the vertebral and basilar arteries by intrinsic or extrinsic disorders and produce symptoms of dizziness (Thiel & Rix, 2005). The commonest intrinsic causes of VBI are atherosclerosis and thrombus. Extrinsic disorders affect the cervical part of the vertebral artery which can be occluded by osteophyte or muscle compression, neck manipulation, neck positions, spontaneous dissection or spasm caused by the vertebral nerve (Thiel & Rix 2005).

Non-dissection VBI is evidenced by symptoms of ischemia of the areas of the brain supplied by the basilar artery (pons, medulla, cerebellum, and vestibular systems).

(Magarey et al. 2004). The ischemic signs and symptoms include the classic symptoms of dizziness, diplopia, dysarthria, dysphagia, drop attacks and nausea, numbness and nystagmus, as well as ataxia. In addition, visual loss, hallucinations, photophobia, diplopia or blurred vision may occur if the occipital lobe is affected (Aspinall, 1989). It is rare for VBI to present with only one symptom, (Rivett, 2004b; Thomas, Rivett, Attia, Parsons, & Levi, 2011) so isolated dizziness would rarely be caused by VBI.

2.2.5.1 The VBI clinical picture

The most common descriptors for VBI dizziness are light headedness, unsteadiness or giddiness (Thomas, et al., 2011). Generally dizziness from posterior circulation dysfunction is not vertigo (spinning of the person or the room), although this could occur (Taylor & Kerry, 2010). It is theorised that full rotation of the cervical spine rather than extension more commonly compromises the vertebral artery (Rivett, 2004b). Usually the symptoms are caused by a transient decrease in cerebral blood flow to the hind brain that does not cause any permanent damage. Unlike BPPV, the dizziness does not improve with continued rotation (Taylor & Kerry, 2010). The dizziness may last from seconds to hours.

It is generally believed that reduced circulation from vertebrobasilar insufficiency is not a common cause of dizziness as these arteries only supply 20% of blood to the brain (Bogduk, 1986; Clements, 2001; Magarey et al., 2004). Even if decreased vertebral artery flow does occur there are usually no symptoms because collateral circulation maintains adequate blood flow to the posterior cranial circulation (Vernon, MacAdam, Marshall, Pion, & Sadowska, 2005). The frequency of vertebrobasilar incidents is extremely rare, with injury to the cervical vasculature following manipulation to the cervical spine event being 1 in 400,000 to 1 in 1.5 million (Hurwitz, Aker, Adams, Meeker, & Shekelle, 1996).

VBI is unfortunately difficult to identify through physical examination and screening procedures. Although various screening tests for identifying VBI have been advocated for pre-manipulative procedures, they have been reported to have low sensitivity and specificity and to be of poor diagnostic value (Thiel & Rix, 2005; Taylor & Kerry, 2010). The tests are also thought to increase risk of VBI as they are designed to reproduce

symptoms, (Di Fabio, 1999; Thiel & Rix, 2005) and so their value has been questioned (Magarey, et al., 2004; Refshauge, 1994; Rivett, 1999; Thomas, et al., 2011) Because there is no simple test to identify VBI, the history of onset of symptoms and clinical features are very important in making the diagnosis. The International Federation of Orthopaedic Manipulative Physical Therapists (IFOMPT) guidelines (2012) recommend that VBI testing should be performed if VBI is suspected and if manipulation or end range manual techniques will be used as part of treatment (Rushton, et al., 2012).

2.2.5.2 Vertebral artery dissection (VAD)

A particular sub-group of VBI conditions is that caused by vertebral artery dissection (VAD). VAD may present as dizziness accompanied by sudden severe neck pain and/or headache, making the differential diagnosis from cervicogenic dizziness or migraine difficult (Thomas, et al., 2011). The dizziness of VAD is usually described as light headedness or a feeling of imbalance, but occasionally it is rotatory (Edlow, Newman-Toker, & Savitz, 2008; Thomas, et al., 2011). The most common initial symptom and sometimes the only symptom, is pain in the head and neck (in almost 90% of cases), often unilateral and sub-occipital (Debette & Leys, 2009). The type and severity of pain associated with VAD is not specific, but is often believed to resemble migraine (moderate to severe and throbbing) or even cluster headache (severe, sharp, piercing pain). Commonly the patient will report they have never experienced a similar pain before. In a study by Thomas et al. (2011), 85% of subjects with vertebobasilar artery dissection had headache, mostly in the occipital area and 67% had neck pain, 67% had unsteadiness/ataxia and only 52% experienced dizziness. Note that headache was not always present and not always severe and dizziness was not present in half of these subjects.

The symptoms of VAD can be divided into non-ischemic (or local, somatic causes) or ischemic (i.e. symptoms of hind-brain ischemia) (Taylor & Kerry, 2010). The non-ischemic initial presentation of VAD is ipsilateral moderate to severe posterior neck pain and/or occipital headache (in almost 90% of cases) (Debette & Leys, 2009). The isolated severe pain is thought to be caused by extracranial vertebral-artery dissection.

The initial non-ischaemic symptoms are then usually followed by the classic ischemic signs and symptoms such as dizziness, diplopia, dysarthria, dysphagia, drop attacks and nausea, numbness and nystagmus as well as ataxia. There could be delayed onset of symptoms of brainstem ischemia from hours to up to 14 days after the dissection. So, in summary, VAD presents initially with localised somatic symptoms and then over time the symptoms from obstructed blood flow develop.

Clinically, the onset is often acute, may be related to mechanical trauma to the neck or be spontaneous. Practitioners should ask about minor mechanical trauma such as a fall, sports activity, sneezing or painting the ceiling (Thomas, et al., 2011). The patient may report a sensation of neck stiffness, but there is often no limitation of cervical range of motion.

The IFOMPT guidelines (2012) recommend if you suspect VAD the physical examination could include:

- blood pressure testing

- craniovertebral ligament testing: consider if prudent and safe

- neurological examination of peripheral nerves, cranial nerves, and for an upper motor neurone lesion

- positional testing to challenge the vascular supply to the brain such as sustained end of range rotation or a sustained pre-manipulative test position (Rivett, 2004a)

- palpation of the carotid artery.

If thought to have a VAD, the patient must be referred urgently to a doctor for appropriate assessment and scanning.

2.2.6 Cardiovascular dizziness

Light headedness, presyncope (impending faint), fainting or wooziness are often symptoms of cardiovascular dysfunction or impaired cerebral perfusion (Bisdorff, et al., 2009; Drachman & Hart, 1972; Newman-Toker, Dy, Stanton, & Robinson, 2008). These symptoms could be due to cardiovascular causes such as postural orthostatic hypotension, cardiac arrhythmia, anaemia, vasovagal problems, anaemia or impaired

cardiac output. Although dizziness from primary cardiovascular disease most commonly presents as presyncope, it can also present as vertigo (Newman-Toker, et al, 2008; Newman-Toker, 2008). The overall prevalence of dizziness in cardiovascular patients is 10% (Newman-Toker, et al., 2008). In a systematic review of cardiovascular dizzy patients, Newman-Toker et al. (2008) found that 63% had some vertigo and 37% had only vertigo. Common cardiovascular red flags include syncope, chest pain and dyspnoea (Newman-Toker, 2008). Cardiovascular dizziness is different to cervicogenic dizziness and BPPV because in these later cases dizziness is provoked by head movements.

2.2.7 Migraine

There is an increased incidence of migraine in people with vertigo and vice versa. The International Headache Society diagnostic criteria for migraine are that it is episodic with at least five attacks without aura and two with aura; the duration should be between four and 72 hours, and it should have at least two of these symptoms: unilateral, throbbing, aggravated by movement, or moderate to severe intensity (Seneviratne, 2009). It should be accompanied by nausea/vomiting and/or photophobia or phonophobia. Having dizziness is not part of the diagnosis of migraine, but general dizziness is present in approximately three quarters migraine sufferers, with one third of people with migraine having vertigo (Handelsman & El-Kashlan, 2006).

The prevalence of migraine has been reported as 6% in men and 18% in women (Furman & Whitney, 2000; Seneviratne, 2009). Migraine associated with vertigo has been reported in 32% of people presenting to an otology practice (Whitney, Wrisley, Brown, & Furman, 2000). People with migraines may have vertigo without the headache or dizziness or they may have vertigo during a migraine headache. The evidence is circumstantial and the diagnosis is often one of exclusion but a family history of migraine or imbalance during complex visual or motion environments are common findings with this condition (Furman & Whitney, 2000). The response to migraine medications in patients with recurrent, isolated vertigo of unknown cause also supports this link between migraines and vertigo.

2.2.8 Red flags

Most importantly when dealing with dizziness, clinicians should identify if there are clinical red flags that indicate a more serious cause for dizziness (such as stroke, encephalitis or neoplasms) which could be irreversible and life-threatening (Newman-Toker, 2008). The presence of central nervous system signs, upper motor neurone signs and symptoms in the absence of a diagnosis or other explanation should be considered a red flag and the patient should be referred immediately to their doctor (Baloh & Halmagyi, 1996; Kristjansson & Treleaven, 2009). Practitioners should specifically ask patients presenting with dizziness about symptoms that may suggest central nervous system pathology. These are repeated falls, ptosis, vertical nystagmus, confusion, episodes of loss of consciousness, inability to stand, severe vomiting, facial numbness, constant vertigo, facial asymmetry, swallowing problems, speech problems, severe headache, changes in sensation, a feeling of being pushed to one side, oculomotor signs/dysfunction (cranial nerves III, IV, VI), double vision, and upper motor neurone signs (Kerber et al., 2011; Wrisley, et al., 2000). Common cardiovascular red flags are chest pain, syncope and dyspnoea (Newman-Toker, 2008). Sudden or sustained severe head and/or neck pain is a red flag for vertebral artery dissection as discussed previously in section 2.2.5.2. Symptoms that require non-urgent referral to an otoneurologist because they are consistent with inner ear pathology are constant dizziness, unilateral hearing loss, new onset of tinnitus, fullness in the ear, ear pain, and transient vertigo (Wrisley, Sparto et al. 2000).

2.3 Cervical spine dysfunction

The lifetime prevalence for an acute attack of neck pain may be as high as 70%, and many people recover with treatment (Fejer, Kyvik, & Hartvigsen, 2006). However, a large proportion of patients presents with chronic neck pain (> 3 months) and recurring relapses and are more challenging to treat (Kristjansson & Treleaven, 2009). The life-long incidence of chronic neck pain may be as high as 15% (Vernon, Humphreys, & Hagino, 2006). The estimated one year incidence of neck pain is between 10.4% and 21.3% (Hoy, Protani, De, & Buchbinder, 2010). People with traumatic neck pain have been shown to have significantly higher resting pain levels and disability scores when compared to idiopathic onset neck pain subjects and healthy controls (Field, Treleaven,

& Jull, 2008; Treleaven, 2011). A sub-group of patients with neck pain may also have dizziness (Borg-Stein, et al., 2001; Kristjansson & Treleaven, 2009).

2.3.1 Cervical spine dysfunction in cervicogenic dizziness

Cervicogenic dizziness is commonly accompanied by a range of symptoms including neck pain, neck stiffness, occipital pain and headache which are believed to be from dysfunction in the upper cervical joints and muscles (occiput to C 3) (Karlberg, Johansson, et al., 1996; Kristjansson & Treleaven, 2009). Less often, it may be accompanied a range of symptoms such as fullness in the ear or sinuses, tinnitus, nausea, sweating, visual disturbances, blurred vision, trouble with swallowing, temporomandibular pain, numbness and paraesthesia, upper extremity radicular symptoms, hyperalgesia and psychological symptoms such as disturbances in concentration and memory (Borg-Stein, et al., 2001; Malmstrom, et al., 2007; Wrisley, et al., 2000).

When assessing people with cervicogenic dizziness (n=22) Malmstrom et al. (2007) found that the majority of patients had bilateral cervical muscle tenderness especially in the dorsal neck muscles, levator scapular and trapezius, and were tight in trapezius and the suboccipital muscles. Fifty per cent of these patients with cervicogenic dizziness stated they had temperomandibular joint pain and 77% had some type of headache. These patients were all tender on palpation of the zygapophyseal joints at several levels of the cervical spine. Seven (out of 22) had cervical segmental hypermobility and ten had reduced mobility mainly at the cervico-throracic junction. Malmstrom et al. report that in some of these patients, dizziness was provoked by neck palpation. There is an increased incidence of osteoarthritis and headaches in people with dizziness and neck pain compared to people with only neck pain. Bjorne & Agerberg (2003) observed that 75% of patients with neck pain and dizziness had osteoarthritis and 65% had headaches, while in patients with neck pain alone, 70% had osteoarthritis and 40% had headaches.

When taking the history of these patients, practitioners should question patients about the type of pain, location, severity (on a scale of 0-10), frequency (is it constant and does it occur daily, weekly or less often), and aggravating and easing factors (Wrisley,

Sparto et al. 2000). Similar information is required about occipital pain, headache and stiffness. To make a diagnosis of cervicogenic dizziness, there must be a correlation between the onset, duration and aggravation of dizziness symptoms with symptoms of neck dysfunction such as pain (Wrisley, Sparto et al. 2000).

2.3.2 Postural alignment

There is some suggestion that people with cervicogenic dizziness have disturbances of physiologic cervical lordosis and posture. Seven of 22 patients with cervicogenic dizziness who were assessed by Malmstrom et al. (2007) were found to have poor cervical postural alignment and 14 had partial imbalance of cervical posture (forward or backward deviations of postural alignment of body segments). In a recent study of 86 young people with cervicogenic dizziness, Rzewnicki and Ianica (2008) found 33-38% had disturbances of cervical lordosis on X-ray compared with the control group of young and healthy people.

2.3.3 X ray findings

Some authors have noted that patients with cervicogenic dizziness have abnormalities on X-rays suggesting this may be a source of dysfunction. Wasilewska, Kotowicz, and Domzal, (1994) noted 90% of a group of people with cervicogenic dizziness had changes in the cervical spine X rays while Strek et al. (1998) found 100% of 130 patients with suspected cervicogenic dizziness had radiographical cervical spine degenerative changes such as discography and osteophytes. Although abnormal X ray findings have been reported, they may not be useful in differentiating causes of dizziness. In a study of 60 patients with whiplash injuries Meenen at al. (1994) concluded that radiological investigations contribute little in differentiating the cause of problems as many may have had pre-existing degenerative disease.

2.3.4 Cervicogenic dizziness triggers

For a diagnosis of cervicogenic dizziness to be made, the dizziness must be produced by neck movements or sustained cervical positions (Wrisley, et al., 2000). There should be a close temporal relationship between the unsteady dizziness and neck pain, including time of onset and duration of episodes of dizziness and cervical symptoms and aggravating movements and triggers (Elies, 1984). However, even when the two

complaints neck pain and dizziness appear together, it is sometimes still difficult to establish a causal relationship (Malmstrom, et al., 2007).

Figure 2.4 Cervicogenic dizziness is triggered by cervical spine movements such as extension

The most common neck movement to bring on cervicogenic dizziness is cervical extension (Figure 2.4) (Mulligan, 1999; Reid, et al., 2008) so activities such as looking up, changing a light bulb, hanging clothes on the line, getting groceries off high shelves or painting the ceiling will be implicated. In a study by Malmstrom et al. (2007), eleven of 22 patients said the same provoking factors such as neck or trunk movement induced dizziness and neck pain and a reduction of neck movements relieved dizziness and pain. Bjorne (2007) states that in his study of patients with vertigo, tinnitus, neck tension, head, neck and shoulder pain and temperomandibular dysfunction, 75% of patients could trigger vertigo with head and neck movements. In the study by Malmstrom et al. (2007) patients with cervicogenic dizziness reported their symptoms were provoked by workload, homework, walking and running, neck muscle tension, head on trunk movements, stress and hormonal variations.

It has been reported that cervicogenic dizziness has no latency period and is usually of short duration (Kondratek et al., 2006). The study by Reid et al. (2008) found 40% of patients reported their dizziness was constant or occurring several times a day.

2.4 Examination of a dizzy patient

Several authors have suggested that a thorough examination can lead to a timely clinical diagnosis of the cause of dizziness without referral to specialists for costly and time consuming investigations (Borg-Stein, et al., 2001; Colledge, et al., 1996; Hanley, et al., 2001; Wrisley, et al., 2000). Hoffman et al. (1999) conducted a structured review of the literature looking at the diagnostic evaluation of dizziness. They concluded a primary care approach to diagnosing the cause of dizziness including a careful history and physical evaluation led to a diagnosis in 75% of cases. Only 10% of patients were left undiagnosed. This is supported by a clinical and electronystagmographic study of 255 patients with BPPV by Katsarkas and Kirkham (1978) who concluded the disorder could be diagnosed on the basis of history and clinical assessment.

2.4.1 History

An important part of the manual therapy approach to the treatment of dizziness is a thorough history and examination, which is necessary in making the diagnosis (Wrisley, Sparto et al. 2000). Since there is no specific test for cervicogenic dizziness, the diagnosis is dependent on detailed questioning, about:

- the type of dizziness
- the presence of neck pain or stiffness
- a close relationship between dizziness and neck symptoms with dizziness being triggered by cervical movements or positions (Meadows & Magee, 1990).

It is important to take a detailed history including types of symptoms, onset, frequency and duration, aggravating and relieving factors. Other causes of dizziness should be ruled out based on the history together with the physical examination and specific vestibular function tests. It should be established if the dizziness and neck pain are related (e.g. similar chronological onset, corresponding behaviour in response to movement). The onset of symptoms should be discussed to see if the dizziness is consistent with the most usual presentation of being linked to a traumatic event or a slow onset over time, perhaps caused by cervical spine degeneration.

As previously mentioned in section 2.2.8 one should always be alert for Red Flags by watching for the presence of central nervous system signs, upper motor neurone signs and symptoms in the absence of a diagnosis (Wrisley, Sparto et al. 2000; Kristjansson and Treleaven 2009).

2.4.2 Physical examination

A cervical spine assessment is recommended including observation of posture, cervical spine active movements, palpation and passive accessory mobilisations (Wrisley, Sparto et al. 2000). A clinical examination may also be performed using some procedures used in oculomotor, neurological and vestibular testing such as the Hallpike manoeuvre, gait and balance tests. Objective testing may help to distinguish between cervicogenic vertigo, vestibular dysfunction such as BPPV and labyrinth problems, vertebrobasilar insufficiency (VBI) and migraine-related vertigo (Bisdorff, et al., 2009; Cronin, 1997).

2.4.2.1 Cervical spine active range of motion

When assessing people with cervicogenic dizziness, active cervical range of motion is likely to be a useful measure, though from the literature it is unclear whether to expect an increase or decrease in range. As well as looking for any increase or decrease in range, therapists should also ask about reproduction of pain or dizziness or any other symptoms (Wrisley, Sparto et al. 2000).

Patients in most neck disability studies (without dizziness) report decreased cervical ROM (Sjölander, Michaelson, Jaric, & Djupsjöbacka, 2008). However, in one study of people with neck pain, reduced neck mobility was reported in those with a traumatic cause such as WAD but not in those with insidious onset neck pain, when compared to normative data (Sjölander, Michaelson et al. 2008). In a more recent study there were reductions in extension in the upper cervical levels and flexion in the lower levels in neck pain patients. These ROM changes were weakly associated to pain and self-rated functioning.

Contrary to this, Malmstrom et al. (2009) found cervical range of motion in participants with cervicogenic dizziness was normal or even larger than age and gender matched

reference values. This may potentially be because their sample was limited to people aged 55 years and younger (mean age 37 years).

2.4.2.2 Palpation and passive joint mobilisation of the cervical spine

Range of motion testing is followed by palpation of the neck muscles and testing of cervical joint passive accessory motion, concentrating on the occiput to C3 area. Passive movement restriction and pain on palpation of zygapophyseal joints in the upper cervical spine has been reported in cervicogenic dizziness (Reid et al, 2008). Malmstrom et al. (2009) report that patients with CD often have reduced cervico-thoracic mobility and were tender at several zygapophyseal joints.

On palpation of the neck, local muscle tenderness and muscle tightness has also been reported in people with cervicogenic dizziness (Hinoki, 1985; Kondratek, 2006). In a case report of a patient with cervciogenic dizziness Wrisley et al. (2000) said the patient presented with tenderness to palpation and possible trigger points in her bilateral upper trapezius, sternocleidomastoid and scalenes. Malmstrom et al. (2009) report that patients with CD often have dorsal neck muscle tenderness and tightness and are poorly stabilised in the neck, shoulders and trunk.

2.4.2.3 Dix-Hallpike manoeuvre

Since cervicogenic dizziness is a diagnosis of exclusion, tests must be performed to exclude other causes of dizziness such as vestibular disorders like BPPV, labyrinthine dysfunction, migraine-related vestibulopathy and Ménière's disease (Wrisley, Sparto et al. 2000). The Dix-Hallpike manoeuvre is a test to identify posterior or anterior semicircular canal BPPV. The patient is taken rapidly from the erect long-sitting position, into left or right cervical rotation (45 degrees) to supine head-hanging (30 degrees below the level of the table). It is repeated to the other side (Wrisley, et al., 2000). The test is positive if it produces paroxysmal positional nystagmus which is high in frequency, has a 3-15 second latency but dissipates within 30 to 60 seconds. It has torsional and linear components and then occurs in the reverse direction when the patient returns to the sitting position. It also has fatigability, that is, with repeated

positioning nystagmus rapidly disappears (Baloh & Honrubia, 1990; Froehling, et al., 1994). With cervicogenic dizziness the test may elicit dizziness but not nystagmus.

2.4.2.4 Peripheral vestibular function testing

Peripheral vestibular function testing (electronystagmography testing) may be performed to assess vestibular dysfunction or neurological problems by measuring nystagmus and other eye movements (Baloh & Honrubia, 1990). Peripheral vestibular function testing includes computerised infrared electro oculography, caloric studies, video head impulse tests, cervical Vestibular Evoked Myogenic Potentials (cVEMPs) and ocular Vestibular Evoked Myogenic Potentials (oVEMPs). If these tests are positive they indicate a central or peripheral vestibular cause for dizziness. Although this testing is useful, it may not be readily available as it is usually performed by a neurologist.

2.4.2.5 The smooth pursuit neck torsion (SPNT) test

People with neck pain and dizziness (and particularly those with whiplash disorders) have been found to have disturbances in smooth pursuit neck torsion tests (Tjell & Rosenhall, 1998; Treleaven, Jull, & LowChoy, 2005a). The smooth pursuit neck torsion test is thought to be a measure of neck afferent influence on eye movement control. Hence, it can be used to identify eye movement disturbances when the dizziness is believed to be from perturbations in sensory afferent pathways. and has been suggested as a way of assessing cervicogenic involvement in dizziness (Tjell & Rosenhall, 1998). To perform this test the person's head is kept in the neutral position while their torso is turned torso 45 degrees to the right or to the left (Figure 2.5). Any reproduction of dizziness is noted. Any unusual eye movement such as nystagmus is noted (Tjell & Rosenhall, 1998). Treleaven et al. (2005a) found subjects with WAD and dizziness had greater deficits in eye movement control during the smooth pursuit neck torsion test than those without dizziness and there were also differences to healthy controls. The smooth pursuit neck torsion test has been found to have high sensitivity and specificity in diagnosing cervicogenic dizziness, especially in patients with whiplash (Tjell & Rosenhall, 1998; Treleaven, et al., 2005a).

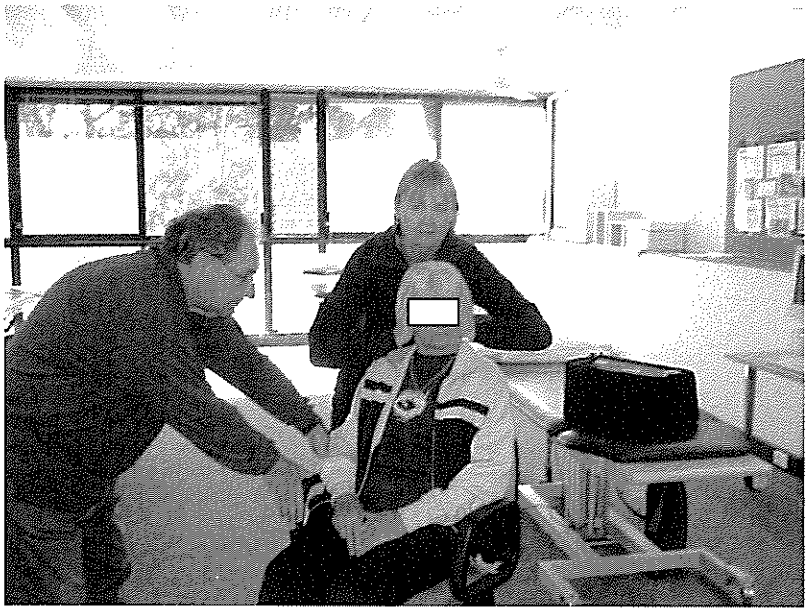

Figure 2.5: Neck torsion test

2.4.2.6 Eye movement tests

To further exclude other forms and causes of dizziness some simple eye movement tests should be performed. Dizziness is often experienced because of perturbation in afferent feedback from the visual, vestibular or proprioceptive systems. Hence, vision is important for balance and perception of motion.

The vestibulo-ocular reflex is a reflex eye movement that stabilizes images on the retina during head movement such as rotation (Baloh & Halmagyi, 1996). It maintains eye fixation during head rotation by moving the eyes in the opposite direction of the head's rotation, at approximately the same speed. It is tested by asking the person to maintain gaze on a stationary target while performing small oscillations of the head up and down, followed by side-to-side. If the person reports dizziness, is unable to maintain the gaze for 60 seconds, has double or blurred vision they may have central or peripheral nervous system dysfunction (Huijbregts & Vidal, 2004).

Smooth pursuit eye movements allow us to fixate and track slowly moving objects. Smooth pursuit requires the coordination of several brain regions. Smooth visual pursuit movement is assessed by the ability to track a slowly moving visual stimulus in a horizontal and vertical direction. The examiner looks for asymmetry of eye

movement or catch-up saccades as this may indicate a central nervous system lesion such as a cerebellar lesion (Huijbregts & Vidal, 2004).

Montfoort et al. (2008) studied the cervico-ocular reflex and vestibulo-ocular reflex in patients with whiplash and concluded that abnormalities in the reflexes and the lack of synergy between them may explain the dizziness these patients experience. Those authors report these tests are useful in assessing subjects with whiplash, especially those complaining of dizziness. In another study, people with neck pain have been shown to have a decreased ability to follow a moving object with head movements (Kristjansson, 2004).

2.4.2.7 Head repositioning accuracy

Head repositioning accuracy (HRA) is the ability to accurately relocate the head to a predetermined position after a maximal active head movement in the horizontal or vertical plane (Rix & Bagust, 2001). It is believed to be a measure of proprioception and to reflect afferent input from the cervical joint and muscle receptors and thus relates well to cervicogenic dizziness (Treleaven, Jull, & Sterling, 2003). The convergence of cues from proprioceptive, visual and vestibular systems controls balance. Cervical proprioception contributes to head position, head orientation in space and thus balance. It is believed a perturbation of cervical proprioception could cause dizziness in people with cervical spine dysfunction. Deficits in neck proprioception have been reported in in those with neck pain and more specifically in those that have suffered a whiplash injury and report dizziness as well as cervical pain (Chen & Treleaven, 2013; Roren et al., 2009; Treleaven, 2010; Treleaven, Jull, & Sterling, 2003).

Proper function of the head-neck system has been shown to rely on proprioception, including joint positioning sense and repositioning sense (Siu, Wing, & Shahidi, 2013). Thus HRA has been used as a measure of cervical proprioception (Rix & Bagust, 2001). Because the joints and deep muscles of the upper cervical spine are involved in proprioceptive input there is an abundance of cervical mechanoreceptors in this area and many connections to the visual, vestibular and central nervous systems (Treleaven, 2008a).

Revel et al. (1991) first proposed the HRA test to quantify the alteration of neck proprioception in participants with neck pain and asymptomatic controls. The Revel protocol involves the subject being seated 90cm away from grid paper attached to the wall and asked to look straight ahead (Revel, Andre-Deshays, & Minguet, 1991). Natural head position (NHP) is achieved and a laser light is fixed to the top of the head (Figure 2.6). Then the subject is told to memorise NHP, because they will be required to duplicate it, after each active head movement. The subject is then allowed a couple of seconds to concentrate and memorise the NHP. The vision of each subject is occluded and a maximal rotation of the head is then performed by the subject firstly to the left and held for approximately two seconds (Figure 2.7). Then the subject tries to relocate the NHP. Revel et al. (1991) found HRA was significantly poorer in participants with neck pain (n=30) compared to asymptomatic participants (n=30), indicating deficits in neck proprioception in those with neck pain. Similar results were also found in other studies suggesting the tests may permit a discriminant classification of people with neck pain (Revel, Minguet, Gregoy, Vaillant, & Manuel, 1994). The Revel studies were important milestones as the method they proposed has been utilised as the standard test in many HRA studies ever since (Hill et al., 2009; Kristjansson & Treleaven, 2009; Roren, et al., 2009; Treleaven, 2008b).

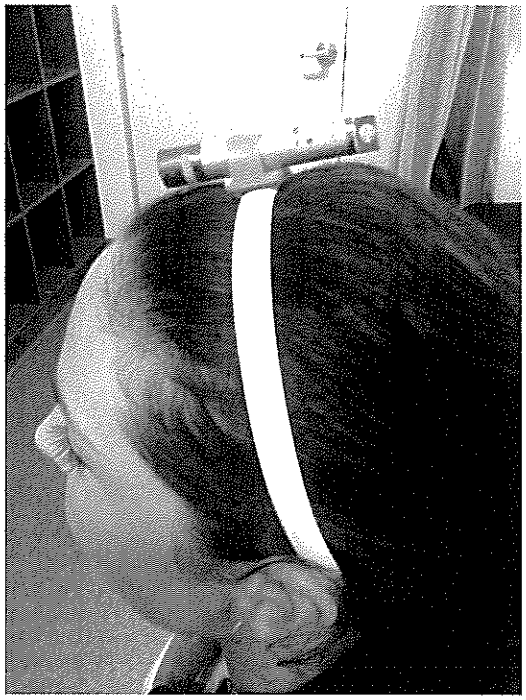

Figure 2.6 The laser light used for testing head repositioning accuracy

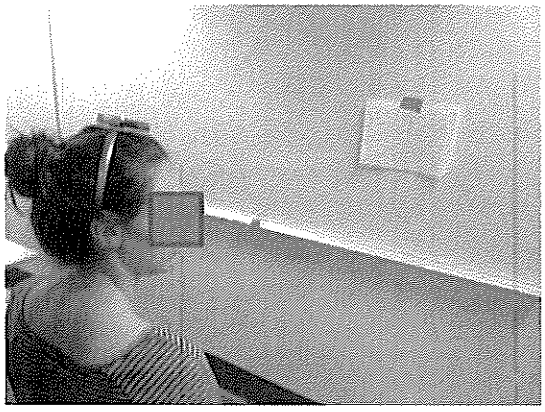

Figure 2.7: Method of testing head repositioning accuracy (Revel 1991)

Methods of measuring head repositioning

Most studies assessing cervical proprioceptive mechanisms use relocation to the NHP (Revel et al. 1991; Hill et al. 2009). Due to the fact that NHP is a commonly performed task and is thus stored in the long-term memory there are suggestions it may be able to be performed without proprioceptive input (Kristjansson, Dall'Alba, & Jull, 2003). This led Kristansson et al. (2003) to compare five different methods of assessing head

relocation in neck pain patients to see which was more reliable. In these studies by Kristjansson et al. relocation inaccuracy was evident in the neck pain groups compared with the asymptomatic group but only with the more traditional test of relocation of the NHP as introduced by Revel et al. They found the more traditional test of relocation of the NHP as described by Revel et al (Revel et al. 1991) was the most reliable test and better than relocating to the 30 degree rotation position (Kristjansson et al. 2003). Hence, their hypothesis that tests which involved more complex tasks might better depict inaccuracies in relocation in neck pain patients because of a greater challenge to the neck proprioceptive system, (Fang, 2010) was rejected as they were unable to differentiate study groups with these more complex tasks. Kristjansson et al. proposed that further study is required with larger sample sizes to better explore the influence of pain versus the influence of injury on cervicocephalic relocation accuracy (Kristjansson et al. 2003).

Most studies investigating proprioception in patients with neck pain have reported greater errors in HRA after cervical movement in those with neck pain than healthy controls (Hill, et al., 2009; Kristjansson, et al., 2003; Revel, et al., 1991; Treleaven, Jull, & Sterling, 2003; Treleaven et al., 2008). Although deficits in HRA have been reported in people with chronic neck pain of both traumatic and insidious origins, there have also been trends for greater head relocation errors in traumatic neck pain subjects compared to those with insidious onset neck pain (Kristjansson, et al., 2003; Sjölander, et al., 2008). Patients with non-traumatic neck pain have been shown to have smaller deficits in HRA than whiplash patients (Sjölander, et al., 2008). A case control study was performed by Kristjansson et al, (2003) to compare HRA in traumatic (whiplash), insidious onset neck pain patients and asymptomatic subjects when targeting a NHP and complex predetermined positions. They found that HRE exists in patients with neck pain with a trend revealing greater deficit in WAD patients compared to insidious onset.

Roren et al. (2009) used two different methods of measuring HRA, the traditional Revel method and a 3D ultrasound method that consists of a specially designed headgear with an ultrasound transmitter to control and measure ROM. Using both methods they found that HRA is significantly higher in healthy groups when compared to groups

with neck-pain, which is in agreement with previous studies results (Heikkilä & Aström, 1996; Revel, et al, 1991; Roren et al., 2009).

Although most studies have reported that patients with neck pain report more head repositioning errors than controls, a study by Rix and Bagust (2001) report no difference between groups. They report that the patient group with chronic, non-traumatic cervical spine pain was no less accurate in head repositioning than the control group for all movement directions except flexion. Thus they concluded that nontraumatic neck pain patients show little evidence of impaired cervicocephalic kinesthetic sensibility. However, these results contrast with studies of chronic cervical pain patients in which the origin was not controlled or involved a cervical whiplash injury. Thus the conclusion of this study may be premature as a result of limited sample size of eleven in the patient group and eleven in the control group. (Rix & Bagust, 2001).

Very few studies to date have assessed HRA in people with cervicogenic dizziness. Most of the previous studies that have shown improvement in head repositioning after treatment have been with whiplash patients. However, Wu et al (2006) showed improvements in head repositioning in people with cervicogenic dizziness after cervical manipulation (Wu, Fang, Hu, Shen, & Jiang, 2006). It has been suggested that cervicogenic dizziness is caused by perturbations to afferent input from proprioceptors in the joints and deep muscles of the upper cervical spine. Hence, HRA should be measured in people suspected of having cervicogenic dizziness as any abnormal findings could aid diagnosis.

2.4.2.1 Balance testing

Patients with cervicogenic dizziness report sensations of unsteadiness of gait, postural imbalance, near falling, falling over, disequilibrium or ataxia (Brandt, 1996). Unsteadiness and dizziness have been reported in 70% of patients with whiplash, with falls occurring in 21% of this cohort (Stokell, Yu, Williams, & Treleaven, 2011; Treleaven, Jull, & Sterling, 2003). To test balance, ideally posturography could be used (Figure 2.8). However, if posturography is not available, the patient should be asked to maintain stance with feet together with eyes open and closed, Romberg, tandem stance

(sharpened Romberg) and single leg stance without upper limb support for at least 30 seconds.

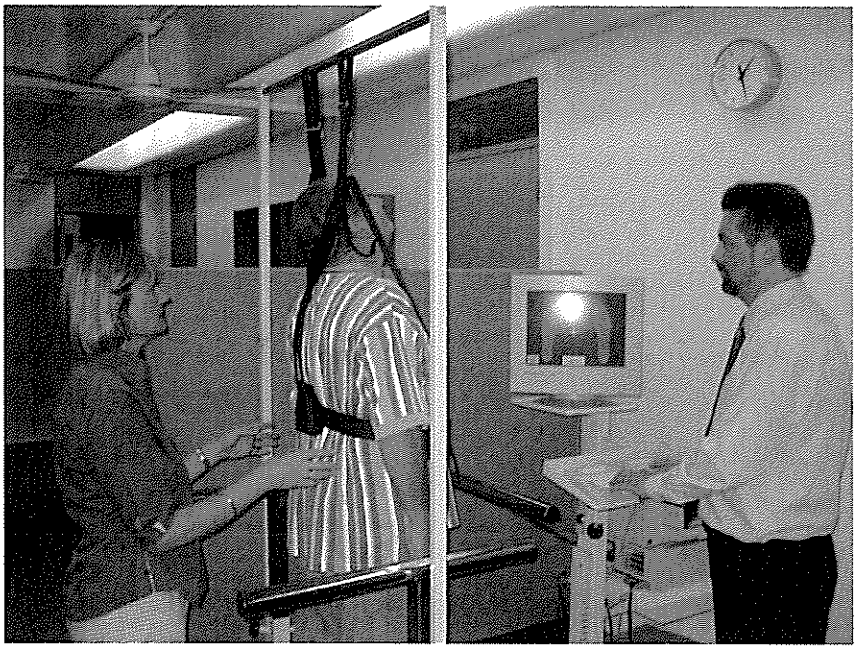

Figure 2.8 Participant having balance measured with the Chattecx posturography balance system

There are suggestions that dynamic balance testing is more relevant than static balance measures in assessing impairments in those with cervical spine dysfunction (Stokell, et al., 2011). The study by Stokell et al. found a correlation between dynamic balance tests and levels of pain, disability and dizziness handicap in traumatic neck pain and dizziness patients, whereas previous studies using static balance measures found few correlations (Treleaven, et al., 2005a). One study showed that static balance did not improve after balance training with visual feedback but dynamic balance did (Hamman, Mekjavic, Mallinson, & Longridge, 1992)

Several studies of patients diagnosed with cervicogenic dizziness reported they had decreased balance, lower equilibrium scores and poorer postural control on posturographic testing than controls (Alund, Ledin, Odkvist, & Sven-Eric, 1993; Karlberg, Magnussen, et al., 1996; Wrisley, et al., 2000; Yahia, et al., 2009). Karlberg et al. (1996) used posturographic assessment of balance to distinguish and identify people

with cervicogenic dizziness from patients with other causes of dizziness. They found that patients with cervicogenic dizziness manifested significantly poorer postural performance than healthy subjects. This led Karlberg et al. (1996) to suggest posturography can distinguish patients with suspected cervicogenic from patients with other causes of dizziness and asymptomatic people. Yahia et al. (2009) measured balance in 32 people with neck pain and dizziness, 30 people with neck pain and no dizziness and 30 healthy controls and found the neck pain and dizziness group had abnormal static and dynamic balance.

Although few studies have examined balance in patients with cervicogenic dizziness, studies from patients with neck pain provide some clues as to the nature of balance disturbance in the presence of neck dysfunction. Several studies have reported significantly worse balance in idiopathic onset neck pain participants than healthy controls (Field, et al., 2008; McPartland , Brodeur, & Hallgren, 1997; Poole, Treleaven, & Jull, 2008; Treleaven, et al., 2008). Field et al. (2008) compared balance responses of subjects with idiopathic neck pain (n=30), to WAD patients (n=30) to healthy controls (n=30). A modified clinical balance test of sensory integration and balance was used to measure standing balance, which has been shown to have excellent test-retest reliability, sensitivity, and validity. Field et al. (2008) found general trends of impaired balance (greater mean total energy of sway) for both the traumatic (whiplash) and idiopathic neck pain groups compared to the control group. Since many of the findings showed general trends rather than statistically significant differences, this study may have been under-powered. These results are supported by Poole et al. (2008) who found that older people (>65) with neck pain had significant gait disturbances with slower selected gait speeds compared to age-matched norms. It has been established that age alone has little effect on both gait and standing balance tests, (Treleaven, Clamaron-Cheers, & Jull, 2011), which suggests that it is neck pain that contributes to the gait disturbances.

An unblinded pilot trial conducted by Stokell et al. (2011) compared dynamic and functional balance in healthy individuals and subjects with persistent WAD. The specific aim was to 'determine whether postural stability differed between subjects with persistent whiplash and healthy controls in selected clinical dynamic and

functional balance measures' (Stokell et al., 2011). This study of twenty subjects with whiplash and twenty healthy controls had extensive exclusion criteria to eliminate those with other causes of poor balance such as vestibular disorders. This study showed deficits for the whiplash subjects compared to healthy controls in balance and gait tasks such as the timed 10m walk test (slower speeds) and the stair test (fewer stairs climbed). Although unsteadiness occurs in subjects with both traumatic (whiplash) and non-traumatic neck disorders, greater unsteadiness has been reported in those with traumatic origin (whiplash injury) than those with insidious-onset neck pain (Field, et al., 2008; Roijezon, Bjorklund, & Djupsjobacka, 2011; Sjostrom et al., 2003; Stokell, et al., 2011).

McPartland et al. (1997) compared standing balance and suboccipital muscle atrophy in seven chronic neck pain subjects and seven asymptomatic controls. The assessments were performed by one partially blinded examiner and one unblinded examiner. Eight clinical tests were performed six times using average velocity of the centre of pressure, average radius of the centre of pressure, and average absolute torque. The partially blinded examiner found larger differences between the subjects and controls on some of the eight tests performed, suggesting that cervical pain may be linked to unsteadiness. Patients with chronic neck pain when compared to the control group, and subjects with significant fatty infiltration and atrophy on MRI of the rectus capitis posterior major and rectus capitis posterior minor muscles had even poorer standing balance (McPartland et al., 1997). The authors concluded that neck muscle atrophy and degeneration may play a substantial role in standing balance and gait disturbances in neck pain sufferers. Nevertheless, there are a few limitations to the research conducted by McPartland et al. (1997), including the unblinding and partial blinding of the examiners. Secondly, several of the results showed only general trends with no statistical significance, indicating insufficient power.

There is a significant difference in standing balance tasks in subjects with neck pain compared to healthy controls (McPartland et al. 1997; Treleaven 2008; Treleaven 2011). If a patient does have imbalance it is important to see if this relates to their dizziness or if it is due to something wrong in the central nervous system (Parkinson's disease, stroke, cerebellar ataxia, multiple sclerosis, Vitamin B12 deficiency and alcoholism), the

peripheral nervous system (neuromuscular problems, peripheral neuropathy, sensory deficits) or the musculoskeletal system (major injuries to the lower limbs) (Borg-Stein, et al., 2001; Brandt & Bronstein, 2001). Cervicogenic dizziness is largely a diagnosis of exclusion, so these other causes for dizziness and unsteadiness must be excluded.

2.5 Treatment of cervicogenic dizziness

Although cervicogenic dizziness is a common problem, the most effective treatment is still debated and thus its management is challenging. Various treatment approaches have been used to treat cervicogenic dizziness. Some authors have reported using a multi-modal approach (Du et al., 2010; Fang, 2010; Karlberg, Johansson, et al., 1996), some have used manual therapy to the cervical spine (Reid, et al., 2008) while others have addressed the sensorimotor deficits (Lystad, et al., 2011). Yet another approach was the use of percutaneous laser to the disc in a study by Yang et al. (2007) of 42 patients with cervical 'vertigo'. Two months postoperative, 28 of the patients reported their dizziness had disappeared (67%), six patients said their dizziness had improved (14%), and for eight patients the dizziness did not improve.

2.5.1 Manual therapy

Several authors have proposed manual therapy interventions for the treatment of dizziness of a cervical origin (Du, et al., 2010; Fang, 2010; Fattori et al., 1996; Galm, Rittmeister, & Schmitt, 1998; Heikkila, et al., 2000; Karlberg, Magnussen, et al., 1996; Lystad, et al., 2011; Malmstrom, et al., 2007; Reid, et al., 2008; Wu et al., 2006; Zhou, Jiang, Li, Zhang, & Wu, 1999). Indeed, it has been suggested that the management of cervicogenic dizziness should be the same as for cervical pain (Brandt & Bronstein, 2001). Manual therapy, which is sometimes also called manipulative therapy, is used worldwide to treat patients with musculoskeletal problems (Bialosky, Simon, Bishop, & George, 2012).

Manual therapy is a structured approach to assessment and treatment of patients with disorders of the neuromusculoskeletal system (Jones & Rivett, 2004; Maitland, 1979a, 1979b). It involves taking a detailed history and performing a careful physical examination of the patient. This is followed by an appropriate treatment that is usually a passive movement technique of either joint mobilisation or manipulation, or a soft

tissue procedure such as massage. The patient is then reassessed to evaluate the success of the procedure, which is either repeated or changed to another technique. Using clinical reasoning and problem solving skills the manual therapist also gives self-management advice which usually includes exercises and education (Corrigan & Maitland, 1983; Jones & Rivett, 2004).

Manual therapy is used to manage pain and other symptoms of musculoskeletal disorders e.g. (headache, dizziness, blurred vision) and to restore normal function and movement (Grieve, 1991; Leaver et al. 2010). Maitland (1979a) states that manual therapy can:

- restore joints to their normal pain-free positions to allow a full range of movement;
- stretch stiff painless joints to restore full range of movement; and
- relieve pain.

Two approaches commonly used with manual therapy are the segmental clinical decision making approach and the responder clinical decision-making approach (Bialosky, et al., 2012). With the former, the aim is to identify a dysfunctional vertebral segment and then apply manual therapy to reduce pain and increase ROM. The second approach, the responder clinical decision-making approach, attempts to characterise patients based on their signs and symptoms that predict their response to manual therapy. The mechanism of action by which manual therapy relieves pain has often been explained as a biomechanical effect of correcting subluxations or re-positioning positional faults (Mulligan, 1994). It is also believed that manual therapy provides adequate input to activate the descending pain inhibitory system (Paungmali & Vincenzio, 2003; Paungmali, Vincenzio, & Smith, 2003). Specifically, it is thought to induce mechanical hypoalgesia involving the non-opioid, descending noradrenergic system (Paungmali, et al., 2003). A review of all RCTs of manual therapy for neck pain found moderate evidence that manual therapy (mobilisation, manipulation and massage) used alone or in combination with other treatment modalities such as hot and cold packs, hydrotherapy, home exercises and electrotherapy was beneficial (Magarey, et al., 2004; Miller, et al., 2010; Nachemson & Jonsson, 2000). However, a systematic

review of randomised trials of manual therapy for neck disorders by Gross et al. in 2002 found that manipulation alone, mobilisation alone, and manipulation combined with mobilisation and treatments including massage showed similar results to placebo, wait period or control (Gross, Kay, & Hondras, 2002). On the other hand, they report that a multimodal approach of manual therapy plus exercise was superior to control treatment for both pain and patient satisfaction.

It has been suggested that there may be a placebo response to manual therapy, which can be used as 'a potential active mechanism' by clinicians to enhance the patient response to manual therapy and further reduce pain (Bialosky, Bishop, George, & Robinson, 2011). Bishop et al (2013) after analysing data from a clinical trial of interventions for neck pain, suggest patient expectations have a strong influence on treatment outcomes and should be considered by physical therapists.

2.5.2 Manual therapy for cervicogenic dizziness

Several authors have proposed using manual therapy as treatment for cervicogenic dizziness (Du, et al., 2010; Kang, Wang, & Ye, 2008; Karlberg, Johansson, et al., 1996; Reid, et al., 2008). It has been hypothesised that when an upper cervical zygapophyseal joint becomes stiff there is a decrease in the number of mechanoreceptors stimulated which could lead to altered sensory input from neck receptors and a mismatch of afferent input to the central nervous system. It has been shown that loss of normal input from Type 1 cervical articular mechanoreceptors leads to dizziness and poor balance (Wyke, 1979). When manual therapy is used, it is believed to be of benefit for this condition because it could restore normal movement, releasing trigger points in the cervical muscles and normalise proprioceptive input (Wrisley, Sparto et al. 2000). Evidence for the increased proprioceptive input is the improved HRA reported by Heikkila et al. (2000) and Wu et al. (2006) after the manual therapy interventions (Wu, et al., 2006).

An earlier systematic review of the literature, to identify clinical trials that have investigated the manual therapy treatment of patients with dizziness of cervical origin, concluded there was limited (Level 3) evidence (Reid & Rivett, 2005). Some of the studies had multi-modal approaches using manual therapy together with acupuncture,

posture correction, electrotherapy and exercises which made it difficult to assess the effect of manual therapy alone. Only one RCT of sound methodological quality was identified by that review. In this study by Karlberg et al. (2007), 17 people with cervicogenic dizziness were found to have reduced dizziness intensity (p=0.007), reduced dizziness frequency (p=0.002), reduced cervical pain (p=0.004) and improved postural performance (p=0.05) after multi-modal physiotherapy. As well as this RCT there were several non-RCT studies of poor methodological quality identified that reported improvements in dizziness after mobilisation and manipulation (Konrad & Gereneser, 1990; Uhlemann, Gramowski, Endres, & Cailles, 1993) or after multi-modal treatment (Bracher, Almeida, Almeida, & Bracher, 2000). Overall, even though the studies generally had low methodological quality, all had a positive outcome, supporting manual therapy as an intervention for cervicogenic dizziness.

Since then Lystad et al. (2011) conducted a systematic review to see the effect of manual therapy as well as vestibular rehabilitation on this condition. This review identified a further 4 RCTs (Du, et al., 2010; Fang, 2010; Kang, et al., 2008; Reid, et al., 2008), several prospective cohort studies (Chen & Zhan, 2003; Strunk & Hawk, 2009; Wu, et al., 2006) and a long-term follow-up of the Karlberg et al. study (Malmstrom, et al., 2007). Lystad et al. (2011) concluded there is now moderate (Level 2) evidence to support the use of cervical manual therapy for cervicogenic dizziness. Six studies (Kang, et al., 2008; Konrad & Gereneser, 1990; Mahlstedt, Westhofen, & Konig, 1992; Reid, et al., 2008; Uhlemann, et al., 1993; Wu, et al., 2006) including two RCTs (Reid et al. 2008, Kang et al. 2008) used only spinal manipulation or mobilisation, or both, as the intervention. The remaining investigations including three RCTs (Karlberg et al. 1996; Fang 2010; Du et al. 2010) utilised a multimodal approach consisting of consisting of several different interventions (spinal manipulation and mobilisation, soft tissue therapy, posture correction, electrotherapy, home exercise programs). The RCT conducted by Reid et al. (2008) was considered by Lystad et al. (2011) to be of 'good methodological quality' and found that participants with cervicogenic dizziness who received treatment with SNAGs had less dizziness, lower scores on DHI, and less cervical pain compared to the placebo group at post-treatment and 6-week follow-up. Balance with the neck in extension and extension range of motion improved in the

SNAG group. The studies by Fang (2010) and Du et al. (2010) ('moderate quality') report significant improvements in dizziness after spinal manipulation and soft tissue therapy. In the 'moderate quality' RCT by Kang et al. (2008), participants were found to have larger reductions in vertebral artery blood flow velocity in the treatment group compared to the control after cervical spine manipulation.

A prospective study of 'moderate quality' of 21 people with cervicogenic dizziness by Strunk and Hawk (2009) reported decreased dizziness and neck pain and improved DHI scores and improved balance after spinal manipulation, myofascial release, post-isometric relaxation, and heat or cold therapy. In a 'poor quality' prospective study Wu et al. (2006) reported improvements in head repositioning accuracy after spinal Tuina manipulation therapy (pressing-kneading manipulation applied continuously to the vertebrae for 5 minutes) of 121 participants believed to have cervicogenic dizziness.

The systematic review of manual therapy treatment for cervicogenic dizziness by Reid and Rivett (2005) identified a study assessing the effects of acupuncture for this condition (Heikkila, et al. 2000). In this case series of people believed to have cervicogenic dizziness, acupuncture was found to improve both dizziness and joint position sense (Heikkila, et al. 2000). In a study by Hawk and Cambron (2009) of elderly patients with poor balance (not actually identified as having cervicogenic dizziness), who underwent a course of 'chiropractic treatment', there were improvements in their dizziness and pain, but not balance.

In conclusion, with this growing body of evidence, with two systematic reviews and several RCTs, there is now level 2 (moderate evidence) for the manual therapy approach to management of this condition.

2.5.3 Sustained Natural Apophyseal Glides (SNAGs)

A SNAG of the cervical spine is one of a group of techniques described by Brian Mulligan, a New Zealand physiotherapist, which have become a popular part of manual therapy treatment internationally (Mulligan, 1989). Although a SNAG is a mobilisation technique mostly used to treat joint restrictions and pain, there is emerging evidence it may be used to treat dizziness as well (Reid, et al. 2008). The technique was first introduced by Mulligan in 1987, as part of a group of techniques

called Mobilisations with Movement (MWM) that aim to restore normal pain-free movement to most joints of the body (Mulligan, 1989). SNAGs are claimed to be safe, painless, gentle and easy to apply (Mulligan, 1991; Wilson, 1996). The clinical acceptance of the cervical SNAG is shown by the fact that the concept is taught by 45 teachers from 17 countries around the world at hundreds of continuing education courses, is described in a number of renowned clinical texts (Boyling & Palastanga, 1994; Grieve, 1991; Hengeveld & Banks, 2014; Petty & Moore, 1998; Vincenzino, Hing, Rivett, & Hall, 2011), and is propagated via an international organization which Mulligan founded (the Mulligan Concept Teachers Association).

Few studies have investigated the effectiveness of SNAGs. A previous study found SNAGs were effective for reducing dizziness, cervical pain and self-perceived handicap after 2-6 treatments, and these effects were maintained for 12 weeks post-treatment (Reid, et al., 2008). Hall et al. (2007) report significantly less headache scores in a group treated with a C1-C2 Self-SNAG compared to a placebo 4 weeks and 12 months after the intervention.

2.5.3.1 Application of SNAGs

A cervical SNAG is applied with the patient seated, in an upright weight-bearing position. The technique is gentle and must remain painless and symptom-free (Mulligan 2010). If rotation is symptomatic, by placing one thumb (reinforced by the other) on the upper vertebra of the implicated joint, the accessory movement can be applied unilaterally (on the articular pillar) (Figure 2.9). If flexion or extension is painful or restricted a postero-anterior (PA) pressure is normally applied centrally to the spinous process of the vertebra (Figure 2.10).

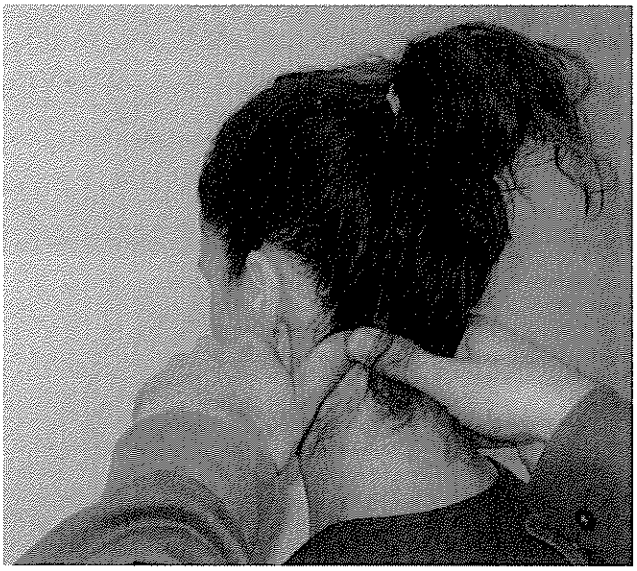

Figure 2.9 SNAG performed into left rotation

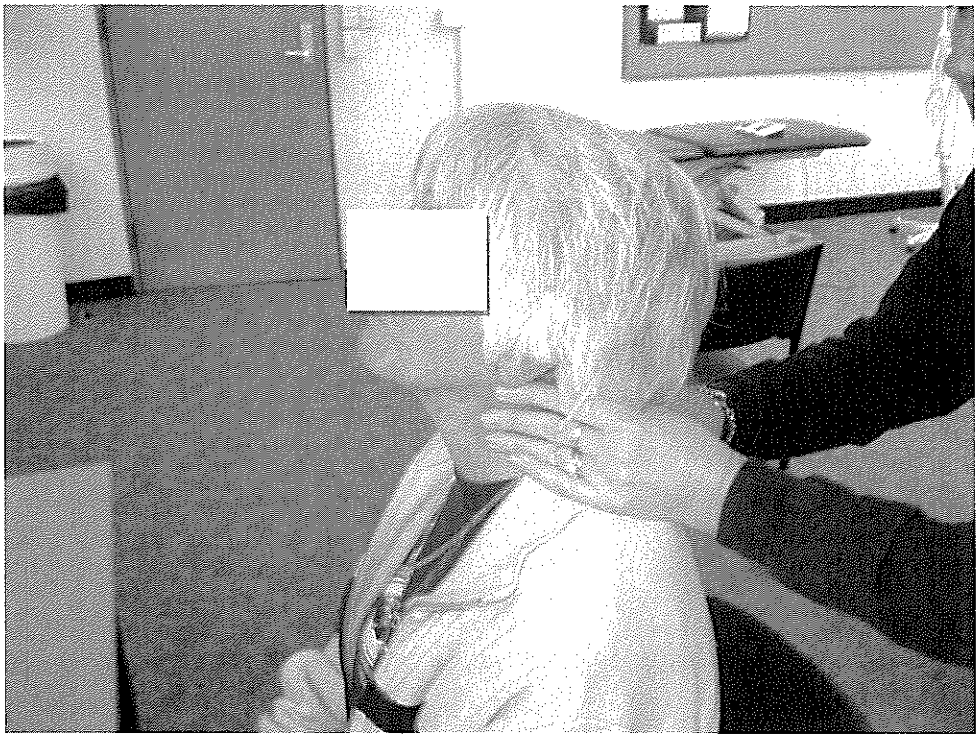

Figure 2.10 SNAG performed into extension

The therapist applies a passive glide of the vertebra in the plane of the zygapophyseal joint. The facet glide is sustained while the patient moves their neck through an active physiological movement to end of range. With cervicogenic dizziness, this would be in the direction that provokes dizziness, but with most patients with cervical dysfunction,

it is usually the painful movement. The patient can then add overpressure to the active movement in an effort to enhance the result (Figure 2.11). After the patient returns to the starting position the therapist releases the glide. If the technique is likely to be effective for the patient, an improvement should be immediately observed (Mulligan, 1999). With this treatment, patients provide real-time and immediate feedback to the therapist regarding its effect.

Figure 2.11 SNAG performed into extension with over-pressure added

2.5.3.2 Self-treatment with SNAGs

Another element of SNAG application that is often required is self-treatment to repeat the SNAG (Vincenzino, et al., 2011). The self-SNAG can be performed as a home exercise using the edge of a towel, the participant's fingers or a strap to perform the glide (Mulligan, 2004) (Figures 2.12-2.15). The participant is usually asked to perform the glide 6-10 times, once a day. The self-SNAG should be symptom-free. The participant is usually asked to perform the glide 6-10 times, once a day. The self-SNAG should also be symptom-free. As mentioned above Hall et al. (2007) used self-SNAGs to the upper cervical spine as the active treatment in their headache study and reported significantly less headache scores in the group treated with the C1-C2 self-SNAG compared to a placebo at four weeks and 12 months post-treatment.

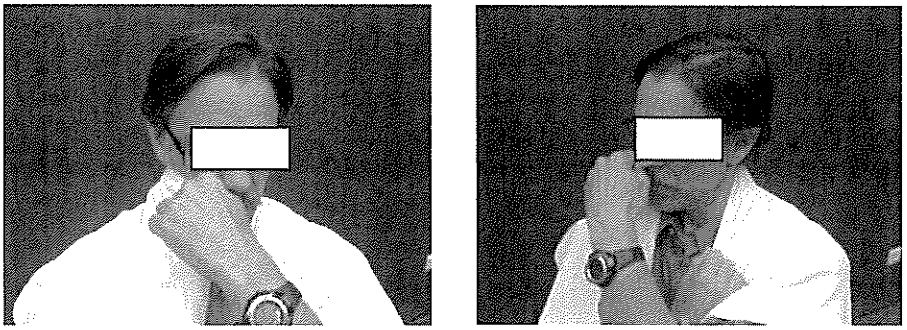

Figure 2.12 Self-SNAG into right rotation using towel

Figure 2.13 Self-SNAG into right rotation using fingers

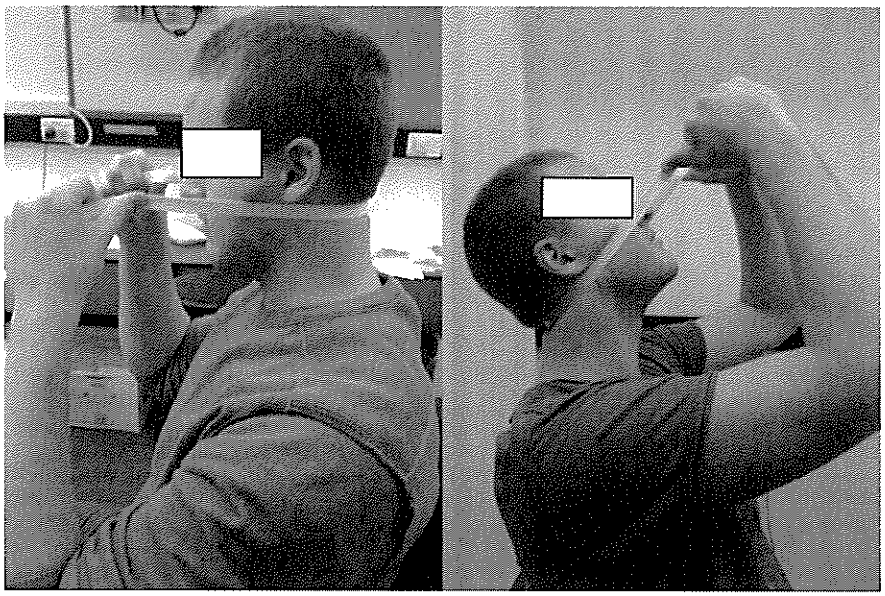

Figure 2.14 Self-SNAG into extension using a strap

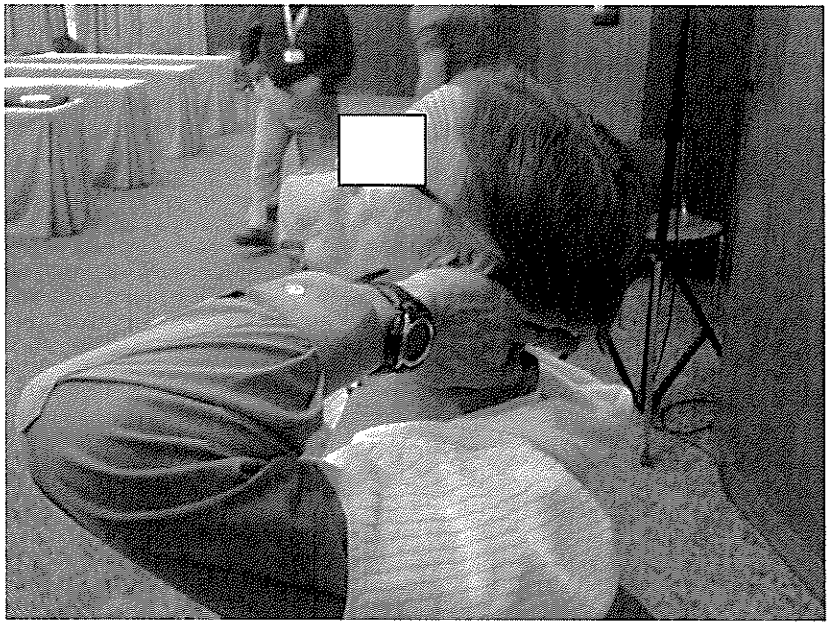

Figure 2.15 Self-SNAG into extension using fingers

2.5.3.3 Mechanism of action

It has been suggested by Mulligan (1991) that SNAGs work by correcting 'minor bony positional faults' or 'mal-tracking' problems. The articular facet is re-positioned and

normal pain-free gliding of the zygapophyseal joint is restored (Mulligan, 1991; Wilson, 1996). Cervical SNAGs are described as producing an accessory glide of the superior facet parallel to the joint surface of the cervical zygapophyseal joint (Mulligan, 1999).

Hearn and Rivett (2002) suggest that because SNAGs are often observed as producing an immediate effect it is unlikely that osseous pathology is affected, but more likely soft tissues in the zygapophyseal joints or the intervertebral discs are implicated. They propose that because of the rapid effect the mechanism is probably mechanical and not a local chemical process or natural resolution. However, even though the mechanism is probably initially mechanical, it may also be theorised that there may be a change in the intra-cellular chemical environment giving the sustained effect from treatment (Hearn & Rivett, 2002). Since treatment of only one specific functional-spinal unit is symptom-free and symptoms remain if other levels are treated, a marked placebo effect is unlikely. The idea of trapped meniscoids being the problem was discounted by Hearn and Rivett, as if meniscoids were the pain source SNAG treatment would be painful as it is performed in a loaded weight bearing position and the ipsilateral active movement of the SNAG would also further compress and irritate the meniscoid. They suggested clinical trials are needed to investigate different combinations of accessory and physiological movements to help explain the mechanism of SNAGs.

In a study where MWM techniques were applied to treat lateral epicondylalgia they were found to result not only in improved grip strength but also increased range of movement at the shoulder joint (Abbott, 2001). Abbott proposed that MWM act neurophysiologically to decrease the contractile activity of the shoulder rotator muscles. MWM techniques have also been found to produce a rapid pain relieving effect that has an accumulative hypoalgesic effect with repeated treatment sessions (Paungmali & Vincenzio, 2003; Paungmali, et al., 2003; Vincenzino, et al., 2011). Some authors have suggested that more than three treatment sessions are required for significant changes in the pain and functional outcome measures (Paungmali & Vincenzio, 2003; Paungmali, et al., 2003).

Mulligan (1994) put forward a theory to help explain the need for ipsilateral physiological rotation during the application of a cervical SNAG. He says that the superior facet of the implicated joint ipsilateral to the side of pain may be jammed postero-inferiorly in an extension or 'closed down' position, resulting in a gliding dysfunction. Ipsilateral rotation would cause pain by further 'closing down' of the zygapophyseal joint. By applying an accessory glide the superior facet may be repositioned supero-anteriorly allowing greater rotation (Mulligan, 1994). Not only is the zygapophyseal joint mobilised but the whole functional-spinal unit including the intervertebral disc is influenced.

2.5.3.4 SNAGs for cervicogenic dizziness

In 1991 Mulligan proposed SNAGs as an effective treatment for cervicogenic dizziness, or more specifically dizziness brought on by active cervical movement. He suggests cervical extension is the most common movement to produce dizziness, followed by rotation and then flexion (Mulligan, 1999). With cervicogenic dizziness, if extension is the aggravating movement, a postero-anterior (PA) pressure is normally applied to the spinous process of C2. As the therapist applies a glide ventrally the patient actively extends their neck (Figure 2.2). The patient can then add overpressure to the active movement in an effort to enhance the result (Figure 2.3). If left rotation is the problem then the ventral glide is applied ipsilaterally on the left C1 transverse process, in an effort to mobilise the C1/2 joint (Figure 2.4). This is followed by active left rotation. If this is not effective, the glide is then performed on the contralateral right transverse process of C1 while active rotation is still performed to the left. Mulligan reports that if the patient has giddiness then thumb placement on the offending side is usually more successful than on the contralateral side (Mulligan, 1999; Mulligan, 2004). If cervical flexion is the aggravating movement then the thumbs should be placed over the spinous process of the superior vertebra. The initial choice would be on C2 as per cervical extension problems. As the patient flexes the direction of glide is horizontal at full flexion (Mulligan, 1999). Mulligan (1999) suggests that often only one or two treatments are required to eliminate dizziness, although he provides no evidence to support this assertion.

A RCT by Reid et al. (2008) found that SNAGs are an effective manual therapy technique for the treatment of cervicogenic dizziness in the short term. SNAGs were shown to have a clinically and statistically significant immediate and sustained (for 3 months) effect in reducing dizziness, neck pain and disability caused by cervical spine dysfunction.

The results of this research is consistent with proposed theories of the mechanism by which SNAGs restore movement (Hearn & Rivett, 2002), which suggest it is an underlying mechanical mechanism. It may be hypothesised that, when normal gliding of the joint surfaces is restored by SNAGs, range of motion improves, stimulation of mechanoreceptors in the joints and muscles is increased, and dizziness and poor balance may be relieved.

The most likely cause of cervicogenic dizziness is either a loss or inadequate stimulation of receptors of the joints and deep muscles in the upper cervical spine (Brandt & Bronstein, 2001). With SNAGs, this deficit is possibly addressed by:

- gliding the joint and therefore increasing stimulation of mechanoreceptors in the joint, and
- active cervical movement which stimulates the muscle spindles especially in the deep, short intervertebral neck muscles.

Despite the widespread use of this technique clinically and the plausible biological theories for its success, the efficacy of the SNAG procedure for cervicogenic dizziness has not been empirically validated in any other research besides that of Reid et al. (2008). A search of Medline (1966 onwards), Cumulative Index to Nursing and Allied Health Literature (CINAHL) (1982 onwards), Physiotherapy Evidence Database (PEDro) and EMBASE (1988 onwards) found no empirical evidence for the efficacy of cervical SNAGs in treating cervicogenic dizziness other than the Reid et al. study. High quality research that examines the efficacy of this type of manual therapy is needed to justify its continued use in treating dizziness over the longer term.

2.5.4 Maitland passive joint mobilisations

Passive joint mobilisations have been described by Geoffrey Maitland to treat cervical pain (1977) and constitute a mainstream treatment with 99.8% of physiotherapists in

one study using this approach (Magarey, et al., 2004). Further, a systematic review of manual therapy and exercise for neck pain found of 17 RCTs, 15 used some form of joint mobilisation (Miller, et al., 2010). Passive joint mobilisation has been found to be an effective technique to treat cervical dysfunction and pain (Magarey, et al., 2004; Miller, et al., 2010). Some manual therapists have also reported they can be used to treat cervicogenic dizziness but to date there is no evidence for this claim.

The term mobilisation means repetitive, rhythmic, passive movement applied to a peripheral or vertebral joint (Grieve, 1981; Maitland, Banks, English, & Hengeveld, 2001). Passive joint mobilisations are described by Maitland (1979a) as consisting of two basic types of passive movements:

- large or small amplitude passive oscillatory movements, at a rate of two or three per second, and applied anywhere in a range of movement;
- small amplitude oscillations applied at the limit of the joint range combined with sustained stretching.

The degree of vigour (grade according to Maitland) and duration of the application are determined by clinical judgement by the therapist, but usually consisted of three 30-60 second applications at each spinal level treated. The technique may utilise the joint's accessory movements (those which a person cannot perform themselves in isolation) or the joint's physiological movements (those that the patient can carry out actively). In the spine these techniques are performed as central passive joint mobilisations over the spinous process (Figure 2.16), or as unilateral passive joint mobilisations over the articular pillar (Figure 2.17). To perform the technique adequately, the therapist must gain the 'feel' of the joint.

Once a treatment movement is chosen it can then be graded according to where it is performed within the available range of that movement and its amplitude and the level of resistance or stiffness felt by the clinician:

Grade I is a small amplitude movement at the beginning of range

Grade II is a large amplitude movement well into range and free of stiffness or muscle spasm

Grade III is also a large amplitude movement but goes into stiffness or muscle spasm

Grade IV is a small amplitude movement stretching into stiffness or muscle spasm (Grieve, 1981; Maitland, et al., 2001).

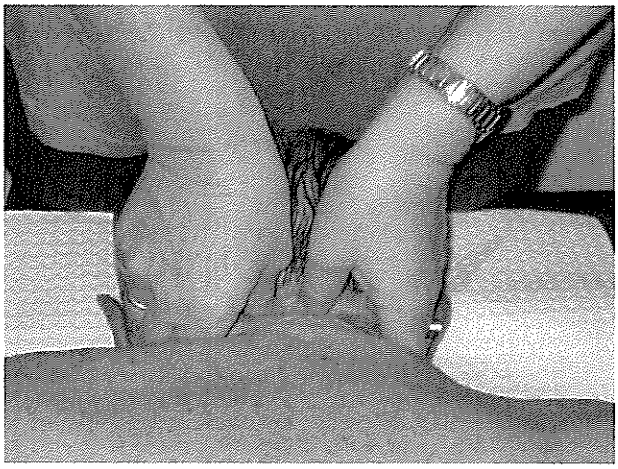

Figure 2.16 Maitland central postero-anterior passive joint mobilisations

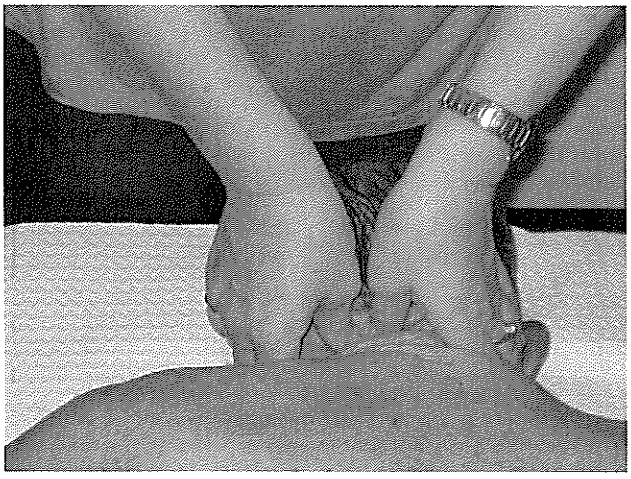

Figure 2.17 Maitland unilateral postero-anterior passive joint mobilisations

The mechanism by which mobilisation reduces pain or symptoms of spinal origin has not been shown. Kaltenborn (1989) suggests that decreased joint gliding can lead to joint hypomobility and therefore impaired joint function and then pain. Hence restoring normal joint movement will encourage pain-free voluntary movement (Maitland, et al., 2001). Mulligan (1994) also stresses that techniques that restore movement to the zygapophyseal joints facilitate normal pain-free function. As

previously discussed, Hearn and Rivett (2002) suggest that when a mobilising technique has an immediate effect, as joint mobilisations often purportedly do, then the underlying mechanism is either mechanical or reflexogenic.

Several possible mechanisms of action of manual therapy induced pain relief have been proposed relating to biomechanical, neurophysiological and psychological mechanisms. The Bialosky et al. (Bialosky, Bishop, Price, Robinson, & George, 2009) model suggests that the mechanical force from manual therapy causes a series of neurophysiological responses from the central and peripheral nervous systems which are then responsible for the clinical outcomes such as pain relief. Although it is commonly claimed that mobilisations work by having a mechanical effect, other authors have suggested that spinal manual therapy may be used to interrupt the pain cycle (Exelby, 1995; Paungmali & Vincenzio, 2003; Paungmali, et al., 2003; Vincenzio, Gutschlag, Collins, & Wright, 1995). Oscillatory mobilisations have been shown to produce an immediate sympatho-excitatory effect (Peterson, Vincenzio, & Wright, 1993). Peterson et al. (1993) used a randomised, repeated, double blind, placebo controlled design to evaluate the effects of a postero-anterior grade III mobilisation on C5. Changes in skin temperature and skin conductance were noted soon after mobilisation indicating cutaneous sympathetic function. Paungmali and Vincenzio (2003) concluded that there was a multi-system effect of the sympathetic nervous system, the motor system and a stimulation-induced analgesic effect which was responsible for manual therapy-induced hypoalgesia. The stimulation-induced analgesia is thought to be caused by stimulation of the lateral-dorsal periaqueductal gray, which produces an immediate non-opioid hypoalgesia, accompanied by sympathoexcitation and motor facilitation.

Mobilisation is advocated in the treatment of cervicogenic dizziness to restore normal movement between the upper cervical vertebrae and at the atlanto-occipital joints (Odkvist & Odkvist, 1988). Consistent with this approach, Oostendorp et al. (1993) suggest that mobilisation of an upper cervical zygapophyseal joint increases the number of responding mechanoreceptors (which decreased when the joint became stiff). This would theoretically increase the afferent information from the joint to the vestibular system and potentially reduce cervicogenic dizziness. As well, it could lead

to normal oculomotor reflexes and postural control that would also help reduce dizziness. Even though Maitland mobilisations are widely used in Physiotherapy practise to treat many cervical disorders there is no evidence for their use with cervicogenic dizziness.

2.5.5 Exercises

There is some evidence for the success of exercises in treatment cervciogenic dizziness. Repetowski et al. (2005) did a study to assess the effectiveness of kinesiotherapy in cervical vertigo treatment. Thirty two patients with cervical vertigo, aged 20 to 75, were examined. The patients performed 'kinesiotherapy exercises' three times a day for four weeks. The effects of therapy were assessed by everyday task self-control cards and ENG examinations. In the present study, objective improvement was noticed in 18.7% patients. Subjective improvement assessed by the patient's everyday task self-control cards reached 62.5%. Kinesiotherapy seems to be the good method of treating neck-related vertigo, but it should be confirmed in further study on the large group of patients.

A RCT conducted in Sweden that used exercise to treat office workers with neck pain report decreased intensity of neck pain with specific resistance training (n = 180) from VAS pain scored of 5.0 ± 0.2 to 3.4 ± 0.2 ($p < 0.0001$), and with all-round physical exercise (n=187) from 5.0 ± 0.2 to 3.6 ± 0.2 ($p < 0.001$), whereas with the control intervention of general health counselling (n=182) there was no change (Andersen et al., 2008).

2.5.6 Sensorimotor rehabilitation

Another treatment approach that is advocated for cervicogenic dizziness is sensorimotor rehabilitation exercises (Sjolander, Michaelson, Jaric, & Djupsjobacka, 2008; Treleaven, 2008b). These are exercise programs that emphasise the importance of the integration of the cervical, visual and vestibular systems (Jull, Falla, Treleaven, Hodges, & Vicenzino, 2007; Kristjansson & Treleaven, 2009; Revel, et al., 1994; Treleaven, 2010). The systematic review by Lystad et al (2011) identified the use of movement habituation exercises, eye/hand coordination exercises, postural control exercises, and exercises desensitising the vestibular system have been used in the

treatment of dizziness. Trubin et al. (1995) found patients with dizziness and balance disturbances following cervical trauma had multi-sensory deficits and rely on accurate visual cues to maintain balance Hence, it has been suggested that exercises that stimulate the sensorimotor system such as those used in vestibular rehabilitation, should be included in treatment of people with balance problems (Hansson & Hakansson, 2009; Lystad, et al., 2011; Sjölander, et al., 2008; Treleaven, 2010; Wrisley, et al., 2000). It has been shown that dynamic but not static balance improved after repeated therapy sessions of balance training using visual feedback in a group of participants without dizziness or pain symptoms (Hamman, et al., 1992). There is some evidence that neck proprioception (HRA and smooth pursuit neck torsion) can be improved with exercises including neck relocation training, gaze stability training, eye head co-ordination, as well as balance practise so these exercises should be included in rehabilitation programs of cervicogenic dizziness patients (Kristjansson & Treleaven, 2009; Treleaven, 2010). Training in joint position error has been shown to decrease that error as well as neck pain intensity and disability (Jull, et al., 2007; Treleaven, 2010). Additionally, manipulation has been shown to improve head repositioning in this population (Heikkila, et al., 2000; Wu et al, 2006).

Vestibular rehabilitation for patients with whiplash-associated disorder has been shown to decrease self-perceived handicap and increase postural control (Hansson & Hakansson, 2009). An RCT of participants with WAD and dizziness found that the group that received vestibular rehabilitation (n=16) had significantly improved balance and DHI scores compared to the control group (n=13) and these effects were maintained for 3 months after the intervention phase (Hansson, Mansson, Ringsberg, & Hakansson, 2006). Hansson et al admit their sample size was small but their attempt to recruit over a period of 2 years and 9 months and the fact that they had 11 dropouts from the study reflect the difficulties of recruiting for running a RCT.

In people with neck pain and dizziness after a whiplash injury, rehabilitation exercise programs that include eye-head coupling have been shown to improve HRA so these exercises should be included in a rehabilitation program if deficits in HRA are found (Jull, et al., 2007; Revel, et al., 1994).

2.5.7 Multi-modal approaches

It has been suggested that a combined multi-modal approach to address not only abnormal cervical afferent input (treated with manual therapy), but also integration of the cervical, visual and vestibular systems (treated with sensorimotor rehabilitation programs) may be needed to treat cervicogenic dizziness (Lystad, et al., 2011; Treleaven, 2010; Wrisley, et al., 2000).

Several multi-modal approaches have been suggested for the treatment of cervicogenic dizziness (Du, et al., 2010; Fang, 2010; Karlberg, Johansson, et al., 1996). The heterogeneity of cervicogenic dizziness, together with the large variety of treatment approaches and poor reporting, makes it difficult to interpret the evidence. The treatment of cervicogenic dizziness has included manual therapy (mobilisation and manipulation), traction, electrotherapy, exercises (balance, vestibular, posture and active range of movement), soft tissue therapy such as massage, trigger point injections, muscle relaxants and the use of a cervical collar (Wrisley, et al., 2000). One RCT study assessing multi-modal treatments, by Karlberg et al. (1996) and its long-term follow-up by Malmstrom et al. (2007), reported reductions in dizziness and neck pain 2 years after the intervention of physiotherapy that was guided by the physical findings. The treatments included soft tissue massage, stretches, local manual therapy, stabilisation exercises, posture correction, home exercises but no vestibular rehabilitation exercises. The RCTs by Fang et al. (2010) and Du et al. (2010) used spinal manipulation as well as soft tissue therapy and report significant improvements in dizziness scores post treatment and at a six-month follow-up.

A multi-modal approach has been advocated by Wrisley et al. (2000) who suggest a combination of manual therapy and exercises (including vestibular rehabilitation) is required for successful results. They report two cases of patients improving with a multi-modal approach which included soft tissue massage (to sternocleidomastoid), mobilisation, TENS, ice and a home program of exercises. The vestibular exercises included eye exercises to improve the efficacy of the vestibular-occular reflex and balance exercises with graded exposure to sensory inputs (Wrisley, et al., 2000). Vestibular rehabilitation aims to promote vestibular adaptation by enhancing gaze stability, improving balance, and improving activities of daily living (Herdman, 2000).

Similarly, Borg-Stein et al. (2001) reviewed outcomes for 15 patients with cervicogenic dizziness who were treated in an outpatient rehabilitation program with various interventions directed at myofascial pain and/or kinaesthetic function. Unfortunately they did not report what the treatment involved. Twenty-seven per cent reported no further episodes of dizziness and 82% of the rest had a decrease in the frequency of episodes of dizziness.

Further, several single case reports lend some support to the multi-modal approach. A 49 year-old man with dizziness after a whiplash injury was given symptomatic relief with a combination of physical therapy (to increase range of motion of the neck and reduce muscle spasm), muscle relaxants and a soft collar (for one to two hours per day) (Furman & Cass, 1996). Three other case reports presented by Cote et al. (1991) describe the successful treatment of cervicogenic dizziness with manual therapy (including mobilisation, massage and manipulation to the cervical spine or temperomandibular joint). In another similar case, Anderson and Yardley (1998) report a 68 year-old lady with dizziness and balance problems who was treated successfully with physiotherapy including education, exercises and a home exercise program including head and neck exercises, balance exercises, relaxation and cognitive behaviour therapy. The exact type of exercise was not reported. These cases show a multi-modal approach can be effective however it is more time consuming for both the therapist and the patient than a few treatments needed with manual therapy (Reid, et al., 2008).

Other factors that may have to be considered are muscle fatigue (Falla, Jull, Russell, Vincenzino, & Hodges, 2007), altered muscle spindle activity due to the presence of inflammatory mediators and the effects of pain locally, in the spinal cord and within the central nervous system (Poole, et al., 2008). More broadly psychosocial modulators of pain and cognitive-behavioural therapies may have to be incorporated into management approaches. There may be a need for stratified and individualised rehabilitation for people with traumatic neck injuries and dizziness to lessen the transition to chronicity (Jull et al., 2011). It is unknown which combinations of treatment are most beneficial for cervicogenic dizziness in a multi-modal treatment approach. Because it is unknown which component is having the beneficial effect, it supports investigating a single intervention at a time.

It has been suggested that manual therapy in conjunction with specific rehabilitation programs that incorporate balance and proprioception retraining with eye-head coupling exercises, gaze stability, head on trunk relocation practice to stimulate the sensory motor system may be the best approach in the management of people with cervicogenic dizziness (Treleaven, 2010, 2011). Although it has been suggested that a combination of manual therapy and vestibular type rehabilitation may be beneficial for cervicogenic dizziness (Schenk, 2006), a systematic review by Lystad et al. (2011) found no observational or experimental studies investigating this combined approach.

As there is still debate as to the best treatment for cervicogenic dizziness, there is a need to investigate individual components of potentially effective treatments in order to determine what works for patients and the effects of treatment components on the various symptoms (dizziness, disability and cervical pain) and signs (ROM, head repositioning accuracy and imbalance) of cervicogenic dizziness. It is thus proposed to assess the effects of manual therapy on cervicogenic dizziness and evaluate its effects over the short and long term. Determining the optimal treatment for patients with this condition is important as cervicogenic dizziness is disabling and significantly affects quality of life.

2.6 References for Chapter Two

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Chapter 3 Efficacy of manual therapy treatments for people with cervicogenic dizziness and pain: protocol of a randomised controlled trial

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Overview

This chapter outlines the design for the randomised controlled trial (RCT) that is the basis for this thesis. This RCT compared the effectiveness of SNAGS to Maitland passive cervical joint mobilisations and to a placebo in reducing signs and symptoms of cervicogenic dizziness and pain immediately post-intervention, at 12 weeks and at 12 months.

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Efficacy of manual therapy treatments for people with cervicogenic dizziness and pain: protocol of a randomised controlled trial

Susan A Reid*, Darren A Rivett, Michael G Katekar and Robin Callister

Abstract

Background: Cervicogenic dizziness is a disabling condition characterised by postural unsteadiness that is aggravated by cervical spine movements and associated with a painful and/or stiff neck. Two manual therapy treatments (Mulligan's Sustained Natural Apophyseal Glides (SNAGs) and Maitland's passive joint mobilisations) are used by physiotherapists to treat this condition but there is little evidence from randomised controlled trials to support their use. The aim of this study is to conduct a randomised controlled trial to compare these two forms of manual therapy (Mulligan glides and Maitland mobilisations) to each other and to a placebo in reducing symptoms of cervicogenic dizziness in the longer term and to conduct an economic evaluation of the interventions.

Methods: Participants with symptoms of dizziness described as imbalance, together with a painful and/or stiff neck will be recruited via media releases, advertisements and mail-outs to medical practitioners in the Hunter region of NSW, Australia. Potential participants will be screened by a physiotherapist and a neurologist to rule out other causes of their dizziness. Once diagnosed with cervciogenic dizziness, 90 participants will be randomly allocated to one of three groups: Maitland mobilisations plus range-of-motion exercises, Mulligan SNAGs plus self-SNAG exercises or placebo. Participants will receive two to six treatments over six weeks. The trial will have unblinded treatment but blinded outcome assessments. Assessments will occur at baseline, post-treatment, six weeks, 12 weeks, six months and 12 months post treatment. The primary outcome will be intensity of dizziness. Other outcome measures will be frequency of dizziness, disability, intensity of cervical pain, cervical range of motion, balance, head repositioning, adverse effects and treatment satisfaction. Economic outcomes will also be collected.

Discussion: This paper describes the methods for a randomised controlled trial to evaluate the effectiveness of two manual therapy techniques in the treatment of people with cervicogenic dizziness for which there is limited established evidence-based treatment.

Trial registration: ACTRN12611000073909

Background

Dizziness is a very common condition in the community that often leads to physical problems such as unsteadiness and falls, as well as social, emotional and financial issues [1]. There are many causes of dizziness, one being a dysfunction in the upper cervical spine [1-4]. In this condition, termed cervicogenic dizziness, the non-rotary dizziness is described as imbalance or unsteadiness and is related to movements or positions of the neck. Cervicogenic dizziness is accompanied by a range of symptoms including neck pain, neck stiffness, headache, and less often visual disturbances, nausea, ear fullness, sweating, tinnitus, problems with swallowing, temporomandibular joint pain, upper extremity radiculopathy, general weakness and psychological symptoms such as anxiety and disturbances in concentration and memory [5]. Although a disabling condition, there is no established treatment. There is some evidence for manual therapy treatment of this condition but good quality randomised controlled trials (RCTs) are scarce [3,6]. The existence of cervicogenic dizziness has been a topic of some controversy [7,8] but more recent studies and

* Correspondence: Sue.Reid@newcastle.edu.au
The Faculty of Health, The University of Newcastle, Callaghan, NSW 2308, Australia

reports have provided evidence in support of its existence [4,9-16].

It has been suggested by Hulse [17] that one third of people with cervical dizziness have their onset due to trauma such as whiplash, one third have an insidious onset following spinal degeneration, and one third are due to other causes. Whiplash injuries are experienced by 0.1% of the population [18] and the incidence of symptoms of dizziness in whiplash sufferers has been variously reported as 20-58% [19], 40-80% [20] and as high as 80-90% [21-23].

Cervical spondylosis, where the cervical zygapophyseal joints are under abnormal mechanical stress, is a major cause of poor balance and dizziness associated with spinal degeneration [19]. This may occur in people with vertebral collapse, decreased cervical disc height or herniated discs, degenerative lesions (e.g. osteoarthritis), inflammatory diseases (e.g. rheumatoid arthritis), vertebral displacement, muscle spasm, or in those wearing cervical collars [19,24,25]. The cervical zygapophyseal joints are the most densely innervated of all the spinal joints [26] with 50% of all cervical proprioceptors occurring in the joint capsules of C1 to C3 [17]. In a study by Colledge et al. [27] investigating the causes of dizziness in the elderly, the authors attributed dizziness to cervical spondylosis in 65% of cases. In support of this theory, extensive degenerative changes such as osteophytes and discopathy on cervical X-rays have been reported in people with this problem [24,28,29]. It is believed that dizziness can also be caused by dysfunction of the deep muscular proprioceptors in the upper cervical spine leading to abnormal input to the vestibular nuclei [19]. In a study by Treleaven, Jull and Sterling in 2003, people with whiplash-associated dizziness and/or unsteadiness (n=102) were shown to have significantly greater joint position errors and a higher neck pain index than control subjects (n=44), consistent with cervical mechanoreceptor dysfunction being a likely cause of the symptoms [30].

Manual therapy treatments

In 1991, Brian Mulligan, a New Zealand physiotherapist, introduced a physical therapy treatment for cervicogenic dizziness called Sustained Natural Apophyseal Glides (SNAGs) [31]. Although this treatment is used clinically and is accepted in the Physiotherapy profession, there has been very little research to evaluate its efficacy for cervicogenic dizziness. SNAGs have been shown to be an effective treatment for this problem in the short term (12 weeks) [4], however no longer term follow-up of this treatment has been undertaken. Geoff Maitland described another form of manual therapy called passive joint mobilisations that are commonly used to treat neck pain, headaches and other neck problems [32]. A systematic review of the literature showed there is a lack of quality studies evaluating the treatment of cervicogenic dizziness with manual therapy and no studies evaluating the efficacy of Maitland mobilisations in the treatment of this condition [3].

Study aims

The aim of this paper is to report the study protocol used to investigate the effectiveness of two manual therapy treatments in reducing the symptoms of cervicogenic dizziness and associated pain over 12 months. This will be investigated by comparing the effects of these treatments to each other and to a placebo intervention. Other aims of the study are: 1) to assess the effects of the interventions on cervical range of motion, head repositioning and balance; 2) assess and compare the cost effectiveness of the interventions; and 3) to report any possible adverse effects and treatment satisfaction.

Methods/Design

A prospective RCT with unblinded treatment and blinded outcome assessment will be conducted in the School of Health Sciences at the University of Newcastle, Australia. Participants (with cervicogenic dizziness) will be randomly allocated to SNAGs, Maitland passive joint mobilisation or placebo groups. Each participant will receive two to six treatments by an experienced physiotherapist over six weeks at the discretion of the treating therapist who will use their clinical judgement to determine the specific dosage based on the participant's response. Treatment will cease if the participant perceives the condition is adequately improved or if the improvement plateaus, that is, no further improvement is evident over three successive visits.

Ethics approval

The study design and procedures were approved by the University of Newcastle Human Research Ethics Committee (Protocol Number: H-2009-0377), and the procedures followed were in accordance with the Helsinki Declaration of 1975, as revised in 2000. Written informed consent will be obtained from all participants prior to enrolment in the study.

Participants and recruitment

Ninety participants with cervicogenic dizziness will be recruited in the Hunter region of NSW, Australia, via media releases and resulting radio interviews and newspaper articles, by advertisements in local newspapers, and by referral from medical practitioners including neurologists (Figure 1 Flow chart). Inclusion and exclusion criteria are summarised in Table 1.

Screening of potential participants for cervicogenic dizziness

A three step process will be followed to identify people with cervicogenic dizziness. Firstly, an initial phone screening

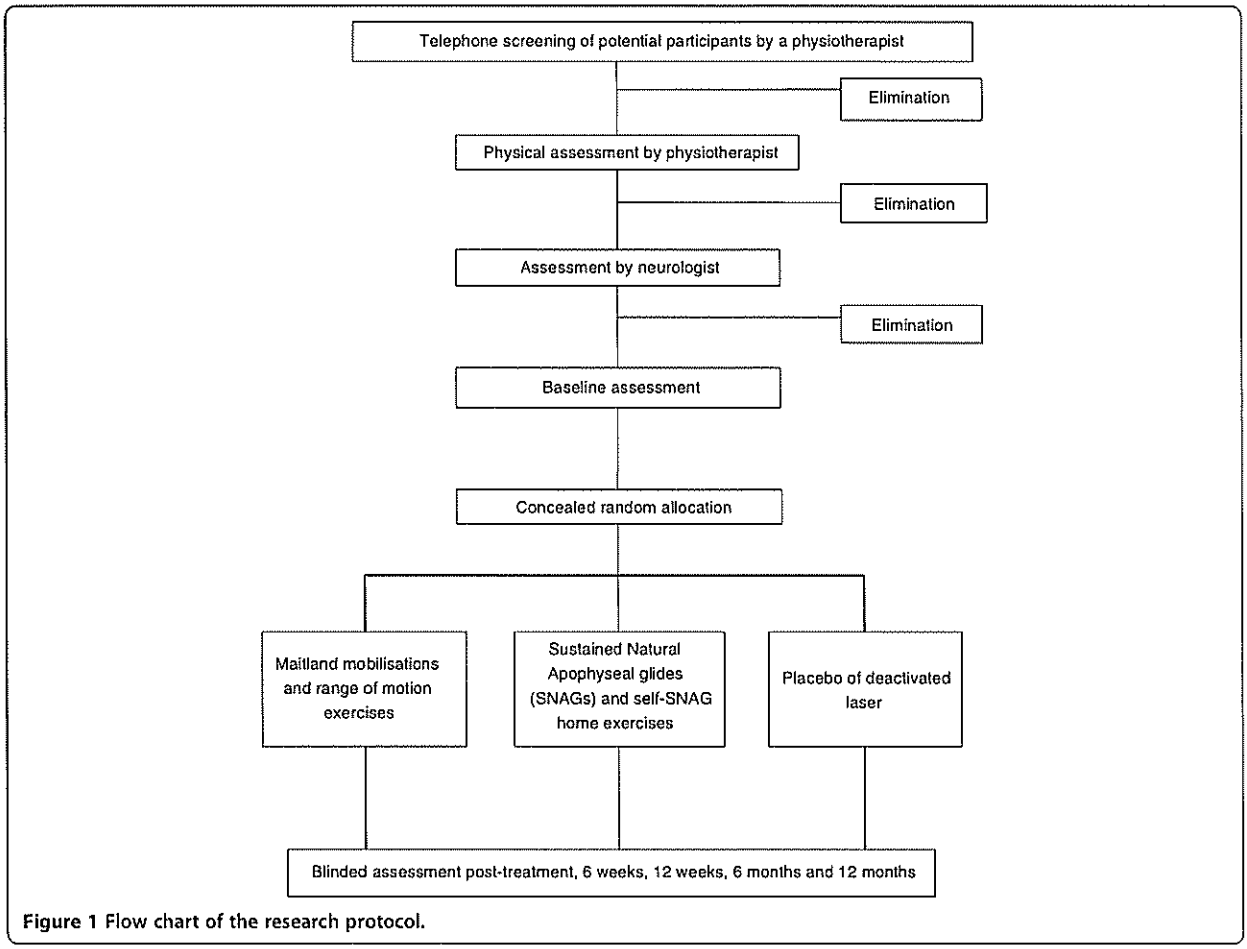

Figure 1 Flow chart of the research protocol.

will be conducted by a physiotherapist. Secondly, if potential participants are still thought to have cervicogenic dizziness after the phone discussion they will then be examined physically by the physiotherapist. Thirdly, if not excluded by the physiotherapist at that stage they will have a clinical examination by a neurologist including peripheral vestibular function testing.

Phone screening

During the phone screening a history will be taken by the physiotherapist to establish that the person does have dizziness described as imbalance or unsteadiness. If the person has any other types of dizziness such as vertigo, migrainous vertigo, pre-syncope, signs and symptoms of vertebral artery ischaemia (dysarthria, drop attacks, facial paraesthesia, syncope), orthostatic hypotension or psychogenic dizziness [33] they will be excluded. If the dizziness is described as imbalance or unsteadiness it must be established that the imbalance is not due to another cause including musculoskeletal problems, neuromuscular problems, and conditions affecting the brain such as Parkinson's disease, stroke, cerebellar ataxia, multiple sclerosis, Vitamin B12 deficiency or alcoholism. Then it must be established that there is a related history of neck pain and or/stiffness. The unsteadiness or poor balance must also be exacerbated by cervical spine movements or positions to be considered to be due to cervicogenic dizziness.

Physical assessment by a physiotherapist

Those who have passed through the phone screening and are thought to have cervicogenic dizziness then undergo a physical assessment by a physiotherapist. This physical examination includes:

- The Dix-Hallpike manoeuvre performed to determine whether the person has dysfunction of the semi-circular canals [34]. In this test, the participant sits on the examination table while the clinician rotates the participant's head to 45 degrees then quickly lays the participant straight back so that their head is extended below the horizontal. The production of nystagmus indicates benign paroxysmal positional vertigo.

Table 1 Inclusion and exclusion criteria

Inclusion criteria:	
-	has dizziness described as imbalance related to neck movements or positions and a stiff and/or painful neck
-	has had the symptoms present for greater than 3 months
-	18-90 years old
Exclusion criteria:	
Known conditions that would put them at risk of injury:	
-	inflammatory joint disease
-	spinal cord pathology
-	cervical spine infection
-	bony disease or marked osteoporosis
-	marked cervical spine disc protrusion
-	cervical spine cancer
-	acute nerve root symptoms (severe pain, weakness, pins and needles or numbness in the arm or hand for less than 6 weeks)
-	recent fracture/dislocation of the neck (in the last 3 months)
-	previous surgery to the upper cervical spine
People will also be excluded if they have the following:	
-	other types or causes of dizziness, such as vertigo, light headedness, psychogenic dizziness, vertebrobasilar insufficiency
-	other causes of poor balance
-	migraines
-	physiotherapy or similar treatment to the neck in the previous month
-	current pregnancy
-	compensable cases
-	inability to speak or read English

- Blood pressure measured in sitting and immediately after rising to standing with a digital sphygmomanometer. A normal blood pressure response to positional change will indicate that neurocardiogenic syncope is an unlikely cause of the dizziness [33]. A drop in systolic blood pressure of > 30mm Hg or a drop of 10mm Hg in diastolic blood pressure is indicative of orthostatic hypotension [33].
- Smooth visual pursuit movements assessed by the ability to track a slowly moving object. The examiner looks for asymmetry of eye movement which may indicate a cerebellar lesion [33].
- The vestibulo-ocular reflex with the participant sitting maintaining a gaze on a stationary target and performing small oscillations of the head side-to-side and up-and-down. Abnormal responses such as an inability to maintain the gaze for 60 seconds due to dizziness, blurry vision or double vision may indicate peripheral or central nervous system dysfunction [33].
- Cervical range of motion assessment to determine whether the participant has a restriction of movement which may indicate cervical spine dysfunction consistent with cervicogenic dizziness [5]. The participant is asked to move their neck into flexion, extension, left rotation, right rotation, left lateral flexion and right lateral flexion and report any symptoms such as dizziness or pain.
- Palpation of the upper cervical spine (occiput to C3) performed to identify any stiff and/or painful joints which may indicate dysfunction in the upper cervical spine [5].
- Decreased balance has been reported in people with cervicogenic dizziness [6]. To assess balance, participants will be asked to hold tandem stance for 30 seconds. Tandem stance is a clinical measure of standing balance considered to assess postural steadiness in a heel-to-toe position [34].

Examination by a neurologist

If the participant is not excluded after this preliminary screening, they are further assessed by a neurologist to exclude central nervous system, vestibular and other non-cervical causes of the dizziness. This examination will include tests for vestibulo-spinal function, the vestibulo-ocular system, disequilibrium such as gait and balance testing, a repeat of the Dix-Hallpike manoeuvre and peripheral vestibular function testing.

Randomisation

Participants who were not excluded during the screening process will be randomly allocated to one of three intervention groups: placebo, Mulligan SNAGS and Maitland mobilisations. An independent statistician will produce a computer generated randomisation sequence which will be placed in sequentially numbered opaque sealed envelopes. The randomisation sequence will contain equal numbers of participants in each group.

Interventions

A physiotherapist with post graduate qualifications and a minimum of 20 years experience in the field of manual therapy will perform all the interventions during the study to all the participants.

Placebo

One group of participants will have a placebo intervention consisting of infrared therapy laser which has been deactivated by the manufacturer. A medical laser is commonly used by physiotherapists to treat musculoskeletal symptoms [35]. To the participant, the placebo laser

device (a Therapower 40mW laser, serial No 020601, Meyer Medical Electronics, Mordialloc, Australia) will appear to operate normally with a light flashing and a beeping sound, but it will not produce any effective emission. The deactivated laser, which has been shown to have a very strong placebo effect [4,35], will be applied for two minutes to each of three sites on the neck, with the pen at a distance of 0.5-1 cm from the skin [35].

Mulligan SNAGs

Another group of participants will receive SNAGs as described by Mulligan [36]. The participant, in the sitting position, is asked to move their head in the direction that particularly produces their symptoms. As the participant moves their head, the physiotherapist gently glides the C1 or C2 vertebra anteriorly and sustains the glide through the movement. During the application of the glide, the participant should stay symptom-free and is instructed to stop moving if any dizziness is produced. This movement is repeated six times at the first treatment session as recommended by Mulligan. At the subsequent treatment sessions provided no dizziness is experienced the SNAG is performed ten times and gentle over pressure can be applied. A second SNAG in another implicated direction of movement may be added to treatment. After the second treatment the participant will be advised to do a self-SNAG (six repetitions) as a home exercise once daily. Written and pictorial instructions for the home exercise will be provided. A second home treatment self-SNAG may be added for another implicated movement direction after the third treatment. The participant will be asked to perform the home exercises once daily for 12 months.

Maitland mobilisations

The physiotherapist will palpate the neck to find the three most dysfunctional joints and then perform passive joint mobilisations to those joints (as described by Maitland et al.) [32]. A passive joint mobilisation is where the therapist uses their thumbs to rhythmically apply pressure to a vertebra usually in a posterior to anterior direction. It is usually applied three times for 30 seconds to dysfunctional joints or determined by the clinical judgement of the physiotherapist [32]. After the second treatment the participant will be advised to perform range of motion exercises into flexion, extension, right rotation, left rotation, left lateral flexion and right lateral flexion, three times in each direction, once a day for 12 months. Written and pictorial instructions for the exercises will be provided.

Outcome measures

Socio-demographic data will be collected at baseline including the participant's age, gender, and time since commencement of dizziness. The primary and secondary outcomes will be measured at baseline, after the last treatment, at six weeks, 12 weeks, six months (questionnaires only), and one year after treatment is completed. All follow-up assessments will be conducted by researchers blinded to the participants' group allocation. The researchers conducting the data entry process will be blinded to group allocation.

Primary outcome

Severity of dizziness (an average level over the previous week) will be measured with a 100 mm horizontal visual analogue scale (VAS). The VAS has been used successfully to measure dizziness in other studies [4,37-39].

Secondary outcomes

1) Frequency of dizziness will be measured on a six-point rating scale (0 = no dizziness, 1 = dizziness less than once per month, 2 = 1–4 episodes of dizziness per month, 3 = 1–4 episodes of dizziness per week, 4 = dizziness once daily, 5 = dizziness more than once a day or constant). This scoring method has been used by several researchers [4,8,37,38] to measure frequency of dizziness.
2) Disability caused by dizziness will be measured with the Dizziness Handicap Inventory (DHI). This is a health status measure specifically designed to assess dizziness. The DHI assesses the quality of life using three subscales evaluating the impact of dizziness on the functional, emotional and physical aspects of everyday life [40]. The highest possible score is 100, indicating maximum self-perceived handicap. The DHI has been shown to be a highly reliable and responsive tool [40-42].
3) Severity of neck pain (an average level over the previous week) will be assessed with a 100 mm VAS. There is much evidence supporting the high validity of the VAS for measuring pain intensity [43-47].
4) Global perceived effect will be used to assess satisfaction with treatment and measured by self-assessment on a six-point scale (0 = no benefit, 1 = minimal benefit, 2 = some benefit, 3 = a lot of benefit, 4 = great benefit, 5 = maximal benefit) as used in other studies [4,48,49].
5) Posturography will be used to identify and quantify disturbances in balance. Body sway will be measured with a Chattecx Balance Dynamic System (Serial No 1001, Chattecx Corporation, the Chattanooga Group, Tennessee). Recordings will be performed during the following tasks.

- standing erect with the neck in the neutral position with eyes open
- standing erect with the neck in the neutral position with eyes closed
- standing erect with the neck extended
- standing erect with the neck in left rotation
- standing erect with the neck in right rotation
- standing on a moving platform.

Posturography has been used in many studies to assess people with dizziness and has been found to have good correlations with the participant's symptoms [4,6,8,37].

6) A Cervical Range of Motion (CROM) goniometer (Performance Attainment Associates, 3550 Lahore Rd, St Paul, MN), which has been shown to be a reliable tool with good validity [50], will be used to measure cervical spine movements. Active flexion, extension, left and right rotation, and left and right lateral flexion will be measured. Each movement will be measured three times and the average taken.
7) Neck repositioning sense will be assessed with the CROM device. This task tests the participant's ability to accurately reposition their head and neck. The participant is first seated with their head in a neutral position. They are then asked to close their eyes and to move their head into rotation. At mid-range of rotation, the participant will be asked to stop, hold their head steady and think about their position. This position is referred to as the 'target position'. After 5 seconds, the participant returns to the starting position and then attempts to find the target position again at which point a reading is taken. The number of degrees difference between the target position and the attempt to find it are calculated. This is performed three times for both right and left rotation and the average taken for each direction of rotation movement.

Information about adverse events will be collected by the treating physiotherapist at each treatment session and by the research assistant at each follow-up measurement session using open-ended questions as per normal clinical practice.

Participants will be given a diary and asked to log medication use, visits to a medical practitioner, visits to other health professionals, time off work, changes in social engagements and adherence to home exercises.

Data analysis

Sample size calculation

The sample size required is based on an analysis using independent t-tests to test for differences between pairs of treatment groups, with alpha set at 0.05. Three comparisons will be made: the SNAG group compared to the placebo group, the Maitland mobilisation group compared to the placebo group and the SNAG group compared to the Maitland mobilisation group. Sample size calculations were based on a difference between the two groups that would be clinically significant for the main outcome measures, supported by the results of previous research where applicable data existed, and clinical expectations for those factors for which no previous data existed. This was estimated by biostatisticians from the Centre for Clinical Epidemiology and Biostatistics, The University of Newcastle, using other studies with the DHI and VAS as outcome measures [41,49,51,52]. The DHI was the primary outcome measure used for sample size calculations, as it is a widely reported measure of self-perceived disability and effect of dizziness on function. It has been shown to have strong validity and short-term test-retest reliability and good internal consistency [53,54]. Visual analogue scales have also been used in many studies to measure pain and the main complaint [49] and been shown to have high reliability and validity, and a calculation of sample size was also based on VAS data.

Assuming that the standard deviation of DHI scores is 15, then 30 participants per group will give the study 80% power to detect a difference of 11 units between groups for each comparison. Thirty participants per group are also required based on a 0–10 VAS scale (e.g. for dizziness), with a standard deviation of 2.4 and a clinically significant difference of 2 units with a power of 80% and a 5% confidence level.

Statistical methods

Biostatisticians from the University of Newcastle will guide and assist with the statistical analyses. Baseline characteristics will be summarized per group using the number of observations, mean, standard deviation, median, minimum and maximum for continuous measures and number of observations and frequency for categorical measures. Primary and secondary outcome measures are either continuous or ordinal in nature and will be analysed using generalized linear mixed models. As an example, for the primary outcome measure of the DHI, the outcome variable will be the DHI and the predictors will be time, treatment group and an interaction term for time by treatment group. The p-value for the interaction term will indicate whether there is a statistically significant difference in change in the DHI over time between the groups. We will use the 'gate keeper' approach to take account of the multiple testing and restrict the overall type I error rate to 5%. This means that we will test the SNAG intervention against the placebo first, then the Maitland mobilisation against the placebo and if those results are statistically significant at the 5% level we will then test the SNAG against the Maitland intervention.

The primary and secondary outcome measures will also be compared between treatment groups at each time point using independent t-tests.

Economic evaluation

The type of economic evaluation by a health economist will depend on the results. It is possible that there will be a difference in efficacy, so a cost effectiveness or cost-utility analysis will be appropriate. If one intervention were more effective and less expensive, an incremental cost effectiveness (utility) ratio (ICER) would not need to be calculated as it would be clear that the more effective intervention is preferred. If one intervention were more effective and more expensive, then an ICER would need to be calculated. If the results show that one intervention is equally effective to the alternative(s), then a cost-minimisation analysis is appropriate. In this case, there is no difference in effectiveness so the economic analysis would be a comparison of costs only; if one intervention is cheaper, it is the preferred alternative.

Controlling bias

To minimise bias randomisation, concealed allocation, specific inclusion and exclusion criteria, blinded outcome assessment, patient blinding, blind data analysis, and intention to treat analysis have been used. It was not possible to blind the physiotherapist performing the interventions.

Discussion

This paper outlines the rationale and design for a RCT that compares the effectiveness and cost effectiveness of:

a) SNAGs to a placebo intervention
b) Maitland passive joint mobilisations to a placebo intervention
c) SNAGs to Maitland passive joint mobilisations

in reducing symptoms of cervicogenic dizziness and associated pain over 12 months. The value of this study will be to determine which of two common manual therapy treatments is most effective for this problem and whether manual therapy is effective in the longer term (up to one year). The study will contribute to evidence-based manual therapy leading to improved clinical decision making in this field of clinical practice.

Competing interests

This study is partly funded by the Mulligan Concept Teachers Association Award. The authors declare that they have no competing interests.

Authors' contributions

SR, DR, RC and MK were responsible for the design of the study. All authors read and approved the final manuscript.

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Chapter 4 Comparison of Mulligan sustained natural apophyseal glides and Maitland mobilizations for treatment of cervicogenic dizziness: a randomized controlled trial

The work presented in this chapter has been published as:

Reid SA, Rivett RA, Katekar MG, Callister R (2014) Comparison of Mulligan sustained natural apophyseal glides and Maitland mobilizations for treatment of cervicogenic dizziness: a randomized controlled trial *Physical Therapy 94:466-476*.

Overview

This paper reports the results from the randomized controlled trial for self-report measures (dizziness intensity, dizziness frequency, disability measured with the Dizziness Handicap Inventory, cervical pain intensity and global perceived benefit of the interventions) at baseline, immediately after the interventions and at 12-weeks post intervention. It was decided to separate these self-report outcomes from the physical outcome measures of cervical range of motion, head repositioning accuracy and balance which are reported in the next chapter (Chapter Five), due to the fact that there were too many outcomes to report in one publication.

Research Report

Comparison of Mulligan Sustained Natural Apophyseal Glides and Maitland Mobilizations for Treatment of Cervicogenic Dizziness: A Randomized Controlled Trial

Susan A. Reid, Darren A. Rivett, Michael G. Katekar, Robin Callister

S.A. Reid, BAppSc(Phty), Grad DipManipTher, MMedSc(Phty), BPharm, School of Physiotherapy, Australian Catholic University, PO Box 968, North Sydney, New South Wales, 2059, Australia. Address all correspondence to Ms Reid at: sue.reid@acu.edu.au.

D.A. Rivett, BAppSc(Phty), MAppSc(ManipPhty), PhD, School of Health Sciences, The University of Newcastle.

M.G. Katekar, MBBS, FRACP, Faculty of Health, The University of Newcastle.

R. Callister, BPharm, MSc, PhD, School of Biomedical Sciences and Pharmacy, The University of Newcastle.

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Background. There is short-term evidence for treatment of cervicogenic dizziness with Mulligan sustained natural apophyseal glides (SNAGs) but no evidence for treatment with Maitland mobilizations.

Objective. The purpose of this study was to compare the effectiveness of SNAGs and Maitland mobilizations for cervicogenic dizziness.

Design. A double-blind, parallel-arm randomized controlled trial was conducted.

Setting. The study was conducted at a university in Newcastle, Australia.

Participants. Eighty-six people with cervicogenic dizziness were the study participants.

Interventions. Included participants were randomly allocated to receive 1 of 3 interventions: Mulligan SNAGs (including self-administered SNAGs), Maitland mobilizations plus range-of-motion exercises, or placebo.

Measurements. The primary outcome measure was intensity of dizziness. Other outcome measures were: frequency of dizziness, the Dizziness Handicap Inventory (DHI), intensity of pain, and global perceived effect (GPE).

Results. Both manual therapy groups had reduced dizziness intensity and frequency posttreatment and at 12 weeks compared with baseline. There was no change in the placebo group. Both manual therapy groups had less dizziness intensity posttreatment (SNAGs: mean difference=−20.7, 95% confidence interval [95% CI]=−33.6, −7.7; mobilizations: mean difference=−15.2, 95% CI=−27.9, −2.4) and at 12 weeks (SNAGs: mean difference=−18.4, 95% CI=−31.3, −5.4; mobilizations: mean difference=−14.4, 95% CI=−27.4, −1.5) compared with the placebo group. Compared with the placebo group, both the SNAG and Maitland mobilization groups had less frequency of dizziness at 12 weeks. There were no differences between the 2 manual therapy interventions for these dizziness measures. For DHI and pain, all 3 groups improved posttreatment and at 12 weeks. Both manual therapy groups reported a higher GPE compared with the placebo group. There were no treatment-related adverse effects lasting longer than 24 hours.

Limitations. The therapist performing the interventions was not blind to group allocation.

Conclusions. Both SNAGs and Maitland mobilizations provide comparable immediate and sustained (12 weeks) reductions in intensity and frequency of chronic cervicogenic dizziness.

The cervical spine should be considered a possible cause of dizziness when dizziness is described as imbalance, occurs with dysfunction in the cervical spine (pain or stiffness, or both), and is aggravated by movements or positions of the neck.[1-9] Mulligan sustained natural apophyseal glides (SNAGs) have been shown to have an immediate and sustained (for 12 weeks) effect in reducing dizziness, neck pain, and disability caused by cervical spine dysfunction.[2] Maitland mobilizations are a commonly used manual therapy technique for management of cervical pain[10-13]; however, there is no published evidence for their use in treating people with dizziness.

Cervicogenic dizziness is often related to upper cervical degeneration or a neck injury, such as whiplash.[5,14] It is thought to result from a perturbation in sensory information from the upper cervical spine.[5,8,15-18] Equilibrium and balance are maintained by an integration of signals from the vestibular system, the visual system, and proprioceptors in the neck, trunk, and lower limbs.[18-21] Normally, balance is controlled subconsciously; however, when a mismatch of afferent input from these systems occurs, a sensation of disequilibrium or dizziness is experienced.[22,23]

Poor balance and dizziness are common in the community, often with extremely disabling consequences.[24,25] The 2008 English Longitudinal Study of Ageing (ELSA), which assessed 2,925 participants aged over 65 years of age, demonstrated that 21.5% (n=619) of the participants had impaired balance and that 11.1% (n=375) experienced dizziness.[26] These conditions often lead to physical problems such as falls, as well as social, emotional, and financial problems.[24,27,28] The incidence of cervicogenic dizziness has been reported to be 7.5% of all dizziness,[29] with many patients having more than one reason for their dizziness.[29,30]

Although many people are affected by cervicogenic dizziness, a large proportion are not offered treatment. To date, the management of this disabling condition has not been widely studied, but there is a slowly growing body of evidence to support its treatment with manual therapy.[2,4,31-36] It is hypothesized that manual therapy applied to the upper cervical spine increases stimulation of proprioceptors in both joints and muscles of this area and normalizes afferent information.[2,37] Clinically, the treatment of cervicogenic dizziness is an emerging area of physical therapist practice.

Although SNAGs, as described by Mulligan,[38] have been shown to be an effective treatment for cervicogenic dizziness in the medium term (12 weeks),[2] the addition of self-administered SNAGs as a home exercise, which reflects clinical practice, has not been studied in treating cervicogenic dizziness. A self-administered SNAG targets cervical spine dysfunction by the patient performing an accessory glide on a vertebra while simultaneously undertaking the dysfunctional spinal active movement. Hall et al[39] provided evidence for the efficacy of the C1-C2 self-administered SNAG technique in the management of cervicogenic headache.

Passive joint mobilization has been described by Maitland as a manual therapy technique to treat people with cervical pain[40] and constitutes mainstream physical therapist practice, with 99.8% of physical therapists in one study using this approach.[11] A systematic review of manual therapy and exercise for neck pain showed that, of 17 randomized controlled trials, 15 used some form of joint mobilization.[12] Some manual therapists have reported anecdotally that this technique also can be used to treat people with cervicogenic dizziness, but to date there is no high-quality evidence for this claim.

The Bottom Line

What do we already know about this topic?

Cervicogenic dizziness is a condition characterized by episodes of potentially disabling dizziness arising from dysfunction of the cervical spine. Mulligan sustained natural apophyseal glides applied to the cervical spine have been shown to help alleviate this dizziness in the short term.

What new information does this study offer?

This study shows that both Maitland mobilizations and Mulligan sustained natural apophyseal glides are beneficial in reducing the intensity of dizziness, dizziness frequency, and disability in people with chronic cervicogenic dizziness, and the effects of these interventions are maintained for 12 weeks after treatment.

If you're a patient, what might these findings mean for you?

This study provides evidence of successful treatment of cervicogenic dizziness with 2 to 6 sessions of physical therapist intervention and some simple home exercises.

The aim of the present study was to determine and compare the effectiveness of Mulligan SNAGs (including self-administered SNAGs) and Maitland mobilizations (plus range-of-motion exercises) on chronic cervicogenic dizziness symptoms immediately and at 12 weeks after treatment. Adverse effects and global perceived effect (GPE) also were assessed.

Method

Design Overview

This study was a 3-arm, double-blind, randomized controlled trial.[11] Participants with cervicogenic dizziness were randomly allocated to 1 of 3 groups: (1) a group that received Mulligan SNAGs (including self-administered SNAGs), (2) a group that received Maitland mobilizations plus range-of-motion exercises, or (3) a group that received a placebo intervention. Participants received 2 to 6 therapist-delivered treatments over 6 weeks at the discretion of the treating therapist, who used clinical judgment to determine the specific number of treatments based on the participant's response and consistent with previous research that used Mulligan SNAGs or Maitland mobilization to treat people with cervicogenic dizziness or neck pain.[2,12,13] An Australia-licensed physical therapist with formal postgraduate training in both the Maitland and Mulligan approaches and more than 30 years of clinical experience using both manual therapy approaches performed all of the interventions.

Setting and Participants

Over a period of 20 months, participants with dizziness were recruited via media releases, advertisements in local newspapers, and letters to general practitioners and neurologists in the Hunter region of New South Wales, Australia. A 3-step process was followed to identify people with cervicogenic dizziness and exclude those who did not have this condition. An initial telephone screening was conducted by a physical therapist asking about the type of dizziness and checking inclusion and exclusion criteria. To be included in the study, participants had to have dizziness described as imbalance (plus a history of neck pain or stiffness, or both) and a history of neck movement or positions provoking the cervicogenic dizziness. They had to be 18 to 90 years of age and have had dizziness symptoms for 3 months or longer. People were excluded if they had other types or causes of dizziness (eg, vertigo, light-headedness, psychogenic dizziness, vertebrobasilar insufficiency, migraines) or other causes of poor balance (eg, stroke, spinal cord pathology, cerebellar ataxia, Parkinson disease). People also were excluded if they had conditions for which manual therapy is contraindicated (eg, inflammatory joint disease, spinal cord pathology, cervical spine infection, marked osteoporosis, cervical spine cancer) or if they were pregnant, receiving workers' compensation payments, or unable to read English.

Potential participants underwent a physical examination by a physical therapist at The University of Newcastle. Palpation and passive accessory mobilizations of the upper cervical spine (occiput to C3) and cervical active range-of-motion measurements were performed to confirm the presence of dysfunction in the cervical spine. Balance also was tested because it has been identified as being impaired in people with cervical spine dysfunction.[4,5,15,42] Testing to exclude other causes of dizziness consisted of smooth visual pursuit movements,[43] the vestibulo-ocular reflex,[43] and blood pressure measurements. The Dix-Hallpike maneuver[13] was performed to identify and eliminate individuals with benign paroxysmal positional vertigo.

Finally, if not previously excluded, the potential participants underwent a clinical examination by an otoneurologist in Newcastle, which consisted of peripheral vestibular function testing to exclude other noncervical causes of dizziness. After these thorough examinations, the identified participants were considered to have a confirmed diagnosis of cervicogenic dizziness. All participants provided written informed consent.

Randomization and Interventions

Participants who met the inclusion criteria were randomly allocated to 1 of 3 intervention groups: (1) a group that received Mulligan SNAGs (including self-administered SNAGs), (2) a group that received Maitland mobilizations plus range-of-motion exercises, or (3) a group that received a placebo intervention. An independent statistician generated a randomization sequence, which was placed in sequentially numbered, opaque, sealed envelopes. Participants were blinded as to whether they received a placebo or active intervention.

One group of participants received SNAGs as described by Mulligan.[44] Each participant, in a seated position, was asked to move his or her head in the direction that produced the dizziness. As the participant moved his or her head, the physical therapist performed a sustained gliding movement to the C1 or C2 vertebra (Fig. 1A). If the provocative direction was flexion or extension, an anterior glide was applied to the C2 spinous process. If rotation produced dizziness, an anterior glide was applied to the C1 transverse process. This movement was repeated 6 times at the first treatment session and had to be symptom-free. At subsequent treatments, gentle overpressure was applied. A second SNAG in another implicated direction was added when clinically justified. After the second treatment, the participant was advised how to self-administer the SNAG using his or her fingers or a strap (6 repetitions) into

the provocative direction as a home exercise once daily (Fig. 1B).

The second group received passive joint mobilizations applied to up to 3 stiff or painful joints in the upper cervical spine based on the clinical judgment of the physical therapist as described by Maitland et al[45] (Fig. 1C). The degree of vigor (grade according to Maitland) and duration of the application were determined by clinical judgment but usually consisted of three 30-second applications at each spinal level treated.[10] After the second treatment, the participant was advised to perform range-of-motion exercises into flexion, extension, rotation, and lateral flexion, 3 times in each direction, once a day.

The third group of participants received a placebo intervention consisting of application of a laser, which had been deactivated by the manufacturer. To the participant, the placebo laser (a Therapower 40-mW laser, Meyer Medical Electronics, Mordialloc, Australia) appeared to operate normally, with a light flashing and a beeping sound, but it did not produce any emission. The deactivated laser was applied for 2 minutes to 3 sites on the neck, with the probe at a distance of 0.5 to 1 cm from the skin. This placebo intervention has been used effectively in previous studies.[2,46]

Outcomes and Follow-up

Demographic data were collected at baseline (Tab. 1). Outcome measurements were obtained at baseline, following the final therapist treatment, and at 12 weeks after the final treatment. All outcome assessments and data entry were performed by a research assistant blinded to group allocation.

The primary outcome measure was intensity of dizziness (averaged over the previous few days), which was measured with a 100-mm visual analog scale (VAS) as in previous studies of cervicogenic dizziness.[2,34,47]

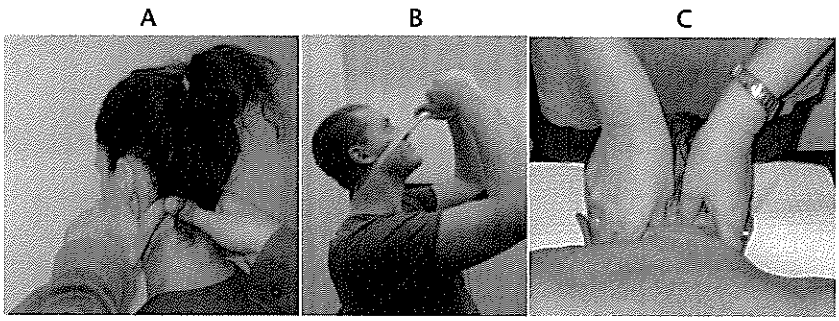

Figure 1.
The manual therapy interventions used in the study: (A) Sustained natural apophyseal glide (SNAG) into left rotation. The physical therapist performs a sustained anterior glide to the left C1 transverse process. The participant turns his or her head to the left as the SNAG is sustained. (B) Self-administered SNAG into extension. The participant uses a strap or his or her fingers to perform a sustained anterior glide to the C2 spinous process while looking up. The glide is maintained until the head returns to the neutral starting position. (C) Maitland central posterior-anterior passive joint mobilization on C2.

Secondary outcome measures were:

1. Frequency of dizziness (0=no dizziness, 1=dizziness less often than once a month, 2=1–4 episodes per month, 3=1–4 episodes per week, 4=dizziness once daily, 5=dizziness more often than once daily or constant dizziness).[2,33,48]

2. Dizziness Handicap Inventory (DHI), a measure of handicap related to dizziness and its impact on daily life.[49] A total score of 0 to 30 indicates mild handicap, of 31 to 60 indicates moderate handicap, and of 61 to 100 indicates severe handicap.[50] It has been suggested that a change in the score of 10% or more is clinically relevant.[17] Also, Tamber et al[51] have suggested that 11 points is the value of the minimal important change (MIC). The DHI was designed for use with patients with vestibular disorders, and its use in studies of cervicogenic dizziness is not well established.

Table 1.
Comparison of Participant Characteristics of the 3 Treatment Groups at Baseline[a]

Characteristic	SNAG Group (n=29)	MM Group (n=29)	Placebo Group (n=28)	p[b]
Sex, female, n (%)	15 (52%)	18 (62%)	10 (36%)	.13
Age (y)	60.0 (10.1)	61.0 (15.7)	65.6 (11.0)	.17
Dizziness duration (mo)	70.3 (61.9)	91.6 (91.0)	91.4 (87.0)	.52
VAS for dizziness	43.3 (21.9)	50.3 (21.2)	47.5 (24.9)	.51
Dizziness frequency	3.1 (1.5)	3.4 (0.9)	3.4 (1.0)	.46
DHI	38.4 (16.3)	44.1 (19.8)	42.8 (16.4)	.44
VAS for pain	41.2 (26.5)	50.9 (22.3)	57.4 (28.1)	.06

[a] Data are mean (SD) unless stated otherwise. SNAG=sustained natural apophyseal glide, MM=Maitland mobilization, VAS=visual analog scale, DHI=Dizziness Handicap Inventory.
[b] Comparison of means among groups (significant at $P<.05$).

3. Intensity of cervical pain, as measured with a 100-mm VAS.[47,52]

4. Global perceived effect, which was used to assess the participant's perceived benefit of the treatment and measured on a rating scale (0=no benefit, 1=minimal benefit, 2=some benefit, 3=a lot of benefit, 4=great benefit, 5=maximal benefit).[2,53,54]

5. Adverse effects, which were identified by asking the participant about any new symptoms after the interventions and if the symptoms persisted for more than 24 hours.

Data Analysis

Sample size calculation. Sample size calculations were based on a difference among the groups that would be clinically significant for the main outcome measures and supported by the results of previous research where applicable data existed and on clinical expectations for those factors for which no previous data existed.[55] The sample size was estimated by biostatisticians from The University of Newcastle using previous studies with VAS (for main complaint) and DHI as outcome measures.[49,54,56-59] Visual analog scales have been used in previous studies to measure dizziness, pain, or the main complaint[2,54,58-60] and have been shown to have high reliability and validity; therefore, a calculation of sample size was based on VAS intensity of main complaint data. It was calculated that a sample size of 30 participants would be required for each group to detect a clinically significant difference of 2 units on a 0-10 VAS between 2 groups, with a power of 80%, a 5% confidence level, and a standard deviation of 2.4.[2,58,60] To allow the study to be adequately powered for secondary outcomes, the DHI also was used for sample size calculations, as it is a widely reported measure of self-perceived disability and effect of dizziness on function. The DHI has been shown to have short-term test-retest reliability and good internal consistency.[61] Assuming that the standard deviation of DHI scores is 15, 30 participants per group would provide 80% power to detect a difference of 11 units between groups for each comparison.[49,56,57]

Statistical methods. Biostatisticians from The University of Newcastle assisted with the statistical analyses. The response variables were found to be consistent with a normal distribution, so parametric statistics were used. Means, standard deviations, and 95% confidence intervals were calculated for all outcome measures. Comparisons of groups at baseline were conducted with one-way analysis of variance (ANOVA). For the main analyses, an intention-to-treat approach using a linear mixed model with repeated-measures ANOVA was used. For missing data, a participant's last observation for each outcome measure was carried forward. Pearson correlation analyses also were performed.

Role of the Funding Source

This study was financially supported by the Mulligan Concept Teachers Association Research Award and The University of Newcastle.

Results

Participants

Six hundred eighty-three people responded to the recruitment strategies between April 2010 and December 2011 (Fig. 2). Most people (n=482; 71%) were excluded because they did not meet the telephone screening inclusion criteria regarding symptoms consistent with cervicogenic dizziness. A further 54 people (8%) were excluded after the physical examinations by the physical therapist, and another 51 people (7%) were excluded after examination by the neurologist, which included vestibular function testing. The most common reasons for being excluded were having rotatory dizziness, central or cardiovascular causes of dizziness, or migraines or not having a related neck problem. Ten individuals (1%) declined to participate. Following screening, 86 people (13%) were identified as having cervicogenic dizziness and entered the study. Twenty-nine participants were allocated to each of the SNAG and Maitland mobilization groups, and 28 participants were allocated to the placebo group. Table 1 presents baseline demographic, dizziness, and pain characteristics. The average age of the participants was 62 years (range=21-85), and 50% of the participants were female. The average time that participants had experienced dizziness before entering the study was 7 years 2 months (range=3 months-30 years). There was a tendency for all measurements (dizziness duration, VAS for dizziness, dizziness frequency, DHI, and VAS for pain) to be lower in the SNAG group at baseline, and the measurements for the VAS for pain approached significance ($P=.06$) (Tab. 1). During the study, 3 participants withdrew due to unrelated medical problems, and 2 dropped out due to moving and were unable to be contacted.

Responses to Interventions

Intensity of dizziness. Analysis of changes in intensity of dizziness over time showed that dizziness intensity was reduced immediately after both manual therapy interventions, and the effects were maintained for 12 weeks (Tab. 2, Fig. 3). There was no reduction in dizziness in the placebo group. Both the SNAG and Maitland mobilization groups had less ($P<.05$) dizziness intensity than the placebo group posttreatment and at the 12-week follow-up (Tab. 3).

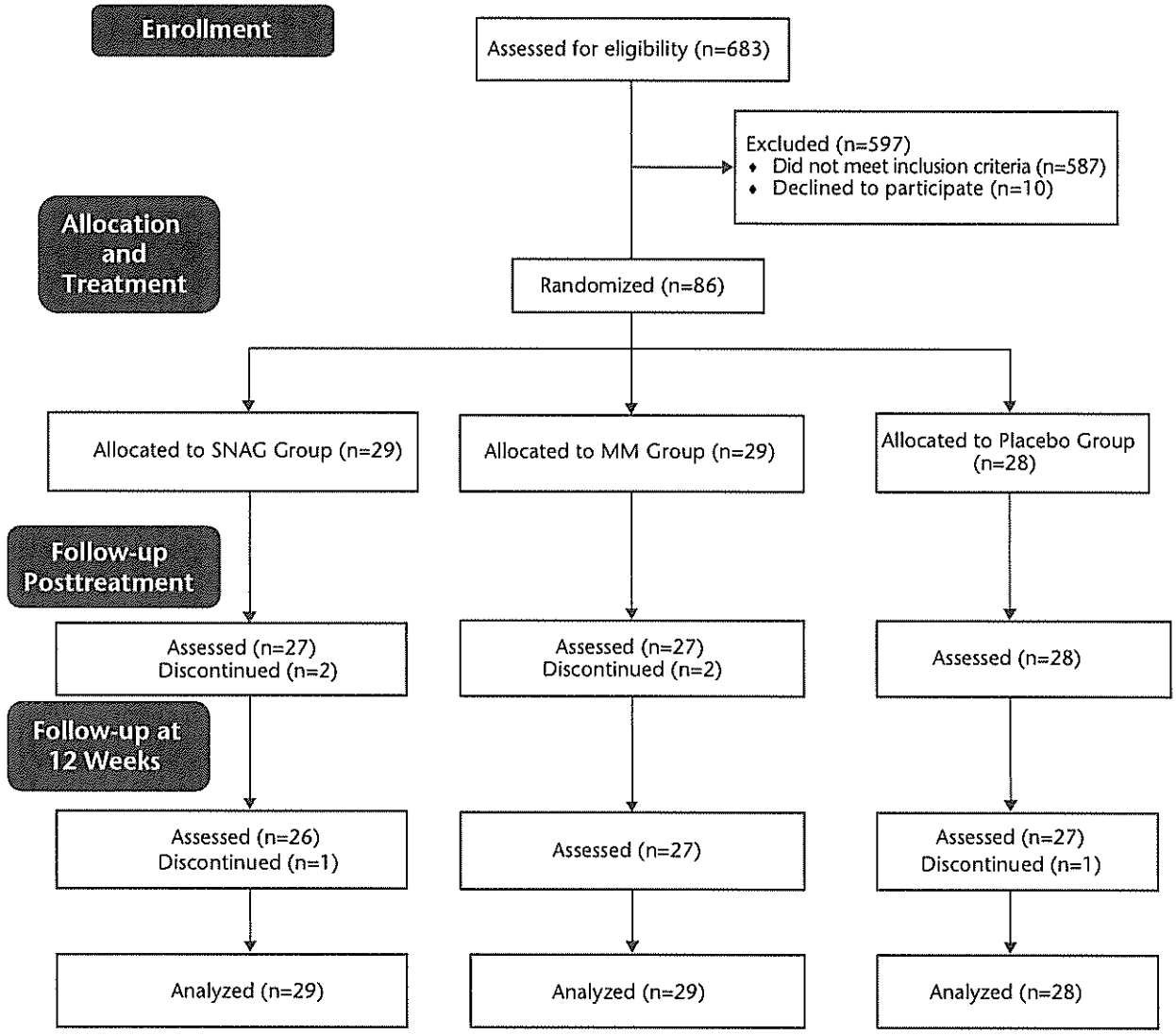

Figure 2.
Flow diagram of participants in the study. An intention-to-treat analysis was performed; therefore, all participants were analyzed at all time points. SNAG=sustained natural apophyseal glide, MM=Maitland mobilizations.

There was no significant difference in dizziness intensity between the SNAG and Maitland mobilization groups after the interventions.

Frequency of dizziness. There were significant reductions in frequency of dizziness after treatment and at 12 weeks in both manual therapy groups compared with baseline but no change in the placebo group (Tab. 2). There were statistically significant lower scores for frequency of dizziness in both the SNAG and Maitland mobilization groups compared with the placebo group at the 12-week follow-up (Tab. 3), but there was no difference between the SNAG and Maitland mobilization groups. The clinical change for the SNAG and Maitland mobilization groups was a reduction in dizziness frequency from dizziness experienced daily or 1 to 4 episodes a week at baseline to dizziness experienced 1 to 4 episodes a month after treatment. For the placebo group, frequency remained at 1 to 4 episodes a week after treatment.

Dizziness Handicap Inventory. At baseline, the DHI scores indicated that dizziness was having a moderate effect on the emotional, social, and physical aspects of the participants' lives in all 3 intervention groups (DHI scores=31–60).[50] There was a significant reduction in DHI scores in all 3 groups posttreatment and at the

Table 2.
Comparison of Changes in Outcome Measures Over Time for Each Treatment Group[a]

Measure	Group	Baseline Mean (SD)	Posttreatment Mean (95% CI)	12 Weeks Mean (95% CI)	Posttreatment vs Baseline		12 Weeks vs Baseline	
					Mean Diff[b] (95% CI)	*P*	Mean Diff[b] (95% CI)	*P*
VAS dizziness	SNAG	43.3 (21.9)	22.3 (12.9, 31.6)	21.7 (12.5, 31.0)	22.5 (13.0, 32.1)	.001*	23.1 (13.7, 32.6)	.001*
	MM	50.3 (21.2)	27.8 (18.6, 36.9)	25.7 (16.4, 34.9)	20.8 (11.5, 30.1	.001*	23.2 (13.7, 32.6)	.001*
	Placebo	47.5 (24.9)	42.9 (34.0, 51.8)	40.1 (31.0, 49.1)	4.2 (−5.1, 13.4)	.38	7.1 (−2.3, 16.4)	.14
Dizziness frequency	SNAG	3.1 (1.5)	2.7 (2.3, 3.1)	2.1 (1.7, 2.5)	0.5 (0.1, 1.0)	.02*	1.0 (0.6, 1.5)	.001*
	MM	3.4 (0.9)	2.9 (2.5, 3.3)	2.3 (1.9, 2.7)	0.5 (0.0, 0.9)	.03*	1.1 (0.7, 1.6)	.001*
	Placebo	3.4 (1.0)	3.0 (2.6, 3.4)	3.0 (2.6, 3.4)	0.4 (0.1, 0.8)	.11	0.4 (−0.1, 0.8)	.11
DHI	SNAG	38.4 (16.3)	32.1 (27.0, 37.2)	30.5 (25.3, 35.7)	8.6 (4.0, 13.2)	.001*	10.2 (5.5, 14.9)	.001*
	MM	44.1 (19.8)	26.7 (21.6, 31.8)	22.9 (17.7, 28.0)	15.2 (10.5, 19.8)	.001*	19.0 (14.3, 23.7)	.001*
	Placebo	42.8 (16.4)	36.9 (31.9, 41.9)	35.2 (30.1, 40.2)	4.6 (0.1, 9.2)	.05*	6.4 (1.8, 11.1)	.006*
VAS pain	SNAG	41.2 (26.5)	28.4 (18.9, 38.0)	31.4 (21.8, 41.1)	15.9 (5.6, 26.2)	.003*	12.7 (2.2, 23.1)	.02*
	MM	50.9 (22.3)	32.7 (23.3, 42.1)	26.2 (16.8, 35.6)	17.9 (7.6, 28.2)	.001*	24.4 (14.1, 34.7)	.001*
	Placebo	57.4 (28.1)	37.8 (28.5, 47.1)	40.5 (31.0, 49.9)	16.7 (6.5, 26.9)	.0001*	13.9 (3.6, 24.3)	.01*

[a] VAS=visual analog scale, SNAG=sustained natural apophyseal glide, MM=Maitland mobilization, DHI=Dizziness Handicap Inventory, 95% CI=95% confidence interval. *$P<.05$.
[b] Mean diff=difference among groups for the least squares mean (adjusted for baseline and missing data).

12-week follow-up compared with baseline (Tab. 2). After treatment and at 12 weeks, the Maitland mobilization group's scores had decreased to indicate mild handicap (DHI scores=1–30),[50] whereas the other 2 groups remained in the moderate range. The reduction in DHI scores reached the MIC of 11 points posttreatment and at 12 weeks for the Maitland mobilization group but not for the other 2 groups. The DHI scores were significantly lower for the Maitland mobilization group compared with the placebo group posttreatment and at 12 weeks and compared with the SNAG group at 12 weeks (Tab. 3). There was no significant difference in DHI scores between the SNAG and placebo groups at any time point (Tab. 3). At baseline, correlations with the DHI scores were as follows: VAS for dizziness intensity, $r=.391$; VAS for frequency of dizziness, $r=.346$; and VAS for pain intensity, $r=.303$.

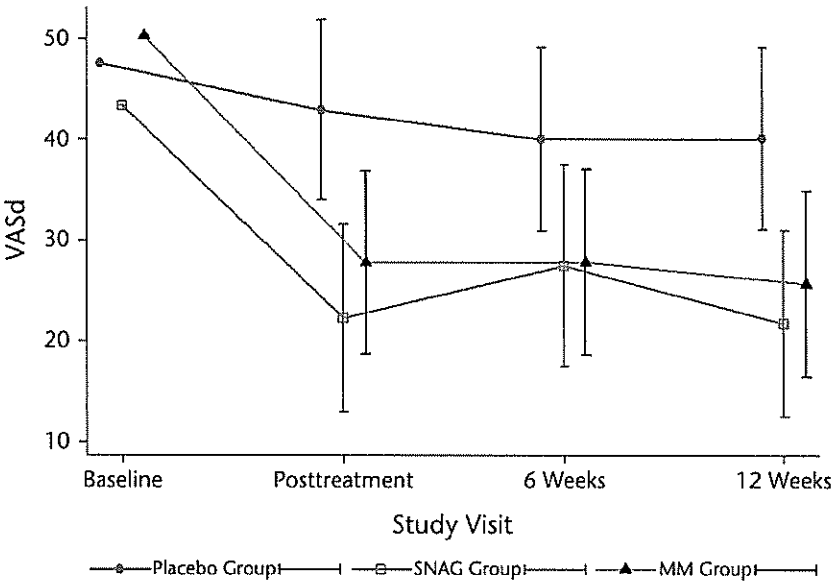

Figure 3.
Changes in mean values for intensity of dizziness (measured on a visual analog scale) over time for each treatment group. The SNAG group received Mulligan sustained natural apophyseal glides, the MM group received Maitland passive joint mobilizations, and the placebo group received deactivated laser. VASd=visual analog scale for intensity of dizziness, 95% CI=95% confidence interval.

Intensity of cervical pain. At baseline, the mean intensity of cervical pain reported by the SNAG and

Table 3.
Differences Among Treatment Groups on Each Outcome Measure Immediately Posttreatment and at 12 Weeks Posttreatment[a]

Measure	Groups	Posttreatment			12 Weeks		
		Mean Diff[b]	95% CI	*P*	Mean Diff[b]	95% CI	*P*
VAS dizziness	SNAG vs Placebo	−20.7	−33.6, −7.7	<.001*	−18.4	−31.3, −5.4	.01*
	MM vs Placebo	−15.2	−27.9, −2.4	.02*	−14.4	−27.4, −1.5	.03*
	MM vs SNAG	5.5	−7.6, 18.6	.41	3.9	−9.2, 17.0	.56
Dizziness frequency	SNAG vs Placebo	−0.4	−0.9, 0.2	.21	−0.9	−1.4, −0.3	<.001*
	MM vs Placebo	−0.1	−0.7, 0.4	.67	−0.7	−1.3, −.0.2	.01*
	MM vs SNAG	0.2	−0.3, 0.8	.41	0.1	0.04, 0.7	.68
DHI	SNAG vs Placebo	−4.8	−12.0, 2.3	.18	−4.7	−11.9, 2.6	.2
	MM vs Placebo	−10.3	−17.4, −3.1	.01*	−12.3	−19.5, −5.1	.01*
	MM vs SNAG	−5.4	−12.7, 1.8	.14	−7.6	−14.9, −0.3	.04*
VAS pain	SNAG vs Placebo	−9.3	−22.8, 4.2	.17	−9.0	−22.7, 4.7	.2
	MM vs Placebo	−5.0	−18.2, 8.1	.45	−14.2	−27.5, −1.0	.04*
	MM vs SNAG	4.3	−9.2, 17.7	.53	−5.2	−18.8, 8.3	.45

[a] VAS=visual analog scale, SNAG=sustained natural apophyseal glide, MM=Maitland mobilization, DHI=Dizziness Handicap Inventory, 95% CI=95% confidence interval. *P<.05.
[b] Mean diff=difference among groups for the least squares mean (adjusted for baseline and missing data).

Maitland mobilization groups was moderate (pain of 30–54 mm on the VAS), whereas the mean severity of pain reported by the placebo group was severe (pain greater than 54 mm on the VAS).[62] There was a significant (P<.05) decrease in pain in all 3 groups after the interventions, and this effect was maintained for 12 weeks (Tab. 2). The Maitland mobilization group had significantly lower pain scores than the placebo group at 12 weeks (Tab. 3). There was a large number of participants (n=10) in the SNAG group with VAS pain scores of less than 20 mm at baseline but only a small number (n=3) in the other 2 groups. There is some thought that participants with VAS pain scores of less than 20 mm should not be included in pain trials, as this low score could be called neck discomfort and not actual pain.[13] When a statistical analysis of changes in pain scores was performed after excluding participants with pain scores of less than 20 mm at baseline, there was a trend for a decrease in pain scores for the SNAG group compared with the placebo group (P=.06) at 12 weeks after the interventions. The clinical change for the manual therapy groups was a reduction in pain intensity from moderate (30–54 mm on the VAS) at baseline to mild (<30 mm on the VAS) posttreatment for the SNAG group and at 12 weeks for the mobilization group (Tab. 2). It remained in the moderate range for the placebo group posttreatment and at 12 weeks (Tab. 2).

GPE. The SNAG and Maitland mobilization treatments were perceived by the participants to be of more benefit than the placebo intervention. The results show that both manual therapy groups had significantly (P<.05) higher GPE ratings compared with the placebo group posttreatment and at 12 weeks. The median GPE score for both the SNAG and Maitland mobilization groups immediately posttreatment and at the 12-week follow-up was 4, indicating "great" benefit. The median score for the placebo group at both time points was 3, indicating "a lot" of benefit.

Adverse effects. Four participants reported mild transient pain in their lower cervical spine or upper arm after SNAGs or self-administered SNAGs. None of the symptoms lasted longer than 24 hours. There were no adverse effects in the Maitland mobilization or placebo groups.

Discussion

This study demonstrated that both SNAGs and Maitland passive joint mobilizations are safe and effective manual therapy interventions for the treatment of cervicogenic dizziness. Both manual therapy treatments reduced the intensity and frequency of dizziness, whereas the placebo intervention had no effect. These reductions in dizziness symptoms were of similar magnitude with both of these manual therapies. The DHI scores and pain intensity ratings also were reduced over time with all of the interventions, although the magnitude of these improvements was greater for Maitland mobilizations. These findings indicate that SNAGs and Maitland mobilizations are effective for the treatment of cervicogenic dizziness,

with more variable effects on any associated handicap or pain.

The effects of the 2 manual therapy treatments on cervicogenic dizziness in this study are consistent with the findings of our previous study,[2] which showed reductions in frequency and intensity of dizziness with treatment using SNAGs manual therapy. Similarly, Karlberg et al[33] found improvements in dizziness after manual therapy, and this effect was maintained for 2 years after treatment.[4] Both Du et al[35] and Fang[36] also reported improvements in dizziness after spinal manipulation and soft tissue therapy. Because these findings show that manual therapy applied to the cervical spine is an effective treatment for cervicogenic dizziness, our study provides indirect evidence that the symptoms can be attributed to cervical structures.

Unlike the changes in dizziness intensity and frequency, which were specific to the intervention groups, all 3 groups had reductions in DHI and pain intensity scores. These findings suggest that the handicap measured by the DHI in this population may not be specific to changes in dizziness symptoms. The DHI was designed for use in people with vestibular pathology and has rarely been used in those with cervicogenic dizziness. The cervicogenic dizziness population tends to be older and have a number of comorbidities, in particular pain, which may influence responses on the DHI, as a number of the items relate to disability and may not be specific to dizziness. The correlations with the DHI at baseline were similar for dizziness intensity, frequency of dizziness, and VAS for pain intensity. This finding suggests that the DHI scores in this population are almost as well correlated with pain ratings as with the dizziness ratings, which may not be surprising given the effects of pain on disability. The reductions in DHI in all 3 groups are consistent with the reductions in pain intensity observed in all 3 groups. In contrast, only the manual therapy interventions resulted in significant improvements in VAS scores for dizziness intensity and frequency of dizziness. Therefore, these dizziness measures are the more appropriate outcomes on which to base conclusions regarding the effects of manual therapy on dizziness symptoms. The DHI was not used as an outcome measure in the studies by Malmström et al,[4] Karlberg et al,[33] Du et al,[35] or Fang,[36] thus precluding any comparison with our results. Further investigation of the DHI in patients with cervicogenic dizziness may be warranted.

For intensity of neck pain, there were no significant differences between the SNAG group and the placebo group at any time point, but there were significant differences for the Maitland mobilization group at 12 weeks. In our previous study, there was a significant difference in pain scores for the SNAG group compared with the placebo group.[2] Karlberg et al[33] and Fang[36] also reported pain reductions after treatment. A potential criticism of the current study is that some participants had very low pain scores (as people were included based on reports of dizziness and either neck pain or stiffness). Ten participants in the SNAG group and 3 participants in the other 2 groups had VAS pain scores of less than 20 mm. It is recognized in pain trials that adequate sensitivity is achieved only if patients experience at least moderate pain (ie, greater than 30 mm on the VAS) before treatment.[62] In the current study, despite randomization, participants in the placebo group tended to have greater pain at baseline compared with the other groups, meaning there was potentially greater scope for improvement in the placebo group. It has been shown that people who had the greatest VAS pain scores at baseline showed the greatest reductions after therapy.[60] Furthermore, the current study was designed to treat only the upper cervical spine. As the average age of the participants was 62 years, they may have had degeneration in the lower cervical spine that remained untreated, resulting in continued pain. This possibility also could explain some of the adverse effects after SNAGs. In clinical practice, the lower cervical spine also may be treated to address pain from lower cervical levels.

To enable the study to better reflect clinical practice, a self-management component was included. The Mulligan concept incorporates self-administered SNAGs for self-management, and evidence for the efficacy of this technique has been demonstrated in the management of cervicogenic headache.[39] Self-administered SNAGs may assist in restoring normal movement by creating desirable movement templates, which are believed to "resculpt" or "retune" the brain with repetition.[63] Interestingly, Jull et al[64] evaluated cervical mobilization and specific exercise for the treatment of patients with cervicogenic headache and found there was a clinically meaningful 10% better response for the participants who received the combined therapy compared with either intervention alone. In a study evaluating the treatment of patients with cervicogenic dizziness, Malmström et al[4] also reported on the use of a home exercise program following the treatment phase.

A major strength of this study was that recruitment was via press release and advertisements in newspapers in the Hunter region, Australia. Hence, the study sample is likely representative of the general population with cervicogenic dizziness in terms of age, sex, intensity of symptoms, and duration of illness, and thus the results of this study are appropriate to translate to people

with this problem in the wider community. Moreover, although the study took place at a university, the study setting was designed to reflect normal physical therapy clinical conditions, further enhancing the generalizability of the findings. The trial design was further strengthened by incorporating several methodological features that minimize bias, including blinded outcome assessment, blinding of participants, intention-to-treat analysis, randomization, and concealed allocation. A further strength of the study design was the use of a convincing placebo intervention, as evidenced by the fact that the placebo group felt this intervention was of "some benefit" and the lack of difference in dropouts between the manual therapy groups and the placebo group.

We acknowledge limitations of the study. The physical therapist performing the treatments was equally trained and experienced in both manual therapy methods but was not blind to group allocation. In an attempt to minimize associated performance bias, the therapist attempted to provide the same amount of attention to all participants. Despite randomization, there was a trend for a difference in pain scores ($P=.06$) at baseline. There was also a tendency for imbalances at baseline among the 3 groups for sex, age, intensity of dizziness, duration of dizziness, and DHI scores. Better allocation balance could be achieved in future studies by stratifying participants before randomization.

It is important to acknowledge that this clinical trial focused only on one aspect of management of cervicogenic dizziness. Many of the participants in the study had experienced dizziness for many years (mean time=7 years), and we recognize that chronic dizziness, pain, and disability are complex problems that clinically might require a wider approach.[63,65,66] Dizziness and cervical pain are very common problems in the community, and the findings of this study have the potential to benefit many people.[15,42] Considering that the participants had experienced dizziness for many years, the fact they could be effectively treated with 2 to 6 sessions indicates that SNAGs and Maitland mobilization are very potent interventions for this condition.

Conclusion

The results of this study provide strong evidence for the effectiveness of 2 common manual therapy treatments for patients with cervicogenic dizziness. There was no difference in effectiveness between the 2 manual therapy interventions, as measured by the changes in intensity and frequency of dizziness. The results provide the first documented evidence for the benefits of Maitland mobilization for cervicogenic dizziness.

All authors provided concept/idea/research design, facilities/equipment, and consultation (including review of manuscript before submission). Ms Reid, Dr Rivett, and Dr Callister provided writing and fund procurement. Ms Reid and Dr Katekar provided data collection and study participants. Ms Reid provided data analysis. Ms Reid and Dr Rivett provided institutional liaisons. This project was conducted with the assistance of Calum Bolton, Andrew Makaroff, and Jane Hake as research assistants.

This study was approved by The University of Newcastle Human Research Ethics Committee (No. H-2009-0377).

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The trial is registered with the Australian New Zealand Clinical Trials Registry (trial registration: ACTRN12611000073909).

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Chapter 5 The effects of cervical spine manual therapy on range of motion, head repositioning and balance in participants with cervicogenic dizziness: a randomized controlled trial

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Overview

This paper reports the results from the randomised controlled trial for the physical outcome measures of cervical range of motion, head repositioning accuracy and balance at baseline, immediately post intervention and at 12-week follow-up.

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ORIGINAL ARTICLE

Effects of Cervical Spine Manual Therapy on Range of Motion, Head Repositioning, and Balance in Participants With Cervicogenic Dizziness: A Randomized Controlled Trial

Susan A. Reid, MMedSc(Phty),[a] Robin Callister, PhD,[b] Michael G. Katekar, MBBS, FRACP,[b] Darren A. Rivett, PhD[b]

From the [a]School of Physiotherapy, Australian Catholic University, North Sydney; and the [b]Faculty of Health and Medicine, The University of Newcastle, Newcastle, Australia.

Abstract

Objective: To evaluate and compare the effects of 2 manual therapy interventions on cervical spine range of motion (ROM), head repositioning accuracy, and balance in patients with chronic cervicogenic dizziness.

Design: Randomized controlled trial with 12-week follow-up using blinded outcome assessment.

Setting: University School of Health Sciences.

Participants: Participants (N=86; mean age ± SD, 62.0±12.7y; 50% women) with chronic cervicogenic dizziness.

Interventions: Participants were randomly assigned to 1 of 3 groups: sustained natural apophyseal glides (SNAGs) with self-SNAG exercises, passive joint mobilization (PJM) with ROM exercises, or a placebo. Participants each received 2 to 6 treatments over 6 weeks.

Main Outcome Measures: Cervical ROM, head repositioning accuracy, and balance.

Results: SNAG therapy resulted in improved ($P \leq .05$) cervical spine ROM in all 6 physiological cervical spine movement directions immediately posttreatment and at 12 weeks. Treatment with PJM resulted in improvement in 1 of the 6 cervical movement directions posttreatment and 1 movement direction at 12 weeks. There was a greater improvement ($P<.01$) after SNAGs than PJM in extension (mean difference, −7.5°; 95% confidence interval [CI], −13° to −2.0°) and right rotation (mean difference, −6.8°; 95% CI, −11.5° to −2.1°) posttreatment. Manual therapy had no effect on balance or head repositioning accuracy.

Conclusions: SNAG treatment improved cervical ROM, and the effects were maintained for 12 weeks after treatment. PJM had very limited impact on cervical ROM. There was no conclusive effect of SNAGs or PJMs on joint repositioning accuracy or balance in people with cervicogenic dizziness.

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Cervicogenic dizziness is described as a sensation of unsteadiness or disequilibrium that occurs together with pain and/or stiffness of the cervical spine and is triggered by neck movements or positions. Dizziness is a common symptom in people with cervical spine dysfunction.[1-5] Management of this condition is challenging because the source of symptoms is difficult to identify. However, there is moderate (level 2) evidence that cervical spine manual therapy is effective in reducing cervicogenic dizziness.[1,5,11] A recent randomized controlled trial[12] by the authors found that 2 common forms of manual therapy, Mulligan sustained natural apophyseal glides (SNAGs) and Maitland low-velocity passive joint mobilization (PJM), reduced both the intensity and frequency of dizziness at 12 weeks compared with a placebo (all $P<.05$).

Previous studies[13-15] have reported that patients with cervical spine dysfunction have reduced cervical range of motion (ROM),

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greater head repositioning errors,[2,13,16-19] and deficits in their balance[3,20-24] compared with healthy controls.[13,15,25] Most of these studies, however, were of patients with whiplash-associated disorder who often report dizziness. In contrast, 1 study[5] of 22 patients (mean age, 37y) with cervicogenic dizziness found that patients had a normal or larger ROM than age- and sex-matched controls.

Although there is evidence that manual therapy reduces self-reported dizziness in patients with cervicogenic dizziness, there is limited evidence for the effect of manual therapy on cervical ROM, balance, and sensorimotor dysfunction in this population.[1,6-9,11,26,27] Cervical ROM has been shown to improve in people with cervicogenic dizziness after treatment with SNAGs, high-velocity thrust manipulation, and using a multimodal approach.[5,7,26,28] Balance has been shown to improve after SNAGs.[1] Additionally, manipulation has been shown to improve head repositioning in this population.[26,27] There have been no randomized controlled trials assessing the effect of PJM on cervicogenic dizziness in spite of PJM's being a commonly used manual therapy technique for cervical dysfunction and pain.[29-31]

The aim of this study was to determine whether Mulligan SNAGs (with self-SNAGs) or Maitland PJMs (with ROM exercises) improves cervical ROM, head repositioning accuracy, and/or balance in people with chronic cervicogenic dizziness, and whether one of these manual therapy approaches was more effective than the other. This current article is a secondary report on the effects of manual therapy on physical measures of participants with cervicogenic dizziness.[12]

Methods

Study design

This study was a double-blind, randomized controlled trial in which participants with cervicogenic dizziness were randomly assigned to receive cervical spine SNAGs, PJM, or a placebo. Each participant was provided with 2 to 6 treatments over 6 weeks, with the number of treatments for each individual determined by the clinical judgment of the treating clinician based on the individual participant's apparent response to treatment. Detailed methods for this study have been previously reported.[12,32] The outcome measures were cervical ROM, head repositioning accuracy, and balance. Measurements were taken at baseline, immediately posttreatment, and at 12 weeks after the intervention. The University of Newcastle Human Research Ethics Committee approved this study (No H-2009-0377), and all participants provided written informed consent.

Participants

Volunteers aged 18 to 90 years with cervicogenic dizziness for 3 months or longer were recruited from April 2010 to December 2011 via media releases and letters to general practitioners and neurologists in the Hunter region of New South Wales, Australia.

List of abbreviations:

CI	confidence interval
CROM	Cervical Range of Motion
NHP	neutral head position
PJM	passive joint mobilization
ROM	range of motion
SNAG	sustained natural apophyseal glide

Those who responded to this call for participants were first screened via a telephone interview to determine whether they had symptoms consistent with cervicogenic dizziness and not symptoms indicating other forms of dizziness such as vertigo. To be included in the study, participants had to report (1) non-rotatory dizziness that was described as imbalance or unsteadiness, and was triggered by neck movements or positions; and (2) a stiff or painful neck, or both. Potential participants who passed this telephone screening then underwent a series of clinical tests with a physical therapist who assessed cervical spine ROM, palpated the cervical muscles, performed passive accessory intervertebral movements to the cervical spine joints, and performed the Dix-Hallpike maneuver (to identify and exclude those with benign paroxysmal positional vertigo). If not excluded, the potential participant was then examined by an otoneurologist and underwent vestibular function testing to diagnose cervicogenic dizziness and exclude other causes of dizziness. This process has been described in more detail previously.[32] Potential participants were also excluded if they were receiving workers' compensation, were pregnant, were unable to read English, or had conditions for which manual therapy was contraindicated, such as inflammatory diseases, spinal cord pathology, and cervical spine cancer.

Randomization

Participants who met the inclusion criteria were randomly allocated to 1 of 3 intervention groups. The randomization sequence was created by an independent statistician and concealed in sequentially numbered envelopes. The randomization sequence contained equal numbers in each group but was otherwise unrestricted.

Blinding

Data collection and data entry were conducted by research assistants who were blinded to treatment allocation. Throughout the study, participants were blinded as to whether they received a placebo or active intervention. It was not possible to blind the physical therapist administering the interventions.

Interventions

All the interventions were performed by an experienced Australian-licensed physical therapist with formal postprofessional training in both the Maitland and Mulligan manual therapy approaches.

Sustained natural apophyseal guides

One group of participants received SNAGs as described by Mulligan.[33] As participants moved their head in the direction that produced dizziness, the physical therapist gently applied a glide to the C1 or C2 vertebra anteriorly and sustained the glide through the movement. This procedure was repeated 6 times at the first treatment session as recommended by Mulligan.[33] During the application of the procedure, the participant should be entirely symptom-free. At the second treatment session, manual therapy was repeated, and the participant was advised how to self-SNAG using a strap placed on the cervical spine, as a home exercise to be performed as 6 repetitions once daily.[33] The participant was asked to perform these exercises for 12 weeks and record compliance in a diary.

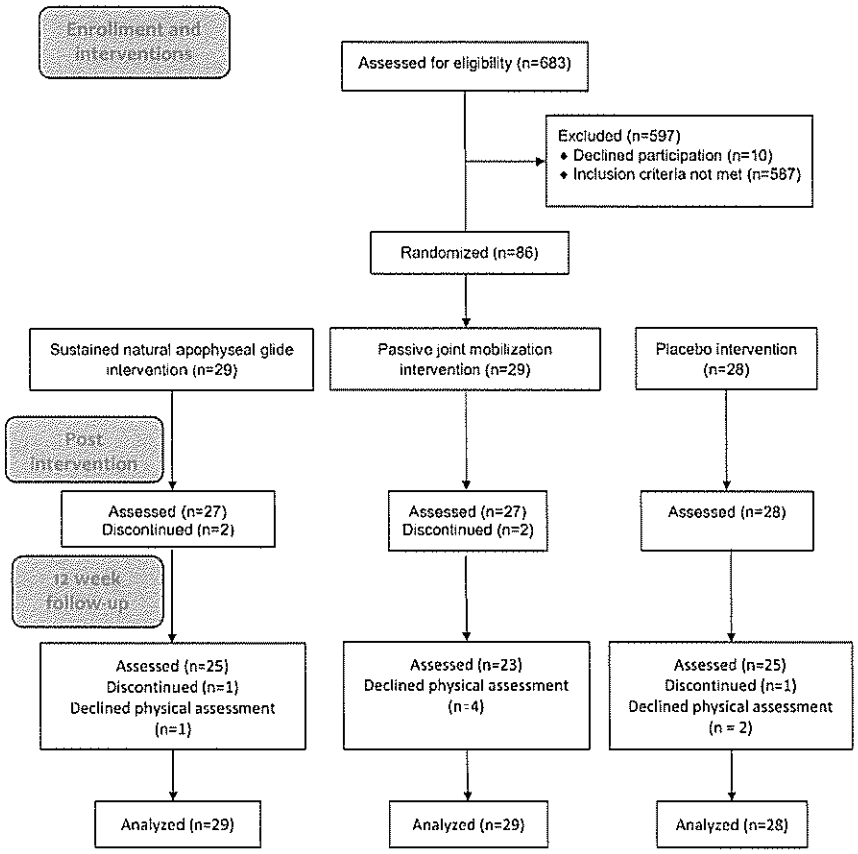


Fig 1 Flow diagram of participants in the study. An intention-to-treat analysis was performed, so all participants were analyzed at all time points.

Passive joint mobilization

The second group received PJM as described by Maitland et al.[34] The mobilization was applied to hypomobile or painful joints, or both, in the upper cervical spine as determined by the treating therapist. After the second treatment, participants were advised to perform ROM exercises into pain-free flexion, extension, left and right rotation, and left and right lateral flexion, 3 times in each direction, once a day. The participant was advised to perform these exercises for 12 months and record the exercises in a diary.

Placebo

The third group of participants received a placebo intervention consisting of a deactivated laser device.[a] The laser probe was positioned at a distance of 0.5 to 1cm from the skin and applied for 2 minutes to each of 3 sites on the neck. This placebo has been used effectively in previous studies.[1,35]

Outcomes and follow-up

To enhance measurement consistency, all research assistants underwent standardized training of the assessment methods.

Outcome measures were cervical ROM, head repositioning accuracy, and balance. Cervical ROM was measured with a Cervical Range of Motion (CROM) instrument,[b] which has been shown to be a reliable and valid tool.[36,37] The maximum range of cervical flexion, extension, left and right rotation, and left and right lateral flexion was measured 3 times and averaged. The SEM with the CROM in participants with neck pain has been reported to be flexion 4.1°, extension 3.0°, left rotation 2.9°, right rotation 3.3°, left lateral flexion 3.9°, and right lateral flexion 2.5°.[38] The minimal detectable change has been reported to be flexion 9.6°, extension 7.0°, left rotation 6.7°, right rotation 7.6°, left lateral flexion 9.1°, and right lateral flexion 5.9° in a population with neck pain.[38]

Head repositioning accuracy was assessed using the CROM device.[19] The participants (blindfolded) started with their head in the neutral head position (NHP) and were asked to actively move to the midpoint of their maximum rotation range (as identified by the research assistant using the CROM device), which was called the "target position." After returning to the NHP, they were then asked to rotate their head to the target position. The difference between the target position and the achieved position was recorded 3 times and averaged. The midpoint position was used rather than the NHP because it was considered a nonlearned position, which may be better for assessing the effectiveness of cervical proprioceptors.[17] In asymptomatic individuals, a 2° to 2.5° error has been reported with this test, while errors of 3° to 4° can indicate a deficit in joint positioning sense.[2,15,39]

Balance was assessed using the Chattecx Balance Dynamic System.[c] The Chattecx posturography system produces a sway index (cm) that reflects the scatter of data about the participant's center of balance and is a measure of postural sway. The sway index was measured during static standing with the head in the neutral position (eyes open); with the head in the neutral position (eyes closed); with the neck extended; with the neck in left and

Table 1 Comparison of the 3 intervention groups at baseline

Characteristic	Total Sample	SNAG (n=29)	PJM (n=29)	Placebo (n=28)	P*
Age (y)	62.0±12.7	60.0±10.1	61.0±15.7	65.6±11.0	.17
Women	43 (50)	15 (52)	18 (62)	10 (36)	.13
VAS dizziness (0-100)	47.05±22.59	43.3±21.9	50.3±21.2	47.5±24.9	.51
Dizziness frequency	3.33±1.17	3.1±1.5	3.4±0.9	3.4±1.0	.46
Dizziness duration (mo)	84.4±80.60	70.3±61.9	91.6±91.0	91.4±87.0	.52
History of trauma	29 (34)	12 (41)	6 (21)	11 (39)	.24
Cervical ROM (deg)					
Extension	45.2±13.0	45.9±14.1	47.1±11.5	42.4±13.4	.37
Flexion	38.3±13.7	36.8±14.6	39.8±14.5	38.2±12.1	.71
Left rotation	50.3±12.8	51.0±12.8	51.8±11.1	48.1±14.7	.53
Right rotation	48.8±13.4	51.6±16.6	50.0±10.3	44.6±11.9	.12
Left lateral flexion	29.6±9.3	30.3±10.8	30.7±8.5	27.7±8.5	.41
Right lateral flexion	26.2±10.4	27.6±12.3	28.6±9.0	22.4±8.9	.05
Head repositioning accuracy (deg)					
Rotation (left)	5.2±3.9	4.7±3.5	5.6±3.9	5.2±4.3	.72
Rotation (right)	4.8±4.6	5.4±4.8	3.8±3.3	5.1±5.6	.35
Balance (sway index; cm)					
Eyes open	0.37±0.22	0.30±0.14	0.44±0.28	0.36±0.20	.05
Eyes closed	0.64±0.37	0.57±0.34	0.73±0.46	0.61±0.28	.23
Extension	1.65±0.69	1.51±0.78	1.75±0.58	1.71±0.69	.37
Left rotation	0.60±0.39	0.58±0.38	0.67±0.47	0.57±0.32	.58
Right rotation	0.47±0.36	0.49±0.48	0.47±0.30	0.44±0.25	.86
Moving base	0.45±0.36	0.45±0.42	0.50±0.37	0.39±0.27	.50

NOTE. Values are mean ± SD, n (%), or as otherwise indicated.
Abbreviation: VAS, visual analog scale.
* Comparison of the means between groups was conducted with 1-way analysis of variance; significance indicated by $P \leq .05$.

right rotation; and on a moving platform (NHP, eyes open). The SEM for the Chattecx system has been reported to be .06cm (95% confidence interval [CI], .16–.40) for static standing (eyes open), .26cm (95% CI, .13–.87) for static standing (eyes closed), and .32cm (95% CI, .36–1.6) for eyes open on a moving platform.[30]

Statistical analysis

Biostatisticians blinded to participant identity performed the statistical analyses. A sample size of 30 participants per group was estimated as necessary to detect a clinically significant difference between the 2 active intervention groups using the primary outcome variable of the study,[12] which was dizziness intensity.[41] The response variables were found to be consistent with a normal distribution, so parametric statistics were used. Mean, SD, and 95% CIs were calculated for all outcome measures. Comparison of groups at baseline was conducted with 1-way analysis of variance. An intention-to-treat approach using a linear mixed model with repeated-measures analysis of variance was used to determine differences between groups after treatment. Because this was an intention-to-treat analysis, all participants are analyzed at all reassessment time points, with missing data imputed via the linear mixed model approach. Significance was accepted at P values $\leq .05$.

Results

Participants

Eighty-six participants were enrolled in the study, with 29 participants allocated to each of the SNAG and PJM groups, and 28 to the placebo group. The flow diagram for participants in the study is presented in figure 1. Table 1 describes participant characteristics, with no differences found among groups for any variable at baseline.

Intervention exposure

The average ± SD number of treatments received by those in the SNAG group was 4.2±.61 sessions; for the PJM group, 4.1±.51; and for the placebo group, 3.9±.26. Diary records regarding compliance with home exercises were obtained from 12 SNAG group and 15 PJM group participants at 12 weeks. There were no adverse effects reported.

Responses to interventions

Cervical spine ROM

Compared with placebo, the SNAG group had greater ROM ($P \leq .05$) in all 6 cervical spine movement directions after treatment and at 12 weeks (table 2). For the PJM group, significant differences from placebo were observed in left rotation after treatment and right rotation at 12 weeks (see table 2). When the 2 manual therapy groups were compared, there were significantly greater ($P \leq .05$) changes in extension and right rotation for the SNAG group compared with the PJM group posttreatment (see table 2), but no differences at 12 weeks. These differences between groups are consistent with the magnitude and time course of change in each group shown in table 3 and figure 2.

Head repositioning accuracy

Neither SNAGs nor PJMs had any meaningful effect on head repositioning accuracy (see table 2), as they were substantially

Table 2 Differences between treatment groups in each outcome measure posttreatment and at 12 weeks

Measures	Posttreatment		12-wk Follow-Up	
	Mean Difference* (95% CI)	*P*	Mean Difference* (95% CI)	*P*
ROM (deg)				
Flexion				
SNAG vs Placebo	6.0 (1.5 to 10.5)	.01	5.8 (1.0 to 10.5)	.02
PJM vs Placebo	4.2 (−0.3 to 8.7)	.07	4.2 (−0.5 to 9.0)	.08
PJM vs SNAG	1.8 (−6.4 to 2.7)	.43	−1.5 (−6.4 to 3.3)	.53
Extension				
SNAG vs Placebo	11.7 (6.1 to 17.2)	<.001	7.4 (1.6 to 13.1)	.01
PJM vs Placebo	4.2 (−1.3 to 9.7)	.14	2.1 (−3.7 to 7.9)	.47
PJM vs SNAG	−7.5 (−13.0 to −2.0)	.01	−5.3 (−11.1 to 0.5)	.07
Left rotation				
SNAG vs Placebo	8.7 (4.2 to 13.1)	<.001	5.6 (0.9 to 10.2)	.02
PJM vs Placebo	6.9 (2.4 to 11.4)	<.001	4.0 (−0.7 to 8.6)	.09
PJM vs SNAG	−1.7 (−6.2 to 2.7)	.44	−1.6 (−6.3 to 3.1)	.50
Right rotation				
SNAG vs Placebo	9.1 (4.4 to 13.9)	<.001	8.2 (3.3 to 13.1)	<.001
PJM vs Placebo	2.4 (−2.3 to 7.1)	.32	7.1 (2.2 to 12.0)	<.001
PJM vs SNAG	−6.8 (−11.5 to −2.1)	<.001	−1.1 (−6.0 to 3.8)	.66
Left lateral flexion				
SNAG vs Placebo	6.4 (2.6 to 10.3)	<.001	3.9 (−0.1 to 7.9)	.05
PJM vs Placebo	3.7 (−0.1 to 7.6)	.06	3.6 (−0.4 to 7.6)	.07
PJM vs SNAG	−2.7 (−6.5 to 1.1)	.17	−0.3 (−4.3 to 3.7)	.89
Right lateral flexion				
SNAG vs Placebo	4.9 (1.0 to 8.8)	.01	5.2 (1.1 to 9.3)	.01
PJM vs Placebo	1.8 (−2.1 to 5.8)	.36	3.9 (−0.2 to 8.1)	.06
PJM vs SNAG	−3.1 (−6.9 to 0.8)	.12	−1.2 (−5.3 to 2.9)	.55
Head repositioning accuracy (deg)				
Left rotation				
SNAG vs Placebo	1.2 (−0.5 to 3.0)	.17	2.4 (0.5 to 4.3)	.01
PJM vs Placebo	2.0 (0.2 to 3.7)	.03	0.1 (−1.8 to 2.0)	.91
PJM vs SNAG	0.7 (−1.0 to 2.5)	.41	−2.3 (−4.2 to −0.4)	.02
Right rotation				
SNAG vs Placebo	1.3 (−0.3 to 2.9)	.10	−1.7 (−3.4 to 0.0)	.05
PJM vs Placebo	1.2 (−0.4 to 2.7)	.14	0.1 (−1.5 to 1.8)	.87
PJM vs SNAG	−0.1 (−1.7 to 1.5)	.88	1.8 (0.1 to 3.5)	.40
Balance (sway index; cm)				
Eyes open				
SNAG vs Placebo	−0.05 (−0.16 to 0.05)	.31	−0.05 (−0.17 to 0.06)	.35
PJM vs Placebo	0.06 (−0.05 to 0.16)	.29	−0.01 (−0.12 to 0.10)	.84
PJM vs SNAG	0.11 (0.00 to 0.22)	.04	0.04 (−0071 to 0.16)	.48
Eyes closed				
SNAG vs Placebo	0.07 (−0.59 to 0.72)	.84	−0.17 (−0.87 to 0.53)	.64
PJM vs Placebo	0.12 (−0.53 to 0.78)	.71	0.03 (−0.74 to 0.67)	.92
PJM vs SNAG	0.06 (−0.61 to 0.72)	.87	0.13 (−0.59 to 0.85)	.72
Extension				
SNAG vs Placebo	0.20 (−0.13 to 0.52)	.24	−0.27 (−0.62 to 0.07)	.12
PJM vs Placebo	0.16 (−0.16 to 0.49)	.32	0.03 (−0.31 to 0.37)	.86
PJM vs SNAG	−0.03 (−0.37 to 0.30)	.84	0.31 (−0.04 to 0.65)	.09
Left rotation				
SNAG vs Placebo	−0.09 (−0.24 to 0.06)	.25	−0.08 (−0.24 to 0.08)	.30
PJM vs Placebo	0.01 (−0.14 to 0.16)	.90	−0.03 (−0.19 to 0.12)	.67
PJM vs SNAG	0.10 (−0.05 to 0.25)	.21	0.05 (−0.11 to 0.21)	.56
Right rotation				
SNAG vs Placebo	−0.06 (−0.17 to 0.05)	.29	0.08 (−0.03 to 0.19)	.17
PJM vs Placebo	−0.04 (−0.15 to 0.06)	.42	0.11 (−0.01 to 0.22)	.06
PJM vs SNAG	0.01 (−0.10 to 0.12)	.81	0.03 (−0.09 to 0.14)	.62

(*continued on next page*)

Table 2 (*continued*)

Measures	Posttreatment		12-wk Follow-Up	
	Mean Difference* (95% CI)	*P*	Mean Difference* (95% CI)	*P*
Moving base				
SNAG vs Placebo	−0.06 (−0.17 to 0.05)	.30	−0.05 (−0.17 to 0.07)	.39
PJM vs Placebo	−0.12 (−0.23 to 0.00)	.04	−0.01 (−0.13 to 0.11)	.85
PJM vs SNAG	−0.06 (−0.17 to 0.06)	.32	0.04 (−0.08 to 0.16)	.51

* Least square mean (adjusted for baseline and missing data) differences from visit to visit.

less than the SEM for the CROM device that was used to measure the head repositioning error.

Balance

There was no conclusive effect for SNAGs or PJMs on balance in people with cervicogenic dizziness (see table 2; table 4). Post hoc analysis revealed that when testing balance with eyes closed, those participants with a history of trauma (n=29) had a mean sway of .72cm, whereas those without trauma (n=57) had a mean sway of .59cm.

Discussion

This study is the first to investigate the effect of PJM on the physical signs of cervical ROM, head repositioning accuracy, and balance in people with cervicogenic dizziness and to compare this manual therapy approach to treatment with SNAGs. Treatment with SNAGs resulted in an improvement in cervical ROM in people with cervicogenic dizziness. Manual therapy had no conclusive effect on joint repositioning accuracy or balance in people with cervicogenic dizziness.

The current study found that there were improvements in cervical spine ROM immediately after treatment with SNAGs and that the improvements were maintained for 12 weeks when the participant was instructed to perform self-SNAG home exercises once daily. The increased ROM after the SNAG intervention of 11° extension and up to 10° rotation constitutes a clinically significant change that would likely make a difference in everyday activities. When the 2 manual therapy groups were compared, the SNAG therapy group had greater ROM improvement than the PJM group in 2 directions posttreatment. This indicates that this Mulligan approach is clinically beneficial in treating reduced cervical spine ROM in patients with cervicogenic dizziness.

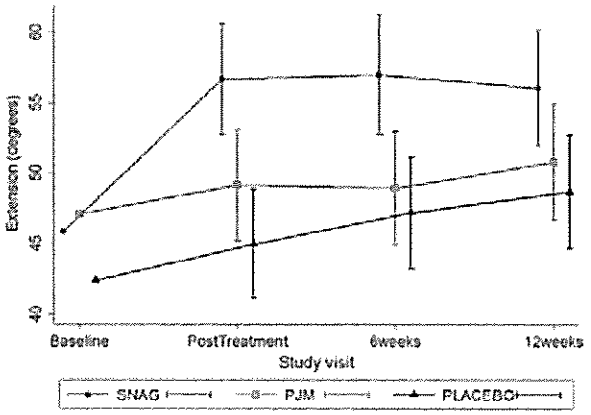
This study found that neither SNAGs nor PJMs had any meaningful effect on joint repositioning accuracy in people with cervicogenic dizziness. A criticism of the study was the use of the CROM to measure head repositioning accuracy. The SEM of the CROM is 2.5° to 4.1°; therefore, it was not sufficiently sensitive to detect the small changes that may occur with head repositioning. For future studies, using a head-mounted laser to measure head repositioning may be more accurate and sensitive.[19,42] Also, relocation to the NHP is potentially more reliable than relocating

Table 3 Comparison of changes in cervical ROM over time for each treatment group

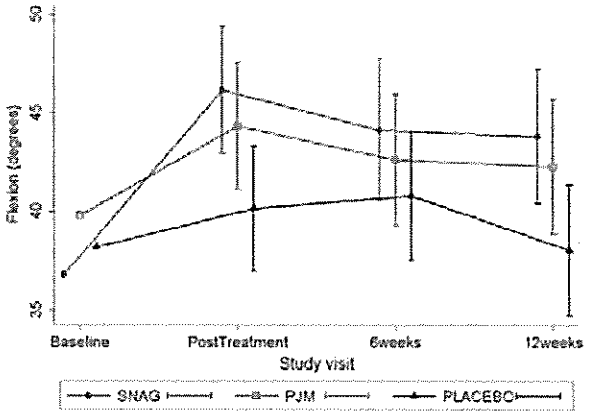
Measures	Group	Baseline	Posttreatment	12wk	Posttreatment vs Baseline		12wk vs Baseline	
					Mean Difference* (95% CI)	*P*	Mean Difference* (95% CI)	*P*
Flexion	SNAG	36.8±14.6	46.2 (43.0–49.4)	43.8 (40.4–47.3)	−8.3 (−11.9 to −4.6)	<.001	−5.9 (−9.8 to −2.1)	.01
	PJM	39.8±14.5	44.4 (41.1–47.6)	42.3 (38.9–45.7)	−4.9 (−8.6 to −1.3)	.01	−2.9 (−6.8–0.9)	.14
	Placebo	38.2±12.1	40.2 (37.0–43.3)	38.1 (34.7–41.4)	−1.7 (−5.3–1.9)	.36	0.4 (−3.4–4.1)	.84
Extension	SNAG	45.9±13.4	56.7 (52.7–60.6)	56.0 (51.9–60.1	−10.6 (−14.5 to −6.6)	<.001	−10.5 (−14.6 to −6.3)	<.001
	PJM	47.1±11.5	49.2 (45.2–53.1)	50.8 (46.7–54.9)	2.7 (−6.6–1.3)	.19	−4.1 (−8.3–0.0)	.05
	Placebo	42.4±13.4	45.0 (41.1–48.8)	48.7 (44.7–52.7)	−0.7 (−4.6–3.2)	.72	−4.4 (−8.5 to −0.4)	.03
Left rotation	SNAG	51.0±12.8	59.2 (56.0–62.4)	55.7 (52.4–59.0)	−8.5 (−11.6 to −5.4)	<.001	−5.1 (−8.4 to −1.8)	.01
	PJM	51.8±11.1	57.4 (54.2–60.6)	54.1 (50.8–57.4)	−8.5 (−11.6 to −5.4)	<.001	−3.2 (−6.5–0.1)	.05
	Placebo	48.1±14.7	50.5 (47.4–53.6)	50.1 (46.9–53.4)	−0.9 (−3.9–2.2)	.59	−0.5 (−3.7–2.7)	.78
Right rotation	SNAG	51.6±16.6	60.3 (56.9–63.6)	56.8 (53.3–60.3)	−10.2 (−13.5 to −6.9)	<.001	−6.8 (−10.3 to −3.3)	<.001
	PJM	50.0±10.3	53.5 (50.2–56.8)	55.7 (52.2–59.2)	−4.1 (−7.4 to −0.7)	.02	−6.4 (−10.0 to −2.9)	<.001
	Placebo	44.6±11.9	51.1 (47.8–54.4)	46.8 (45.2–52.0)	−3.7 (−7.0 to −0.4)	.03	−1.2 (−4.6–2.2)	.49
Left lateral flexion	SNAG	30.3±10.8	35.2 (32.5–37.9)	32.9 (30.0–35.7)	−5.1 (−7.8 to −2.5)	<.001	−2.7 (−5.5–0.1)	.05
	PJM	30.7±8.5	32.5 (29.8–35.2)	32.6 (29.7–35.4)	−2.3 (−5.0–0.3)	.09	−2.4 (−5.2–0.4)	.09
	Placebo	27.7±8.5	28.8 (26.1–31.5)	29.0 (26.2–31.7)	0.6 (−2.0–3.2)	.65	0.5 (−2.2–3.2)	.73
Right lateral flexion	SNAG	27.6±12.3	31.8 (29.0–34.5)	30.6 (27.7–33.5)	−5.3 (−7.9 to −2.4)	<.001	−4.0 (−6.9 to −1.1)	.01
	PJM	50.0±10.3	53.5 (50.2–56.8)	55.7 (52.2–59.2)	−1.9 (−4.6–0.9)	.18	2.6 (−5.5–0.3)	.08
	Placebo	44.6±11.9	51.1 (47.8–54.4)	48.6 (45.2–52.0)	−1.4 (−4.1–1.3)	.30	0.1 (−2.8–2.8)	.99

NOTE. Values are mean ± SD, mean (95% CI), or as otherwise indicated.

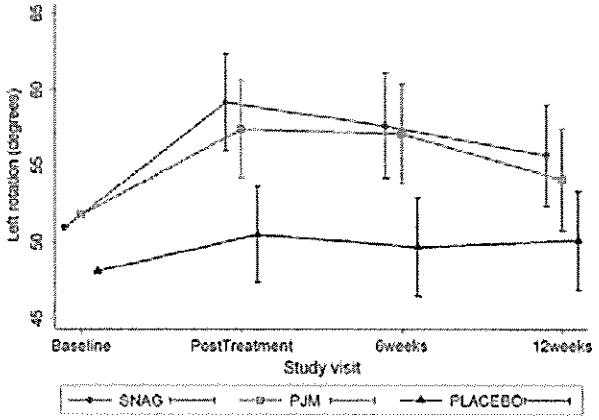
* Least square mean (adjusted for baseline and missing data) differences from visit to visit.



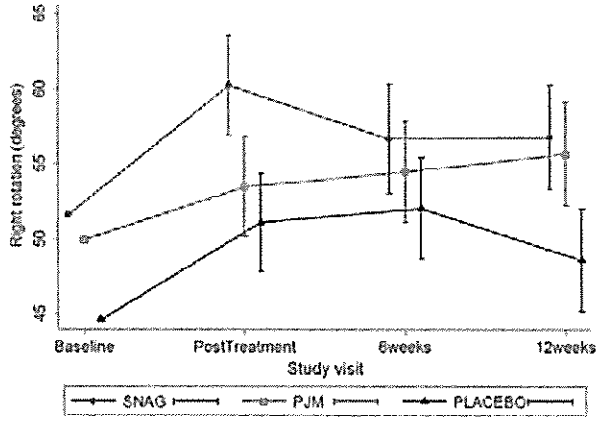
Extension



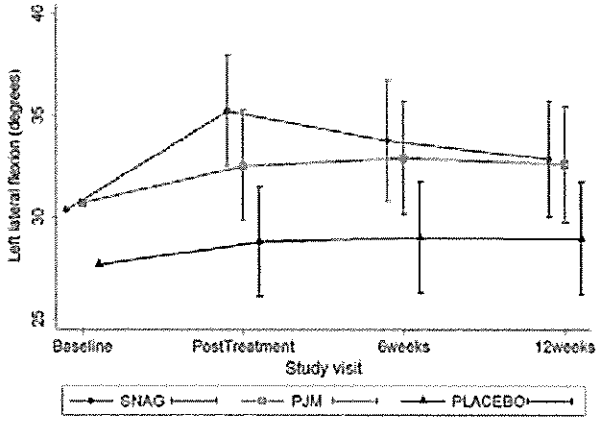
Flexion



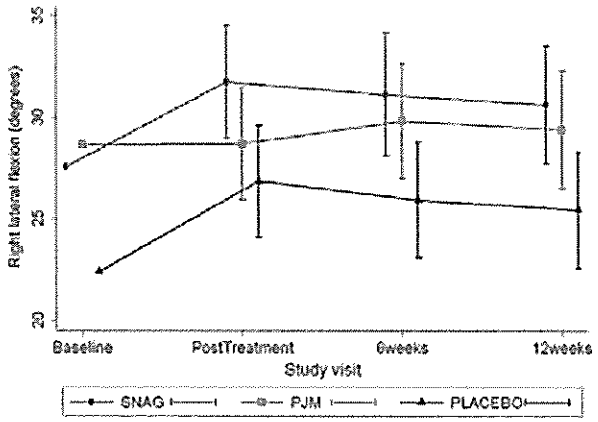
Left Rotation



Right Rotation



Left lateral flexion



Right lateral flexion

Fig 2 Changes in mean values (95% CI) for cervical ROM (measured with a CROM device) over time for each treatment group. The SNAG group received Mulligan SNAGs, the PJM group received Maitland PJM, and the placebo group received deactivated laser.

Table 4 Comparison of changes in head repositioning accuracy and balance over time for each treatment group

Measures	Group	Baseline	Posttreatment	12wk	Posttreatment vs Baseline Mean Difference* (95% CI)	P	12wk vs Baseline Mean Difference* (95% CI)	P
Head repositioning accuracy (deg)								
Left rotation	SNAG	4.7±3.5	5.3 (4.0–6.5)	5.7 (4.3–7.0)	−0.4 (−2.1–1.2)	.59	−0.8 (−2.5–0.9)	.33
	PJM	5.6±3.9	6.0 (4.8–7.3)	3.4 (2.0–4.7)	−0.6 (−2.2–1.0)	.48	2.1 (0.4–3.8)	.02
	Placebo	5.2±4.3	4.1 (2.8–5.3)	3.2 (1.9–4.5)	1.1 (0.05–2.7)	.16	2.0 (0.3–3.6)	.02
Right rotation	SNAG	5.4±4.8	4.2 (3.1–5.3)	2.5 (1.3–3.7)	1.2 (−0.4–2.8)	.15	2.9 (1.2–4.6)	<.001
	PJM	3.8±3.3	4.0 (2.9–5.2)	4.3 (3.1–5.5)	−0.1 (−1.7–1.5)	.92	−0.3 (−2.0–1.4)	.71
	Placebo	5.1±5.6	2.9 (1.8–4.0)	4.1 (3.0–5.3)	2.2 (0.6–3.8)	.01	0.9 (−0.8–2.6)	.29
Balance (sway index—cm)								
Eyes open	SNAG	0.3±0.1	0.3 (0.2–0.4)	0.3 (0.3–0.4)	0.0 (−0.1–0.1)	.82	0.0 (−0.1–0.1)	.76
	PJM	0.4±0.3	0.4 (0.4–0.5)	0.4 (0.3–0.5)	0.1 (−0.1–0.1)	.84	0.0 (−0.1–0.1)	.43
	Placebo	0.4±0.2	0.4 (0.3–0.4)	0.4 (0.3–0.5)	0.0 (−0.1–0.1)	.82	0.0 (−0.1–0.1)	.53
Eyes closed	SNAG	0.6±0.3	0.5 (0.1–1.0)	0.5 (−0.1–1.0)	0.1 (−0.5–0.6)	.84	0.1 (−0.4–0.7)	.64
	PJM	0.7±0.5	0.6 (0.1–1.1)	0.6 (0.1–1.1)	0.1 (−0.5–0.7)	.72	0.1 (−0.5–0.7)	.71
	Placebo	0.6±0.3	0.5 (0.0–0.9)	0.6 (0.1–1.1)	0.1 (−0.4–0.7)	.60	0.0 (−0.6–0.6)	.99
Extension	SNAG	1.5±0.8	1.6 (1.4–1.9)	1.2 (1.0–1.5)	−0.1 (−0.4–0.1)	.42	0.3 (0.1–0.6)	.02
	PJM	1.7±0.6	1.6 (1.4–1.8)	1.5 (1.3–1.8)	0.1 (−0.2–0.3)	.50	0.2(−0.1–0.5)	.17
	Placebo	1.7±0.7	1.5 (1.2–1.7)	1.5 (1.3–1.7)	0.2 (0.0–0.5)	.07	0.2 (−0.1–0.4)	.17
Left rotation	SNAG	0.6±0.4	0.4 (0.3–0.5)	0.5 (0.3–0.6)	0.2 (0.0–0.3)	.02	0.1 (0.0–0.3)	.08
	PJM	0.7±0.5	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.1 (0.0–0.3)	.06	0.1 (0.0–0.3)	.05
	Placebo	0.6±0.3	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.1 (−0.1–0.2)	.35	0.0 (−0.1–0.2)	.57
Right rotation	SNAG	0.5±0.5	0.4 (0.3–0.4)	0.4 (0.3–0.5)	0.1 (0.0–0.2)	.01	0.1 (0.0–0.2)	.26
	PJM	0.5±0.3	0.4 (0.3–0.4)	0.4 (0.4–0.5)	0.1 (0.0–0.2)	.04	0.0 (−0.1–0.1)	.64
	Placebo	0.4±0.2	0.4 (0.3–0.5)	0.3 (0.3–0.4)	0.0 (−0.1–0.1)	.44	0.1 (0.0–0.2)	.03
Moving base	SNAG	0.4±0.4	0.4 (0.3–0.4)	0.3 (0.3–0.4)	0.1 (0.0–0.2)	.13	0.1 (0.0–0.2)	.05
	PJM	0.5±0.4	0.3 (0.2–0.4)	0.4 (0.3–0.5)	0.2 (0.1–0.3)	<.001	0.1 (0.0–0.2)	.06
	Placebo	0.4±0.3	0.4 (0.3–0.5)	0.4 (0.3–0.5)	0.0 (−0.1–0.1)	.83	0.0 (−0.1–0.1)	.65

NOTE. Values are mean ± SD, mean (95% CI), or as otherwise indicated.
* Least square mean (adjusted for baseline and missing data) differences from visit to visit.

to the midrotation position[17,19] and should be considered for future studies.

In this study, there was no conclusive effect for SNAGs or PJMs on balance in people with cervicogenic dizziness. Most of the balance tests performed in this study were static balance tests. Recent studies[23] suggest that dynamic balance testing may be more sensitive than static balance testing in assessing impairments in those with neck injuries, and this should be considered in future research of patients with cervicogenic dizziness. Moreover, balance and postural control are known to decline with age.[24] Since the cohort for this study had a mean age of 64 years, which is older than the population in many other studies,[3,5] there may have been other factors contributing to balance impairment in this group, in addition to cervical spine dysfunction, that were not addressed by manual therapy.

People with a traumatic onset of neck symptoms have greater unsteadiness and greater head repositioning errors than those with insidious onset.[13,17,43,44] Most of the participants in the current study (66%) had an insidious onset of dizziness, which may have been a factor in the limited improvements observed in some balance tasks and in head repositioning accuracy after manual therapy treatment. Indeed, we observed greater balance deficits in participants with a history of trauma compared with those without this history.

The results of this study can be translated to people with cervicogenic dizziness in the wider community because recruitment was via press release in the Hunter region, Australia, making the study sample representative of the general population with this condition.

Study limitations

A limitation of the study was the use of the CROM device to measure head repositioning accuracy, since it may not be sensitive enough to detect small changes. A limitation of the analysis is that group sizes were established using the primary study outcome of intensity of dizziness.[12] Consequently there may be a compromised ability to detect small differences in the outcome measures reported in this article because of limited statistical power.

Conclusions

Treatment with SNAGs and a recommendation of ongoing self-SNAG exercises resulted in improvement in cervical spine ROM in people with chronic cervicogenic dizziness, and these effects were maintained for 12 weeks after treatment. There were no consistent or sustained effects from PJMs on cervical spine ROM. There was no conclusive effect for SNAGs or PJMs on head repositioning accuracy or balance in people with cervicogenic dizziness.

Suppliers

a. Therapower 40mW laser, serial no. 020601; Meyer Medical Electronics, 34 Percy St, Mordialloc, Victoria 3195, Australia.

b. Performance Attainment Associates, 12805 Lake Blvd, Lindstrom, MN 55045.
c. Chattecx Corp, Chattanooga Group, 101 Memorial Dr, Chattanooga, TN 37405.

Keywords

Cervical vertebrae; Musculoskeletal manipulations; Neck pain; Rehabilitation

Corresponding author

Susan A. Reid, MMedSc(Phty), School of Physiotherapy, The Australian Catholic University, 173 Pacific Hwy, North Sydney, NSW 2060, Australia. *E-mail address:* Sue.Reid@acu.edu.au.

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Chapter 6 Manual therapy treatment of cervicogenic dizziness: long-term outcomes of a randomised controlled trial

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Overview

This manuscript reports the long term results from the randomized controlled trial that was conducted for this thesis by reporting on all outcomes at 12 months after the interventions. It also reports the results of the head-to-head comparison of SNAGs (with self- administered SNAGs) and PJMs (with ROM exercises) for treating cervicogenic dizziness.

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Original article

Manual therapy for cervicogenic dizziness: Long-term outcomes of a randomised trial

Susan A. Reid [a, *], Robin Callister [b], Suzanne J. Snodgrass [b], Michael G. Katekar [b], Darren A. Rivett [b]

[a] Physiotherapy, Australian Catholic University, North Sydney, NSW 2060, Australia
[b] Faculty of Health and Medicine, The University of Newcastle, Callaghan, NSW 2038, Australia

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ABSTRACT

Manual therapy is effective for reducing cervicogenic dizziness, a disabling and persistent problem, in the short term. This study investigated the effects of sustained natural apophyseal glides (SNAGs) and passive joint mobilisations (PJMs) on cervicogenic dizziness compared to a placebo at 12 months post-treatment. Eighty-six participants (mean age 62 years, standard deviation (SD) 12.7) with chronic cervicogenic dizziness were randomised to receive SNAGs with self-SNAGs (n = 29), PJMs with range-of-motion (ROM) exercises (n = 29), or a placebo (n = 28) for 2–6 sessions over 6 weeks. Outcome measures were dizziness intensity, dizziness frequency (rated between 0 [none] and 5 [>once/day]), the Dizziness Handicap Inventory (DHI), pain intensity, head repositioning accuracy (HRA), cervical spine ROM, balance, and global perceived effect (GPE). At 12 months both manual therapy groups had less dizziness frequency (mean difference SNAGs vs placebo −0.7, 95% confidence interval (CI) −1.3, −0.2, p = 0.01; PJMs vs placebo −0.7, −1.2, −0.1, p = 0.02), lower DHI scores (mean difference SNAGs vs placebo −8.9, 95% CI −16.3, −1.6, p = 0.02; PJMs vs placebo −13.6, −20.8, −6.4, p < 0.001) and higher GPE compared to placebo, whereas there were no between-group differences in dizziness intensity, pain intensity or HRA. There was greater ROM in all six directions for the SNAG group and in four directions for the PJM group compared to placebo, and small improvements in balance for the SNAG group compared to placebo. There were no adverse effects. These results provide evidence that both forms of manual therapy have long-term beneficial effects in the treatment of chronic cervicogenic dizziness.

1. Introduction

Cervicogenic dizziness is non-rotatory dizziness described as unsteadiness that is associated with neck pain and/or stiffness and is triggered by cervical movements or positions (Heikkila et al., 2000; Wrisley et al., 2000). It is a common and potentially disabling condition, often persisting for many years and leading to psychological and emotional stress, a negative impact on quality of life and reduced productivity (Colledge et al., 1996; Yardley et al., 1998). The 2008 English Longitudinal Study of Ageing (>65 years) reported that 11.1% of aged individuals experience dizziness (Stevens et al., 2008) while another study found that 30% had dizziness and of those, 66% had cervical spine involvement (Colledge et al., 1996).

There is a growing body of evidence that manual therapy is beneficial in the treatment of cervicogenic dizziness in the short term (Karlberg et al., 1996; Reid and Rivett, 2005; Reid et al., 2008; Du et al., 2010; Fang, 2010; Lystad et al., 2011; Reid et al., 2014b), however there is very limited evidence of the benefits of manual therapy for this condition in the long term. To date there has been only one randomised controlled trial (RCT) by Malmstrom et al. (2007) who used a multi-modal treatment approach and reported reduced pain and dizziness two years after the interventions.

Previous studies (Reid et al., 2008, 2014b) demonstrated that Mulligan's sustained natural apophyseal glides (SNAGs) (Mulligan, 2004) and Maitland's passive joint mobilisations (PJMs) (Maitland, 2001) can produce reductions in severity and frequency of dizziness after treatment and that these results are sustained for 12 weeks post intervention. It was also found that the SNAG intervention resulted in improved cervical range of motion (ROM)

* Corresponding author. Physiotherapy, Australian Catholic University, PO Box 968, North Sydney, NSW 2060, Australia. Tel.: +61 2 97392267; fax: +61 2 97392132.
E-mail address: sue.reid@acu.edu.au (S.A. Reid).

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in all six movement directions immediately post-treatment and at 12 weeks compared to the placebo intervention, while the PJM treatment improved ROM in one direction (Reid et al., 2014a). To address the need for long-term follow-up studies, in this current paper we report on the symptoms and signs of cervicogenic dizziness in patients 12 months after the application of these two types of manual therapy treatment. We also compare the effects of these two commonly used types of manual therapy to each other as this has not been done before over the long term.

Accordingly, the research questions for the present study were:

1. For people with chronic cervicogenic dizziness, what are the effects of SNAGs (with self-administered SNAGs) and PJMs (with ROM exercises) compared to a placebo at 12-months post-treatment?
2. Is one form of manual therapy different to the other in the long term?

2. Method

2.1. Design

A randomised controlled trial (RCT) was conducted in a physiotherapy research laboratory at an Australian university. A detailed protocol has been published elsewhere (Reid et al., 2012). Briefly, participants with chronic cervicogenic dizziness were randomised using concealed allocation to one of three groups: Mulligan's SNAGs together with self-SNAGs, Maitland's PJMs with ROM exercises, or a placebo intervention of detuned laser. An independent statistician produced a computer generated randomisation sequence without stratification, which was placed in sequentially numbered, opaque sealed envelopes by a research assistant. Participants received between 2 and 6 intervention sessions over six weeks, the number determined by the treating physiotherapist based on the individual's response. Outcomes were measured at baseline, immediately post-treatment, at 12 weeks and at 12 months after the interventions. The post-treatment and 12-week follow-up outcomes have been published previously (Reid et al., 2014a,b).

Baseline and follow-up data were collected by research assistants who were blind to group allocation. The participant was also blind to whether they received an active or placebo intervention. The physiotherapist administering the interventions was not blind to group allocation. Adverse events were monitored at treatment sessions and follow-up assessment sessions.

2.2. Participants, therapists, centres

Participants were recruited from the general community via press releases, advertisements in local newspapers and letters to general practitioners and neurologists in the Hunter region of New South Wales, Australia. Phone screening, followed by thorough clinical examinations by a physiotherapist and a neurologist (including vestibular function testing), identified people with cervicogenic dizziness (Reid et al., 2014a,b). Included participants had non-rotary dizziness (described as unsteadiness) provoked by cervical spine movements, plus a history of neck pain and/or stiffness. They were aged between 18 and 90 years and had experienced dizziness symptoms for greater than 3 months. Potential participants were excluded if they had other types or causes of dizziness, such as vertigo, psychogenic dizziness, vertebro-basilar insufficiency, cardiovascular dizziness or migraines; or other causes of unsteadiness such as spinal cord pathology, stroke, or Parkinson's disease. They were also excluded if they had conditions for which manual therapy was contraindicated, if they were unable to read English, were pregnant, or receiving workers' compensation payments.

2.3. Interventions

A musculoskeletal physiotherapist recognised by the Australian Physiotherapy Association as a titled member for twenty-five years and who had formal post-graduate training in both the Maitland and Mulligan manual therapy approaches performed all the interventions.

Participants received one of the following interventions:

SNAGs: If active cervical spine flexion or extension produced dizziness, the treatment consisted of the physiotherapist applying a sustained glide anteriorly to the C2 spinous process as the participant actively moved their head in the symptomatic direction, as per Mulligan's recommendations (Mulligan, 2004). If rotation produced their dizziness, a sustained anterior glide was applied to the ipsilateral articular pillar as the participant rotated their head in the provocative direction. The SNAG, which was always performed without symptoms, was repeated six times and over-pressure was added to the end of the active movement at the second and subsequent treatments. After the second treatment, the participant was asked to self-SNAG into the provocative direction using their finger or a strap to assist the glide (Mulligan, 2004), for six repetitions once daily and to record compliance in a diary. They were asked to continue the self-SNAG once daily until the 12 month follow-up.

PJM: Mobilisation as described by Maitland et al. (Maitland, 2001) was applied to up to three hypomobile and/or painful joints in the upper cervical spine. This usually consisted of three 30–45 second oscillatory movement applications at each spinal level treated. The PJM could be a unilateral mobilisation over the articular pillar or a central pressure on the spinous process. After the second treatment ROM exercises into symptom-free flexion, extension, left and right rotation, and left and right lateral flexion, three times in each direction, once a day were commenced as a home exercise to be continued for 12 months, and recorded in a diary.

Placebo: A placebo intervention consisting of a deactivated laser device (Therapower 40 mW laser, serial No 020601, Meyer Medical Electronics, Mordialloc, Australia) was applied once (at each intervention session) for 2 min to each of three sites on the posterior neck, keeping the probe 0.5–1 cm from the skin (Reid et al., 2008, 2012).

2.4. Outcome measures

2.4.1. Primary outcome

The primary outcome was intensity of dizziness measured with a 100 mm visual analogue scale (VAS).

2.4.2. Secondary outcomes

1. frequency of dizziness measured on a six-point categorical rating scale (0 = no dizziness, 1 = dizziness < once per month, 2 = 1–4 episodes per month, 3 = 1–4 episodes per week, 4 = dizziness once daily, 5 = dizziness > once a day or constant).
2. the Dizziness Handicap Inventory (DHI) (0 = no handicap, 100 = maximum handicap)
3. intensity of cervical spine pain (100 mm VAS)
4. cervical spine ROM (flexion, extension, left and right rotation, left and right lateral flexion) measured three times for each direction with the Cervical Range Of Motion (CROM) device (Performance Attainment Associates, 3550 Lahore Rd, St Paul, MN) and averaged.

5. head repositioning accuracy (a measure of neck proprioception) into left and right rotation measured three times for each direction with the CROM device and averaged (Revel et al., 1991).
6. balance measured with a Chattecx balance dynamic system (Serial No 1001, Chattecx Corporation, The Chattanooga Group, Tennessee). Recordings were obtained under the following conditions:
   - standing on a stationary platform with the neck in the neutral position with eyes open (EO)
   - standing (stationary platform) with the neck in the neutral position with eyes closed (EC)
   - standing (stationary platform) with the neck extended (EO)
   - standing (stationary platform) with the neck in left rotation (EO)
   - standing (stationary platform) with the neck in right rotation (EO)
   - standing on a moving platform (EO).

   One trial of 15 s was performed for each condition.
7. global perceived effect (a measure of perceived benefit from the intervention) was measured by self-assessment on a five-point scale (0 = no benefit, 1 = minimal benefit, 2 = some benefit, 3 = a lot of benefit, 4 = great benefit, 5 = maximal benefit) as used in other studies (Koes et al., 1992a; Reid et al., 2008).

2.5. Data analysis

Using data from previous dizziness studies that used VAS (for main complaint) (Koes et al., 1992a,b) and DHI (Enloe and Shields, 1997; Storper et al., 1998; O'Reilly et al., 2000), it was determined that a sample size of 30 participants was required for each group to detect a clinically significant difference of 2 units on a 0–10 VAS scale between groups, with a power of 80%, a 5% confidence level, and a standard deviation of 2.4. To allow the study to be adequately powered for secondary outcomes, the DHI was also used for sample size calculations. Assuming that the standard deviation of DHI scores is 15, then 30 participants per group would provide 80% power to detect a difference of 11 units between groups for each comparison (Enloe and Shields, 1997; Storper et al., 1998; O'Reilly et al., 2000). Biostatisticians who were blind to participant identity performed the statistical analyses. Significance was accepted for p values of <0.05. The response variables were found to be consistent with a normal distribution so parametric statistics including t-tests and repeated measures analyses of variance (ANOVA) were used. To determine if there was change within each group from baseline to 12 months or from post-treatment to 12 months, and to assess differences between groups at the 12-month follow-up, a Linear Mixed Model ANOVA was used. To determine treatment effect (GPE), a repeated measures mixed effect model was used. An intention-to-treat analysis was used, with all participants included in the analysis at all time points. Data are reported as mean ± standard deviation.

3. Results

3.1. Flow of participants through the study

Participants were recruited from April 2010 to December 2011, with the follow-up period continuing until December 2012. Of the 683 volunteers who responded to recruitment, 86 entered the trial with 29 participants allocated to each of the SNAG and PJM groups, and 28 to the placebo group. The main reasons for exclusion were having symptoms inconsistent with cervicogenic dizziness, or no related neck problems. The flow of participants through the trial and loss to follow-up of three participants from the SNAG group, three from the PJM group and one from the placebo group are presented in Fig. 1. All dropouts were unrelated to treatment responses.

The average age of participants was 62 ± 12.7 (range 21–85) years, 50% were female and they reported having dizziness for a mean of 7.2 ± 6.8 years (range 3 months–30 years) before entering the study. There were no significant differences between groups for any outcome measures at baseline. At baseline, mean dizziness intensity was 43.3 (standard deviation (SD) 21.9) for the SNAG group, 50.3 (21.2) for the PJM group and 47.5 (24.9) for the placebo group, with comparison of means non-significant ($p = 0.51$). For the DHI the baseline values were 38.4 (16.3) for the SNAG group, 44.1 (19.8) for the PJM group and 42.8 (16.4) for the placebo group, with no significant differences when the means were compared ($p = 0.44$).

3.2. Effects of interventions

Participants in the SNAG group received an average of 4.2 ± 0.6 treatment sessions over the six-week intervention period, with the PJM group receiving 4.1 ± 0.5 and the placebo group 3.9 ± 0.3 sessions. Seven participants in each group were still keeping a diary at 12 months, with five people in each of the manual therapy groups indicating they were still doing the prescribed exercises daily.

3.2.1. Intensity of dizziness

There were no significant differences between the groups for intensity of dizziness at the 12-month follow up (Fig. 2, Table 1). At 12 months the intensity of dizziness was in the mild range (less than 30 mm on the VAS) for the two manual therapy groups, but still in the moderate range (30–54 mm) for the placebo group. By the 12-month follow-up there was a significant reduction in intensity of dizziness compared to baseline for all three groups with 43% and 53% reduction in the SNAG and PJM groups respectively compared to a 28% reduction with placebo (Fig. 2, Table 2). There was no significant change from post-treatment to 12-month follow-up for any group, though for the placebo group 67% of the change occurred in this period.

3.2.2. Frequency of dizziness

At the 12-month follow up there were significantly lower scores for frequency of dizziness for both the SNAG and PJM groups compared to the placebo, but no difference between the SNAG and PJM groups (Fig. 2, Table 1). There were significant reductions in the frequency of dizziness for all three groups at the 12-month follow up compared to baseline (Fig. 2, Table 2). There were significant improvements from post intervention to the 12-month follow up for both the manual therapy groups but not for the placebo group (Table 2).

3.2.3. Dizziness Handicap Inventory

The DHI scores for the SNAG and PJM groups were significantly lower than the placebo group at 12 months, but there was no difference between the two manual therapy groups (Fig. 2, Table 1). There were significant reductions in DHI scores at the 12-month follow-up compared to baseline in all three groups (Fig. 2, Table 2). The magnitude of these reductions was 38% for the SNAG group and 46% for the PJM group, compared to 15% for the placebo. The DHI scores at baseline indicated that for all three groups dizziness was having a moderate effect on participants' lives (DHI scores of 31–60). The scores had decreased at 12 months to indicate mild handicap (DHI scores 1–30) for both manual therapy groups whereas the placebo group remained in the moderate range (Fig. 2).

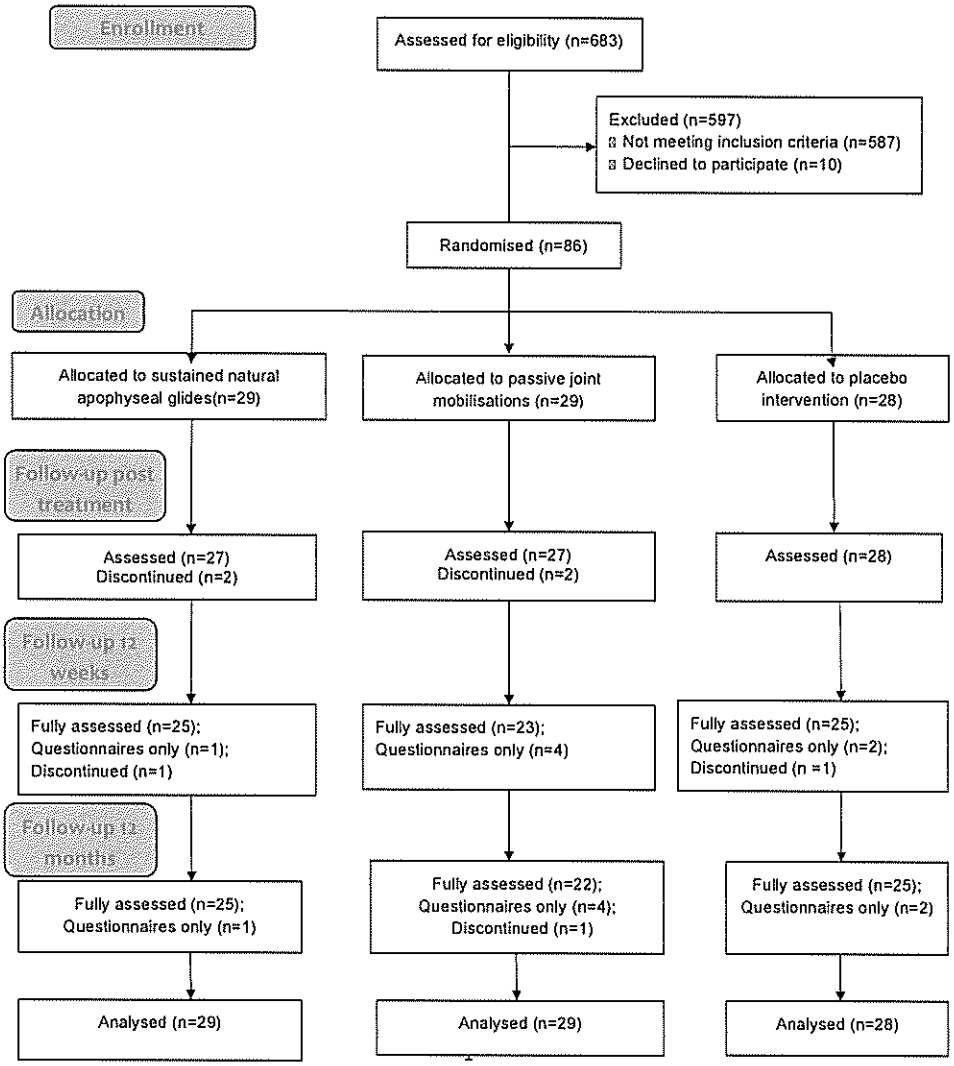

Fig. 1. Flow diagram of participants in the study. An intention to treat analysis was performed so all participants were analysed at all time points.

At 12 months the reductions in the DHI scores reached the minimal important change of 11 points for the SNAG and PJM groups, but not for the placebo group (Tamber et al., 2009).

3.2.4. *Intensity of cervical spine pain*

At 12 months there were no differences in pain intensity between any of the groups (Table 1). There was a significant ($p < 0.05$) decrease in intensity of cervical pain in all three groups at the 12-month follow-up compared to baseline (Fig. 2, Table 2). By 12 months pain levels for the SNAG and PJM groups were in the mild range (VAS scores of <30 mm) whereas the placebo group was still in the moderate range (30–54 mm) (Collins et al., 1997) (Fig. 2, Table 1). The minimal clinically important change (MCIC) for pain using a VAS has been reported to be 20 mm (Vernon et al., 2007). The MCIC for pain was reached for the PJM group with a change of 22.6 mm from baseline to 12 month follow-up, but the changes did not reach the MCIC for the other two groups.

3.2.5. *Cervical spine ROM*

Compared to placebo, the SNAG group had greater ROM in all six directions at 12 months and for the PJM group in four directions (Fig. 3, Table 1). There was no difference between the manual therapy groups at 12 months (Fig. 3, Table 1). Analysis of changes in cervical ROM over time found that ROM increased in five directions at the 12-month follow-up compared to baseline values after both the SNAG and PJM treatments and in two directions for the placebo group (Fig. 3, Table 3).

3.2.6. *Head repositioning accuracy*

There were no differences in head repositioning accuracy among groups at the 12-month follow-up (Table 1). Any changes were small (0.1–2.9°) and less than the standard error of measurement (SEM) with the CROM for rotation of 2.4–5.1° (Fletcher and Bandy, 2008).

3.2.7. *Balance*

At 12 months the SNAG group had significantly better balance than the placebo group with the neck in right rotation on a stationary base and with the head in neutral on a moving base (Table 1). At 12 months, there was improved balance compared to baseline for the SNAG group with the neck in right rotation and for the PJM group with the neck extended (both on a

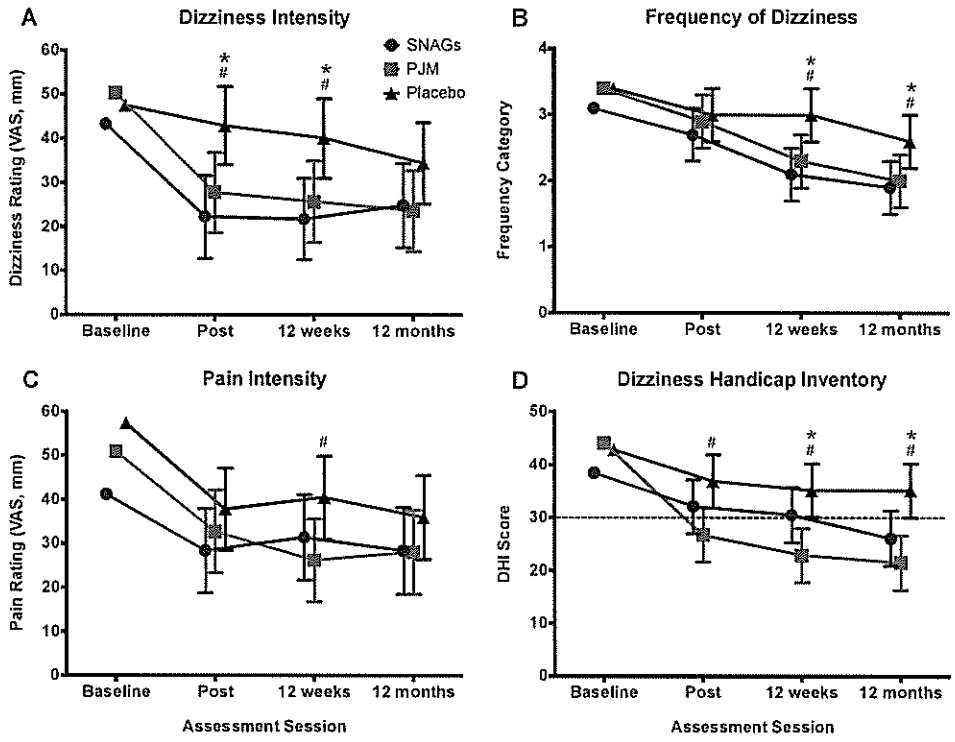

Fig. 2. Changes in mean (95% confidence interval) values for dizziness intensity, frequency of dizziness, pain intensity and Dizziness Handicap Inventory (DHI) over time (baseline, post-treatment, 12-week follow-up, 12-month follow-up) for each treatment group. The SNAG group received Mulligan's sustained natural apophyseal glides (SNAGs), the PJM group received Maitland's passive joint mobilisations (PJM) and the placebo group received deactivated laser. * Significant ($p < 0.05$) difference SNAG group to placebo group. # Significant ($p < 0.05$) difference PJM group to placebo group. DHI score ≤ 30 indicates Mild level of handicap.

stationary base), but no change in any positions for the placebo group (Table 3).

3.2.8. Global perceived effect

Both SNAG and PJM groups had significantly ($p < 0.05$) higher GPE ratings compared to the placebo group at 12 months (Table 1). The least square mean GPE score for the SNAG group at 12 months was 2.7 (95% confidence interval (CI) 2.2, 3.2) indicating 'a lot of benefit' and for the PJM group 2.3 (1.9, 2.8) ('some benefit') whereas for the placebo group GPE was 1.6 (1.1, 2.0) (between 'minimal benefit' and 'some benefit') (Koes et al., 1992a; Reid et al., 2008).

3.2.9. Adverse effects

There were no adverse effects that lasted longer than 24 h after any of the interventions.

4. Discussion

This study has shown that 2–6 treatments of two commonly used forms of manual therapy (SNAGs and PJM) provide both short and long-term benefits for people with chronic cervicogenic dizziness. Manual therapy treatment provides an immediate reduction in the intensity of dizziness, which is maintained 12 months later. Dizziness frequency and handicap continue to improve post-treatment and are substantially reduced at 12 months. There were no differences in the effectiveness of the two manual therapy treatments on these measures. Improvements in cervical range of motion were observed with both manual therapies although SNAG treatment provided greater and more immediate benefits than PJMs when compared to the placebo intervention. When the two manual therapy groups were compared to each other, there was no significant difference on any of the outcome measures at 12 months.

Although there were changes in the intensity of dizziness in all three groups by 12 months from baseline, the time course of the improvements differed between the manual therapy treatments and placebo. The improvements in the SNAG and PJM groups were observed immediately after the interventions and were then maintained for 12 months whereas the placebo group improvement was much more gradual and did not reach significance until 12 months after the intervention. Although not significantly different, the magnitude of improvement with manual therapy was almost double that of the placebo. The improvement in the placebo group at 12 months could be due to the placebo effect or natural resolution. It is also possible that following assessment and a diagnosis of a benign cervicogenic cause for their dizziness, participants were reassured that nothing more serious was responsible and subsequently developed better coping strategies. It is reasonable to conclude that the greater and more immediate improvement in dizziness after manual therapy is preferable to a slower and more limited resolution.

Frequency of dizziness improved in all groups but improved significantly more with manual therapy than the placebo. In contrast to intensity of dizziness, frequency of dizziness continued to improve after the treatment sessions were completed for both the manual therapy groups, with at least 60% of the improvement occurring over the ensuing 12 months. It is likely that such a change in frequency of dizziness from 1 to 4 times a week at baseline to 1 to 4 times a month at 12 months for both the manual therapy groups would make a meaningful difference to a person's quality of life.

The parallel reductions in frequency of dizziness and DHI scores from post intervention to the 12 month follow-up for both

Table 1
Differences between treatment groups in each outcome measure at 12 months.

Self-report measures			
		12 months	
		Mean diff (95% CI)	*p*
VAS dizziness	SNAG vs placebo	−9.6 (−22.8, 3.6)	0.15
	PJM vs placebo	−10.8 (−23.8, 2.2)	0.1
	PJM vs SNAG	−1.2 (−14.5, 12.0)	0.85
Dizziness frequency	SNAG vs placebo	−0.7 (−1.3, −0.2)	0.01*
	PJM vs placebo	−0.7 (−1.2, −0.1)	0.02*
	PJM vs SNAG	0.1 (−0.5, 0.7)	0.77
DHI	SNAG vs placebo	−8.9 (−16.3, −1.6)	0.02*
	PJM vs placebo	−13.6 (−20.8, −6.4)	<0.001*
	PJM vs SNAG	−4.7 (−12.0, 2.7)	0.21
VAS pain	SNAG vs placebo	−7.6 (−21.4, 6.3)	0.28
	PJM vs placebo	−7.9 (−21.3, 5.6)	0.25
	PJM vs SNAG	−0.3 (−14.1, 13.4)	0.96
Global perceived effect	SNAG vs placebo	1.1 (0.5, 1.8)	<0.001*
	PJM vs placebo	0.8 (0.1, 1.4)	0.02*
	PJM vs SNAG	−0.4 (−1.0, 0.3)	0.27
Physical measures			
		12 months	
		Mean diff (95% CI)	*p*
ROM (degrees)			
Flexion	SNAG vs placebo	4.9 (0.2, 9.7)	0.04*
	PJM vs placebo	4.7 (−0.1, 9.5)	0.06
	PJM vs SNAG	−0.2 (−5.1, 4.6)	0.93
Extension	SNAG vs placebo	10.4 (4.6, 16.1)	<0.001*
	PJM vs placebo	5.6 (−0.3, 11.4)	0.06
	PJM vs SNAG	−4.8 (−10.6, 1.0)	0.11
Left rotation	SNAG vs placebo	8.3 (3.7, 13.0)	<0.001*
	PJM vs placebo	8.2 (3.5, 13.0)	<0.001*
	PJM vs SNAG	−0.1 (−4.8, 4.6)	0.96
Right rotation	SNAG vs placebo	5.6 (0.7, 10.5)	0.03*
	PJM vs placebo	5.8 (0.9, 10.7)	0.02*
	PJM vs SNAG	0.2 (−4.7, 5.1)	0.93
Left lateral flexion	SNAG vs placebo	7.0 (3.0, 11.0)	<0.001*
	PJM vs placebo	5.9 (1.8, 9.9)	<0.001*
	PJM vs SNAG	−1.1 (−5.1, 2.9)	0.58
Right lateral flexion	SNAG vs placebo	5.6 (1.5, 9.7)	0.01*
	PJM vs placebo	6.5 (2.4, 10.7)	<0.001*
	PJM vs SNAG	0.9 (−3.2, 5.0)	0.66
Head repositioning accuracy (degrees)			
Left rotation	SNAG vs placebo	0.4 (−2.4, 2.3)	0.64
	PJM vs placebo	0.8 (−1.1, 2.7)	0.42
	PJM vs SNAG	0.3 (−1.6, 2.3)	0.72
Right rotation	SNAG vs placebo	−0.9 (−2.6, 0.7)	0.28
	PJM vs placebo	−0.5 (−2.2, 1.2)	0.57
	PJM vs SNAG	0.4 (−1.3, 2.1)	0.63
Balance (sway index, cm)			
Eyes open	SNAG vs placebo	−0.01 (−0.12, 0.10)	0.87
	PJM vs placebo	−0.01 (−0.12, 0.11)	0.91
	PJM vs SNAG	0.00 (−0.11, 0.12)	0.96
Eyes closed	SNAG vs placebo	−0.18 (−0.88, 0.52)	0.62
	PJM vs placebo	−0.02 (−0.74, 0.70)	0.95
	PJM vs SNAG	0.16 (−0.56, 0.88)	0.67
Extension	SNAG vs placebo	−0.05 (−0.39, 0.30)	0.79
	PJM vs placebo	−0.06 (−0.40, 0.29)	0.74
	PJM vs SNAG	−0.01 (−0.36, 0.34)	0.95
Left rotation	SNAG vs placebo	−0.09 (−0.25, 0.07)	0.25
	PJM vs placebo	−0.05 (−0.22, 0.11)	0.51
	PJM vs SNAG	0.04 (−0.12, 0.20)	0.64
Right rotation	SNAG vs placebo	−0.15 (−0.26, −0.03)	0.01*
	PJM vs placebo	−0.04 (−0.16, 0.07)	0.49
	PJM vs SNAG	0.11 (−0.01, 0.22)	0.07
Moving base	SNAG vs placebo	−0.14 (−0.25, 0.02)	0.03*
	PJM vs placebo	−0.12 (−0.24, 0.00)	0.05
	PJM vs SNAG	0.01 (−0.11, 0.13)	0.83

VAS = visual analogue scale, SNAG = sustained natural apophyseal glide, PJM = passive joint mobilisation, DHI = dizziness handicap inventory, ROM = range of motion, 95% CI = 95% confidence interval. Mean diff = differences between groups for the least square mean (adjusted for baseline and missing data), *$p < 0.05$.

manual therapy groups suggest that handicap may have been closely linked to how often participants were experiencing dizziness. DHI scores had improved more with manual therapy than with the placebo at 12 months with 30–40% of this improvement occurring after treatment sessions were completed. It is plausible that some of the emotional aspects measured by the DHI, such as depression, frustration, and stress may have continued to improve as confidence that the reduction in dizziness would persist became more evident.

Similar reductions in the intensity of pain were observed post-treatment and maintained at 12 months in all three groups. Notably some participants had very low pain scores, as people were included based on reports of dizziness and either neck pain or stiffness, and there may not have been adequate sensitivity to show differences between groups.

The SNAG intervention had a greater and more immediate effect than PJM on cervical ROM. Improvements in ROM for those receiving the SNAG treatment were evident immediately after the intervention and then maintained for 12 months, whereas for the PJM group it was a slower, steady improvement over time, with only three directions increasing immediately after the intervention but a further two directions improving in the 12-month follow-up period. It is possible that the self-SNAGs and ROM exercises may have helped to maintain this improvement in ROM after the intervention phase with further restoration of normal movement over the 12 months, although this cannot be confirmed given the low compliance in completing the diaries. It has been hypothesised that by increasing cervical ROM, information from cervical sensory afferents is normalised and thus dizziness of cervical origin is reduced (Mulligan, 1991; Furman and Cass, 1996; Wilson, 1996). In contrast to some other studies (Heikkila et al., 2000; Wu et al., 2006) the present study did not find significant improvements in disturbed head movement precision after manual therapy, which may be a consequence of the insensitivity of the CROM as a measuring device.

There were significantly greater improvements ($p < 0.05$) in balance in the SNAG group compared to the placebo group at 12 months when participants were tested standing on a stationary platform with the neck in right rotation and when standing on a moving platform, both with eyes open. There was no difference between the PJM and placebo groups or between the two manual therapy groups.

As SNAGs and PJMs were both found to be beneficial in the long term, and because one was not found to be different to the other in reducing dizziness intensity or frequency or their related handicap, a clinician managing a patient with chronic cervicogenic dizziness, could initially apply either SNAGs or PJMs for 2–6 treatments with equal confidence. If there was no improvement, then the clinician could then try the other type of manual therapy. If the patient presents with decreased cervical ROM, it is recommended that SNAGs with self-SNAGs should be applied first, as this manual therapy approach was found to be more beneficial for improving ROM.

A major strength of this study is that the results can be generalised to people with cervicogenic dizziness in the general community. Recruitment was via press releases and advertisements in newspapers in the Hunter region, Australia and referral was from general practitioners and neurologists, therefore it is likely the study sample is representative of the wider general population with cervicogenic dizziness, in terms of gender, age, duration and intensity of symptoms. Recruitment was from a wide geographical area that includes a major metropolitan centre, several large regional towns, smaller rural centres and farming districts resulting in the study population being representative of the general population with cervicogenic dizziness. Although the study took place in a research laboratory within a university, the

Table 2
Comparison of changes in self-reported outcome measures over time for each treatment group.

	Group	Baseline	Post-treatment	12 months	Post-treatment vs baseline		12 months vs Baseline		12 months vs Post-treatment	
					Mean diff (95% CI)	*p* Value	Mean diff (95% CI)	*p* Value	Mean diff (95% CI)	*p* Value
VAS dizziness	SNAG	43.3 (21.9)	22.3 (12.9, 31.6)	24.8 (15.3, 34.3)	22.5 (13.0, 32.1)	0.001*	20.0 (10.3, 29.7)	0.001*	−2.6 (−12.6, 7.4)	0.61
	PJM	50.3 (21.2)	27.8 (18.6, 36.9)	23.6 (14.4, 32.8)	20.8 (11.5, 30.1)	0.001*	25.3 (15.8, 34.7)	0.001*	4.5 (−5.1, 14.0)	0.36
	Placebo	47.5 (24.9)	42.9 (34.0, 51.8)	34.4 (25.2, 43.6)	4.2 (−5.1, 13.4)	0.38	12.8 (3.3, 22.2)	0.008*	8.6 (−0.9, 18.1)	0.08
Freq dizziness	SNAG	3.1 (1.5)	2.7 (2.3, 3.1)	1.9 (1.5, 2.3)	0.5 (0.1, 1.0)	0.02*	1.3 (0.9, 1.8)	0.001*	0.8 (0.3, 1.3)	<0.001*
	PJM	3.4 (0.9)	2.9 (2.5, 3.3)	2.0 (1.6, 2.4)	0.5 (0.0, 0.9)	0.03*	1.4 (1.9, 1.9)	0.001*	0.9 (0.5, 1.4)	<0.001*
	Placebo	3.4 (1.0)	3.0 (2.6, 3.4)	2.6 (2.2, 3.0)	0.4 (0.1, 0.8)	0.11	0.8 (0.3, 1.2)	0.001*	0.4 (−0.1, 0.9)	0.08
DHI	SNAG	38.4 (16.3)	32.1 (27.0, 37.2)	26.1 (20.9, 31.3)	8.6 (4.0, 13.2)	0.001*	14.6 (9.8, 19.3)	0.001*	5.9 (1.2, 10.7)	0.01*
	PJM	44.1 (19.8)	26.7 (21.6, 31.8)	21.5 (16.3, 26.6)	15.2 (10.5, 19.8)	0.001*	20.4 (15.8, 25.1)	0.001*	5.3 (0.6, 10.0)	0.03*
	Placebo	42.8 (16.4)	36.9 (31.9, 41.9)	35.1 (30.0, 40.2)	4.6 (0.1, 9.2)	0.05*	6.6 (1.9, 11.3)	0.006*	2.0 (−2.7, 6.6)	0.41
VAS pain	SNAG	41.2 (26.5)	28.4 (18.9, 38.0)	28.4 (18.6, 38.2)	15.9 (5.6, 26.2)	0.003*	15.6 (5.0, 26.1)	0.004*	−0.3 (−10.9, 10.3)	0.95
	PJM	50.9 (22.3)	32.7 (23.3, 42.1)	28.1 (18.6, 37.6)	17.9 (7.6, 28.2)	0.001*	22.6 (12.2, 33.0)	0.001*	4.8 (−5.8, 15.3)	0.37
	Placebo	57.4 (28.1)	37.8 (28.5, 47.1)	36.0 (26.4, 45.5)	16.7 (6.5, 26.9)	0.001*	18.4 (8.0, 28.9)	0.001*	1.7 (−8.8, 12.2)	0.75

VAS = visual analogue scale, SNAG = sustained natural apophyseal glide, PJM = passive joint mobilisation, DHI = dizziness handicap inventory, SD = standard deviation, 95% CI = 95% confidence interval, Mean diff = least square mean (adjusted for baseline and missing data) differences from visit to visit, *$p < 0.05$.

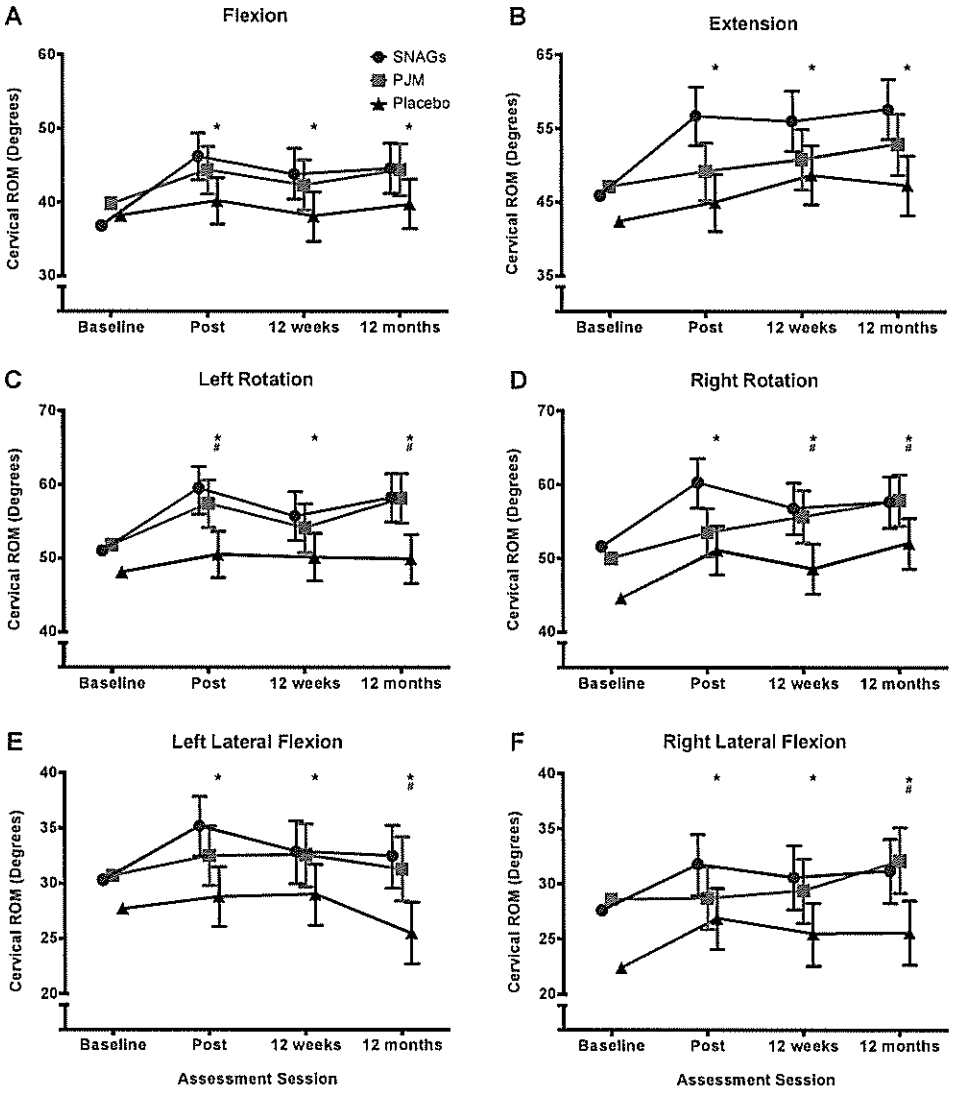

Fig. 3. Changes in mean (95% confidence interval) values for cervical range of motion (measured with a CROM device) over time (baseline, post-treatment, 12-week follow-up, 12-month follow-up) for each treatment group. The SNAG group received Mulligan's sustained natural apophyseal glides (SNAGs), the PJM group received Maitland's passive joint mobilisations (PJM) and the placebo group received deactivated laser. * Significant ($p < 0.05$) difference SNAG group to placebo group. # Significant ($p < 0.05$) difference PJM group to placebo group.

Table 3
Comparison of changes in physical outcome measures over time for each treatment group.

	Group	Baseline	Post-treatment	12 months	Post-treatment vs baseline		12 months vs Baseline		12 months vs Post-treatment	
					Mean diff (95% CI)	*p* Value	Mean diff (95% CI)	*p* Value	Mean diff (95% CI)	*p* Value
Cervical ROM (degrees)										
Flexion	SNAG	36.8 (14.6)	46.2 (43.0, 49.4)	44.6 (41.2, 48.0)	−8.3 (−11.9, −4.6)	<0.001*	−6.8 (−10.6, −3.0)	<0.001*	1.5 (−2.3, 5.3)	0.44
	PJM	39.8 (14.5)	44.4 (41.1, 47.6)	44.4 (40.9, 47.9)	−4.9 (−8.6, −1.3)	0.01*	−5.0 (−8.9, −1.1)	0.01*	−0.1 (−4.0, 3.9)	0.97
	Placebo	38.2 (12.1)	40.2 (37.0, 43.3)	39.7 (36.4, 43.1)	−1.7 (−5.3, 1.9)	0.36	−1.3 (−5.1, 2.5)	0.51	0.4 (−3.4, 4.2)	0.83
Extension	SNAG	45.9 (13.4)	56.7 (52.7, 60.6)	57.6 (53.6, 61.7)	−10.6 (−14.5, −6.6)	<0.001*	−12.0 (−16.1, −7.9)	<0.001*	−1.4 (−5.5, 2.7)	0.5
	PJM	47.1 (11.5)	49.2 (45.2, 53.1)	52.9 (48.7, 57.0)	2.7 (−6.6, 1.3)	0.19	−6.3 (−10.5, −2.0)	0.004*	−3.6 (−7.9, 0.6)	0.1
	Placebo	42.4 (13.4)	45.0 (41.1, 48.8)	47.3 (43.2, 51.3)	−0.7 (−4.6, 3.2)	0.72	−3.0 (−7.1, 1.1)	0.15	−2.3 (−6.4, 1.8)	0.27
Left rotation	SNAG	51.0 (12.8)	59.2 (56.0, 62.4)	58.2 (54.9, 61.5)	−8.5 (−11.6, −5.4)	<0.001*	−7.6 (−10.9, −4.4)	<0.001*	0.9 (−2.4, 4.2)	0.59
	PJM	51.8 (11.1)	57.4 (54.2, 60.6)	58.1 (54.8, 61.5)	−8.5 (−11.6, −5.4)	<0.001*	−7.3 (−10.6, −3.9)	<0.001*	0.7 (−4.1, 2.6)	0.67
	Placebo	48.1 (14.7)	50.5 (47.4, 53.6)	49.9 (46.6, 53.2)	−0.9 (−3.9, 2.2)	0.59	−0.3 (−3.6, 3.0)	0.855	0.6 (−2.7, 3.8)	0.74
Right rotation	SNAG	51.6 (16.6)	60.3 (56.9, 63.6)	57.7 (54.2, 61.1)	−10.2 (−13.5, −6.9)	<0.001*	−7.7 (−11.2, −4.2)	<0.001*	2.5 (−1.0, 6.0)	0.16
	PJM	50.0 (10.3)	53.5 (50.2, 56.8)	57.9 (54.4, 61.4)	−4.1 (−7.4, −0.7)	0.02*	−8.6 (−12.2, 0.5.0)	<0.001*	−4.5 (−8.1, −0.9)	0.01*
	Placebo	44.6 (11.9)	51.1 (47.8, 54.4)	52.1 (48.6, 55.5)	−3.7 (−7.0, −0.4)	0.03*	−4.7 (−8.2, −1.2)	0.008*	−1.0 (−4.5, 2.4)	0.56
Left lateral flexion	SNAG	30.3 (10.8)	35.2 (32.5, 37.9)	25.5 (22.7, 28.3)	−5.1 (−7.8, −2.5)	<0.001*	−2.3 (−5.1, 0.4)	0.1	2.8 (0.0, 5.6)	0.05
	PJM	30.7 (8.5)	32.5 (29.8, 35.2)	31.3 (28.4, 34.2)	−2.3 (−5.0, 0.3)	0.09	−1.2 (−4.0, 1.6)	0.41	1.1 (−1.7, 4.0)	0.44
	Placebo	27.7 (8.5)	28.8 (26.1, 31.5)	25.5 (22.7, 28.3)	0.6 (−2.0, 3.2)	0.65	4.0 (1.2, 6.7)	0.005*	3.4 (0.6, 6.1)	0.02*
Right lateral flexion	SNAG	27.6 (12.3)	31.8 (29.0, 34.5)	31.2 (28.3, 34.1)	−5.3 (−7.9, −2.4)	<0.001*	−4.6 (−7.4, −1.7)	0.002*	0.6 (−2.3, 3.5)	0.68
	PJM	50.0 (10.3)	53.5 (50.2, 56.8)	32.1 (29.2, 35.1)	−1.9 (−4.6, 0.9)	0.18	−5.3 (−8.2, −2.3)	<0.001*	−3.4 (−6.4, −0.5)	0.02*
	Placebo	44.6 (11.9)	51.1 (47.8, 54.4)	25.6 (22.7, 28.5)	−1.4 (−4.1, 1.3)	0.3	−0.1 (−3.0, 2.7)	0.92	1.3 (−1.6, 4.1)	0.38
Head repositioning accuracy (degrees)										
Left rotation	SNAG	4.7 (3.5)	5.3 (4.0, 6.5)	3.9 (2.6, 5.2)	−0.4 (−2.1, 1.2)	0.59	0.9 (−0.7, 2.6)	0.27	1.4 (−0.3, 3.1)	0.11
	PJM	5.6 (3.9)	6.0 (4.8, 7.3)	4.2 (2.9, 5.6)	−0.6 (−2.2, 1.0)	0.48	1.2 (−0.5, 2.9)	0.17	1.8 (0.0, 3.5)	0.046*
	Placebo	5.2 (4.3)	4.1 (2.8, 5.3)	3.4 (2.1, 4.8)	1.1 (0.05, 2.7)	0.16	1.8 (0.1, 3.4)	0.04*	0.6 (−1.1, 2.3)	0.47
Right rotation	SNAG	5.4 (4.8)	4.2 (3.1, 5.3)	4.1 (2.9, 5.3)	1.2 (−0.4, 2.8)	0.15	2.2 (0.5, 3.9)	0.01*	1.0 (−0.7, 2.7)	0.26
	PJM	3.8 (3.3)	4.0 (2.9, 5.2)	3.2 (2.0, 4.4)	−0.1 (−1.7, 1.5)	0.92	0.3 (−1.4, 2.1)	0.7	0.4 (−1.3, 2.2)	0.64
	Placebo	5.1 (5.6)	2.9 (1.8, 4.0)	4.1 (2.9, 5.3)	2.2 (0.6, 3.8)	0.01*	0.9 (−0.8, 2.6)	0.28	−1.2 (−2.9, 0.4)	0.15
Balance (sway index, cm)										
Eyes open	SNAG	0.30 (0.14)	0.32 (0.25, 0.40)	0.41 (0.33, 0.49)	0.01 (−0.09, 0.10)	0.89	−0.08 (−0.18, 0.02)	0.1	−0.09 (−0.19, 0.01)	0.08
	PJM	0.44 (0.28)	0.43 (0.36, 0.51)	0.41 (0.33, 0.49)	0.01 (−0.10, 0.08)	0.84	0.01 (−0.09, 0.11)	0.78	0.02 (−0.08, 0.13)	0.64
	Placebo	0.36 (0.20)	0.43 (0.36, 0.45)	0.42 (0.34, 0.50)	0.01 (−0.10, 0.08)	0.82	−0.05 (−0.15, 0.05)	0.3	−0.04 (−0.14, 0.06)	0.41
Eyes closed	SNAG	0.57 (0.34)	0.54 (0.07, 1.01)	0.49 (0.00, 0.99)	0.06 (−0.51, 0.62)	0.84	0.11 (−0.48, 0.69)	0.72	0.05 (−0.54, 0.64)	0.87
	PJM	0.73 (0.46)	0.60 (0.13, 1.06)	0.65 (0.13, 1.17)	0.10 (−0.46, 0.67)	0.72	0.05 (−0.55, 0.65)	0.87	−0.05 (−0.66, 0.55)	0.86
	Placebo	0.61 (0.28)	0.47 (0.01, 0.93)	0.67 (0.18, 1.16)	0.15 (−0.42, 0.71)	0.6	−0.05 (−0.64, 0.54)	0.86	−0.20 (−0.79, 0.39)	0.5
Extension	SNAG	1.51 (0.78)	1.65 (1.42, 1.88)	1.44 (1.20, 1.69)	−0.10 (−0.36, 0.15)	0.42	0.11 (−0.16, 0.37)	0.43	0.21 (−0.06, 0.47)	0.12
	PJM	1.75 (0.58)	1.62 (1.38, 1.85)	1.43 (1.18, 1.68)	0.10 (−0.16, 0.35)	0.46	0.29 (0.02, 0.56)	0.03*	0.20 (−0.07, 0.47)	0.15
	Placebo	1.71 (0.69)	1.45 (1.23, 1.68)	1.49 (1.25, 1.73)	0.23 (−0.02, 0.48)	0.07	0.19 (−0.07, 0.46)	0.15	−0.04 (−0.30, 0.23)	0.78
Left rotation	SNAG	0.58 (0.38)	0.42 (0.32, 0.53)	0.49 (0.37, 0.60)	0.16 (0.02, 0.30)	0.02*	0.10 (−0.05, 0.24)	0.18	−0.06 (−0.21, 0.08)	0.4
	PJM	0.67 (0.47)	0.52 (0.41, 0.63)	0.52 (0.41, 0.64)	0.13 (−0.01, 0.27)	0.06	0.12 (−0.03, 0.27)	0.1	−0.01 (−0.16, 0.14)	0.91
	Placebo	0.57 (0.32)	0.51 (0.41, 0.61)	0.58 (0.47, 0.69)	0.07 (−0.07, 0.21)	0.35	0.00 (−0.15, 0.15)	0.99	−0.07 (−0.21, 0.08)	0.36
Right rotation	SNAG	0.49 (0.48)	0.35 (0.27, 0.43)	0.31 (0.23, 0.39)	0.13 (0.03, 0.23)	0.01*	0.17 (0.06, 0.27)	0.002*	0.04 (−0.07, 0.14)	0.47
	PJM	0.47 (0.30)	0.36 (0.29, 0.44)	0.42 (0.33, 0.50)	0.11 (0.01, 0.21)	0.04*	0.05 (−0.05, 0.16)	0.31	−0.05 (−0.16, 0.06)	0.34
	Placebo	0.44 (0.25)	0.41 (0.33, 0.48)	0.46 (0.38, 0.54)	0.04 (−0.06, 0.14)	0.44	−0.01 (−0.11, 0.10)	0.89	−0.05 (−0.15, 0.06)	0.37
Moving base	SNAG	0.45 (0.42)	0.37 (0.29, 0.45)	0.35 (0.27, 0.43)	0.08 (−0.02, 0.18)	0.13	0.10 (−0.01, 0.20)	0.72	0.02 (−0.09, 0.13)	0.74
	PJM	0.50 (0.37)	0.31 (0.23, 0.39)	0.36 (0.28, 0.45)	0.18 (0.07, 0.28)	<0.001*	0.12 (0.01, 0.23)	0.31	−0.05 (−0.17, 0.06)	0.33
	Placebo	0.39 (0.27)	0.43 (0.35, 0.51)	0.48 (0.40, 0.57)	0.01 (−0.11, 0.09)	0.83	−0.07 (−0.17, 0.04)	0.22	−0.06 (−0.16, 0.05)	0.31

VAS = visual analogue scale, SNAG = sustained natural apophyseal glide, PJM = passive joint mobilisation, DHI = dizziness handicap inventory, SD = standard deviation, 95% CI = 95% confidence interval. Mean diff = least square mean (adjusted for baseline and missing data) differences from visit to visit, *$p < 0.05$.

study setting was designed to reflect everyday normal physical therapy clinical conditions, further enhancing the generalisability of the findings.

One limitation of this study is that the extent to which participants regularly performed their home exercises is unknown, as only a low proportion recorded this in their diaries. It therefore cannot be determined whether the home exercises contributed to the post-treatment improvements observed in dizziness frequency and DHI scores. A second limitation, consistent with all manual therapy randomised controlled trials, is that it was not possible to blind the treating therapist to group allocation.

This study has provided evidence for the first time that a small number of manual therapy treatments combined with recomm endations to perform simple home-based exercises can make a significant difference over the long term to patients experiencing chronic cervicogenic dizziness. Since cervicogenic dizziness is a relatively common problem in the community, the findings of this study have the potential to benefit many patients.

5. Conclusion

These results provide evidence that both forms of manual therapy have long-term beneficial effects in the treatment of chronic cervicogenic dizziness. There was a greater and more immediate effect on improving cervical spine ROM with the SNAG intervention than with PJM and ROM exercises.

Ethical clearance statement

Ethics approval was granted by the Human Research Ethics Committee at The University of Newcastle. No. H-2009-0377.

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Chapter 7 Identification of individuals with chronic cervicogenic dizziness for participation in a randomised controlled trial

The work presented in this chapter has been submitted as:

Reid S, Callister R, Katekar M, Rivett D. Identification of individuals with chronic cervicogenic dizziness for participation in a randomised controlled trial. *Manual Therapy* (under review).

Overview

This chapter provides a more comprehensive report of the screening process that was used to identify people with chronic cervicogenic dizziness to participate in the RCT described in Chapters Three, Four, Five and Six. This work has been presented at a number of conferences and the decision to submit it was based on supportive feedback from presentation at the conferences. There is a need for more education to assist in identifying those with cervicogenic dizziness and this screening process may have the potential to inform the development of a clinical screening tool. It is presented as Chapter Seven after the other papers and manuscripts as it includes data from the RCT findings described in the previous chapters. As a high proportion of participants benefited from cervical spine manual therapy, it provides some indirect evidence that this was a successful screening process to identify individuals with cervicogenic dizziness.

Identification of individuals with chronic cervicogenic dizziness for participation in a randomised controlled trial

Susan A. Reid[a] MMedSc(Phty), BAppSc(Phty), GradDipManipTher, BPharm

Lecturer in Physiotherapy, Australian Catholic University, North Sydney, Australia

Robin Callister[b] BPharm, MSc, PhD

Professor in School of Biomedical Science and Pharmacy, The University of Newcastle, Australia

Michael G. Katekar[b] MBBS, FRACP

Consultant Neurologist & Conjoint Senior Lecturer in Faculty of Health and Medicine, The University of Newcastle, Australia

Darren A. Rivett[b] BAppSc(Phty), MAppSc(ManipPhty), PhD

Professor and Head of School of Health Sciences, The University of Newcastle, Australia

Institutional address

[a] Physiotherapy, Australian Catholic University, North Sydney, NSW 2060 Australia

[b] Faculty of Health and Medicine, The University of Newcastle, Callaghan, NSW 2308 Australia

Correspondence to: Susan Reid

Physiotherapy, Australian Catholic University, PO Box 968, North Sydney, NSW. 2059. Australia.

Phone: +61 2 97392267, Fax: +61 2 97392132. E-mail: sue.reid@acu.edu.au

ABSTRACT

A screening process was developed to identify people with chronic cervicogenic dizziness for inclusion in a randomised controlled trial. This process may be of assistance to clinicians or researchers for identification of this condition. Participants with dizziness described as unsteadiness and with neck pain/stiffness were recruited from the Hunter region, Australia. Firstly, potential participants were screened in a telephone interview to determine if their dizziness was possibly of cervical spine origin based on the type and behaviour of dizziness, presence of cervical symptoms, triggers such as neck movements, and elimination of other causes for dizziness. Secondly, the potential participant underwent physical assessment by a physiotherapist including cervical active movements, cervical palpation, and the Dix-Hallpike manoeuvre. They were then assessed by an oto-neurologist to exclude other non-cervical causes of dizziness. Recruitment yielded 683 potential participants. After telephone screening, 482 people (71%) were excluded. Ten individuals (1%) declined to participate. A further 54 (8%) were excluded after physical examination by the physiotherapist and 51 (7%) by the oto-neurologist (for having Benign Paroxysmal Positional Vertigo, central causes or migraines). Eighty-six (13%) participants were identified as having cervicogenic dizziness. Fifty-eight individuals identified using this process received manual therapy to the cervical spine and for 44 (76%) the intensity of their dizziness symptoms improved $\geq$20/100 on the visual analogue scale. This supports the effectiveness of the screening process in identifying those with a cervical contribution to their dizziness. These findings are a first step in developing a screening process to identify cervicogenic dizziness.

Keywords: dizziness, manual therapy, randomised controlled trial, cervical spine

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INTRODUCTION

Cervicogenic dizziness is a potentially disabling and ongoing condition with significant impact on the sufferer's quality of life (Reid, Rivett, et al., 2014; Yardley, et al., 1998). There is evidence that manual therapy and a multimodal approach can reduce the signs and symptoms of this condition (Du, et al., 2010; Fang, 2010; Karlberg, Johansson, et al., 1996; Lystad, et al., 2011; Malmstrom, et al., 2007; Reid, et al., 2008; Reid, Rivett, et al., 2014). Despite this evidence, many sufferers of cervicogenic dizziness are not offered treatment, which may in part be due to the difficulty of identifying this condition (Newman-Toker, 2007).

Hoffman et al. (1999) performed a structured review of the literature related to the diagnostic evaluation of dizziness and concluded that a primary care approach, including a careful history and thorough physical evaluation led to a diagnosis in 75% of cases. That said, there is often confusion and the rate of misdiagnosis has been reported to be as high as 50% (Newman-Toker, 2008; Newman-Toker, et al., 2007). One of the problems is that more than one type or cause of dizziness can be experienced concurrently (Newman-Toker, et al., 2007).

To date the management of cervicogenic dizziness has been challenging and arguably poor, with some authors even suggesting the condition does not exist (Brandt, 1996b). There is a clear need to better identify people with this disabling disorder so they can receive appropriate treatment. As there is no single 'gold standard' clinical test for diagnosing cervicogenic dizziness, it is necessary to establish a diagnostic process to better identify those with this complaint. Hence, the aim of this paper is to describe the screening process that was used to select participants with cervicogenic dizziness for a randomised controlled trial (RCT) as this may be of assistance to other researchers who want to categorise and isolate this particular type of dizziness for further studies. The process may also benefit clinicians in recognising this type of dizziness so they can offer evidence-based treatment.

METHODS

Participant recruitment and screening

Potential participants with cervicogenic dizziness were recruited for the RCT via newspaper advertisements and a letter introducing the study to medical practitioners, in the Hunter region of New South Wales, Australia. People who had dizziness described as imbalance, together with a stiff and/or painful neck were asked to contact the researchers if interested in participating in a clinical trial being conducted at the University of Newcastle.

The screening process to identify people with cervicogenic dizziness was developed based on the published literature. A three-step process, comprising a patient screening interview conducted via telephone, examination by a physiotherapist and examination by an oto-neurologist (including vestibular function testing) was followed to identify individuals with cervicogenic dizziness and exclude those who did not have this disorder.

Step 1: Telephone screening

The first step involved telephone screening of potential participants by a physiotherapist. There were five components to the telephone-screening interview (Table 1). The primary purpose of these questions was to distinguish between symptoms consistent with cervicogenic dizziness and symptoms indicative of one of the other types or causes of dizziness such as vertigo, vertebrobasilar insufficiency (VBI), migraines, and dizziness of cardiovascular origin. Questions were first asked about the type and behaviour of dizziness, then cervical symptoms, followed by additional questions related to non-cervicogenic origins of dizziness. Finally, additional study inclusion and exclusion criteria were checked. Questioning was performed in the order listed in Table 1. At any stage that a person met an exclusion criterion, questioning was stopped and the person was excluded.

Table 1: Questions for telephone interview

1a: Questions about dizziness	Include*	Exclude**
Do you have or have you recently had dizziness?	Yes	No
Could you describe what the dizziness feels like?		
Is the dizziness described as imbalance/unsteadiness?	Yes	No
Is the dizziness ever a spinning sensation (vertigo)?	No	Yes
Do you have it if you roll over in bed or get in and out of bed?	No	Yes
How often do you have it?		
How long does it last when you have an attack?		
Is it episodic or persistent?		
Is it worse at certain times of the day?		
Is there any neck movement or position that brings on your dizziness or makes it worse?	Yes	No
Do neck movements or positions make the dizziness worse?	Yes	No
Is there any neck movement or position which eases your dizziness?		
When was the first time you experienced dizziness?		
Was it linked to some incident (trauma/whiplash)?		
1b: Questions about other cervical symptoms		
Do you have a painful neck?	Yes	
Do you have a stiff neck?	Yes	
Do you have pain at the back of the head or other headache?	Yes	
Do you feel the dizziness and neck symptoms are connected, i.e. do they come on at the same time?	Yes	
Do you have or have yourecently had sudden, severe headache or neck pain (check for vertebral artery dissection)		Yes
1c: Exclusion of other types/causes of dizziness		
Check if there are any symptoms or signs of vertebrobasilar insufficiency (VBI) by asking about the 5Ds, 3 Ns, ataxia.		Yes
Have you ever had migraines?		Yes
Do you have anxiety/ panic attacks?		Yes
Do you feel like you may faint?		Yes
Do you have dizziness when you stand up suddenly (light headed, perhaps due to a drop in blood pressure).		Yes
Do you have a past history of stroke, peripheral neuropathy, cerebellar ataxia, Parkinson's disease or other neurological c		Yes
The other general inclusion and exclusion criteria are checked:		
1d: Inclusion criteria:		
18-90 years old	Yes	
has had symptoms three months or more	Yes	
1e: Exclusion criteria:		
The participant should be free of any known conditions which might put them at risk of injury with manual therapy:		
a history of inflammatory joint disease		Yes
spinal cord pathology		Yes
cervical spine infection		Yes
bony disease or marked osteoporosis		Yes
marked cervical spine disc protrusion		Yes
cervical spine cancer		Yes
acute nerve root symptoms (severe pain, weakness, pins and needles or numbness in the arm or hand for less than 6 weeks)		Yes
recent fracture/dislocation of the neck (in the last 3 months)		Yes
previous surgery to the upper cervical spine		Yes
physiotherapy or similar treatment to the neck in the previous month		Yes
pregnancy		Yes
compensable case in dispute		Yes
inability to speak or write in English		Yes

*Include column: Yes = participant included when they answered 'yes' to the item; No = participant included when they answered 'no' to the item; empty cells = decision about inclusion not made based on response to the item.

**Exclude column: Yes = participant excluded when they answered 'yes' to the item; No = participant excluded when they answered 'no' to the item; empty cells = decision about exclusion not made based on response to the item.

History and presence of unsteady dizziness

At the beginning of the telephone screening, it was first established that the potential participant had dizziness. Dizziness is a sensation of disturbed or impaired spatial orientation, without a false sense of motion (Bisdorff, et al., 2009). The person was asked to describe their dizziness to see if they used words consistent with unsteadiness such as poor balance, disequilibrium, being 'wobbly', near falls or a fear they would fall over (Oostendorp et al., 1992). If the dizziness was not described in a manner consistent with unsteadiness, the potential participant was excluded. Questions were then asked about the behaviour of the dizziness; if it was episodic or constant, how often they experienced it, and how long it lasted (Table 1a). Cervicogenic dizziness is usually episodic or intermittent of short duration although it can vary from minutes to hours (Kondratek, 2006; Wrisley, et al., 2000). These questions were asked to obtain a general description of cervicogenic dizziness. For example, if the dizziness was constant, it was more likely to be due to a central cause than cervicogenic dizziness. The potential participant was also asked if the dizziness was worse at certain times of the day to determine if it coincided with the occurrence of other cervical symptoms. An indication of the dizziness severity was sought, as cervicogenic dizziness is generally a less severe form of dizziness compared to vertigo (Kristjansson & Treleaven, 2009).

Exclusion of other types and causes of dizziness

Cervicogenic dizziness is largely a diagnosis of exclusion therefore it was necessary to identify anyone with other types and causes of dizziness so they could be excluded (Heikkila, 2004; Schenk, 2006; Wrisley, et al., 2000). In practice it is not uncommon to have more than one type of dizziness concurrently (Newman-Toker, 2008), but for this study it was necessary to isolate those who had cervicogenic dizziness exclusively.

i) Vertigo

Questions were asked (Table 1a) to identify whether the potential participant experienced a feeling of self-motion that was a false spinning sensation (vertigo)

(Bisdorff, et al., 2009; Hanley, et al., 2001; Wrisley, et al., 2000). Other descriptors of vertigo included swaying, rolling, rocking, tilting or bobbing (Bisdorff, et al., 2009). This sensation is often severe and disabling, and may be accompanied by nausea and vomiting. Benign Paroxysmal Positional Vertigo (BPPV) is the most common cause of vertigo (20-30% of all vertigo) and was identified by asking if the dizziness was triggered by rolling over in bed or getting in/out of bed (Table 1a) (Borg-Stein, et al., 2001; Caplan, 1996). BPPV typically has a 3-10 second latency, in contrast to cervicogenic dizziness, which has no latency period (Baloh and Honrubia, 1990; Caplan, 1996). Attacks of vertigo generally last less than 30 seconds, although they may last for up to several minutes (Oostendorp, et al., 1999; Wrisley, et al., 2000).

iii) Vertebro-Basilar Insufficiency (VBI)

To identify anyone with VBI, questions were asked about the presence of symptoms or signs of VBI including the classic '5 Ds' (dizziness, diplopia, dysarthria, dysphagia, drop attacks), '3 Ns' (nausea, numbness, nystagmus), and ataxia (Table 1c) (Aspinall, 1989; Magarey, et al., 2004). It is rare for VBI to present with only one symptom as it usually presents as a cluster of symptoms (Kerry, Taylor, Mitchell, & McCarthy, 2008), so dizziness without other symptoms is unlikely to be caused by VBI (Caplan, 1996).

Whether the dizziness onset was accompanied by sudden severe neck pain or headache was also asked (Table 1b) in an attempt to identify a particular sub-group of VBI that is caused by vertebral artery dissection (Thomas & Rivett, 2011). With vertebral artery dissection, the initial non-ischaemic (local, somatic) symptoms of ipsilateral posterior neck pain and/or occipital headache are usually followed by the ischemic signs and symptoms as the dissection, and therefore the occlusion, of the vertebral artery progresses (Taylor & Kerry, 2010). There can be a delay in the presentation of ischemic symptoms for up to 14 days after the dissection commences.

iv) Migraine

Each potential participant was asked if they had ever suffered migraines. Migraines are often accompanied by dizziness so anyone suffering from migraines must be excluded for a diagnosis of isolated cervicogenic dizziness. Migraine is usually accompanied by nausea/vomiting and/or photophobia or phonophobia (Whitney, et al., 2000). Migraine associated with vertigo has been reported in 32% of patients presenting to an otology practice (Whitney, et al., 2000). Occasionally, the diagnosis of migraine becomes confusing if the individual has a migraine presenting as dizziness without a headache (Furman & Whitney, 2000).

v) Cardiovascular causes of dizziness

If the dizziness was described as light headedness or pre-syncope (impending faint) further questions were asked to exclude those with a cardiovascular cause from impaired cerebral perfusion in conditions such as postural hypotension, vasovagal attacks, impaired cardiac output or cardiac arrhythmia (Table 1c) (Drachman & Hart, 1972; Newman-Toker, et al., 2008). Common cardiovascular red flags sought included syncope, chest pain and dyspnoea (Newman-Toker 2008).

vi) Other causes of unsteadiness

Once it was established that the dizziness symptoms were consistent with cervicogenic dizziness, it was necessary to eliminate other possible reasons for reported imbalance by asking about a past history of neurological conditions such as stroke, spinal cord pathology, peripheral neuropathy, cerebellar ataxia and Parkinson's disease (Table 1c).

Presence of neck pain and/or stiffness

Cervicogenic dizziness is usually accompanied by symptoms of neck pain, discomfort or stiffness, and/or occipital pain or headache (Table 1b) (Schenk, 2006; Wrisley, et al.,

2000). People who have BPPV, cardiovascular dizziness or psychogenic dizziness would usually not report related neck pain or stiffness.

A question was asked about the first time the person experienced the symptoms of neck pain and dizziness, and whether it was caused by trauma. This information was used to further support a link between the cervical spine and dizziness because there are two main presentations of cervicogenic dizziness: traumatic or insidious onset (Wrisley et al., 2000). Those with a traumatic cause may have suffered a neck injury such as a whiplash injury or a blow to the top of the head (Wrisley et al., 2000). Cervicogenic dizziness can present days to months after a neck injury (Pfaltz, 1984; Wrisley, et al., 2000). In non-traumatic cases with an insidious onset, the dizziness presents with a slow onset of milder pain, morning stiffness and decreased neck movement and is often associated with degeneration of the cervical spine (possibly evident on X-rays) (Ryan & Cope, 1955).

Cervical movements or positions trigger dizziness

It was necessary to establish whether the dizziness or unsteadiness was triggered by cervical movements or positions, consistent with dizziness of cervical origin (Table 1b). The most common neck movement to elicit dizziness in cervicogenic dizziness is cervical extension (Mulligan, 1999; Reid, et al., 2008) including activities such as looking up or hanging the washing on the line. Sometimes the dizziness is only elicited following sustained neck positions, such as painting a ceiling or changing a light bulb. The second most common provocative movement is cervical rotation (Reid, et al., 2008).

Step 2: Physiotherapist physical examination

If the potential participant was not excluded after the telephone screening, a physical examination by a physiotherapist was undertaken to test for the presence or absence of dysfunction in the upper cervical spine consistent with cervicogenic dizziness (Table

2). This examination was designed to exclude those who had no signs of stiffness or symptoms of pain or dizziness produced on examination of the neck.

Table 2: Physical examination by the physiotherapist and related study inclusion/exclusion criteria for participants

Test	Include*	Exclude**
Active cervical spine ROM		
Pain/spasm on palpation of cervical muscles	Yes	
PAIVM testing O-C3		No signs
a) Central PA pain or stiffness	Yes	
b) Unilateral PA pain or stiffness	Yes	
Dix-Hallpike manoeuvre positive		Yes

ROM = range of motion; PAIVM = passive accessory intervertebral movement testing; PA = posterior to anterior

*Include column: Yes = participant included when they answered 'yes' to the item

**Exclude column: Yes = participant excluded when they answered 'yes' to the item

Cervical active movements

First, cervical active movements into flexion, extension, left and right rotation and left and right lateral flexion were tested to detect limitations indicative of cervical spine dysfunction. The presence of any dizziness and/or pain during this testing was recorded. There have been reports of cervical movements being restricted in people with cervicogenic dizziness (Bjorne & Agerberg, 2003; Reid, Callister, Katekar, & Rivett, 2014b), although Malmstom et al. (2007) found a normal or greater range of motion compared to age matched normative values.

Cervical spine palpation and accessory movement testing

Palpation and passive accessory intervertebral movement testing (PAIVM) (centrally to the spinous process and unilaterally to the articular pillar) were performed to the upper cervical spine (occiput to C3) to determine if there were any signs of abnormality (Maitland, 1977). Reproduction of pain or dizziness, or a feeling of restricted joint movement was considered a positive sign and consistent with cervicogenic dizziness (Malmstrom, et al., 2007; Reid, et al., 2008). A finding of local muscle tenderness or tightness of the dorsal neck muscles was also considered to be consistent with cervicogenic dizziness (Hinoki, 1985; Kondratek, 2006; Malmstrom, et al., 2007; Wrisley, et al., 2000). Any findings on cervical spine palpation and PAIVM testing were considered consistent with a cervical problem.

Dix-Hallpike manoeuvre

The Dix-Hallpike manoeuvre was performed to identify those with BPPV who were not already excluded with telephone questioning (Halker, Barrs, Wellik, Wingerchuk, & Demaerschalk, 2008; Wrisley, et al., 2000). This is a test for positional nystagmus, induced by taking the patient rapidly from the erect long-sitting position into left or right cervical rotation (45 degrees) and then into the supine head hanging position (Bronstein, 2003). People were excluded if they demonstrated the typical nystagmus of BPPV: high frequency, torsional, latent, fatigable and reversible (Halker, et al., 2008). In cases of cervicogenic dizziness this test may elicit dizziness due to cervical movement, but will not produce nystagmus (Halker, et al., 2008).

Eye movement tests

To further exclude peripheral or central nervous system sources of dizziness, both the smooth visual pursuit movement and the vestibular-ocular reflexes were tested to identify visually-induced dizziness or vestibulo-visual symptoms (Baloh & Honrubia, 1990; Bisdorff, et al., 2009).

Smooth visual pursuit movements are slow tracking movements of the eyes designed to keep a moving stimulus on the fovea. These movements are under voluntary control because the person can choose whether or not to track a moving stimulus. This movement is assessed by the ability of the participant to track a slowly moving target being moved up and down and side to side. The examiner looks for asymmetry of eye movement or catch-up saccades as this may indicate a cerebellar lesion (Huijbregts & Vidal, 2004).

The vestibulo-ocular reflex is important for stabilizing vision during head movement.To test this, the participant was seated and attempted to maintain their gaze on a stationary target while performing small oscillations of the head side-to-side and up-and-down. Abnormal responses such as an inability to maintain the gaze for 60 seconds due to dizziness, blurred or double vision, may indicate peripheral or central nervous system dysfunction (Huijbregts & Vidal, 2004). If there was a positive result with either of these tests the person was excluded from the study.

Blood pressure

Blood pressure was measured to check for postural hypotension as this is a common reason for light headedness and dizziness (Huijbregts & Vidal, 2004). It has also been suggested by Taylor and Kerry (2013) that blood pressure recording is an important objective test prior to assessment and treatment with manual therapy, especially where dizziness is reported. Blood pressure was measured in sitting and immediately after rising to standing. A normal blood pressure response (reduction in systolic blood pressure < 20 mmHg and/or a reduction in diastolic BP < 10 mmHg) on standing would indicate that cardiogenic syncope is an unlikely cause of the dizziness (Huijbregts & Vidal, 2004). If a potential participant was found to have postural hypotension or abnormally high or low high blood pressure they were excluded from the study.

Step 3) Examination by an oto-neurologist

Those who were not excluded by telephone screening and the physiotherapist assessment then underwent a clinical examination by an oto-neurologist to exclude other non-cervical causes of dizziness. This examination included tests for vestibulo-spinal function, the vestibulo-ocular system, disequilibrium using gait and balance testing, evaluation of cerebellar, pyramidal and extrapyramidal motor systems, eye movement examination including smooth pursuit and saccades, and a comprehensive search for posterior or horizontal canal BPPV, positional or gaze evoked nystagmus, and bedside head impulse tests. Detailed peripheral vestibular function testing was performed, including computerised infrared electro oculography, caloric studies, video head impulse tests, cervical Vestibular Evoked Myogenic Potentials (cVEMPs) and ocular Vestibular Evoked Myogenic Potentials (oVEMPs). If a potential participant was found to have any central or peripheral vestibular cause for their dizziness, they were excluded from the study.

After this series of rigorous clinical assessments, the remaining participants were considered to have a confirmed diagnosis of cervicogenic dizziness and were eligible to enter the RCT.

Brief summary of the RCT

Detailed methods of the interventions and outcome measures for this RCT have been reported previously (Reid et al., 2012). Participants identified as having cervicogenic dizziness were randomly allocated to receive either Mulligan Sustained Natural Apophyseal Glides (SNAGs) including self-administered SNAGs, Maitland passive joint mobilisation (PJM) plus cervical spine range of motion exercises, or a placebo intervention of deactivated laser (Reid, et al., 2012). Outcome measures included self-report questionnaires to assess dizziness intensity (a 100-mm visual analogue scale VAS), dizziness frequency, handicap (measured with the Dizziness Handicap Inventory), pain intensity (a 100-mm VAS) and global perceived benefit. Other physical outcome measures were cervical range of motion, head repositioning accuracy and balance.

RESULTS

Six hundred and eighty three individuals responded to recruitment strategies between April 2010 and December 2011 (Figure 1) (Reid, Rivett, et al., 2014). The structured telephone screening resulted in 71% (n=482) of potential participants being excluded (Table 3). Reasons for exclusion were rotatory dizziness (39 %, n=189), not having neck pain or stiffness (18 %, 88), symptoms consistent with cardiovascular dizziness (11 %, 54), a history of migraines (10 %, 49), having a central cause for dizziness (9 %, 43), medical problems for which manual therapy was not indicated (7 %, 34), symptoms of anxiety/disorientation (3%, 13) and suspected VBI (1 %, 4). Eight (2%) people recovered spontaneously. Ten (1%) people declined to participate.

After the physical examination by a physiotherapist, a further 54 (8%) potential participants were excluded (Reid, Rivett, et al., 2014). The most common reasons were co-morbidities, absence of a related cervical problem, and a diagnosis of BPPV on testing with the Dix-Hallpike manoeuvre.

Fifty-one potential participants (7%) were excluded by examination (including vestibular function testing) by the oto-neurologist. The most common reasons for being excluded by the oto-neurologist were having other neurological conditions, exhibiting signs or symptoms of BPPV, or co-morbidities (Figure 1).

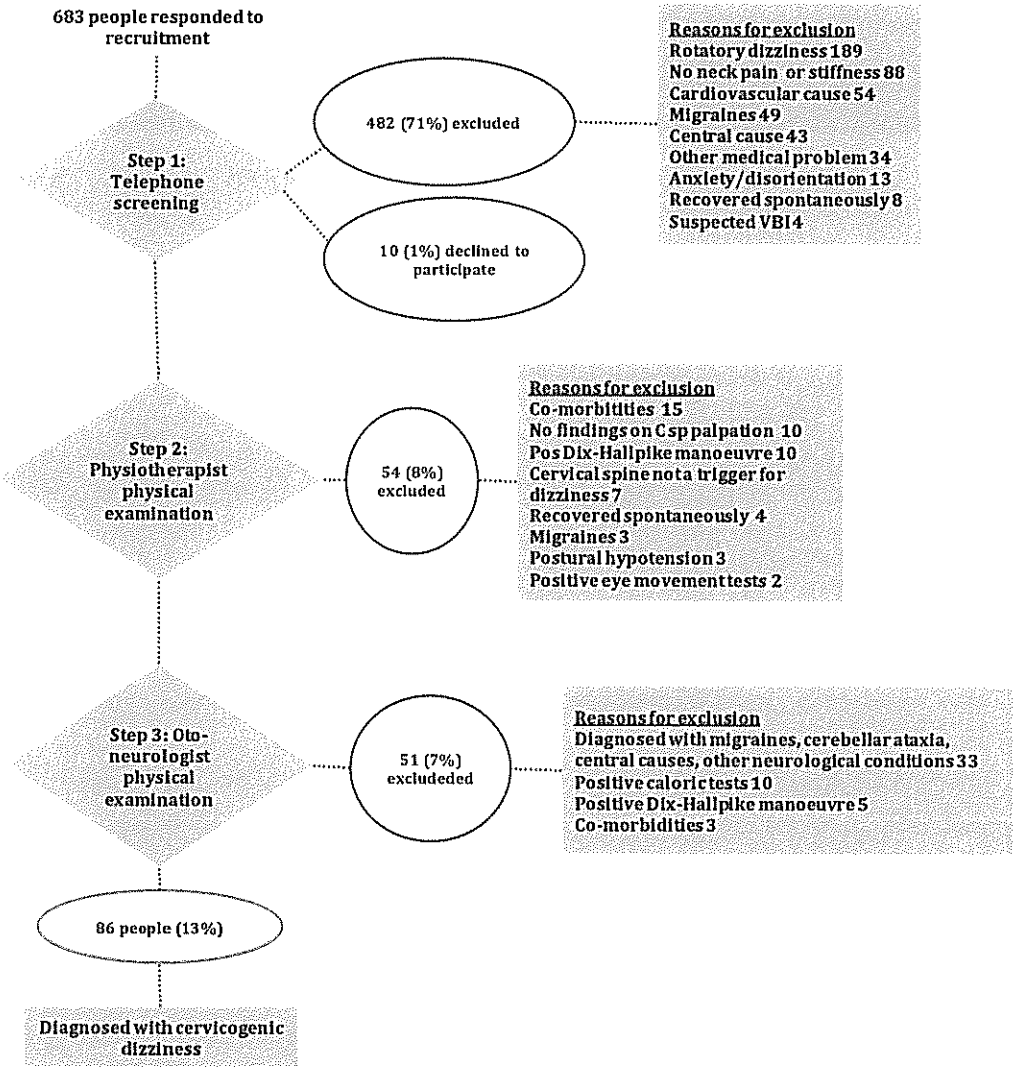

Figure 1. Results of implementing the screening process for participation in the RCT

Eighty-six (13% of the initial response) people were identified as having cervicogenic dizziness with no other forms of dizziness or contraindications to manual therapy. Those identified with cervicogenic dizziness had generally experienced their dizziness for many years (mean 7.0 ± 6.7 years, range 3 months to 30 years), with 66% having an insidious and 34% a traumatic onset of symptoms. The mean intensity of reported dizziness on a visual analogue scale was 47/100 ± 23. The mean age was 62 ± 13 (range 21-85) years and 50% were female (S Reid, Rivett, et al., 2014).

Outcomes of the RCT

Fifty-eight participants in this RCT, who were regarded as having cervicogenic dizziness and no other form of dizziness, received manual therapy to the upper cervical spine and had a favourable response with significantly greater improvements in dizziness observed with manual therapy than placebo. After the manual therapy interventions (either SNAGs or PJMs), 44 participants out of the 58 (76%) reported a decrease in intensity of dizziness of ≥ 20mm on the 100mm VAS. This change in the VAS is considered a clinically significant change (Bird & Dickson, 2001). Immediately post-treatment there were significant ($p<0.05$) reductions in the mean values of intensity of dizziness for both manual therapy intervention groups compared to baseline values and this was maintained for 12 weeks (Reid, Rivett, et al., 2014). The intensity of dizziness measured on a 100mm VAS decreased from 43.3 (SD 21.9) at baseline to 22.3 (SD11.5) post-treatment for the SNAG group, and from 50.3 (SD 21.2) to 27.8 (SD14.5) 36.9) for the PJM group (Reid, Rivett, et al., 2014). Frequency of dizziness also decreased in both manual therapy groups from dizziness experienced daily or up to 4 times a week at baseline to dizziness experienced 1 to 4 episodes a month after treatment (Reid, Rivett, et al., 2014). These reductions in dizziness intensity and frequency observed following manual therapy treatment to the upper cervical spine support the hypothesis that the cervical spine was contributing to their dizziness and suggest that the screening process was successful in identifying those with a cervical cause of their dizziness.

DISCUSSION

A screening process is described that distinguishes people who have dizziness primarily of cervical origin from those with dizziness from other causes. This screening process was implemented and used to identify individuals with symptoms and signs consistent with cervicogenic dizziness who then participated in a RCT assessing treatment of this condition. From this screening process 13% of the people who answered a call for participants having neck pain and unsteady dizziness were identified as having cervicogenic dizziness. The main reasons for exclusion were

having co-morbidities, having BPPV, not having a cervical spine disorder, and being diagnosed with other neurological conditions.

The procedure described in this manuscript may be useful in identifying people with cervicogenic dizziness and not another type or cause of dizziness. Those with vertigo, particularly BPPV, were excluded at each of the three steps of the assessment process, first by telephone questioning and then when the Dix-Hallpike manoeuvre was performed by the physiotherapist. As this test can have false negatives, when it was repeated in the third step by the oto-neurologist more people were identified as having BPPV, and further still with peripheral vestibular function testing.

This screening process may be of interest to other researchers or to clinicians to assist in identifying individuals with cervicogenic dizziness who may potentially benefit from treatment with manual therapy to the cervical spine.

The researchers acknowledge that the examination presented here is not a complete examination that an oto-neurologist would use to examine someone with dizziness, nor is it the full musculoskeletal examination that would typically be conducted by a physiotherapist on someone with nonspecific neck pain. It is an outline of the key components of a procedure that was used to recruit participants with cervicogenic dizziness for the purpose of a clinical trial. As there is no single 'gold standard' test to identify cervicogenic dizziness, this screening procedure could possibly be further developed and validated to become a formal screening tool to help identify those with this disorder.

CONCLUSION

This study has outlined a successful screening process used in a RCT to identify people with cervicogenic dizziness and to exclude those with other dizziness disorders.

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Chapter 8 Comparison of cervical range of motion, head repositioning accuracy and balance in people with cervicogenic dizziness to normative values

8.1 Background

In the previous chapter, the process that was used to identify people with chronic cervicogenic dizziness for inclusion in this RCT was detailed. To further describe this condition, some of the physical attributes of the participants diagnosed with cervicogenic dizziness that were measured in this study will now be further analysed as this will help to characterise this population and may aide in their identification. To date, the characteristics of patients with suspected cervicogenic dizziness have not been extensively described in the literature, which may contribute to it being a poorly understood condition (Malmstrom, et al., 2007). For cervicogenic dizziness to be recognised and appropriately treated, a detailed description of the condition needs to be documented so that clinicians are aware of the signs and symptoms and can confidently diagnose patients with this condition. If diagnostic guidelines could be established, it might enable practitioners to better differentiate between people with cervicogenic dizziness and those with migraine, cardiovascular dizziness, central or peripheral vestibular dysfunction (such as BPPV), and the more serious conditions of VBI, craniocervical arterial dissection and stroke. In this chapter, the findings for the physical measures of cervical spine ROM, head repositioning accuracy (as a measure of proprioception) and balance in people with cervicogenic dizziness are compared to published normative data to determine if this sample differs from asymptomatic people.

8.1.1 Cervical ROM

In order to inform this comparative analysis it is useful to review what is known about these measures. Previous studies have reported that patients with cervical spine dysfunction (generalised neck pain) have reduced cervical range of motion (ROM) compared to healthy controls (Sjölander, et al., 2008; Woodhouse & Vasseljen, 2008). Several studies have described patients with whiplash-associated disorder (WAD),

who often complain of dizziness as well as neck pain, and were found to have decreased ROM compared to asymptomatic controls (Dall'Alba, Sterling, Treleaven, Edwards, & Jull, 2001; Woodhouse & Vasseljen, 2008).

There have been very few studies of patients with cervicogenic dizziness. Wrisley et al. (2000) report in two case studies that one participant (a 49 year-old female) had normal ROM while the other (another 49 year-old female) had 25% of normal cervical flexion, extension, right rotation and right lateral flexion range, and 50% of normal left rotation and left lateral flexion. In a study of elderly patients over 65 years of age with dizziness (n=149) where 98 (66%) were believed to have cervical spondylosis, the patients with dizziness had significantly limited neck movement when compared to healthy controls (n=97), although this was not measured or quantified (Colledge, et al., 1996). In contrast to the above papers that report a decrease in cervical ROM, Malmstrom et al. (2007) in a study of 22 patients (mean age 37 years, range 25-49) with cervicogenic dizziness reported the patients had normal or greater cervical ROM than age and gender matched controls. Due to the younger age of the participants in the study by Malmstrom et al., they may not have had cervical spine degeneration, which could explain the lack of hypomobility. They do however report some reduced ROM at the cervico-thoracic junction.

8.1.2 Head repositioning accuracy

Cervical proprioception has great importance for postural control and is thus of interest when describing a population with unsteadiness and cervical spine dysfunction (Roren, et al., 2009). Cervical proprioception has commonly been measured using a test of head repositioning accuracy (Chen & Treleaven, 2013; Malmstrom, et al., 2007; Revel, et al., 1991). The test is a measure of the ability to relocate the head to a target position after active cervical spine movement such as rotation, flexion or extension. Previous studies have reported that patients with cervical spine pain have greater errors in head repositioning accuracy compared to healthy controls (Kristjansson, 2004; Kristjansson, et al., 2003; Revel, et al., 1991; Sjölander, et al., 2008; Treleaven, Jull, & Sterling, 2003; Treleaven, et al., 2008). This suggests that people with cervical spine pain have proprioceptive deficits in their necks. A case controlled study was performed by Kristjansson et al. (2003) to compare

head repositioning accuracy between patients with a traumatic onset of neck pain (for example, due to WAD) (n=22), insidious onset neck pain (n=20) and asymptomatic subjects (n=21) when targeting the neutral head position (NHP) and complex predetermined positions. They established that head repositioning error exists in patients with neck pain, with a trend (p=0.07) suggesting a greater deficit in WAD patients compared to those with insidious onset neck pain (Kristjansson, et al., 2003). Perhaps this study was underpowered to detect the influence of pain versus the influence of injury (traumatic or non-traumatic) on head repositioning accuracy. A study by Rix and Bagust (2001) found no difference between those with neck pain and healthy subjects but only had 11 people in each group.

Only one previous study compares head repositioning accuracy results for cervicogenic dizziness patients to asymptomatic controls (Heikkila, et al., 2000). They found participants with cervicogenic dizziness (n=14, mean age 36 years) had significantly worse ($p<0.001$) head repositioning accuracy in all directions (flexion, extension and left and right rotation) compared to asymptomatic controls of similar ages (mean age 35 years). This was a robust study with the participants carefully screened to identify those with cervicogenic dizziness and exclude those with extra-cervical causes of their dizziness.

8.1.3 Balance

Participants with neck pain have been found to have larger postural sway (poorer balance) in standing compared to healthy controls (Field, et al., 2008; McPartland, et al., 1997; Poole, et al., 2008; Stokell, et al., 2011). Larger postural sway was reported in participants with higher pain intensities (Ruhe, Fejer, & Walker, 2013). These findings appear to suggest that neck pain may alter the afferent input from the proprioceptors in the joints and muscles in the cervical spine, resulting in poor head on trunk orientation and deficits in balance (Kristjansson & Treleaven, 2009). An observational study of 40 women over the age of 65 years by Poole et al. (2008) using computerised dynamic posturography, demonstrated a significant ($p < 0.05$) increase in postural sway in the anteroposterior direction in participants with insidious neck pain of greater than 3 months (n=20) compared to healthy controls (n=20). The study included medication use as a co-variant so that it did not distort the findings as greater

medication use in the pain group may have adversely affected their balance. The study may have been underpowered as there were trends for increased sway in all the static standing tests in the neck pain group, but it only reached significance in two of the eight tests. In a study of patients with persistent whiplash, Treleaven et al. (2005b) report greater sway in those with dizziness (n=50) than those without (n=50), which lead them to conclude that the postural disturbances possibly originated from abnormal cervical afferent input. The study did not perform any vestibular assessment and so did not exclude those who may have vestibular pathology which sometimes occurs after a WAD injury (Treleaven, et al., 2005b).

Greater unsteadiness has been described for people with a traumatic onset of neck symptoms compared to those with an insidious neck pain onset (Field, et al., 2008; Kristjansson, et al., 2003; Roijezon, et al., 2011). An observational study by Field et al. (2008) directly compared 30 participants with insidious neck pain, to 30 with WAD and 30 controls. It was found that participants with WAD had a larger postural sway compared to participants with insidious neck pain in standard and narrow stances with eyes opened and closed (Field et al., 2008). Both the participants with WAD and those with insidious neck pain had a larger postural sway compared to healthy controls.

There have been very few studies that have compared balance in participants diagnosed with cervicogenic dizziness to normative data. Karlberg et al. (1996) measured balance deficits in patients with cervicogenic dizziness and showed that patients with cervicogenic dizziness could be distinguished from healthy subjects ($p<0.001$) and from those with vestibular pathology ($p<0.001$) using posturography (Karlberg, Johansson, et al., 1996).

Thus, there is evidence that people with cervical spine pain and those with whiplash-associated disorders have deficits in cervical ROM, head repositioning accuracy and balance compared to controls. However there is a lack of information about these physical attributes for the cervicogenic dizziness population.

Hence, the aim of this chapter was to determine whether this sample with established cervicogenic dizziness have differences in cervical ROM, head repositioning accuracy

and balance compared to published normative data. This should assist in describing the characteristics of people with this condition.

8.2 Method

After a call for participants and a thorough screening process (described in Chapter Seven), 86 participants who were determined to have cervicogenic dizziness and no other co-existing cause for their dizziness entered the RCT that is the basis of this thesis. The mean age of participants was 62 years (SD 12.7, range 21-85 years), 50% were female, and 29% had a traumatic onset.

Physical outcome measures that were assessed at baseline included:

i) Cervical ROM measured with a Cervical Range of Motion (CROM) device (Performance Attainment Associates 12805 Lake Blvd, Lindstrom, Minnesota. USA). The maximum ranges of cervical flexion, extension, left and right rotation, and left and right lateral flexion were measured three times and averaged.

ii) Head repositioning accuracy assessed using the CROM device. The blindfolded participant was seated with their head in the neutral starting position. They were asked to actively move to the midpoint of their maximum left rotation range (as previously measured by the research assistant using the CROM device) with verbal feedback from the research assistant that they were at the mid-position, which was called the 'target position'. They were asked to memorise this position so they could find it again after a head movement. After returning to the starting position, they were then asked to rotate their neck to the left and find the 'target position' without verbal feedback. The difference between the target position and the achieved position was recorded three times and averaged. This was then repeated into the right rotation. In asymptomatic individuals, 2-2.5 degrees error has been reported with this test, and the mean error was found in previous studies to be >4.5° for 89% of neck pain patients (Revel, et al., 1991; Treleaven, 2008a; Treleaven, Jull, & Sterling, 2003; Woodhouse & Vasseljen, 2008).

iii) Balance was assessed using the Chattecx Balance Dynamic System (Serial No 1001, Chattecx Corporation, 101 Memorial Dr, Chattanooga, Tennessee. USA). The Chattecx posturography system produces a measure of postural sway called the sway

index (measured in centimeters). Balance was measured during static standing with the head in the neutral position (eyes open); with the head in the neutral position (eyes closed); with the neck extended; with the neck in left and right rotation; and on a moving platform (NHP, eyes open). The SEM for the Chattecx system has been reported to be 0.06cm (95% CI 0.16-0.40) for static standing (eyes open), 0.26 cm (0.13-0.87) for static standing (eyes closed) and 0.32cm (0.36-1.6) for eyes open on a moving platform (Mattacola, Lebsack, & Perrin, 1995) .

8.2.1 Statistical Analysis

Mean, standard deviation and 95% confidence intervals were calculated for the outcome measures. Baseline outcome measures were compared using independent sample t-tests to published normative data using the average of male and female norms when both were available. In the current study, 50% of participants were female.

8.3 Results

Participants with chronic cervicogenic dizziness were found to have differences in their cervical ROM, head repositioning accuracy and balance compared to normative data.

8.3.1 Cervical ROM

The mean values for ROM at baseline are presented in Table 8.1. When these mean values were compared to published normative values for a similar age group (60-69 years) (Youdas, Garrett, Suman, & Bogard, 1992), it was found that those with cervicogenic dizziness had significantly less ROM in all directions (Table 8.1).

8.3.2 Head repositioning accuracy

Participants of the present study, who had all been identified as having chronic cervicogenic dizziness were found to have mean head repositioning errors of 5.2° (SD 3.9) into left rotation and 4.8° (SD 4.6) into right rotation (Table 8.1). These errors in head repositioning accuracy were significantly greater in participants with cervicogenic dizziness than for asymptomatic individuals (mean age 34.1 yrs ±1.8 years; 66% female), where 2-2.5° error has been reported (Table 8.1) (Treleaven, Jull et al. 2003).

8.3.3 Balance

The results for testing balance at baseline are reported in Table 8.1. It was only possible to find published mean values (Mattacola, et al., 1995) for the sway index measured with the Chattecx Balance Dynamic System for asymptomatic normals in static standing with eyes open and closed. When the mean values for participants with cervicogenic dizziness were compared to these published normative data (mean age 24.7 years ± 3.3 years; 83% female), those with cervicogenic dizziness were found to have increased sway during double leg static standing with both eyes open and closed (Table 1) (Mattacola, et al., 1995). Post hoc analysis revealed that when testing balance with eyes closed, those participants with a history of trauma (n=29) had a mean sway of 0.72 cm that was significantly worse ($p<0.05$) than those without trauma (n=57) who had a mean sway of 0.59 cm.

Table 8.1. Comparison of participant characteristics at baseline to published data for asymptomatic individuals. Data are presented as means (SD) or N (%).

Characteristic	Study mean	Normative value	P value*
Cervical ROM (degrees)§			
Extension	45.2 (13.0)	61.3 (11.9)	<0.0001
Flexion	38.3 (13.7)	52 (8.6)	<0.0001
Left rotation	50.3 (12.8)	58.2 (7.9)	<0.0001
Right rotation	48.8 (13.4)	59.4 (8.6)	<0.0001
Left lateral flexion	29.6 (9.3)	32.4 (6.4)	<0.0001
Right lateral flexion	26.2 (10.4)	31.3 (7.5)	<0.0001
Head repositioning accuracy (degrees)§			
Rotation (left)	5.2 (3.9)	2 (0.2)	<0.0001
Rotation (right)	4.8 (4.6)	2.5 (0.2)	<0.0001
Balance (sway index, cm)§			
Eyes open	0.37 (0.22)	0.28 (0.11)	0.0003
Eyes closed	0.64 (0.37)	0.37 (0.23)	<0.0001

ROM= range of motion ;* significance = $p < 0.05$

§Normative values using cervical range of motion device (CROM) (Youdas, et al., 1992), head repositioning error (Treleaven, Jull, & Sterling, 2003), balance (Chattecx system) (Mattacola, et al., 1995)

8.4 Discussion

Participants with a diagnosis of chronic cervicogenic dizziness were found to have decreased cervical ROM, head repositioning accuracy and balance compared to asymptomatic individuals. This is an important finding because the characteristics of this group of patients have not been extensively described in the literature. This is the first time these three measures have been reported in the same cohort of cervicogenic dizziness patients. It is hoped this may add further information that could provide a better understanding of patients with cervicogenic dizziness and may assist in recognition of this condition.

The study found that participants with chronic cervicogenic dizziness have decreased cervical ROM compared to asymptomatic individuals of a similar age. This is in agreement with the majority of previous studies, which found that ROM was reduced in patients with cervical spine dysfunction of both insidious onset and with WAD (Sjolander et al. 2008; Dakll'alba et al. 2001; Woodhouse 2008). However the results are in contrast to those of Malmstom et al. (2007) who report that in a group of 22 people with suspected cervicogenic dizziness, ROM was equal to or larger than age matched values. These differing results may be because participation in the study by Malmstrom et al. was limited to people aged 55 years and younger (mean age 37 years, range 25-49 years), whereas in the current study the mean age was 62 years (SD 12.7, range 21-85 years).

Further, this study demonstrated that people with chronic cervicogenic dizziness have deficits in head repositioning accuracy when compared to asymptomatic individuals. This is in agreement with Heikkila et al. (2000) who also report increased errors in those with cervicogenic dizziness. Most previous studies of patients with neck pain have reported larger head repositioning errors in patients with neck pain compared to healthy controls (Kristjansson et al. 2003; Treleaven et al. 2003; Kristjansson 2004; Sjolander et al. 2008; Treleaven et al. 2008) however Rix & Bagust (2001) only found a difference into cervical flexion so the results to date are inconclusive. The conflicting results may be related to the varied study methodologies including differing measurement methods, or to different study populations including varying age groups and types of neck pain (insidious onset versus traumatic onset). There are indications

the deficit in head repositioning accuracy is greater in patients with traumatic onset of neck pain (e.g. WAD) rather than insidious onset (Kristjansson, et al., 2003; Sjölander, et al., 2008).

The present investigation also found that people with cervicogenic dizziness had poorer balance with both eyes open and closed compared to asymptomatic individuals. This is in agreement with previous studies of neck pain patients. Since the sample for this study had a mean (SD) age of 62 (13) years, which is older than the population in many other studies (Sjostrom, et al., 2003; Stokell, et al., 2011; Treleaven, et al., 2006), it could be argued perhaps that aging contributed to poor balance rather than cervical spine dysfunction. However Poole et al. (2008) reported that an elderly population (65 years or older) with neck pain had greater balance and gait impairments than asymptomatic elderly patients, suggesting that neck pain further compromises disturbances in balance that occur with normal aging. The balance tasks that were assessed in the current study were mainly static posturography tests. In future studies, dynamic balance tests could be used as these are thought to be more provocative and challenging and may better expose balance deficits in the cervicogenic dizziness population compared to asymptomatic individuals (Alund, et al., 1993).

By measuring, for the first time, the three physical outcome measures (cervical ROM, head repositioning accuracy and balance) in the same sample of participants with cervicogenic dizziness and showing they are all reduced compared to normative values, this study opens discussion as to the hypothesis of the cause of cervicogenic dizziness. As dicussed in Chapter Two, these three outcomes measures have been linked with cervical spine dysfunction. It has long been theorised that the most likely cause of unsteadiness and sensations of dizziness in patients with cervicogenic dizziness is a pertubation of cervical afferent input from cervical joint and muscles proprioceptors (Brandt & Bronstein, 2001; de Jong, et al., 1977; Terrahe, 1979; Wyke, 1979). The findings in this study suggest that since there is decreased cervical ROM compared to normal, there could be perturbation of afferent feedback to the central nervous system from cervical joints and muscles. There is also evidence in this study of decreased proprioceptive feedback from joints and muscles, demonstrated by significant differences in head repositioning accuracy compared to normal

asymptomatic people. This abnormal sensory afferent input from cervical proprioceptors could also lead to a mismatch with inputs from the vestibular and visual systems and result in unsteadiness or poor balance (also demonstrated in this cohort) and sensations of dizziness (Treleaven, Jull, & Sterling, 2003; Wrisley, et al., 2000).

A criticism of the comparison of these physical measures to published normative data is that the results are not compared to individuals matched for age and gender, but to mean values. The mean age of participants in this study was 62.0 years (SD 12.7). For ROM measures it was possible to obtain mean values for a similar age bracket (60-69 years), but for head repositioning accuracy and balance the published normative data is not specific to a similar older age group. The mean ages of the asymptomatic controls in the studies of normative values was 34.1 years (SD 1.8) for head repositioning accuracy, and 24.7 years (SD 3.3 years) for balance using the Chattecx. The effect of aging on head repositioning accuracy is unknown but an age-related decline in balance is well-supported in the literature (Haber, Erbas, Hill, & Wark, 2008). Therefore, not having aged matched controls could be a confounder of these results.

8.5 Conclusion

This is the first time that a population with a clear diagnosis of cervicogenic dizziness has been shown to have deficits in cervical ROM, head repositioning accuracy and balance when compared to published normative values. These findings will help to characterise this population which may provide a better understanding and recognition of this condition. This is also the first time that deficits in all three physical measures have been established in the same sample with established cervicogenic dizziness.

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Chapter 9 Discussion and conclusions

9.1 Overview

This thesis is a series of manuscripts that report the results of a RCT conducted to assess the effects of SNAGs, PJMs and a placebo intervention as treatments for chronic cervicogenic dizziness. It is the first time that the use of PJMs for cervicogenic dizziness have been assessed. The effectiveness of the treatments was evaluated by assessments of self-reported outcomes post-treatment and at 12 weeks (presented in Chapter Four), as well as of physical outcomes (Chapter Five). For the first time, the long-term (12 month) benefits of treatment of cervicogenic dizziness with manual therapy are reported (Chapter Six). A head-to-head comparison was made at each of these time points to determine whether one of these commonly used types of manual therapy is superior to the other for chronic cervicogenic dizziness. There have been no previous RCTs to compare a Mulligan technique to a Maitland technique for cervicogenic dizziness. The process used to identify people with this condition for inclusion in this RCT is described in Chapter Seven, and the results of that selection process discussed. To further aid in the identification of this condition, in Chapter Eight the findings for cervical ROM, head repositioning accuracy and balance in this sample at baseline were compared to published normative values to further describe this group of patients.

9.2 Summary of study findings

The key findings of this thesis were:

- Manual therapy is effective in the short, medium and long term for chronic cervicogenic dizziness
- PJMs were found for the first time to be effective in treating cervicogenic dizziness
- Both forms of manual therapy (SNAGs and PJMs), together with associated exercise advice are equally effective treatments for chronic cervicogenic dizziness
- There was a clinically important improvement after manual therapy of 20mm out of 100mm on a VAS for the main outcome (severity of dizziness) in 76% of participants

- Manual therapy reduced the frequency of cervicogenic dizziness over the medium and long term
- There were marked reductions in disability in this cohort after both types of manual therapy
- There were increases in cervical ROM in both manual therapy groups compared to placebo over the long term (at 12 months); these improvements were observed earlier in the SNAG group
- No adverse effects of treatment with manual therapy for cervicogenic dizziness were reported lasting longer than 24 hours
- There is preliminary support for a clinical screening process for this condition which may have the potential to be developed further
- Participants with cervicogenic dizziness were found to have deficits in their cervical ROM, head repositioning and balance compared to normative data
- The findings provide further indirect evidence for the existence of cervicogenic dizziness.

9.2.1 Hypotheses

The two hypotheses for this thesis were accepted.

1. Manual therapy is better than a placebo intervention for people with chronic cervicogenic dizziness.

2. There is no difference in outcomes for people with chronic cervicogenic dizziness who receive SNAGs (including self-SNAGs) or Maitland PJMs (plus ROM exercises).

9.2.2 Effects of manual therapy on cervicogenic dizziness

The manuscripts in Chapters Four to Eight detail the evidence underpinning these conclusions. The second publication (Chapter Four) reports the short to medium term results from the RCT (post-treatment and at 12 weeks), using self-report measures to assess intensity of dizziness, dizziness frequency, dizziness handicap, intensity of cervical pain and perceived benefit from the interventions. Both the SNAG and PJM

groups had lower dizziness intensity compared to the placebo group immediately post-treatment and at 12 weeks, and less frequency of dizziness at 12 weeks. When an analysis of change over time was performed, both intensity and frequency of dizziness were reduced immediately after both manual therapy interventions compared to baseline and the effects were maintained for 12 weeks, while no change occurred in the placebo group. For dizziness handicap (measured with the DHI), the PJM group had lower scores than the placebo group after treatment and at 12 weeks, but there was no difference between the SNAG and placebo groups. For the DHI scores over time, all three groups improved post-treatment and at 12 weeks, compared to baseline. Those participants receiving either type of manual therapy reported the treatment was more beneficial than did those receiving the placebo, both post-treatment and at the 12-week follow-up assessment. The PJM group also experienced lower pain intensity than the placebo group at 12 weeks. Changes over time showed a decrease in pain scores in all three groups post-treatment and at 12 weeks. There were no differences between the two manual therapy groups in any of the self-reported outcomes except for the lower DHI score in the PJM group compared to the SNAG group at the 12-week follow-up. These results constitute the first documented evidence for the benefits of PJMs in improving the symptoms of chronic cervicogenic dizziness.

The results of this study are consistent with those of a previous study by the authors investigating the effect of SNAGs on cervicogenic dizziness (Reid, Rivett, Katekar, & Callister, 2008). It found significantly reduced dizziness severity, and DHI scores post-treatment and at 6 weeks after SNAGs treatment, compared to a placebo intervention. However in the previous study there was no significant difference in the frequency of dizziness between the SNAG and placebo groups at any of the assessment times (post-treatment, 6 weeks and 12 weeks). The only treatment difference in the current study for the SNAG group was the addition of self-SNAGs from the second intervention session. It is possible that the addition of self-SNAGs was beneficial in reducing the frequency of dizziness, as there was no difference in frequency immediately post-treatment but reduced frequency of dizziness at 12 weeks in the SNAG group compared to the placebo, which suggests it may have been the home treatment that helped. The beneficial findings for manual therapy treatment of cervicogenic dizziness

are supported by the RCTs conducted by Kang, Wang and Ye (2008), Fang (2010), and Du et al. (2010) who all used spinal manipulation and reported improvements in clinical outcomes, such as improved dizziness intensity scores and reduced neck/shoulder pain intensity scores. There is strong evidence from a RCT by Karlberg et al. (1996) that manual therapy as part of a multimodal approach, including soft tissue therapy, stabilisation techniques and body awareness therapy improved dizziness intensity. Other non-RCT studies have shown that 'chiropractic care' (Strunk and Hawk 2009) and cervical manipulation (Heikkila, Johansson et al. 2000) had positive effects on reducing dizziness in people believed to have cervicogenic dizziness, though these studies did not have a control group as they were both single-group studies, nor did they report any follow-up outcome measures.

The effects of SNAGs and PJMs in this RCT over the long term (12 months) are reported in the fourth publication (Chapter Six). Although there was no significant difference in intensity of dizziness between the three groups at 12 months, there were significant decreases for all three groups compared to baseline. The time courses were however different for the two manual therapy groups compared to the placebo. The improvements in both manual therapy groups were evident immediately after the interventions and were then maintained for 12 months, whereas improvement was much slower for the placebo group, and did not reach statistical significance until 12 months post-intervention. At 12 months both manual therapy groups reported lower dizziness frequency scores than the placebo group, and had relatively lower scores on the DHI. There were no differences between the three groups for intensity of cervical pain at 12 months. Participants in both manual therapy groups reported at the 12-month follow-up that they found the treatment more beneficial (measured with the GPE questionnaire) than those who received the placebo. The only other study (Malmstrom, Karlberg, Melander, Magnusson, & Moritz, 2007) measuring medium (6 months) and long-term (2 year) outcomes after multimodal treatment (that included cervical manual therapy) for cervicogenic dizziness used a questionnaire to assess 'How do you feel right now?" (no symptoms, better, same or worse than before treatment) and 'Did the treatment help you?' Sixteen of the 17 participants stated they had benefited from the treatment two years after the interventions (Malmstrom, et al.,

2007). Four patients reported no dizziness and seven 'had improved'. However, there was no quantitative measure of dizziness intensity or frequency, or comparison to a control group at the re-assessment points.

9.2.3 Comparison of manual therapy interventions

Another key objective of the thesis was the head-to-head comparison to determine for the first time, whether Mulligan SNAGs or Maitland PJMs are more effective in treating chronic cervicogenic dizziness. Essentially there were no significant differences in outcomes between the two manual therapy approaches. Post-treatment there was greater cervical ROM in two movement directions in the SNAG group compared to the PJM group. At the 12-week follow-up, there was also a lower DHI score for the PJM group compared to the SNAG group. Other than these two findings, there were no substantial differences in outcomes between the SNAG and PJM groups. This means that both forms of manual therapy are equally effective treatments for this condition. This is the first time these two commonly used physiotherapy interventions have been compared for this condition.

9.2.4 Effects of manual therapy on cervical pain associated with cervicogenic dizziness

There is Level 1 and Level II evidence that manual therapy alone is beneficial in pain reduction in the treatment of mechanical neck disorders (Gross, Aker, Goldsmith, & Peloso, 1996), with PJMs shown to be of short term benefit for neck pain (Hurwitz, Aker, Adams, Meeker, & Shekelle, 1996). However, there is conflicting evidence as to whether there is any benefit for pain in those with cervicogenic dizziness.

In the current study it was not possible to draw a conclusion regarding the effect of manual therapy on cervical pain experienced by the participants with cervicogenic dizziness. The primary aim of this study was to determine the effects of manual therapy on cervicogenic dizziness, and not pain, which was why pain was not used as an inclusion or exclusion criterion. As pain was not a criterion for inclusion in this study, participants with no pain or very low levels of pain were included. Notably, 16 participants (18%) had baseline pain scores of less than 20mm (on a 100mm scale). A VAS score in excess of 30 mm is considered to be moderate pain, and in excess of 54

mm to be severe pain (Collins, Moore, & McQuay, 1997). Some pain studies do not include participants with pain scores of less than 20mm as such a relatively low score is thought to indicate neck discomfort rather than pain (Leaver et al., 2010; Leaver, Maher, McAuley, Jull, & Refshauge, 2012). Because participants were not stratified by neck pain intensity before randomisation, there was a strong trend for a difference in pain scores (p=0.06) between groups at baseline.

In a previous study of the effects of SNAGs on participants with cervicogenic dizziness, there was a significant change in cervical pain post-treatment and at the 6-week follow-up after the SNAG treatment compared to the placebo intervention. In that study the mean VAS scores for cervical pain at baseline were the same for both groups, with a mean VAS score for cervical pain of 4.7 (SD ±2.7) for the SNAG group and 4.7 (±2.2) for the placebo group. This adds support for the possibility that differences in pain scores at baseline between groups may have been a confounder in the current study. The participants in the Karlberg et al. study (1996) also reported reduced cervical pain intensity after multi-modal treatment including manual therapy. In contrast, in a single–group study by Strunk and Hall (2009) patients with suspected cervicogenic dizziness showed no significant change on the NDI, which was used to measure neck pain after two treatments per week for 8 weeks with common chiropractic interventions such as manipulation, soft tissue therapy and heat or cold therapy. Hence, it is not possible to draw a firm conclusion on the effectiveness of these interventions for cervical pain in people with cervicogenic dizziness.

9.2.5 Effects of manual therapy on physical indicators of cervicogenic dizziness

The third publication (Chapter Five) reports the short and medium term (post-intervention and at 12 weeks) results of the RCT in relation to the physical measures of cervical ROM, head repositioning accuracy and balance. Compared to a placebo, the SNAG group had greater ROM in all six cervical spine movement directions after treatment and at 12 weeks. For the PJM group, greater ROM was observed in one direction post-treatment and at 12 weeks compared to the placebo group. When the two manual therapy groups were compared, there was greater movement in two directions for the SNAG group compared to the PJM group post-treatment. When

changes over time were analysed, cervical ROM was shown to increase in participants with cervicogenic dizziness immediately after they were treated with both manual therapy interventions and this increased ROM was maintained at the 12-week follow-up, however those treated with SNAGs showed greater increases than those treated with PJMs at both time points.

It has been hypothesised that by increasing cervical ROM, information from sensory afferents in the cervical spine is normalised, potentially contributing to a reduction in cervicogenic dizziness (Furman & Cass, 1996; Mulligan, 1991; Wilson, 1996). The results of the current study are consistent with this hypothesis, as the increase in cervical ROM of motion occurred alongside a decrease in dizziness intensity. However in the previous study by the authors there were no significant increases in cervical range of motion after the SNAG intervention when compared to the placebo, even though there were decreases in dizziness intensity in the SNAG group compared to the placebo post-treatment and at 6 weeks (Reid, et al., 2008). This may suggest that the introduction of the self-SNAG in the current study, which was introduced at the second therapist session, made a meaningful difference to neck movement. Malmstrom et al. (2007) reported that those people with cervicogenic dizziness who had restricted cervical ROM or cervico-thoracic mobility and were treated with joint mobilisation, were then found to have increased ROM after treatment, although this was not compared to a control group. Manipulation improved ROM in a study of cervicogenic dizziness patients (n=14) by Heikkila et al. (2000), but again this study was not placebo controlled.

There was no conclusive effect for SNAGs or PJMs on head repositioning accuracy or balance at the short term or medium term follow-up in the current study. Heikkila et al. (2000) reported improvements in head repositioning accuracy with both manipulation and acupuncture in their single-group study, but there was no control group or follow up. Similarly, in two single-group studies by Strunk and Hawk (2009) and Hawk and Cambron (2009), patients with suspected cervicogenic dizziness had improved balance as measured with the SF-BBS (a 7-item functional test of balance) after chiropractic intervention. However, neither study had a control group or any follow-up.

Changes in the physical measures were also reported in the current RCT at the 12 month follow-up. This is the first study to measure physical outcomes of patients with cervicogenic dizziness over the longer term. At 12 months there was greater cervical ROM in all six cervical spine physiological movement directions for the SNAG group and four directions for the PJM group compared to the placebo. There were no differences between the groups for head repositioning accuracy at 12 months. The SNAG group had better balance than the placebo group with the neck in both right rotation and also on a moving base at 12 months, while balance in the PJM group was not different to that for the placebo group. On a head-to-head comparison between the two manual therapy groups at 12 months, there were no significant or meaningful differences in any physical outcomes.

9.2.6 Identification of patients with cervicogenic dizziness

In the fifth publication (Chapter Seven), the process that was used to identify people with cervicogenic dizziness for inclusion in this RCT is comprehensively described. This description may be of some assistance for future studies and for clinicians in identifying those patients with cervicogenic dizziness. It includes data from the RCT described in the previous chapters, which show a high proportion of participants benefitted from the manual therapy interventions, thus providing some indirect evidence that the screening process was successful in identifying participants with cervicogenic dizziness. In clinical practice, if a patient presents with a neck problem and non-rotary dizziness that is triggered by cervical movements or positions, the results of this RCT suggest it would be appropriate to trial treatment with cervical spine PJMs or SNAGs. If they subsequently report decreased dizziness, it would support the clinical hypothesis that the cervical spine is contributing to their dizziness. This approach could help avoid time consuming and costly investigations to establish a diagnosis of cervicogenic dizziness.

9.2.7 Evidence of physical disturbances associated with cervicogenic dizziness

To further describe the characteristics of the cervicogenic dizziness population to aid in recognition of this condition, Chapter Eight reports the baseline mean values for some of the physical indicators of cervicogenic dizziness (cervical ROM, head repositioning

accuracy, balance) for the participants of this study and compared these to published normative data for these measures. Participants with cervicogenic dizziness were found to have significantly decreased cervical ROM compared to published reference values for a similar age group (60-69 years) who were asymptomatic. For the participants of this study, there were also greater errors in head repositioning accuracy compared to published data for asymptomatic healthy individuals, which is a similar finding to that of Heikkila et al. (2000). The findings in this current study for balance are also similar to those of Karlberg et al. who measured balance deficits in patients with cervicogenic dizziness (Karlberg, Johansson, et al., 1996; Karlberg, Magnussen, Malmstrom, Melander, & Moritz, 1996). In the Karlberg et al. study, patients with cervicogenic dizziness were distinguished using posturography from healthy subjects ($p<0.001$) and from those with vestibular pathology ($p<0.001$). This lead Karlberg et al. (1996) to propose that posturographic assessment could be developed to be a possible future tool to differentiate dizziness of cervical origin from other forms of dizziness.

This is the first time that these three measures (cervical ROM, head repositioning accuracy, balance) have been reported for the same group of participants with cervicogenic dizziness. The findings add support to the theory that cervicogenic dizziness results from abnormal sensory afferent input from the proprioceptors of the joints and muscles of the upper cervical region, which then results in disparity of integration with inputs from the vestibular and visual systems and consequently results in balance problems and feelings of dizziness (Wrisley et al. 2000; Treleaven et al. 2003).

9.3 Strengths and limitations of thesis

A major strength of this thesis is that it is based on a RCT study design, which ranks highly in the hierarchy of strength of evidence assessing effectiveness of clinical treatments. Participants were randomly allocated to groups and were unaware of whether they received an active or placebo intervention. The research assistants performing all the outcome assessments were also blind to group allocation. There was adequate statistical power to detect significant differences for the main outcome measures, however, because group sample sizes were determined using the outcome

measures of intensity of dizziness and the DHI, the study may have been under-powered to detect a significant difference between groups for some of the other outcome measures.

In interpreting the findings of this thesis, some methodological limitations must be acknowledged. One limitation, as with all RCTs in the field of manual therapy, was that it was not possible to blind the treating therapist to group allocation, which may introduce bias by the therapist unknowingly influencing participant expectations. Past work has suggested that in RCTS the outcomes can be influenced by personal equipoise. However the treating therapist who has postgraduate training in both the Mulligan and Maitland approaches, attempted to treat all participants equally.

There were shortcomings with some of the measurement tools used. The CROM used to measure head repositioning accuracy was not sufficiently sensitive to detect very small changes in cervical ROM. Another possible limitation of the study was the use of fixed force-plates for most of the posturographic testing of balance, rather than dynamic force-plates. It is thought that dynamic testing is more provocative and sensitive in assessing postural stability (Alund, Ledin, Odkvist, & Sven-Eric, 1993). Changes were reported at 12 months for some of the more challenging tests, such as standing on a moving base, and with the head rotated or extended. However, if all the balance tests had been performed on a moving platform, which is arguably more challenging, it may have been possible to detect other improvements after therapy.

A further limitation is that it is not known how many participants completed their home exercises as only a small proportion of participants regularly kept diaries to monitor compliance, so it is not possible to establish whether home exercises contributed to the changes observed. Only five participants in each manual therapy group reported that they were still performing the exercises at 12 months; more regular follow-ups of participants with phone calls may have encouraged better exercise compliance. It is not possible to say whether people stopped performing the exercises if they had received substantial relief of their symptoms.

9.4 Generalisability of the findings

Since cervicogenic dizziness and associated cervical spine dysfunction are common problems in the community (Colledge, Barr-Hamilton, Lewis, Sellar, & Wilson, 1996), the findings of this study have the potential to benefit many people with this condition. Recruitment for the current study involved a wide geographical area incorporating a major metropolitan centre, several large regional towns, smaller rural centres and farming districts. Thus, the study population is arguably representative of the broader population with cervicogenic dizziness and thus the results can be translated to the wider community.

9.5 Implications of the research

This thesis has the potential to make a difference to how patients with cervicogenic dizziness are managed. Many participants reported the dizziness was having a substantially negative impact on their daily lives (with mean DHI scores being in the moderate handicap range) before the interventions, and after the manual therapy interventions this was significantly reduced (to mild handicap). Moreover, the mean frequency of dizziness episodes decreased from an average of 1 to 4 episodes per week at baseline to an average of 1-4 episodes per month at the 12-week and 12-month follow-ups for both the manual therapy groups. The thesis also adds indirectly to the evidence that cervicogenic dizziness exists, which has been a subject of much debate (Brandt, 1996; Brandt & Bronstein, 2001). A screening process has been described and some of the physical characteristics of this cohort have been compared to normative values, which may aid in the identification of people with this condition.

9.5.1 Implications for clinical practice

When people present with symptoms consistent with cervicogenic dizziness they are often told by their medical practitioner that there is no treatment as the imbalance and neck pain are a normal part of aging, or alternately they may be referred to a neurologist which results in a lengthy, complex and expensive approach where many tests are ordered before a diagnosis is made (Colledge, et al., 1996; Wrisley, Sparto, Whitney, & Furman, 2000). These expensive tests lack diagnostic specificity and are rarely helpful (Colledge, et al., 1996; Newman-Toker et al., 2007; Newman-Toker &

Pronovost, 2009). Even if a diagnosis of cervicogenic dizziness is made, often there is still no treatment offered (Wrisley, et al., 2000).

The primary implication for clinical practice arising from this thesis is that there is evidence that 2-6 sessions of either of two commonly used manual therapy techniques can decrease symptoms of dizziness and improve the physical impairments of decreased cervical ROM, and to a lesser extent balance, over the long term, in people with chronic cervicogenic dizziness. This suggests that at least one of these two forms of manual therapy should be incorporated into the management approach for this condition. Not only are these techniques proven to be effective, but they are simpler, quicker and potentially cheaper to administer than being referred to a specialist. The study thus contributes to the evidence base for manual therapy, which may assist clinicians in their clinical decision making regarding treatment selection.

To date the general consensus for treatment of cervicogenic dizziness has been a multimodal approach incorporating balance and gait exercises, as well as vestibular rehabilitation with habituation exercises (Hansen et al., 2011; Hansson, Månsson, Ringsberg, & Håkansson, 2006; Lystad, Bell, Bonnevie-Svendsen, & Carter, 2011; Shepard, Smith-Wheellock, Telian, & Raj, 1993). In the RCT by Hannson et al. (2006) participants who attended 50 minute group rehabilitation sessions twice a week for six weeks showed improvements in balance tasks and on the DHI compared to a control group at 6 and 12 weeks after the intervention. Additionally, a sensorimotor approach including exercises to improve cervical joint position sense, oculomotor function and postural stability (with general and specific balance exercises) has been shown to be beneficial (Kristjansson & Treleaven, 2009; Lystad, et al., 2011; Treleaven, 2008, 2010). More broadly it has been suggested that as well as physiotherapy and exercises to treat chronic dizziness, an integrated, wider, multidisciplinary approach in a cognitive behavioural framework may be required (Anderson & Yardley, 1998; Butler, 2012; Moseley, Nicholas, & Hodges, 2004). The implication of this thesis is that 2-6 sessions of manual therapy with a recommendation to perform simple mobilising exercises for the neck can have significant effects on the symptoms and signs of chronic cervicogenic dizziness. Hence, this study provides evidence for an alternative treatment option, which may mean a multi-modal treatment approach is unnecessary in some cases. Two

to six sessions of manual therapy would probably be quicker, cheaper and simpler than a multi-modal approach. The emphasis of management should be on relief of symptoms such as dizziness and cervical spine pain, and increasing cervical ROM, as well as balance and proprioception. This should then reduce any handicap caused by this form of dizziness and improve the quality of life for patients suffering from this condition.

As this study found no lasting adverse effects from manual therapy treatment of cervicogenic dizziness, it may be recommended to first trial manual therapy when a patient meets the clinical criteria outlined in Chapter Seven. Physiotherapists can safely treat cervicogenic dizziness with a choice of two commonly used manual therapy approaches, without serious adverse effects, as long as they conduct appropriate screening and regularly monitor symptoms. The participants in the current study were carefully selected to have only cervicogenic dizziness (by excluding those with any other causes of dizziness or two concurrent types of dizziness) to meet the aims of this study. In reality, people may often present with two or three forms of dizziness concurrently (Newman-Toker, et al., 2007). The two manual therapy treatments were found to be safe, supporting that their use be trialled in any patient with suspected cervicogenic dizziness even if another benign cause of dizziness co-exists, so long as dizziness symptoms are monitored (and the more serious conditions of VBI or craniocervical arterial dissection have been ruled out).

The other practice implication is that the steps that were used in this RCT to identify people with cervicogenic dizziness have been outlined and may benefit clinicians in diagnosing this condition. In summary, a new approach to management, supported by the results of this study, may be beneficial for patients with this disabling condition who to date have often been told by their doctor that there are few or no treatment options.

9.6 Suggestions for further research

To further determine the efficacy of manual therapy in the treatment of cervicogenic dizziness and to improve identification of people with this condition, more research is required.

9.6.1 Treatment of more complex dizziness conditions

Although it was found in this study that manual therapy was effective in treating participants identified as having cervicogenic dizziness exclusively (as those with two co-existing causes for dizziness were excluded), further research into the treatment of more complex cases of cervicogenic dizziness, especially those individuals with a combination of this condition and other causes of dizziness may be helpful in the future, as patients commonly present with two or more co-existing types of dizziness (Newman-Toker and Cannon 2007). For example, those participants who had positive peripheral vestibular function tests, as well as a diagnosis of cervicogenic dizziness could be treated with manual therapy to the cervical spine to determine whether improving one deficit (i.e., the pertubation of cervical afferent input) will still have a positive effect on dizziness outcomes. If one sensory deficit is 'normalised' it is possible it may enhance the fine-tuning or balance between proprioceptive, vestibular and visual inputs and reduce the overall sensation of dizziness. This may provide another treatment option for those with coexisting vestibulopathy and cervical afferent deficits. Possible patient groups that this approach may benefit include firstly, those with WAD who may have damage to both their vestibular system and their cervical spine due to forces in the accident and secondly, the ageing population who may have concurrent vestibular degeneration and cervical spine spondylosis and be at increased risk of falls (Chen and Treleaven 2013).

9.6.2 Determine the contribution of home-based exercises

In the present study recommendations to perform self-management strategies (self-SNAGs, self-ROM exercises) were added to the therapist delivered interventions in the treatment of cervicogenic dizziness. This extends previous research where only therapist administered SNAGs were investigated (Reid, Rivett et al. 2008). However, as a large proportion of participants in the current study did not keep diaries to record exercise adherence, it is unknown what proportion of participants followed the exercise advice and for how long. Consequently, the effectiveness of the self-SNAG or self-ROM home exercises could not be accurately assessed. Future research could examine more closely the contribution of home exercises and self-treatments in the improvement in signs and symptoms of people with cervicogenic dizziness. In a future

study, more emphasis could be placed on participants regularly keeping a diary, possibly completing it online so it could be monitored externally, or perhaps closer monitoring of exercise adherence by regular phone calls could be included. Questions could be asked as to when and why the participant ceased the exercises as some may simply cease them when they obtain substantial symptom relief. This will enable a more comprehensive analysis relating to exercise compliance to be undertaken.

Other competing interventions may have been as useful as manual therapy and deserve investigation in the future. Interventions which could be evaluated are neck strengthening, neck kinaesthetic exercises and the benefit of medications.

9.6.3 Improve measurement of head repositioning accuracy

Although an effort was made in this study to determine the effect of manual therapy on head repositioning accuracy as a measure of neck proprioception, it was found that the CROM measuring device was not sufficiently sensitive to detect small changes in movement. Because of the inconclusive results in this study, it would be useful to repeat this assessment using a more sensitive measurement tool such as a head-mounted laser or the Fastrak system. Both the Revel laser method and the three-dimensional Fastrak system have been well documented and used in many other studies to measure head repositioning accuracy (Chen & Treleaven, 2013; Heikkila, et al., 2000; Revel, Andre-Deshays, & Minguet, 1991; Rix & Bagust, 2001; Treleaven, Jull, & Sterling, 2003). Both methods were found to be accurate and able to detect very small movement changes (laser ±0.5°, Fastrak ±0.2°) (Pearcy & Hindle, 1989; Revel, et al., 1991).

In the current study it was decided to use the mid position of the participants available cervical ROM as the 'target position' to assess head repositioning accuracy as it was considered a non-learned position. It was thought that because the neutral head position is a commonly performed task, it may be stored in the long-term memory and thus may be able to be performed without proprioceptive input. However, returning to the NHP as described by Revel et al. (1991), rather than the mid-position of cervical spine rotation, may be more useful as it has been used in many other studies and may be a more reliable 'findable' position (Chen & Treleaven, 2013; Kristjansson, Dall'Alba,

& Jull, 2003; Revel, et al., 1991; Treleaven et al., 2008). Most studies assessing cervical propriocepive mechanisms use relocation to the neutral head position (Revel, et al., 1991). Perhaps future studies could compare the mid position to the neutral head position to see which method is more challenging, yet still accurate.

9.6.4 Cost effectiveness of manual therapy treatments for cervicogenic dizziness

A cost effectiveness study could be performed for each of the interventions to compare pre- and post-intervention costs (medication use, visits to general practitioners, visits to other health professionals etc.) to determine whether medical costs are reduced after the interventions to further support these manual therapy treatment approaches. As both forms of manual therapy were found to be equally beneficial, a cost comparison analysis could be made for treatment with each form of manual therapy to determine whether one approach is less costly than the other. Treatment of cervicogenic dizziness with manual therapy could be compared to treatment with vestibular rehabilitation, which has been shown to be beneficial but could take longer and require more therapist sessions, and thus be more costly. In the study by Hannson et al. (2006) participants received 12 x 50 minutes sessions of vestibular rehabilitation, however as these were group sessions the cost may have been less overall. It may be useful to compare the cost of management by a physiotherapist (diagnosis and treatment) to that of seeing a neurologist and undertaking the tests they may request in order to make a diagnosis of this condition (Colledge, et al., 1996).

9.6.5 Validate a procedure for the diagnosis of cervicogenic dizziness

To date there is no 'gold standard' test to identify patients with cervicogenic dizziness. The process that was used to identify people with cervicogenic dizziness in this study was found to be effective and has been described in Chapter Seven. This screening process could be further developed and validated to become a formal screening tool to help identify those individuals with this disorder. The characteristics of patients with cervicogenic dizziness have not been extensively described in the literature as it is a poorly understood condition and a sometimes controversial diagnosis. If more specific descriptions of this condition and diagnostic guidelines could be established, it might enable practitioners to better differentiate between people with cervicogenic dizziness,

migraine, cardiovascular dizziness, vestibular dizziness (such as BPPV) and the more life threatening conditions of vertebrobasilar insufficiency, arterial dissection and stroke.

9.6.6 Determine whether a cervicogenic dizziness handicap tool is desirable

Although the DHI was a useful tool in this study to assess the effect dizziness was having on the day to day lives of those suffering from chronic cervicogenic dizziness and the results showed that self-perceived handicap (measured with the DHI) improved after treatment with manual therapy, it may be possible to modify the DHI to be more responsive for the cervicogenic dizziness population. The DHI questionnaire, in its present form, was designed by Newman and Jacobson (1993) using patients with vestibular dysfunction who demonstrated positive responses to caloric testing, resulting in some questions not being relevant to the cervicogenic dizziness population. In addition to differences in many dizziness characteristics such as a sensation of vertigo/spinning with vestibular dysfunction versus unsteadiness with cervicogenic dizziness, the cervicogenic dizziness population tends to be older and thus have more co-morbidities, in particular pain, which may influence responses on the DHI (as a number of the items relate to disability that is not specific to dizziness). Further, the DHI has been shown to be poorly correlated with balance measures such as posturography (Vereeck, Truijen, Wuyts, & Van de Heyning, 2006). Together this suggests that the DHI in its present format may have limitations for use with the cervicogenic dizziness population and could probably be revised to be more responsive to this population. The DHI has three subgroups or domains of questions (emotional, functional, physical) and some may be more relevant to people with cervicogenic dizziness than others (Treleaven, 2006). It would be interesting to further breakdown the results from the DHI for this cohort of dizziness patients, considering their responses to these domains and to specific questions. It would be useful to see the proportion of participants who scored high or low on certain components of the DHI. Examples of questions that may not be relevant to the cervicogenic dizziness population include 'Because of your problem, do you avoid heights?' and 'Because of

your problem, are you afraid to stay home alone?' A new or modified DHI specifically intended for the cervicogenic population could be designed and validated.

9.6.7 Determine the effect of manual therapy on cervical pain in participants with cervicogenic dizziness

There is still inconclusive evidence for the impact of manual therapy on cervical pain in participants with cervicogenic dizziness. In future studies it is recommended that participants are assigned to groups by stratifying for dizziness and for cervical pain so both dizziness intensity and pain intensity are equivalent between groups at baseline.

9.7 Summary of the thesis

Cervicogenic dizziness is a commonly presenting musculoskeletal disorder that is responsible for significant disability and reduction in quality of life, as well as potentially placing a financial burden on society. It is recognised as difficult to diagnose.

This thesis provides evidence that 2-6 treatments of either of two commonly used forms of manual therapy (SNAGs and PJMs) provide both short and long-term benefits for people with chronic cervicogenic dizziness. Both manual therapy treatments provided an immediate reduction in the intensity of dizziness, which is maintained 12 months later. Dizziness frequency and handicap continue to decrease post-treatment and are significantly less that for a placebo intervention at 12 months. There were no differences in the effectiveness of the two manual therapy treatments on these self-reported dizziness measures, except for lower DHI scores in the PJM group compared to the SNAG group at the 12-week follow-up. Increases in cervical ROM were observed with both manual therapies, although the SNAG treatment provided slightly more immediate and greater benefits. There were no differences in the effectiveness of the two manual therapy interventions on head repositioning accuracy or balance.

The findings of this thesis indicate that clinicians can incorporate manual therapy (with related exercise) as part of an effective long-term treatment for chronic cervicogenic dizziness. The process that was used to identify people with cervicogenic dizziness for inclusion in this study may also assist the clinician in the diagnosis of cervicogenic dizziness, as well as the researcher in identifying a homogeneous group of study

participants. The dissemination of results from this thesis will lead to benefits for patients suffering from cervicogenic dizziness by improving our understanding of this condition and providing high level evidence for its effective management.

9.8 References for Chapter Nine

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Appendices

APPENDIX A: Co-author statements

Statement from Darren A. Rivett relating to papers published with Susan Reid

I, Darren A. Rivett, attest that Research Higher Degree candidate, Susan Reid contributed to the listed publications by contributing to the conception and design of the studies, conducting and writing up the literature review, the collection of data, undertaking the statistical analysis in collaboration with a statistician, description and interpretation of the results, and writing the discussion and conclusions.

1. Reid, SA, Rivett, DA, Katekar, M, Callister, R. (2012) Efficacy of manual therapy treatments for people with cervicogenic dizziness and pain: protocol of a randomised controlled trial. BMC Musculoskeletal Disorders 13: 201-208.

2. Reid, S. A. Rivett, D. A.Katekar, M & Callister, R. (2014) Comparison of Mulligan sustained natural apophyseal glides and Maitland mobilizations for treatment of cervicogenic dizziness: a randomized controlled trial. Physical Therapy 94: 466-476.

3. Reid, SA. Rivett, DA. Katekar, M & Callister, R. (2014) The effects of cervical spine manual therapy on cervical range of motion, head repositioning and balance in participants with cervicogenic dizziness: a randomized controlled trial. Archives of Physical Medicine and Rehabilitation DOI: http://dx.doi.org/10.1016/j.apmr.2014.04.009.

4. Reid SA, Callister R, Snodgrass S, Katekar MG, Rivett RA (2014) Manual therapy treatment of cervicogenic dizziness: long-term outcomes of a randomised controlled trial. Manual Therapy (Accepted 27/6/2014. In Press)

5. Reid SA, Callister R, Katekar MG, Rivett RA, (2014) Identification of individuals with cervicogenic dizziness with evidence from recruitment for a randomised controlled trial. Manual Therapy (Under review)

Professor Darren A. Rivett **Date:**

.....

Susan Reid **Date:**

.....

Professor Robert Callister **Date:**

Assistant Dean Research Training, Faculty of Health and Medicine

Statement from Robin Callister relating to papers published with Susan Reid

I, Robin Callister, attest that Research Higher Degree candidate, Susan Reid contributed to the listed publications by contributing to the conception and design of the studies, conducting and writing up the literature review, the collection of data, undertaking the statistical analysis in collaboration with a statistician, description and interpretation of the results, and writing the discussion and conclusions.

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.....

Professor Robin Callister **Date:**

Susan Reid **Date:**

.....

Professor Robert Callister **Date:**

Assistant Dean Research Training, Faculty of Health and Medicine

Statement from Michael Katekar relating to papers published with Susan Reid

I, Michael Katekar, attest that Research Higher Degree candidate, Susan Reid contributed to the listed publications by contributing to the conception and design of the studies, conducting and writing up the literature review, the collection of data, undertaking the statistical analysis in collaboration with a statistician, description and interpretation of the results, and writing the discussion and conclusions.

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5. Reid SA, Callister R, Katekar MG, Rivett RA, (2014): Identification of individuals with cervicogenic dizziness with evidence from recruitment for a randomised controlled trial. Manual Therapy (Under review)

.....

Dr Michael Katekar **Date:**

Susan Reid **Date:**

.....

Professor Robert Callister **Date:**

Assistant Dean Research Training, Faculty of Health and Medicine

Statement from Suzanne Snodgrass relating to a paper published with Susan Reid

I, Suzanne Snodgrass, attest that Research Higher Degree candidate, Susan Reid contributed to the listed publication by contributing to the conception and design of the studies, conducting and writing up the literature review, the collection of data, undertaking the statistical analysis in collaboration with a statistician, description and interpretation of the results, and writing the discussion and conclusions.

Reid SA, Callister R, Snodgrass S, Katekar MG, Rivett RA (2014): Manual therapy treatment of cervicogenic dizziness: long-term outcomes of a randomised controlled trial. Manual Therapy (Accepted 27/6/2014. In Press)

Dr Suzanne Snodgrass **Date:**

Susan Reid **Date:**

.....

Professor Robert Callister **Date:**

Assistant Dean Research Training, Faculty of Health and Medicine

APPENDIX B: Permission letter regarding copyright

American Physical Therapy Association

1111 North Fairfax Street
Alexandria, VA 22314-1488
703 684 2782
703 684 7343 fax
www.apta.org

December 12, 2013

Sue Reid
Australian Catholic University
Level 3, 173 Pacific Hwy
North Sydney NSW 2060, Australia
E-mail: sue.reid@acu.edu.au

APTA Request Reference: PTJ 147/13; PTJ-2012-0483; Thesis

Dear Ms Reid
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February 3-6, 2014
Las Vegas, NV

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Michele Tillson VA,
Publishing and Member Communications Specialist
December 12, 2013

APPENDIX C: University media release for recruitment

Volunteers wanted for physiotherapy study

Researchers at the University of Newcastle are looking for volunteers who suffer from poor balance or dizziness due to a stiff or painful neck for the next stage of a physiotherapy study.

PhD student Sue Reid said the study aimed to determine whether physiotherapy procedures were effective in relieving dizziness and neck pain.

"Our earlier study showed that manual Physiotherapy was effective in the short term," Ms Reid said. "Now we will investigate if gentle manual therapy and/or exercises are effective in the long term."

You would not be suitable for the study if your dizziness is a spinning sensation, if you have a current Workers Compensation claim or if you have conditions that prevent you from having normal physiotherapy such as a fractured neck, cancer etc.

The University's health and medical researchers work in collaboration with the Hunter Medical Research Institute (HMRI). HMRI is a partnership between the University of Newcastle, Hunter New England Health and the community.

For more information or to volunteer for the study, contact Ms Sue Reid on 02 4921 5925. Media contact: Ms Sue Reid on 02 4921 5925.

Kate Reid
Media and Public Relations
The University of Newcastle
P: +61 2 4921 5351
M: 0438 745 098
F: +61 2 4921 6400
W: www.newcastle.edu.au
E: Kate.Reid@newcastle.edu.au

APPENDIX D: Advertisement for recruitment

THE UNIVERSITY OF
NEWCASTLE
AUSTRALIA

Do you have poor balance or dizziness from a neck problem?

Researchers at the University of Newcastle are looking for volunteers who suffer from dizziness described as unsteadiness or poor balance and also have a stiff or painful neck. The unsteadiness may be caused by neck movements or positions such as looking up or turning the head. If you have this type of dizziness you may wish to be part of a physiotherapy study at the University of Newcastle. Sue Reid (a PhD student under the supervision of Professor Darren Rivett and Associate Professor Robin Callister) from the Faculty of Health says that the study will determine whether physiotherapy procedures are effective in relieving or eliminating the symptoms of neck dizziness and pain in the long term.

"We are testing physiotherapy techniques to see if they relieve the patient's dizziness and neck pain," said Sue. "We have previously conducted a study that showed one of these physiotherapy treatments was very effective in the short term in treating dizziness, pain and disability in patients with this problem. Now we will be comparing it with another form of physiotherapy treatment, adding some home exercises and looking at the long term effect of the interventions."

All people entering the trial will be assessed and screened by a neurologist first. Researchers will measure each participant's dizziness, neck movements and balance before and after the treatment.

People interested in participating in the study should call (02) 4921 5925.

APPENDIX E: Recruitment letter to doctors

Discipline of Physiotherapy

School of Health Sciences
Faculty of Health
University Drive, Callaghan
NSW 2308 Australia
Phone: +61 2 4921 7904
Fax: +61 2 4921 7902

January 14, 2010

Dear Doctor,
We are currently conducting a placebo controlled clinical trial of physiotherapy procedures in patients with suspected cervicogenic dizziness. We are recruiting patients with positionally induced or aggravated dizziness, which is not due to benign positioning vertigo or labyrinthine disease and in whom clinical features suggest a possible cervicogenic cause.

Participants should have:
Dizziness:

- described as imbalance, disequilibrium or unsteadiness (not rotatory vertigo)

AND

- related to either movements or positions of the cervical spine, or occurring with a stiff or painful neck

The study is being conducted by Mrs Sue Reid, as part of her PhD degree in Physiotherapy at The University of Newcastle.

Patients participating in the study will undergo detailed neurological assessment and vestibular function testing by Dr Michael Katekar (neurologist). Dr Katekar will see the patients promptly and free-of-charge. If selected as suitable (i.e. they have cervicogenic dizziness) they will be randomized to undergo a physiotherapy procedure. The aim of the study is to investigate the effectiveness of physiotherapy procedures used in the management of dizziness originating in the neck.

If you have any patients who might be suitable, we would be interested to discuss the study with them. Could you please give them a copy of the Initial Letter of Invitation (enclosed). They could phone Sue Reid on 49215925. Many thanks for your assistance.

Yours sincerely

Susan Reid (PhD student)
Professor Darren Rivett (Principal Supervisor)
Assoc. Prof. Robin Callister (Co-supervisor)
Dr Michael Katekar (Co-investigator)

Version No. 1 14/01/10

APPENDIX F: Invitation to participate

Professor Darren Rivett
School of Health Sciences
Faculty of Health
University Dr, Callaghan
NSW 2308
Phone 49217220; FAX 49217053
Darren.Rivett@newcastle.edu.au

Invitation to participate in the Research Project:
The identification and treatment of patients with cervicogenic dizziness and pain
Version 2 (29/01/10)

Chief Investigators: Prof Darren Rivett (Project Supervisor), A/Prof Robin Callister, Dr Michael Katekar. Student Researcher: Ms Susan Reid

29th January, 2010

Dear Sir/Madam,

I would like to invite you to participate in a study that investigates the effectiveness of physiotherapy procedures used in the management of dizziness and pain coming from the neck.

Eligibility

Your doctor has examined you and believes your dizziness may be coming from a neck problem so you may be eligible to participate in the study.

To participate in the study you must:
- be between 18-90 years old
- have dizziness which is related to movements or positions of your neck OR
- have dizziness with a stiff neck and/or pain produced by your neck
- have had the symptoms for greater than three months.

If you have or have had any of the following you may not be able to participate in the study:
- a history of inflammatory joint disease e.g. rheumatoid arthritis
- spinal cord pathology
- cervical spine infection
- bony disease or marked osteoporosis
- marked cervical spine disc protrusion
- cervical spine cancer

- severe pain, weakness, pins and needles or numbness in your arm or hand for less than 6 weeks duration
- recent fracture or dislocation of the neck (in the last 3 months)
- previous surgery to the neck
- physiotherapy or other treatment directed to the neck in the last month
- pregnancy
- Worker's Compensation case in progress
- inability to speak or write English.

If you are unsure if one of these criteria applies to you, please phone and ask Susan Reid or one of the other investigators.

If you would like to find out more information about the study please phone Susan Reid in the Discipline of Physiotherapy, University of Newcastle on 4921 5925.

You will be sent some written information to read before you make a decision about participating in the study. You will be allowed time to consider your decision at leisure and independent of the doctor and researchers. Whatever you decide in relation to participation in the study, your treatment by your referring doctor or Dr Katekar won't be affected. Your participation in this study is voluntary and you can withdraw from the study at any time.

Yours sincerely,

Prof. Darren Rivett
Project supervisor

Susan Reid
PhD Research student

Complaints about this research

This project has been approved by the University's Human Research Ethics Committee, Approval No. H-2009-0377

Should you have concerns about your rights as a participant in this research, or you have a complaint about the manner in which the research is conducted, it may be given to the researcher, or, if an independent person is preferred, to the Human Research Ethics Officer, Research Office, The Chancellery, The University of Newcastle, University Drive, Callaghan NSW 2308, Australia, telephone (02) 49216333, email Human-Ethics@newcastle.edu.au.

APPENDIX G: Participant information statement

Professor Darren Rivett
School of Health Sciences
Faculty of Health
University Dr, Callaghan
NSW 2308
Phone 49217220; FAX 49217053
Darren.Rivett@newcastle.edu.au

Information Statement for the Research Project:
The identification and treatment of patients with cervicogenic dizziness and pain
Version 2 (14/01/10)

Chief Investigators: Prof Darren Rivett (Project Supervisor), A/Prof Robin Callister, Dr Michael Katekar
Student Researcher: Ms Susan Reid

You are invited to participate in the research project identified above which is being conducted in the Faculty of Health at the University of Newcastle. The research is part of Susan Reid's PhD studies and is supervised by Prof. Darren Rivett, Assoc Prof. Robin Callister and Dr Michael Katekar.

Why is the research being done?
The purpose of the research is to determine the effectiveness of three procedures performed by physiotherapists as treatment for dizziness and pain that is brought on by movement of the neck or associated with neck pain or stiffness.

Dizziness is a common complaint. It can lead to falls, anxiety and an inability to perform normal activities. Many different problems may cause dizziness. In some cases, dizziness is thought to be due to a problem in the neck or cervical spine. Physiotherapy is often used to treat the neck to alleviate dizziness in such cases.

Who can participate in the research?
We are seeking people who have dizziness that is described as imbalance together with a stiff and/or painful neck. Certain neck positions, such as looking up or turning your head, may bring on your dizziness.

To participate in the study you must:
- have dizziness which is related to movements or positions of your neck OR
- have dizziness with a stiff neck and/or pain produced by your neck
- be between 18-90 years
- have had the symptoms for greater than three months

If you have or have had any of the following you may not be able to participate in the study:
- a history of inflammatory joint disease e.g. rheumatoid arthritis
- spinal cord pathology
- cervical spine infection
- bony disease or marked osteoporosis

- marked cervical spine disc protrusion
- cervical spine cancer
- severe pain, weakness, pins and needles or numbness in your arm or hand for less than 6 weeks duration
- recent fracture or dislocation of the neck (in the last 3 months)
- previous surgery to the neck
- physiotherapy or other treatment directed to the neck in the last month
- pregnancy
- Workers Compensation case in progress
- inability to speak or write English

If you are unsure if one of these criteria applies to you, please ask an investigator.

What choice do you have?
Participation in this research is entirely your choice. Only those people who give their informed consent will be included in the project. Whether or not you decide to participate, your decision will not disadvantage you.

If you do decide to participate, you may withdraw from the project at any time without giving a reason and have the option of withdrawing any data which identifies you.

What would you be asked to do?
If you agree to participate, you will be screened by the Student Researcher Susan Reid in the Discipline of Physiotherapy in the Hunter Building at the University of Newcastle at Callaghan to see if you have cervicogenic dizziness (dizziness caused by a neck problem). Susan Reid is a Musculoskeletal Physiotherapist with thirty years of clinical experience. You will be provided with a map and parking vouchers for the University to attend this screening.

This screening will involve talking about your dizziness and a standard clinical test will be performed to see whether you have vertigo due to an inner ear problem. Blood pressure will be measured. If it is determined that you possibly have cervicogenic dizziness, you will then be assessed by Dr Michael Katekar (neurologist) at Charlestown who will examine you to exclude any other cause of your dizziness. There will be no financial charge for this visit. If Dr Katekar diagnoses cervicogenic dizziness you will then proceed to the intervention phase of the study. If you were referred into the study by Dr Katekar you will not be required to do the above screening.

The intervention phase

- On the first day you will be assessed by filling in questionnaires, completing dizziness and pain scales, and having your balance and neck movements measured. You will also be asked about your medications and how often you take them.
- Following the measurements, you will be randomly allocated to one of three possible groups by choosing a numbered envelope. Each group will receive a different procedure from the physiotherapist. You are requested not to discuss your procedure with other people in the study.
  - You will be given this same procedure two to six times over three-to-six weeks. The number of actual treatments you receive and the time frame will be determined by the physiotherapist.
  - You may be asked to do some simple home exercises.

- You will be asked to keep a simple diary of visits to your doctor, medication use and performance of exercises.
- The first session will take about 60 minutes. Each other session will take about 30 minutes, except the last, which will also take about 60 minutes.
- After the last application of the procedure you will be asked to again complete the questionnaires, the pain and dizziness rating scales, and your neck movements and balance will be measured.
- Six weeks, twelve weeks, six months, one year and two years after the completion of your last treatment session, you will be reassessed with the questionnaires, dizziness and pain scales and have your balance and neck movements measured. Each of these sessions will take about 60 minutes.

The procedures

One group will receive therapeutic laser to the upper neck region. Normally there is no sensation felt with laser. If you experience any pain or dizziness you must tell the physiotherapist.

One of the two manual therapy groups will be treated with a gentle mobilisation technique to the upper neck region while you are seated on a chair. The physiotherapist will place her thumbs on one of the vertebrae and gently press, as you move your neck. This movement is repeated a number of times, as long as you have no symptoms during the procedure.

The other manual therapy group will receive an intervention that involves the physiotherapist pushing gently on the vertebrae in your upper neck while you lie on your stomach on the bed. This normally takes several minutes and you may feel slight discomfort.

What measurements will be performed?

- Questionnaire. You are asked to complete some questions regarding difficulties you may be experiencing because of your dizziness. This is a standard easy-to-use questionnaire.
- Pain rating scale. You will be asked to rate how bad the neck pain you may experience is on a scale of 0 to 10.
- Dizziness rating scale. You will be asked to rate the severity of your dizziness on a scale of 0 to 10.
- You will be asked how often you get dizzy.
- Neck movements. You will be asked to move your head, as far as you can comfortably move, by looking up and down and by turning and tilting left and right. Your movements will be measured using a comfortable and well-accepted measuring device.
- Balance. You will be asked to stand on two balance machines, which will measure your balance reactions. You can hold onto rails if you feel unsteady. You may have a harness on to stop you falling as a safety precaution, if you feel unstable. There will be two people, the physiotherapist and an assistant, present to help you. This machine is very safe and is often used to assess and treat patients with dizziness.
- You will be asked to turn your neck to the side and then bring it back to the starting position. Your ability to return to the starting position will be measured.

All the tests are stopped if you feel dizzy or have pain. There will be about a 5 minute rest between tests.

You will be asked to do all of these on the first day, after the final application of the procedure and at the six week, twelve week, six month, one year and two year follow up visits. At the other treatment sessions, the rating scales for pain and dizziness and measurement of your neck movements will be performed.

How much time will it take?

Each visit will take between 30 and 60 minutes.

What are the risks and benefits of participating?

The procedures in this study are used routinely in clinical practice, and there have been no published reports of adverse effects or problems with any of these procedures, to the best of our knowledge.

You will receive a procedure of unknown benefit. The researchers anticipate that some participants will benefit from the procedures used, but not all. As this is a placebo controlled trial there is a chance you could be allocated to a placebo control group. Previous studies have shown that participants can still benefit from being in the placebo group. If you receive no benefit from your procedure, and if one of the other procedures is shown to be beneficial, you will be offered the other procedure free of charge, after completion of your participation in the study. There will be no cost to you for any of the procedures you may receive in this trial or for any other part of your participation.

How will your privacy be protected?

Information that is provided will be confidential to the researchers. If these results are reported at a scientific meeting or published in a journal or thesis, no names will be used. Publication of results will be in general terms and will not allow for the identification of individuals. Name and address information will be removed from the forms within one week and data will be recorded against a code number. Only the consent form will contain your name and this will be stored separately, in a locked cabinet in the office of the School of Health Sciences. All the data will be stored for 20 years after completion of the study and then shredded and deleted.

How will the information collected be used?

The results of this study will be submitted in a thesis for Susan Reid's PhD and may be presented at conferences and in scientific journals. Individual participants will not be identified in any reports arising from this project. Participants will be offered a summary of the results of this project in lay language.

What do you need to do to participate?

Please read this Information Statement and be sure you understand its contents before you consent to participate. If there is anything you do not understand, or you have questions, contact the researcher.

If you would like to participate, please complete the attached Consent Form and return it in the reply paid envelope provided. I will then contact you to arrange a time convenient to you for the screening.

Funding

This study has been partially funded by The Mulligan Concept Teachers Association.

Further information

If you would like further information please contact Susan Reid 49215925 or sue.reid@newcastle.edu.au or Prof Darren Rivett 49217220 Darren.Rivett@newcastle.edu.au.

Thank you for considering this invitation.

Prof. Darren Rivett
Project supervisor

Susan Reid
Research student

Complaints about this research

This project has been approved by the University's Human Research Ethics Committee, Approval No. H-2009-0377

Should you have concerns about your rights as a participant in this research, or you have a complaint about the manner in which the research is conducted, it may be given to the researcher, or, if an independent person is preferred, to the Human Research Ethics Officer, Research Office, The Chancellery, The University of Newcastle, University Drive, Callaghan NSW 2308, Australia, telephone (02) 49216333, email Human-Ethics@newcastle.edu.au.

APPENDIX H: Consent Form

Consent Form for the Research Project:

The identification and treatment of patients with cervicogenic dizziness and pain

Version 2 (14/01/10)

Chief Investigators: Prof Darren Rivett (Project Supervisor), A/Prof Robin Callister, Dr Michael Katekar

Student Researcher: Ms Susan Reid

I agree to participate in the above research project and give my consent freely.

I understand that the project will be conducted as described in the Information Statement, a copy of which I have retained.

I understand I can withdraw from the project at any time and do not have to provide any reason for withdrawing.

I consent to:

- Being tested to determine whether I have cervicogenic dizziness and/or pain. (Participants referred by Dr Katekar do not require additional testing for diagnosis of cervicogenic dizziness.)
- Receiving an intervention which may treat my dizziness and/or pain

I understand that my personal information will remain confidential to the researchers and that data collected from my participation may be used in journal publications, conference presentations and theses.

I have had the opportunity to have questions answered to my satisfaction. I have the right to withdraw my information at any time.

Print Name: _____

Signature:_____ **Date:** _____

Contact Details: Phone _____

(Email) _____

Address if prefer results to be mailed: _____

_____

APPENDIX I: Self-report forms

VISUAL ANALOGUE SCALE FOR DIZZINESS

Code: _____ Date:_____

Please place a mark on the line below to show how **intense** your average level of dizziness has been in the last few days.

I_____I

No dizziness

Worst dizziness imaginable

DIZZINESS HANDICAP INVENTORY

Form 1

CODE: _____ D.O.B.: _____ Date: _____

Score Totals: _____; E _____; F _____; P _____
(100) (36) (36) (28)

Instructions: Please *circle* the correct response:

1. I have dizziness/unsteadiness:

[1] 1 per month [2] >1 but <4 per month [3] more than one per week

2. My dizziness/unsteadiness is:

[1] mild [2] moderate [3] severe

Instructions: (Please read carefully): The purpose of the scale is to identify difficulties that you may be experiencing because of your dizziness or unsteadiness. Please answer "Yes", "Sometimes", or "No" to each question. Answer each question as it pertains to your dizziness or unsteadiness problem only.

	YES	SOMETIMES	NO
1. Does looking up increase your problem?	☐	☐	☐
2. Because of your problem, do you feel frustrated?	☐	☐	☐
3. Because of your problem, do you restrict your travel for business or recreation?	☐	☐	☐
4. Does walking down the aisle of a supermarket increase your problem?	☐	☐	☐
5. Because of your problem, do you have difficulty getting into or out of bed?	☐	☐	☐
6. Does your problem significantly restrict your participation in social activities such as going out to dinner, going to movies, dancing, or to parties?	☐	☐	☐
7. Because of your problem, do you have difficulty reading?	☐	☐	☐
8. Does performing more ambitious activities like sports, dancing, household chores such as sweeping or putting dishes away increase your problem?	☐	☐	☐
9. Because of your problem, are you afraid to leave your home without someone accompanying you?	☐	☐	☐
10. Because of your problem, have you been embarrassed in front of others?	☐	☐	☐
11. Do quick movements of your head increase your problem?	☐	☐	☐
12. Because of your problem, do you avoid heights?	☐	☐	☐
13. Does turning over in bed increase your problem?	☐	☐	☐
14. Because of your problem, is it difficult for you to do strenuous house work or yard work?	☐	☐	☐
15. Because of your problem, are you afraid people may think you are intoxicated?	☐	☐	☐
16. Because of your problem, is it difficult for you to go for a walk by yourself?	☐	☐	☐
17. Does walking down a sidewalk increase your problem?	☐	☐	☐
18. Because of your problem, is it difficult for you to concentrate?	☐	☐	☐
19. Because of your problem, is it difficult for you to walk around your house in the dark?	☐	☐	☐
20. Because of your problem, are you afraid to stay home alone?	☐	☐	☐
21. Because of your problem, do you feel handicapped?	☐	☐	☐
22. Has your problem placed stress on your relationships with members of your family or friends?	☐	☐	☐
23. Because of your problem, are you depressed?	☐	☐	☐
24. Does your problem interfere with your job or household responsibilities?	☐	☐	☐
25. Does bending over increase your problem?	☐	☐	☐

Code:_____ Date:_____

FREQUENCY OF DIZZINESS

Indicate how often you get dizziness by circling a number from 0 to 6 where:

0 = no dizziness

1 = dizziness less than once a month

2 = dizziness a few times per month

3 = dizziness a few times a week

4 = dizziness once daily

5 = dizziness more than once per day

6 = constant dizziness

CODE: _____ Date: _____

VISUAL ANALOGUE SCALE FOR PAIN

Please place a mark on the line below to show how **intense** your usual pain has been in the last few days.

|_____|

No pain

Worst pain imaginable

GLOBAL PERCEIVED EFFECT

CODE:_____ DATE:_____

Indicate by circling the most appropriate statement below.

Do you feel the treatment you received had:

0=No benefit

1=Minimal benefit

2=Some benefit

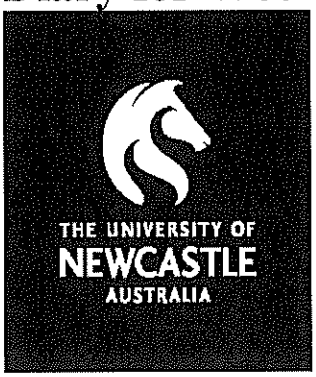
3=A lot of benefit

4=Great benefit

5=Maximal benefit

APPENDIX J: Diary for weeks 1-6 (during intervention phase)

Diary for Weeks 1-6



Patient No:

Date Diary commenced:

This diary is to be used to record aspects of your symptoms that will help us with the study.
The diary has a page for each week for the first 6 weeks after your intervention starts. After the first 6 weeks you will be given another diary with one page for each month to record any changes that take place.

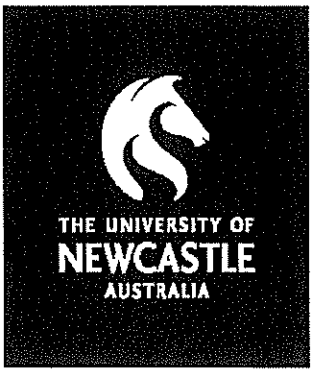
You are asked to record any time that your dizziness or neck pain or headaches results in:

-use of tablets or medication. Write "M"
-time off work. Write "W"
-changes in social engagements or outings. Write "S"
-visits to doctors or other health professionals. Write "D"

Weekly Diary Week 1 Week of _____

MONDAY	TUESDAY
Medications	Medications
Time off work	Time off work
Doctors visits	Doctors visits
Changes to social life	Changes to social life
WEDNESDAY	THURSDAY
Medications	Medications
Time off work	Time off work
Doctors visits	Doctors visits
Changes to social life	Changes to social life
FRIDAY	SATURDAY
Medications	Medications
Time off work	Time off work
Doctors visits	Doctors visits
Changes to social life	Changes to social life
SUNDAY	
Medications	
Time off work	
Doctors visits	
Changes to social life	

APPENDIX K: Diary for 12 month follow-up phase



Participant No:_____

Date Diary commenced:_____

This diary is to be used to record aspects of your symptoms that will help us with the study.
The diary has a page for each month after the first weeks following the intervention.

You are asked to record any time that your dizziness or neck pain or headaches results in:

-use of tablets or medication. Write "M"
-time off work. Write "W"
-changes in social engagements or outings. Write "S"
-visits to doctors or other health professionals. Write "D"

January 2009

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Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
				1	2	3
4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	31

Goals

☑ To Do

Remember

Notes

APPENDIX L: External grant advice

MULLIGAN CONCEPT

Mulligan Concept Teachers Association (MCTA)
Research Committee
c/o Associate Professor Wayne Hing
School of Physiotherapy
Auckland University of Technology
Private Bag 92006, Auckland, New Zealand
wayne.hing@aut.ac.nz

5th May 2009

Professor Darren Rivett, Susan Reid, Associate Professor Robin Callister
School of Health Sciences
The University of Newcastle
Callaghan, NSW 2308, Australia

Re: Application for Mulligan Concept Teachers Association Research Award
Dear Darren,

Thank you for applying to the Mulligan Concept Teachers Association for the above Research Award.

The Scientific Committee are pleased to advise you that your application for funding has been successful.

You have been awarded the amount of $ 40,000.00 (Australian Dollars)

It is a requirement of this grant that you acknowledge the MCTA for this research funding in any publications or conference presentations that arise from this work.

Additionally, reports updating the progress of the project are required at six months and on the completion of the study.

This confirmation letter has also been sent to Frank Gargano, the treasurer of the organisation (garganof@ Frank Gargano (garganof@sbcglobal.net), so he is aware of the outcome.

Upon receipt of the start date of the research, when you will be requiring the funds and your relevant bank details he will organise payment of the grant.

Sincerely

Dr Wayne Hing
Chair of Scientific Research Committee MCTA

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For the journals:

- BMC Musculoskeletal
- Archives of Physical Medicine and Rehabilitation

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