RED FLAG SCREENING FOR SERIOUS PATHOLOGY PRESENTING IN CERVICAL SPINE MUSCULOSKELETAL DISORDERS

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Abstract

Aim
To develop a list of red flag clinical indicators for possible serious pathology masquerading as, or presenting alongside, neck related musculoskeletal disorders in the adult population.

Background
Musculoskeletal physiotherapists provide assessment and treatment for pain and functional impairments relating to musculoskeletal disorders, such as back and neck pain. In order to apply safe and effective treatment to these conditions it is vitally important that any underlying serious complaints have been excluded. Clinical indicators known as 'red flags' have been developed for diagnostic triage in back pain to help identify serious underlying conditions, such as cancer and infection. Red flags for serious pathology in neck pain or neck related pathology has not received the same level of attention as red flags in back pain. A literature review identified inconsistent evidence for clinical tests and clinical indicators for serious pathology in neck related musculoskeletal disorders. This presents a serious clinical challenge for musculoskeletal physiotherapists.

Method
A mixed method study design was developed involving: a) Qualitative descriptive method through Physiotherapy focus group; and, b) Three round Delphi survey method involving consultant neurologists and consultant neurosurgeons. The Delphi method involves combined qualitative and quantitative data phases. Thematic content analysis was used to analyse the qualitative data. A combined descriptive and inferential (non-parametric) statistical analysis was used to analyse the quantitative data. Kendall’s W (Kendall’s coefficient of concordance) was used to evaluate the level of consensus across all participants for the quantitative phase of the Delphi method.

Findings
A list of neck related red flag clinical indicators within five specific categories were developed: 1. progressive pain; 2. cancer, infection, trauma; 3. neurological deficit (spinal cord compromise); 4. headache (associated with neck pain/stiffness); 5. brainstem, cervical arterial and cranial nerve dysfunctions. An increase in Kendall’s W was demonstrated between Rounds 2 and 3 in four out of five categories, indicating an increase in consensus levels between participants. This process highlights the complexity of interpreting clinical features within musculoskeletal presentations.

Key words
Cervical spine red flags; neck pain red flags; cervical arterial dissection; cervical arterial dysfunction; cervical spondylotic myelopathy; cord compression; serious spinal pathology.
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<th>Abbreviation</th>
<th>Definition</th>
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<tbody>
<tr>
<td>AE</td>
<td>Adverse events</td>
</tr>
<tr>
<td>CAD</td>
<td>Cervical Arterial Dissection</td>
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<tr>
<td>CADy</td>
<td>Cervical Arterial Dysfunction</td>
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<tr>
<td>CKS</td>
<td>Clinical Knowledge Summaries</td>
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<tr>
<td>CNT</td>
<td>Cranial Nerve Testing</td>
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<tr>
<td>CSAG</td>
<td>Clinical Standards Advisory Group</td>
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<tr>
<td>ESP</td>
<td>Extended Scope Practitioner</td>
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<tr>
<td>FG</td>
<td>Focus Group</td>
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<tr>
<td>ICA</td>
<td>Internal Carotid Artery</td>
</tr>
<tr>
<td>ICAD</td>
<td>Internal Carotid Artery Dissection</td>
</tr>
<tr>
<td>ISI</td>
<td>Increased Signal Intensity (in Magnetic Resonance Imaging)</td>
</tr>
<tr>
<td>Min(s)</td>
<td>Minute(s)</td>
</tr>
<tr>
<td>NIC</td>
<td>No identifying code</td>
</tr>
<tr>
<td>PI</td>
<td>Principal Investigator</td>
</tr>
<tr>
<td>Q</td>
<td>Question/statement (in focus group discussion)</td>
</tr>
<tr>
<td>QD</td>
<td>Qualitative Description</td>
</tr>
<tr>
<td>R</td>
<td>Researcher</td>
</tr>
<tr>
<td>RA</td>
<td>Rheumatoid Arthritis</td>
</tr>
<tr>
<td>S</td>
<td>Supervisor</td>
</tr>
<tr>
<td>SIGN</td>
<td>Scottish Intercollegiate Guideline Network</td>
</tr>
<tr>
<td>sICAD</td>
<td>spontaneous Internal Carotid Artery Dissection</td>
</tr>
<tr>
<td>sVAD</td>
<td>spontaneous Vertebral Artery Dissection</td>
</tr>
<tr>
<td>VA</td>
<td>Vertebral Artery</td>
</tr>
<tr>
<td>VAD</td>
<td>Vertebral Artery Dissection</td>
</tr>
<tr>
<td>VBI</td>
<td>Vertebrobasilar Insufficiency</td>
</tr>
<tr>
<td>5Ds And 3Ns</td>
<td>(Framework that includes: dizziness, diplopia, dysarthria, dysphagia, drop attacks, ataxia, nausea, numbness, nystagmus)</td>
</tr>
</tbody>
</table>
Glossary of Technical Terms

Arterial dissection  Separation of the layers of the artery wall
Ataxia  Gross lack of coordination of muscle movement
Dizziness  Light-headedness, a lack of mental clarity, or frank vertigo (feeling of you or your surroundings are moving)
Drop attacks  Sudden spontaneous falls while standing or walking, with complete recovery in seconds or minutes
Diplopia  Double vision
Dygeusia  Distortion or disturbance in the sense of taste
Dysarthria  Motor speech disorder
Dysphagia  Difficulty in swallowing
Haemodynamics  Movements and forces involved in circulation of the blood
Horner’s syndrome  Drooping of upper eye lid; decreased pupil size on affected side (miosis); decreased or absent sweating on affected side of face (anhidrosis)
Lower motor neuron lesion  condition affecting nerve fibres travelling from spinal cord to muscles
Nystagmus  Involuntary eye movement
Paraesthesia  Abnormal sensation e.g. tingling, burning, prickling
Presyncope  An episode of near fainting - May include light-headedness, dizziness, blurred vision of severe weakness
Ptosis  Drooping upper eyelid
Syncope  A faint – temporary loss of consciousness
Upper motor neuron lesions  conditions affecting motor neurons in the brain or spinal cord e.g. stroke, brain injury, cerebral palsy
Chapter 1. Introduction

Musculoskeletal physiotherapists provide assessment and treatment for pain and functional impairments relating to the musculoskeletal system, for example back and neck pain. In order to apply safe and effective treatment to these conditions it is vitally important that any underlying serious complaints have been excluded. Clinical indicators or danger signs known as ‘red flags’ have been developed as a diagnostic triage or screening tool to help identify any possible serious underlying condition, such as cancer. This process helps reduce the risk of physiotherapy treatment causing further harm and improve the chance of early detection of more serious conditions presenting as a musculoskeletal problem.

Whilst red flags for musculoskeletal back pain have been developed (e.g. Clinical Standards Advisory Group - CSAG 1994) and widely accepted, a change in provision of a Scottish-based regional musculoskeletal spinal service (adult population) has given rise for the need to include more specific red flag indicators or diagnostic screening for neck related pain or functional impairment.

Development of local clinical guidelines that included neck or cervical spine red flag screening focused around Coman’s (1986) 5Ds framework (dizziness, diplopia, dysarthria, dysphagia and drop attacks). These clinical indicators are frequently quoted in physiotherapy literature regarding screening for signs and symptoms of vascular insufficiency, which have potential to develop as an adverse event, such as transient ischemic attack (TIA), stroke, or potentially death following treatment to the cervical spine. Although such events are a rare occurrence, the outcome for both patient and carer are catastrophic.

Discussions with a medical advisor (consultant neurologist) regarding the development of the regional guidelines identified that the 5Ds were not reliable indicators of serious cervical vascular pathology. On reviewing the evidence base used to inform these discussions it became apparent that the information was extracted from physiotherapy-based literature. This literature was limited to clinical commentary or masterclass type publications (e.g. Kerry and Taylor 2006), which do however, use medical-based literature to inform their recommendations, and single case reports (e.g. Taylor and Kerry 2005).
Indeed, the medical advisor’s opinion on the limitations of the 5Ds framework is supported by a commissioned report (Kerry et al 2007). However, this framework continues to be used within the physiotherapy evidence base, albeit with progressive development since its initial introduction following the publication of Coman (1986). This process now includes additional clinical features (ataxia, nausea, numbness, and nystagmus) to form an extended framework known as the ‘5Ds And 3Ns’.

A literature review designed to investigate this framework and other neck related serious pathologies, such as cord compression, is explored in chapter 2. This review identifies that a number of limitations and significant inconsistencies exist within the evidence base.

Kerry and Taylor’s (2006; 2008) physiotherapy masterclass type publications demonstrate that improvements have been made within recent years by the physiotherapy profession to better understand serious pathology related to the cervical spine. However, there appears to be a lack of high level observational evidence originating from the musculoskeletal physiotherapy-based journals to suitably inform physiotherapy practice. In addition, no systematic reviews were identified that specifically examines cervical spine red flags for serious pathology.

Therefore, this suggests that a knowledge gap remains in physiotherapy screening methods for musculoskeletal neck related problems with specific reference to local neurological and neurovascular pathology. This presents a serious clinical problem where physiotherapists administering therapeutic intervention may be faced with patients presenting with early signs of stroke or who may be at risk of developing stroke through the presence of benign arterial pathology. This scenario requires a high level of knowledge to detect potential serious pathology and prevent progression to an adverse event (Kerry and Taylor 2006; 2008).

As stated earlier, red flags for back pain have been developed (CSAG 1994) and widely accepted, as exemplified through integration in more recent guidelines for low back pain (van Tulder et al 2006; NICE 2009). Although the red flag list developed by CSAG (1994) contains components that apply to the whole spine, such as cancer
or infection, the uncertainty that exists for the cervical spine emphasises a need to review and develop the physiotherapy knowledge base for neck related red flag indicators.

**Aim**

The aim of this study is to develop an evidence based list of red flag indicators for neck related problems that would equate to, or complement, the list of red flags for back pain. This screening process for early recognition of potentially serious neurological and neurovascular pathology could enhance safe application of treatment to meet quality ambitions, such as NHS Scotland’s (Scottish Government 2010) quality strategy to deliver safe, effective and person centred care. The key ambition behind this study is summarised in the following overarching research question to be addressed within this thesis:

What pathologies, including their signs and symptoms and risk factors, should be considered as red flags when screening for serious pathology in neck related musculoskeletal disorders?

This study is designed to combine a literature review with engagement from expert physiotherapists and medical consultants to develop a process that addresses the knowledge gap within this overarching research question.
Chapter 2. Literature Review

2.1 Literature review methodology

The Scottish Intercollegiate Guideline Network (SIGN) (http://www.sign.ac.uk/guidelines/fullt/50/index.html) methodology for conducting a literature review was adapted for use by a single reviewer. Key question(s) are defined followed by a systematic review process involving a literature search for existing evidence based guidelines, systematic reviews and meta-analyses. Literature is graded according to the SIGN levels of evidence (section 2.1.1). If sufficient evidence is not available to answer the specific question(s), then the literature search is expanded to include studies of quality as detailed within the inclusion criteria point 1 (Section 2.1.3).

2.1.1 SIGN Guidelines: Levels of evidence

(http://www.sign.ac.uk/pdf/sign50.pdf)

1++ High quality meta-analyses, systematic reviews of Randomised Controlled Trials (RCTs), or RCTs with a very low risk of bias

1+ Well conducted meta-analyses, systematic reviews, or RCTs with a low risk of bias

1 - Meta-analyses, systematic reviews, or RCTs with a high risk of bias

2++ High quality systematic reviews of case control or cohort studies

High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal

2+ Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal

2 - Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal

3 Non-analytic studies e.g. case reports, case series

4 Expert opinion

2.1.2 Literature review search strategy

The databases searched were EBSCO CINAHL, MEDLINE, and the Cochrane Library. Searches were performed using a combination of key words (Refer to Appendix A for detailed search strategy and summary of selected studies used to inform the main literature review). The search was reduced to include peer-
reviewed articles with abstracts available and published in English from January 2002 to October 2012. In addition, the initial searches were supplemented by manually searching the reference sections in those studies retrieved from the initial search.

Studies were selected to inform three phases of this project (Refer to chapter 3 for full details of phases). The main function of the literature review within these three phases was for use in preparatory work for the following:

a. Focus group information pack (within phase 1). This included summaries of key studies used to inform the focus group discussion.

b. To inform construction of a draft clinical chart (within phase 2) that would provide context of the clinical scenario for Delphi survey participants.

c. To inform construction of the Delphi survey (within phase 3).

(This work is discussed in chapter 3 alongside signposting to relevant appendices).

Studies were selected on the basis of the inclusion/exclusion criteria as outlined in section 2.1.3, below.

2.1.3 Inclusion criteria were as follows:

1. Literature selected in the following hierarchical order (Adapted from SIGN: http://www.sign.ac.uk/pdf/sign50.pdf):

   A: Guidelines, meta-analyses, systematic reviews. If evidence is not sufficient to address the questions under consideration, progress to B.
   
   B: Randomised control trials. If evidence is not sufficient to address the questions under consideration, progress to C and D.
   
   C: Case control or cohort studies.
   
   D: Non-analytic studies: Case series reporting 5 or more cases (Haneline and Lewkovich 2004).

2. Studies describing signs, symptoms, clinical tests and/or risk factors for pathologies included in the research questions.

3. Adults (≥16 years).
2.1.4 Exclusion criteria were as follows:
1. Literature graded as lower level of quality:
   A: Case series of less than 5 cases (Haneline and Lewkovich 2004).
   B: Single case studies.
   C. Expert opinion.
   **Note:** Exclusions A-C may be used to provide supplementary statements within the literature review or to support areas within the clinical chart where studies meeting the inclusion criteria are considered to have limited supporting evidence.
2. Literature not describing signs, symptoms, clinical tests and/or risk factors to answer the specific questions informing the literature search strategy.
3. Paediatric (infants, children or adolescent <16 years) based studies.

2.2 Literature review introduction
In order to make a valid risk assessment prior to therapeutic intervention, knowledge development of haemodynamic principles, pathophysiology, risk factors and clinical signs of Cervical Arterial Dissection (CAD) are considered essential (Kerry and Taylor 2006; 2008). This presents a significant clinical challenge to clinicians as demonstrated by Rubinstein et al’s (2006) attempt to aid understanding and clinical reasoning of the pathogenesis of CAD through their theoretical model outlining major risk categories. Although CAD can present initially with benign clinical signs and symptoms it is a major cause of stroke in young to middle age adults (35-50 years of age), therefore, early recognition and appropriate management is of paramount importance (Leys et al 2002; Debette and Leys 2009). This has added importance as there is a tendency for clinicians to believe that more gentle manual treatment techniques in neck pain are relatively risk free (Sweeney and Doody 2010). This appears as a reasonable assumption; however, there is no empirical data to support this statement.

Similarly, serious neurological conditions, such as cervical myelopathy (spinal cord compression) that may result from cervical spondylosis (degenerative spine) may present only after delayed diagnosis and operations for misdiagnosis e.g. carpal tunnel syndrome (Meyer et al 2008). Cervical degenerative changes are the most likely cause of cervical myelopathy, however other considerations for differential diagnosis may include intracranial pathology, intradural tumour or syrinx, multiple
sclerosis, amyotrophic lateral sclerosis or Guillain Barre syndrome (Edwards et al 2003). Unfortunately, a delayed diagnosis could have a detrimental effect on an individual’s functional recovery e.g. gait, bladder control and hand function if not detected at an early stage (Meyer et al 2008). Screening tests, therefore, are utilised to achieve early exclusion of selected diagnosis or impairments. These should be cheap, relatively accurate and not cause further complications during their application (Cook et al 2007).

The range of neurological and neurovascular presentations are too numerous to discuss in this review. This literature review will rather briefly outline the epidemiology and pathogenesis of neurological and neurovascular complications or masqueraders linked with Coman’s (1986) 5D’s, as a screen for cervical arterial pathology, and other clinical presentations that create challenging differential diagnosis scenarios in musculoskeletal physiotherapy practice. Examples of such additional challenges are cervical myelopathy, headaches and dizziness presentations. As stated in Chapter 1, dizziness forms part of the 5Ds. Headaches and dizziness are included as they may feature as patient symptoms, but have multiple causes of onset, such as CAD or upper cervical spine dysfunction. In addition, adverse events associated with clinical practice will be considered to provide context for this review. Together, these highlight the significant clinical challenge of differential diagnosis and the requirement to consider a list of clinical features that would be considered as red flags for serious pathology.

The review will examine two key areas; cervical arterial dysfunction and cervical myelopathy. The review will specifically consider cardinal signs and symptoms, main risk factors (where applicable), including theoretical modelling that may contribute to the screening process, and relevant screening tests with an objective to identify any inconsistencies or gaps in the current knowledge base. The findings will direct this research project, which has an overarching aim to support clinical decision making skills through developing appropriate screening methods for potential neurological and neurovascular conditions or complications presenting as, or parallel to, cervical spine musculoskeletal disorders.
2.3 Cervical arterial dysfunction (includes dizziness and headaches)

2.3.1 Epidemiology and pathogenesis

The cervical arteries are more vulnerable to injury compared to vessels of similar size as they are more mobile in the cervical spine (Schievink 2001). Spontaneous CAD (sCAD), which may involve the internal carotid artery (ICA) and/or vertebral artery (VA) can affect all age groups, but mainly affects young and middle-aged adults between the ages of 35-50 years (Schievink et al 1994; Schievink 2001). Community based studies in the USA and France have reported the annual incidence of spontaneous ICA dissection (sICAD) as 2.5 per 100,000 and 3 per 100,000 per year (Schievink et al 1993; Giroud et al 1994). The annual incidence of VA dissection (VAD) has been estimated to be 1-1.5 per 100,000 per year (Schievink 2001). These ranges of incidence are probably an underestimation of CAD as cases with reduced clinical signs may remain undiagnosed (Debette and Leys 2009).

A combination of an underlying arteriopathy and an additional factor, such as minor trauma is a probable mechanism of CAD onset (Schievink and Debette 2011). Thanvi et al’s (2005) review paper of CAD, however, questions the theory of an underlying arteriopathy as it does not explain specific sites of dissections, low recurrence rates, and rare cases of familial history of CAD. Case control studies involving biopsies of CAD patients compared to patients free of vascular disease e.g. accident victims, identified signs of tissue weakening along the Tunica Media (middle layer) and Tunica Adventitia (outer layer) junction of the artery in all of the spontaneous CAD patients, but not in any of the control samples. These findings suggest a generalised arteriopathy leading to haematoma formation. This formation can be a source of somatic head/neck pain and can further cause arterial wall instability in sCAD patients, which can lead to dissection (tearing) (Volker et al 2005; 2011). It is this mechanism that can result in stroke onset, which may be accompanied by additional symptoms, such as dizziness. There are several limitations to Volker et al’s (2005; 2011) studies with low numbers, non-blinded investigators and the use of the superficial temporal artery as a surrogate (vessel representing a similar function) for the cervical arteries (Schievink and Debette 2011). In addition, these studies use autopsy samples used as controls (e.g. accident victims free of vascular disease) obtained from a separate European country. This is acknowledged by the authors.
This presents a clinical problem of how to identify patients with an arteriopathy if they present to musculoskeletal clinics complaining of head and/or neck pain with or without associated symptoms, such as dizziness. Dizziness accounts for approximately 5% of reasons for attendance at primary care clinics and can be difficult to differentially diagnose from other conditions as the symptoms are frequently non-specific (Post and Dickerson 2010). Therefore, a high level of clinical awareness is required to detect a potential CAD (Thanvi et al 2005; Greenhalgh and Selfe 2006; Kerry and Taylor 2006, 2008).

2.3.2 Clinical presentation (cervical arterial dysfunction) – Background problem of ‘Classical’ cardinal symptoms and signs

Rivett et al (2006 p.3) recommends:

“In every patient presenting with upper quadrant dysfunction, questioning is specifically directed to determine the presence of dizziness which is the most common presenting symptom of VBI. If dizziness is present, other symptoms associated with VBI should be sought...”

No references are provided to support the cardinal signs and symptoms provided in Rivett et al (2006), which is a similar list referred to by Kerry and Taylor (2006 p.244-245) as “Classically, signs and symptoms related to hindbrain ischaemia are considered as the “5 Ds And 3Ns” of Coman (Coman 1986)”. This list is Dizziness (vertigo, giddiness, light-headedness), Drop attacks, Diplopia, Dysarthria, Dysphagia (+ hoarseness/hiccups), Ataxia, Nausea, Nystagmus, Numbness (unilateral). Coman’s (1986) list of cardinal signs and symptoms is not referenced. Kerry and Taylor (2006), however, advise that unreasoned adherence to this list may result in an incomplete understanding of the patient’s presentation, and in a commissioned report, Kerry et al (2007) state there is no support for Coman’s 5 Ds.

Table 2.1 summarises literature findings that questions the suitability of the 5D And 3N framework as an appropriate screening aid in neck pain/headache presenting at physiotherapy musculoskeletal clinics. This further supports the medical advisor’s opinion as outlined in Chapter 1 (Introduction). Kerry and Taylor (2006, 2008) and Kerry et al (2007), are the only authors to question the 5Ds And 3Ns framework through a literature review approach.
<table>
<thead>
<tr>
<th>Reference</th>
<th>Symptoms and signs</th>
<th>Study authors’ comments</th>
<th>Researcher comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coman (1986)</td>
<td>5Ds</td>
<td></td>
<td>Book chapter. No supporting references.</td>
</tr>
<tr>
<td>Grant (1994)</td>
<td>5Ds (Coman) plus visual disturbances, visceral and vasomotor disturbances (e.g., nausea, faintness, light-headedness), perioral sensory changes</td>
<td>Cite Coman (1986) and Williams and Wilson (1962).</td>
<td>Book chapter. Williams and Wilson (1962) not available, but is reported as a review of 20 major and 65 minor cases of basilar insufficiency.</td>
</tr>
<tr>
<td>Thiel and Rix (2005)</td>
<td>5Ds And 3Ns framework.</td>
<td>State ‘adapted from Sturzenegger (1993), Saeed et al (2000).</td>
<td>Papers cited are small studies with patients presenting at neurological units with stroke. Mainly formed part of presenting features i.e. at development of stroke rather than early warning signs.</td>
</tr>
<tr>
<td>Rivett et al (2006)</td>
<td>Similar to 5Ds And 3Ns.</td>
<td></td>
<td>Clinical guidelines. No references for these signs and symptoms.</td>
</tr>
<tr>
<td>Kerry and Taylor (2006)</td>
<td>5Ds And 3Ns - Unreasoned adherence may result in an incomplete understanding</td>
<td></td>
<td>Masterclass type publication.</td>
</tr>
</tbody>
</table>

**Conclusion:** It is therefore questionable whether the 5D And 3N framework is an appropriate screening aid in neck pain/headache presenting at a physiotherapy musculoskeletal clinical setting.
Furthermore, Debette, Grond-Ginsbach et al’s (2011) large multicentre prospective study presents differential features of carotid and vertebral artery dissections. Unfortunately, this report does not offer specific detail on aspects of pain presentation or ischemic signs that may aid musculoskeletal physiotherapists in their identification of such pathologies. In addition to the uncertainties suggested above with regards to the 5Ds And 3Ns framework, this recent study highlights the difficulty in establishing patterns of presenting features to assist physiotherapy clinical decision making.

2.3.3 Clinical presentation (cervical arterial dysfunction) – Literature review for ‘Classical’ cardinal symptoms and signs

A systematic review process was used to investigate the signs and symptoms relative to the 5D And 3Ns framework and ascertain if this framework is a reasonable clinical approach for identifying possible serious neurovascular pathology. To enhance clarity of discussion the local symptoms of neck pain, headache, and tinnitus are addressed separately in section 2.3.5.

In addition to headache and neck pain, Thomas et al's (2011) retrospective case control study of a younger patient group (<55-years) comparing cervical arterial dissection cases with non-dissection causes of stroke identified the following symptoms and signs:

“Symptoms: Dizziness, visual disturbance, paraesthesia (face, upper and lower limb).


In general, visual disturbances were reported, however, observations of nystagmus was rarely recorded. Ataxia or balance problems were quite frequent in the VBAD group (18:67%) and in less than half of ICAD cases (9:45%).
This study was designed for the purpose of informing clinical practice of manual therapists e.g. physiotherapists, therefore it is useful to have symptoms and signs separated. As a retrospective study, it is however, vulnerable to bias. The authors acknowledge that medical records are not always detailed and that negative responses to questions in the history may not always be recorded. Thomas et al (2011) also observed that details of blood results and radiological imaging were sometimes limited.

Vertebrobasilar ischemia typically presents with a collection of symptoms and signs, such as motor or oculomotor signs, and rarely causes only one symptom (Savitz and Caplan 2005). Savitz and Caplan’s (2005) review paper is linked to Caplan et al’s (2004) large prospective study (n=407) in which <1% of patients with vertebrobasilar ischemia had only one presenting symptom or sign. Frequent symptoms of vertebrobasilar-artery occlusive disease are dizziness, vertigo, headache, vomiting, double vision, loss of vision, ataxia, numbness, and weakness with bilateral body structure involvement (Savitz and Caplan 2005). These features support Thomas et al’s (2011) retrospective observations. Savitz and Caplan (2005), however, also state that dizziness and drop attacks are often incorrectly apportioned to posterior-circulation (vertebral artery) ischemia. Pelkonen et al (2004) in reporting on pulsatile tinnitus also reported that dysgeusia (taste disturbance) was observed in two cases (from 16 pulsatile tinnitus cases). Dysgeusia is not mentioned by Thomas et al (2011) or Savitz and Caplan (2005).

Bassi et al’s (2003) prospective study (n=49) of arterial dissections mainly spontaneous in nature, reported local neurological manifestations were present in 15 patients (30.6%): this represents less than one-third of patients. The majority of patients (41:83.6%) had ischemic cerebral symptoms. No specific detail is provided on the latter. Eighty per cent of strokes are ischemic with one-quarter of ischemic events being apportioned to posterior (vertebrobasilar) circulation (Savitz and Caplan 2005).

Debette, Grond-Ginsbach et al’s (2011) large multi-centred prospective study subdivides cerebral ischemia into four components: a. Ischemic stroke; b. TIA; c. transient monocular blindness; and, d. Subarachnoid haemorrhage. Ischemic cerebral symptoms are reported as the main presenting complaint in a number of
patient-centred studies of varying levels of quality (Chaves et al 2002; Dziewas et al 2003; Arnold, Bousser et al 2006; Lee et al 2006; Chandra et al 2007; Huang et al 2009; Gui et al 2010). The nature of these ischemic components suggests that the majority of patients presented with more severe signs and symptoms that are very unlikely to present at musculoskeletal clinics.

Drop attack also forms part of the 5Ds. Savitz and Caplan (2005) reporting on Caplan et al (2004) identified that no episodes of drop attack occurred in isolation within this large study (n=407). Savitz and Caplan (2005) consider that weakness of the legs is more likely to be persistent if caused by brain-stem ischemia with its affect on corticospinal tracts and motor control of the legs. This questions the rationale behind drop attacks being included within the 5Ds And 3Ns framework for identifying vertebrobasilar insufficiency. Similarly, loss of consciousness is more likely related to seizures and syncope (a faint) than cerebrovascular disease.

Thomas et al (2011) additionally reported the signs of ptosis (Horner’s sign/syndrome), facial palsy and upper and lower limb weakness were the most common signs in ICAD (anterior circulation system). This supports Baumgartner et al (2001) who identified the main significant local signs in ICAD without ischemic development (n=55) was Horner’s syndrome and lower cranial nerve palsies. Ptosis or Horner’s sign/symptom or oculosympathetic palsy was reported in approximately 25-30% of cases (Bassi et al 2003; Dziewas et al 2003; Lee et al 2006). Ptosis or Horner’s sign and facial palsy, however, are not included in the 5Ds And 3Ns framework. Within the literature there appears to be an interchangeable use of the terms ptosis, Horner’s syndrome and Horner’s sign (drooping eyelid).

2.3.4 Dizziness
Dizziness is a symptom within the 5Ds And 3Ns framework. The term ‘dizziness’ may also encompass light headedness, a lack of mental clarity or frank vertigo, and is reported to be a frequent symptom of vertebro-basilar-artery occlusive disease (Savitz and Caplan 2005). Tarnutzer et al (2011) similarly use the term dizziness to encompass vertigo, presyncope, unsteadiness, and other non-specific forms of dizziness. However, of note, is Bhattacharyya et al’s (2008) interchangeable use of the terms dizziness and vertigo, and may mention light-headedness alongside these terms within their clinical practice guideline for Benign Paroxysmal Positional Vertigo (BPPV). This inconsistency in use of terminology highlights a further potential
source of confusion for physiotherapists in attempting to improve their differential diagnosis knowledge of peripheral and central causes of dizziness.

Savitz and Caplan (2005) consider that vertigo indicates dysfunction of the peripheral vestibular or central vestibulocerebellar system. This is significant as Tarnutzer et al’s (2011) systematic review reports that vertebrobasilar ischaemic stroke may closely mimic peripheral vestibular disorders, with obvious focal neurologic signs absent in greater than half of patients presenting with acute vestibular syndrome due to stroke.

Furthermore, Thomas et al (2011) identify that dizziness is often emphasised as a primary clinical indicator of vertebrobasilar flow insufficiency (e.g. Rivett et al 2006; Maitland 2005 in Thomas et al 2011). However, Thomas et al’s (2011) retrospective case control study revealed that dizziness presented in only 32% (15) of total dissection cases versus 7% (3) of non-dissection cases. This study is based within a specialty setting. Bhattacharyya et al’s (2008) clinical guidelines reports that evaluation of patients presenting with vertigo in a non-specialty setting found that BPPV and vestibular neuritis, accounted for most of the cases with 42% and 41%, respectively. The remaining causes were apportioned to Ménière's disease (10%), vascular causes (3%), and other causes (3%).

Missed diagnosis of stroke at first medical contact within emergency departments is often linked to dizziness with 35% of cerebrovascular events in patients with any dizziness and 44% in those with isolated dizziness reported to be have been missed at this stage (Tarnutzer et al 2011). The authors add that available data suggests that patients with misdiagnosis are at particularly high risk of poor outcomes. This emphasises the requirement for accurate screening for potentially serious pathology that may present at musculoskeletal physiotherapy clinics.

Vertebral-artery disease can cause transient attacks of vertigo; however, this is usually accompanied by other brain-stem or cerebellar symptoms. Light-headedness typically indicates presyncope related to circulatory, systemic, or cardiac disease rather than vertebral artery disease (Savitz and Caplan 2005). Savitz and Caplan’s (2005) review following Caplan et al’s (2004) posterior circulation registry (n=407) observed that isolated episodes of vertigo continuing for more than three weeks was almost never caused by vertebrobasilar disease, and
that only 7% (of n = 407) described light-headedness. No patients presented with light-headedness as an isolated symptom. This type of information may help guide differential diagnosis at first point of contact. These relatively low numbers questions the accuracy of using the symptom of dizziness as a primary indicator of cervical arterial pathology and perhaps the focus should be on a combination of symptoms/signs. Caution, however, should be exercised if focusing on a combination of symptoms/signs as demonstrated by the earlier report of Tarnutzer et al's (2011) systematic review identifying that missed diagnosis of stroke at first medical contact within emergency departments is often linked to dizziness.

As previously mentioned, Savitz and Caplan (2005) consider that vertigo indicates dysfunction of the peripheral vestibular or central vestibulocerebellar system. BPPV is a peripheral vestibular dysfunction. Clinical practice guidelines have been developed for BPPV that include guidance on diagnosing and differentiating peripheral and potential central neurological causes for dizziness (e.g. migraine-associated vertigo, vertebrobasilar insufficiency, and intracranial tumors), as central causes may have more serious medical implications. This presents a significant clinical challenge for differential diagnosis (Bhattacharyya et al 2008). Bhattacharyya et al (2008) outline other less serious or self limiting causes for differential diagnosis are; otological (e.g. Meniere’s disease, vestibular neuritis or labyrinthitis), and other entities (e.g. Anxiety or panic disorders, cervicogenic vertigo, medication side effects, and postural hypotention).

Two systematic reviews were identified that considered differential diagnosis of peripheral and central causes of dizziness (Dros et al 2010; Tarnutzer et al 2011).

Dros et al (2010) conducted a systematic review with a clearly defined question and methodology to investigate tests used to evaluate dizziness in primary care to determine differentiation between self limiting conditions and serious conditions requiring referral or immediate treatment. Most musculoskeletal physiotherapy clinics, including those sited within hospitals, are likely to operate in a primary care format e.g. accepting General Practitioner or self-referrals in addition to those generated from medical consultant sources. Therefore these systems require careful consideration to their clinical ability to diagnose dizziness within primary care settings.
Dros et al (2010) report four tests for neuro-otologic conditions that were evaluated in more than one study: a. Dix-Hallpike manoeuvre; b. Head-shaking nystagmus test; c. Head impulse test; and d. Vibration-induced nystagmus test. This review concluded the following:

1. “Studies on diagnosing dizziness have been conducted in highly selected homogeneous groups of patients only e.g. within secondary care. Secondary care settings are likely to have higher prevalence rates and severity of conditions compared to primary care due to it receiving specific conditions” (p.E621);

2. “Evidence to support the diagnostic process in primary care is scarce” p.E621). Studies were not diagnostic or of poor methodological quality, and;

3. Two tests (head-shaking nystagmus and head impulse tests) however, differed to this report. Dros et al (2010 p.E630) report that these two tests demonstrated a similar prevalence of peripheral vestibular dysfunction in primary care based patients complaining of dizziness when compared to other target groups.

Tarnutzer et al’s (2011) systematic review similarly considered the differential diagnosis of an acute peripheral vestibular syndrome and stroke.

“A three-component bedside oculomotor examination – HINTS (horizontal head impulse test, nystagmus and test of skew) is reported to identify stroke with high sensitivity and specificity in patients with acute vestibular syndrome and rules out stroke more effectively than early diffusion-weighted MRI in the acute phase” (Tarnutzer et al’s 2011 p.1025).

These findings are encouraging. However, clinicians also need to consider if these tests are appropriate for non-medical musculoskeletal clinical settings.

Dros et al (2010) provide web-links to the tests covered in their study. On review, clinical experience suggests that these tests (head-shaking nystagmus and head impulse tests) are too aggressive to perform on a patient complaining of neck pain.
Furthermore, if from a musculoskeletal screening perspective the aim is to exclude potentially serious pathology then the application of vigorous tests has potential to prematurely progress a dissecting artery. Tarnutzer et al (2011) recommends that no provocative tests, including the gentler Dix-Hall pike test, should be applied in an acute setting.

Tarnutzer et al’s (2011) systematic review is primarily aimed at establishing differential diagnosis of acute vestibular syndrome of peripheral cause from a central cause. Acute vestibular syndrome typically presents with dizziness onset of 24-48 hours, rather than the shorter transient dizziness as seen in other conditions e.g. TIA, BPPV.

Tarnutzer et al (2011 p.1031) conclude:

“Red flags for stroke probably include a history of multiple transient prodromal episodes of dizziness over weeks or months; auditory symptoms; and headache, neck pain or recent trauma. Best evidence suggest that nearly two-thirds of patients with stroke lack focal neurologic signs that would be readily apparent to a non-neurologist and one-third lack signs that would be readily apparent to a neurologist”.

It is highly unlikely that acute vestibular patients may present at a physiotherapy department. However, as it aims to differentiate from a central cause, the information on the central type presentations has potential for extraction for clinical use by non-medical musculoskeletal clinicians.

2.3.5 Pain (Background problem and cervicogenic headache)

Cervicogenic headache is considered a disorder that is manageable by the physical therapies (Jull 1997). This disorder has been recognised by the International Headache Society (IHS 2004). Part of the criteria referred to by the IHS is pain referred from a source in the neck and felt in one or more regions of the head and/or face. Differential diagnosis of cervicogenic headache, however, can be difficult to separate from other causes of headache unless additional features are presented. Some physiotherapy studies have attempted to address this differential diagnosis problem by comparing examination findings in headache groups of cervicogenic and migraine with aura, and asymptomatic controls and have identified upper cervical
spine mechanical dysfunction (Zito et al 2006; Ogince et al 2007). The most crucial differential diagnosis is headache from CAD due to the heightened risk of adverse events with potentially near fatal consequences following manipulation (Bogduk and Govind 2009).

An additional problem with headache differentiation is that neurologists differ in their agreement of cervicogenic headache as having a nosological identity making the concept of cervicogenic headache controversial (Leone et al 1998; Zhou 2008; Bogduk and Govind 2009). This adds greater complexity to clinicians navigating their clinical reasoning processes when a patient presents complaining of neck pain and headache.

2.3.5.1 Pain (Overview - Cervical arterial dysfunction)
Neck pain and/or headache symptoms are the most frequent local symptoms of CAD (Silbert et al 1995; Savitz and Caplan 2005; Taylor and Kerry 2005; Arnold, Cumurciuc et al 2006; Kerry and Taylor 2006; Chandra et al 2007; Hardmeier, Gobbi et al 2007; Morelli et al 2008; Rigamonti et al 2008; Tobin and Flitman 2008; Thomas et al 2011), and additionally, can be the only presenting symptoms (Bioussse et al 1992; Bioussse et al 1994; Guillon et al 1998; Arnold, Cumurciuc et al 2006). Furthermore, VAD has also been reported as presenting as a fifth cervical nerve root (C5) radiculopathy (Arnold, Bousser et al 2006; Hardmeier, Haller et al 2007). Note, Rivett et al (2006) stated that dizziness was the most frequent symptom. However, they also advise to check for presence of neck pain or headache. Savitz and Caplan (2005) state that the cardinal symptom in patients with vertebral dissections is occipital or posterior neck pain with diffuse headache also occurring; however, no supporting data is presented. Savitz and Caplan’s (2005) review paper is linked to Caplan et al’s (2004) large prospective study (stroke registry).

For VBI, Rivett et al (2006 p.3) cite Haldeman et al (2002) and Krespi et al (2002) to add that pain is “Specifically, sudden, severe, sharp pain located in the ipsilateral postero-superior region of the neck and occiput and for which there is no past history should be regarded as suspicious”. This does not include ICAD. Kerry and Taylor (2006) describe acute onset neck pain/headache as “unlike any other”, but are these patterns and distributions definitive in CAD?
In considering clinical recommendations (e.g. Rivett et al 2006; Kerry et al 2007), Thomas et al (2011) noted that headache was not always present or severe in either VBAD or ICAD subjects. However, headache was more prevalent in VBAD (85%) and ICAD (75%) subjects when compared to an age-matched control group of non-dissection stroke patients (51%). This latter point differs to Debette et al (2011) who reported that headache was more prevalent in ICAD > VAD, which supports Chandra et al’s (2007) small retrospective study (n=20) reported the most common symptom on presentation in both SCD and SVD patients was headache (83% SCD, 78% SVD).

Thomas et al (2011) and Debette, Gronsbach et al (2011), however, both reported that neck pain was more likely in VBAD than ICAD subjects. Neck pain and headache are symptoms that can present at musculoskeletal clinics without prior attendance at a medical practitioner, or could be the reason for referral from the medical practitioner. Therefore, more specific detail of description and pattern of such pain presentations may assist in the differential diagnosis of more serious causes.

2.3.5.2 Neck pain and headache in cervical arterial dysfunction (patterns and characteristics)
Arnold, Cumurciuc et al (2006) examined cases of sCAD that presented with pain only (without additional neurologic manifestations). By using a hospital-based registry 20 from 245 patients (8%) mean age 39-years (±8) were identified. Six patients presented with headache only, 2 with neck pain only, and 12 with both. Twelve had VAD, 3 had ICAD and 5 had multiple dissections. There was no clear pattern of headache and neck pain characteristics. This supports Silbert et al (1995) and Biousse et al (1994) who considered varying descriptors of pain: distribution, mode of onset, quality and evolution (constant or intermittent).

Silbert et al (1995) specifically studied the characteristics of headache in 161 consecutive patients presenting at the same location. The mean age of ICAD patients (n=135) and VAD (n=26) was 47 years and 40.7 years, respectively. Biousse et al (1994) investigated pain in non-traumatic ICAD patients (n=65 mean age: 43-years range 14-67) headache was the presenting symptom in 38 (58.5%)
and present at any point throughout the duration in 48 patients (74%). Facial pain and neck pain also presented independently as the initial symptom and a combination of all three was present in 17% of patients (Biousse et al 1994). No clear pattern was demonstrated in CAD related head and neck pain.

Chaves et al’s (2002) report on 10 cases identified that 9 had a stroke (1 had an associated subarachnoid hemorrhage), whereas 1 patient had only transient ischemic attacks. Severe headache (usually retro-orbital, frontal and/or temporal) followed by contralateral hemiparesis was the most common initial clinical symptoms (80%). Although this pain was severe in all cases, the distribution is not specific. Bassi et al (2003) reported headache and neck pain occurred in 32 patients (65.3%), and Lee et al (2006) retrospective study (n=48) reported the occurrence of neck pain in 13 (27%) and headache in 33 (69%). Unfortunately, no information on pattern, distribution or temporal components is provided.

In contrast, Huang et al’s (2009) retrospective study (n=73) identified 22 (55%) had accompanying headache and/or neck pain lateralized to the dissection side. This is supported by a small case series (3 male/4 female age range 35-79 mean +/- 16.2 years: 6VAD, 1 ICAD) that reported all except one had a unilateral distribution of headache or neck pain only (Maruyama et al 2012). Huang et al (2009) also describe pain in patients presenting with sub-arachnoid haemorrhage (SAH) as intense and lateralized to the dissection side. Gui et al’s (2010) prospective study over a 6 year period reported serious parieto-occipital pain with symptoms of posterior-circulation ischemia were the most common manifestations in 10 cases (63%). Headache was the initial symptom in 8 patients (53%). One patient had SAH. However, patients with SAH and acute unilateral hemiparesis are highly unlikely to present at musculoskeletal clinics.

Silbert et al (1995) reported 65 of ICAD patients considered their headache as 'unique' but 45 did not consider a significance difference to previous experience of headache. This does not support Kerry and Taylor’s (2006) statement as having headache “unlike any other”. Cases of CAD presenting with pain only may be under-diagnosed, particularly if it presents similar to previous episodes of pain (Biousse et al 1992; Mirza et al 1998; Arnold, Cumurciuc et al 2006). Silbert et al (1995) also reported that 132 from 135 ICAD patients had accompanying focal
neurological manifestations. Only three had headache only, with/without neck or facial pain. Additionally, Silbert et al (1995) report from 135 ICAD patients, 35 (26%) had neck pain at time of dissection with gradual onset in 25 patients and sudden in 7. Neck pain was the first symptom in only 9 patients.

These studies suggest there is no clear pattern of headache or neck pain that may occur in either VAD or ICAD and demonstrates inconsistency in literature reporting pain presentations related to CAD. These studies typically use a retrospective and/or prospective methodology. For example, Biousse et al (1994) utilises a combined methodological approach with retrospective recording to 1988 followed by a prospective method to 1990. Silbert et al (1995) specifically studied the characteristics of headache in CAD and provide a sound description of a follow-up direct interview with patients, unless a detailed history of the headache according to diagnostic criteria set by the International Headache Society (1988) was recorded by the neurologist.

The study by Debette, Grond-Ginsbach et al (2011) is a high quality large prospective study that reports presenting differential features of carotid and vertebral artery dissections. No specific detail on pattern, distribution or temporal aspect of pain presentation is provided. This study highlights the difficulty for physiotherapists in establishing patterns of presenting features to assist clinical decision making processes. In addition, patients are recruited through large neurological centres that are unlikely to receive patients with local signs, or mild cerebral or retinal ischemia, the type of clinical picture which is more likely to present at a musculoskeletal physiotherapy clinic.

Furthermore, the literature suggests if clinicians focus solely on either the anterior or posterior circulatory system, rather than consider the cervical arteries as a group e.g. Rivett et al's (2006) guidelines (refer to section 2.3.2) highlighting occipital or posterior neck pain with VBI (posterior circulation), then there is a possibility that an ICAD could be undetected if the patient presents with pain in other regions, such as temporal or retro-orbital pain.

As previously stated, Debette, Grond-Ginsbach et al (2011) and Thomas et al (2011) report neck pain is more likely in VAD, but both reports differed with regards
to headache. However, from a physiotherapy clinical perspective it is questionable if this information is relevant to musculoskeletal clinical decision making. Clinical experience suggests that it would be too difficult for a physiotherapist to be able to differentiate between these two arteries, unless additional advanced practice training was received. It may therefore be a reasonable suggestion that physiotherapists should focus solely on whether the presence of a serious arterial pathology exists, rather than trying to further complicate the examination by attempting to ascertain which of the cervical arteries is in a dysfunctional state. This additional information is unlikely to alter the clinician’s subsequent management i.e. refer on to a medical specialist. One counter argument to this suggestion is that this additional information may assist the specific targeting of any further investigations e.g. Duplex ultrasound.

2.3.5.3 Headache – red flags
The Scottish Intercollegiate Guideline Network (SIGN) Guideline 107 Diagnosis and management of headache in adults is a national (NHS Scotland) clinical guideline. Guideline 107 states that secondary headache (i.e. headache caused by another condition other than a primary cause) should be considered in patients presenting with new onset headache or headache that differs from their usual headache.

In addition, observational studies have highlighted the following warning signs or red flags for potential secondary headache which requires further investigation:

Red flag features (SIGN Guideline 107):
- "new onset or change in headache in patients who are aged over 50
- thunderclap: rapid time to peak headache intensity (seconds to 5 mins)
- focal neurological symptoms (e.g. limb weakness, aura <5 min or >1 hr)
- non-focal neurological symptoms (e.g. cognitive disturbance)
- change in headache frequency, characteristics or associated symptoms
- abnormal neurological examination
- headache that changes with posture
- headache wakening the patient up (NB migraine is the most frequent cause of morning headache)
- headache precipitated by physical exertion or valsalva manoeuvre (e.g. coughing, laughing, straining)
- patients with risk factors for cerebral venous sinus thrombosis
- jaw claudication or visual disturbance
- neck stiffness
- fever
- new onset headache in a patient with a history of human immunodeficiency virus (HIV), infection
- new onset headache in a patient with a history of cancer” (SIGN 107 p.9).

SIGN guideline 107 outlines what clinical evaluation should take place e.g. neurological testing including cranial nerve testing and fundoscopy. Cranial nerve testing is advocated by a small number of physiotherapy based publications (Taylor and Kerry 2010; Thomas et al 2011). Unfortunately, clinical observation suggests that cranial nerve testing is not routinely utilised within physiotherapy and fundoscopy is a medical based skill. Therefore, physiotherapists would benefit from additional medical guidance as to what specific features should be considered as being a potential indicator of serious pathology, relative to a musculoskeletal clinic setting. For example, thunderclap type headache is a medical emergency that will not typically present at a musculoskeletal clinic.

2.3.5.4 Pulsatile tinnitus

Pulsatile tinnitus is almost exclusively related to the sound of non-laminar blood flow transmitted to the inner ear occurring from alteration in haemodynamics e.g. arterial dissection, systemic disease, or local disorders within or in close proximity to the petrous bone (Pelkonen et al 2004). Pulsatile tinnitus presented as a symptom in 16 patients within a prospective study (n=136; Pelkonen et al 2004). Ten cases presented with subjective (only heard by the patient), 5 with objective tinnitus (audible to auscultation) and 1 case of it being the only presenting symptom. In one additional case reported within this study, pulsatile tinnitus in a patient with bilateral ICAD was reported to have occurred 3-months after initial symptoms occurred (Pelkonen et al 2004). Pelkonen et al (2004) also reported that 12 patients had headache, 1 with headache and neck ache, and 1 patient with neck ache. Arnold, Bousser et al (2006) and Dziewas et al (2003) also reported tinnitus occurring in a small number of patients 7 (5%) and 8 (6%), respectively. This symptom is not included within the 5Ds And 3Ns framework.
2.4 Temporal aspects
One additional consideration should be given to the temporal aspect of pain onset to additional manifestations in CAD, which could significantly aid physiotherapists' clinical decision making knowledge. This has been reported as a mean 8.8 days delay in ICAD patients and mean 12-day delay in VAD patients (Silbert et al 1995) and from several minutes to 1-month (Mas et al 1987; Bioussé et al 1995). Bioussé et al (1995) reported ≤7 days in 82% of cases. Gui et al (2010) reported neurologic deficits with the onset of sVAD, and symptoms of posterior-circulation ischemia were apparent within 4 days of onset of headache (n=14, 88%). Dziewas et al (2003) reported that 75 (78 %) of patients with stroke reported preceding warning symptoms with 54 patients (56 %) recognising symptoms only minutes prior to the onset of stroke, whereas 42 patients (44 %) noticed between 12 hours and 14 days (median 3 days) before the onset of stroke. Similarly, Chaves et al (2002) reported that neurological signs occurred in most patients (9: 90%) immediately after severe headache (usually retro-orbital, frontal and/or temporal).

With regards to pain duration, Bioussé et al (1994) reported all pain had resolved in less than 30 days: Headache had a duration of 90-minutes to 30-days (mean 5.4 days ± 7.5); neck pain duration from 1-13 days (mean 5.9 ± 4.3); and facial pain lasted 2-hours-15-days (mean 5.3 ± 5.8-dyas). Arnold, Cumurciuc et al (2006) reported all pain resolved within 3-months.

The publications in section 2.3-2.4 referring to signs and symptoms, such the ‘5Ds And 3Ns framework’, including dizziness and headache presentations highlights the difficulty physiotherapists face in clinic when considering differential diagnosis. This creates the question of what subjective (questioning) and objective procedures e.g. cranial nerve testing, should musculoskeletal practitioners use to support awareness of detecting more subtle aspects of neurological change in order to prevent occurrences of an adverse event?

2.5 Adverse events (AE) associated with physical therapies
Identifying AE associated with treatment of neck pain in adults has been problematic due to low quality data in clinical trials and lack of agreement on standardised AE terminology (Carlesso et al 2010). Ernst’s (2007) systematic review, however, concluded that spinal manipulation was frequently associated with mild-moderate
AE and can result in more serious events such as VAD. This is highlighted by Lee et al (1995) who surveyed California, USA based neurologists to gain an estimation of adverse neurological complications following chiropractic manipulation over a 2-year period. From 177 (29%) responses: 55 strokes (1 death), 16 myelopathies and 30 radiculopathies were reported, most of which involved the cervical spine. Despite limitations to this survey, such as it being questionnaire based without validation of the clinical details provided by the respondents, it nonetheless highlights the serious nature of such events. However, defining AE in the physical therapies to enable consistent reporting has proven difficult without context and detail (Carnes et al 2010).

More recently, Sweeney and Doody’s (2010) postal-survey of Manipulative Physiotherapists (n=127) in Ireland to determine the use of cervical spine manual treatment and to describe adverse events associated with these interventions reported the most serious adverse events were associated with more gentle non-manipulation techniques. These included one TIA, one fainting, and one drop attack. There was moderate use of vertebro-basilar insufficiency (VBI) functional screening tests as outlined by Rivett et al (2006). However, of the 26% (n=33) of respondents that experienced an adverse event, 24% (n=8) had conducted VBI testing, whilst 58% (n=19) did not conduct testing. This questions the validity of functional screening tests (section 2.6) as outlined by Rivett et al (2006).

2.6 Clinical tests, functional screening tests, and blood flow studies
Rivett et al (2006) published evidence-based guidelines for assessment of VBI prior to the application of manipulation and mobilisation of the cervical spine, which include provocative testing for patients who report symptoms associated with VBI (e.g. dizziness) during the subjective examination (refer to sections 2.3.3-2.3.4). It is hypothesised that a mechanically induced stress on the VA causing altered blood flow with decreased perfusion to the brainstem will initiate VBI signs and symptoms (Westaway et al 2003). Thiel and Rix (2005) question the continued use of functional pre-manipulation testing of the cervical spine as it could cause added arterial compromise to an underlying vascular pathology resulting in an AE. As stated earlier, screening tests should not cause further harm (Cook et al 2007).
Provocative functional testing, as outlined by Rivett et al (2006), consists of end-of-range cervical/neck rotation held for a minimum 10 seconds, simultaneously examining the eyes for nystagmus and checking for any additional symptoms. Rivett et al (2006) further suggest that symptoms of dizziness provoked by cervical spine causes can be differentiated from a vestibular cause by trunk rotation sustained and moving, whilst keeping the head steady. Also, from a physician’s perspective, Post and Dickerson (2010) report that differential diagnosis of dizziness can be narrowed down with clinical tests, such as evaluating for nystagmus, Dix-Hallpike manoeuvre (a positional test for the vestibular system) and orthostatic blood pressure testing, but 20% of cases will remain undiagnosed beyond these tests. Bhattacharyya et al (2008) suggests several clinical features may suggest a central cause of vertigo rather than BPPV, one of which is nystagmus. Nystagmus may occur in both peripheral and central causes of vertigo. The latter is more strongly suggested if; down-beating nystagmus on the Dix-Hallpike manoeuvre, direction-changing nystagmus occurring without changes in head position (i.e. periodic alternating nystagmus), or baseline nystagmus manifesting without provocative manoeuvres.

These tests could be used by physiotherapists, with the Dix-Hallpike manoeuvre in particular being utilised by clinicians specialising in managing dizziness caused by vestibular dysfunction. Evaluation of nystagmus, however, requires a high level of interpretative skills (Patten 1998). Patten (1998) states that “the importance of testing nystagmus correctly and recording the quality, direction and other features is not sufficiently appreciated……can demonstrate poor clinical technique and a failure to distinguish this from a true physical sign” (p.103).

Additionally, Rivett et al’s (2006) statement oversimplifies the clinical scenario of differential diagnosis for causes of dizziness when compared to Newman-Tolker et al’s (2008) cross sectional analysis investigating the spectrum of dizziness visits to United States Emergency Departments. A total of 9472 dizziness cases over a 13-year period were grouped into the following diagnostic categories of dizziness: otologic/vestibular (32.9%), cardiovascular (21.1%), respiratory (11.5%), neurologic (11.2% including cerebrovascular/stroke), injury/poisoning (10.6%), psychiatric (7.2%), digestive (7.0%), genitourinary (5.1%), and infectious (2.9%). Although these were acute onset cases it highlights the complexities presented to
physiotherapists when attempting to address dizziness as part of a patient’s complaint and should also be considered alongside the current evidence base on cervical artery blood flow studies

A number of blood flow studies investigating the effects of provocative testing on the arterial flow have produced inconsistent results (Kerry and Taylor 2008).

Several studies have reported reduced blood flow in functional testing positions (Rivett et al 1999; Mitchell et al 2003, 2004; Arnold et al 2004). Mitchell et al (2003) reported reduced contra-lateral blood flow in the intracranial VA on full rotation and in a later study Mitchell et al (2004) reported reduced intracranial VA blood flow in healthy participants, but no VBI signs and symptoms. Despite using healthy participants and recreating no signs and symptoms, Mitchell et al (2004) stated that their study supports the use of the VBI test. This is difficult to accept as the reduced blood flow does not correlate with a reproduction of signs or symptoms.

In contrast, several studies have reported no change in flow (Thiel et al 1994; Zaina et al 2003; Bowler et al 2011). Bowler et al (2011) considered both the ICA and VA blood flow in the simulated manipulation position and reported no reduced blood flow in healthy participants. The authors are currently developing this study on patients with signs and symptoms of vascular pathology on pre-manipulative testing. However, blood flow studies of this nature cannot factor in the effect of a manipulative thrust or repeated less forceful manual mobilisations on a potentially dissecting artery.

These examples provide an indication of the inconsistency in blood flow studies and neurological clinical tests. Therefore clinicians also need to enhance awareness of neurological and neurovascular pathology through other methods, such as identifying risk factors.

2.7 Risk factors (Vascular pathology)

2.7.1 Theoretical modelling

Thomas et al (2011) consider that a consensus has not been achieved on definitive risk factors for CAD. Additionally, there is inconsistent evidence between studies investigating risk factors for CAD (Kerry et al 2008; Thomas et al 2011).
Kerry and Taylor (2006) consider CAD to be intrinsically linked to two inter-related principles of an underlying pathology (including atherosclerosis) and mechanical forces. Other features linking to underlying pathology or risk have been suggested as: genetic (e.g. connective tissue disorders, fibromuscular dysplasia (non-inflammatory disease of medium sized arteries), or family history) and environmental origins (e.g. major and minor trauma, infections, smoking, hypertension, oral contraceptives, iatrogenic causes such as, surgery or medical intervention) are also considered a risk (Thanvi et al 2005; Debette and Leys 2009;)

In an attempt to aid understanding and clinical reasoning of the pathogenesis of CAD Rubinstein et al (2006) present a theoretical model that outline four major risk categories of CAD: 1) genetic predisposition/underlying familial disorder; 2) environmental exposure e.g. infection, oral contraceptive; 3) trivial trauma e.g. normal neck movements, sports injury, neck manipulation; and 4) common risk factors associated with atherosclerosis. Rubinstein et al (2006) propose that a genetic predisposition must be present, plus an additional necessary trigger for a CAD event to occur. They argue that a CAD is highly unlikely in an otherwise healthy individual free from this combination of factors. In a thorough review focusing on sCAD Debette and Leys (2009) concluded that studies on genetic association with CAD have been underpowered.

Rubinstein et al (2006) acknowledge that based on poor methodological processes identified in a systematic review (Rubinstein et al 2005), the true risk of CAD to the population remains unknown. Therefore, if manipulation could be a contributing factor, as opposed to a principal cause of CAD, the problem of identifying a young/middle aged person at risk from CAD still remains. This provides a challenging problem to clinicians in how to identify these patients.

2.7.2 Manual therapies and minor trauma

Haldeman et al (2002) concluded after reviewing 64-medical/legal (medical related compensation claims) cases that risk factors could not be identified and that dissection was an unpredictable event. Manipulation of the neck, however, is considered as having a strong association as a risk factor for CAD (Rubenstein et al 2005; Ernst 2007). In addition to Sweeney and Doody (2010) and Lee et al (1995)
(section 2.4), Volker et al (2005) and Mas et al (1987) report cases of VAD following manipulation. Thomas et al (2011) found a statistically significant association between minor mechanical trauma and CAD. The minor trauma included manual therapy (chiropractic, osteopathy, physiotherapy, massage) to the neck; however, what type and extent of therapy was not recorded. Similarly, Kerry and Taylor (2006) consider that altered mechanical forces, such as movement or positions can influence the blood flow.

2.7.3 Cardiovascular system
Kerry and Taylor (2006) recommend undertaking a systems based approach to identify those at risk of CAD. This system includes assessing for cardiovascular risk factors. A number of studies have considered cardiovascular risk factors relative to CAD, which question this recommendation (e.g. Arnold et al 2008; Thomas et al 2011), although all cardiovascular components cannot be excluded at this time (e.g. Baumgartner et al 2001).

Arnold et al (2008) conducted a case control study to examine vascular risk factors in 239 CAD patients (ICAD 150:63%; sVAD 71:30%; and 18:7% with both) compared with 516 age and sex matched healthy controls. There was no significant difference in other cardiovascular factors previously considered as a risk for vascular pathology: frequency of hypertension, diabetes, current smoking, past smoking and hypercholesterolemia. This supports Thomas et al (2011) who reported that cardiovascular risk factors were not considered significant in a CAD group compared with age (<55-years) and sex matched controls of patients with stroke from other causes, and Bioussé et al (1994) who reported no difference between 65 ICAD patients with (48) or without pain (17). Hypercholesterolemia, however, was reported as a significant risk factor (p<0.05) in sICAD patients with ischaemic events compared to patients without such developments (Baumgartner et al 2001). Similarly, Arnold, Bousser et al (2006) reported a higher rate of hypercholesterolemia in a prospective study of 165 VAD patients compared to other studies. The authors considered that although no control groups were used, this feature could not be dismissed as a significant risk factor in VAD.

In a further report Arnold et al (2010) compared the characteristics of consecutive patients with sVAD with cerebral ischemia (n=165) versus patients with local signs
and symptoms only (head/neck pain, cervical spine radiculopathy; n=21) and concluded that older patients (mean 43.6 ±9.9 v’s 38.6±9 years) and smokers were more likely to develop cerebral ischemia. Arnold et al (2010) hypothesise that this age finding may be related to an increase in inflammation and vulnerability to thrombosis with increasing age, which is a recognised risk factor for ischemic stroke. The patient group from Arnold, Bousser et al (2006) was also included this paper. Arnold et al (2008) concluded that sCAD patients tended to be taller and have a lower body weight than the control group. These features were previously unreported in other studies, however, the measurements were self reported, which exposes the data to personal bias e.g. inserting a lower weight. Nevertheless, it is an interesting aspect as general health knowledge informs us that obesity causes further risk to cardiovascular complications.

Thomas et al’s (2011) control group (n=43 age 43.6 years ± 7.3) and Debette, Metso et al’s (2011) control group (n=556 44.7±10.5 years; 39.9% women) of non-CAD ischemic stroke, had stroke from causes other than dissection. Therefore, from an initial screening perspective, this questions the process of trying to separate potential symptoms caused by a dissection from other causes, if stroke from non-dissection causes can also occur in the younger population (<55 years). Thomas et al’s (2011) control group had considerably more cardiovascular risk factors and other co-morbidities (average 3.23, SD 1.6) compared to the dissection group (1.4, SD 1.3). Clinically, this is too difficult to differentiate; therefore, it is a potential risk focusing on dissection pathology alone. This opinion supports the recommendation to consider cardiovascular risk factors (e.g. Taylor and Kerry 2010).

2.7.4. Migraine
Migraine is defined by the International Classification of Headache Disorders (2004). Debette, Grond-Ginsbach et al’s (2011) large observational study (2004-2009, 20 centres in 9 countries) reported migraine in 221 (36.3%) ICAD and 123 (38.1%) VAD patients with no significant difference in frequency between the two dissection sites (p=0.6 odds ratio (OR) (95% CI) = 1.09 (0.81-1.47). Silbert et al (1995) investigated characteristics of headaches in CAD and reported a history of migraine was present in 24 (8%) ICAD and 6 (23%) VAD cases. Rist et al (2011) conducted a systematic review and meta-analysis to evaluate the association between migraine or migraine subtypes (e.g. with aura) and CAD. Five case control studies
were included that were published through 2010. A pooled analysis revealed that migraine doubled the risk of CAD (pooled OR = 2.06, 95% CI 1.33-3.19). Migraine with aura showed slightly weaker association compared to without aura; however, there was no evidence that aura status modifies association between migraine and CAD (met-regression on aura status p=0.58). Arnold, Cumuric et al (2006) investigated characteristics of pain as the only symptom of CAD. Twenty from 245 consecutive cases diagnosed with sCAD were included, of which 50% had a history of migraine (8 without and 2 with aura).

2.7.5 Oral contraceptives
Thomas et al (2011) observed use of oral contraceptives in 5 (14%) of CAD patients and 4 (9%) of control subjects (<55 years stroke of non-dissection cause). Unfortunately, the statistical analysis is between the groups, rather than considering this as an isolated risk of stroke. Caplan’s (1985) uncontrolled 5-case series reported one patient was also using oral contraceptives. Oral contraceptives were not considered in Debette, Metso et al’s (2011) large multi-centred observational study. Guillon et al (2003) reported 58.3% of n=47 CAD and 40% of n=52 control subject with cerebral ischemic event unrelated to CAD had baseline characteristics of oral contraceptives. Unfortunately, there is no mention if this was current or past use. There appears to be weak evidence to support inclusion of oral contraceptive use as a risk factor for stroke, but not specifically dissection.

2.7.6 Infection
Recent infection is considered as a risk factor that could be a trigger for sCAD (Guillon et al 2003; Debette, Grond-Ginsbach et al 2011). Both studies provide definitions of infection, however, Debette, Grond-Ginsbach et al (2011) consider their results, ICAD n=131 (21.7%), VAD n=47(14.6%) p=0.009 OR (95% CI) 1.59 (1.09-2.31), are possibly an over-estimation of infection within the previous week as their definition is broadly defined i.e. “….one typical feature of infection combined with fever (≥38 degrees) or one typical symptom with corresponding investigative findings indicating infection or two typical symptoms indicating infection” (p.1175). Guillon et al’s (2003) case control study investigating infection occurring within the previous 4-weeks as risk factor of sCAD (n=47) compared to a control group (n=52) with cerebral ischemic event unrelated to SCAD reported that infection was present in 31.9% sCAD and 13.5% control subjects (crude OR 3.0 (95% CI) 1.1-8.2
Clinical staff were not blinded to the patient groups; however, a structured questionnaire was used in the diagnosis of infection.

### 2.7.7 Genetic

Genetic risk factors are thought to play a role in the aetiology of sCAD; however, familial CAD is rare (Grond-Ginsbach et al 2012). Debette and Markus (2009) conducted a thorough systematic review of all published data from 1966-2008 on genetic factors for CAD and performed a meta-analysis of association studies with a polymorphism report. Debette and Markus (2009) concluded that studies on genetic association with CAD have been underpowered and that monogenic connective tissue disease is rarely associated with CAD (Debette and Markus 2009). Ehlers Danlos syndrome is the main connective tissue disease. However, Debette and Markus (2009) report that there are several arguments for association of “sporadic” CAD with connective tissue abnormalities as part of a multifactorial predisposition. A meta-analysis identified an overall significant association of the MTHFR 677TT genotype and CAD (OR 1.67 (95% CI) 1.21 – 22.31). This genotype is associated with elevated homocysteine levels (refer to 2.7.8), which could contribute to arterial wall damage (Debette and Markus 2009). Unfortunately, identifying a MTHFR 677TT genotype is not conducted within a musculoskeletal clinic. This review is performed by the study lead of the Cervical Artery Dissection and Ischemic Stroke Patients (CADISP) study, a large multi-centred prospective study involving 9 countries and 20 centres, which is an indication of the methodological quality.

Martin et al (2006) examined 7 families (n=15, 9 female) with 15 dissections to establish if any specific features existed and identified that familial CAD families are young at first dissection (mean age 36.2 years, median age 32 years, range 18-59). This younger age group of first onset familial CAD is supported by Grond-Ginsbach et al (2012) reporting a mean age of 38.4 years (± 13.3, n=32). Grond-Ginsbach et al (2012) suggest that a specific predisposition for familial history does exist; however, the age of onset and site of dissection differs between families making the familial CAD profile heterogeneous.

### 2.7.8 Hyperhomocysteinemia

Mild Hyperhomocysteinemia has been reported as a risk factor for both arterial dissection and ischemic stroke without dissection (e.g. case-control studies of Pezzini et al 2002; Arauz et al 2007; Benninger et al 2009). However the underlying
mechanism behind this risk factor remains unclear and requires additional investigation (Benninger et al 2009). Additionally, the practicalities of assessing this risk factor are not feasible within musculoskeletal clinics. Arauz et al (2007) however, reported that high plasma concentrations of homocysteine and low plasma levels of folate were associated with an increased risk of CAD and concluded that in a Mexican population that deficiencies in nutritional status may contribute to the relatively high incidence of CAD in Mexico. This feature has potential consideration during assessment e.g. in poor socioeconomic areas.

2.7.9 Styloid process length
Raser et al (2011) conducted a single centre retrospective (2001-2009) case control study of patients with cervical carotid artery dissection (n=38, male=18, age 50.6 years, ±11.5, range 21-77 years) with an equal no. of age and sex matched controls to investigate a potential association between length of styloid process and CAD. The styloid process of the temporal bone is variable in length, angulation and proximity to the carotid artery. This study revealed no significant difference in angulation, however, there was a significant difference for styloid process being longer ipsilateral to the dissection than in control subjects (30.3mm v 26.6mm, p=0.33). Dissection was associated with increasing styloid length with OR 1.08/mm (95% CI 1.002 to 1.17, p=0.04).

Eagle syndrome involves an elongated styloid process that produces a range of cervical related symptoms (Das et al 2008; Piagkou et al 2009 in Raser et al 2011), including potential compromise of the carotid arteries (Piagkou et al 2009 in Raser et al 2011). Figure 2.1 displays elongated styloid processes through ossification of the stylohyoid ligament.
Raser et al (2011) concluded that CAD is associated with a longer styloid process suggesting that mechanical injury from the styloid may contribute to the pathogenesis of CAD. Although the study is retrospective, with potential bias to recall of information, the methodology is detailed and measurements were recorded by blinded observers with high inter-observer correlation coefficients for all measures (0.88 for length and proximity, 0.91 for proximal angulation, and 0.89 for caudal angulation). All subjects had CT angiogram that allows evaluation of both bone and vascular tissue. Unfortunately, in the absence of radiological investigation an elongated styloid process is likely to go undetected if the clinician does not possess a high level of palpation skills and increased awareness to suspect its presence.

2.7.10 Post-partum

Cervicocephalic artery dissection in mothers following childbirth is considered rare. Arnold et al (2008) conducted a case control study to determine differences between post-partum (childbirth within 6-weeks previous) and non-postpartum CAD. A total of 102 women <50 years (6 post-partum, 96 non-postpartum) from 245 female patients held on single centre CAD register (1997–2005) were identified. Arnold et al (2008) concluded that post-partum CAD patients and associated conditions should be considered in women with unusual headache after childbirth. All post-partum CAD patients had neck and or headache as the first symptom onset ranging from 7 days to 18 days after delivery. There was a mixture of risk factors present.
and 3 had ischemic events following pain onset (2 TIA and 1 cerebral infarction). One also had Horner’s syndrome. There was no significant explanation behind the underlying mechanisms. In addition to this study there are a number of single case reports on natural post-partum CAD.

2.7.11 Seasonal variability
Seasonal variability has been reported in CAD. Paciaroni et al (2006) examined seasonal variability in a prospective study of 352 patients with 380 spontaneous CAD (361 symptomatic; 305 carotid and 75 vertebral artery dissections) admitted to 2 Swiss hospitals (1985 – 2004). Most patients presented with ischemic stroke (241 / 63%), followed by TIA in 40 (11%), retinal ischemia in 7 (2%), and non-ischemic in 73 (19%). Nineteen (5%) were asymptomatic spontaneous CAD. A higher frequency of CAD was observed in winter (31.3%; 95% CI; 26.5 to 36.4; p=0.021) compared to spring (25.5% (95% CI)) 21.1 to 30.3), and summer (23.5% (95% CI) 19.3 to 28.3), and autumn (19.7% (95% CI) 15.7 to 24.1). Although a seasonal pattern is reported this does not appear to be constant i.e. spring and summer displays a greater prevalence than autumn. Paciaroni et al (2006) report that the cause of dissection in winter is unclear with possible increased contribution from winter peaks of infection, hypertension, and aortic dissection. There was no additional data to support this hypothesis other than observing season variation. Therefore, it would appear that further evidence is required to accept seasonal variation as a risk factor for CAD.

2.7.12 Risk factors - conclusion
Knowledge of potential risk factors associated with CAD may help early detection of an underlying serious pathology occurring. Similarly, this information may help guide physiotherapeutic management if serious pathology is not imminently suspected. It would appear that some risk factors discussed in section 2.7 have either weak or inconclusive evidence to support routine inclusion as risk factors. In addition, some risk factors, such as length of styloid process, mild hyperhomocysteinemia, and MTHFR 677TT genotype would be very difficult if not impossible to detect within a musculoskeletal clinic setting. However, the presence of cardiovascular risk factors, infection, history of migraine, use of oral contraceptives, awareness of family history of previous cervical arterial pathology or stroke, and headache onset soon after childbirth accompanied by symptoms and
signs of CAD as discussed within this review should raise the index of suspicion for the presence of an underlying serious pathology.

2.8 Cervical myelopathy

2.8.1 Cervical myelopathy - pathogenesis

Cervical myelopathy (CM) is a clinical diagnosis arising from compression of the spinal cord at cervical spine level. Myelopathy can also occur at the thoracic and lumbar spine sections. The most common cause is degenerative changes related to progressive spondylosis that narrows the spinal canal (disc degeneration/protrusion, osteophyte formation, thickening of the ligamentum flavum, and facet joint hypertrophy) (Meyer et al 2008). The subsequent cord compression can result in a range of neurological signs and symptoms that makes differential diagnosis a challenge. The cord compression can cause functional dysfunction and pain that may require urgent surgical intervention.

Cervical myelopathy is reported to peak between the ages of 50 and 60-years; however, the incidence of progression from spondylosis to cord compression myelopathy is unknown (Cook et al 2007; Meyer et al 2008). Chiles et al (1999) retrospectively examined patterns of neurological deficit and recovery following anterior cervical decompression resulting from cervical spondylotic myelopathy (n=76. Male 47:62%) and identified a mean age of 56-years (range 29-87). This was further sub-grouped to myelopathy primarily due to soft disc herniation (mean age 51.3-years, range 29-77) and spondylitic ridges (57.6-years, range 33-87). No additional statistical analysis is provided to identity any significant age related difference between disc and spondylotic changes.

2.8.2 Cervical myelopathy – clinical challenge

The clinical features of myelopathy can overlap with a radiculopathy problem, which is generally compression of a single nerve root as it exits the spinal canal. This is not a medical emergency and is more responsive to conservative management. Furthermore, spinal cord pathologies, such as cervical myelopathy, multiple sclerosis and amyotrophic lateral sclerosis may have similar presenting signs and symptoms that may include upper and lower motor neuron signs, pain, paraesthesia, functional impairment (Cook et al 2007). This presents a significant challenge to practitioners to ensure correct differential diagnosis is obtained with
subsequent selection of safe management strategies. This challenge is exemplified by the findings of Heffez et al (2004) and Rhee et al (2009).

Twenty-one per cent (8) of 39 patients in a prospective controlled study with MRI confirmed cervical myelopathy and subsequent progression to surgery did not demonstrate a single myelopathic sign at their initial presentation (Rhee et al 2009). Rhee et al (2009) was included in Cook et al’s (2011) systematic review described in section 2.8.4. This achieved a score of 6 using the Quality Assessment of Diagnostic Accuracy Studies (QUADAS) tool (score 0-14).

Heffez et al (2004) demonstrated clinical evidence for cervical myelopathy due to Chiari 1 malformation (cerebellar tonsillar herniation; 20%) and clinically significant spinal canal stenosis with mild extension (46%) in n=270 patients (female (86%), mean age 44 years; SD=11 years) who had previously received a diagnosis of fibromyalgia. This initial diagnosis was made by external medical practitioners (Rheumatologist (in 66% of cases), neurologist, or primary care physician). Some patients had previous MRI investigation identifying a degree of stenosis and/or cerebellar tonsillar ectopia, however, this was not considered significant at that time. There is no mention of timeframe or comparison of previous imaging and the most recent MRI confirming cervical myelopathy. Heffez et al (2004) acknowledge that patients were not randomly selected from fibromyalgia sufferers i.e. patients had self referred in this study; therefore, the prevalence of cervical myelopathy Chiari 1 malformation and spinal stenosis within fibromyalgic patients remains unknown. As similar signs and symptoms may exist in these conditions Heffez et al (2004) recommend that a detailed neurological evaluation should be conducted in patients diagnosed with fibromyalgia to exclude cervical myelopathy resulting from cord compression.

2.8.3 Myelopathy – symptoms and signs
2.8.3.1 Multiple level involvements
As previously reported the most common cause of cervical myelopathy is degenerative changes that narrow the spinal canal (Meyer et al 2008). The nature of degenerative changes suggests that pathology may not be restricted to one specific level of the cervical spine; therefore, clinicians should exercise caution when interpreting presenting symptoms and signs.
Vyas et al (2004) prospectively studied the C3-4 level in cervical spondylotic myelopathy (n=14, all male, mean age 50.6 years, range 24-77, identified from 137 CM patients). This study focused on the C3/4 level due to infrequent reporting of involvement at this level and its compromise reportedly being generally in an older patient age group. This mean age (50.6 years) is considered by the authors as younger than previously reported. As an age comparison, Taylor et al (1991) retrospectively observed surgical treatment of cervical spondylotic myelopathy in elderly patients (n=17; Female = 6), where a mean age 71 years (range 65-82) was recorded and additionally observed that coexisting medical conditions (ischemic heart disease (5), significant RA/OA (5, 4 of which with arthroplasties) were common in elderly patients. This is a small sample size and Taylor et al (1991) considered that presenting signs and symptoms (not co-morbidities) were similar to those in younger patients, but no comparison or control group is used to support this. Vyas et al (2004) is also a small sample size due to the focus on a specific level and was restricted to an Indian population, therefore this aspect may not transfer to a broader population. Although anterio-posterior cord compression ratio showed significant compression compared to lower cervical levels and all patients demonstrated pyramidal signs, this study is unable to state if these features are related to the C3/4 level. The presenting symptoms and signs lack specific detail.

Eleven from 14 patients also had lower cervical level involvement (Vyas et al 2004) and Kim et al’s (2007) retrospective study (n=26; male =20, mean age 44.8 years range 23-61) reports the most frequent level as C5/6 with 4 patients having more than 2 levels of involvement. Vyas et al (2004) consider the effect of lower cervical spine level involvement predisposes upper cervical levels to increased mobility and spondylotic change. Therefore, these studies suggest that physiotherapists should exercise caution in interpreting a patient’s presentation as multiple level involvement may be occurring with potential to contribute to an array of signs and symptoms.

2.8.3.2 Sphincter (bladder/bowel) and sexual dysfunction
Bladder and/or bowel dysfunction and erectile/sexual dysfunction are signs that may indicate potential spinal cord compromise e.g. compression through cervical myelopathy (e.g. Bednarik et al 1999; Vyas et al 2004) or malignancy (Greenhalgh and Selfe 2003; 2009). Personal experience and clinical observations suggests
that specific detail of these features may be poorly understood by physiotherapists, unless advanced level or specialist training has been undertaken. Similar to the 5Ds discussed earlier, in presentations that may include bladder dysfunction the clinician’s level of questioning may be limited to enquire solely on bladder dysfunction or disturbance without any clarification beyond this. Unfortunately, there is no empirical data to support this observation. However, there are examples within the evidence base that may contribute to this observation.

Studies, in the main, reporting on cervical myelopathy tend to use generic terms such as, bladder or sphincter dysfunction when reporting such changes (e.g. Bednarik et al 1999; Vyas et al 2004). These terms lack specific detail to suitably inform clinicians as part of their decision making processes. This lack of specific detail may result in delayed diagnosis (Cook et al 2007; Meyer et al 2008). Therefore, if physiotherapists become consistently more aware of the range of features that may appear within sphincter dysfunction this may help earlier decision making.

One study, however, does provide such specific detail. Sakakibara et al (1995) studied the location of the paths subserving micturition in patients with cervical myelopathy through a prospective design (n=95 identified from 128 cervical myelopathy patients). Micturitional symptoms were classified as either: irritative (diurnal or nocturnal urinary frequency; sensation of urgency or incontinence) n=61, or; obstructive (urinary hesitation, prolongation, difficulty of voiding and urinary retention) n=71. Urinary incontinence was found in 25 patients and urinary retention in 22 patients.

Urodynamic studies (residual volume, water cystometry and simultaneous sphincter EMG) were undertaken alongside neurological examination (disturbed deep sensation of lower extremities (position and vibration; n=55), disturbed superficial sensation of lower body including perineal area (pin prick; n=63) and pyramidal signs (weakness and hyperreflexia of lower extremities, and Babinski sign; n=96). Uninhibited contraction was more common with all 3 pyramidal signs (p<0.05). Bladder capacity was smaller in patients with pyramidal signs, and with Babinski sign (p<0.05).
All participants referred for assessment of micturitional state were included in this study. From a clinical perspective, this study may be useful to highlight patterns of micturition disturbance. As most studies tend to use the generic terms such as, bladder or sphincter dysfunction, this study could inform clinical practice by highlighting the micturitional sub-groups of irritative and obstructive features. No specific studies on bowel dysfunction were identified during the search.

He et al (2006) examined improvement of sexual function in male patients (average age 56.3-years; range 43-72) treated surgically for cervical spondylotic myelopathy through prospective follow-up. Symptoms of sexual dysfunction were reported with difficulty in penile erection or ejaculation. Twenty-two subjects (plus 2 unable to attend follow-up) were identified from 753 patients diagnosed with cervical myelopathy. All participants reported normal function 6-months pre-surgery. The dysfunctions were classified as either reflexogenic erection (n=4) or psychogenic erection (n=18). Post surgery: 20 from 22 (91%) improvement was measured in International Index of Erectile Function. This study highlights the low frequency of sexual dysfunction, which is more likely to be psychogenic compared to reflexogenic. This supports Chiles et al (1999) in commenting that sphincter and sexual dysfunction were relatively infrequent and usually in far-advanced myelopathy after they identified the incidence of bowel dysfunction (4: 5.3%), bladder dysfunction (8: 10.5%), and sexual dysfunction (5: 6.6% men only).

Therefore, this low rate may indicate that routine questioning in relation to sphincter or sexual dysfunction may not need to occur; however, questioning for these dysfunctions may be utilised in advanced level questioning should more information be required. This could be combined with awareness of duration of this dysfunction. All patients with sexual dysfunction reported normal function 6-months pre-surgery (He et al 2006).

2.8.3.3 Gait
Similar to generic terms used in clinical practice for bladder or sexual dysfunction, personal clinical observation suggests that gait disturbances are also a broad term used for checking for such dysfunctions with specific detail lacking on the components of gait disturbance. Although there is no empirical data to support this observation this is frequently used within the evidence base (e.g. Bednarik et al...
1999; Chiles et al 1999; Kim et al 2007). Kim et al (2007) report patients (20:77%) with myelopathy caused by soft cervical disc herniation had difficulty with walking, Bednarik et al (1999) reported 56 from 60-patients in a prospective case control study, and Chiles et al (1999) reported gait disturbance in 61 (80.3%) patients with cervical myelopathy. These studies do not provide specific detail, such as kinematic and linear parameters, for these dysfunctions.

A number of studies, however, have investigated spatiotemporal or linear parameters and kinematic parameters for specific gait disturbances within this pathology (Kuhtz-Buschbeck et al 1999; Kim et al 2010; Lee et al 2011). Three dimensional gait analysis, which provides a quantitative measurement was included in all three studies.

Spatiotemporal or linear parameters comprised of; step width, gait velocity, cadence (step rate per minute), step length, stride length (distance between both feet), step length (distance between contact point same foot), stance time (single foot), and double support time (both feet). Kinematic data comprised of pelvic or hip, knee and ankle joint range of movement (ROM) (Kuhtz-Buschbeck et al 1999; Kim et al 2010; Lee et al 2011). Consistent findings (disturbances) were reported between these studies:

Disturbances of linear parameters include:

- Slow gait.
- Decreased step/stride length
- Increased step width and double support.
- Decreased single limb support.

Disturbances in kinematic parameters include:

- Decreased maximal knee flexion (swing phase).
- Increased ankle dorsi-flexion (swing phase).
- Decreased plantar-flexion at push-off.

These kinematics features are indicative of spasticity (Kim et al 2010). Furthermore, Lee et al's (2011) prospective case control study (n=38 (control=36); male (21), age 56.2-years ± 15.2, mean duration of symptoms 4.3 months ± 4.2 months) identified an increase in leg muscle tone (1 to 1+ Ashworth scale) in all CM patients. This
supports Kim et al (2010) who also identified mild-moderate spasticity within their study and state that decreased knee joint flexion during swing phases and ankle joint motion during stance phase are indicative of a spastic gait pattern. Gait deviations in cervical myelopathy patients with mild spasticity are considered to mainly result from instability caused by impairment of afferent proprioceptive signal delivered by the dorsal column. Decreasing gait velocity and step length and increasing step width and double support time were identified as compensatory strategies to stabilise dynamic balance (Lee et al 2011).

Kuhtz-Buschbeck et al (1999), Kim et al (2010) and Lee et al (2011) do not provide specific detail of the selection process; however, detailed information on gait analysis procedures is provided and although MRI films were reviewed independently by one radiologist in Lee et al (2011), this practitioner was blinded to clinical information before grading levels of compression. Kim et al’s (2010) study used two radiologists to sub-group subjects into 3 groups (0, 1 and 2): Group 0, No Increased Signal Intensity (ISI) (n=13, age 54.5 ± 9.6-years); Group 1, Faint ISI (n=14, age 58.6 ± 8.3-years); or, Group 2, Intense ISI (n=9, age 62.9-years ± 7.3) to evaluate the relationship between increased signal intensity (ISI) on magnetic resonance imaging (MRI) and gait function in cervical spondylotic myelopathy (n=36, Male=26). This is a retrospective analysis therefore risks recall bias and the authors acknowledge limitations in statistical analysis e.g. limited statistical power from a small sample size; however, sub-grouping into levels of ISI and correlated with gait analysis using Spearman rank correlation coefficient is detailed.

Patients with ISI on MRI compared to those without ISI had significantly slower gait speed, longer step time, decreased single limb support time, increased double-limb support time, and reduced knee flexion in swing phase and increased ankle DF ROM, which the authors report as being indicative of spasticity.

Kuhtz-Buschbeck et al’s (1999) cervical myelopathy group (n=12, age 49 ± 5-years), within a case control study demonstrated significant 2-month post-surgical improvement in spatiotemporal parameters. Velocity was increased (mean 10%) in all patients, except one (p<0.01). This is a small sample, however an age and anthromorphically matched healthy control group (n=14) is utilised. Pre-post
surgical gait analysis helps support the findings that the gait disturbances are related to myelopathic pathology.

The three gait analysis studies described above are useful studies to help inform clinical practice to identify specific aspects of gait, rather than simply considering generic difficulties with gait. More defined detail would enable clinicians to guide subjective questioning for any changes reported by patients and to objectively observe with greater knowledge.

2.8.3.4 Upper motor neuron (spasticity) signs
In addition to gait analysis, Kim et al (2010) also checked for presence of: neck pain, increased tendon reflex, ankle clonus, Babinski sign (upgoing plantar response), parasthesia, sensory changes, bowel/bladder symptoms. Regression tree analysis observed upper motor neuron signs, such as ankle clonus and Babinski sign were important in classification of ISI groups. These signs, in addition to other pathologic reflexes, such as Hoffman sign, and inverted radial reflex are pyramidal or long tract signs consistent with cord compression (Edwards et al 2003). Upper motor neuron findings (spasticity) may occur in both upper and lower limbs. Cranial nerve dysfunctions or hyperactive jaw reflexes may indicate brainstem or intracranial lesions (Edwards et al 2003).

Other examples of varying quality of studies reporting these signs of spasticity are displayed in Table 2.2:
Table 2.2: Additional examples of studies reporting signs of spasticity in cervical myelopathy

<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>Study information / clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taylor et al (1991)</td>
<td>17</td>
<td>Reflex changes in both upper and lower (generally hyperreflexia in lower limbs, with brisk or diminished upper limb depending on level of lesion), lower limb weakness associated with spasticity (13)</td>
</tr>
<tr>
<td>Bednarik et al (1999)</td>
<td>60</td>
<td>Gait disturbances (56), spasticity and/or weakness of the lower and/or upper extremities (55).</td>
</tr>
<tr>
<td>Chiles et al (1999)</td>
<td>76</td>
<td>Spastic gait (52: 68.4%), ankle clonus (25: 32.9%), Babinski reflex (31: 40.8%), hyperreflexia (58: 76.3%).</td>
</tr>
<tr>
<td>Heffez et al (2004)</td>
<td>270</td>
<td>Hyperreflexia (64%), inverted radial reflex (57%), Hoffman sign (26%), Clonus (25%), weakness in ≥1 limb (22%).</td>
</tr>
<tr>
<td>Kim et al (2007)</td>
<td>26</td>
<td>Reported walking difficulty (20: 77%), spasticity (15:58%) and central cord syndrome (4:15%) without providing any additional specific information.</td>
</tr>
</tbody>
</table>

Kim et al (2007) is a retrospective study, therefore potential recall bias may occur. The primary aim of this study was to establish clinical characteristics, radiological findings, and improvement post surgery for CM caused by soft cervical disc herniation. Twenty-six myelopathy patients due to soft cervical disc herniation were selected from n=456 undergoing surgery during a 7-year period, with n=111 identified as being soft disc herniation during this period. However, no specific detail is provided on how the signs/symptoms of the study group differed from the n=111, also with soft disc herniation.

Heffez et al (2004) provide details on their standard pre-assessment conducted by a neurologist and/or neurosurgeon (n=138 assessed by both). However, a statistically significant difference (p=0.02) was noted between the two specialists with the neurologist recording a higher prevalence of findings in hyperreflexia, Hoffman sign, Romberg sign, and impaired tandem walking. Heffez et al’s (2004) analysis of this difference was a higher threshold required by the neurosurgeon as they were required to consider surgery. Bednarik et al’s (1999) prospective case control (n=60; Male (43), Female (17); mean age 52.6 ± 8.1-years range 32-76) provides details on the selection process and control group (probable vascular cerebral involvement), however, they simply state this group did not show signs of CM.
2.8.3.5 Motor deficit

Chiles et al (1999) reported motor deficit in 152 muscle groups from n=76 patients. Motor deficit in the upper limbs typically occurred in the hands and triceps first; Hand intrinsics (86: 56.6%), triceps (44: 28.9%), biceps (18: 11.8%), and deltoid (16: 10.5%). Motor deficit in the lower limb was recorded more frequently in the hip flexor and knee extensor muscle groups; iliopsoas (59: 38.8%), quadriceps (40: 26.3%), dorsiflexion (28: 18.4%), plantarflexion (24: 15.8%). Heffez et al (2004; n=270) reported weakness in ≥1 limb (22%). Weakness in the hands may also be described as clumsy or useless (Bednarik et al 1999; Taylor et al 1991). Bednarik et al (1999; n=60) reported weakness of the hands/upper limbs (14) and lower limb weakness associated with spasticity (13).

The work of Taylor et al (1991) is a retrospective analysis, and therefore vulnerable to potential recall bias. The small sample is specific to elderly population. Taylor et al (1991) state the presenting features are similar to younger patients, but no direct comparison group is provided. Objective functional improvement was recorded in 58% upper limb and 71 % lower limb post-surgical intervention. Taylor et al (1999) devised their own functional grading system for upper and lower limb and allocated a score to each patient after obtaining information from patient notes. No information on validity/reliability of this system is provided.

It is hypothesised that motor deficit occurs from anterior horn cell loss, rather than nerve root compression, resulting from spondylotic obstruction of spinal cord venous drainage (Chiles et al 1999). There is no mention of a standardised subjective screening process or the method of individual muscle testing. The same surgeon performed surgery, but it is not clear if this surgeon also conducted assessment of all patients. However, validated functional measures are used pre/post surgery. There is no comparison for the presenting signs/symptoms e.g. against a control group of cervical radiculopathy, but significant post-surgery improvement helps support pre-operative test findings.

2.8.3.6 Sensory deficit

Sensory deficit is a common finding in cervical myelopathy (e.g. Chiles et al 1999; Bednarik et al 1999). Chiles et al (1999; n=76) reported upper limb sensory
complaints (sensory loss, dysesthesias, paraesthesias) occurred in 63 (82.9%) patients, and lower limb sensory complaints 34 (44.7%).

Chiles et al (1999) observed that upper limb symptoms usually occurred slightly before the onset of gait difficulties, and in all (except 6) patients the upper limb sensory complaints or difficulties typically started distally in the finger tips, thus affecting fine movements, and then progressed proximally with time. Unfortunately, no timelines are provided. These symptoms rarely had a radicular distribution. The primary aim of Chiles et al's (1999) study was to establish patterns of neurological deficit and recovery post surgery for CM. Although this study used a retrospective analysis of presenting signs and symptoms, with potential recall bias, the information regarding upper limb dysfunction occurring pre-gait dysfunction as early signs, and most experiencing difficulty with fine hand movements before progressing proximally could be particularly helpful from a clinical perspective when differentiating from other possible diagnoses.

Taylor et al's (1991) retrospective study involving elderly patients (n=17) reported paraesthesiae in hands and upper limbs, often with asymmetrical distribution (16), impairment of pin prick sensation in hands and upper limb (12), lower limb (10), severe proprioceptive loss in legs (1). Bednarik et al's (1999) prospective case control (n= 60. Male (43), female (17); mean age 52.6 ± 8.1 range 32-76) reported sensory disturbance corresponding to cervical spinal cord involvement (n=37). The primary aim of study was to investigate presence of median nerve mononeuropathy in CM.

2.8.3.7 Pain

Neck, and upper and lower limb pain has been reported to occur in cervical myelopathy (e.g. Bednarik et al 1999; Chiles et al 1999; Kim et al 2007; 2010). Edwards et al's (2003) clinical review type publication advises that myelopathy may occur simultaneously to radiculopathy as a result of progressive cervical spondylosis causing foraminal stenosis. However, specific aspects of pain in studies on cervical myelopathy were generally poorly reported. Chiles et al (1999 n=76) reported the occurrence of; neck pain (21:27.6%), upper extremity pain (18: 23.7%), and lower extremity pain (7:9.2%). Taylor et al (1991) also reported neck pain (5 from 17). Radicular pain was reported by Kim et al (2007; 9:35%) and Kuhtz-Buschbeck et
al's (1999) in upper limbs (9) without specific detail provided. Bednarik et al (1999) reported Lhermitte sign (n=9), an electric shock type phenomenon occurring down the spine or limbs on neck flexion.

2.8.3.8 Cervical myelopathy - Mean duration of symptoms and signs
The studies reviewed in this section had reported a large variation of duration of symptoms that suggest a largely chronic and progressive presentation; however, caution should be reserved for acute or rapid deterioration of clinical features. Examples of these variations in mean duration or time-range of features are: Vyas et al (2004) ranged from 1 to 36-months pre-spinal surgery; Lee et al (2011) 4.3 months ± 4.2 months; Kuhtz-Buschbeck et al (1999) 10-months ± 3-months; Heffez et al (2004) 8-years (SD=6.3-years); Taylor et al (1991) symptoms duration 1 to 18 months (mean 9).

2.8.3.9 Summary of cervical myelopathy presenting features
The following is a summary of the presenting features for cervical myelopathy:

UMN - Pyramidal signs (corticospinal tracts - motor)
- Spasticity e.g. limbs / gait / reflexes (hyper)
- Weakness
  - Upper Limb (UL): intrinsics>triceps are 1st onset
    - Grip, fine motor skills
  - Lower Limb (LL): Hip flexor>knee Extensors (quads) are 1st
- Hyperreflexia
  - LL / UL

LMN - Sensory deficit
- Hyporeflexia
  - UL at level of compression
- UL/LL numbness/paraesthesia/dysaesthesia
  - UL usually begin in digits and move up
- Proprioception, balance, fine motor e.g. fastening buttons
- Pain: May have concomitant radiculopathy therefore mixed picture. Pain not always present.
2.8.4 Cervical myelopathy - Clinical tests for screening and diagnosis

A range of clinical tests are used to screen for neurological conditions, such as cervical myelopathy (cord compression). However, some neurological clinical tests (e.g. finger escape sign and clonus) have not been investigated for diagnostic accuracy, whilst others (e.g. the Hoffman sign, Lhermitte sign, and plantar response) have been investigated, but with inconsistent levels of methodological quality that affects their diagnostic accuracy values (Cook et al 2007 p.1237).

Cook et al’s (2011) systematic review of 12 studies followed Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to examine clinical tests for screening and diagnosis of cervical spine myelopathy.

All studies were assessed using Quality Assessment of Diagnostic Accuracy Studies (QUADAS) tool. No RCTs were included within this review and the authors acknowledge that not many studies met their inclusion criteria, which are outlined. This appears appropriate; therefore, the process relied heavily on hand searches. All studies were double assessed and no authors assessed any of their own previous work.

This review is a comprehensive collection of the clinical tests commonly used for screening for CM and highlights the generally low-moderate quality of studies assessing the clinical utility of such testing. These tests demonstrated high levels of specificity and low levels of sensitivity (see additional comments below).

The authors have sub-divided the tests into 3 categories of tests:

1. Those associated with gait or balance analysis;

2. Those associated with upper motor neuron or “pathological” signs; and,

3. Those associated with deep tendon reflexes.

Cook et al’s (2011) tables 1-4 and descriptions/positive findings summary is presented (with permission) in Appendix A. The authors recommend further evaluation of these tests and exploration of clustered findings. Tests with low sensitivity are unlikely to identify the condition early in the examination (cannot rule it
out) and may result in a false negative. Thus clinicians may apply treatment techniques under false assumptions of safety, which may further compromise the spinal cord.

One further test identified that is not included in Cook et al’s (2011) review is the foot tapping test (FTT; Refer to Appendix A for description) as a simple quantitative objective assessment of cervical myelopathy (Numasawa et al 2012;). The FTT strongly correlated with Japanese Orthopaedic Association Lower Extremity, a reliable functional scale \( r=0.696, p<0.0001 \) and correlated with a reference test, grip /release test \( r=0.571, p<0.0001 \).

Numasawa et al (2012) consider this test as an easily applied quantitative test for patients with cervical myelopathy, especially those with limited walking ability. However, on review, easy replication may be counteracted with difficulty to achieve a meaningful clinical interpretation unless a comparative quantitative base measure is used e.g. <30 repetitions derived as a base comparative measure that may be developed following additional testing, including healthy volunteers. This is because both lower limbs may be affected making it difficult to compare limb to limb. In addition, Numasawa et al (2012) identify that the test is not specific to cervical myelopathy, but rather is a test for UMN diseases and may not be readily differentiated from LMN disorders such as, peroneal palsy or lumbar spinal stenosis. This test, however, appears suitable for non-ambulatory patients, where gait disturbances are difficult to assess (Numasawa et al 2012).

2.9 Literature review conclusion

This literature review identifies that there is a risk of neurological/neurovascular pathology, such as cervical arterial dissection and cervical myelopathy, presenting as neck and/or head pain or functional impairment at musculoskeletal clinics. Currently, the extent of adverse events related to such presentations is unknown. Although the problem is not considered common, there is limited evidence to support this assumption. Therefore, clinical decision making processes should be sufficiently robust to establish a correct early diagnosis or to identify those patients at risk of developing an adverse event following manual treatment. Failure to identify such cases may have catastrophic consequences for the individual and carer through complications, such as permanent gait or bladder dysfunctions, stroke or potentially, death.
The literature review identifies limitations and inconsistent evidence for current functional provocative testing and clinical tests, risk factors, and, cardinal signs and symptoms traditionally used as a screening process by musculoskeletal physiotherapists to differentially diagnose serious neurological and neurovascular pathology from musculoskeletal disorders. Additionally, the review highlights the difficulties in differentiating neck and head pain from non-musculoskeletal causes. This presents a serious challenge for physiotherapists and requires a high level of awareness to suspect such pathology (Thanvi et al 2005; Greenhalgh and Selfe 2006; Kerry and Taylor 2006, 2008). Therefore, an improved process aimed at detecting serious neurological and neurovascular pathology of the cervical spine would be of clinical value.

It is of paramount importance that physiotherapists attain the required evidence based knowledge that may be applied to their musculoskeletal clinical practice. This includes correct interpretation of the medical literature and engagement with medical professionals to achieve high quality information and clinical credibility in any screening process. In order to develop an improved red flag screening process for serious pathology related to the cervical spine the following research aim and questions have been developed to achieve this process. Chapter 3 describes the methodological processes developed to address the research aim and questions.

2.10 Research aim
The overarching aim of this study is to develop a list of red flag clinical indicators of possible serious cervical spine related pathology presenting as a musculoskeletal disorder.

2.11 Research questions (RQ)
RQ1. “What pathologies including their signs and symptoms and risk factors should be considered as red flags when screening for serious pathology in neck related musculoskeletal disorders in the adult population?”

a) “What risk factors and signs and symptoms may indicate the presence of Cervical Arterial Dysfunction (CAD)?”
b) “What features of headache and/or neck pain are likely to indicate the presence of serious pathology e.g. CAD?”

c) “What features of dizziness and what clinical tests would aid MSK physiotherapists' differential diagnosis of a peripheral versus central cause of dizziness?”

d) “What clinical tests, signs and symptoms of cervical myelopathy (CM) have been identified in this presentation that should be used to screen for this pathology?”
Chapter 3. Methodology

3.1 Development of the research methodology

One method of contributing to the knowledge base is to seek a consensus of the medical profession on key issues regarding screening and differentiation of conditions, such as cervical arterial dysfunction and cervical myelopathy. The Delphi technique and Nominal Group Techniques are consensus methods. The latter uses a highly structured meeting to gain information from experts. This method was not considered practical for this study due to the anticipated difficulties and complexities involved in recruiting and convening a group of specialist medical consultants. Therefore, this option was rejected. The Delphi technique, however, was considered as a suitable method, which is designed to gain a consensus where there is incomplete knowledge or uncertainty in clinical issues (Jones and Hunter 1995; Powell 2003; Skulmoski et al 2007; Hung et al 2008). An example is Smart et al's (2010) Delphi approach using expert clinicians to achieve a consensus in establishing clinical indicators of pain mechanisms in musculoskeletal disorders.

The Delphi technique is an iterative survey process used to collate expert opinion over a series of sequential rounds, which are interspersed by feedback from the previous round (Powell 2003; Skulmoski et al 2007; Hung et al 2008). However, immediate progression to a Delphi survey approach would be making an assumption that there was indeed a problem with red flag screening for serious pathology in neck pain or neck related disorders. As the literature review was conducted by a single researcher, additional input was considered appropriate to gain opinions from physiotherapists on three key components before progressing to a Delphi survey (The Delphi technique is discussed in section 3.3). Therefore, a subset of aims was constructed specifically for the focus group. This subset is listed in 3.1.1:

3.1.1 Subset of aims specific to focus group

1) Gain additional opinions on the researcher’s suggestion that a knowledge gap exists for red flag screening of serious pathology in neck pain or neck related disorders.

2) Seek opinion on relevant key sub-topics within this red flag screening process.
3) Seek opinion on a format for which to present these key sub-topics that will aid clinical decision making.

4) Seek opinion to establish if gaining input from medical experts would be considered beneficial to addressing areas of incomplete knowledge or uncertainty in clinical areas. This could help support the literature review findings.

3.1.2 Mixed methodology research design
A mixed qualitative and quantitative methodological design was considered appropriate to address the research questions within this study, as listed in section 2.11.

The study’s aim to inform clinical practice by developing a clinical screening list of red flag clinical indicators of possible serious cervical spine related pathology presenting as a musculoskeletal disorder would be created through a 3-phase process that followed the literature review:

Phase 1 – Expert physiotherapists focus group that informs phase 2 and 3

Phase 2 - Develop draft clinical chart based on information gained through focus group discussion and findings established from literature review. The aim of the clinical chart is to present pathology related clinical symptoms and signs within one accessible resource that supports clinical decision making for physiotherapists. This clinical information will provide context and detail to support the final list of red flag features established through phase 3

Phase 3 – 3-round Delphi survey of medical experts (including pilot for 1st round).

The research design, including phases 1-3, is displayed in Figure 3.1:
This process aimed to achieve a clinical consensus on main clinical features, and if applicable, subjective questioning with subsequent actions or tests to enhance early
detection of red flag neurological and neurovascular pathologies presenting as, or alongside, a musculoskeletal disorder. Strengths in the qualitative aspect of this research design are; it allows flexibility that enables the researcher to adjust their focus on data collection processes, and it allows the researcher to be close to the data and research material, which can be tested in subsequent quantitative studies if required (Bowling 2010 p.380).

Within a qualitative approach researchers using a Qualitative Description (QD) method are considered to be closer to their data compared to researchers conducting grounded theory, phenomenologic, ethnographic or narrative studies (Sandelowski 2000; Neergaard et al 2009). Sandelowski (2000) considers that QD involves reporting the facts of the event or situation in the everyday language of these occurrences, whereas other qualitative approaches (e.g. grounded theory, phenomenologic, ethnographic or narrative studies) require the researcher to have a reflective or interpretive interplay on their observations, thus reporting or representing such events in different terms (Sandelowski 2000; Neergaard et al 2009). Qualitative description is not free from interpretation. However, less inference is generated and the results are likely to achieve a consensus from researchers (Sandelowski 2000). A low level of inference could be construed as a limitation of QD as it reduces the depth of discussion in general terms (Neergaard et al 2009). Additionally, the QD approach remains flexible and may be utilised with aspects of other qualitative approaches (e.g. grounded theory, phenomenologic, ethnographic or narrative) integrated into the study design, dependent on its objectives (Sandelowski 2000; Neergaard et al 2009).

QD is the least theoretical of the qualitative approaches; however, it is founded on existing knowledge or evidence base with thoughtful conduits to such work and experience of the research group (Neergaard et al 2009). This is a valuable component of this research design, which aims to inform clinical practice through several stages of interactions with two professional groups; Physiotherapists and medical consultants, where opinions and recommendations on clinical practice and clinical indicators would be received. However, flexibility should not be sought at the expense of sacrificing methodological organisation and rigour (Bowling 2010 p.379).
The results of a QD study offer great potential in the provision of information to develop service delivery, which is a reason for increasing use of this methodological approach by clinical expert nurses (Sullivan-Bolyai et al 2005). However, Sullivan-Bolyai et al’s (2005) study used a QD approach to develop and refine interventions in persons with health disparities, whereas this study aims to use this approach with service providers to develop construction of a questionnaire. The QD design provides a method whereby participants’ constructive suggestions may be identified and utilised to inform development of clinical practice (Sullivan-Bolyai et al 2005).

QD is the method of choice if data is not generated or interpreted from the existing evidence base. However, the subsequent descriptive summaries for example, the findings from focus groups, can be used to generate future theory-based research (Sandelowski 2000; Neergaard et al 2009). Reporting in QD describes the experiences or opinions in a language similar to the informants or participants. This study’s participants are senior clinicians, therefore lending itself to the inclusion of technical descriptions within the summaries, if considered appropriate. This creates added potential for development of the third phase (Delphi method) of this project. QD methodology is an appropriate form of qualitative enquiry in a mixed method design prior to questionnaire development (Sandelowski 2000; Sullivan-Bolyai et al 2005; Neergaard et al 2009; Peerman et al 2013).

3.2. Focus groups

Focus groups are a method used to collect qualitative data from a focused discussion that is both inductive and naturalistic. This method can be used for constructing questionnaires (Krueger and Casey 2000 p. 18-19) or as a Delphi study first round to generate qualitative comments (Keeney 2000 in Keeney et al 2006; Hasson and Keeney 2011). Focus groups are useful if a range of views or opinions are needed on a specific issue. For example, they have been used to good effect before designing a questionnaire for patients’ priorities regarding outpatient physiotherapy care (Peerman et al 2013). Pagé et al’s (2012) qualitative approach to investigate psychosocial services associated with congenital heart disease combined a focus group and survey method with the same participants. Geller and Holtzman (1995) used focus group methodology in a medical context with physicians to gain their perceptions on genetic testing then subsequently summarised these perceptions.
3.2.1 Focus group construction

A focus group method involves a group interview, typically consisting of 6-10 participants and lasting between 1-2 hours duration (Petty et al 2012). Stewart et al (2000 p.37) also report that focus groups may consist of 8-12 participants. Whereas, Krueger and Casey (2000) consider the ideal group size to be between 6-8 participants in non-commercial topics, and observe that smaller numbers of 4-6 are becoming more popular due to easier to recruit and to host. However, fewer than 6 participants may be considered not ideal for optimal discussion (Stewart et al 2007 p.58). Participants may be a homogeneous group with similar backgrounds or a heterogeneous group with different backgrounds or experiences (Petty et al 2012). The focus group is usually audio or video-recorded for subsequent analysis.

Focus group methodology typically consists of 3-4 groups with any one type of participant (Krueger and Casey 2000 p. 26) or 4-12 groups (Litosselliti 2003) to achieve a point of “saturation”. Saturation is a term used to describe the point where no new data or ideas have been generated (Krueger and Casey 2000 p.26). However, 1-2 focus groups may be appropriate dependent on the purpose of the study and where the focus group sits within the research agenda to which they are applied. The key aspect of successful integration of a focus group is ensuring that it is consistent with the objectives and purpose of the study (Stewart et al 2000 p.39).

The inconsistencies in reporting the number of focus group discussions conducted within individual studies was addressed in a methodological study of sample size reporting in focus group studies (Carlsen and Glenton 2011). Carlsen and Glenton (2011) identified 220 papers involving focus group methodology in various forms and considered there was poor reporting of sample sizes. The number of focus groups conducted within these papers varied considerably (mean 8.4, median 5, range 1 to 96). Eleven studies used one focus group and typically involved questionnaire design. Carlsen and Glenton (2011) conclude that further evidence based guidance is required to assist in deciding on sample size. The authors also considered that "saturation" point was inconsistent or unconvincing reports of the iterative process in reaching this point, although there were several examples of adequate reporting. In relation to this red flag project, it is important to consider Stewart et al's (2000 p.39) considerations that successful integration of a focus group is ensuring that it is consistent with the objectives and purpose of the study.
Stewart et al (2000 p.40) cites Krippendorf (2004) to describe two types of data: emic and etic. Emic data arises in a natural or indigenous form, whereas etic data represents a researcher's imposed view. Focus group interviews are likely to be closer to the emic end of the continuum, whereas a survey design is closer to the etic end of the spectrum. This concept helps consider how focus groups may be used for both exploration and confirmation purposes if questioned against the choice of other research tools, such as a questionnaire design. An exploratory purpose is typically used at the early phase of a research project (Stewart et al 2000 p.40). A focus group approach in this study is utilised in an exploratory context to gain opinion on particular clinical issues within physiotherapy practice.

3.2.2 Focus group bias

Researchers should be cautious of introducing bias into the focus group approach (Krueger and Casey 2000; Stewart et al 2000). Selection bias may develop in ways that are not obvious which has potential to reduce the quality of the study. To counteract this, Krueger and Casey’s (2000 p.80-81) recommendation is that all of the target population has an equal chance of selection and that randomisation is only effective if prospective participants meet the selection criteria.

Stewart et al (2000) also advise caution on introducing moderator bias; however, there is no single most effective style or type of moderator to lead a focus group. This role should be adapted in order that the strategy and moderator suit the function of the group. Kennedy (1976 in Stewart et al 2007 p.85) identifies three different sources of moderator bias that may potentially affect moderator objectivity:

- **Personal bias**; reinforcing points of view commensurate with the moderator’s own perspectives. This was considered as a risk within this study.
- **Unconscious needs to “please” the client**; reinforcing points of view commensurate with the moderator’s own perspectives. This was not considered as a risk of bias within this study.
- **The need for consistency**; the predisposition to reinforcing points of view that are internally consistent. This was considered as a risk within this study.
3.2.3 Focus group analysis – How much?

The extent of focus group analysis varies with the purpose of the research, complexity of research design and the extent of which how efficiently the researcher may reach conclusions’ based on simple analyses (Stewart et al 2007 p.109). This range of analyses may vary from requiring no formal documentation due to quickly ascertained obvious conclusions to more detailed reports, such as to inform product design or service delivery. Therefore, the depth of analysis and level of rigour will be dependent on the purpose of the research project combined with the cost-benefit of carrying out analysis at a given level (Stewart et al 2007 p.110). Refer to section 3.6.2.5 for a more detailed discussion on focus group analysis.

3.3 Delphi technique

As previously stated, the Delphi technique is an iterative survey process used to collate expert opinion over a series of sequential rounds, which are interspersed by feedback derived from previous responses (Powell 2003; Skulmoski et al 2007; Hung et al 2008).

The Delphi method is considered as an effective method for this systematic collection and aggregation of informed judgement e.g. achieving a consensus or to forecast future events, by a group of experts on specific questions and issues (Hung et al 2008; Reid 1988 in Hasson and Keeney 2011 p. 7). An example of such issues or situations is where frequent clinical or practical judgements may be encountered in the presence of incomplete empirical evidence to support evidence-based decision making (Cook et al 2005).

There are various forms of Delphi technique, such as ‘modified Delphi’ where panellists are presented with pre-selected items; the ‘policy Delphi’ designed to inform policy decision-makers; ‘Technological Delphi’ with the aims varying dependent on research design, such as predicting future events; and the ‘classical Delphi’ designed to elicit opinion and gain consensus (Keeney et al 2006; Hasson and Keeney 2011). This study is interested in the ‘classical Delphi’, however, Hasson and Keeney (2011) consider that whichever approach is taken the generic aim is to determine, predict and explore group attitudes, needs and priorities.
3.3.1 Delphi technique critique

The variations in design are likely to have different characteristics with different aims that are reflective of the situation, target participants and administrative requirements (Hasson and Keeney 2011). For example the number of rounds, level of anonymity and feedback provided, inclusion criteria, methods of sampling and analysis. These differing methods will also have accompanying variances in quality of outcomes. This combination has contributed to considerable criticism of this technique with regards to its ability to achieve acceptable levels of reliability and validity (Powell 2003; Hasson and Keeney 2011). Hasson and Keeney (2011) also identify the Delphi technique to contain other challenges, such as defining a consensus level, the expert level, and the number of Delphi method variations that are available.

Strengths and weaknesses of the Delphi method have been discussed in a number of studies (e.g. Jones and Hunter 1995; Kennedy 2003; Powell 2003; Hung et al 2008; Hasson and Keeney 2011). These are highlighted below:

3.3.1.1 Delphi weaknesses / criticisms

- Anonymity could lead to a lack of accountability of views or perhaps expressing opinions without full thought; however, this critique could also be applied to any type of questionnaire design e.g. postal, and the engagement of participants over sequential rounds is likely to negate this critique.
- Consensus approach may lead to a diluted version of best opinion.
- Concerns over administration and analyses involved over three rounds.
- A consensus does not equate to achieving the correct answer nor is the method a replacement for rigorous scientific reviews.
- Criticised as a method that forces a consensus with no scope for experts to elaborate on responses. Therefore, this study included multiple opportunities for comment after each section. This helped keep comments fresh and relevant to thought process at this time.
- Criticised in relation to its reliability and validity.
- Feedback mechanism may lead to conformity rather than consensus. Possible problems in developing initial questionnaire.
- No accepted guidelines for establishing consensus, sample size or sampling techniques;
• Time delays between rounds (data collection process); requires time/participant commitment, particularly over typical 3-rounds.

3.3.1.2 Delphi strengths
• Anonymity counteracts dominant character effect as may be observed in meetings and avoids direct confrontation.
• Possibly motivational and educational for participants e.g. an opportunity to compare their opinions to peers without judgement.
• Focused, avoiding distraction and combines a collective opinion. Allows thoughtful consideration, particularly compared to a meeting setting.
• Validity – content is driven by the panel. Applicable where clinical uncertainty exists and for informing on planning future studies
• The Delphi technique enables contact with a group of relevant experts without geographical restriction and high costs. However, low costs benefits may be counter-argued with the extensive time commitment required to administer a Delphi method.

3.3.1.3 Delphi technique methodological rigour
The criticism of the Delphi method in relation to its reliability and validity may be levelled at any qualitative research method (Keeney et al 2001). Hasson and Keeney (2011) explore the rigour of the Delphi technique with consideration to the “holy grail” of research is establishing methodological rigour as the researcher’s responsibility to ensure procedures are conducted to produce dependable results. Hasson and Keeney (2011) provide a useful deconstruction of the methodological rigour for quantitative methods and how this transfers to qualitative studies. Within quantitative studies this involves reliability and validity, whereas in qualitative studies, methodological rigour is measured by applying components of trustworthiness. This consists of credibility (comparable to internal validity), dependability (in lieu of reliability referring to stability of data), confirmability (conveying neutrality) and transferability (external validity and application to other settings) (Hasson and Keeney 2011).

Hasson and Keeney (2011) report key methodological and contextual challenges to establishing rigour in a Delphi study with measurement and continuing modifications being key examples. Reliability and validity are aligned with a positivist or scientific
approach (seeking prediction); whereas trustworthiness is more aligned with an interpretive perspective (exploring and understanding situations). Thus, “transferring such measurements between paradigms is problematic, since both are based on different underlying philosophies” (Hasson and Keeney 2011 p.1696). In addition, Hasson and Keeney (2011) consider that the frequent variations of modifications of this method create challenges to testing rigour. Methodological rigour is explored further in section 3.5.3.

3.4 Ethics
A change in NHS ethics policy was implemented in September 2011, which states that NHS ethics approval is not required for questionnaires/surveys involving NHS staff. However, the NHS ethics policy officer (Scotland) recommends submitting the research proposal to the ethics office, where a letter of confirmation will be issued. This was obtained (Appendix B). In addition, the Researcher’s host NHS health board’s Research and Development (R&D) office was informed in accordance with local procedures. The study was assessed as not requiring R&D Management approval. However, it had been noted by the Research Governance Committee (Appendix C).

The host university’s Divisional Research Ethics Committee (DivREC) procedures were followed (Appendix D) with consent achieved (Appendix E). The Researcher made minor amendments to the study’s original recruitment strategy and subsequently informed DivREC as per local policy (Appendices F and G). These changes were accepted (Appendix H).

3.5. Objectives
Following the detailed literature review used to develop materials for a focus group, the objectives for each phase of this study are:

Phase 1:
1. Facilitate a focus group (physiotherapists) that will prioritise the research literature and review main findings (from a musculoskeletal physiotherapy perspective) for inclusion in a red flag clinical chart. This will also reveal areas of consensus and lack of consensus in preparation for presenting material in the Delphi process.
Phase 2:
2. Construct a draft clinical chart to provide physiotherapists with an initial list of red flags indicators for possible serious pathology in neck pain or neck related disorders. The chart additionally aims to provide information from the literature review to inform clinical decision making that underpins these red flag features.

Phase 3:
3. Conduct a Delphi process with medical experts where iterative exploration of the findings formulated from 1 and 2 will allow these to be refined.

Final outcome:
4. Formulate a list of red flags that may be applied to clinical practice to help detect early indicators of serious pathology presenting as, or alongside, cervical spine musculoskeletal disorders. This list may be presented in a final version of the clinical chart (point 2.)

3.6 Application of the study design
3.6.1 Study design overview
The objectives outlined in section 3.5 will be achieved by the study design displayed in Figure 3.1 (section 3.1.2) and detailed below for phases 1-3.

3.6.2. Phase 1: Focus group
3.6.2.1 Participants: Inclusion criteria
Inclusion criteria for focus group participants were detailed as follows:

1. UK based physiotherapists:
   - National Health Service (NHS) clinical consultant / senior grade or private practitioner with 5-years post-graduate clinical experience; or university physiotherapy lecturer with publications in relevant areas; or lecturer in spinal component of post-graduate Master of Science (MSc) degree in Neuromusculoskeletal Physiotherapy/Manual Therapy;
(An NHS physiotherapy senior grade will typically complete 2-3 years post-graduate rotational work in a junior physiotherapy role before being considered suitable for selection to a senior physiotherapy position).

And;


The MACP is a clinical sub-group of the Chartered Society of Physiotherapy. In addition to their undergraduate training, MACP members undertake extensive postgraduate study to reach a recognised standard of excellence in neuromusculoskeletal physiotherapy (http://www.macpweb.org).

3.6.2.2 Participants: exclusion criteria

Exclusion criteria for focus group participants were detailed as follows:

1. Non-UK based physiotherapists, or

2. UK based physiotherapists not meeting professional experience detailed in inclusion criteria point 1, or

3. Non-members of the MACP.

3.6.2.3 Recruitment

A purposive sampling method was used as the recruitment strategy. Permission was gained from the MACP to contact their membership. The MACP circulated the focus group recruitment call (Appendix I) to its membership. Interested members were requested to contact the researcher directly for additional information (Appendix J).

Twenty-nine prospective participants contacted the researcher to request additional information or to acknowledge the research call for participants. This number progressively reduced due to logistical issues, such as unable to attend for geographical reasons or other prior commitments. In addition, a number of interested participants did not have access to video-link. Three did not re-contact
following receipt of the information sheet. The final number of prospective participants reduced naturally to seven confirmed participants. Therefore, no additional selection process was required. Table 3.1 summarises the prospective participants’ responses with final status of participation or non-participation alongside reasons cited for this outcome:

Table 3.1: Prospective participants’ responses with final status of participation or non-participation with reasons cited for this outcome

<table>
<thead>
<tr>
<th>Prospective participant no’s</th>
<th>Participated or Reasons cited for non-participation</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Participated</td>
</tr>
<tr>
<td>1</td>
<td>Withdrawal without reason pre-focus group</td>
</tr>
<tr>
<td>9</td>
<td>Distance too far to focus group location (Edinburgh) and unable to establish video-link facilities</td>
</tr>
<tr>
<td>9</td>
<td>Acknowledged research call for participants expressing best wishes with project, but cited unavailable due to various reasons; work/course commitments, work part-time only, based outwith UK (USA), specialist interests had developed in other areas.</td>
</tr>
<tr>
<td>3</td>
<td>No follow-up</td>
</tr>
</tbody>
</table>

All prospective participants were based within the National Health Service except:

- 4 were employed as university physiotherapy lecturers,
- 2 worked within independent healthcare,
- 1 worked in private physiotherapy practice, and
- 1 was based overseas (on career break, but maintaining specialist knowledge)

Figure 3.2 outlines the focus group recruitment process.
Table 3.2 displays the focus group participants’ demographic information. These positions are all classed as consultant or senior positions.
<table>
<thead>
<tr>
<th>Identifier</th>
<th>Experience</th>
<th>Years qualified</th>
<th>Education level</th>
<th>Geographic location</th>
<th>Participation Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Clinical (Lead MSK Physiotherapist)</td>
<td>&gt;10</td>
<td>PhD</td>
<td>South East Scotland</td>
<td>Attendance</td>
</tr>
<tr>
<td>2</td>
<td>Clinical (Extended scope practitioner – ESP)</td>
<td>&gt;10</td>
<td>PhD</td>
<td>North England</td>
<td>Attendance</td>
</tr>
<tr>
<td>3</td>
<td>Clinical (Consultant)</td>
<td>&gt;10</td>
<td>MSc</td>
<td>West Scotland</td>
<td>Attendance</td>
</tr>
<tr>
<td>4</td>
<td>Clinical (Combined manager and clinical role)</td>
<td>&gt;10</td>
<td>MSc</td>
<td>North England</td>
<td>Video-link</td>
</tr>
<tr>
<td>5</td>
<td>Clinical (Senior)</td>
<td>&gt;10</td>
<td>Post-graduate diploma</td>
<td>North England</td>
<td>Video-link</td>
</tr>
<tr>
<td>6</td>
<td>Clinical (Principal Physiotherapist)</td>
<td>&gt;10</td>
<td>MSc</td>
<td>South East England</td>
<td>Video-link</td>
</tr>
<tr>
<td>7</td>
<td>Clinical (ESP)</td>
<td>&gt;10</td>
<td>MSc</td>
<td>South West England</td>
<td>Video-link</td>
</tr>
</tbody>
</table>

3.6.2.4 Focus group procedure

Focus group participants may be asked to prepare for the group discussion (Krueger and Casey 2000 p.55). An information pack was prepared and circulated to participants to assist in their preparations for discussion. This pack, which is part-displayed in Appendix L, outlined the background problems, research questions, summaries of research findings relative to these questions, and a format for the discussion.
The aims of the focus group were to:

1) Identify priority areas for inclusion in a clinical screening chart;
2) Discuss any inconsistencies in the literature; and,
3) Discuss the potential structure of the clinical chart (Figure 3.3 is the initial clinical chart design provided within the information pack and presented on screen during the focus group discussion).

Keeney et al (2006) report several studies with their personal involvement that used the literature to inform the basis of their interviews or focus groups as part of a Delphi process. Although Keeney et al (2006) provided opportunity to raise new issues or ideas, they do however, note caution on introducing bias with participants feeling psychologically pressured in not altering their opinions against peer-reviewed literature.

The focus group was conducted through a combination of attendance for those participants able to attend and video link for participants based further afield. Three
participants attended the host venue and four participants engaged through three-video-links in England. The discussion was facilitated by the researcher as a single moderator.

Videoconference (VC) facility was provided through the host university. Connections were established using Integrated Services Digital Network (ISDN) or via the JANET Videoconferencing Service (JVCS), dependent on participants’ VC facility. A VC test was conducted 5-days before the focus group discussion to ensure quality of visual and video connections. Following testing, two support audio-links were established as reserve in the event of loss of VC connection.

The information pack was discussed alongside contributions of participants’ experiential knowledge and opinions (Bohnsack 2004). The focus group discussion was recorded through audio and video means. The video-recording was downloaded whilst logged onto the secure JANET server and transferred to an encrypted information systems device. The audio recording was transferred to the same encrypted system before file deletion from the recording device.

3.6.2.5 Focus group data analysis
Analysing the obtained data is considered as the most difficult phase of focus group research. This requires decisions on who will perform the analysis, what method will be used and to what extent of analysis will be conducted (Litoselliti 2003). Various methods of analysis may be chosen dependent on the anticipated outcomes of the project or guided by time or budget constraints (Krueger and Casey 2000 p. 130-131; Litoselliti 2003)

Qualitative content analysis is the strategy of choice in Qualitative Descriptive (QD) design (Sandelowski 2000). Although content analysis is a specific research tool, it is composed of features similar to other types of research, such as data making, reduction, analysis and validation (Stewart et al 2007 p.120). This type of analysis takes a dynamic form that is shaped to summarise the informational content of the data. Codes or themes are generated from data, which differs to quantitative content analysis where the researcher systematically applies a pre-existing set of codes to the data (Sandelowski 2000). However, in qualitative content analysis the researcher may also start with pre-existing codes. This coding system may be fluid or undergo change throughout the analysis e.g. dependent on how the information
best represents its source (Sandelowski 2000). This was particularly relevant to this study. The focus group information (literature) pack containing formulated questions with sub-categories of literature with flow of pathologies and associated symptoms served as pre-existing codes. The content and organisation of this information is considered as the least interpretive of the qualitative analysis processes; however, it is presented in a way that best suits the data as it is from its own source (Sandelowski 2000).

A transcript-based analysis may involve an unabridged (full) transcript or it may use abridged transcripts. The researcher will typically read the full transcript and make notes, code sections or develop categories of the group discussion (Krueger and Casey 2000 p. 130-131; Litoselliti 2003). However, unitising all of the discussion is generally not practical or is a regular requirement (Stewart et al 2007 p. 122). An abridged transcript should be used by a member of the research team with a thorough understanding of the purpose of the project as this involves constructing a transcript of the relevant and useful portions of the discussion to form a condensed version (Krueger and Casey 2000). This method allows for removal of irrelevant components of the discussion.

A notes based method is based on the researcher's field notes or observations, but may use the audio or video recording as back-up for clarification. The advantage of this method is speed (Krueger and Casey 2000). However, as a single researcher the notes based method was not considered robust as the degree of note taking was limited due to performing the function of moderator. This reduced the capacity to record detailed notes. The researcher may have a number of functions in the focus group: moderator, listener, observer, and analyst using an inductive process to foster an understanding of the topic for discussion, as opposed to testing a hypothesis (Krueger and Casey 2000 pp.11-12). Krueger and Casey (2000) also describe a memory based approach, which requires considerable skill and experience in this methodology; and a long table approach involving the laying-out and cutting of the transcript and compiled into themes. This low-technology method was considered too laborious for the purpose of this phase.

The method selected, therefore, was a combined approach incorporating a full transcript compiled of the group discussion followed by an abridged version. As a
single researcher this provided an additional check with each version constructed from the audio and/or video-recording.

Litoselliti (2003) recommends the analyst works closely with the transcriber to be closer to the data and to read the transcript for general impression before focusing on the substantive aspects of the transcript related to the research questions. However, Litoselliti (2003) also outlines the advantage of the researcher analysing their own focus group data. This creates an opportunity to think about the material as it is collected and processed. The latter method of the researcher processing the data was used in this project to facilitate a thorough analysis and recollection of observations to occur at an early stage. Reading the transcript for an initial impression enabled a seamless extension of early analysis as the researcher transcribed this discussion.

A final stage of data making is recording data in a way to ensure reliability and meaningfulness (Stewart et al 2007). A thematic content was established as described above from the formulated questions with sub-categories of literature to prioritise clinical sections for inclusion in a clinical screening chart and identify a potential suitable format for presentation. Thematic content is a method frequently used in focus group analyses and provides a framework for a more systematic analysis of this data type. This method of analysis should be led by the focus of the research and the researcher’s ability to achieve reliability in the coding system (Stewart et al 2007 p.120). The condensed version / summary of the discussion was circulated to the group participants. This served a dual purpose of providing feedback to participants with the opportunity to respond should any discrepancies be identified, and to enhance the trustworthiness of the data through providing this feedback mechanism.

The outcomes from the focus group discussion were used to inform construction of phases two (development of draft clinical chart) and three (Delphi survey) of this study.
3.6.3 Phase 2: Development of draft clinical chart

As outlined in sections 3.1.2 and 3.5, phase 2 involves development of a draft clinical chart designed to provide physiotherapists (or other non-medical practitioners) with pathology related clinical symptoms and signs within one accessible resource that supports clinical decision making. This clinical information will provide context and detail to support the final list of red flag features produced as an outcome of phase 3 (Delphi survey).

An initial chart design (Figure 3.3 section 3.6.2.4) was presented to the focus group participants for their opinion regarding its potential for development as a tool to support clinical decision making. This draft clinical chart was developed using Mindjet Manager Version 9 for Windows (http://www.mindjet.com) to include information obtained through literature review. The original file was saved and converted to an interactive pdf. format (Appendix M – electronic version has embedded pdf. Hard copy - refer to CD attachment inside rear cover for full view) for distribution to the expert panel. Examples of images extracted from this clinical chart are displayed in Figures 3.4 and 3.5. Figure 3.4 is displayed to a size that provides readability of the core features, whilst Figure 3.5 is reduced to demonstrate the drop-down feature within the main chart. The draft clinical chart was forwarded to Delphi participants as preparation for phase 3. The final version of the full clinical chart will be progressed outwith this study to identify the red flag features through phase 3.
Figure 3.4: Image of draft clinical chart showing quick reference red flag section with main sub-topics below
Figure 3.5: Image of draft clinical chart showing a drop-down feature of a main clinical sub-topic
3.6.4 Phase 3: Delphi survey

3.6.4.1 Delphi survey overview

As previously stated the Delphi technique is typically a 3-round process considered as a suitable method to gain a consensus where there is incomplete knowledge or uncertainty in clinical issues (Jones and Hunter 1995; Powell 2003; Skulmoski et al 2007; Hung et al 2008). This ‘classic’ approach involves presenting a questionnaire to a panel of experts or informed individuals within a specific field to gain a consensus (Keeney et al 2006). Questionnaires are returned, analysed and 2nd round questionnaire developed from responses. This is forwarded to the same participants for completion. The second-round responses are analysed and used to construct the third-round questionnaire. This is forwarded to participants with group feedback returned to provide participants’ with the opportunity to reconsider their responses relative to the group response (Jones and Hunter 1995; Powell 2003; Keeney et al 2006; Skulmoski et al 2007; Hung et al 2008). Keeney et al (2006) suggest that individual responses are returned with group responses to participants for the first and second rounds. However, this first round return method was not employed by Rushton and Moore (2010) and Smart et al (2010). In addition, the anonymity provided to participants through the web-based survey method in this study does not provide individual identities for this to occur. Feedback is recommended in the form of summary statistics e.g. histograms or graphical representations. This enables participants to consider their rankings relative to other participants (Jones and Hunter 1995).

3.6.4.2 Participants – selection and numbers required

3.6.4.2.1 Participants: Delphi inclusion criteria

Inclusion criteria for Delphi survey participants were detailed as follows:

1. Consultant neurosurgeon or neurologist
2. Currently practising within respective specialty
3. Based within the UK or Ireland
3.6.4.2.2 Participants: Delphi exclusion criteria

Exclusion criteria for Delhi survey participants were detailed as follows:

1. Neurology or neurosurgical grades below consultant level e.g. specialist registrar
2. Non-practising consultants e.g. retired
3. Based out with the UK or Ireland

The selection of qualified participants is critical to a successful Delphi. Participants or ‘experts’ should be selected for their knowledge within a chosen field and commitment to a group process. Clinicians within a specific clinical field may be considered as experts and considered appropriate for this selection process (Gordon 1994; Powell 2003; Skulmoski et al 2007; Hung et al 2008). Selection bias should be avoided, such as selection on the basis of acquaintance (Powell 2003; Mitchell 1991 in Hung et al 2008 p.192;). Skulmoski et al (2007 p.4) identifies four requirements for “expertise”:

1. Knowledge and experience with the issues under investigation;
2. Capacity and willingness to participate;
3. Sufficient time to participate in the Delphi; and
4. Effective communication skills (p.4).

Consultant neurologists and consultant neurosurgeons are medical specialists in the relevant areas of this study (e.g. CAD and cervical myelopathy) and form part of a group of medical specialists within the ‘clinical neurosciences’. Consultant neurologists provide non-surgical management for central nervous problems. Consultant neurosurgeons may provide surgical or non-surgical management for central nervous system problems. Both consultant specialties are appropriate medical experts relevant to this study.

3.6.4.2.2 Participants: numbers required

The numbers required to make up an expert panel vary significantly (Gordon 1994; Powell 2003; Keeney et al 2006; Skulmoski et al 2007; Hung et al 2008). The scope of the problem and available resources may influence the number of participants in a Delphi (Powell 2003). In homogeneous groups a sample number between ten to
fifteen participants is considered appropriate (Skulmoski et al. 2007). A group of experts selected from within the clinical neurosciences was considered as a homogenous sample for the purpose of this study. Therefore, this number (10-15) was used as guide to form an expert panel of medical consultants for this study.

3.6.4.3 Participants – recruitment strategy
A purposeful sampling strategy was utilised to recruit this group of medical experts, consisting of consultant neurologists and neurosurgeons (Skulmoski et al. 2007). Skulmoski et al. (2007) advise on the challenges around recruitment of prospective participants who are true experts in their respective fields due their busy schedules. Therefore clear communication and concise questions should be used.

Participants were recruited via two methods;

1) Email distribution direct to two professional organisations’ members of their respective society (Smart et al 2010). The following organisations agreed to circulate a research call (Appendix N) to their respective neurologist and/or neurosurgeon members:

a) The Scottish Association of Neurological Sciences (SANS) (http://www.dcn.ed.ac.uk/sans/). Membership includes neurologists, neurosurgeons, neuropathologists and clinical neurophysiologists working in Scotland, trainees as well as consultants. SANS aim is to improve the care of patients with neurological problems in Scotland, to enhance the training and education of everyone involved in that care, and to promote research.

b) The Society of British Neurological Surgeons (SBNS) (http://www.sbns.org.uk/). The purpose of the Society is the study and advancement of Neurosurgery; and

2) Email distribution direct to consultant neurologists and neurosurgeons (Gordon 1994). This strategy, however, was restricted to Scotland due to limitations in identifying suitable prospective participants. Skulmoski et al. (2007) also suggest a “snowball” sampling technique to generate additional participants after an initial group of experts has been identified. This was achieved through requesting a
consultant to share the information with their consultant colleagues if considered appropriate.

Figure 3.6 outlines the Delphi recruitment process.

Delphi study participant recruitment process

Forward research call (Appendix N) for participants to specialist organisations (SANS and SBNS).
Administrator circulates research call to members

Direct email contact with consultant identified within areas of clinical interest.

Prospective participant contacts researcher to register their interest / request more information

Ensure information sheet provided (Appendix O), plus oral explanation if requested

Participant confirms intention to participate

Draft clinical chart circulated plus issued with final instruction to access web-based Delphi study 1st round (Consent form contained in page 1 Delphi 1st round)

Informed consent (Appendix P) considered as gained when participant clicks web-link to Delphi study 1st round - a method used in a published Delphi study

Participant formally recruited

Figure 3.6: Delphi study participant recruitment process
The participants’ demographic information is detailed in Table 3.3.

Table 3.3: Delphi participants’ demographic information

<table>
<thead>
<tr>
<th>Identifier</th>
<th>Consultant</th>
<th>Years qualified</th>
<th>Geographic location</th>
<th>Main professional setting</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Neurologist</td>
<td>5-9</td>
<td>South East Scotland</td>
<td>NHS</td>
<td>Academia, Research</td>
</tr>
<tr>
<td>2</td>
<td>Neurosurgeon</td>
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<td>3</td>
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<td>North East Scotland</td>
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<td>4</td>
<td>Neurosurgeon</td>
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<td>South East Scotland</td>
<td>NHS</td>
<td>Independent healthcare</td>
</tr>
<tr>
<td>5</td>
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<td>&gt;20</td>
<td>London</td>
<td>NHS</td>
<td>Independent healthcare, Military</td>
</tr>
<tr>
<td>6</td>
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<td>&gt;20</td>
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<td>NHS</td>
<td>Research</td>
</tr>
<tr>
<td>7</td>
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<td>10-19</td>
<td>West Scotland</td>
<td>NHS</td>
<td>Independent healthcare, Academia, Research</td>
</tr>
<tr>
<td>8</td>
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<td>North West England</td>
<td>NHS</td>
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<td>9</td>
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<td>10</td>
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<td>11</td>
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<td>12</td>
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<td>10-19</td>
<td>North East England</td>
<td>NHS</td>
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3.6.4.4 Pilot Delphi

A Delphi pilot is not an obligatory part of the Delphi process. However, it may be applied to test and adjust the Delphi questionnaire to improve comprehension and
address any procedural issues or ambiguities (Powel 2003; Skulmoski et al 2007). A pilot Delphi method was adopted to test these purposes. Three senior medical consultants within the host health board agreed to participate in this process; Consultant Physician, Consultant Radiologist, and General Practitioner. The pilot Delphi recruitment process is outlined in Figure 3.7. Circulation of draft clinical chart and informed consent followed the same process as outlined in main Delphi study.

Figure 3.7 Pilot Delphi study - participant recruitment process

- Gain permission from NHS Borders to undertake the following process
- Circulate pilot study research call via poster/email or verbal for sample of convenience to NHS Borders medical practitioners
- Prospective participant contacts researcher to register their interest / request more information
- Ensure information sheet provided, plus oral explanation if requested
- Participant confirms intention to participate
- Draft clinical chart issued with final instruction to access web-based Delphi study 1st round
- Informed consent considered as gained when participant clicks web-link to Delphi study 1st round – a method used in a published Delphi study
- Participant formally recruited
3.6.4.5 Main Delphi Procedure

Delphi participants within the main study were forwarded the draft clinical chart as discussed in section 3.6.3. Consultants were advised that comments on the chart would be requested during the Delphi 1st round survey (Appendix Q).

To assist reliability or consistency in the interpretation of the questions, a parallel-form testing method was adapted as a pilot process. Parallel-form testing is a process that is recommended to improve stability or reliability of the Delphi technique (Hasson and Keeney 2011). In this study the 1st round questions were submitted to three medical practitioners of consultant or experienced General Practitioner level to explore respondents’ interpretations of the questions (Gordon 1994; Hasson and Keeney 2011).

There is no clear guidance for undertaking pilot tests before implementation; for example, if this should take place before each round or before the first round (Keeney et al 2001). Due to anticipated recruitment and retention challenges associated with busy medical staff it was decided to perform one such pilot test before round one. This was considered as the most appropriate position as round one is related to open questions required to obtain suitable information to inform the construction of questionnaire contained within round two. In addition, achieving a high quality and clear opening round was considered important to enhance retention.

A small number of consultants opted to provide initial feedback through email correspondence. This early feedback was combined with the pilot Delphi feedback to inform development of the main Delphi study. With consideration to cautions outlined by several authors (e.g. Powell 2003; Skulmoski et al 2007; Hung et al 2008) regarding the difficulties of recruitment and retention of experts the researcher opted to remain flexible to these methods of receiving feedback; however, all feedback from email communication was incorporated with the Delphi survey comments for analysis.

An internet based system ‘Bristol Online Surveys’ (https://www.survey.bris.ac.uk/) was used as the data collection system over three rounds.
Round one consisted of three initial short questions to obtain participant demographic data (Table 3.2), followed by a number of questions based on presented information obtained from the literature review and focus group. Appendix Q contains the web-based version of this questionnaire. As consultants had committed time to viewing the clinical chart, the 1st round survey was constructed with a simple strategy to request: chart feedback; consultant’s thought on red flag list with associated conditions that these features may indicate; plus an additional information section if required. This allowed for any narrative around the features

Open ended questions are considered to enhance the content of the data collected, which will then undergo qualitative analysis, and subsequently used to inform the structure of the questionnaire in round two (Powell 2003). The third and final round questionnaire was a repeat of round two with minor modifications applied for clarity. These minor changes were based on additional comments received from round two responses. A reminder scenario (Appendix R) was provided alongside the final round. Appendix S contains the web-based version of this final round questionnaire.

3.6.4.6 Delphi data analysis
A mixed method (qualitative and quantitative) approach is utilised within the Delphi method. The first round is a qualitative design using a thematic content analysis approach to identify key themes (Carlesso et al 2011). A similar approach to the focus group analysis was employed with the coding system developed from the data source. However, the Delphi first round responses were developed and assessed for inter- and intra-coder reliability in the analysis of the 1st round responses. Inter-coder reliability was checked by the researcher and one research supervisor coding the same response data from 3 randomly selected transcripts (Smart et al 2010). Intra-coder reliability was checked by the researcher coding 3 randomly selected transcripts on two separate occasions with a minimum of 2-days a part (Smart et al 2010).

Rounds 2 and 3 utilised a quantitative approach based on responses from round 1. This comprised of a set of questions/statements designed by the researcher. The responses followed a five-point likert scale format (5 = strongly agree, 4 = agree, 3 = no preference, 2 = disagree, 1 = strongly disagree) and subsequently analysed
using descriptive and non-parametric statistics. Kendall’s W (Kendall’s coefficient of concordance) was used to evaluate the level of consensus across all participants’. This method has been used in previous Delphi studies (Rushton and Moore 2010; Smart et al 2010). Round 2 format will also allow for optional additional comments. As round 2 is formulated from thematic content analysis of round 1 data response, this enhances validity of the researcher’s interpretations by providing a facility to respond to any discrepancies identified by participants. Hasson and Keeney (2011) recommend providing the expert panel with the opportunity to perform a check on the 1st round interpretation to improve construct validity. This allows for any necessary amendments by the researcher to occur for round three, if considered appropriate. Round three contained feedback to the participants in the form of graph representation of the highlighting the percentage distribution of participants’ responses (Gordon 1994).

3.6.4.7 Consensus level and non respondents
There are no recognised guidelines for setting a consensus level (Powell 2003; Keeney et al 2006). However, Keeney et al’s (2006) paper to inform research using the Delphi technique recommends a minimum 75%. Therefore, this level was selected for gauging the consensus point. Consensus information gained from open questions was compared with existing literature as a method to help gauge generalisability (external validity) or transferability of the findings (Hasson and Keeney 2011) and provides further guidance to clinicians specialising in musculoskeletal clinics. However, caution should be exercised in interpretation of Delphi outcomes as consensus does not necessarily mean that the correct answers have been found.

Keeney et al (2001) and Hung et al (2008) report poor response rates are characteristic of the iterative process, with Keeney et al (2006) specifying the Delphi final round being susceptible to participant drop-out. Therefore, email reminders were sent on two occasions during each round post-initial circulation as a reminder to non-respondents (Smart et al 2010). This was performed at the two and three week stages.

Hung et al (2008) identify that motivating panellists is one method to address this issue with quick turnaround times in data collection recommended. A one-week
turnaround time was implemented from the closure of round two to the distribution of the final round. The time between the first and second round required an extended period to allow for the qualitative analysis and construction of the second round survey to occur. Keeney et al (2006) suggest that it is critical for the panel to feel they are partners in the study. Engagement in the clinical chart from the outset and incorporating a ‘reminder’ scenario (Appendix R) at the beginning of round three communications were strategies that helped maintain interest. Although this was a small study, a non-response was observed in one case only with no follow through to round 2 from round 1. Opportunistic communication with this consultant reported this reason be due to time pressures. Powell (2003) and Skulmoski et al (2007) note the difficulties in recruitment and retention of experts due to their busy demands from the very nature of their expertise in demand elsewhere.
4. Results

4.1 Introduction

Inconsistencies and limitations within the existing evidence base on the main clinical indicators for potential serious pathology presenting as, or alongside, cervical spine musculoskeletal disorders has been discussed in chapter 2. Chapter 3 subsequently outlined a study design aiming to contribute to this evidence base to improve musculoskeletal physiotherapy screening for serious pathology.

The main findings from two phases within this study design are presented in this chapter: phase 1 focus group with experienced physiotherapists, and phase 3 Delphi survey (3-round method) involving medical consultants. Phase 2 involved the draft clinical chart, which linked phases 1 and 3. The progression of this chart was presented in chapter 3 (methodology). Figure 3.3 (section 3.6.2.4) is the initial chart design presented to the physiotherapy focus group with Figures 3.4 and 3.5 displaying examples of the chart content presented to the Delphi participants in preparation for the Delphi survey method. This may be viewed in Appendix M (electronic version has embedded pdf. Hard copy - refer to CD attachment inserted inside rear cover).

Table 4.1 provides an overview of the main themes presented in this chapter. These themes were identified and explored through phases 1 and 3. The prevalent findings that have emerged from these themes will be identified throughout this chapter. The relationship of these findings to the current evidence base with implications for clinical practice will be discussed in chapter 5.

This chapter contains a number of sections of literature presented as direct quotes or summaries by the researcher in order to facilitate or prompt discussion. These sections are indented to assist clarity in this presentation and to differentiate this information from additional commentary provided by the researcher. Similarly, all discussion based comments / responses are indented and coded by respective speaker for clarity (Researcher or Focus Group participants 1-7 [FG1-7]). An exception to this indentation method is the presentation of any subsequent sub-questions or specific statements generated by the researcher through discussion on the main problems. These
questions or statements are presented in a contemporaneous order with standard alignment and pre-fixed with ‘Q’ to denote a question or statement.

Table 4.1: Overview of the main themes identified and explored during phase 1 (focus group) and phase 3 (Delphi surveys)

<table>
<thead>
<tr>
<th>Main themes</th>
<th>Phase 1: Focus group - Physiotherapists</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Current status on red flags</td>
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<tr>
<td></td>
<td>Cervical arterial dysfunction</td>
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<tr>
<td></td>
<td>Clinical features</td>
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<td></td>
<td>Balance and cranial nerve testing</td>
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<td></td>
<td>Drop attacks</td>
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<td></td>
<td>Neck pain/headache</td>
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<td></td>
<td>Dizziness</td>
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<td>Nystagmus</td>
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<td></td>
<td>Cervical myelopathy</td>
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<td></td>
<td>Draft clinical chart</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Main themes</th>
<th>Phase 3: Delphi Surveys - Medical consultants</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Feedback on draft clinical chart</td>
</tr>
<tr>
<td></td>
<td>Red flag clinical features, which were sub-grouped into:</td>
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<td></td>
<td>o Progressive pain</td>
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<tr>
<td></td>
<td>o Cancer etc.</td>
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<tr>
<td></td>
<td>o Neurological</td>
</tr>
<tr>
<td></td>
<td>o Headache</td>
</tr>
<tr>
<td></td>
<td>o Brainstem, vascular, cranial nerve dysfunction</td>
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</tbody>
</table>

4.2 Phase 1: Focus group

4.2.1 Current status on red flags

The researcher summarised the current status on red flags within clinical practice and the existing evidence base. This primarily included the Clinical Standards Advisory Group list of red flags for low back pain alongside locally developed departmental screening forms incorporating other comorbidities, such as Diabetes Mellitus, cardiac
and respiratory problems. In addition, local departments may adapt variations of the 5Ds And 3Ns framework as discussed in chapter 2.

With reference to specific red flags for the cervical spine, the health information website Patient.co.uk is a resource supplying evidence based information to patients and health professionals and provides a list of red flags for neck pain. A section on ‘cervical radiculopathy’ cites the Clinical Knowledge Summaries (2009) as its source. CKS was a National Institute for Health and Clinical Excellence supported website provide information to support clinical practice. CKS provides a number of references with Binder (2007) found to be the most relevant reference provided. Binder (2007) provides a list of red flags, which is poorly referenced. This work has been adapted for use by Patient.co.uk. This website describes itself as

“…one of the most trusted medical resources in the UK, supplying evidence based information on a wide range of medical and health topics to patients and health professionals” (www.Patient.co.uk).

Patient.co.uk provides the following red flag list:

“Red flags for neck pain
A serious underlying cause is more likely in people presenting with
Red flags suggesting possible malignancy, infection or inflammation
Red flags suggesting myelopathy (compression of the spinal cord)
Red flags suggesting severe trauma/skeletal injury
Red flags suggesting vascular insufficiency” (www.Patient.co.uk).

The low back pain red flags are integrated within specific cervical red flag information. However, due to limited supporting evidence, questions remain on the selection of the clinical indicators and tests as being reliable or of sufficient detail to inform clinical practice.

The researcher, however, suggested this list appears reasonable, but has potential for improvement. For example, this list could be developed. Alternatively, if the list is not amended then the process of providing a list resulting from a more robust supporting structure could provide clinicians’ with enhanced confidence in the red flag information. One such example are red flags suggesting vascular insufficiency include:
- Dizziness and blackouts (restriction of vertebral artery) on movement, especially on extension of the neck with upward gaze
- Dizziness, drop attacks

These clinical indicators form part of the 5Ds And 3Ns framework discussed in chapter 2, which outlined limitations with this framework. This highlights a lack of clear information on cervical spine red flags.

The researcher asked the group the following question:

**Q: Do you have any thoughts or opinions on how to use this current information, for example, do we list the CSAG low back pain red flag list then ask a series of specific questions for the cervical spine or should we integrate the information as in patient.co.uk?**

There were four responses received during this phase of discussion indicating that it was better to have specific groups of pathologies that encouraged clinicians to consider such conditions within their clinical decision making or screening process. There were no signs or expressions from the remaining participants indicating disagreement to these comments. The responses were:

- I would certainly think that it would be more useful to be thinking about specific pathologies and problems when you are going through your risk factors [FG2].

The researcher sought further clarification by asking:

**Q: Do we include the low back pain list?**

Depends on what the use is. If you are thinking of a screening questionnaire for your average MSK department I think you make them do everything, then it is covered from a managers point of view on a clinical governance, but perhaps from an ESP clinic then you will have reasoned that if it is a neck that you will pick out the questions that you need to cover. So I think it depends on the context [FG1].

I like the way that it is separated out into different pathologies. Perhaps having it set out separately will make the clinicians think more carefully as to why they are actually asking these questions, rather than having a tick box screening tool. It makes them think more about why they are asking the questions [FG6].
A back clinic and neck clinic will depend on the patient you have in front of you. There will be some overlap of questions. For example, infection is applicable to both low back and neck pain, where for example bladder and bowel problems will always be asked in low back presentation, but only in neck if suspecting a cervical myelopathy presentation in neck pain. Similarly, wouldn’t be thinking about headache as red flag for low back pain [FG7].

These comments suggest that grouping pathologies would better inform clinical decision making. However, these should be specific to each situation.

4.2.2 Clinical problems
A series of clinical problems (sections 4.2.2.1 – 4.2.2.5) were read by the researcher and discussed within this group. These problems were provided in advance to participants within the focus group information pack alongside relevant literature (Appendix L) to inform this discussion. The findings identified during this discussion are presented in the remainder of this section (4.2.2). As stated in 4.1 additional researcher-led sub-questions and statements evolved from discussions on the main problems and are presented in a contemporaneous order. These are pre-fixed with ‘Q’.

4.2.2.1 Problem 1: What signs and symptoms indicate the presence of CADysfunction? Studies appear to be based in neurological centres; therefore it is questionable as to how relevant the findings are to MSK clinics? Admission to neurological centres indicates significant progression of an arterial event. Taylor and Kerry (2010 masterclass) provide excellent information on CADy from a manual therapy perspective. However, the following studies highlight the difficulty in identifying features relevant to MSK clinics. Other than single case reports, there are no identifiable studies within MSK settings (excluding adverse events based reports).

4.2.2.1.1 The presence of pain and/or stiffness
The researcher cited Thomas et al (2011) in reporting that headache, neck pain and dizziness was not always present in patients within their retrospective study of cervical arterial dissections. Therefore, the researcher proposed the following to the group:
Q: For the purpose of musculoskeletal clinics, it is reasonable to assume that neck pain, headache and/or upper limb dysfunction would feature, otherwise the patient may not present at a musculoskeletal clinic.

A participant responded to this:

Well, do people sometimes come into clinic with stiffness rather than pain as their main complaint? May be unusual, but neck stiffness is possible rather than pain [FG6].

There were three to four general agreements to this comment indicated through head nodding actions with one additional comment received that also agreed this was reasonable to accept that stiffness may feature as a complaint without the presence of pain.

Yes, stiffness is possible [FG3].

There were no signs or expressions of disagreement to this comment.

The researcher added that it is important to consider what other local symptoms and signs may be present. Therefore, the researcher provided a summary of literature provided for this section that was contained within the focus group information pack (Appendix L). This summary included symptoms and signs from Thomas et al (2011):

“**Symptoms:** Pain, dizziness, Visual disturbances, paraesthesia face/UL/LL

**Signs:** unsteadiness/ataxia, weakness (UL/LL), dysphasia/dysarthria/aphasia, facial palsy, nausea/vomiting, dysphagia, drowsiness, confusion, loss of consciousness” Thomas et al (2011 p.355) [Researcher].

And in addition, Chandra et al 2007:

“Other associated symptoms such as amaurosis (loss of vision 1 eye), anisocoria (unequal pupil size), ipsilateral facial droop, partial ipsilateral Horner’s syndrome, and neck pain were seen to occur in one-third or fewer patients in each group” (Chandra et al 2007 p.180) [Researcher].

There were no additional comments received to this prompt.
4.2.2.1.2 Balance testing in neck pain, headache or dizziness

The researcher reported that Kerry and Taylor (2010) and Thomas et al (2011) recommend incorporating balance and cranial nerve testing (CNT) into the routine musculoskeletal examination to detect potential symptoms and signs of cervical arterial dysfunction. Therefore the researcher asked the following question:

Q: Do you or your departmental colleagues currently consider balance testing in neck pain or headache?

Approximately three initial responses indicated that this was not used routinely, unless dizziness or falls were identified during the subjective assessment as part of the presentation. Thereafter, balance or an alternative e.g. Romberg’s test is incorporated into the assessment. Testing for balance, however, was also reported as being very variable between staff members and locations, typically dependent on level of training or location of specific staff e.g. with vestibular rehabilitation experience being more likely to perform balance testing.

I could say that we don’t use it routinely, but if dizziness was part of the picture then we would use it. It would depend on the context of the patient’s subjective [FG3].

The researcher explored this further with a scenario:

Q: If a patient complained of neck pain or headache and dizziness featured, you would test balance?

Yes [FG3].

Approximately four other participants agreed with this statement through “yes” and head nodding actions. There was one additional comment stating that Romberg’s test would be included for people with dizziness.

Yes, we do that for people suffering from dizziness. We do Romberg’s usually [FG4].
Other comments on integrating balance into neck assessment were provided:

We do all sorts of things. I brought along a comprehensive chart..., but it includes the sort of things that you are probably going to bring up [FG1].

This was confirmed as a document specifically developed for this participant’s local department use, which included testing for balance in this scenario.

Testing for balance is very very patchy here (video-link participant) depending on the kind of in-service training or external courses the physios have been on. I mean I have worked with physios who have been on loads of extra courses on vestibular problems and they always incorporate it into their tests and they will obviously get other people doing it as well, but that is very patchy and only occurs in some places [FG6].

The researcher clarified this phase of discussion did not relate to a patient referred in with a specific complaint of dizziness or for vestibular rehabilitation, but rather a musculoskeletal setting in which the person complained of dizziness you would test for balance?

Four responses were noted to indicate agreement to this through stating “Yes” or head nodding action, that balance would be tested in this scenario. One additional extended comment was received:

But if they didn’t complain of dizziness, no, unless there was some other indication like falls that would send you that way, but then it would be a routine [FG1].

The researcher clarified this situation that if clinical reasoning or decision making in the scenario indicated a need for testing, then this would be incorporated.

Yes [FG1].
4.2.2.1.3 Cranial nerve testing

The researcher reported that Thomas et al (2011) recommend cranial nerve testing (CNT) due to ICAD findings in their retrospective study. However, symptoms and signs referred to were also present in VAD. In addition, the physiotherapists’ Taylor and Kerry’s (2010) masterclass type paper published in an osteopathic journal also recommend conducting a simple eye exam and CNT. This paper was omitted from the information pack due to it not containing patient observations.

The group was asked the following question on CNT:

Q: Do you or your departmental colleagues currently do cranial nerve testing in neck pain/headache presentations?

Four “No” responses were received plus two participants reported their respective locations had received training in cranial nerve testing (including crib sheet) but this was not performed frequently (estimated ≤ 3 times per year). One location was in an Extended Scope Practitioner (ESP) role, while all musculoskeletal staff within the 2nd location were trained in performing CNT. However, this was rarely used. Comments also suggested that there may be a confidence issue if not performing the test on regular basis.

In my ESP clinic I have been taught to do cranial nerve testing and I do have a little crib sheet to help do it if I need to, and I think that is quite appropriate in that setting, but I probably wouldn’t expect the rest of the physio dept unless I have gone through that with them to be very adept at doing it, but I don’t feel very confident at doing it myself unless you are doing it all the time, and a lot of time you only need to do it when you need to clinically, so that is very rare actually [FG7].

The researcher prompted expansion on this response through further enquiry:

Q: How often was this type of examination conducted over the past twelve months?

Probably about 2 or 3 times in the past year [FG7].
We had ........(name deleted) here doing a training session with all members of the physio staff and I would say that nobody uses it on a routine basis at all and it is very rare for some to find the sheet that tells us how to do it. I would say less than three times a year [FG4].

There were no additional comments received for this phase of the discussion.

4.2.2.1.4 Temporal component
Q: Another component identified in literature is the temporal or time factor i.e. A time period that exists from preceding warning symptoms to the onset of more serious development. Do you think that is an important aspect that could help guide our clinical reasoning process? A clinical example is a patient reporting the presence of dizziness alongside neck pain/headache or stiffness. Is the duration or length of time that dizziness has been present of relevance to you?

There were four responses indicating (head nodding action) and/or stating:

Yes

The researcher asked participants about the relevance of what this information would provide you with.

Q: What else would it add?

Well it would depend if the onset of dizziness coincided with the onset of their problem or if they have always been dizzy. You have to clarify, don’t you? [FG5].

Approximately four “Yes” or head nodding actions indicating agreement were received with extended comments detailed as follows:

If it was progressive then you would take action. The majority of people that come to see us may have actually had dizziness for a long time before they actually see us. It is about the onset [FG5].

Participant added:

Reading through some of the literature there was such variance from the literature from minutes to days to weeks that it was very difficult that it might be useful to know and ask the question by episode or by how long they have had a continual headache and did it coincide with their other symptoms. But you would
need to be quite clear back to the literature on what parameters you were going to use because it was so varied that I think that would be quite difficult to pin down [FG3].

The other thing to consider is that if they have got a severe headache they will be taking medication and the effects of the medication, whether that is actually giving them a headache and giving them dizziness because they will be taking presumably opiates or something to do with that [FG5].

To invite additional comments the researcher suggested that:

**Q:** It would appear the temporal component is of relevance with potential to add value to physiotherapy clinical decision making processes.

A further response was received:

Can I say that it only adds to our reasoning process only if we know what proportion go on to develop a serious adverse event at that stage [FG2].

The researcher added:

This aspect was not evident from the literature review. One potential problem in ascertaining this information is the likelihood of quick medical intervention should symptoms be detected at an early stage by appropriate clinical staff [Researcher].

There were no additional comments received for this phase of the discussion.

**4.2.2.1.5 Presenting symptoms and signs and their relevance within current physiotherapy screening methods**

The researcher summarised Savitz and Caplan (2005), which is based on findings from New England Medical Centre Posterior Circulation Registry. This is cited within physiotherapy based literature (e.g. Kerry and Taylor 2008) and was provided in the focus group information pack (Appendix L and summarised in Appendix A). This information was provided as a prompt for discussion on aspects such as, symptoms and signs presenting in isolation versus likely to be accompanied by other features, and the relevance of any of the following features within current physiotherapy screening methods:
4.2.2.1.5.1 Atherosclerotic stenosis and occlusion (The researcher read the following)

“Near the origin of a vertebral artery in the neck.....often presents as brief TIAs (Transient Ischemic Attacks), consisting of dizziness, difficulty focusing visually, and loss of balance” Savitz and Caplan (2005 p.2619) [Researcher].

No comments were received. Therefore, the researcher added the following question:

Q: Regarding TIA, does it always have these three components present or just part of it?

The Group were unable to provide further clarification.

The researcher continued with the summary from Savitz and Caplan (2005):

“Intracranial vertebral artery.....most often causes symptoms and common signs referred to as the Wallenberg, or lateral medullary syndrome” (Savitz and Caplan 2005 p.2619-2620) [Researcher].

Researcher added:

Serious pathology of this level or full stroke is highly unlikely to present in MSK clinics. This is acknowledged by Taylor and Kerry (2010) who state that it is unlikely full stage will present to manual therapist, but subtle retinal ischemia might [Researcher].

Three nodding actions were noted indicating agreement to this. There were no signs or expressions indicating disagreement.

4.2.2.1.5.2 Arterial dissection (The researcher read the following)

“The cardinal symptom in patients with vertebral dissections is pain, most often in the posterior part of the neck or occiput, spreading into the shoulder. Diffuse, mostly occipital, headache also occurs” (Savitz and Caplan 2005 p.2620) [Researcher]

Researcher added:

No supporting data is presented for this within this particular paper [Researcher].
“Dizziness, diplopia, and signs of lateral medullary or cerebellar infarction can ensue from embolism or extension of the dissection to the intracranial vertebral artery” (Savitz and Caplan 2005 p.2620) [Researcher].

Researcher added:

This may suggest a later presentation, yet the literature also suggests that a TIA (consisting of dizziness) may be early. This potentially creates confusion for clinicians. Therefore, dizziness could be considered as possibly presenting early or later [Researcher].

The group were invited for any comments or opinions on these components. No additional comments were received at this stage.

4.2.2.1.5.3 Drop attacks (The researcher read the following)

“Drop attacks have inappropriately been attributed to transient ischemia of the posterior circulation. Not a single patient in the NEMC-PCR had a drop attack as the only symptom” (Savitz and Caplan 2005 p.2621) [Researcher].

Researcher added:

Q: Therefore, if asking this question, are we screening for another reason? For example, cardiovascular rather than a cervical arterial dysfunction problem.

No additional comments were received at this stage. The following question was then asked:

Q: Does this call into question the inclusion of drop attacks within the 5Ds And 3Ns framework when considering cervical arterial dysfunction?

Well I would ask, what are people asking when they ask about drop attacks. Within the department there is a huge range of what people will actually say to the patient from “have you ever collapsed?” to “have you ever found yourself on the floor unconscious and not knowing why?” You know, it depends on how they establish what people say to establish that their drop attack has occurred. I don’t know if everyone else has had this experience with their staff or not. And certainly I would say there is a huge range or variation in what people are actually saying or asking [FG4].

I sometimes think the understanding of what a drop attack is not always accurate, which is in the type of questions that are asked [FG6].
It would therefore appear that the issue of drop attacks and loss of consciousness lacks clarity. No additional comments were received at this stage.

4.2.2.1.5.4 Differentiating ICAD and VAD sites of pathology

The researcher reported that physiotherapy literature suggests a clinical reasoning approach to identify if the arterial problem is ICAD or VAD in origin (e.g. Taylor and Kerry 2010). Taylor and Kerry (2010) recommend use of palpation skills for the carotid artery in times of suspicion as one method to assist differential diagnosis. Therefore, the researcher asked the following question:

Q: From the perspective of screening for potential red flags in cervical arterial dysfunction is there clinical value in trying to reason out if it is ICAD or VAD, versus grouping the two together?

I just don’t think that it is appropriate for us to try and do this……we just need to consider if there is some serious pathology. That is not for us [FG5].

I think at the end of the day, what we are looking at when we are assessing a patient is we are looking towards what we can do for that patient and what that treatment is going to look like and how safe is that treatment going to be. So I don’t think it is that relevant to know what artery it is that we are looking at [FG3].

Q: Do you think that palpating the artery will add anything more as a physiotherapist?

I don’t think that is a skill that is relevant to us and I think we would be laughed off the podium by our medical colleagues if we were trying to diagnose from that [FG3].

Two additional comments agreed with this through “yes” and/or head nodding action, with one additional participant offering an alternative perspective:

I was just going to say we do palpate for abdominal aortic aneurysm so we do have a history in saying that we are able to do that and we can use that in our diagnostic process, so I am afraid that I don’t know if there is something that we could palpate in a carotid artery that would give us any information that would be useful, but if there was something we could do, then I would not be averse to
learning how to do it. So I don’t think it is outside our scope of practice because we have got a precedent with abdominal aortic aneurysms [FG4].

This point identifies that there could be a training issue present. However, there are reservations regarding the profession’s credibility on palpating the carotid artery for diagnostic purposes. Participants were asked any additional opinions on separating the internal carotid and vertebral artery from a musculoskeletal perspective?

Personally, I can’t see the value in that. I think as said, as musculoskeletal physiotherapists we are looking to see is there anything we can do to add to that person’s journey and if it is not a musculoskeletal problem then we can pass them onto a professional who can deal with them. So it is just about being able to identify, from an extended scope capacity, which professional would be of use to them. So that is why we need the information. So for me, I don’t think that we need to differentiate the two [FG4].

I agree with…(deleted name inserted FG4) and I was thinking as well that with some physiotherapists using the ultrasound scan that they would be appropriate to them to be able to scan the artery, but for the majority of people it is not [FG7].

This phase of the discussion highlights inconsistencies and uncertainties in the information interpreted from the symptoms, signs and diagnostic skills on identifying arterial dysfunction and what aspects are relevant to physiotherapists. This suggests there is potential benefit seeking direction from the medical profession.

4.2.2.2 Pain

Pain is a symptom that has been mentioned previously. Therefore the following problem was read to the group by the researcher:

Problem 2: Neck pain and/or headache symptoms are reported as the most frequent local symptoms of cervical arterial dissection. There are suggestions that onset of headache is described as “unlike any other”, that pain associated with vertebral artery dissection (VAD) is typically posterior neck pain/occipital headache, and that pain associated with internal carotid artery dissection (ICAD) is typically upper-mid neck pain/ipsilateral front-temporal headache.

Q: Do you think these descriptors for onset and distribution of CAD related pain are sufficiently evidenced for use as clinical indicators?
Purely based on what you have presented to us, I don’t think we can say that [FG2].

I don’t think so [FG3].

Yes. I would agree with that. You cannot tell from the location of the pain, what you would be dealing with [FG4].

Or the intensity of it [FG2].

I thought the description of it was vague as well. Was for the reason, if you were asking someone about the uniqueness of headache or that it is unlike any other, or its intensity, in terms of pain that is only valid for the time that they say it. It is not valid – it depends on their stress levels on the day, their emotions on the day, because it is very subjective, so I didn’t think it was very robust [FG3].

Or if you have ever had a headache before [FG1].

Yes [FG3].

The general opinion appears to be that no clear patterns of pain exist. However, caution should be noted with this interpretation as this was part influenced by the literature and not entirely on participant observations as demonstrated by FG2’s opening comment in this section of discussion stating this response was based on the literature provided.

4.2.2.3 Dizziness

Dizziness is listed as a symptom of arterial pathology (e.g. Thomas et al 2011) and forms part of the 5Ds And 3Ns framework. Therefore, the following problem was read to the group:

Problem 3: Dizziness is reported as a significant symptom potentially indicating serious pathology e.g. Australian guidelines 2006. The term “dizziness” may encompass vertigo, presyncope, unsteadiness, and other non-specific forms of dizziness (Tarnutzer et al 2011). Dizziness may be the result of a peripheral condition (e.g. benign paroxysmal positional vertigo, vestibular neuritis, Meniere’s syndrome, or postural hypotension) or a more serious central cause
(e.g. ischaemic stroke in brainstem or cerebellum, or tumour of posterior fossa). What features of dizziness and what clinical tests would aid MSK physiotherapists’ differential diagnosis of a peripheral versus central cause of dizziness?

Two additional points extracted from the literature (Tarnutzer et al 2011) were read to the group by the researcher before inviting comments. This information was considered from the perspective of learning new skills to enhance the screening process.

1. “Vertebrobasilar ischaemic stroke may closely mimic peripheral vestibular disorders, with obvious focal neurologic signs absent in greater than half of patients presenting with acute vestibular syndrome due to stroke” (Tarnutzer et al 2011 p.1025) [Researcher].

2. “A three-component bedside oculomotor examination – HINTS (horizontal head impulse test, nystagmus and test of skew) identifies stroke with high sensitivity and specificity in patients with acute vestibular syndrome and rules out stroke more effectively than early diffusion-weighted MRI” (Tarnutzer et al 2011 p.1025) [Researcher].

Q: Could this be applied to MSK clinic screening or is it for more serious presentations?

This topic generated several detailed responses:

I see some patients referred from the ENT clinic in…(place deleted) and they have a dizzy clinic which is specifically designated to see dizzy patients. And I would say that we have to be very careful with this as they use a whole battery of investigations in order to identify why the patients are dizzy, so they use prism glasses, caloric testing, MRIs on top of using things like Hallpike, using the history, to differentiate. I mean they will say for example that in BPPV that dizziness will last for less than a minute and always reproduced by position, so the history is one thing, but they use a lot of investigations on the patient before they make a decision that they have benign dizziness. So I do think we have to be quite careful how we structure ours, whether we take on board the ability to assess dizziness [FG4].

I have the reverse that they do come from the consultant. They get one minute if they are lucky. They’re never allowed to speak then they all come down with disequilibrium for vestibular rehab. It doesn’t really matter about – or TMJ they can come down with, and that’s it. Now the starter is that they have waited a long time to see the consultant and then sit on our waiting list for a while, so I suspect that if they were going to have stroke they would have had it. So from
that point of view there is a little bit of safety in the length of time that they have had it. But we do treat them and do get very good results from them, but we are working now with some of the audiologists, who as ...(deleted name inserted FG4) has said do a lot more than we do and have lot more sophisticated tests. But we have put together, and I have brought that today, a proforma for some basic testing and probably these are ones with benign dizziness, but again, many of the patients that come to us, I would say have not been properly screened. But I take your point ....(deleted name inserted FG4) that there are so many causes of dizziness. However, I would say that having treated for a large number of years now I think that once you have done that you can pick out the benign ones a little bit more easily. And then I do think it is case of referring on those that you think that you are not happy with. But I think our waiting lists probably help in terms of the seriousness of it [FG1].

I think in terms of the three component bedside examination the caveat I have there is that is a bedside examination that these patients have been admitted so they have probably actually had some event and that’s why it is sensitive and specific. Where the patients that we see, we’re wondering is there a threat of pathology here or is there likelihood, so I don’t know if that is particularly relevant to us as MSK physiotherapists. I would need to know more [FG3].

For me the whole question of dizziness testing is the context. If you are treating a patient then yes you probably do need these skills and you are probably in setting where you have back-up from experts and investigations. When you are in the MSK clinic you presumably treating for neck pain or stiffness predominantly, I don’t think this is appropriate [FG2].

In response to FG1, the researcher suggested that there is a safety net because the patients are referred from a consultant. This generated the following response:

They don’t all come, but a large amount of them do......But I think there is also a context in terms of, it is one thing to have patient sent to what is a routine physio list, where you have got 45 minutes, you have got time, you can think about it, you phone a GP. Where it is a different context in an ESP clinic where they may be coming through the doors at vast rate and then you have to decide if they have got something serious, and they also may have been sent by the GP because they are not sure, so there are contexts for both. So if we see these patients, we give them a thorough screening and we go through all the different tests and try an make a decision at the end of that, which I think is quite different to a clinic where I don’t know how long your appointments are, but it could be 20 minutes, half an hour, so there is context to both. So what is appropriate for one setting is probably not appropriate for the next one [FG1].
The researcher commented that the aim was not purely for an advanced clinic setting, but could be applied to a standard outpatient clinic, before further adding the following analysis on the tests:

One test was conducted in a primary care setting indicating specificity and sensitivity in this environment. However, having reviewed the tests in action through web-based video the most specific and sensitive test, the horizontal head impulse test involves high acceleration / high velocity with 20 to 30 degrees of rotation. If a patient is presenting with neck pain or neck stiffness this test is likely to introduce added complications rather than assist in a screening process [Researcher].

One participant added agreement to this comment [FG3]. An expanded response to an earlier comment was added from FG1:

Well I chose depending on what sort of age and co-morbidities there were, I would choose as what sort of tests I would apply a reasoning process to it, for example, I am not going to do a Hallpike, but I might do the alternate Hallpike on an older patient. I won’t do an Epleys’ I might send them home doing BrandtDaroff exercises instead, so there is a bit of reasoning, but again if you are trying to get something against how much reasoning there is, what level people are at, again it is a basic screen against a more advanced screen [FG1].

The researcher clarified that the focus of this particular process was to improve clinical reasoning or decision making at the early stage in identifying how serious the presentation is, as opposed to aiming to diagnose and provide ongoing management. The following extract was presented from Tarnutzner et al (2011):

“Best evidence suggest that nearly two-thirds of patients with stroke lack focal neurologic signs that would be readily apparent to a non-neurologist and one-third lack signs that would be readily apparent to a neurologist” (Tarnutzner et al (2011 p.1031) [Researcher].

The researcher suggested:

**Q:** This statement cautions against clinicians being led into a false sense of security that serious pathology is not occurring and may be a case where non-medical practitioners may not be sufficiently skilled to detect such pathology. The group were asked for comments on this information and if it was reasonable
to highlight that we (physiotherapists) are very limited in our skills to differentially diagnose this problem.

It is, but then we run the risk of saying that every dizzy patient that walks through the door that we end up saying that we better not do anything with them. And it is getting that balance and that is the tricky bit [FG2].

There were no additional comments at this stage; therefore, the researcher added that the literature review provided patterns that may help in this process with the following identified (The researcher read the following summary information from Bhattacharrryya et al (2008) to the group before asking a question):

- **Peripheral (BPPV)**
  - BPPV less than one minute (also mentioned by FG4 earlier in discussion)
  - Relative to gravity
  - No hearing loss
  - Physical exam: vertigo and/or nystagmus from DIX-Hallpike

- **Otological**
  - Usually hours, present at rest
  - Disabling
  - Hearing deficit likely
  - Plus or minus nausea vomiting

- **Central**
  - Usually less than thirty minutes
  - Isolated and transient
  - Aware that it maybe weeks/months pre-stroke (Summary from Bhattacharrryya et al 2008) [Researcher].

**Q:** Do you think it would it be reasonable to put that sort of information to a neurology group - is there any value in us trying to use that sort of information to try and work out if dizziness is linked to a central or peripheral cause. Any thoughts on that?

But there are so many causes of dizziness. I am thinking postural hypotension that could mimic; I think they will say, yes this could provide a little bit of guidance, but it is not going to give you any definitions or diagnosis [FG6].

I think the way the person describes their dizziness can certainly give you some indication as where their dizziness is coming from and what’s its cause. Definitely, if they are lying down and the room is spinning or if it is positional
lasting thirty seconds to one minute when they turn in bed. I think that can help, yes [FG6].

Just from my perspective of treating them for long time, the time definitely helps in deciding, but there is also a whole range of other factors, but for me I would consider the timing just in terms of how they have described it. I would agree [FG1].

Because we ask patients do you suffer from dizziness, and dizziness covers such a huge range of things, doesn’t it. And sometimes patients don’t understand what you are talking about dizziness. Is it light headed dizziness, is the room spinning, are they unsteady so helping to clarify in question of what we actually mean by dizziness or get the patient to clarify clearly what they are feeling [FG6].

I agree with that. If anything it comes back to what questions physiotherapists are asking with the phrasing of the questions making a difference to the weight of usefulness of that piece of information. So if you are going to take the point and put it into some sort of programme, questions that should be asked or could be asked in terms of risk management and as (Name deleted) did with her red flag book and weight them, it might be a useful question to ask that you might give it low weighting so this is low probability, but this increases the probability where if someone describes as spinning lasting for thirty minutes it might be a high probability. But I think the way people ask the questions is going to be the key thing as to how useful the answer is [FG4].

The researcher added:

So if I bring this back to our current evidence base that we are using, as in Coman’s 5Ds and simply stating dizziness, it is just not appear to be enough information. We are trying fine tune some of these components. We might be limited with using something like the 5Ds, but can we then define it a little bit more that may help us, and I think that is what is coming out here. If we can provide other information like position or temporal aspects it could give us better information [Researcher].

There were no additional comments at this stage of the discussion.

4.2.2.3.1 Nystagmus

Nystagmus forms part of the 3Ns framework. Patten (1998) was cited to describe nystagmus to the group as:
A sign, not a disease, with involuntary movement of the eyes (Patten 1998) [Researcher].

In addition:

Bhattacharryya et al (2008) describe directional patterns to aid differential diagnosis of central versus peripheral causes of this sign [Researcher].

The group were asked:

**Q: Are we sufficiently skilled to interpret this?**

I think it takes training [FG1].

I think probably not. Ok. I think we can learn to look at the Dix-Hallpike test and recognise normal nystagmus there, but if you read the literature there is just so many different types of nystagmus as to what is going on and I don’t think an ordinary MSK clinician, well not an ordinary one, but a highly skilled one, MSK clinician, can be expected to read nystagmus as they would in an expert neurology clinic. Because it goes upwards, sideways, downwards, backwards and frontwards, you know, I mean we can say if it is there, but we can’t really differentiate as to what it is indicating, I don’t think [FG6].

Two agreements were noted to this statement with a further expanded response:

It probably needs to be interpreted alongside other tests we wouldn’t be performing in any case in our clinics. So, along with other investigations [FG7].

The researcher added:

Referrals may come from a safe environment, for example, specialist consultant after all tests completed and serious pathology excluded. However, we need to consider if we are presented with that patient for the first time for example through self referral or a GP [Researcher].

Participant FG3 added:

Or NHS24.

Patten 1998 is a medical text on neurological differential diagnosis, which states that the interpretation and reporting of nystagmus is poor. Therefore, as physiotherapists, recognising such limitations may be important [Researcher].

The researcher suggested a recommendation should be made that:
If nystagmus is present then onward referral for specialist opinion is considered appropriate. The patient may be re-accepted for further management once cleared from a central or serious cause [Researcher].

Q: The group were asked if this was a reasonable approach and invited comments on this:

Approximately 5 ‘Yes’ responses or head nodding actions were received indicating agreement to this statement. There were no signs or expressions noted to indicate disagreement.

4.2.2.4. Risk factors
Manipulation and minor trauma were included within the focus group information pack as a risk factor for cervical arterial problems. Therefore, the researcher highlighted a recent ‘Head to Head’ discussion article “Should we abandon cervical spine manipulation for mechanical neck pain?” (BMJ 2012;344doi: http://dx.doi.org/10.1136/bmj.e3679) for further reading. This article was not discussed within this focus group; however, one additional point was raised by the researcher on recognising limitations in vascular screening blood flow studies to support positional tests (e.g. rotation/extension). This information was provided within the information pack (Appendix L):

The other point that I would like to make is that within physiotherapy it is recognised that our vascular screening blood flow studies to support those tests are inconsistent or conflicting evidence [Researcher].

The group were invited to make any comments or express thoughts/opinions on this aspect. No responses were received.

4.2.2.5 Cervical myelopathy (cord compression)
The following problem was read to the group:

Problem 4: It is a clinical challenge to identify cervical myelopathy (CM). What signs and symptoms have been identified in this presentation that should be used to screen for this pathology?
Similar to the background problem on the 5Ds And 3Ns framework the aim of addressing this issue was to refine the information on these presenting features e.g. gait disturbances or bladder dysfunction. For example, bladder dysfunction is typically the basis of questioning within the subjective examination, however, clarification on the components considered as a dysfunction is often lacking in the literature as identified in chapter 2. Therefore, increased awareness of the range of features that may appear within bladder dysfunction or gait disturbances may enhance physiotherapists clinical decision making [Researcher].

There were no additional comments at this stage.

The researcher further presented additional information:

Rhee et al’s (2009) prospective controlled study highlight the potential difficulty in identifying this pathology where: 21% (8) of 39 Cervical Myelopathy patients did not demonstrate a single myelopathic sign at their initial presentation. These patients had confirmed myelopathy with subsequent surgical intervention [Researcher].

Due to time constraints the researcher proposed to summarise main findings from the information pack where participants could offer thoughts/opinions as they arise. The Group agreed.

4.2.2.5.1 Sexual dysfunction
The researcher read the following:
  o Sexual dysfunction
    o It is low frequency/prevalence; subjective difficulty in penile erection or ejaculation.

Focus group preparations revealed that a group participant [FG2] previously conducted PhD level research on lumbar spinal stenosis; therefore, the researcher explored this further by asking:

Q: Did your research reveal any additional information on the frequency of this problem or indicate a requirement to include this within routine questioning?

No [FG2].

4.2.2.5.2 Bladder dysfunction
The researcher read the following:
o Bladder dysfunction was identified as having two components (Sakakibara et al. 1995).
  o Irritative with subgroups;
    o Frequency, urgency, incontinence, and
  o Obstructive, with subgroups;
    o Hesitancy, prolongation, difficulty voiding, retention

The researcher then directed the following question to FG2 before inviting group comments on this prompt for bladder dysfunction:

**Q: Were any additional findings identified in this area during your research?**

Nothing quantitative and it is a real problem with stenosis patients that they quite often are getting older [FG2].

Another participant sought clarification

Going more frequent? [FG1]

Yes, and quite often the men have got prostate problems. So no, nothing quantitative on that [FG2].

The researcher added:

I (personal) think it is helpful to be aware that it can be any of those types of dysfunctions, rather than simply stating bladder dysfunction. Would that be helpful to physios? [Researcher].

Participant FG6 responded:

I think just general education about all the different causes of bladder dysfunction is absolutely essential because people get themselves wound up into so many problems about bladder dysfunction and erectile dysfunction problems, and I am sure that patients are sent of left, right and centre unnecessarily because the clinician hasn’t really understood the answer or have re-phrased the question wrongly. So we definitely need clarification as to how they should be addressed I think [FG6].
Four group members indicated agreement with this comment through nodding action and/or stating “Yes”. There were no actions or expressions indicating disagreement.

The researcher added that no specific information was identified on bowel dysfunction. Therefore, the group were asked for any additional information on bowel dysfunction. This generated one response:

   Nothing obvious [FG1].

No additional information was identified by the researcher on patterns of bowel dysfunction. Similarly, no additional group comments were received.

4.2.2.5.3 Gait disturbances
The researcher read the following summary for gait disturbances:

   Three studies were identified in the literature with consistent findings on gait disturbances. Gait disturbances were identified as having two main components:
   o Linear parameters with sub-groups;
     o Slow gait
     o Increased step/stride length/step width/double support
     o Decreased single limb support, and
   o Kinematic parameters with sub-groups;
     o Decreased knee flexion
     o Increased (ankle) Dorsiflexion
     o The latter 2 are suggestive of spastic gait pattern

The researcher explored opinions on the breakdown component parts of gait through the following comment and question:

   As a clinician I thought this would be useful to know rather than just saying “have you any gait disturbances?” I think traditionally, asking “are you tripping over yourself?” or, “are you moving slowly because of pain?” is not clear. Whereas, with this sort of information I have a better idea as to what it is I am assessing or asking questions to do with gait [Researcher].

Q: Would that be reasonable?
Participants’ expressed the visual assessment of gait was more important compared with questions:

Yes, but I think it is something that is much easier to establish by assessing or observation. For me, I don’t ask gait questions too much. I look. I might ask some preliminary questions [FG2].

Again with the questions, I mean how you phrase it when you are asking the patient really. Are you unsteady on your feet? Do your legs give way? Do you veer off to one side? You know, there are so many different connotations of this question. I have picked up several patients with cervical myelopathy. Two had a wide based spastic gait, and the other two had little shuffling type gait. And how do you phrase that into a question. I think looking is important here. Observation is the word [FG6].

So, questioning is difficult, but the actual assessment or observation is the more important perspective of the two [Researcher].

I think so, yes [FG6].

No additional responses were received.

4.2.2.5.4 Upper motor neuron (UMN) and lower motor neuron (LMN) features

The researcher read the following summary from the literature review to highlight two additional points:

1. Neck pain/stiffness was not always present. Similarly, upper/lower limb pain was not always present. Therefore, patient may present with another type of upper limb dysfunction, which physiotherapists should consider i.e. not exclude cervical myelopathy prematurely [Researcher].

2. Upper limb symptoms usually occurred slightly pre-gait disturbance (Chiles 1999), however a clinical commentary type paper not included in information pack reported that gait disturbance followed by fine motor disturbance (in Wang et al 2010). Although it is the reverse, it suggests that it may be useful to consider this should both feature present e.g. a sensory deficit alongside gait disturbance, regardless of order of onset may indicate myelopathic changes [Researcher].

Five nodding actions were noted indicating acknowledgement or approval to this suggestion.
Q: What clinical tests should be used for screening and diagnosis of cervical myelopathy?

The researcher read the following summary of Cook (2007) extract:

A range of clinical tests are used to screen for neurological conditions, such as cervical myelopathy (cord compression). However, some neurological clinical tests (e.g. finger escape sign and clonus) have not been investigated for diagnostic accuracy, whilst others (e.g. the Hoffman sign, Lhermitte sign, and plantar response) have been investigated, but with inconsistent levels of methodological quality that affects their diagnostic accuracy values (summary of Cook et al 2007 p.1235-1236) [Researcher].

The researcher cited Cook et al (2007, 2011) to suggest that from the outset, recognition, is important that clinical tests are limited as to the information they may provide. Therefore, relying solely on a single test is not advisable for example due to low sensitivity rendering such tests as not reliable for ruling in pathologies.

The researcher added:

Q: Would that be a reasonable synopsis based the information that I have given you so far (information pack)? So I think I would be suggesting to insert a footnote (into clinical chart) to physiotherapists that the cervical myelopathy tests are of limited value, therefore consider this in the context of your presentation. Is that reasonable?

Participants indicated that they agreed with this synopsis and supported inclusion of a footnote into a clinical chart to inform

Four “Yes” responses or head nodding actions were noted to indicate a general agreement to this statement.

One additional response was noted:

I think reflexes. Simple upper and lower limb reflexes are one of the most important physical tests. I would agree that some of the others are very operator dependent [FG6].

One additional comment was received that generated further discussion on checking sphincter tone in presentations of suspected cervical myelopathy:
You didn’t mention sphincter tone [FG2].

No. It was not coming up in the literature. Are you talking about physios checking sphincter tone? [Researcher].

It is just that it is done routinely in medical practice. That is all [FG2].

Can I just ask… (Name deleted and inserted FG2) do your consultants do sphincter tone when they are looking for cervical myelopathy? [FG4].

If they are thinking cord problems, then yes, they will do. But obviously more for cauda equina [FG2].

Right, but do they do it routinely [FG4].

They do it routinely for a neurological examination [FG2].

Wow. Ok [FG4].

No further discussion developed on this topic.

4.2.3 Draft clinical chart

A suggestion for a draft clinical chart was produced on screen (Figure 3.3 section 3.6.3.4). This was discussed as to how this may be developed. When asked:

**Q: Do you consider this initial idea as a reasonable way to progress with chart development?**

Participants indicated approval with five comments:

Yes (and /or head nodding action).

FG6 added

It is very clear.

The focus group participants were thanked for their participation and provided with an opportunity to add any additional information being the discussion was closed. No additional information was forthcoming.
4.3 Phase 3: Delphi survey

4.3.1 Pilot Delphi survey

The pilot Delphi survey was conducted as described in Chapter 3. This process was combined with early consultant feedback to inform refinement of the main Delphi survey following circulation of the draft clinical chart.

I would consider using your Delphi to ask people what they think the red flags should be based on a (shortish!) list that you provide (and allowing them to add more). I would also try to keep the language simple and avoid neuro lingo (like dysarthria) that could be misinterpreted [D1].

Construction of the survey focused on three keys areas outlined below (Refer to Appendix Q for full Delphi Round 1 survey):

1. Draft clinical chart - Feedback
2. Developing list of red flags
3. Any other comments?

4.3.2 Delphi survey Round 1 (Qualitative)

4.3.2.1 Chart feedback

Consultants were requested to provide feedback on the draft clinical chart and were informed that the chart aimed to provide a ‘quick reference’ section with a specific list of key red flags. This component would be developed through the Delphi process. The red flag list is accompanied by a detailed background information section in the form of main clinical sub-topics with drop-down menus offering additional information designed to provide context for these features to assist physiotherapists’ with their interpretation and subsequent clinical decision making. Feedback suggested a favourable response to the concept of the design; however, reservations were expressed with regards to its complexity which detracted from its usability and transfer to clinical practice:

Having looked at it again tonight I think the chart is trying to do too many things. In places it is a textbook of diagnosis, in others it is trying to alert people to red flags. For example, there is no need to describe hemicrania continua in a chart
about neck problems. If a patient has that, they will mainly have a headache [D1].

I think it would benefit from massive simplification targeted at the title of your email. What are the truly red flag symptoms associated with neck pain or neck problems? And are you interested in both of these things because they are a different set of red flags [D1].

May be slightly too complex. Likely to work better in the longer term if eventual chart is relatively simple. Otherwise take-up and general acceptance may be limited [D10].

The sub-menus are comprehensive, but may contain too much information to make them easily useable - I might be inclined in a final version, or at least in the applicable version, to concentrate on main features and diagnoses, omitting very rare conditions. It may be of course that your intention is to make it complete. Is the intention to allow non-medics (physiotherapists) to identify red flag signs? If they are to do so and then identify a possible diagnosis, then very rare conditions will confuse. In symptoms and signs of cord compression, I could not see Lhermitte's phenomenon/sign mentioned. This is very typical in some patients with critical cord compression. Maybe I missed it. Central Cord Syndrome is very important in cervical injuries, and it is important to identify this. There is also a phenomenon of high cord injuries that leads to a dissociated sensory/motor effect due to fibre tracts that cross high in the cord and lower brain stem [D12].

I like the quick reference guide which obviously is not yet complete. I am glad that you have relegated cervical degenerative disease as a cause of "dizziness", this is a common misunderstanding in referrals I receive [D2].

I like the concept but find the chart too visually cumbersome and 'overpopulated' to be a simple sorting device [D5].

4.3.2.2 Developing list of red flags

Consultants were requested to list the key red flag symptoms/signs that they consider to exist for neck pain or neck problems alongside which pathology the features may indicate e.g. cord compression. The following categories were themed from the subsequent responses:

1. Progressive pain;
2. Cancer, infection, inflammatory arthritis/spondyloarthopathies, trauma (includes...
risk of instability in this section);
3. Neurological deficit;
4. Headache (may present alongside neck pain/stiffness);
5. Brainstem, cervical arterial, and cranial nerve dysfunctions.

Participant responses from Round 1 were used to develop Round 2 and Round 3 surveys (Refer to Appendix S for web-page version of Round 3 survey). Responses to support these categories (1-5) are provided below:

4.3.2.2.1 Progressive pain
Progressive pain [D1].

New severe unremitting neck pain; Pain not responding to normal analgesics; severe pain on movement, or gross cervical spasm and reluctance to move neck at all (malignancy, instability, infection) [D12].

...recent acute onset neck pain failing to ease with simple measures AND associated with red flag neurological symptoms [D5].

Local pain in neck and arm nerve root pain getting worse over a week, disrupting sleep, worse on movement and when travelling e.g. in car, in the presence of Red flags ....[D6].

4.3.2.2.2 Cancer, infection, inflammatory arthritis/spondyloarthropathies, trauma
Malaise, fever, unexplained weight loss. History of inflammatory arthritis, cancer, tuberculosis, immunosuppression, drug abuse, AIDS, or other infection [D1]

Lymphadenopathy [D1]

Severe neck pain associated with fever, sweats, lethargy, or malaise (infection) [D12].

....in the presence of Red flags are: known cancer; fever; infection; age greater than 40 [D6].

Note if: cancer; age>50 yrs; or fever/infection present [D7].

Neck pain: instability, neoplastic lesion/ trauma [D8].
4.3.2.2.3 Headache

...changes with posture or exertion for headache - should also mention cough and strain induced headache seen with Chiari malformation [D2].

Signs and symptoms of raised intracranial pressure (in capitals) (suggests other site of lesion) Headache (in presence of neck pain/stiffness) with features of: new onset (e.g. <1 month); change in usual pattern; changes with posture or exertion [D7].

Headache (in presence of neck pain/stiffness) with features of: new onset (e.g. <1 month); change in usual pattern; changes with posture or brought on by exertion, cough, laugh or straining. Note caution if: cancer; age>50 yrs; or fever/infection present [D9].

4.3.2.2.4 Neurological

Weakness of arms or legs...gait disturbance, clumsy or weak hands, loss of sexual, bladder, or bowel function. Lhermitte's sign Upper motor neuron signs in the lower limbs Lower motor neuron signs in the upper limbs [D1].

Most crucial are those aspects of the history that suggest spinal cord compromise. So Lhermitte’s phenomenon, foot drop, loss of leg strength or bladder/bowel disturbance [D10].

Neurological deficit a] Radicular: Numbness, pain, paraesthesia, weakness, hyporeflexia in a particular dermatome b] Cord: Lhermitte's; Loss of manual dexterity, weakness in hands; diffuse numbness, paraesthesia - bilateral, non-dermatomal; hyperreflexia (Hoffman's, Finger flexion/extension jerks, clonus, stiff gait, myoclonus); up-going plantar response; loss of proprioception [D12].

All new progressive unusual neurological symptoms/signs, irrespective of neck pain, need investigation. Bladder/Bowel/Erectile dysfunction is more common in lumbar disorders (cauda equina) and comparatively rare in neck disorders except after severe injury or in the presence of profound motor/sensory deficit in the limbs. Bowel and bladder dysfunction are the last features to develop in cord compression at a very late stage and should not be emphasised too much [D12].

Cord compression - early on patients often complain of stiffness in the legs (spasticity) Bladder and bowel disturbance is relatively late in cervical cord compression [D2].

I believe testing for hyperreflexia is more relevant than hyporeflexia (especially in patients who present with symptoms reminiscent of carpal tunnel syndrome) [D4].

Cord compression symptoms/signs (with or without neck pain/stiffness) [D6].
If there is hand or arm weakness or numbness or clumsiness or gait disturbance I would consider that late, but clearly important. Progressive features: bilateral hand and/or feet pins/needles or numbness; upper or lower limbs spasticity or weakness; reflexes exaggerated or reduced; bladder/bowel disturbance (incontinence or retention); erectile dysfunction (rarely) [D6].

Signs and symptoms of myelopathy (in capitals) Neck pain/stiffness/headache with cord compression symptoms/signs : a - limbs - hand weakness or clumsiness, gait disturbance, paraesthesia, spasticity, increased reflexes, b - sphincter disturbance (incontinence or retention); erectile dysfunction [D7].

...gait disturbance: cervical myelopathy upper or lower limbs spasticity or weakness; cervical myelopathy reflexes exaggerated cervical myelopathy bladder/bowel disturbance (incontinence or retention)cervical myelopathy [D8].

Cord compression symptoms/signs (with or without neck pain/stiffness): early features: hand weakness or clumsiness; gait disturbance progressive features: bilateral hand and/or feet pins/needles or numbness (need not be symmetrical ); upper or lower limbs spasticity or weakness; reflexes exaggerated in legs and reduced in arms (or exaggerated in arms too); wasting of hand muscles, bladder/bowel disturbance (incontinence or retention); erectile dysfunction (rarely) [D9].

4.3.2.2.5 Brainstem-vascular-cranial nerve

Horners sign [D1]

Secondly, those features that may indicate a vascular pathology such as arterial dissection in the neck (dizziness, cranial nerve symptoms etc.) but I'm not sure that the latter is in the remit of this study [D10].

In my opinion, if you include dizziness, slurred speech, double vision, horner's syndrome, and other cranial nerve features, every patient with almost any neurological symptom or condition, even with common neck pain, will be referred for urgent assessment, and this is not appropriate [D12].

Signs and symptoms of brainstem dysfunction(in capitals) (Suggests other site of origin) slurred speech; double or loss of vision; drooping eyelid; pulsatile tinnitus or sudden loss of hearing; facial numbness or weakness; taste disturbance; nausea/vomiting; nystagmus [D7].

Neck pain/stiffness/headache with brainstem symptoms/signs - may be subtle and typically within 1 month onset: dizziness (typically episodic and between >1min and <30mins duration); slurred speech; double or loss of vision; drooping eyelid; pulsatile tinnitus or sudden loss of hearing; unilateral limb clumsiness or reduced balance; facial numbness or weakness; taste disturbance; nausea ... nausea alone would be too non-specific I think... I'd say vomiting; nystagmus [D9].
4.3.2.6 Other comments


As I mentioned I think it’s a short list. For example dizziness is only a red flag in neck pain if the patient also has other brainstem symptoms/signs (eg double vision, slurred speech or unilateral limb clumsiness). It turns out that “brainstem symptoms and signs” are a red flag generally, and actually you can forget about dizziness on its own being a red flag [D1].

I think it is important to differentiate red flags that focus on C Spine and those which divert the search for pathology elsewhere [D7].

I’m partly putting this effort in to replying as I agree that knowledge of neck problems and how these present in neurology is often not correct among physios. For example, many still do “VBI tests” even though there are few neurologists in the UK who believe that you can produce vertebral basilar ischaemia from turning your neck. Tingling down the arm is often interpreted as root irritation when usually it’s referred muscle pain or carpal tunnel syndrome (which doesn’t behave as its supposed to in the books) etc. [D1].

4.3.2.3 Intra and Inter-coder reliability

As outlined in section 3.5.4.6 three sets of Delphi first round responses were assessed for intra and inter-coder reliability of coding performed by the researcher (R) and one research supervisor (S). Table 4.2 displays the percentage agreement and Kappa coefficients for inter-coder and intra-coder reliability suggesting excellent agreement for both. A score of ‘0’ indicates no agreement, whilst a score of ‘1’ indicates full agreement between coders. A score above 0.75 indicates excellent agreement (NVivo 9; www.qsrinternational.com). However, due to the limited volume of text within the responses the extent of coding was limited to 2-3 sentences only for each of the three participants selected for coding. Therefore, this significantly reduced potential for error meaning there was greater potential for agreement.
Table 4.2: Intra and inter-coder reliability analysis for Delphi 1st round

<table>
<thead>
<tr>
<th>Coder</th>
<th>% Agreement</th>
<th>Kappa coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>R (repeated)</td>
<td>100</td>
<td>1</td>
</tr>
</tbody>
</table>

Intra-coder reliability

<table>
<thead>
<tr>
<th>Coders</th>
<th>% Agreement</th>
<th>Kappa coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>R and S</td>
<td>100</td>
<td>1</td>
</tr>
</tbody>
</table>

R - Researcher; S - Supervisor

4.3.3 Delphi survey rounds 2 and 3

4.3.3.1 Clinical indicators and consensus levels

As outlined in section 3.5.4.7 there are no recognised guidelines for setting a consensus level (Powell 2003; Keeney et al 2006). A minimum 75% level was selected a priori for gauging the consensus point. As questions were not compulsory allowances were factored in to accommodate missing responses. Therefore, this was adjusted to require a minimum n=9 (from a total of n=11 participants) to complete a question for this to be considered for consensus calculation. The consensus levels increased to a minimum 80% strongly agree/agree to achieve the consensus point. Tables 4.3 to 4.7 display the clinical indicators achieving this consensus level. These clinical indicators were selected for inclusion in a list of red flags for potential serious pathology presenting as, or alongside, a cervical spine musculoskeletal disorder. The complete round 3 survey responses to questions with 5-point likert scale ratings, plus ‘age’ question, may be viewed in Appendix T. Refer to Appendix U for round 3 descriptive statistics. Raw data for rounds 2 and 3 may be viewed in Appendices V and W, respectively.

Furthermore, the Delphi rounds 2 and 3 responses were not identifiable to consultant coding allocated for round 1 analysis, therefore any additional responses obtained during these rounds are coded as ‘NIC’ (No Identifying Code) to denote this position.
### Table 4.3: Progressive pain (Neck and/or radicular pain. May be accompanied by headache) indicators achieving red flag consensus level

<table>
<thead>
<tr>
<th>1. Progressive pain:</th>
<th>Total responses n=11</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.b. May be associated with others features (e.g. history of cancer, trauma, presence of hard neurological signs, suspected atlanto-axial instability or infection).</td>
<td>Strongly agree / Agree 90.9% 10</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6. Severe pain on movement; reluctance to move; gross cervical spasm or torticollis (cancer, infection, atlanto-axial instability).</th>
<th>Total responses n=10</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Strongly agree / Agree 80.0% 8</td>
</tr>
</tbody>
</table>

**Additional comments on pain:**
In terms of pain, the most excruciating pain is most frequently encountered with cervical radiculopathy. With the exception of pain at rest and occipital neuralgia, I don't generally associate it with sinister pathologies [NIC].

### Table 4.4: Cancer, infection, inflammatory arthritis/spondyloarthropathies, trauma indicators achieving red flag consensus level

<table>
<thead>
<tr>
<th>9. Cancer Previous history of cancer; unexplained weight loss; lymphadenopathy.</th>
<th>Total responses n=11</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Strongly agree / Agree 90.9% 10</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>11. Infection Malaise, fever, sweats, lethargy.</th>
<th>Total responses n=11</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Strongly agree / Agree 81.8% 9</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>12. Tuberculosis, immunosuppression, drug abuse, HIV/AIDS, or other (significant) infection.</th>
<th>Total responses n=11</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Strongly agree / Agree 90.9% 10</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>14. Trauma (recent onset)</th>
<th>Total responses n=11</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Strongly agree / Agree 100% 11</td>
</tr>
</tbody>
</table>
### Table 4.5: Neurological deficit (e.g. spinal cord compromise) indicators achieving red flag consensus level

<table>
<thead>
<tr>
<th></th>
<th>Neurological deficit (e.g. spinal cord compromise) indicators achieving red flag consensus level</th>
<th>Total responses n=11</th>
</tr>
</thead>
<tbody>
<tr>
<td>16.</td>
<td>Quick guide Upper motor neuron symptoms/signs (in lower limbs more than upper limbs). Lower motor neuron symptoms/signs (in upper limbs more than lower limbs).</td>
<td>Strongly agree / Agree</td>
</tr>
<tr>
<td>17.</td>
<td>Hands: clumsy/loss of dexterity or weakness.</td>
<td>Strongly agree / Agree</td>
</tr>
<tr>
<td>20.</td>
<td>Loss of proprioception.</td>
<td>Strongly agree / Agree</td>
</tr>
<tr>
<td>21.</td>
<td>Lhermitte's phenomenon / sign.</td>
<td>Strongly agree / Agree</td>
</tr>
<tr>
<td>22.</td>
<td>Hyperreflexia: (Increased/exaggerated reflexes in lower limbs more than upper limbs; Hoffman's reflex; finger flexion-extension jerks; clonus; myoclonus/spasticity in lower limbs &gt; upper limbs; upgoing plantar response).</td>
<td>Strongly agree / Agree</td>
</tr>
<tr>
<td>23.</td>
<td>Gait disturbance e.g. stiff, slow, broad based.</td>
<td>Strongly agree / Agree</td>
</tr>
<tr>
<td>25.</td>
<td>Very late stage</td>
<td>Strongly agree / Agree</td>
</tr>
<tr>
<td>25.a.</td>
<td>Sphincter disturbance (bladder and/or bowel) disturbance (retention or incontinence).</td>
<td>Strongly agree / Agree</td>
</tr>
<tr>
<td>26.</td>
<td>General progressive neurological deficit Any new progressive and/or unusual neurological symptoms/signs (irrespective of neck pain/stiffness).</td>
<td>Strongly agree / Agree</td>
</tr>
</tbody>
</table>

An additional comment was received in the neurological section:

Physical signs too poorly done in general to be useful in assessment by most people. Some neurological progressive symptoms may already have reliably
indicated the true diagnosis and so excluded spinal pathology - it depends on the quality of the history [NIC].

Table 4.6: Headache (accompanying neck pain/stiffness) indicators achieving red flag consensus level

<table>
<thead>
<tr>
<th></th>
<th>Total responses n=9</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>30.</strong> Headache changes:</td>
<td></td>
</tr>
<tr>
<td><strong>30.b.</strong> With posture or brought on by exertion, cough, laugh or straining.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>100%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Total responses n=10</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>31.</strong> Sudden (unexplained) onset.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>80.0%</td>
</tr>
</tbody>
</table>

Headache demonstrated the most notable change in consensus levels between Rounds 2 and 3 (refer to 4.3.3.2). Additional comments on headache included:

Headache, unless new onset, severe an unremitting, might indicate a problem that merits referral/investigation but I would not regard as red flag sign in relation to cervical pathology [NIC].

New persistent headache with evolving signs is most predictive of underlying pathology. Chronic severe headache is mostly primary. Need an option for sudden headache neck pain to cover SAH and arterial dissection (vertebral) [NIC].

There are many other pathological and non-pathological reasons for headache to be a symptom, irrespective of spinal pathology. Do not ‘overload’ it in this context [NIC].

Distinguish/separate posture and other exacerbators [NIC].

Headache suggesting more serious pathology is usually acute, severe, unresponsive to normal analgesics, constant and unusual to the patient. Progressively increasing headache over weeks is also suggestive. Chiari headache is specifically cough related. Raised ICP usually has a postural component - worse lying down and wakes the patient during the night/first thing in the morning [NIC].

The main type of headache that should be Ix is occipital neuralgia, signifying C1/2 instability or FM compaction [NIC].
Table 4.7: Brain stem, cervical arterial (occlusion/stenosis/dissection) and cranial nerve dysfunctions indicators achieving red flag consensus level

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Total responses n=10</th>
</tr>
</thead>
<tbody>
<tr>
<td>33.a.ii. Slurred speech.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>90.0%  9</td>
</tr>
<tr>
<td>33.a.iii. Double or loss of vision.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>90.0%  9</td>
</tr>
<tr>
<td>33.a.iv. Drooping eyelid / Horner's sign.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>88.9%  8</td>
</tr>
<tr>
<td>33.a.v. Pulsatile tinnitus or sudden loss of hearing.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>80.0%  8</td>
</tr>
<tr>
<td>33.a.vi. Unilateral limb clumsiness or reduced balance.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>80.0%  8</td>
</tr>
<tr>
<td>33.a.vii. Facial numbness or weakness.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>100%  9</td>
</tr>
<tr>
<td>33.a.viii. Nystagmus.</td>
<td></td>
</tr>
<tr>
<td>Strongly agree / Agree</td>
<td>90.0%  9</td>
</tr>
</tbody>
</table>

This final section was accompanied by the following comments:

I have highlighted which neurological symptoms are most worrying (Loss of vision, slurred speech, facial weakness, drooping eyelid) not because they necessarily all relate to brain stem dysfunction, but because they may suggest serious pathology outside the neck that justifies urgent referral/investigation. Blurred vision, double vision, vomiting, nystagmus, and tinnitus are not likely to indicate an acute decompensating chiari that requires immediate investigation, but are symptoms that should be brought to the GP’s attention and assessed by him [NIC].
These are mostly worrying signs but not of cervical spine pathology (except rarely) [NIC].

Horner's is the main finding (often ignored) [NIC]. Lacks neurological clarity [NIC].
In relation to the possibility of dissection, Horner’s is invariably present in my experience. What generally concerns me is the presence of abnormal semiology, of upper motor Neuron type [NIC].

One final additional comment of note perhaps summarises the complexity of this topic:

Leave neurology to neurologists [NIC].

Other findings of note were three indicators achieving near consensus level (between 70% to 79%):

1. Weakness (widespread) of arms or legs.
   Additional comment:
   Functional (psychological) problems often present with widespread give way weakness and diffuse paraesthesia. Focal signs are much more predictive of underlying pathology [NIC].

2. Lower motor neuron symptoms/signs (in upper limb more than lower limb). Radicular pattern in particular dermatome: Numbness; paraesthesia (pins/needles); weakness; hyporeflexia (reduced reflexes) in particular dermatome.
   Additional comment:
   You have to differentiate myelopathy (spinal cord compromise) from radiculopathy (nerve root compromise). The former requires urgent assessment; the latter is very common in degenerative cervical disease, and an isolated root problem doesn’t necessarily require urgent assessment. The important thing is appreciating the LOWER limb symptoms (not pain), particularly if bilateral and related to a neck disorder must be due to cord compromise [NIC].

3. Taste disturbance.
Two additional clinical indicators of note that did not achieve either the consensus level or near-consensus level were dizziness and age. Dizziness generated a number of detailed responses within the focus group. However, this indicator achieved a low strongly agree/agree level of 40% (n=10 Mean 3.0 SD 0.94). Similarly, Age (and increased risk of serious pathology occurring.....) received a split response for age > 50 years (n=6; 54.5%) versus no age group exempt/association (n=5; 45.5%). During round 2, one participant stated ‘age >40 years’ as a risk. However, in round 3 this option was not selected. One additional comment accompanied this section:

I have reconsidered the age question and since degenerative disease becomes much more common with age, serious disease can probably occur in any age group and is unlikely to be necessarily linked with increasing age [NIC].

4.3.3.2. Level of agreement

Kendall’s W (Kendall’s coefficient of concordance) ranges from 0 (no agreement) to 1 (complete agreement). This was calculated for each of the five categories (outlined in 4.3.2.2) using IBM SPSS Statistics version 19 and demonstrated an increase in Kendall's W between Rounds 2 and 3 in four out of five categories (Refer to Table 4.8). This indicates an increase in consensus levels between participants over these two rounds. The most notable change was found in the ‘Headache’ category with an increase from 0.13 in Round 2 to 0.63 in Round 3. Caution should be noted in interpreting this calculation due to the small sample size.
Table 4.8: Kendall’s W (Kendall’s coefficient of concordance) and P value for Delphi survey rounds 2-3

<table>
<thead>
<tr>
<th>Categories</th>
<th>Round 2</th>
<th>Round 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P value</td>
<td>K</td>
</tr>
<tr>
<td>Progressive pain</td>
<td>0.13</td>
<td>0.25</td>
</tr>
<tr>
<td>Cancer, infection, inflammatory arthritis/spondyloarthropathies, trauma</td>
<td>0.25</td>
<td>0.25</td>
</tr>
<tr>
<td>Neurological</td>
<td>0.11</td>
<td>0.26</td>
</tr>
<tr>
<td>Headache</td>
<td>0.28</td>
<td>0.13</td>
</tr>
<tr>
<td>Brainstem-vascular-cranial nerve</td>
<td>0.00</td>
<td>0.40</td>
</tr>
</tbody>
</table>

P value ≤ 0.05; K= Kendall’s W (Kendall’s coefficient of concordance)

Appendices X and Y display examples of graphical representation for Kendall’s coefficient of concordance for Progressive pain and Headache, respectively.
Chapter 5. Discussion

5.1 Introduction
The principal motivating factor behind this study design arose through development of a national health board’s regional spinal service (adult population) that questioned the diagnostic utility of red flag indicators traditionally used within physiotherapy practice for the screening of possible serious pathology presenting in musculoskeletal neck pain or neck related complaints. A subsequent literature search identified that red flags for serious pathology in the cervical spine or neck pain did not receive the same level of attention as red flags for serious pathology in back pain. Although some features within the recognised list of red flags for serious pathology in back pain (e.g. CSAG 1994) will relate to the whole spine, such as cancer or infection, there are however, specific clinical considerations to the cervical spine that creates uncertainty for physiotherapy practitioners’ decision making regarding clinical features and their importance relative to the serious nature of the clinical presentation. There is therefore, a requirement to address such uncertainties that ensures vigilance and actions in the process of early detection of serious pathology is both measured and appropriate throughout the assessment and management systems.

CSAG (1994) use the term serious spinal pathology to include spinal tumour, infection, inflammatory disease e.g. ankylosing spondylitis, structural deformity, such as scoliosis, and widespread neurological disorders. However, Negrini (2007) considers that the neck is not the back. In relation to red flag indicators, cervical arterial dysfunction is one such category that would differentiate the neck from the back in terms of possible serious pathology. Negrini’s (2007) editorial on new developments in neck pain estimates that neck pain lags approximately 20-years behind research on low back pain, which includes clinical behavioural differences. Negrini (2007) calls for research by well trained clinicians and rehabilitation professionals in neck pain to address this gap. This study design aims to contribute to this call.

The prognostic variables or diagnostic indicators developed by CSAG (1994) for possible serious spinal pathology have been widely integrated into clinical practice for the management of low back pain. This list is displayed in Table 5.1. These clinical indicators are risk factors identified during the subjective examination (clinical history
taking) and objective examination (physical examination) and are associated with a higher risk of serious disorders causing low back pain compared to patients without these characteristics. Further investigation may be required to exclude serious pathology, such as cancer, inflammatory arthritis or infection, in the presence of such clinical features (Greenhalgh and Selfe 2009).

Table 5.1: Red flag indicators for low back pain (CSAG 1994; Greenhalgh and Selfe 2006, 2010)

<table>
<thead>
<tr>
<th>Red flag indicators for low back pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset less than 20 years or more than 55 years</td>
</tr>
<tr>
<td>Recent history of violent trauma</td>
</tr>
<tr>
<td>Constant progressive, non mechanical pain (no relief with bed rest)</td>
</tr>
<tr>
<td>Thoracic pain</td>
</tr>
<tr>
<td>Past medical history of malignant tumour</td>
</tr>
<tr>
<td>Prolonged use of corticosteroids</td>
</tr>
<tr>
<td>Drug abuse, immunosuppression, HIV</td>
</tr>
<tr>
<td>Systemically unwell</td>
</tr>
<tr>
<td>Unexplained weight loss</td>
</tr>
<tr>
<td>Widespread neurological symptoms (including cauda equina syndrome)</td>
</tr>
<tr>
<td>Structural deformity</td>
</tr>
<tr>
<td>Fever</td>
</tr>
</tbody>
</table>

With reference to specific red flags for the cervical spine, the health information website Patient.co.uk is a resource supplying evidence based information to patients and health professionals and provides a list of red flags for neck pain (http://www.patient.co.uk/doctor/Neck-Examination.htm). This section ‘Neck pain (Cervicalgia) and Torticollis’ references the Clinical Knowledge Summaries (CKS 2009) as its source for this list of red flags (http://cks.nice.org.uk/neck-pain-cervical-radiculopathy#!/topicsummary). CKS is a National Institute for Health and Clinical Excellence supported website that provides information to support clinical practice. This web-based resource, which has not been updated since 2009, provides a number of references with Binder (2007a, b) found to be the most relevant citations provided.
Binder (2007a, b) provides a list of red flags, which is poorly referenced. This work has been adapted for use by Patient.co.uk with the following red flag list:

**Red flags for neck pain**
- A serious underlying cause is more likely in people presenting with
- Red flags suggesting possible malignancy, infection or inflammation
- Red flags suggesting myelopathy (compression of the spinal cord)
- Red flags suggesting severe trauma/skeletal injury
- Red flags suggesting vascular insufficiency

The focus group participants considered the construction of this list that divides into groups of pathologies, could assist physiotherapists’ clinical decision making by enhancing the thought process behind the pathology, as opposed to simply thinking of a clinical indicator in isolation. This is a different format to the method presented in Table 5.1, the traditional red flag list for low back pain as developed by CSAG (1994).

Chapter 4 identified that the list of red flags for low back pain is integrated within specific cervical red flag information contained in the Patient.co.uk list summarised above. However, due to limited supporting evidence, questions remain on the selection of cervical spine clinical indicators and tests as being reliable or of sufficient detail to inform clinical practice. This chapter discusses how these clinical indicators within specific groups of pathologies e.g. vascular insufficiency and myelopathy, relate to the findings from this study alongside the current evidence base. This will include current physiotherapy screening processes, specifically the clinical indicators forming the 5Ds And 3Ns framework, neurological features, age and pain as discussed in chapter 2. This chapter uses Delphi participant comments to support discussion points. As stated in chapter 4, the Delphi rounds 2 and 3 responses were not identifiable to consultant coding allocated for round 1 analysis, therefore these are coded as ‘NIC’ (No Identifying Code) to denote this position.

Figure 5.1 displays the categories and clinical indicators identified from the Delphi method that achieved the consensus point.
Figure 5.1: Cervical spine red flag categories with clinical indicators achieving Delphi consensus point
5.2 Clinical indicators

5.2.1 Age

Greenhalgh and Selfe (2009) reported this established and well recognised red flag was not considered as relevant by an expert panel of seven senior palliative care clinicians participating in a qualitative investigation of red flags for serious spinal pathology. Greenhalgh and Selfe (2009), however, note a weakness with their participant group that routinely cared for patients with a confirmed diagnosis of serious spinal pathology, therefore this may not be representative of the general patient population seen within a primary care setting. In this current study three age options were generated by consultant responses: age greater than 40-years; age greater than 50-years; and, age has no association with the onset of serious pathology occurring naturally. The Delphi 3rd and final round revealed no consensus was achieved with 45.5% (n=5) supporting Greenhalgh and Selfe’s (2009) expert panel indicating that age was not relevant. The remaining 55.5% (n=6) of selected age greater than 50-years, which follows the original CSAG (1994) guidance. Given that cervical arterial pathology, such as spontaneous CAD (sCAD), comprising of the internal carotid artery (ICA) and vertebral artery (VA) can affect all age groups, but mainly affects young and middle-aged adults between the ages of 35-50-years (Schievink 2001; Schievink et al 1994) and the Greenhalgh and Selfe’s (2009) findings this suggests that caution should be used if relying on age as an indicator. It would appear that age is not a clear indicator for the onset of serious pathology within the cervical spine.

5.2.2 Pain

Chapter 2 identified that neck pain and/or headache symptoms are the most frequent local symptoms of CAD (Silbert et al 1995; Savitz and Caplan 2005; Taylor and Kerry 2005; Arnold, Cumurciuc et al 2006; Kerry and Taylor 2006; Chandra et al 2007; Hardmeier, Gobbi et al 2007; Morelli et al 2008; Rigamonti et al 2008; Tobin and Flitman 2008; Thomas et al 2011), and additionally, can be the only presenting symptoms (Biousse et al 1992; Biousse et al 1994; Guillon et al 1998; Arnold, Cumurciuc et al 2006). Furthermore, VAD has also been reported as presenting as a fifth cervical nerve root (C5) radiculopathy (Arnold, Bousser et al 2006; Hardmeier, Haller et al 2007;). However, how does the presentation of pain within this evidence
base compare with CSAG’s (1994) description of constant, progressive, non-mechanical pain, and the clinical indicators identified from this study?

CSAG (1994) identify that constant progressive, non-mechanical pain (no relief with bed rest) is a red flag indicator for serious spinal pathology. Non-mechanical pain is unrelated to physical activity. The Delphi panel also identified progressive pain as a red flag indicator with a number of accompanying descriptors. Rounds 2 and 3 sought to achieve clarity on these descriptors, such as how progressive pain may present or develop.

Two descriptors of progressive pain achieved the consensus level:

a) May be associated with other features e.g. (history of cancer, trauma, presence of objective neurological signs, suspected atlanto axial instability or infection); and

b) Severe pain on movement, reluctance to move, gross cervical spasm or torticollis.

The descriptors in (b) were suggested to indicate the presence of cancer, infection or atlanto-axial instability.

Descriptors that did not achieve the consensus point were progressive pain: in isolation; progressively worse e.g. over 1-week period; not responding to simple analgesia; onset may be a new 1st episode of acute/sub-acute pain (e.g. following trauma or arterial dissection); may be acute/sub-acute on chronic pain; severe and/or unremitting; disrupting sleep. However, severe and/or unremitting pain and pain with onset of new 1st episode (e.g. trauma or cervical arterial dissection) each received 63.7% strongly agree/agree (n=7, mean score 3.5 SD 1.04 and 3.7 SD 1.13, respectively). These scores indicate a trend towards reaching a consensus point. Therefore, the latter two descriptors should be reconsidered within further studies.

One participant offered additional comment indicating that, with the exception of two descriptors, pain in general was not a clinical indicator of serious pathology:
In terms of pain, the most excruciating pain is most frequently encountered with cervical radiculopathy. With the exception of pain at rest and occipital neuralgia, I don't generally associate it with sinister pathologies [NIC].

5.2.3 Headache

5.2.3.1 Clinical indicators of headache achieving the consensus point

Pain associated with occipital neuralgia was suggested to be linked with atlanto-axial instability and foramen magnum compaction.

The main type of headache that should be Ix (investigated) is occipital neuralgia, signifying C1/2 instability or FM (Foramen magnum) compaction [NIC].

Williams (1977) report a case of foramen magnum impaction in a rare case of congenital osseous dysplasia causing basilar invagination from defective bone formation. This change caused occipital headache and progressive neurological deterioration of cerebellar function and lower cranial nerve dysfunction. Occipital or posterior neck pain with diffuse headache is considered as a main feature of vascular pathology of the posterior circulation system (Savitz and Caplan 2005; Rivett et al 2006).

Chiari malformation type 1 is the most frequent of the Chiari malformations (Grazzi and Andrasik 2012). This involves inferior displacement of the cerebellar tonsillas through the foramen magnum. Most cases are congenital in origin with less frequent onset after birth with formations involving excessive drainage of spinal fluid due to infection, injuries or exposure to harmful substances. A rare cause may be chronic sub-dural haematoma (Grazzi and Andrasik 2012). Robertson and Stanley (2008) report a single case of Chiari 1 malformation in a 25-year-old female involved in a road traffic accident (RTA) approximately 4-month previous to examination, but with progressively worse symptoms over the previous 6-weeks.

Two clinical indicators within the headache section achieved the Delphi consensus point:
a) Changes with posture or brought on by exertion, cough, laugh or straining (100%, n=9 mean score 4.2 SD 0.4); and

b) Sudden (unexplained) onset (90% n=9, mean score 4.1 SD 1.0).

Participant comments added:
Chiari headache is specifically cough related. Raised ICP usually has a postural component - worse lying down and wakes the patient during the night/first thing in the morning [NIC].

New persistent headache with evolving signs is most predictive of underlying pathology. Chronic severe headache is mostly primary. Need an option for sudden headache neck pain to cover SAH and arterial dissection (vertebral) [NIC].

This latter comment demonstrates how the headache section was modified for round 3 with the inclusion of ‘sudden (unexplained) onset’ following comments in round 2. This process allowed refinement of the clinical features that resulted in a change in consensus levels demonstrated by an increase in Kendall’s W from 0.13 in Round 2 to 0.63 in Round 3. This indicates an increase in consensus levels between participants over these two rounds.

The Chiari 1 malformation clinical presentation may be precipitated by cough and/or Valsalva manoeuvre; occipital and/or sub-occipital headache; associated with symptoms/signs of brainstem, cerebellar and/or cervical cord dysfunction. Evidence of posterior fossa dysfunction is based on at least 2 of the following: otoneurological symptoms (e.g. dizziness, disequilibrium, nystagmus); transient visual symptoms (e.g. diplopia, visual blurring); and, clinical signs of cervical cord, brainstem or lower cranial nerves or of ataxia or dysmetria) (Robertson and Stanley 2008; ICHD 1998 in Grazzi and Andrasik 2012). Robertson and Stanley (2008) report dizziness. However, dizziness is reported as a possible symptom from whiplash associated disorder. It is unclear when this dizziness onset occurred in relation to the RTA reported by Robertson and Stanley (2008) with the original symptom being neck pain. This article
is a short communication presenting magnetic resonance imaging (MRI) for this case, therefore lacks in-depth detail. Cough headache is one of the most frequent headache forms; however, migraine and tension type is also reported (Grazzi and Andrasik (2012).

5.2.3.2 Clinical indicators of headache not achieving the consensus point

The Scottish Intercollegiate Guideline Network (SIGN) Guideline 107 Diagnosis and management of headache in adults is a national (NHS Scotland) clinical guideline outlined in chapter 2. Guideline 107 states that secondary headache (i.e. headache caused by another condition other than a primary cause) “…should be considered in patients presenting with new onset headache or headache that differs from their usual headache” (p.9).

This guideline contrasts with headache features not achieving consensus points within this study. These were headache of new onset e.g. less than 1 month (20% n=2); severe and persistent (50% n=5); and, headache that changes from usual pattern (20%, n=2). Interestingly, these features, or similar descriptors, were also included in comments e.g.

Headache suggesting more serious pathology is usually acute, severe, unresponsive to normal analgesics, constant and unusual to the patient. Progressively increasing headache over weeks is also suggestive [NIC].

However, these did not achieve the consensus level as seen in ‘sudden’ onset. It should be noted that the comment ‘Progressively increasing…’ was received in the final round, therefore this is not specifically defined within the multiple choice questions.

Additional comments also suggested that clinical reasoning should not be overly inclusive of headache features within cervical pathology. This may explain reasons behind round 2 not achieving the consensus point. This was indicated in both the chart feedback and rounds 2/3 comments:
There are many other pathological and non-pathological reasons for headache to be a symptom, irrespective of spinal pathology. Do not 'overload' it in this context [NIC].

Headache, unless new onset, severe and unremitting, might indicate a problem that merits referral/investigation but I would not regard as red flag sign in relation to cervical pathology [NIC].

During clinical chart feedback:

……there is no need to describe hemicrania continua in a chart about neck problems. If a patient has that, they will mainly have a headache [D1].

These comments alongside several components of the SIGN Guideline 107 not achieving a Delphi consensus agreement may suggest that headache should not be considered in the context of neck pathology, and possibly not included within the list of red flag clinical indicators generated from this study. Similarly, it may indicate that headache of such severity will not present at a non-medical musculoskeletal clinic. This latter interpretation is highly plausible; however, the overall conclusion may be too simplistic to separate headache from cervical spine related pathology as patients presenting with musculoskeletal neck pain may also complain of headache.

As discussed in chapter 2 cervicogenic headaches is considered a disorder that is manageable by the physical therapies (Jull 1997). This disorder has been recognised by the International Headache Society (IHS 2004). Part of the criteria referred to by the IHS is pain referred from a source in the neck and felt in one or more regions of the head and/or face. Differential diagnosis of cervicogenic headache, however, can be difficult to separate from other causes of headache unless additional features are presented. An additional problem with headache differentiation is that neurologists differ in their agreement of cervicogenic headache as having a nosological identity making the concept of cervicogenic headache controversial (Leone et al 1998; Zhou 2008; Bogduk and Govind 2009). This adds greater complexity to clinicians navigating their clinical reasoning processes when a patient presents complaining of neck pain and headache.

It is therefore considered appropriate to include features of headache within this study. However, these should be restricted to key features. The Delphi participant [D1]
comment during the chart feedback phase was utilised to inform development of the ‘headache’ section within the Delphi 2\textsuperscript{nd} round survey. This included; headache ‘accompanying neck pain/stiffness’. This terminology was employed to indicate that a complaint of neck pain/stiffness should be present to help clarify that physiotherapists are not receiving specific complaints of headache that are likely to present at a neurology clinic, except in the case of cervicogenic headache where the source of pain is considered to be from the neck region (IHS 2004).

Chapter 2 highlighted that in relation to neck pathology the most crucial differential diagnosis is headache from CAD due to the heightened risk of adverse events with potentially near fatal consequences following manipulation (Bogduk and Govind 2009). In considering clinical recommendations (e.g. Rivett et al 2006; Kerry et al 2007), Thomas et al (2011) noted that headache was not always present or severe in either VBAD or ICAD subjects. Debette, Gronsbach et al (2011) and Thomas et al (2011), however, both reported that neck pain was more likely in VBAD than ICAD subjects. Neck pain and headache are symptoms that can present at musculoskeletal clinics without prior attendance at a medical practitioner, or could be the reason for referral from the medical practitioner.

Silbert et al (1995) reported 65 of ICAD patients considered their headache as ‘unique’ but 45 did not consider a significance difference to previous experience of headache. This does not support Kerry and Taylor’s (2006) statement as having headache “unlike any other”. Silbert et al (1995) also reported that 132 from 135 ICAD patients had accompanying focal neurological manifestations. Only 3 had headache only, with/without neck or facial pain. Additionally, Silbert et al (1995) report from 135 ICAD patients, 35 (26%) had neck pain at time of dissection with gradual onset in 25 patients and sudden in 7. Neck pain was the first symptom in only 9 patients. Whilst it is possible for cervical arterial pathology to present with pain only, this appears to be in a small number of cases within a relatively rare condition. Although, this condition is relatively rare, the outcomes unfortunately may be catastrophic to both patient and carer. Debette and Leys (2009) advocate caution regarding incidence rates as CAD cases with reduced clinical signs may remain undiagnosed.
With regards to pain and cervical arterial dysfunction a review of the evidence base suggest there is no clear pattern of headache or neck pain and that may occur in either VAD or ICAD and demonstrates inconsistency in literature reporting pain presentations related to CAD (e.g. Biousse et al 1994; Silbert et al 1995; Arnold, Cumurciuc et al 2006).

5.2.3.3 Differential diagnosis of arterial causes cause of pain / headache

The literature review identified that Debette, Grond-Ginsbach et al (2011) and Thomas et al (2011) report neck pain is more likely in VAD, but both reports differed with regards to headache. However, from a physiotherapist’s clinical perspective it is questionable if this information is relevant to musculoskeletal clinical decision making. Clinical experience suggests the concept of differential diagnosis within cervical arterial pathology would be highly challenging for a physiotherapist to correctly select between these two arteries as the source of an underlying pathology, unless additional medical-led advanced practice training was received.

Physiotherapy literature suggests a clinical reasoning approach to identify if the arterial problem is ICAD or VAD in origin, such as using palpation skills for the carotid artery in times of suspicion (e.g. Taylor and Kerry 2010). This aspect was discussed within the physiotherapy focus group that indicated a mixed opinion. The differing opinions considered that differential diagnosis skills may be advantageous if appropriate training was applied versus expressions on reservations regarding concerns for the profession’s credibility with the medical profession on the issue of palpating the carotid artery for diagnostic purposes. In the absence of such training and a need for further debate involving medical and non-medical musculoskeletal practitioners it appears reasonable to suggest that physiotherapists should focus solely on identifying the presence of a serious arterial pathology, rather than introducing additional complexity to the examination through attempts to ascertain which of the cervical arteries are in a dysfunctional state.

It is important for the credibility of this thesis to ensure that conclusions are based on the combination of the focus group discussion and Delphi panel’s input through comments and multiple choice responses alongside the evidence base. In the context
of this section key examples from the evidence base are Silbert et al’s (1995) identification of n=132 from 135 CAD patients had accompanying neurological manifestations and Savitz and Caplan (2005) reporting that vertebrobasilar ischemia typically presents with a collection of symptoms and signs, such as motor or oculomotor signs, and rarely causes only one symptom. Therefore, it could plausibly be argued that a more practicable approach to considering the presentation of pain from an arterial pathology origin is to screen for additional local neurological features, whilst retaining an outside caution with pain only presentations. This would also relegate any recommendations to palpate cervical arteries with the view to identifying a pathological state, unless such processes are advocated by the medical community. It is important that physiotherapists’ progress their assessment systems based on evidence based practice and development to ensure wider clinical credibility is achieved and maintained.

5.2.4 Brain stem, cervical arterial dysfunction (occlusion/stenosis/dissection) and cranial nerve dysfunctions
To facilitate discussion on this section Table 5.2 displays the clinical indicators in two groups; achieving and not achieving the consensus point.
Table 5.2 Brain stem, cervical arterial dysfunction and cranial nerve dysfunctions consensus levels

<table>
<thead>
<tr>
<th>Clinical indicator achieving consensus point</th>
<th>n Agreement</th>
<th>% Agreement</th>
<th>Mean score</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slurred speech</td>
<td>9</td>
<td>90</td>
<td>3.9</td>
<td>0.7</td>
</tr>
<tr>
<td>Double or loss of vision</td>
<td>9</td>
<td>90</td>
<td>4.1</td>
<td>0.8</td>
</tr>
<tr>
<td>Drooping eyelid / Horner’s sign</td>
<td>8</td>
<td>88.9</td>
<td>4.6</td>
<td>0.7</td>
</tr>
<tr>
<td>Pulsatile tinnitus or sudden loss of hearing</td>
<td>8</td>
<td>80</td>
<td>4.0</td>
<td>0.9</td>
</tr>
<tr>
<td>Unilateral limb clumsiness or reduced balance</td>
<td>8</td>
<td>80</td>
<td>3.8</td>
<td>1.0</td>
</tr>
<tr>
<td>Facial numbness or weakness</td>
<td>9</td>
<td>100</td>
<td>4.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>9</td>
<td>90</td>
<td>4.4</td>
<td>0.7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical indicator not achieving consensus point</th>
<th>n Agreement</th>
<th>% Agreement</th>
<th>Mean score</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>New or recent onset of symptoms/signs e.g. 1 month</td>
<td>4</td>
<td>57.2</td>
<td>3.9</td>
<td>0.7</td>
</tr>
<tr>
<td>Dizziness</td>
<td>4</td>
<td>40</td>
<td>3.0</td>
<td>0.9</td>
</tr>
<tr>
<td>Taste disturbance</td>
<td>7</td>
<td>70</td>
<td>3.5</td>
<td>0.9</td>
</tr>
<tr>
<td>Vomiting</td>
<td>6</td>
<td>60</td>
<td>3.5</td>
<td>1.0</td>
</tr>
</tbody>
</table>

5.2.4.1 Clinical indicators achieving consensus point

Slurred speech, double or loss of vision, drooping eyelid/Horner’s sign, pulsatile tinnitus or sudden loss of hearing, unilateral limb clumsiness or reduced balance, facial numbness or weakness, and nystagmus all achieved the consensus point within this study. A recommendation within the qualitative phase of the Delphi survey was to avoid confusion and misinterpretation by not using neurological terminology, such as ‘dysphasia’. This recommendation was adopted within this section, for example Delphi responses included ‘slurred speech’ rather than ‘dysphasia’, ‘double or loss of vision’
rather than ‘diplopia’, ‘reduced balance’ in place of ‘ataxia’, and ‘drooping eyelid’ replacing ‘ptosis’. ‘Nystagmus’ does not appear to have an obvious simplified alternative name. Dysphasia, diplopia, ataxia and nystagmus form part of the 5Ds And 3Ns framework.

These features were identified within the literature review to investigate the signs and symptoms relative to the 5D and 3Ns framework and to ascertain if this framework is a reasonable clinical approach for identifying possible serious neurovascular pathology. For example, Thomas et al’s (2011) retrospective case control study of a younger patient group (<55-years) comparing cervical arterial dissection cases with non-dissection causes of stroke identified the symptoms; visual disturbance, paraesthesia (face, upper and lower limb), and signs; Unsteadiness/ataxia, weakness upper and lower limb; dysphasia/dysarthria/aphasia, facial palsy, ptosis (Horner’s sign). Thomas et al’s (2011) study was designed with consideration to informing clinical practice of non-medical practitioners.

5.2.4.1.1 Nystagmus

Nystagmus forms part of the 3Ns framework and was described to the group as a sign, not a disease with involuntary movement of the eyes (Patten 1998). Bhattacharyya et al (2008) describe directional patterns to aid differential diagnosis of central versus peripheral causes of this sign. Nystagmus achieved the consensus point. This clinical sign generated discussion around the physiotherapy skills level required to sufficiently interpret this clinical feature.

Assessment of nystagmus may be performed by physiotherapists undertaking vestibular assessment and rehabilitation (e.g. Herdman 2007 ch.7 pp.108-124), such as the Dix-Hallpike manoeuvre (a positional test for the vestibular system). As outlined in chapter 2, evaluation of nystagmus, however, requires a high level of interpretative skills. Patten (1998 p.103) states that “…the importance of testing nystagmus correctly and recording the quality, direction and other features is not sufficiently appreciated, which can demonstrate poor clinical technique with potential failure to achieve a true differential diagnosis”.
Therefore, unless specific additional training has been undertaken, physiotherapists should exercise caution if attempting to interpret this sign. Indeed, the focus group participants’ considered that recognising such limitations were important with onward referral for specialist opinion being considered appropriate. This is summarised by the following two participants’ comments when asked if physiotherapists are sufficiently skilled to interpret this.

I think probably not. Ok. I think we can learn to look at the Dix-Hallpike test and recognise normal nystagmus there, but if you read the literature there is just so many different types of nystagmus as to what is going on and I don’t think an ordinary MSK clinician, well not an ordinary one, but a highly skilled one, MSK clinician, can be expected to read nystagmus as they would in an expert neurology clinic. Because it goes upwards, sideways, downwards, backwards and frontwards, you know, I mean we can say if it is there, but we can’t really differentiate as to what it is indicating, I don’t think [FG6].

It probably needs to be interpreted alongside other tests we wouldn’t be performing in any case in our clinics........ [FG7].

Participant FG6’s comments identifying the many different types of nystagmus is exemplified by Herdman (2007 p. 117) in listing the following types of vestibular nystagmus due to central lesions; torsional, downbeat, upbeat, seesaw, periodic alternating nystagmus, and latent. This highlights the challenges presented to non-medical practitioners in assessing nystagmus. It would, therefore, appear reasonable to recommend that a patient presenting to a musculoskeletal physiotherapy clinic with signs of nystagmus in the absence of a previous medical diagnosis should receive an onward referral for specialist opinion. The urgency of this referral may depend on the presence of additional symptoms and signs. The patient may be re-accepted for further physiotherapy-led management once diagnosis confirms no serious central pathology as the underlying cause.

5.2.4.1.2 Drooping eyelid / Horner’s sign, Facial numbness or weakness, and pulsatile tinnitus or sudden loss of hearing

Several clinical features out with the 5Ds And 3Ns framework were included in this section with subsequent consensus agreement; Drooping eyelid / Horner’s sign, Facial numbness or weakness, and pulsatile tinnitus or sudden loss of hearing.
Thomas et al. (2011) additionally reported the signs of ptosis (Horner’s sign/syndrome), facial palsy and upper and lower limb weakness were the most common signs in ICAD (anterior circulation system). This supports Baumgartner et al. (2001) who identified the main significant local signs in ICAD without ischemic development (n=55) was Horner’s syndrome and lower cranial nerve palsies. Ptosis or Horner’s sign/symptom or oculosympathetic palsy was reported in approximately 25-30% of cases (Bassi et al., 2003; Dziewas et al., 2003; Lee et al., 2006).

Pulsatilie tinnitus is almost exclusively related to the sound of non-laminar blood flow transmitted to the inner ear occurring from alteration in hemodynamics e.g. arterial dissection, systemic disease, or local disorders within or in close proximity to the petrous bone (Pelkonen et al., 2004). Pulsatile tinnitus presented as a symptom in 16 patients within a prospective study (n=136; Pelkonen et al., 2004). Similarly, Dziewas et al. (2003) and Arnold, Bousser et al., (2006) also reported tinnitus occurring in a small number of patients with 8 (6%) and 7 (5%), respectively.

Ptosis or Horner’s sign, facial palsy and pulsatile tinnitus or sudden loss of hearing, however, are not included in the 5Ds And 3Ns framework. The exclusion of these features suggests that this framework is not a reliable aide memoire for cervical arterial pathology. The following Delphi participant adds further insight to features within this section:

I have highlighted which neurological symptoms are most worrying (Loss of vision, slurred speech, facial weakness, drooping eyelid) not because they necessarily all relate to brain stem dysfunction, but because they may suggest serious pathology outside the neck that justifies urgent referral/investigation. Blurred vision, double vision, vomiting, nystagmus, and tinnitus are not likely to indicate an acute decompensating chiari that requires immediate investigation, but are symptoms that should be brought to the GP’s attention and assessed by him [NIC].

5.2.4.2 Clinical indicators not achieving consensus point

New or recent onset of symptoms/signs was included to reflect the evidence base indications of a temporal component for the onset of neurological manifestations from
the initial feature presenting in CAD. For example, this has been reported as a mean 8.8 days delay in ICAD patients and mean 12-day delay in VAD patients (Silbert et al 1995) and from several minutes to 1-month (Mas et al 1987; Biousse et al 1995). Such information may assist physiotherapy clinical decision making. However, similar to the ‘headache’ category indicator ‘new onset (e.g. less than 1 month)’, this did not achieve the consensus point. This question received a low response rate of n=7 from 11 participants. On reflection, this may be related to the placement of this question at the beginning of this section that refers to the onset of the symptoms/signs listed below. This question may have been more effective if placed at the end of this section. However, the intention for placement at the beginning was to set the scene for the remaining symptoms/signs occurring with an acute/sub-acute onset.

The remaining questions in this category received n=9 or n=10 responses, the latter being the maximum return for this section. The literature suggests that a temporal component could be useful within a clinical decision making framework. Therefore, given the limitation for potential misinterpretation of this question or not returning to complete this question after viewing the subsequent indicators, it may be premature to dismiss this on the basis of not achieving the consensus point and warrants further investigation within future studies.

Dysphagia or difficulty with swallowing did not feature within the qualitative phase of the Delphi survey. Therefore, this was not included within the multiple choice rounds (2 and 3). Thomas et al (2011) reported that dyshpagia occurred in 8 (17%) cervical arterial dissection cases versus 2 (5%) non-dissection causes of stroke in a retrospective case control study of a younger patient group (<55-years). Taste disturbance (dysgeusia), however, was included within the quantitative phase. Dysgeusia is not within the 5Ds And 3Ns framework. Although taste disturbance did not achieve the consensus point, it did, however, achieve a near consensus level of 70% (n=7, mean score 3.5, SD 0.7). Therefore, further consideration should be given to this component.

Nausea, which forms part of the 3Ns received a Delphi participant comment suggesting this feature was too vague for inclusion. Therefore, vomiting was the recommended indicator for inclusion within the multiple choice, quantitative rounds 2 and 3. Vomiting
did not achieve the consensus point (n=6, 60% mean score 3.5, SD 1.0). Savitz and Caplan’s (2005) review paper is linked to Caplan et al’s (2004) large prospective study (n=407) reported vomiting as a frequent symptom of vertebrobasilar-artery occlusive disease. However, Savitz and Caplan (2005) also report that such presentations typically presents with a collection of symptoms and signs, therefore it is questionable or unlikely that vomiting in this context would present to a musculoskeletal physiotherapy clinic. Savitz and Caplan (2005), however, also state that dizziness and drop attacks are often incorrectly apportioned to posterior-circulation (vertebral artery) ischemia.

5.2.4.2.1 Dizziness
Dizziness forms part of the 5Ds And 3Ns framework as a symptom of arterial pathology (e.g. Thomas et al 2011). Dizziness is an imprecise term used to describe a variety of symptoms’ (Herdman 2007 p.108). The term ‘dizziness’ may also encompass light headedness, a lack of mental clarity or frank vertigo, and is reported to be a frequent symptom of vertebro-basilar-artery occlusive disease (Savitz and Caplan 2005). Savitz and Caplan (2005) consider that vertigo indicates dysfunction of the peripheral vestibular or central vestibulocerebellar system. This is significant as Tarnutzer et al’s (2011) systematic review reports that vertebrobasilar ischemic stroke may closely mimic peripheral vestibular disorders, with obvious focal neurologic signs absent in greater than half of patients presenting with acute vestibular syndrome due to stroke.

Herdman (2007 pp.108-110) describe the components of tempo, symptoms, and the circumstances of the complaint are used to help with the diagnosis of dizziness. ‘Tempo’ refers to whether the complaint is acute (within 3-days or less) or chronic (more than 3-days) or spells of dizziness; ‘symptoms’ refers to the patient’s description of their complaint; whilst ‘circumstance’ relates to how the onset of a patient’s dizziness occurs, such as spontaneous, positional, or movement related onset (Herdman 2007).

Table 5.3 lists disorders with dizziness (adapted from Herdman 2007 p.109). This list highlights the complexity and challenge in differentiating a peripheral from a central cause of dizziness.
Rivett et al (2006) state that dizziness was the most frequent symptom of VBI, however, they also advise to check for presence of neck pain or headache.

Rivett et al (2006 p.3) recommends:

“In every patient presenting with upper quadrant dysfunction, questioning is specifically directed to determine the presence of dizziness which is the most common presenting symptom of VBI. If dizziness is present, other symptoms associated with VBI should be sought...”

Thomas et al’s (2011) retrospective case control study revealed that dizziness presented in only 32% (15) of total dissection cases versus 7% (3) of non-dissection cases. This study is based within a specialty setting, therefore, it is difficult to extrapolate this prevalence to a primary care based musculoskeletal clinic. Bhattacharyya et al’s (2008) clinical guidelines reports that evaluation of patients presenting with vertigo in a non-specialty setting found that vascular causes represented 3% of cases. Missed diagnosis of stroke at first medical contact within emergency departments is often linked to dizziness with 35% of cerebrovascular events in patients with any dizziness and 44% in those with isolated dizziness reported to be have been missed at this stage (Tarnzutzer et al 2011). The authors add that available data suggests that patients with misdiagnosis are at particularly high risk of poor
outcomes. This emphasises the requirement for accurate screening for potentially serious pathology that may present at musculoskeletal physiotherapy clinics.

However, dizziness did not achieve the consensus point within this study (40% n=4, mean score 3.0, SD 0.94). Caution should be noted with this finding as ‘dizziness’ in this survey was presented as ‘Dizziness (central cause typically episodic and between >1min and <30min duration. Not occurring in isolation)’. Therefore, it is unclear if it is ‘dizziness’ or the statement within the parenthesis was the reason for not achieving the consensus point. This statement was adapted from clinical practice guidelines developed for BPPV (Bhattacharyya et al 2008). These guidelines include guidance on diagnosing and differentiating peripheral and potential central neurological causes for dizziness (e.g. migraine-associated vertigo, vertebrobasilar insufficiency, and intracranial tumors), as central causes may have more serious medical implications (Bhattacharyya et al 2008). This presents a significant clinical challenge for differential diagnosis.

Chapter 2 outlined that Rivett et al (2006) further suggest that symptoms of dizziness provoked by cervical spine causes can be differentiated from a vestibular cause by trunk rotation sustained and moving, whilst keeping the head steady.

A Delphi participant made comment regarding the musculoskeletal system as a cause of dizziness within the draft clinical chart:

I am glad that you have relegated cervical degenerative disease as a cause of "dizziness", this is a common misunderstanding in referrals I receive [D2].

From a Physician’s perspective, Post and Dickerson (2010) report that differential diagnosis of dizziness can be narrowed down with clinical tests, such as evaluating for nystagmus, Dix-Hallpike manoeuvre (a positional test for the vestibular system) and orthostatic blood pressure testing, but 20% of cases will remain undiagnosed beyond these tests.

Vertebral-artery disease can cause transient attacks of vertigo; however, this is usually accompanied by other brain-stem or cerebellar symptoms (Savitz and Caplan 2005). This appears significantly different to Tarnutzer et al’s (2011) earlier statement that 44%
in those with isolated dizziness is reported to be related to a missed diagnosis of stroke at first medical contact within emergency departments.

Light-headedness typically indicates presyncope related to circulatory, systemic, or cardiac disease rather than vertebral artery disease (Savitz and Caplan 2005). Savitz and Caplan’s (2005) review following Caplan et al’s (2004) posterior circulation registry (n=407) observed that isolated episodes of vertigo continuing for more than three weeks was almost never caused by vertebrobasilar disease, and that only 7% (of n=407) described light-headedness. No patients presented with light-headedness as an isolated symptom. This type of information may help guide differential diagnosis at first point of contact. These relatively low numbers questions the accuracy of using the symptom of dizziness as a primary indicator of cervical arterial pathology and perhaps the focus should be on a combination of symptoms/signs. Caution, however, should be exercised as demonstrated by Tarnutzer et al (2011) reporting missed diagnosis at first medical contact:

“Best evidence suggest that nearly two-thirds of patients with stroke lack focal neurologic signs that would be readily apparent to a non-neurologist and one-third lack signs that would be readily apparent to a neurologist” (Tarnutzner et al 2011 p.1031).

Alongside identifying accompanying neurological symptoms/signs to the complaint of dizziness, perhaps this physiotherapy focus group participant’s approach in relation to the context or setting of the examination is equally important. This comment was extracted when considering differentiating tests for dizziness:

For me the whole question of dizziness testing is the context. If you are treating a patient then yes you probably do need these skills and you are probably in setting where you have back-up from experts and investigations. When you are in the MSK clinic you presumably treating for neck pain or stiffness predominantly, I don’t think this is appropriate [FG2].

5.2.4.2.2 Drop attacks

This clinical sign forms part of the 5Ds. Binder (2007a,b) report drop attacks, especially when moving the neck, as a red flag for neck pain. Similar to the origin of the 5Ds And 3Ns framework (Coman 1986), Binder (2007 a,b) is un referenced for inclusion of this sign. Drop attacks were not identified as a feature by the consultant expert panel within
this study. The 5Ds And 3Ns framework list was provided to Delphi participants in a 'More Info' window within the survey as an additional opportunity for potential inclusion through the various comments sections. A review of the literature identified that drop attacks have inappropriately been attributed to transient ischemia of the posterior circulation. Not a single patient in the NEMC-PCR had a drop attack as the only symptom (Savitz and Caplan 2005). It would therefore appear that physiotherapists inappropriately check for this sign to screen for potential cervical arterial compromise. Patten (1998 pp.392-393) describe drop attacks as being almost exclusive to females and having no identifiable pathological state. In addition, there does not appear to be any associated dizziness, confusion or impairment of consciousness present.

The following physiotherapy focus group comments highlight the uncertainty surrounding the rationale behind checking for drop attacks:

Well I would ask, what are people asking when they ask about drop attacks. Within the department there is a huge range of what people will actually say to the patient from “have you ever collapsed?” to “have you ever found yourself on the floor unconscious and not knowing why?” You know, it depends on how they establish what people say to establish that their drop attack has occurred. I don’t know if everyone else has had this experience with their staff or not. And certainly I would say there is a huge range or variation in what people are actually saying or asking [FG4].

I sometimes think the understanding of what a drop attack is not always accurate, which is in the type of questions that are asked [FG6].

Therefore it would appear that drop attacks do not have sufficient clinical rationale for inclusion within a red flag list for the cervical spine.

5.2.4.3 Cranial nerve testing
SIGN guideline 107 outlines what clinical evaluation should take place in relation to headaches e.g. neurological testing including cranial nerve testing and fundoscopy. Cranial nerve testing (CNT) is advocated by a small number of physiotherapy-based publications on cervical arterial dysfunction (Taylor and Kerry 2010; Thomas et al 2011). Unfortunately, clinical observation suggests that cranial nerve testing is not
routinely utilised within physiotherapy and fundoscopy is a medical based skill. Therefore, physiotherapists would benefit from additional medical guidance as to what specific features should be considered as being a potential indicator of serious pathology, relative to a musculoskeletal clinic setting. For example, thunderclap type headache is a medical emergency that will not typically present at a musculoskeletal clinic.

The focus group discussed cranial nerve testing within physiotherapy practice. Thomas et al (2011) recommend cranial nerve testing due to ICAD findings in their retrospective study. However, symptoms and signs referred to were also present in VAD. In addition, Taylor and Kerry’s (2010) masterclass type publication directed for non-medical practitioners, also recommend conducting a simple eye exam and CNT.

Two participants reported their respective locations had received training in cranial nerve testing, however, such testing was performed approximately ≤ 3 times per year. This questions knowledge and skills competency levels for such testing, which may impact on practitioner confidence if not performing cranial nerve testing on a regular basis:

In my ESP clinic I have been taught to do cranial nerve testing and I do have a little crib sheet to help do it if I need to, and I think that is quite appropriate in that setting, but I probably wouldn’t expect the rest of the physio dept unless I have gone through that with them to be very adept at doing it, but I don’t feel very confident at doing it myself unless you are doing it all the time, and a lot of time you only need to do it when you need to clinically, so that is very rare actually [FG7].

A counter-argument to continue with cranial nerve testing is to perform this examination with a different focus to seeking diagnosis. Perhaps the aim of performing a basic cranial nerve examination may be to identify the presence of features that are atypical of a neuromusculoskeletal presentation, suggesting the requirement for further medical assessment, as opposed to attempting to diagnose a specific condition. It is vitally important that physiotherapists recognise any knowledge and skills limitations that may
exist within their clinical examination. This is turn may support physiotherapy credibility with the medical profession. One Delphi participant comment is of particular note:

Leave neurology to neurologists [NIC].

5.3 Cancer, infection, inflammatory arthritis/spondyloarthropathies, trauma

5.3.1 Inflammatory arthritis/spondyloarthropathies and upper cervical instability

Table 5.4 displays the agreement levels for the section Cancer, infection, inflammatory arthritis / spondyloarthropathies, and trauma.

<table>
<thead>
<tr>
<th>Clinical Indicator</th>
<th>n Agreement</th>
<th>% Agreement</th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer: Previous history; unexplained weight loss; lymphadenopathy</td>
<td>10</td>
<td>90.9</td>
<td>4.4</td>
<td>0.9</td>
</tr>
<tr>
<td>Infection: Malaise, fever, sweats, lethargy</td>
<td>9</td>
<td>81.8</td>
<td>3.9</td>
<td>1.0</td>
</tr>
<tr>
<td>Tuberculosis, immunosuppression, drug abuse, HIV/AIDS, or other (significant) infection</td>
<td>10</td>
<td>90.9</td>
<td>4.1</td>
<td>0.5</td>
</tr>
<tr>
<td>Inflammatory arthritis/spondyloarthropathies</td>
<td>5</td>
<td>50</td>
<td>3.4</td>
<td>1.0</td>
</tr>
<tr>
<td>Trauma (recent onset)</td>
<td>11</td>
<td>100</td>
<td>4.4</td>
<td>0.5</td>
</tr>
</tbody>
</table>

The most notable finding is inflammatory arthritis / spondyloarthropathies not achieving the consensus point (n=5, 50%) indicating that this clinical presentation was not considered a red flag for serious pathology within the cervical spine.
This is a significant finding as CSAG (1994) consider that serious spinal pathology includes inflammatory disease, such as ankylosing spondylitis. Atlantoaxial dislocation or subluxation is a common and significant development of rheumatoid arthritis (RA) or ankylosing spondylitis patients with involvement of the cervical spine (Uitvlught and Indenbaum 1988). Down’s syndrome is also considered as a frequent cause of atlantoaxial instability (Swinkels et al 1996; Cattrysse et al 1997). Yurube et al (2011) conducted a 5-year prospective cohort study of cervical spine instabilities as a complication of RA in 21 facilities (United States of America) and reported that from n=267 (42.1% follow-up rate from initial n=634), 52.4% had no instability measured at the beginning of the study. This decreased to 29.6% by the end of this study indicating that over 20% of study participants that were followed up had experienced an increase in measured cervical spine instability. N=11 follow-ups had received surgical intervention due to progression to myelopathy.

Chen et al (2011) report a single case study of an 18-year old man who sustained a vertebral artery dissection caused by arterial compression from atlantoaxial dislocation. This case presented with a history of sudden severe headache, neck pain, unconsciousness, and irritating cough with no obvious inducing factors. Niere and Torney (2004) report that bony impingement or compression of neural or vascular structures may occur from major instability of the cervical spine following disruption to the passive restraining system. However, Chen et al (2011) report knowledge of only 10 previous cases of arterial dissection following atlantoaxial dislocation, suggesting vascular compromise following atlantoaxial disruption is a rare occurrence. Similarly, Dickman et al (1995) report 37 from 39 patients sustaining trauma to the transverse atlantal ligament were neurologically intact. One patient died with the remaining patient presenting with mild quadripareisis.

Cook et al (2005) conducted a Delphi study of physical therapists to consider identifiers suggestive of cervical spine instability and concluded that this is difficult to diagnose. Symptoms reaching highest consensus were: intolerance to prolonged static postures; fatigue and inability to hold head up; better with external support, including hands or collar; frequent need for manipulation; feeling of instability, shaking or lack of control;
frequent episodes of acute attacks; and sharp pain. Occipital neuralgia was reported as a symptom of atlantoaxial instability within this study’s Delphi study of consultant neurosurgeons and neurologists:

The main type of headache that should be Ix is occipital neuralgia, signifying C1/2 instability…. [NIC].

5.3.2 Cancer, Infection and trauma
Cancer, Infection and trauma of recent onset all achieved the consensus point. These indicators are included within the CSAG (1994) list.

5.3.2.1 Infection
Recent infection is considered as a risk factor that could be a trigger for sCAD (Guillon et al 2003; Debette, Grond-Ginsbach et al 2011). Both studies provide definitions of infection, which are very useful as a reference to the range of infections that may present, particularly as some may be over-looked e.g. sinusitis. The key feature from these studies is the acute/subacute nature of infection alongside a clinical presentation suggesting potential cervical arterial compromise. Debette, Grond-Ginsbach et al (2011), however, consider their description to be broadly defined and occurring within the past week. Guillon et al’s (2003) case control study investigates infection occurring within the previous 4-weeks as risk factor of sCAD (n=47) compared to a control group (n=52) with cerebral ischemic event unrelated to SCAD reported that infection was present in 31.9% sCAD and 13.5% control subjects (crude odds ratio 3.0 95% CI 1.1-8.2 p=0.032).

Debette, Grond-Ginsbach et al (2011) consider “the presence of at least one typical symptom of infection in combination with fever (≥38 °C) or the presence of at least one typical symptom of infection with corresponding serologic, culture or radiologic findings indicating an acute infection or the combination of at least 2 typical corresponding symptoms, infection occurring in the previous week of dissection onset” (p.1175). Guillon et al (2003) define infection as “symptoms within 4 weeks preceding vascular event and was diagnosed when with a positive history of fever (≥38 °C), a subfebrile
state (37.5 to 37.9°C), or chills, accompanied by 1 or more of the following features…” (p.e79), displayed in Table 5.5.

Table 5.5: Clinical features contributing to the diagnosis of infection (Adapted from Guillon et al 2003 p.e79)

- otalgia (otitis)
- cough with purulent sputum (upper respiratory tract infection such as tonsillitis, pharyngitis, laryngitis, sinusitis, bronchitis, or pneumonia if chest roentgenogram showed parenchymal consolidation)
- headache
- myalgia, (flu syndrome)
- nausea, vomiting and/or diarrhea (gastroenteritis),
- urinary frequency, dysuria and/or positive urine culture (lower urinary tract infection)
- back pain with pyuria, bacteruria, or positive urine culture (pyelonephritis).

Infection was also considered present if “….physical exam and/or laboratory studies performed on admission revealed sepsis, pneumonia, bacterial endocarditis, renal or urinary tract infection, skin or soft tissue infection, gingivitis or dental abscess, septic arthritis, or osteomyelitis” (Guillon et al 2003 p.e79).

Osteomyelitis is an inflammatory condition typically affecting a single bone and caused by an infecting organism (McNally and Nagarajah 2010). McNally and Nagarajah (2010) consider that intravenous drug abuse and immuno-compromised patients from HIV have presented new challenges and that bone infections from surgical interventions, injury, peripheral vascular disease, diabetes are increasing. Vertebral osteomyelitis caused by Myobacterium tuberculosis mainly affects the thoracic spine (McNally and Nagarajah 2010; Cheung and Luk 2011;). Early stages of this disease may be slow progressive constitutional symptoms, such as, generalized weakness, malaise, night sweats, fever and weight loss. Radiculopathy or spinal cord compromise may occur. In addition, spinal complications may occur years after the infection e.g. due to compensation from kyphotic deformity causing hyper-extension of adjacent levels resulting in early spinal degeneration, spinal stenosis and neurological deficits (Cheung and Luk 2011). These adjacent levels could be cervical spine degeneration with complications such as cervical myelopathy.
5.3.2.2 Trauma (Recent onset)

Trauma achieved 100% consensus (n=11) and is included within CSAG’s (1994) guidance. This indicator is potentially open to large variation in interpretation as to what constitutes trauma. Bandiera et al (2003) conducted a large prospective multicentre cohort study (10 Canadian urban academic emergency departments; 18 month, n=6265 mean age 36.6 yrs range 16 to 97, male 50.1%) report use of the Canadian C-Spine rule performs better than unstructured physician judgment at detecting clinically important injuries with sensitivity 100% v’s 92.2% and sensitivity 44% v’s 53.9%. The Canadian C-Spine rule is reported as being previously validated in a USA based study. This rule is for alert (Glasgow Coma Scale score=15) and stable trauma patients when cervical spine injury is a concern. The Canadian C-Spine rule is outlined below (Bandiera et al 2003):

1. “Any high risk factor that mandates radiography?”
   
a. Age ≥ 65 yrs, or, Dangerous mechanism, or, paraesthesias in extremities. If YES – radiography
   
i. (Dangerous mechanism: Fall from height ≥ 3ft/5 stairs; Axial load to head, e.g. diving; High speed vehicle accident (>100km/h), rollover, ejection; Motorised recreational vehicles; Bicycle crash).

2. Any low risk factor that allows safe assessment of range of motion?
   
a. Simple rear end vehicle accident, or, sitting position in department, or, ambulatory at any time, or, delayed onset (i.e. not immediate) neck pain, or, absence of midline cervical spine tenderness. If NO – Radiography.
   
i. (Simple rear end vehicle accident excludes: Pushed into oncoming traffic; Hit by large bus/truck; Rollover; Hit by high speed vehicle).

3. Able to actively rotate neck?
   
a. 45 degrees bilateral. If UNABLE: Radiography” (Bandiera et al 2003 p.397).
This rule does not mention identifying risk factors for fracture e.g. osteoporosis (e.g. Poole et al 2009).

5.4 Neurological deficit (e.g. cervical spondylotic myelopathy / spinal cord compromise)

5.4.1 Neurological indicators for spinal cord compromise achieving consensus point

Five items within the neurological section achieved 100% agreement (n=11). These clinical indicators are listed in Table 5.6. In addition, gait disturbance had full participant response with all except one reporting strongly agree/agree response (n=10, 91%, mean score 4.2, SD 0.9).

Table 5.6: Neurological clinical indicators achieving 100% consensus

<table>
<thead>
<tr>
<th>Clinical Indicator</th>
<th>n Agreement</th>
<th>% Agreement</th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quick guide: UMN symptoms/signs in LL&gt;UL; LMN symptoms/signs in UL&gt;LL</td>
<td>11</td>
<td>100</td>
<td>4.6</td>
<td>0.5</td>
</tr>
<tr>
<td>Hands: clumsy/loss of dexterity or weakness</td>
<td>11</td>
<td>100</td>
<td>4.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Lhermitte's phenomenon/sign</td>
<td>11</td>
<td>100</td>
<td>4.7</td>
<td>0.5</td>
</tr>
<tr>
<td>Hyperreflexia</td>
<td>11</td>
<td>100</td>
<td>4.6</td>
<td>0.5</td>
</tr>
<tr>
<td>General progressive neurological deficit</td>
<td>11</td>
<td>100</td>
<td>4.5</td>
<td>0.7</td>
</tr>
</tbody>
</table>

Two other clinical indicators achieved the consensus point; Loss of proprioception (n=9, 81.8%, mean score 4.0, SD 0.9) and Sphincter disturbance (bladder and/or bowel retention or incontinence: n=8, 80%, mean score 4.1, SD 1.2).
5.4.1.1 Sphincter (bladder/bowel) and sexual dysfunction

Bladder and bowel dysfunction generated Delphi participant comments indicating that this problem was a late presentation in cervical cord compression. This is supported by Wang et al (2010).

Bladder and bowel disturbance is relatively late in cervical cord compression [D2].

Bowel and bladder dysfunction are the last features to develop in cord compression at a very late stage and should not be emphasised too much [D12].

These comments informed development of the Delphi survey rounds 2 and 3 with ‘very late stage’ included within the descriptor. This example highlights the benefits of receiving consultant level input to this project. The inclusion/exclusion and refinement of the clinical descriptors enables physiotherapists to provide more informed clinical judgment to enhance patient care. Whilst this project does not specifically address the various causes of sphincter dysfunction a physiotherapy focus group participant’s comment highlights a common problem faced by non-medical practitioners:

I think just general education about all the different causes of bladder dysfunction is absolutely essential because people get themselves wound up into so many problems about bladder dysfunction and erectile dysfunction problems, and I am sure that patients are sent of left, right and centre unnecessarily because the clinician hasn’t really understood the answer or have re-phrased the question wrongly. So we definitely need clarification as to how they should be addressed I think [FG6].

This supports the Researcher’s personal experience and clinical observations suggesting that specific detail of these features may be poorly understood by physiotherapists, due to generic terms being used to describe this dysfunction as outlined below and in chapter 2, unless advanced level or specialist training has been undertaken. Unfortunately, there is no empirical data to support this observation. However, there are examples within the evidence base that may contribute to this statement.
Bladder and/or bowel dysfunction and erectile/sexual dysfunction are signs that may indicate potential spinal cord compromise e.g. compression through cervical myelopathy (e.g. Bednarik et al 1999; Vyas et al 2004) or malignancy (Greenhalgh and Selfe 2003; 2009). Similar to the 5Ds discussed earlier, in presentations that may include bladder dysfunction the clinician’s level of questioning may be limited to enquire solely on bladder dysfunction or disturbance without any clarification beyond this.

Studies, in the main, reporting on cervical myelopathy tend to use generic terms such as, bladder or sphincter dysfunction when reporting such changes (e.g. Bednarik et al 1999; Vyas et al 2004). These terms lack specific detail to suitably inform clinicians as part of their decision making processes. This lack of specific detail may result in delayed diagnosis (Cook et al 2007; Meyer et al 2008). Therefore, if physiotherapists become consistently more aware of the range of features that may appear within sphincter dysfunction then this may help earlier decision making.

The detail established in this study with bladder/bowel dysfunction being a late feature will contribute to this decision making process. This information may then be enhanced through use of the evidence base as explored in chapter 2 for inclusion in the clinical chart to provide more detailed clinical information that underpins the main red flag list. For example, although the evidence has been critiqued for using generic terms, such as bladder/bowel dysfunction, there was however, one particular exception to this poor reporting on sphincter dysfunction. Sakakibara et al (1995) describes the micturitional sub-groups of irritative and obstructive features. This study could inform clinical practice by highlighting more specific detail. No specific studies on bowel dysfunction were identified during the search.

5.4.1.2 Lhermitte’s phenomenon/sign and upper motor neuron (spasticity) signs

Lhermitte’s phenomenon/sign was listed by several consultants. This clinical indicator is described as:

“Consists of tingling in all 4 limbs or electric shock-like feelings down the back on flexing the neck if cervical cord is damaged by multiple sclerosis, cervical spondylosis or any other condition that distorts or inflames the cervical spinal cord. Reverse Lhermitte’s: hyperextension of the neck causes similar signs as cord may be squeezed between spondylitic bar and buckled ligaments” (Patten 1998 p.259).
However, Wang et al (2010) state that Lhermitte’s sign is not specific to cervical spondylotic myelopathy, but rather is related to posterior column abnormalities. Patten (1998) identifies multiple sclerosis as one such pathological state. This would therefore indicate that physiotherapists should remain vigilant to the presence of other neurological pathologies out with the neurological compromise suspected from a degenerative spine condition.

5.4.1.3 Quick guide
The ‘quick guide’ indicator (UMN symptoms/signs in LL>UL; LMN symptoms/signs in UL>LL) was developed following consultant comments e.g:

….Upper motor neuron signs in the lower limbs Lower motor neuron signs in the upper limbs [D1].

This is supported by Edwards et al (2003) who suggest that the physical examination may frequently reveal a mixed picture or central cord and peripheral nerve (radiculopathy) compromise. Upper motor neuron findings (spasticity or ‘myelopathic signs’) may present in both upper and lower limbs, whereas lower motor neuron dysfunction will occur at the level of the cord or root compression (Edwards et al 2003; Wang et al 2010). The degenerative spine condition may also present with concomitant myelopathic and radiculopathy features e.g. spinal stenosis or single nerve root compromise with origin in the lumbar spine alongside a cervical spondylotic myelopathy presentation (Edwards et al 2003; Wang et al 2010). The potential for mixed clinical presentations highlights the caution required by physiotherapists in correct interpretation of clinical findings that ensures more serious complications are detected. This red flag study will contribute to enhanced awareness of such features.

Kim et al’s (2010) gait analysis study also checked for presence of: increased tendon reflex, ankle clonus, babinski sign (upgoing plantar response), paraesthesia, sensory changes, bowel/bladder symptoms. Regression tree analysis observed upper motor neuron signs, such as ankle clonus and Babinski sign were important in classification of groups with increased signal intensity (ISI) on magnetic resonance imaging (MRI).
These signs, in addition to other pathologic reflexes, such as Hoffman sign, and inverted radial reflex are pyramidal or long tract signs consistent with cord compression (Edwards et al 2003).

Hyperreflexia was developed within the Delphi rounds 2 and 3 following participant responses, for example:

hyperreflexia (Hoffman's, Finger flexion/extension jerks, clonus, stiff gait, myoclonus); up-going plantar response; loss of proprioception [D12].

upper or lower limbs spasticity or weakness; reflexes exaggerated in legs and reduced in arms (or exaggerated in arms too) [D9].

Chapter 2 highlighted a number of studies of varying quality reporting signs of spasticity, for example; Chiles et al (1999; n=76) spastic gait (52: 68.4%), ankle clonus (25: 32.9%), Babinski reflex (31: 40.8%), hyperreflexia (58: 76.3%); and, Heffez et al (2004; n=270) hyperreflexia (64%), inverted radial reflex (57%), Hoffman sign (26%), Clonus (25%), weakness in ≥1 limb (22%).

5.4.1.4 Progressive neurological deficit and hands dysfunction (clumsy/loss of dexterity or weakness)

Weakness in the hands may also be described as clumsy or useless (Bednarik et al 1999; Taylor et al 1991).

All new progressive unusual neurological symptoms/signs, irrespective of neck pain, need investigation [D12].

If there is hand or arm weakness or numbness or clumsiness or gait disturbance I would consider that late, but clearly important. Progressive features: bilateral hand and/or feet pins/needles or numbness; upper or lower limbs spasticity or weakness; reflexes exaggerated or reduced; bladder/bowel disturbance (incontinence or retention); erectile dysfunction (rarely) [D6].

This comment [D6] highlighting the late onset of hand and gait dysfunction, albeit remaining important, differs from Wang et al (2010) and Chiles (1999). In addition, another Delphi participant’s response also considered the reverse to occur:
Chiles et al (1999) reported motor deficit in 152 muscle groups from n=76 patients. Motor deficit in the upper limbs typically occurred in the hands and triceps first. Chiles et al (1999) observed that upper limb symptoms usually occurred slightly before the onset of gait difficulties. Wang et al (2010) report that subtle gait disturbances or problems maintaining balance may occur in the early stage of myelopathy. Wang et al (2010 p.182) cite Gorter et al’s (1976) large study in which >1000 cases of cervical spondylotic myelopathy were reviewed with the loss of fine motor control accompanied by numbness was identified as following subtle gait disturbances, the most frequent presentation. It is hypothesised that motor deficit occurs from anterior horn cell loss, rather than nerve root compression, resulting from spondylotic obstruction of spinal cord venous drainage (Chiles et al 1999). Sensory deficit is a common finding in cervical myelopathy (e.g. Bednarik et al 1999; Chiles et al 1999). Chiles et al (1999; n=76) reported upper limb sensory complaints (sensory loss, dysesthesias, paraesthesias) occurred in 63 (82.9%) patients, and lower limb sensory complaints 34 (44.7%).

When considering ‘progressive neurological deficit’ the literature review identified a large variation in duration of symptoms that suggest a largely chronic and progressive presentation; however, caution should be reserved for acute or rapid deterioration of clinical features. Examples of these variations in mean duration or time-range of features are: Vyas et al (2004) ranged from 1 to 36-months pre-surgery; Lee et al (2011) 4.3-months ± 4.2 months; Kuhtz-Buschbeck et al (1999) 10-months ± 3 months; Heffez et al (2004) 8-years (SD=6.3 years); Taylor et al (1991) Symptoms duration 1 to 18-months (mean 9).

5.4.1.5 Gait

Chiles et al (1999) observed that upper limb symptoms usually occurred slightly before the onset of gait difficulties, whilst Wang et al (2010) report subtle gait disturbances as the earlier feature. However, gait disturbances were consistently reported by the Delphi panel with all except one participant indicating strongly agree/agree to inclusion as a red flag neurological sign.
The physiotherapy focus group participants’ expressed the visual assessment of gait was more important compared with questions. Most studies tend to use generic terms when reporting gait disturbances (e.g. Bednarik et al 1999; Chiles et al 1999; Kim et al 2007). Therefore, the literature review explored studies to help identify specific features of gait dysfunction that may better inform such examinations.

A number of studies have investigated spatiotemporal or linear parameters and kinematic parameters for specific gait disturbances within this cervical myelopathy (Kuhtz-Buschbeck et al 1999; Kim et al 2010; Lee et al 2011). Three dimensional gait analysis, which provides a quantitative measurement was included in all three studies. Consistent findings (disturbances) were reported between these studies:

Disturbances of linear parameters include:
- Slow gait.
- Decreased step/stride length
- Increased step width and double support.
- Decreased single limb support.

Disturbances in kinematic parameters include:
- Decreased maximal knee flexion (swing phase).
- Increased ankle dorsi-flexion (swing phase).
- Decreased plantar-flexion at push-off.

These kinematic features are indicative of spasticity (Kim et al 2010).

The three gait analysis studies described above are useful studies to help inform clinical practice to identify specific aspects of gait, rather than simply considering generic difficulties with this component. More defined detail would enable clinicians to guide subjective questioning for any changes reported by patients and to objectively observe with greater knowledge.
5.4.2 Neurological indicators for spinal cord compromise not achieving consensus point

The remaining neurological symptoms/signs achieved relatively high levels of agreement without meeting the consensus point. This suggests that these features should not be included in a short-list of red flag indicators. However, awareness of these features should remain within broader clinical decision making. Table 5.7 displays these indicators:

Table 5.7: Neurological indicator not achieving the consensus point

<table>
<thead>
<tr>
<th>Clinical Indicator</th>
<th>n</th>
<th>% Agreement</th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weakness (widespread) of arms or legs</td>
<td>7</td>
<td>70</td>
<td>3.8</td>
<td>1.1</td>
</tr>
<tr>
<td>Diffuse numbness or paraesthesia</td>
<td>7</td>
<td>63.7</td>
<td>3.5</td>
<td>1.1</td>
</tr>
<tr>
<td>LMN symptoms/signs</td>
<td>7</td>
<td>70</td>
<td>3.6</td>
<td>1.2</td>
</tr>
<tr>
<td>Very late stage: Erectile dysfunction (rare occurrence)</td>
<td>6</td>
<td>60</td>
<td>3.4</td>
<td>1.3</td>
</tr>
</tbody>
</table>

5.4.2.1 Weakness (widespread) of arms or legs and diffuse numbness or paraesthesia

These features may occur in functional problems (e.g. Stone 2006, 2009). However, caution should be exercised in the interpretation of such findings problems. This clinical indicator generated an additional comment within the Delphi study:

Functional (psychological) problems often present with widespread give way weakness and diffuse paraesthesia. Focal signs are much more predictive of underlying pathology [NIC].

5.4.2.2 Lower motor neuron (LMN) symptoms/signs

LMN features also generated comments. It would appear that the Delphi panel generally agrees on the components of LMN symptoms/signs, without achieving the
consensus point for inclusion in the red flag list. Additional comments suggest why this aspect is not considered as a red flag indicator:

You have to differentiate myelopathy (spinal cord compromise) from radiculopathy (nerve root compromise). The former requires urgent assessment; the latter is very common in degenerative cervical disease, and an isolated root problem doesn't necessarily require urgent assessment. The important thing is appreciating the LOWER limb symptoms (not pain), particularly if bilateral and related to a neck disorder must be due to cord compromise [NIC].

I believe testing for hyperreflexia is more relevant than hyporeflexia (especially in patients who present with symptoms reminiscent of carpal tunnel syndrome) [NIC].

LMN dysfunction (e.g. fasciculations and hyporeflexia or reduced reflexes) are indicative of a peripheral nerve compromise, for example radiculopathy (Edwards et al 2003). Numbness or paraesthesiae in the upper limbs from cervical spondylotic myelopathy origin is typically non-specific, whereas a single dermatomal distribution may be a radiculopathy presentation (Wang et al 2010), which as stated by a Delphi participant does not necessarily require an urgent assessment. In relation to back pain, less than 5% is attributable to true nerve root pain with only a small proportion requiring surgical intervention (Waddell 2004 p.11). CSAG (1994) advise that nerve root pain usually arises from a single nerve root caused by disc prolapse or degenerative changes e.g. spinal stenosis, or scar tissue. Similarly, pain typically follows a dermatomal distribution radiating distally towards the foot or toes and is worse in this distal distribution compared with any concomitant back pain. This presentation does not typically require an urgent referral for further investigation, unless the presentation deteriorates or does not respond to conservative management (CSAG 1994).

5.4.2.3 Erectile dysfunction

A Delphi participant [D12] commented that bladder/bowel/erectile dysfunction is comparatively rare in neck disorders, except following severe injury. Therefore, these features should not be emphasised too much. This comment is supported by two studies included in chapter 2 (He et al 2006; Chiles et al 1999). He et al (2006) examined improvement of sexual function in male patients (Average age 56.3 years;
range 43-72) treated surgically for cervical spondylotic myelopathy through prospective follow-up. Twenty-two subjects (plus 2 unable to attend follow-up) were identified from 753 patients diagnosed with cervical myelopathy and underlying cause was identified as being more likely psychogenic origin compared to reflexogenic. This study highlights the low frequency of sexual dysfunction, which further supports Chiles et al (1999) in commenting that sexual dysfunction was relatively infrequent and usually in far-advanced myelopathy after they identified the incidence of sexual dysfunction (5:6.6% men only and some inactive).

Therefore, this low rate combined with the Delphi comment indicates that routine questioning is not required. However, broader awareness of this outside caution for use in advanced level questioning may be appropriate should more information be needed.

**5.5 Implications for clinical practice**

**5.5.1 Adverse events (AE) associated with physical therapies**

The literature review highlighted that identifying adverse events (AE) associated with treatment of neck pain in adults has been problematic due to low quality data in clinical trials and lack of agreement on standardised AE terminology (Carlesso et al 2010). Ernst’s (2007) systematic review, however, concluded that spinal manipulation was frequently associated with mild-moderate AE and can result in more serious events such as vertebral artery dissection. This is a contentious issue as demonstrated by a recent ‘Head to Head’ discussion article “Should we abandon cervical spine manipulation for mechanical neck pain?” (BMJ 2012;344doi: http://dx.doi.org/10.1136/bmj.e3679).

Sweeney and Doody’s (2010) postal-survey of Manipulative Physiotherapists (n=127), based in Ireland, to determine the use of cervical spine manual treatment and to describe adverse events associated with these interventions reported the most serious adverse events were associated with more gentle non-manipulation techniques. These included one TIA, one fainting, and one drop attack. There was moderate use of vertebro-basilar insufficiency (VBI) functional screening tests as outlined by Rivett et al (2006). However, of the 26% (n=33) of respondents that experienced an adverse event,
24% (n=8) had conducted VBI testing, whilst 58% (n=19) did not conduct testing. This questions the validity of functional screening tests (section 2.6) as outlined by Rivett et al (2006).

5.5.2 Functional screening and clinical tests
A number of blood flow studies investigating the effects of provocative testing on the arterial flow have produced inconsistent results (Kerry and Taylor 2008). Several studies have reported reduced blood flow in functional testing positions (Rivett et al 1999; Mitchell et al 2003, 2004; Arnold et al 2004). In contrast, several studies have reported no change in flow (Thiel et al 1994; Zaina et al 2003; Bowler et al 2011). These examples provide an indication of the inconsistency in blood flow studies and neurological clinical tests. Therefore physiotherapists also need to enhance awareness of neurological and neurovascular pathology through other methods, such as identifying risk factors.

It is hypothesised that a mechanically induced stress on the VA causing altered blood flow with decreased perfusion to the brainstem will initiate VBI signs and symptoms (Westaway et al 2003). Thiel and Rix (2005) question the continued use of functional pre-manipulation testing of the cervical spine as it could cause added arterial compromise to an underlying vascular pathology resulting in an AE. As stated earlier, screening tests should not cause further harm (Cook et al 2007).

Provocative functional testing, as outlined by Rivett et al (2006), consists of end-of-range cervical/neck rotation held for a minimum 10 seconds, simultaneously examining the eyes for nystagmus and checking for any additional symptoms. Rivett et al (2006) further suggest that symptoms of dizziness provoked by cervical spine causes can be differentiated from a vestibular cause by trunk rotation sustained and moving, whilst keeping the head steady. This current red flag study has highlighted the complexity associated with interpretation of nystagmus (Patten 1998 p.103) and the associated limitations expressed by an experienced physiotherapy focus group. Similarly, Delphi participants’ expressed their concern with such provocative testing and interpretation of dizziness as symptom:
I’m partly putting this effort in to replying as I agree that knowledge of neck problems and how these present in neurology is often not correct among physios. For example, many still do “VBI tests” even though there are few neurologists in the UK who believe that you can produce vertebrobasilar ischaemia from turning your neck…… [D1].

I am glad that you have relegated cervical degenerative disease as a cause of "dizziness", this is a common misunderstanding in referrals I receive [D2].

Furthermore, a range of clinical tests are used to screen for neurological conditions, such as cervical myelopathy (cord compression). However, some neurological clinical tests (e.g. finger escape sign and clonus) have not been investigated for diagnostic accuracy, whilst others (e.g. the Hoffman sign, Lhermitte’s sign, and plantar response) have been investigated, but with inconsistent levels of methodological quality that affects their diagnostic accuracy values (Cook et al 2007, 2011). These tests have been identified for use by the Delphi medical panel and supported by a number of studies (e.g. Chiles et al 1999; Heffez et al 2004). The following is an example of a Delphi response:

hyperreflexia (Hoffman’s, Finger flexion/extension jerks, clonus, stiff gait, myoclonus); up-going plantar response; loss of proprioception [D12].

It is therefore important that physiotherapists recognise the limitations of such tests. There is, however, a knowledge shift in this area with Taylor and Kerry (2010) and Cook et al (2011) being physiotherapy-based publications. Taylor and Kerry (2010) in particular, highlight the inconsistency in the evidence behind functional screening tests for VBI with such tests having poor diagnostic utility. Taylor and Kerry (2010) and Cook et al (2011) are excellent examples of isolated work occurring within specific pathologies, however the collection of red flag screening for the cervical spine is limited. Similarly, the ‘5Ds And 3Ns’ framework continues to be used, albeit Taylor and Kerry (2010 p.86) state that “unreasoned adherence to these cardinal ‘classic’ signs and symptoms can, however, be misleading and result in an incomplete understanding of patient presentations”. Kerry et al (2007) state there is no support for Coman’s 5 Ds. Kerry and Taylor (2006, 2008), Kerry et al (2007) and Taylor and Kerry (2010) are the
only identified authors to question the 5Ds and 3Ns framework through a literature review approach.

These physiotherapy ‘masterclass’ type articles provide excellent advancement in non-medical practitioner knowledge, however, these publications are limited by; not being observational based studies and do not state what specific features lack sufficient evidence for inclusion in a red flag list, with the exception of Taylor and Kerry (2010) stating that dizziness does not always occur. Therefore, the 5Ds And 3Ns framework continues to be used. This creates a knowledge gap in understanding the rationale behind these clinical indicators. One method of addressing these areas is to engage with medical experts in ascertaining the importance of such features, alongside the evidence base that would provide further navigation towards a fuller understanding and improved screening process. For example, to address the continued use of dizziness and drop attacks as clinical indicators for serious cervical vascular pathology.

5.5.3 Clinical indicators

This study has identified poor evidence to support inclusion of drop attacks and no Delphi consensus was achieved for the inclusion of dizziness. This is not to state that such features should be ignored; however, it may suggest that it is how these features are ‘weighted’ in terms of importance or relevance that may be the direction for the future. No studies were identified that specifically examines the 5Ds And 3Ns framework with engagement from both non-medical and medical personnel to seek opinion on these components and their relevance to non-medical practitioner clinical practice.

Whilst red flags for musculoskeletal low back pain have been developed (e.g. CSAG 1994) and widely accepted, a change in provision of a regional musculoskeletal service had given rise to the need to include more specific red flag indicators or diagnostic screening for neck related pain or functional impairment. The literature review identified the limitations of the references to specific red flags for the cervical spine. For example, the health information website, Patient.co.uk, adaptation of Binder’s (2007 a,b) publications with limited references supporting the identified red flags. However, the suggested red flag list provides a very useful point for development. This current study
provides a platform for progression of this work, albeit with several methodological limitations.

Figure 5.2 displays the red flag categories with subsequent clinical indicators that achieved the consensus point within this study. The clinical indicators have been integrated with the CSAG (1994) list of red flags for back pain and incorporated into the draft clinical chart to display how these findings may be presented to the clinical community through future publication. These clinical indicators, with appropriate supporting literature, have potential to be used with immediate effect to inform clinical decision making. However, the draft clinical chart requires further development as part of an extended project to support the findings of this study.
Figure 5.2: Cervical spine red flag clinical indicators integrated with CSAG (1994) red flags for back pain and displayed in draft clinical chart
5.6 Future research suggestions

1. The clinical indicators would benefit from additional investigation to provide more robust evidence base for inclusion in a red flag list. For example, few indicators achieved 100% consensus within this small sample size, whilst others narrowly failed to achieve the consensus point. This could be achieved initially by a focused systematic review for specific components of the categories identified within this study.

2. Extend the study design to target a larger medical participation group and refinement of the survey questions. The focus of any such approach should be medical input to inform physiotherapy clinical decision making.

3. Greenhalgh and Selfe (2006, 2010) introduced a ‘weighting’ system for serious spinal pathology with a focus around the red flags developed by CSAG (1994). This approach could be utilised to progress this study by weighting the importance of the red flag indicators identified for the cervical spine.

5.7 Study limitations

1. The SIGN methodology for conducting a literature review was adapted for use by a single researcher, thus limiting the breadth of critical analysis.

2. The focus group was conducted by a single researcher, which limits the capacity to make extensive observational notes. However, viewing the video recording soon after the focus group enhanced close working with the data. Stewart et al (2007) advises the transcript may not reflect the full character of the discussion. Therefore, this may require supplemented material such as, moderator notes. The video recording helped address this limitation.

3. This study employed one focus group. A specific focus group study will typically use 3-4 groups. However, this study used a single group to inform development of the main Delphi survey, rather than form part of a main focus group approach.

4. The consultant sample size within the Delphi survey was relatively small (n=11). Skulmoski et al (2007) advise that 10-15 participants from a homogenous group is considered acceptable for this type of post-graduate level study. This sample size reflects the challenges experienced in
recruiting expert medical level participants for this type of study. However, having gained initial medical input this may facilitate future recruitment strategies for additional studies associated with this project.

5. The Delphi technique has undergone continual modifications of its concept and design, which makes the process of testing rigour problematic (Hasson and Keeney 2011).

6. Achieving the set Delphi consensus level that enables clinically meaningful production of a screening process that is considered suitable to inform clinical practice is a challenge. This was selected by the single researcher based on a similar study and guidance on conducting Delphi method projects. The consensus levels for meaningful transferability is potentially affected by the small sample size and a number of questions were not completed by the full panel. This appeared to be in areas of uncertainty e.g. the 'headache' and 'brainstem - cervical vascular – cranial nerve dysfunction' sections. However, the headache section participation improved from round 2 to 3 with refinement of the indicators following participant feedback.

7. Delphi methodology: Overall, it is unclear how methodological rigour should be established as each study design, sample and consensus process is different (Hasson and Keeney 2011 p.1700). Trustworthiness is considered as more appropriate than reliability and validity to gauge effectiveness and appropriateness of a Delphi study (Hasson and Keeney 2011). Therefore, the following four main strategies as outlined by Hasson and Keeney (2011 p.1700) were adapted to establish trustworthiness:

   a. Credibility: Ongoing iteration and feedback was provided to Delphi participants.

   b. Dependability: The study included a representative sample of experts.

   c. Confirmability: A detailed description of Delphi collection and analyses process was maintained.

   d. Transferability: Comparing the Delphi study findings alongside the current evidence base helps enhance applicability of survey findings.
Chapter 6. Conclusion

6.1 Study Overview
Musculoskeletal physiotherapists provide assessment and treatment for pain and functional impairments relating to the musculoskeletal system. Examples of such problems are back and neck pain. In order to apply safe and effective treatment to these conditions it is vitally important that any underlying serious complaints have been excluded. Prognostic variables or diagnostic indicators for possible serious spinal pathology, commonly known as ‘red flags’, have been developed by the Clinical Standards Advisory Group (CSAG1994) and widely integrated into clinical practice for the management of low back pain. The intended outcome of this thesis was to develop a red flag screening process or equivalent clinical indicators for the cervical spine. The motivating factors behind this study design arose following development of a musculoskeletal clinical service. This process identified that screening for red flag indicators of possible serious pathology presenting in musculoskeletal neck pain or neck related complaints did not receive the same level of attention as red flags for serious pathology in back pain.

The literature review identifies there is a risk of neurological/neurovascular pathology, such as cervical arterial dissection and cervical myelopathy, presenting as neck and/or head pain or functional impairment at musculoskeletal clinics. Currently, the extent of adverse events related to such presentations is unknown. Although the problem is not considered common, there is limited evidence to support this. If the clinical decision making processes are not sufficient in making a correct early diagnosis or in identifying those patients at risk of developing an adverse event following manual treatment then this has potentially catastrophic consequences for the individual, which could result in complications such as, permanent gait or bladder dysfunctions and stroke, with potential for death from the latter example.

The literature review identifies limitations and inconsistent evidence for current functional provocative testing and clinical tests, risk factors, and cardinal signs and symptoms traditionally used as a screening process by musculoskeletal therapists to differentially diagnose serious neurological and neurovascular pathology from musculoskeletal disorders. Additionally, the review highlights the difficulties in
differentiating neck and head pain from non-musculoskeletal causes. This presents a serious challenge for musculoskeletal physiotherapists and requires a high level of awareness to suspect such pathology (Thanvi et al 2005; Greenhalgh and Selfe 2006; Kerry and Taylor 2006, 2008). Therefore, an improved process aimed at detecting serious neurological and neurovascular pathology of the cervical spine would be of clinical value.

This study contributes to the knowledge base through the mixed method study design aimed to combine physiotherapy and medical expert input to develop a list of red flag clinical indicators applicable to clinical practice that informs physiotherapy clinical decision making. A list of neck related red flag clinical indicators within five specific categories were developed: 1. progressive pain; 2. cancer, infection, trauma; 3. neurological deficit (spinal cord compromise); 4. headache (associated with neck pain/stiffness); 5. brainstem, cervical arterial and cranial nerve dysfunctions. An increase in Kendall’s W was demonstrated between Rounds 2 and 3 in four out of five categories, indicating an increase in consensus levels between participants.

Whilst a short-list of clinical indicators within specific categories has been developed, the focus group discussion and subsequent Delphi study with consultant neurologists and consultant neurosurgeons has highlighted the complexity of the clinical features within musculoskeletal presentations. For example, the presence of headaches in the context of neck complaints and the basic level of description attached to the descriptors in the ‘brainstem-cervical vascular-cranial nerve dysfunction’ section generated input to suggest that physiotherapists should not over-emphasise these aspects, which should be clinically assessed and managed by medical practitioners.

An additional benefit of this study design that engages both physiotherapy and medical consultants is to enhance the future credibility of the former through engagement of the latter. This will provide guidance to ensure that musculoskeletal physiotherapy screening processes develop to appropriate and effective clinical standards. An example is the Delphi participant response, ‘leave neurology to neurologists’. This comment is interpreted as meaning medical level, not specifically neurologists. The benefits of the focus group discussion part support this statement.
through recognition of the limitations in physiotherapy based skills e.g. interpreting signs, such as nystagmus. There will be, however, a number of highly skilled physiotherapists with such sufficient skills. This scenario will be dependent on specialist settings and working closely alongside medical consultants. In relation to mainstream musculoskeletal physiotherapists, these skills are likely to be limited. Therefore, the key aspect of this approach is for physiotherapists to have sufficient knowledge and skills to recognise at an early stage the potentially subtle atypical clinical features suggesting an underlying pathological state masquerading as, or presenting alongside, a cervical spine musculoskeletal presentation.

Whilst this study has several methodological limitations the findings from the focus group discussion and subsequent Delphi survey will contribute to the understanding of the challenges faced by physiotherapists in screening for serious pathology as frontline practitioners. This is vitally important as there is a high likelihood of physiotherapists increasingly becoming the first contact clinician as national services continue to redesign, for example self referral to physiotherapy departments without General Practitioner or medical consultant input. It is therefore critical that musculoskeletal physiotherapy knowledge develops with support from the medical profession to deliver safe, effective and person-centred care in line with national drivers, such as the Scottish Government’s Healthcare Quality Strategy for NHSScotland (2010). This collaborative approach will enhance physiotherapy clinical credibility with the medical profession and facilitate appropriate onward referrals for the right person to the right place at the right time in line with the Healthcare Quality Strategy (2010).

6.2 Progressing the research findings

The study design was developed to have immediate clinical applicability. Whilst there are limitations to this design the findings are considered to be useful to physiotherapists at this time. In addition to informing future project design the present findings will inform current practice and understanding of clinical indicators. The draft clinical chart requires development out with this current thesis. Both physiotherapy and medical consultant feedback suggest the concept of the clinical chart would be a suitable vehicle to convey the findings of this project. However, valuable medical input recommends simplifying this platform to ensure uptake and usability.
Having identified a number of central red flag categories with respective components, the clinical chart is aimed at providing physiotherapists with the supporting knowledge base within one interactive resource. This plan would address the problems of publications, such as Cook et al (2011) on cervical spondylotic myelopathy and Taylor and Kerry et al (2010) on cervical arterial dysfunction remaining separate and not easily accessible, whilst providing a detailed evidence base to support the clinical indicators. The latter point is a limitation of the evidence base supporting the Patient.co.uk website, the health information resource supplying evidence based information to patients and health professionals and the Clinical Knowledge Summaries (2009), a resource supported previously by the National Institute for Health and Clinical Excellence.

Important directions for study development are to continue with the combined medical and physiotherapy involvement, and to refine the clinical features through progression of the evidence base for the respective categories. In addition, introducing a 'weighting' system to help inform clinicians of the importance of the individual clinical indicators or combination of these features would be a valuable feature. For example, dizziness alone did not achieve the Delphi consensus point; however, dizziness in the presence of other neurological findings may alter clinical interpretation. This approach could also address those clinical features that achieved a near consensus point.

The combined physiotherapy and medical expert approach to this study provides a platform to inform the future development of physiotherapy clinical decision making skills for the benefits of patient safety relating to the assessment and management of musculoskeletal neck problems.
References


Negrini, S. 2007. The neck is not the back: obvious, but the research gap should be reduced. *Europa Medicophysico*, ed. 43, pp.75-77.


Appendix A: Clinical questions, search strategy and summary of selected studies

Question 1. What pathologies including their signs and symptoms and risk factors should be considered as red flags when screening for serious pathology in neck related musculoskeletal disorders?

Search strategy
Search terms: Red flags OR serious pathology AND neck pain OR cervical spine.

Results: 172. Title checks reduced to: 4. Abstracts check reduced to 0.

Additional search
Additional search of medical website: patient.co.uk containing revealed evidenced based section with 9 references. One article contained relevant information, but was graded as an expert opinion, which included limited references.

This article together with clinical experience was used to form the following key sub-questions:

Question 2. What risk factors and signs and symptoms may indicate the presence of Cervical Arterial Dysfunction (CAD)?

Search strategy
Search terms: Risk factors AND cervical arterial dissection OR cervical artery atherosclerosis OR carotid artery atherosclerosis OR vertebral artery atherosclerosis OR carotid artery dissection OR vertebral artery dissection.

Search reduction
Results: 1874. Reduced by selection of academic journals192. Title checks reduced to 134. Full article/abstract checks 119 (including hand search): Selected: 36

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>SIGN evid. Level</th>
<th>n</th>
<th>Clinical presentation / Main findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albuquerque et al 2011</td>
<td>case series</td>
<td>3</td>
<td>13</td>
<td><strong>Risk:</strong> presented with craniocervical dissections following chiropractic manipulation. Symptoms of neurological deficit, head or neck pain, or both had atypical onset within hours or days of manipulation. In this case series 31% (n=4) were left permanently disabled or died as a result of their arterial injuries.</td>
</tr>
<tr>
<td>Arnold, Bousser et al 2006</td>
<td>Prospective observational</td>
<td>2+</td>
<td>169</td>
<td><strong>Symptoms/signs:</strong> TIA 17 (10%), and Occipital head and/or neck pain alone 21 (12%) SAH without ischemia 3 (2%) cervical radiculopathy C5/C6 1 (1%). 118 (88%) of 134 patients with ischemic or hemorrhagic symptoms had also occipital head and/or neck pain and 7 (5%) a pulsatile tinnitus.</td>
</tr>
<tr>
<td>Arnold, Cumuric et al 2006</td>
<td>Prospective case series</td>
<td>3</td>
<td>20</td>
<td><strong>Risk:</strong> Reported a higher rate of hypercholesterolemia in a prospective study of 165 VAD patients compared to other studies. The authors considered that although no control groups were used, this feature could not be dismissed as a significant risk factor in VAD. Investigated characteristics of pain as the only symptom of CAD. 20 from 245 consecutive cases diagnosed with sCAD were included. 50% had history of migraine (8 without and 2 with aura).</td>
</tr>
<tr>
<td>Study</td>
<td>Design</td>
<td>Case/Control</td>
<td>N</td>
<td>Risk</td>
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<tr>
<td>Arnold et al 2008</td>
<td>Case control</td>
<td>2-</td>
<td>102</td>
<td>Risk: Cervicocephalic artery dissection after childbirth is considered rare. To determine differences between postpartum (childbirth within 6-weeks previous) and non-postpartum CAD. A total of 102 women (&lt;50 years) from 245 female patients held on single centre CAD register between 1997 – 2005. Arnold et al (2008) concluded that post partum CAD patients and associated conditions should be looked for in women with unusual headache after childbirth. All postpartum CAD patients had neck and or headache as the 1st symptom onset ranging from 7 days to 18 days after delivery.</td>
</tr>
<tr>
<td>Arnold et al 2009</td>
<td>Case control</td>
<td>2+</td>
<td>239</td>
<td>Risk: There was no significant difference in other cardiovascular factors previously considered as a risk for vascular pathology: frequency of hypertension, diabetes, current smoking, past smoking and hypercholesterolemia. sCAD patients tended to be taller and have a lower body weight than the control group.</td>
</tr>
<tr>
<td>Arnold et al 2010</td>
<td>Prospective</td>
<td>2-</td>
<td>186</td>
<td>Risk: compared the characteristics of consecutive patients with sVAD with cerebral ischaemia versus patients with local signs and symptoms only (head/neck pain, cervical spine radiculopathy) and concluded that older patients (mean 43.6 ±9.9 vs 38.6±9) and smokers were more likely to develop cerebral ischaemia.</td>
</tr>
<tr>
<td>Arauz et al 2007</td>
<td>Case control</td>
<td>2-</td>
<td>39</td>
<td>Risk: Mild Hyperhomocysteinemia has been reported as a risk factor for both arterial dissection and ischemic stroke without dissection. Reported that high plasma concentrations of homocysteine and low plasma levels of folate were associated with an increased risk of CAD and concluded that in a Mexican population that deficiencies in nutritional status may contribute to the relatively high incidence of CAD in Mexico.</td>
</tr>
<tr>
<td>Bassi et al 2003</td>
<td>Prospective multicentre study</td>
<td>2-</td>
<td>49</td>
<td>Symptoms/signs: Headache and neck pain occurred in 32 patients (65.3%). Local neurological manifestations were present in 15 patients (30.6%). Ischemic cerebral symptoms were present in 41 patients (83.6%). 36 ICAD (incl. 2 with associated VAD), 24 (56.6%) had headache, 10 (28.5%) had oculosympathetic palsy, and 27 (77.1%) had symptoms of cerebral ischemia. 8 (16.3%) had only local symptoms. Of 13 with VAD (incl. 2 with ICAD), 9 (69.2%) had headache and 12 (92.3%) had symptoms of cerebral ischemia. Asymptomatic VAD* was detected in 4 patients (11.4%) during the evaluation of symptomatic ICAD.</td>
</tr>
<tr>
<td>Baumgartner et al 2001</td>
<td>Prospective</td>
<td>2+</td>
<td>181</td>
<td>Risk: Reported a statistically significant finding (p&lt;0.05) in sCAD patients with ischemic events compared to patients without such developments. and Baumgartner et al 39% from n= had a history of smoking.</td>
</tr>
<tr>
<td>Author et al.</td>
<td>Study Type</td>
<td>N</td>
<td>Control</td>
<td>Risk:</td>
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<tr>
<td>Benninger et al 2009</td>
<td>Case control</td>
<td>2+</td>
<td>346</td>
<td>Mild Hyperhomocysteinemia has been reported as a risk factor for both arterial dissection and ischemic stroke without dissection. However the underlying mechanism behind this risk factor remains unclear and requires additional investigation. Hyperhomocysteinemia in SCAD (N=33,38%) versus healthy controls (n=23,23%; p=0.034).</td>
</tr>
<tr>
<td>Biouss et al 1994</td>
<td>Prospective series</td>
<td>2-</td>
<td>65</td>
<td>Risk: cardiovascular risk factors were reported as no difference between ICAD patients with (n=48) or without pain (n=17).</td>
</tr>
<tr>
<td>Caplan et al. 2004</td>
<td>Prospective</td>
<td>2+</td>
<td>407</td>
<td>Symptoms/signs: All patients TIA or stroke within previous 6 months. Coronary artery disease in 143 (35%). &lt;1% of patients with vertebrobasilar ischemia had only a single presenting symptom or sign. 13 patients had hemodynamically sensitive ischemia, with multiple brief episodes of dizziness, veering, perioral paraesthesias, and diplopia. Only 7% of patients described light-headedness, and none presented with light-headedness as an isolated symptom. No patients had a drop attack as the only symptom.</td>
</tr>
<tr>
<td>Chandra et al 2007</td>
<td>Retrospective</td>
<td>2-</td>
<td>20</td>
<td>Symptoms/signs: Most common symptom on presentation was headache (83% SCD, 78% SVD). A significantly higher incidence of nausea was reported in the SVD group (67% SVD vs. 33% SCD; p&lt;0.01). Vertebrobasilar symptoms (vertigo, dysarthria, loss of consciousness, or diplopia) occurred in a majority of patients in each group (67% SCD, 56% SVD) with a relatively higher rate of hemispheric symptoms occurring in the SCD group (33% SCD vs. 25% SVD). Other associated symptoms such as amaurosis (loss of vision in 1 eye), anisocoria (unequal pupil size), ipsilateral facial droop, partial ipsilateral Horner's syndrome, and neck pain were seen to occur in 1/3 or fewer patients in each group.</td>
</tr>
<tr>
<td>Chaves et al 2002</td>
<td>Case series</td>
<td>3</td>
<td>10</td>
<td>Symptoms/signs: 9 had a stroke (1 had an associated subarachnoid hemorrhage), whereas 1 patient had only TIA. Severe headache (usually retro-orbital, frontal and/or temporal) followed by contralateral hemiparesis was the most common initial clinical symptoms (80%). Neurological signs occurred in most pts (90%) immediately after headache. No patient had vascular risk factors or a history of neck or head trauma.</td>
</tr>
<tr>
<td>Debette and Markus 2009</td>
<td>Systematic review / and a meta-analysis</td>
<td>2++</td>
<td></td>
<td>Risk: on genetic factors for CAD and a meta-analysis of association studies with a polymorphism, concluded that studies on genetic association with CAD have been underpowered. Case reports and genetic association studies on &lt;20 CAD patients, or where studied in a post-hoc subgroup analysis were excluded. Monogenic connective tissue disease is rarely associated with CAD. Ehlers-Danlos syndrome is the main one; however, in the large majority of cases of CAD, there is no evidence of for a known monogenic disease. However, Debette and Markus (2009) report that there are several arguments for</td>
</tr>
</tbody>
</table>
association of "sporadic" CAD with connective tissue abnormalities as part of a multifactorial predisposition. A meta-analysis identified an overall significant association of the MTHFR 677TT genotype and CAD (OR1.67; 95% CI, 1.21 – 2.31).

<table>
<thead>
<tr>
<th>Reference</th>
<th>Study Type</th>
<th>N</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Debette, Grond-Ginsbach et al 2011</td>
<td>Large observational</td>
<td>2++</td>
<td>946</td>
</tr>
<tr>
<td>Symptoms/signs:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. cervical pain</td>
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<tr>
<td>2. Headache</td>
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<tr>
<td>3. Cerebral ischemia</td>
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<tr>
<td>a. Ischemic stroke</td>
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<tr>
<td>b. TIA</td>
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<tr>
<td>c. transient monocular blindness</td>
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<tr>
<td>d. Subarachnoid haemorrhage</td>
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<tr>
<td>ICAD Vs VAD</td>
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<tr>
<td>1. ICAD n=231 (38.7%); VAD n=212 (66%)</td>
<td>0.001; OR(95% CI) 0.27 to 0.48</td>
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<tr>
<td>2. ICAD n=405 (67.8%); VAD n=207 (64.5%)</td>
<td>0.3; OR(95% CI) 1.36 (1.01 to 1.84)</td>
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<tr>
<td>3. ICAD n=453 (73.2%); VAD n=295 (90.2%)</td>
<td>0.0001 OR(95% CI) 0.32 (0.21 to 0.49)</td>
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<tr>
<td>Risk:</td>
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<tr>
<td>The following were found to have experienced minor mechanical trauma defined as not requiring a medical visit attendance at a hospital, within the previous 1 month: ICAD n=177 (29.2%) and VAD n=118 (36.3%) p=0.02 (adjusted for univariate) OR (95% CI) 0.75 (0.56 to 1.02) p=0.05 (adjusted for age / gender). Major trauma defined as requiring a medical visit or attendance at a hospital did occur, but did not achieve significance (p=0.87 adjusted for age/gender).</td>
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<tr>
<td>Migraine present in 221 (36.3%) ICAD and 123 (38.1%) VAD p=0.6 OR (95% CI) = 1.09 (0.81-1.47).</td>
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<tr>
<td>Debette et al (2011 p.1175) defined infection &quot;...occurring in the previous week of dissection onset and corresponding to the presence of at least one typical symptom of infection in combination with fever (≥38 °C) or the presence of at least one typical symptom of infection with corresponding serologic, culture or radiologic findings indicating an acute infection or the combination of at least 2 typical corresponding symptoms...&quot;, ICAD n=131(21.7%), VAD n=47(14.6%) p=0.009 OR (95% CI) 1.59 (1.09-2.31). The authors consider this as possibly an overestimation of infection within the previous week as their definition is broad.</td>
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<table>
<thead>
<tr>
<th>Reference</th>
<th>Study Type</th>
<th>N</th>
<th>OR (95% CI)</th>
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<tbody>
<tr>
<td>Debette, Metso et al 2011</td>
<td>Prospective</td>
<td>2++</td>
<td>690 (CAD) and 556 non-CAD ischemic stroke and 1170 referants</td>
</tr>
<tr>
<td>Study</td>
<td>Study Type</td>
<td>Risk</td>
<td>Symptoms/Signs</td>
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</table>
| Dziewas et al 2003 | Retrospective     | 2-126  | **Symptoms/Signs:** Major presenting complaint. Cerebral infarction (96; 76%), Transient ischemic attack (15;12%); Local signs only (15;12%)
|               |                   |        | **Associated Features** Neck pain (73;58%), headache (57;45%), tinnitus (8;6%), Partial Horner’s syndrome (29;23%).
|               |                   |        | Risk: 40% CAD patients had hypercholesterolemia and a history of smoking. |
| Ernst 2007    | Systematic review | 2++    | Risk: Manipulation of the neck, however, is considered as having a strong association as a risk factor for CAD. |
| Ernst 2010    | Systematic review | 3      | Risk: To establish the numbers of fatalities following chiropractic intervention and reported a total of 26-deaths were published in the medical literature, but further states there is reason to believe that under-reporting is substantial and reliable incidence figures do not exist. |
| Grond-Ginsbach et al 2012 | Prospective | 2-32   | Risk: To show specific factors for familial CAD. Nine new patients added to 23 patients from previous study. Mean age 38.4 yrs +/- 13.9 yrs. Twenty-six female (62.5%). Twelve suffered multiple dissections. Four recurrent dissections after 1 yr. Patients with familial history were younger (p=0.018). Conclusion: high prevalence multiple dissections and of longterm (>1yr) recurrent patients indicates specific predisposition for familial CAD exists. |
| Gui et al 2010 | Prospective       | 2-16   | **Symptoms/Signs:** No history of head / neck trauma, TIA or signs of spinal cord ischaemia. Initial symptom headache in 8 (53%). Most (n = 14, 88%) had neurologic deficits with the onset of symptoms of ischemia apparent within 4 days of onset of headache. 13 presented with symptoms of posterior-circulation ischemia, 1 with SAH and 2 with serious occipital lacerating pain. Serious parietooccipital pain with symptoms of posterior-circulation ischemia were the most common manifestations (n = 10, 63%). |
| Guillon et al 2003 | Case control     | 2-47    | **Risk:** Reported 58.3% CAD and 40% of control subject with cerebral ischemic event unrelated to CAD had baseline characteristics of oral contraceptives. Guillon et al (2003) case control study investigating infection as risk factor of sCAD (n=47) compared to a control group (n=52) with cerebral ischemic event unrelated to SCAD. Reported that infection was present in 31.9% sCAD and 13.5% control subjects (crude odds ratio 3.0 95% CI 1.1-8.2 p=0.032), concluding that recent infection is a risk factor and could be a trigger for sCAD. |
| Haldeman et al 2002 | Retrospective | 2-64   | concluded after reviewing medicolegal (medical related compensation claims) cases that risk factors could not be identified and that dissection was an unpredictable event. |
| Huang 2009    | Retrospective     | 2-73   | **Symptoms/Signs:** N=40 patients presenting with ischemic stroke, 22 (55%) had accompanying headache and/or neck pain lateralized to the dissection side. All patients presenting with SAHemorrhage or combined ischemia and SAH had headache. |
4 patients with headache only, the pain described as intense and lateralized to the dissection side.

Preceding trauma or specific activities before arterial dissection were found in 10 cases (13.7%). Prior history of hypertension was recorded in only 12 patients (16.4%).

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>n</th>
<th>Symptoms/signs:</th>
</tr>
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<tbody>
<tr>
<td>Lee et al 2006</td>
<td>Retrospective</td>
<td>2-48</td>
<td>In CAD n=(%): Asymptomatic 3 (6); Pain 38 (80); Neck pain 13 (27); HA 33 (69); Horner syndrome 12 (25); Cerebral ischemia/stroke or TIA 32 (67); TIA 11 (23); Stroke 27 (56). The average annual incidence rate for CAD in Olmsted County was 2.6 per 100,000 population (95% CI, 1.86 to 3.33). The average annual incidence rate for CAD in the city of Rochester was 3.01 per 100,000 population (95% CI, 1.86 to 3.33).</td>
</tr>
<tr>
<td>Martin et al 2006</td>
<td>observational</td>
<td>2-7</td>
<td>Risk: Genetic risk factors are thought to play a role in the aetiology of scAD; however, familial CAD is rare. dissections to establish if any specific features existed. They concluded that familial CAD families are young. Mean age (n=15, 9 women) at first dissection was 36.2 years (median age 32 years, range 18-59). Skin biopsies were performed on 11 patients and conclude that ultrastructural alterations in the dermal connective tissue might not be an important risk factor.</td>
</tr>
<tr>
<td>Maruyama et al 2012</td>
<td>Case series</td>
<td>3</td>
<td>Symptoms/signs: 6 VAD / 1 ICAD / 1 combined All but 1 patient, headache and neck pain were unilateral. All VAD complained of posterior cervical or occipital pain. 1 ICAD had temporal pain, and 1 patient with co-existing VAD had posterior cervical pain. Acute onset in 5, thunderclap in 1, and 1 gradual and progressive. Pain severe in all cases. 5 continuous pain, 2 intermittent pain. Quality of the pain throbbing in 5 and constrictive in 2. Pain duration ≥1 week in 6 patients. Suspect CAD if intense unilateral posterior cervical and occipital pain or temporal pain.</td>
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<tr>
<td>Miley et al 2008</td>
<td>Review (structured evidence based clinical neurologic practice review)</td>
<td>2++</td>
<td>Risk: Conclude there is weak to moderately strong evidence to support causation between CMT and VAD and associated stroke.</td>
</tr>
<tr>
<td>Paciaroni et al 2006</td>
<td>prospective study</td>
<td>2+</td>
<td>Risk: Examined seasonal variability. Most patients presented with ischemic stroke (241 / 63%), followed by TIA in 40 (11%), retinal ischemia in 7 (2%), an non-ischemic in 73 (19%); 19 (5%) were asymptomatic spontaneous CAD. A higher frequency of CAD was observed in winter (31.3%; 95% CI; 26.5 to 36.4; p=0.021) compared to spring (25.5%; 95% CI; 21.1 to 30.3), and summer (23.5%; 95% CI; 19.3 to 28.3), and autumn (19.7%; 95% CI; 15.7 to 24.1). Although there was seasonal pattern present, the cause in unclear with possible increased contribution from winter peaks of infection, hypertension, and aortic dissection. There was no additional data to support this hypothesis other than observing season variation.</td>
</tr>
<tr>
<td>Pezzini et al 2006</td>
<td>prospective, case-control study</td>
<td>2-153</td>
<td>Risk: Reported a trend towards significant association was observed when the prevalence of hypertension was compared among patients with spontaneous CAD and control group ((26.8% v 17%; OR 1.79; 95% CI, 0.98 to 3.27, p=0.058).</td>
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</tbody>
</table>
### Mild Hyperhomocysteinemia

Mild Hyperhomocysteinemia has been reported as a risk factor for both arterial dissection and ischemic stroke without dissection.

### Raser et al 2011

**Risk:** To investigate for association of length of styloid process and CAD. The styloid process of the temporal bone is variable in length, angulation and proximity to the carotid artery. This study revealed no significant difference in angulation, however, there was a significant difference for styloid process being longer ipsilateral to the dissection than in control subjects 930.3mm vs 26.6mm, p=0.33. Dissection was associated with increasing styloid length with OR 1.08/mm (95% CI 1.02 to 1.17, P=0.04). Comparing the top quartile of length (>31.15mm) with bottom 3 quartiles revealed an OR for dissection of 4.0 (95% CI 1.3 to 14.2, P=0.03). Control subjects 13% (n=5/38) had styloid length associated with risk. Raser et al (2011) concluded that CAD is associated with a longer styloid process suggesting that mechanical injury from the styloid may contribute to the pathogenesis of CAD.

### Rist et al 2011

**Risk:** To evaluate the association between migraine or migraine subtypes (e.g. with aura) and CAD. Five case control studies included that were published through 2010. Pooled analysis, migraine doubled the risk of CAD (pooled odds ratio (OR) = 2.06, 95% CI 1.33-3.19). Migraine with aura showed slightly weaker association compared to without aura; however, no evidence that aura status modifies association between migraine and CAD (met-regression on aura status p=.58).

### Rubenstein et al 2005

**Risk:** Manipulation of the neck is considered as having a strong association as a risk factor for CAD.

### Silbert et al 1995

**Risk:** investigated characteristics of headaches consecutive symptomatic patients with spontaneous CAD (n=135 ICAD, n=26 VAD) reported a history of migraine in 24 (8%) ICAD and 6 (23%) VAD patients. No other statistical analysis presented. Medical records check and follow-up (letter, telephone call or clinical evaluation).

### Thomas et al 2011

**Symptoms/signs:**
- Signs: Unsteadiness/ataxia: 26 (55%):21 (47%), weakness upper limb: 22 (47%):32 (74%) weakness lower limb: 21 (45%):26 (60%); dysphasia/dysarthria/aphasia: 21 (45%):30 (70%) ; Facial palsy: 18 (38%):20 (47%); ptosis: 17 (36%):2 (5%); Nausea/vomiting: 13 (28%):6 (14%); Dysphasia: 8 (17%):2 (5%); Drowsiness: 5 (11%):1 (2%); Confusion: 5 (11%):6 (14%); Loss of consciousness: 8 (17%):2 (5%)

**Risk:** statistically significant association between minor mechanical trauma and CAD patients compared to a control group: 23 (64%) v’s 3(7%) OR (95% CI) 26.67 (6.83 to 104.17) adjusted OR 25.29 (6.04 to 105.82) p<0.000.
cardiovascular risk factors were not considered significant in a CAD group compared with age (<55-years) and sex matched controls of patients with stroke from other causes. Observed in 5 (14%) of CAD patients and 4 (9%) of control subjects (<55 years stroke of non-dissection cause). Infection had borderline statistical significance with CAD.

**Question 3.** What features of dizziness and what clinical tests would aid MSK physiotherapists’ differential diagnosis of a peripheral versus central cause of dizziness?

**Search strategy**  
Dizziness AND Differential AND Diagnosis.

**Search reduction**  
Research Article: 329. Title checks reduced to 30. Full article/Abstract checks reduced to: 12. Selected: 3.

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>SIGN Evid Level</th>
<th>Main findings</th>
</tr>
</thead>
</table>
| Bhattacharyya et al 2008. | Guidelines | 2++ | **Statement 1a. Diagnosis of Posterior Canal BPPV**  
*Strong recommendation*  
Posterior semicircular canal BPPV is diagnosed when:  
1) patients report a history of vertigo provoked by changes in head position relative to gravity, and when  
2) on physical examination, characteristic nystagmus is provoked by the Dix-Hallpike manoeuvre bringing the patient from an upright to supine position with the head turned 45 degrees to one side and neck extended 20 degrees.  

**2a. Differential Diagnosis of BPPV**  
Clinicians should differentiate BPPV from other causes of imbalance, dizziness, and vertigo.  
Other causes of vertigo confused with BPPV can be divided into otological, neurological, and other entities. In a nonspecialty setting evaluation of patients presenting with vertigo, BPPV has been found to account for 42 percent of cases followed by vestibular neuritis (41%), Ménière’s disease (10%), vascular causes (3%), and other causes (3%). The most common diagnoses that require distinction from BPPV because their natural history, treatment, and potential for serious medical sequelae differ significantly.  

**Neurological Disorders**  
Key issue facing clinicians’ differentiation of vertigo between peripheral causes and CNS causes of vertigo.  
Several clinical features may suggest a central cause of vertigo rather than BPPV.  
- Nystagmus findings that more strongly suggest a neurological cause for vertigo, rather than a peripheral cause such as BPPV, include down-beating nystagmus
on the Dix-Hallpike manoeuvre, direction-changing nystagmus occurring without changes in head position (ie, periodic alternating nystagmus), or baseline nystagmus manifesting without provocative manoeuvres.

Central causes of vertigo that should be differentiated from BPPV are migraine-associated vertigo, vertebrobasilar insufficiency, and intracranial tumors.

**Failure to respond to conservative management should raise concern that the underlying diagnosis may not be BPPV.**

<table>
<thead>
<tr>
<th>Author</th>
<th>Type</th>
<th>Level</th>
<th>Summary</th>
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</table>
| Dros et al 2010. | Systematic review | 2++ | 1. Studies on diagnosing dizziness have been conducted in highly selected homogenous groups of patients only.  
2. Evidence to support the diagnostic process in primary care is scarce.  
3. An exception is the head impulse test: +ve test diagnostic of peripheral vestibular dysfunction and –ve test result diagnostic of central peripheral dysfunction.  
Accurate evaluation of diagnostic tests should be based on the results of more than one study. Therefore, the authors describe 4 tests, all targeted for neuro-otologic conditions that were evaluated in more than 1 study.  
a. Dix-Hallpike manoeuvre, b. Head-shaking nystagmus test, c. Head impulse test, and d. Vibration-induced nystagmus test. (weblinks provided by authors) |
| Tarnutzer et al 2011. | Systematic review | 2++ | Vertebrobasilar ischaemic stroke may closely mimic peripheral vestibular disorders, with obvious focal neurologic signs absent in >1/2 of patients presenting with acute vestibular syndrome due to stroke.  
A 3-component bedside oculomotor examination – HINTS (horizontal head impulse test, nystagmus and test of skew) identifies stroke with high sensitivity and specificity in patients with acute vestibular syndrome and rules out stroke more effectively than early diffusion-weighted MRI. |
**Question 4.** What clinical tests, signs and symptoms of cervical myelopathy (CM) have been identified in this presentation that should be used to screen for this pathology?

**Search strategy**

Cervical spondylosis OR Cervical myelopathy AND Signs OR Symptoms OR Examination OR Diagnosis NOT Surgery.

Narrow by subject major headings: Spinal Osteophytosis complications; Spinal Osteophytosis; Spinal Osteophytosis diagnosis; Spinal Cord Diseases; Spinal Cord Diseases diagnosis; Cervical Vertebrae pathology; Spinal Cord Compression diagnosis; Physical Examination; Cervical Vertebrae.

**Search reduction**


<table>
<thead>
<tr>
<th>Study</th>
<th>Study design</th>
<th>Evid. level</th>
<th>Sample size</th>
<th>Symptoms</th>
<th>Signs</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bednarik, J., Kadanka, Z. and Vohanka, S. 1999. Median nerve mononeuropathy in spondylotic cervical myelopathy: double crush syndrome? <em>Journal of Neurology</em>, 246 pp.544-551.</td>
<td>Prospective, case control</td>
<td>2-</td>
<td>60</td>
<td>Gait disturbances (n=56), spasticity and/or weakness of the lower and/or upper extremities (n=55), clumsy hand (n=13), sensory disturbance corresponding to cervical spinal cord involvement (n=37), bladder, bowel or sexual disturbances (n=15), Lhermitte sign (n=9).</td>
<td>Exclusion: other possible causes of clinical signs/symptoms (multiple sclerosis, motor neuron disease, cervical spinal canal and/or posterior fossa expansive lesions), known systemic disease e.g. RA</td>
<td>CM diagnosis confirmed with MRI. Nerve conduction studies, EMG, and median nerve somatosensory evoked potentials.</td>
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<tr>
<td>Chiles, B., Leonard, M., Choudri, H., and Cooper, P. 1999. Cervical spondylotic myelopathy: patterns of neurological deficit and recovery after anterior cervical decompression. <em>Neurosurgery</em>, 44 (4), pp.762-769.</td>
<td>Retrospective (Osteophytic ridge (54.76%) or soft disc herniation 22:29%)</td>
<td>2-</td>
<td>76</td>
<td>N=76 patients Neck pain (21:27.6%), upper extremity pain (18:23.7%), lower extremity pain (7:9.2%), upper extremity sensory complaints (loss, dysesthesias, paraesthesias, pain) (63:82.9%), lower extremity sensory complaints (34:44.7%), hand use deterioration (957:75%), gait dysfunction (61:80.3%), bowel dysfunction Motor deficit (n=152 muscle groups): Upper limb: Deltoid (16:10.5%), Biceps (18:11.8%), Triceps (44:28.9%), hand intrinsics (86:56.6%) Lower limb: Iliopsoas (59:38.8%), quadriceps (40:26.3%),</td>
<td>Authors comments: UE symptoms usually slightly before onset gait difficulties; all except 6, had UE sensory complaints or difficulties with fine movements when 1st seen. UE sensory symptoms generally begin finger tips, then extend proximally with time, but rarely radicular distribution. Motor deficit (UE) usually in hands and triceps first. Hypothesised that this occurs from anterior horn cell loss rather than</td>
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<tr>
<td>Study</td>
<td>Design</td>
<td>Participants</td>
<td>Sexual Dysfunction</td>
<td>Abnormal</td>
<td>Exclusions</td>
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<tr>
<td>He, S., Hussain, N., Zhao, J., Fu, Q. and Hou, T. 2006. Improvement of sexual function in male patients treated surgically for cervical spondylotic myelopathy. Shisheng. Spine, 31(1), pp.33-36.</td>
<td>Prospective follow-up 2+</td>
<td>22 (identified from 753 CM patients)</td>
<td>Sexual dysfunction (subjective difficulty in penile erection or ejaculation). All had normal function 6 months pre-surgery.</td>
<td>Abnormal: reflexogenic erection (4) / psychogenic erection (18)</td>
<td>Exclusions: No report of sexual dysfunction; Patients with other diseases, such as brain damage, thoracic and lumbar spinal disease, peripheral neuropathy, endocrinopathy, or specific depression</td>
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<tr>
<td>Heffez, D., Ross, R., Shade-Zeldow, Y., Kostas, K., Shah, S., Gottschalk, R., Elias, D., Shepard, A., Leurgans, S. and Moore, C. 2004. Clinical evidence for cervical myelopathy due to Chiari malformation and spinal stenosis in a non-randomised group of patients with the diagnosis of fibromyalgia. European Spine Journal, 13 pp.516-523.</td>
<td>Prospective non-randomised 2-</td>
<td>270</td>
<td>Fatigue (96%), Neck/back pain (95%), Cognitive impairment (92%), generalised weakness (92%), headache (90%), gait instability (85%), grip weakness (83%), photophobia (83%), hand clumsiness (80%), paraesthesiae (80%), irritable bowel syndrome (77%), dizziness (71%), numbness (69%), blurred vision or diplopia (65%), disorientation (54%), chronic nausea (40%)</td>
<td>Sensory level (83%), hyperreflexia (64%), recruitment – inverted radial reflex (57%), absent gag reflex (57%), Romberg sign (28%), Hoffman sign (26%), Clonus (25%), impaired tandem walk (23%), weakness in ≥1 limb (22%), impaired position sense (14%), Cranial nerve V (8%), ataxia (8%), nystagmus (6%), Cranial nerve X11 (4%).</td>
<td>Exclusion: no previously diagnosed neurological disease including CM (some had previous MRI identifying degree of stenosis and/or cerebellar tonsillar ectopia, but not considered significant).</td>
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<td>Kim, Y., Oh, S., Yi, H., Kim, Y, Ko, Y. and Oh, S. Myelopathy caused by soft Retrospective (soft disc herniation)</td>
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<td></td>
<td>Walking difficulty (20; 77%), spasticity (15;58%), chest abdominal</td>
<td>Muscle atrophy (7;26%), spinothalamic deficits (7;26%), sphincter</td>
<td>Standard assessment: Multidisciplinary (including neurologist and/ or neurosurgeon, and rehab team (PT/OT/SLT) and psychologist. MRI brain/cervical spine and CT cervical spine.</td>
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<tr>
<td>Study</td>
<td>Method</td>
<td>Sample Size</td>
<td>Description</td>
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<tr>
<td>Kim et al., 2010. Gait analysis for evaluating the relationship between increased signal intensity on T2 – weighted magnetic resonance imaging and gait function in cervical spondylotic myelopathy. Archives of Physical and Medical Rehabilitation, 91 pp.1587-92.</td>
<td>Retrospective - gait analysis laboratory</td>
<td>2+ 36</td>
<td>Examination also included: neck pain, increased tendon reflex, ankle clonus, Babinski sign, paraesthesia, sensory changes, bowel/bladder symptoms.</td>
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<tr>
<td>Kuhtz-Buschbeck et al., 1999. Analysis of gait in cervical myelopathy. Gait and Posture, 9 pp.184-189.</td>
<td>Case control (n=14)</td>
<td>2+ 26</td>
<td>Additional: Radiculopathy in upper limbs (9), Sensory symptoms (paraesthesia) affecting legs 3, sphincter dysfunction 2 (not specified). Upper motor Neuron involvement included a mild increase of leg muscle tone (0.9 ± 0.3 Ashworth scale). Pre-surgery: significantly reduced gait velocity and step length, prolonged double support, increased step width and reduced ankle plantarflexion. Hip and knee no difference to controls. Gait recorded on walkway (13-m) and treadmill.</td>
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<tr>
<td>Lee et al., 2011. The characteristics of gait disturbance and</td>
<td>Prospective case control</td>
<td>2+ 38</td>
<td>Leg muscle tone in all CM patients was increased (1 to 1+ Ashworth scale). Graded levels of compression: garde 0 – no impingement on spinal cord, garde 1 – some</td>
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Three dimensional gait analysis: linear parameters - step width, gait velocity, cadence (step rate per minute), step length, stride length (distance between both feet), stance time (single foot), and double support time (both feet); Kinematic data – pelvis, hip, knee, ankle angles. Exclusions: unable to walk; other diseases that could affect walking e.g. stroke, traumatic brain injury, or myelitis; previous cervical or lumbar surgery.

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>n</th>
<th>Type of Symptoms</th>
<th>Exclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taylor, J., Johnston, R. and Caird, F. 1991. Surgical treatment of cervical spondylotic myelopathy in</td>
<td>Retrospective</td>
<td>17</td>
<td>Paraesthesiae in hands and upper limbs, often asymmetrical distribution (16). Weak hands (11). Four commented clumsy and</td>
<td>Reflex changes in both upper and lower (generally hyperreflexia in lower limbs, with brisk or diminished</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Coexisting medical conditions were common: ischemic heart disease (5), significant RA/OA (5, 4 of which with arthroplasties).</td>
</tr>
</tbody>
</table>

Control group n=36 (no significant difference)

Impingement, but deformity on spinal cord is absent or minimal, grade 2 – evident deformity of spinal cord with an obviously reduced cross-sectional area, and, grade 3 – grade 2 and high signal intensity area within the spinal cord. Grade 2 or 3 at minimum 1 level considered as positive MRI sign indicative of CM.

Patients also divided into 2 groups related to posterior tibial somatosensory evoked potentials. Abnormal PTSEP results in 20 patients of CM group (52.6%) length, stride length (distance between both feet), step length (distance between contact point same foot), stance time (single foot), and double support time (both feet); Kinematic data – hip, knee, ankle jtROM.

Six trails of 10-m walkway. Posterior tibialsomato-sensory evoked potentials

Exclusions: previous CM surgery, other neurological disorders, coexisting peripheral neuropathy, or orthopaedic lower limb joints that could alter gait.
elderly patients.  
*Age and Ageing, 20*  
pp.407-412.

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Study Design</th>
<th>No. of Patients</th>
<th>Symptoms</th>
</tr>
</thead>
</table>

|  |  |  | Not specified |
|  |  |  | All 14 had pyramidal signs; 13 sensory involvement; 5 posterior column involvement; 3 distal hand muscle wasting; 3 sphincteric dysfunction. No additional specific detail presented. |

The authors considered presenting signs and symptoms (not co-morbidities) were similar to those in younger patients.

useless), deterioration in gait (11), neck pain (5), prone to falls (4), sphincter disturbance (1). upper limb depending on level of lesion), weakness of hands and upper limbs (14), Reduced power lower limb, associated with spasticity (13), impairment of pin prick sensation in hands and upper limb (12), lower limb (10), severe proprioceptive loss in legs (1). Additional: only 4 patients mobile without any aid, upper limb function independence (2).

**Reviewer comments:** Well conducted systematic review following Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. All studies assessed using Quality Assessment of Diagnostic Accuracy Studies (QUADAS) tool. No RCT’s within studies and authors acknowledge that not many studies met their inclusion criteria, which is outlined and appears appropriate; therefore the process relied heavily on hand searches. All studies double assessed and no authors assessed any of their own previous work. Nonetheless, this paper is a good collection of the clinical tests commonly used for screening for CM and highlights the generally low-moderate quality of studies assessing the clinical utility of such testing. These tests demonstrated high levels of specificity and low levels of sensitivity (see additional comments below). The authors have sub-divided into 3 categories of tests: 1. associated with gait or balance analysis; 2. associated with upper motor neuron or “pathological” signs; and, 3. associated with deep tendon reflexes. Permission has been gained to reproduce Cook et al’s (2011) tables 1-4 and descriptions/positive findings summary within this presentation to help inform readers. These are presented below. The authors recommend further evaluation of these tests and

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Systematic review</td>
<td>12 studies</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
exploration of clustered findings. Tests with low sensitivity are unlikely to identify the condition early in the exam (cannot rule it out) and may result in a false negative. Thus clinicians may apply treatment techniques under false assumptions of safety, which may further compromise the spinal cord.

Table 1: Diagnostic accuracy of test findings associated with gait or balance analysis

<table>
<thead>
<tr>
<th>Study</th>
<th>Reliability</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>LR+</th>
<th>LR-</th>
<th>QUADAS Score (0-14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormally wide-based gait, ataxia, or spastic gait</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cook et al38</td>
<td>NT</td>
<td>19 (14-24)</td>
<td>94 (91-97)</td>
<td>3.4 (1.6-7.3)</td>
<td>0.85 (0.78-0.94)</td>
<td>10</td>
</tr>
<tr>
<td>Static or dynamic Romberg sign</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kiely et al39*</td>
<td>NT</td>
<td>NT</td>
<td>100</td>
<td>NT</td>
<td>NT</td>
<td>2</td>
</tr>
</tbody>
</table>

NT: not tested; * Confidence intervals were not reported in the article

Adapted with permission from Cook et al (2011)

Authors comments: Unable to pool results as QUADAS <10. Both tests very specific. Both tests were evaluated by 1 author only.
### Table 2: Diagnostic accuracy of test findings associated with upper motor neuron or “pathological” signs

<table>
<thead>
<tr>
<th>Study</th>
<th>Reliability</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>LR+</th>
<th>LR−</th>
<th>QUADAS score (0-14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hoffmann sign</td>
<td>NT</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Denmo and Meadows19**</td>
<td>NT</td>
<td>94</td>
<td>NT</td>
<td>NA</td>
<td>NA</td>
<td>9</td>
</tr>
<tr>
<td>Sung and Wong19**</td>
<td>NT</td>
<td>82</td>
<td>NT</td>
<td>NA</td>
<td>NA</td>
<td>6</td>
</tr>
<tr>
<td>Wong et al16**</td>
<td>NT</td>
<td>58</td>
<td>74</td>
<td>2.23</td>
<td>0.57</td>
<td>8</td>
</tr>
<tr>
<td>Glasser et al17 (unblinded tests)*</td>
<td>NT</td>
<td>28</td>
<td>71</td>
<td>0.96</td>
<td>1.01</td>
<td>8</td>
</tr>
<tr>
<td>Glasser et al17 (blinded test)*</td>
<td>NT</td>
<td>44</td>
<td>(28-58)</td>
<td>75</td>
<td>(63-86)</td>
<td>1.8 (0.8-4.1)</td>
</tr>
<tr>
<td>Cook et al16**</td>
<td>NT</td>
<td>59</td>
<td>84</td>
<td>3.69</td>
<td>0.49</td>
<td>6</td>
</tr>
<tr>
<td>Cook et al16**</td>
<td>NT</td>
<td>31</td>
<td>(25-38)</td>
<td>73</td>
<td>(59-84)</td>
<td>4.9 (2.6-9.6)</td>
</tr>
<tr>
<td>Kiely et al17**</td>
<td>NT</td>
<td>90</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>2</td>
</tr>
<tr>
<td>Houen and Nocce41 (bilateral or unilateral)*</td>
<td>NT</td>
<td>68</td>
<td>NT</td>
<td>NA</td>
<td>NA</td>
<td>6</td>
</tr>
<tr>
<td>Chikuda et al19**</td>
<td>NT</td>
<td>81</td>
<td>(72-87)</td>
<td>NT</td>
<td>NA</td>
<td>9</td>
</tr>
<tr>
<td>Babinski sign</td>
<td>NT</td>
<td>18</td>
<td>NT</td>
<td>NA</td>
<td>NA</td>
<td>6</td>
</tr>
<tr>
<td>Hinchcliff et al17**</td>
<td>NT</td>
<td>23</td>
<td>NT</td>
<td>NA</td>
<td>NA</td>
<td>6</td>
</tr>
<tr>
<td>Chumpu</td>
<td>NT</td>
<td>13</td>
<td>100</td>
<td>Inf</td>
<td>0.87</td>
<td>6</td>
</tr>
<tr>
<td>Cook et al16**</td>
<td>NT</td>
<td>7 (4-8)</td>
<td>100 (98-100)</td>
<td>Inf</td>
<td>(2-9-Inf)</td>
<td>0.93 (0.93-0.97)</td>
</tr>
<tr>
<td>Kiely et al17**</td>
<td>NT</td>
<td>100</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>2</td>
</tr>
<tr>
<td>Houen and Nocce41*</td>
<td>NT</td>
<td>23</td>
<td>NT</td>
<td>NA</td>
<td>NA</td>
<td>6</td>
</tr>
<tr>
<td>Chikuda et al19**</td>
<td>NT</td>
<td>53</td>
<td>(44-62)</td>
<td>NT</td>
<td>NA</td>
<td>9</td>
</tr>
<tr>
<td>Lhermitte sign</td>
<td>NT</td>
<td>3</td>
<td>97</td>
<td>1</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Clonus</td>
<td>NT</td>
<td>13</td>
<td>100</td>
<td>Inf</td>
<td>0.87</td>
<td>6</td>
</tr>
<tr>
<td>Cook et al16**</td>
<td>NT</td>
<td>11 (5-16)</td>
<td>96 (90-99)</td>
<td>2.7</td>
<td>(0.4-20.1)</td>
<td>0.9 (0.8-1.1)</td>
</tr>
<tr>
<td>Cook et al16**</td>
<td>NT</td>
<td>7 (4-8)</td>
<td>90 (97-99)</td>
<td>5.4</td>
<td>(1.2-23.6)</td>
<td>0.04 (0.09-0.90)</td>
</tr>
<tr>
<td>Chikuda et al19 (stretched ankle clonus)</td>
<td>NT</td>
<td>35</td>
<td>(26-64)</td>
<td>NT</td>
<td>NA</td>
<td>9</td>
</tr>
<tr>
<td>Gonda-Allen sign</td>
<td>NT</td>
<td>90</td>
<td>NT</td>
<td>NA</td>
<td>NA</td>
<td>9</td>
</tr>
<tr>
<td>Allen-Clarkley sign</td>
<td>NT</td>
<td>82</td>
<td>NT</td>
<td>NT</td>
<td>NA</td>
<td>9</td>
</tr>
<tr>
<td>Inverted supinator sign</td>
<td>NT</td>
<td>81</td>
<td>51</td>
<td>2.6</td>
<td>0.60</td>
<td>6</td>
</tr>
<tr>
<td>Cook et al16**</td>
<td>NT</td>
<td>18 (14-19)</td>
<td>90 (97-99)</td>
<td>29.1</td>
<td>(0.1-171.5)</td>
<td>0.02 (0.81-0.84)</td>
</tr>
<tr>
<td>Kiely et al17**</td>
<td>NT</td>
<td>NT</td>
<td>72.4</td>
<td>NT</td>
<td>NT</td>
<td>2</td>
</tr>
<tr>
<td>Wong et al17**</td>
<td>NT</td>
<td>53</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>6</td>
</tr>
<tr>
<td>Finger escape sign</td>
<td>NT</td>
<td>100</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>2</td>
</tr>
<tr>
<td>Hand withdrawal reflex</td>
<td>NT</td>
<td>55</td>
<td>NT</td>
<td>NT</td>
<td>NT</td>
<td>6</td>
</tr>
<tr>
<td>Cook et al16**</td>
<td>NT</td>
<td>41 (25-58)</td>
<td>63 (51-75)</td>
<td>1.1</td>
<td>(0.3-2.3)</td>
<td>0.9 (0.6-1.5)</td>
</tr>
<tr>
<td>Croust uppogog toe sign (cat)</td>
<td>NT</td>
<td>31</td>
<td>96</td>
<td>7.8</td>
<td>0.72</td>
<td>6</td>
</tr>
</tbody>
</table>

* NT, not tested; NA, not applicable; Inf, infinite.
* Confidence intervals were not reported in the articles.

Table 2: Authors comments – 12 studies met criterion. Hoffmann sign and Babinski most commonly investigated. Tests are more specific than sensitive. Clonus also highly specific. Inverted supinator sign is the most sensitive sign (4 studies investigated). Unable to pool results (only 1 with QUADAS >10).

Adapted with permission from Cook et al (2011)

---

### Table 3: Diagnostic accuracy of test findings associated with deep tendon reflex changes

(Example Table 3)
<table>
<thead>
<tr>
<th>Study</th>
<th>Reliability</th>
<th>Sensitivity (95% CI)</th>
<th>Specificity (95% CI)</th>
<th>LR+ (95% CI)</th>
<th>LR- (95% CI)</th>
<th>QUADAS Score (0-14)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Biceps or triceps hyperreflexia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cook et al&lt;sup&gt;36&lt;/sup&gt;</td>
<td>89% Agreement</td>
<td>44 (28-59)</td>
<td>71 (59-82)</td>
<td>1.5 (0.7-3.4)</td>
<td>0.8 (0.5-1.2)</td>
<td>12</td>
</tr>
<tr>
<td>Cook et al&lt;sup&gt;38&lt;/sup&gt;</td>
<td>NT</td>
<td>18 (13-22)</td>
<td>96 (93-96)</td>
<td>4.8 (2.0-11.7)</td>
<td>0.85 (0.79-0.93)</td>
<td>7</td>
</tr>
<tr>
<td><strong>Suprapatellar reflex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cook et al&lt;sup&gt;36&lt;/sup&gt;</td>
<td>84% Agreement</td>
<td>56 (39-72)</td>
<td>33 (22-46)</td>
<td>0.8 (0.5-1.3)</td>
<td>1.3 (0.6-2.8)</td>
<td>12</td>
</tr>
<tr>
<td>Cook et al&lt;sup&gt;38&lt;/sup&gt;</td>
<td>NT</td>
<td>22 (17-25)</td>
<td>97 (94-99)</td>
<td>6.9 (2.8-17.5)</td>
<td>0.81 (0.76-0.89)</td>
<td>7</td>
</tr>
<tr>
<td><strong>Achilles tendon hyperreflexia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cook et al&lt;sup&gt;36&lt;/sup&gt;</td>
<td>NT</td>
<td>15 (11-17)</td>
<td>98 (96-99)</td>
<td>7.8 (2.5-25.4)</td>
<td>0.87 (0.84-0.93)</td>
<td>7</td>
</tr>
<tr>
<td>Rhee et al&lt;sup&gt;34&lt;/sup&gt;</td>
<td>NT</td>
<td>26</td>
<td>81</td>
<td>1.37</td>
<td></td>
<td>0.91</td>
</tr>
<tr>
<td><strong>Infrapatellar reflex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chikuda et al&lt;sup&gt;44&lt;/sup&gt;</td>
<td>NT</td>
<td>94 (88-97)</td>
<td>NT</td>
<td>NA</td>
<td>NA</td>
<td>9</td>
</tr>
<tr>
<td>Rhee et al&lt;sup&gt;34&lt;/sup&gt;</td>
<td>NT</td>
<td>33</td>
<td>76</td>
<td>1.37</td>
<td></td>
<td>0.88</td>
</tr>
</tbody>
</table>

Authors comments: 4 studies. Measures appear very specific and diagnostic. I low quality study (Chikuda et al) indicated infrapatellar reflex as sensitive. Unable to pool results (only 1 with QUADAS >10).

Adapted with permission from Cook et al (2011)

### Table 4 Clustered findings for diagnosis of CSM<sup>38</sup>

<table>
<thead>
<tr>
<th>Study</th>
<th>Reliability</th>
<th>Sensitivity (95% CI)</th>
<th>Specificity (95% CI)</th>
<th>LR+ (95% CI)</th>
<th>LR- (95% CI)</th>
<th>QUADAS Score (0-14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cook et al&lt;sup&gt;36&lt;/sup&gt;</td>
<td>NT</td>
<td>0.94 (0.89-0.97)</td>
<td>0.31 (0.27-0.32)</td>
<td>1.4 (1.2-1.4)</td>
<td>0.18 (0.12-0.42)</td>
<td>7</td>
</tr>
<tr>
<td>Cook et al&lt;sup&gt;38&lt;/sup&gt;</td>
<td>(2 of 5 positive test results)</td>
<td>0.39 (0.33-0.46)</td>
<td>0.88 (0.84-0.92)</td>
<td>3.3 (2.1-5.5)</td>
<td>0.63 (0.59-0.79)</td>
<td>7</td>
</tr>
<tr>
<td>Cook et al&lt;sup&gt;44&lt;/sup&gt;</td>
<td>(3 of 5 positive test results)</td>
<td>0.19 (0.15-0.20)</td>
<td>0.99 (0.97-0.99)</td>
<td>30.9 (5.5-181.8)</td>
<td>0.81 (0.79-0.87)</td>
<td>7</td>
</tr>
<tr>
<td>Cook et al&lt;sup&gt;45&lt;/sup&gt;</td>
<td>(4 of 5 positive test results)</td>
<td>0.09 (0.06-0.09)</td>
<td>1.0 (0.98-1.0)</td>
<td>Inf (3.9-Inf)</td>
<td>0.91 (0.90-0.95)</td>
<td>7</td>
</tr>
</tbody>
</table>

Five tests are included in the rule: (1) gait deviation, (2) +Hoffmann test, (3) inverted supinator sign, (4) +Babinski test, and (5) age more than 45 years. The associated posttest probability values are based on a pretest probability of 35%.

Authors comments: 1 low quality study clustered test findings. Diagnostically, failure of a -ve finding in 1 of 5 tests resulted in strong screening combination yielding a sensitivity of 0.94 and LR- of 0.18. A finding that included 3 of +ve tests results yielded an LR+ of 30.9 (95% CI=5.5-181.8) and a post-test probability of 94%.

Adapted with permission from Cook et al (2011)
<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
<th>Positive Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gait abnormality</td>
<td>The examiner asks the patient to ambulate while the examiner observes from different angles.</td>
<td>Wide based gait, spastic gait, or ataxia, all typically symmetrical</td>
</tr>
<tr>
<td>Static or Dynamic Rhomberg Sign</td>
<td>Static: With the patient in standing, instruct the patient to stand with their feet together, eyes closed, and hands by his or her side. Dynamic: same position as static but with a light external moment applied to the patient.</td>
<td>Balance disruption</td>
</tr>
<tr>
<td>Hoffman’s Sign</td>
<td>With the patient in sitting or standing, the examiner stabilizes the middle finger just proximal to the distal interphalangeal joint and cradles the patient’s hand. The examiner then either nips the patient’s fingernail between his or her thumb and index finger, or flicks the middle fingernail with the examiner’s fingernail.</td>
<td>Adduction of the thumb and flexion of the fingers</td>
</tr>
<tr>
<td>Babinski’s Sign</td>
<td>With the patient in supine and the foot held in neutral by the examiner, the examiner applies stimulation with the blunt end of a reflex hammer to the plantar aspect of the foot (laterally to medial from heel to metatarsal).</td>
<td><em>electrical-type</em> sensation in the midline and occasionally into the extremities during flexion</td>
</tr>
<tr>
<td>Lhermitte’s Sign</td>
<td>With the patient in sitting or supine, the patient is instructed to flex the neck with emphasis on lower cervical flexion. Some examiners have advocated use of hyperextension to produce Lhermitte’s response. Query the patient for “electrical-type” response during flexion or extension.</td>
<td>An “electrical-type” sensation in the midline and occasionally into the extremities during flexion</td>
</tr>
<tr>
<td>Clonus</td>
<td>This technique can be applied to either the ankle or wrist. With the patient in supine or sitting, the examiner takes slack of the wrist into extension or the ankle into dorsiflexion. The examiner then applies a quick overpressure with maintained pressure.</td>
<td>Repeated beats of 3 or greater</td>
</tr>
<tr>
<td>Gonda-Alien Sign</td>
<td>With the patient in supine, the examiner provides a forceful downward stretch or snaps the distal phalanx of the 2nd or 4th toe. The examiner may also press on the toe nail, twist the toe, and hold for a few seconds.</td>
<td>The extensor toe sign (great toe extension) Similar to positive Babinski’s sign</td>
</tr>
<tr>
<td>Allen-Checkley Sign</td>
<td>With the patient in supine, the examiner provides a sharp upward flick of the 2nd toe or pressure over the distal aspect or ball of the toe.</td>
<td>The extensor toe sign</td>
</tr>
<tr>
<td>Inverted Supinator Sign</td>
<td>With the patient in sitting, the examiner places the patient’s forearm in slight pronation on his or her forearm to ensure relaxation. The examiner applies a series of quick strikes near the styloid process of the radius at the attachment of the brachioradialis and the tendon.</td>
<td>Finger flexion or slight elbow extension</td>
</tr>
<tr>
<td>Finger Escape Sign</td>
<td>With the patient seated, the patient is asked to flex both elbows to 90° and keep them at his or her side. The forearms ar then pronated and all fingers are adducted.</td>
<td>Inability of the patient to maintain adduction of the 5th digit which will start to drift in an ulnar and volar direction.</td>
</tr>
<tr>
<td>Hand Withdrawal Reflex</td>
<td>With the patient in either sitting or standing, the examiner grasps the patient's palm and strikes the dorsum of the patient's hand with a reflex hammer.</td>
<td></td>
</tr>
<tr>
<td>Crossed Uppgoing Toe Sign (Cut sign)</td>
<td>With the patient in supine, the examiner passively raises the opposite limb into hip flexion. The examiner then instructs the patient to hold the leg in flexion. The examiner applies a downward force against the leg. The examiner needs to visually inspect the opposite great toe.</td>
<td>Upgoing toe sign of great toe of the opposite toe</td>
</tr>
<tr>
<td>Biceps or Triceps Hyperreflexia</td>
<td>Biceps: With the patient in sitting, the examiner slightly supinates the patient’s forearm and places it on his or her own forearm. The examiner’s thumb is then placed on the patient’s bicep tendon and is struck with quick strikes of a reflex hammer. Triceps: With the patient in sitting, the patient’s shoulder is elevated to 90° with the elbow passively flexed to 90°. The examiner’s thumb is placed over the distal aspect of the patient's triceps tendon and a series of quick strikes is applied with a reflex hammer to the back of the examiner's thumb.</td>
<td>Brisk, exaggerated finding or hyperreflexia</td>
</tr>
<tr>
<td>Suprapatellar Reflex</td>
<td>With the patient seated and his or her feet off the ground, the examiner applies quick strikes of the reflex hammer to the suprapatellar tendon.</td>
<td>Brisk, exaggerated finding or hyperreflexia</td>
</tr>
<tr>
<td>Test</td>
<td>Procedure</td>
<td>Findings</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>---------------------------------------</td>
</tr>
<tr>
<td>Achilles Tendon Hyperreflexia</td>
<td>With the patient seated and his or her feet off the ground, the examiner uses a reflex hammer to either strike the Achilles tendon itself or use the plantar strike technique. If the reflex is absent, ask the patient to plantarflex the foot, tightly close the eyes and pull their clasped hand apart just prior to striking.</td>
<td>Brisk, exaggerated finding or hyperreflexia</td>
</tr>
<tr>
<td>Infrapatellar reflex</td>
<td>With the patient seated and the his or her feet off the ground, the clinician uses a reflex hammer to deliver quick strikes to the infrapatellar tendon</td>
<td>Brisk, exaggerated finding or hyperreflexia</td>
</tr>
</tbody>
</table>

Adapted with permission from Cook et al (2011)
<table>
<thead>
<tr>
<th>Study</th>
<th>Study design</th>
<th>Evid. Level</th>
<th>Sample size</th>
<th>Population characteristics</th>
<th>Index Test</th>
<th>Reference Test</th>
<th>Reliability</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>QUADAS Score (0-14)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numasawa, N., Ono, A., Wada, K., Yamasaki, Y., Yokoyama, T., Aburakawa, S., Takeuchi, K., Kumagai, G., Kudo, H., Uneda, T., Nakaji, S. and Toh, S. 2012. Simple foot tapping test as a quantitative objective assessment of cervical myelopathy. Spine, 37(2), pp.108-103</td>
<td>Cohort</td>
<td>2++</td>
<td>252 (126 tested pre-post op).</td>
<td><strong>CM group:</strong> Male (168: 65.9%), female (86: 34.1%) Mean age 64.8 years (male range 32-84; female 43-82).</td>
<td>Foot tapping test (FTT)</td>
<td>Grip and release test (see Cook et al 2011 description).</td>
<td>FTT: right side (r=0.934, p&lt;0.0001); left side (r=0.899, p&lt;0.0001).</td>
<td>12</td>
<td>CM group: FTT mean value 23.8±7.2</td>
<td>FTTS scores improved by surgery.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Control group n=792</td>
<td>Control group: Male (279 (35.2%), female (513: 64.8%). Mean age 57.5 years (male range 20-83; female 21-83)</td>
<td>Functional lower limb measure: modified Japanese Orthopaedic Association Lower Extremity (JOALE)</td>
<td></td>
<td></td>
<td></td>
<td>Control group: FTT mean value 31.3±6.5. FTT strongly correlated with JOALE (r=0.696, P&lt;0.0001) and correlated with grip /release test (r=0.571, P&lt;0.0001).</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Test description:** Foot tapping test (FTT): Participant seated on a chair with comfortable posture (hips and knees at approx 90°). The value of FTT was measured bilaterally by having sole of the foot tap as many times as possible for 10 seconds while keeping the heel in contact with the floor. The mean no. of times was adopted for the data analysis. Slowness of foot tapping is a phenomenon that reflects upper motor neuron weakness with cerebral or spinal cord disorders (p.106).
Appendix B: South East Scotland Research Ethics Service letter

South East Scotland Research Ethics Service
Waverley Gate
24 Waterloo Place
Edinburgh
EH1 3EG

Name: Colin Redmond
Address: Principal Spinal Physiotherapist
Physiotherapy Department
Borders General Hospital
Minto
TD6 0BS

Date: 08/02/2012
Your Ref: NR/1201A393
Our Ref: Alex Bailey
Enquiries to: Direct Line: 0131 448 6776
Email: alex.bailey@nhslothian.scot.nhs.uk

Dear Colin,

Full title of project: Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders

You have sought advice from the South East Scotland Research Ethics Service on the above project. This has been considered by the Scientific Officer and you are advised that, based on the submitted documentation (email correspondence and 1201105 QMU Doctoral Research Proposal Colin Redmond (NHS Borders) - Ethics-1), it does not need NHS ethical review under the terms of the Governance Arrangements for Research Ethics Committees in the UK. The advice is based on the following:

- The project is an opinion survey seeking the views of NHS staff on a healthcare issue.

If this project is being conducted within NHS Lothian you should inform the relevant local Quality Improvement Team(s).

This letter should not be interpreted as giving a form of ethical approval or any endorsement of the project, but it may be provided to a journal or other body as evidence that ethical approval is not required under NHS research governance arrangements. However, if you, your sponsor/under or any NHS organisation feels that the project should be managed as research and/or that ethical review by a NHS REC is essential, please write setting out your reasons and we will be pleased to consider further. Where NHS organisations have clarified that a project is not to be managed as research, the Research Governance Framework states that it should not be presented as research within the NHS.

You should retain a copy of this letter with your project file as evidence that you have sought advice from the South East Scotland Research Ethics Service.

Yours sincerely,

[Signature]

Alex Bailey
Scientific Officer
South East Scotland Research Ethics Service
Appendix C: NHS Borders Research and Governance Committee letter

NHS Borders
Research Administration
Clinical Governance

Mr Colin Redmond
2 Cranston Road
Lauder
TD2 6TU

Date 17th March 2012
Our Ref SE46
Enquiries to Extension
Email research.governance@borders.scot.nhs.uk

Dear Mr Redmond

Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders

Thank you for sending details of your study to NHS Borders. Given that ethics has deemed your study an evaluation, your study does not require R&D Management approval. However, it has been noted by our Research Governance Committee.

You may proceed with your study in the NHS Borders area. We ask that you inform the R&D Office when the study is completed.

May I take this opportunity to wish you every success with your project. Please do not hesitate to contact the R&D Office should you require any further assistance.

Yours sincerely

Name deleted for thesis
Associate Medical Director (Clinical Governance)
APPLICATION FOR ETHICAL APPROVAL
FOR A RESEARCH PROJECT 2011/12

This is an application form for ethical approval to undertake a piece of research. Ethical approval must be gained for any piece of research to be undertaken by any student or member of staff of QMU. Approval must also be gained by any external researcher who wishes to use Queen Margaret students or staff as participants in their research.

Please note, before any requests for volunteers can be distributed, through the moderator service, or externally, this form MUST be submitted (completed, with signatures) to the Secretary to the Research Ethics Panel.

You should read QMU’s chapter on “Research Ethics: Regulations, Procedures, and Guidelines” before completing the form. This is available at: http://www.qmu.ac.uk/quality/rs/default.htm

Hard copies are available from the Secretary to the Research Ethics Panel.

The person who completes this form (the applicant) will normally be the Principal Investigator (in the case of staff research) or the student (in the case of student research). In other cases of collaborative research, e.g. an undergraduate group project, one member should be given responsibility for applying for ethical approval. For class exercises involving research, the module coordinator should complete the application and secure approval.

The completed form should be typed rather than handwritten. Electronic signatures should be used and the form should be submitted electronically wherever possible.

**Applicant details**

1. Researcher’s name: Colin Redmond
2. Researcher’s contact email address: 09001905@qmu.ac.uk
3. Category of researcher (please tick and enter title of programme of study as appropriate):

<table>
<thead>
<tr>
<th>QMU undergraduate student</th>
<th>Title of programme:</th>
</tr>
</thead>
<tbody>
<tr>
<td>QMU postgraduate student – taught degree</td>
<td>Title of programme:</td>
</tr>
<tr>
<td>QMU postgraduate student – research degree (Professional Doctorate)</td>
<td>✔</td>
</tr>
<tr>
<td>QMU staff member – research degree</td>
<td></td>
</tr>
</tbody>
</table>
4. School: Health Sciences

5. Division: Dietetics, Nutrition, Biological Sciences, Physiotherapy, Podiatry and Radiography

6. Name of Supervisor or Director of Studies (if applicable): Dr Fiona Macmillan; 2nd supervisor Dr Jan Gill

7. Names and affiliations of all other researcher who will be working on the project:

Research details

8. Title of study: Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders.

9. Expected start date: March 2012

10. Expected end date: June 2013

11. Details of any financial support for the project from outside QMU: NHS National Education Scotland (NES) is contributing up to £200.00 towards focus group expenses. NES and NHS Borders have additionally contributed funds towards academic fees 2011/12.

12. Please detail the aims and objectives of this study (max. 400 words)

<table>
<thead>
<tr>
<th>Research aim and objectives</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aim</strong></td>
</tr>
<tr>
<td>The overarching aim of the study is to formulate a red flag screening algorithm for clinical indicators of possible serious cervical spine related pathology presenting as, or alongside, a musculoskeletal disorder.</td>
</tr>
</tbody>
</table>

**Objectives**

1. Conduct a detailed literature review that will be used to develop materials for a focus group.

2. Facilitate a focus group (physiotherapists) prioritising the research literature review main findings (from a musculoskeletal physiotherapy perspective) for inclusion in a red flag screening tool. This will also reveal areas of consensus and lack of consensus in preparation for presenting material in a Delphi process.

3. Construct and implement a Delphi survey with medical experts where iterative exploration of the findings formulated from 1 and 2 will allow these to be refined.

4. Formulate final draft cervical spine red flag screening algorithm suitable for use in a clinical setting.
Methodology

13. Research procedures to be used: please tick all that apply.

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Tick if applicable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Questionnaires (please attach copies of all questionnaires to be used)</td>
<td>✔ (part of phase 2 and will be developed following phase 1)</td>
</tr>
<tr>
<td>Interviews (please attach summary of topics to be explored)</td>
<td></td>
</tr>
<tr>
<td>Focus groups (please attach summary of topics to be explored / copies of materials to be used)</td>
<td>✔</td>
</tr>
<tr>
<td>Experimental / Laboratory techniques (please include full details under question 14)</td>
<td></td>
</tr>
<tr>
<td>Use of email / internet as a means of data collection (please include full details under question 14)</td>
<td>✔</td>
</tr>
<tr>
<td>Use of questionnaires / other materials that are subject to copyright (please include full details under question 14 and confirm that the materials have been / will be purchased for your use)</td>
<td></td>
</tr>
<tr>
<td>Use of biomedical procedures to obtain blood or tissue samples (please include full details under question 14 and include subject area risk assessment forms, where appropriate)</td>
<td></td>
</tr>
<tr>
<td>Other technique / procedure (please include full details under question 14)</td>
<td></td>
</tr>
</tbody>
</table>

14. Briefly outline the nature of the research and the methods and procedures to be used (max. 400 words).
Methodology

Introduction

The study’s aim to inform clinical practice by developing a clinical screening algorithm will be created through a 2-phase process that follows a literature review: Phase 1 - Physiotherapy focus group that informs phase 2; Phase 2 - Delphi survey of medical experts. This process seeks to achieve a clinical consensus on main clinical features, subjective questioning and subsequent actions or tests to enhance early detection of serious pathologies presenting as, or alongside, a musculoskeletal disorder. The Delphi technique is considered as a suitable method to gain a consensus where there is incomplete knowledge or uncertainty in clinical issues.

Phase 1: Focus group

Participants: Approximately 6-10 UK based physiotherapists (Recruitment process - refer to Appendix K).

Procedure and Data Analysis: The Principal Investigator (PI) will circulate the topics for discussion and summary of the key components of the literature review to the group participants (Refer to example Appendix A). The focus group will be conducted within QMU (subject to gaining permission). This will combine attendance for locally based (within South-Central Scotland) and video link for participants out with this location. A transcript will be compiled. Thematic content will be established. This will be used to prioritise clinical sections for inclusion in a screening algorithm and formulate accompanying questions for inclusion in phase 2 round 1.

Phase 2: Delphi study (including pilot study)

Pilot Delphi study: To assist reliability or consistency in the interpretation of the questions, the 1st round questions will undergo a pilot phase. The questions will be submitted to three medical practitioners using the same procedure as outlined below (Recruitment process - refer to Appendix L). The purpose is to explore respondents’ interpretations of the questions achieved through follow-up discussion with the PI. The questions will be amended accordingly for the main study.

Main Delphi study participants: Consultant neurologists. Neurologists are medical specialists in the relevant areas (e.g. cervical arterial dysfunction and cervical myelopathy). A sample number between 10-15 participants is considered appropriate (Recruitment process - refer to Appendix M).

Procedure and data analysis: An internet based survey system http://www.survey.bris.ac.uk/ will be used as the data collection system over 3 rounds. Round 1 will consist of a small (<5) number of initial short questions to obtain demographic data, followed by a number of questions based on presented non-sensitive information obtained from the literature review and focus group.

Main Delphi study: Main study round 1 data will undergo a thematic content analysis and subsequently used to inform the survey content in round 2. Inter-coder reliability will be checked by the PI and one research supervisor coding the same response data from 3 randomly selected transcripts. This will provide verification of the content analysis. Intra-coder reliability will be checked by the PI coding 3 randomly selected transcripts on two separate occasions with a minimum of 2-days a part. Rounds 2 and 3 data analysis will utilise a quantitative approach.

Non-respondents: Email reminders will be sent on two occasions after each round (after 2 and 3 weeks, respectively).

15. Does your research include the use of people as participants? Please delete as appropriate. Yes
16. Does your research include the experimental use of live animals? Please delete as appropriate. No

17. Does your research involve experimenting on plant or animal matter, or inorganic matter? Please delete as appropriate. No

18. Does your research include the analysis of documents, or of material in non-print media, other than those which are freely available for public access? Please delete as appropriate. No

19. If you answered ‘Yes’ to question 18, give a description of the material you intend to use. Describe its ownership, your rights of access to it, the permissions required to access it and any ways in which personal identities might be revealed or personal information might be disclosed. Describe any measures you will take to safeguard the anonymity of sources, where this is relevant:

This text box will expand as required.

20. Will any restriction be placed on the publication of results? Please delete as appropriate. No

21. If you answered ‘Yes’ to question 20, give details and provide a reasoned justification for the restrictions. (See Research Ethics Guidelines Section 2, paragraph 7)

This text box will expand as required.

22. Will anyone except the named researchers have access to the data collected? Please delete as appropriate. No

23. Please give details of how and where data will be stored, and how long it will be retained for before being destroyed. (See Research Ethics Guidelines Section 1, paragraph 2.4.1)

Data will be stored on NHS encrypted information systems (memory stick, laptop and health board’s network) and in accordance with NHS Borders Data Protection Policy (2006), which incorporates the Data Protection Act 1998. In accordance with QMU Code of Research Practice recommendations all data will be stored for 5-years post completion of the study (http://www.qmu.ac.uk/research_knowledge/docs/Code%20of%20Practice-Aug%202011.doc Appendix A).

24. Please highlight what you see as the most important ethical issues this study raises (eg. adverse physical or psychological reactions; addressing a sensitive topic area; risk of loss of confidentiality; other ethical issue. If you do not think this study raises any ethical issues, please explain why).

The main ethical issue is to prevent loss of participant confidentiality. However, the encrypted information systems offer the best available measure against this loss occurring. The Delphi survey participants will also be informed that their identity will be withheld from other participants. This is to ensure that an individual’s responses will not be influenced directly by another participant. Due to the nature of the focus group this aspect is not possible, therefore all participants will be introduced. However, the focus group data will also be stored in the same encrypted systems and names replaced by coder identifiers.
25. If you have identified any ethical issues associated with this study, please explain how the potential benefits of the research outweigh any potential harms (eg. by benefiting participants; by improving research skills; other potential benefit).

Participant responses are their own professional opinions on clinical topics that are considered important to developing a clinical screening process, as opposed to opinions on clinically sensitive patient information. Participant opinions on the research topics are considered as non-sensitive with no greater risk than discussing such non-sensitive information during a formal or informal meeting that could take place as part of an individual’s professional role. The developed screening process will be used to promote patient safety through early identification of potentially serious pathology presenting in musculoskeletal disorders. This benefit is considered to significantly outweigh any potential harm.

Protection for the Researcher

26. Will the researcher be at risk of sustaining either physical or psychological harm as a result of the research? Please delete as appropriate. No

27. If you answered ‘Yes’ to question 26, please give details of potential risks and the precautions which will be taken to protect the researcher.

Research Involving Human Participants

You should only complete this section if you have indicated above that your research will involve human participants.

28. Please indicate the total number of participants you intend to recruit for this study from each participant group:

<table>
<thead>
<tr>
<th>Participant Group</th>
<th>Please state total number</th>
</tr>
</thead>
<tbody>
<tr>
<td>QMU students</td>
<td></td>
</tr>
<tr>
<td>QMU staff</td>
<td></td>
</tr>
<tr>
<td>Members of the public from outside QMU</td>
<td></td>
</tr>
<tr>
<td>NHS patients</td>
<td></td>
</tr>
<tr>
<td>NHS employees</td>
<td>Refer to Appendix N: (NHS) Research Ethics Service letter – confirming NHS ethical review is not required.</td>
</tr>
<tr>
<td>Children (under 18 years of age)</td>
<td></td>
</tr>
<tr>
<td>People in custody</td>
<td></td>
</tr>
<tr>
<td>People with communication or learning difficulties</td>
<td></td>
</tr>
<tr>
<td>People with mental health issues</td>
<td></td>
</tr>
<tr>
<td>People engaged in illegal activities (eg. illegal drug use)</td>
<td></td>
</tr>
<tr>
<td>Other (please specify):</td>
<td></td>
</tr>
</tbody>
</table>

20-30 (additional Delphi participants will be accepted if volunteers are forthcoming)

* Please declare in section 32 where the participant group may necessitate the need for standard or enhanced disclosure check
29. Please state any inclusion or exclusion criteria to be used. (See Research Ethics Guidelines Section 1, paragraph 2.4)

<table>
<thead>
<tr>
<th>Inclusion criteria:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Focus group</strong> – Physiotherapist plus one of the following; clinical/academic staff with publications in relevant areas; consultant level; senior specialist spinal role; or lecturer in spinal component of post-graduate MSc in Neuromusculoskeletal Physiotherapy/Manual Therapy.</td>
</tr>
<tr>
<td><strong>Pilot Delphi Study</strong> – NHS Borders medical practitioner at Consultant or GP level</td>
</tr>
<tr>
<td><strong>Delphi survey</strong> - Consultant neurologist and/or neurologists with publications in relevant areas.</td>
</tr>
<tr>
<td>No added exclusion criteria.</td>
</tr>
</tbody>
</table>

30. Please give details of how participants will be recruited:

<table>
<thead>
<tr>
<th>Focus group: Physiotherapists – Refer to Appendix K</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pilot Delphi study: NHS Borders Medical Practitioners - Refer to Appendix L</td>
</tr>
<tr>
<td>Main Delphi study: Consultant neurologists - Refer to Appendix M</td>
</tr>
</tbody>
</table>

31. Please describe how informed consent will be obtained from participants. (See Research Ethics Guidelines Section 1, paragraphs 2.1.2 – 2.1.5)

<table>
<thead>
<tr>
<th>Focus group: Refer to Appendix E. Participants will be instructed to ‘Please copy /paste this completed form to your work email address and return to the researcher’s university email address’. This method is adapted from the procedure outlined below in the Main Delphi study.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pilot Delphi study: Refer to Appendix F (as Main Delphi study below)</td>
</tr>
<tr>
<td>Main Delphi study: Refer to Appendix G. Participants will be informed that clicking the web link to online data collection tool <a href="http://www.survey.bris.ac.uk/">http://www.survey.bris.ac.uk/</a> will be considered as gaining informed consent. Participants will be informed of this procedure. This process has been used in a published Delphi survey.</td>
</tr>
</tbody>
</table>

32. Ethical Principles incorporated into the study (please tick as applicable):

<table>
<thead>
<tr>
<th>Ethical Principles</th>
<th>Tick as applicable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Will participants be offered a written explanation of the research?</td>
<td>✓</td>
</tr>
<tr>
<td>Will participants be offered an oral explanation of the research? (Applicant added: opportunity offered via participant information sheets – Appendices B-D).</td>
<td>✓</td>
</tr>
<tr>
<td>Will participants sign a consent form? (Applicant added: Refer to section 31. Focus group participants will provide consent by responding through personalised work email; and, Delphi participants’ consent will be gained by accessing the web-based survey).</td>
<td>✓</td>
</tr>
<tr>
<td>Will oral consent be obtained from participants?</td>
<td></td>
</tr>
<tr>
<td>Will participants be offered the opportunity to decline to take part?</td>
<td>✓</td>
</tr>
<tr>
<td>Will participants be informed that participation is voluntary?</td>
<td></td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>--------</td>
</tr>
<tr>
<td>Will participants be offered the opportunity to withdraw at any stage without giving a reason?</td>
<td>✓</td>
</tr>
<tr>
<td>Will independent expert advice be available if required?</td>
<td>✓</td>
</tr>
<tr>
<td>Will participants be informed that there may be no benefit to them in taking part?</td>
<td>✓</td>
</tr>
<tr>
<td>Will participants be guaranteed confidentiality?</td>
<td>✓</td>
</tr>
<tr>
<td>Will participants be guaranteed anonymity? <em>(Applicant added: Delphi survey participants will be anonymous to all except the Principal Investigator. Due to the format of the focus group, participants will be introduced to one another. However, for the purpose of all reporting, group participants will be assigned a coder identifier. In the event of achieving future journal publication, participants may be offered the opportunity to be ‘acknowledged’. Further consent will be gained if this opportunity arises).</em></td>
<td>✓</td>
</tr>
<tr>
<td>Will the participant group necessitate a standard or enhanced disclosure check?</td>
<td></td>
</tr>
<tr>
<td>Will the provisions of the Data Protection Act be met?</td>
<td>✓</td>
</tr>
<tr>
<td>Has safe data storage been secured?</td>
<td>✓</td>
</tr>
<tr>
<td>Will the researcher(s) be free to publish the findings of the research?</td>
<td>✓</td>
</tr>
<tr>
<td>If the research involves deception, will an explanation be offered following participation?</td>
<td>✓</td>
</tr>
<tr>
<td>If the research involves questionnaires, will the participants be informed that they may omit items they do not wish to answer?</td>
<td>✓</td>
</tr>
<tr>
<td>If the research involves interviews, will the participants be informed that they do not have to answer questions, and do not have to give an explanation for this?</td>
<td>✓</td>
</tr>
<tr>
<td>Will participants be offered any payment or reward, beyond reimbursement of out-of-pocket expenses?</td>
<td>✓</td>
</tr>
</tbody>
</table>
# Risk Assessment

**School / Division:** Health Sciences / Dietetics, Nutrition, Biological Sciences, Physiotherapy, Podiatry and Radiography  
**Location:** QMU / NHS Borders  
**Date:** 08/02/2012

**Assessed by:** C Redmond  
**Job Title:** Prof Doc student / Principal Spinal Physiotherapist

**Activity / Task:** Prof Doc Research  
**Total Number exposed to risk:** 20-30  
**Review Date:**

## Reference

Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders.

<table>
<thead>
<tr>
<th>Ref no.</th>
<th>Hazards</th>
<th>People at risk</th>
<th>Likelihood</th>
<th>Severity</th>
<th>Total risk</th>
<th>Existing control measures</th>
<th>Adequate controls?</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Focus group participants: dominant character effect – offends other participants.</td>
<td>6-10</td>
<td>Improbable</td>
<td>Minor</td>
<td>2</td>
<td>Explain ground rules re. Professional conduct i.e. respect of different</td>
<td></td>
</tr>
<tr>
<td></td>
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<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Loss of participant confidentiality.</td>
<td>20-30</td>
<td>✓</td>
<td>✓</td>
<td>1</td>
<td>Data secured within NHS encrypted systems and adherence to principles of Data Protection Act (1988).</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Availability of respondents to participate is significantly poorer than expected / there is a higher than expected drop out rate.</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>3</td>
<td>Focus group: ensure potential focus group participants confirm their acceptance or rejection of invitation. Approach additional participants if suitable numbers not achieved. Continue recruitment strategy of Delphi phase until sufficient numbers achieved. Include all respondents (in excess of basic number required) in Delphi phase, if inclusion criteria are met.</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Respondents become unavailable for focus group or withdraw from Delphi survey</td>
<td></td>
<td>✓</td>
<td>✓</td>
<td>3</td>
<td>Risk minimised by interim communication with respondents between recruitment and focus group date, and throughout the Delphi survey with feedback.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Description</td>
<td>Likelihood</td>
<td>Severity</td>
<td>Risk Value</td>
<td>Summary</td>
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</tr>
<tr>
<td>5</td>
<td>Data lost due to network malfunction</td>
<td>✓</td>
<td>✓</td>
<td>3</td>
<td>Principal Investigator to make daily backups of project information.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Unable to access workstation due to property damage/theft</td>
<td>✓</td>
<td>✓</td>
<td>1</td>
<td>Principal Investigator will be able to access back-up project material from multiple sites.</td>
<td></td>
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<tr>
<td>7</td>
<td>Maintaining contact with participants</td>
<td>✓</td>
<td>✓</td>
<td>1</td>
<td>Principal Investigator’s email, plus alternative email, address and two telephone numbers will be issued to participants should they wish to discuss any aspect of the study.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Complaints procedure</td>
<td>✓</td>
<td>✓</td>
<td>1</td>
<td>QMU Code of Research Practice will be followed. In the first instance the issue should be addressed to the Principal Investigator, supported by the research supervisor. If the complaint can not easily be resolved in a timely manner, the QMU escalation procedure will be employed.</td>
<td></td>
<td></td>
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**Risk value (RV)**

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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</table>

Total risk = Likelihood (RV) x Severity (RV)

- Total risk of 1 – 4 = 'L', low risk
- Total risk of 6 – 9 = 'M', medium risk
- Total risk of 12 – 16 = 'H', high risk
Remedial action required

<table>
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<th>Action required</th>
<th>Target date</th>
<th>Action by:</th>
<th>Date completed</th>
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<tr>
<td>5.</td>
<td></td>
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</tbody>
</table>
Declarations

34. Having completed all the relevant items of this form and, if appropriate, having attached the Information Sheet and Consent Form plus any other relevant documentation as indicated below, complete the statement below.

- I have read Queen Margaret University’s document on “Research Ethics: Regulations, Procedures, and Guidelines”.

- *In my view* this research is:

  - Non-invasive
  - Minor invasive using an established procedure at QMU
  - Minor invasive using a NEW procedure at QMU
  - Major invasive

- I request Ethical Approval for the research described in this application.

  Name *(if you have an electronic signature please include it here)*

  Colin Redmond___ Date ___08/02/2012____

Documents enclosed with application:

```
<table>
<thead>
<tr>
<th>Document</th>
<th>Enclosed (please tick)</th>
<th>Not applicable (please tick)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Copy of consent form(s)</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Copy of information sheet(s)</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Sample questionnaire</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Example interview questions</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Copy of proposed recruitment advert(s)</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Letters of support from any external organisations involved in the research</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Evidence of disclosure check</td>
<td></td>
<td>✓</td>
</tr>
<tr>
<td>Division risk assessment documentation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any other documentation (please detail below)</td>
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<td></td>
</tr>
<tr>
<td>Risk Assessment</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>NHS Research Ethics Service letter</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>See appendices list next page</td>
<td>✓</td>
<td></td>
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Appendices

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35. If you are a student, show the completed form to your supervisor/Director of Studies and ask them to sign the statement below. If you are a member of staff, sign the statement below yourself.

- I am the supervisor/Director of Studies for this research.
- In my view this research is:

<table>
<thead>
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<th>See Research Ethics Guidelines Section 6</th>
<th>Please tick</th>
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</thead>
<tbody>
<tr>
<td>Non-invasive</td>
<td>√</td>
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<tr>
<td>Minor invasive using an established procedure at QMU</td>
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<tr>
<td>Minor invasive using a NEW procedure at QMU</td>
<td></td>
</tr>
<tr>
<td>Major invasive</td>
<td></td>
</tr>
</tbody>
</table>

- I have read this application and I approve it.

Name (if you have an electronic signature please include it here)

Dr Fiona S Macmillan________________________ Date 13th Feb 2012_________
36. **For all applicants**, send the completed form to your Head of Division or Head of Research Centre or, if you are an external researcher, submit the completed form to the Secretary to the QMU Research Ethics Panel. **You should not proceed with any aspect of your research which involves the use of participants, or the use of data which is not in the public domain, until you have been granted Ethical Approval.**

<table>
<thead>
<tr>
<th>FOR COMPLETION BY THE HEAD OF DIVISION/HEAD OF RESEARCH CENTRE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Either</strong></td>
</tr>
<tr>
<td>I refer this application back to the applicant for the following reason(s):</td>
</tr>
</tbody>
</table>

Name *(if you have an electronic signature please include it here)*

_______________________________________        Head of Division / Research Centre

Date ______________

Please return the form to the applicant.

Or

Please tick one of the alternatives below and delete the others.

- I refer this application to the QMU Research Ethics Panel.
- I find this application acceptable and an application for Ethical Approval should now be submitted to a relevant external committee.
- I grant Ethical Approval for this research.

Name *(if you have an electronic signature please include it here)*

_______________________________________        Head of Division / Research Centre

Date ______________

Please send one copy of this form to the applicant and one copy to the Secretary to the Research Ethics Panel, Quality Enhancement Unit, Registry.

**Date application returned: ____________________**
Appendix E: Queen Margaret University Student project release form (ethical approval)

DIVISION OF DIETETICS, NUTRITION, BIOLOGICAL SCIENCES, PHYSIOTHERAPY, PODIATRY and RADIOGRAPHY

STUDENT PROJECT RELEASE FORM

This form is designed to notify each student of the DivREC response to individual Dissertation Proposals. A copy of the form will also be retained by the Committee to record each decision and to monitor resource requirements. StudentsPlease complete a – d below

a. PROJECT TITLE: _Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders._

b. STUDENT(S): ____Colin Redmond______________________________

c. SUPERVISOR: __ Dr Fiona Macmillan; 2nd supervisor Dr Jan Gill__________________________

d. SITE FOR DATA COLLECTION ____NHS Borders_________________________ (if not QMU state where)

e. APPROXIMATE DATES FOR DATA COLLECTION __March 2012 – June 2012_________________

All students should refer to Committee Response below and Comments overleaf

COMMITTEE RESPONSE

<table>
<thead>
<tr>
<th>Decision</th>
<th>✓ / X</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Project proposal and Ethical approval granted</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>2. Proceed with minor modifications to the project proposal (as noted in response overleaf)</td>
<td>✓</td>
<td>ASAP</td>
</tr>
<tr>
<td>3. Resubmit revised proposal by (insert date...........)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Resubmit revised ethics by (insert date .........)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Submit for further ethics scrutiny (QMU / external)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Project documentation incomplete</td>
<td></td>
<td></td>
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</tbody>
</table>

Please note – you can not proceed to dissertation unless response box 1 or boxes 1 and 2 ticked ✓

GENERAL COMMENTS:

This project has been granted ethical approval provided some minor changes are made and the DivREC committee informed by letter that these changes have been made.

This form will only be signed by Head of Division once project and ethical approval granted
<table>
<thead>
<tr>
<th>ISSUES ADDRESSED</th>
<th>COMMITTEE RESPONSE</th>
<th>ACTION</th>
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<tbody>
<tr>
<td>Student complete this column</td>
<td>ok</td>
<td>None</td>
</tr>
</tbody>
</table>

**i. Subjects**

<table>
<thead>
<tr>
<th>Number of subjects</th>
<th>N=</th>
<th>ok</th>
<th>None</th>
</tr>
</thead>
<tbody>
<tr>
<td>QM University students or other? (Please state)</td>
<td>ok</td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

**ii. Access to QMU facilities**

| ?Which labs / rooms (if known) | N/A | N/A |
| ?When is access required (dates and/or times) | N/A | N/A |

**iii. Training requirements**

| Technician support required | Yes / No / NA | N/A |
| Supervisor to provide training | Yes / No /NA | N/A |

**iv. Equipment requirement**

| State precise equipment requirements | ok | None |

**v. Costs**

| (e.g. reprographics / postage / consumables) | ok | Student |

---

**DivREC representative to complete sections vi. – xv, after proposal scrutiny**

<table>
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<th>RESPONSE</th>
<th>ACTION REQUIRED</th>
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</thead>
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<tr>
<td><strong>vi. Recruitment Procedure (any advert should be included by student)</strong></td>
<td>Need to add where interview tapes will be stored, who will have access to theses and when they will be destroyed.</td>
<td>Student</td>
</tr>
<tr>
<td><strong>vii. Intervention / Investigation Procedure</strong></td>
<td>ok</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Outcome measures</td>
<td>ok</td>
</tr>
<tr>
<td>---</td>
<td>------------------</td>
<td>----</td>
</tr>
<tr>
<td>ix.</td>
<td>Risk Assessment completed</td>
<td>ok</td>
</tr>
<tr>
<td>x.</td>
<td>Safety concerns (COSSH etc)</td>
<td>ok</td>
</tr>
<tr>
<td>xi.</td>
<td>Information sheet</td>
<td>Focus group information leaflet – need to add information on storage, handling and maintaining confidentiality of tape recordings. Pilot Delphi information leaflet – “why have I been chosen?” rephrase 1st sentence of the paragraph. Delphi survey information leaflet – “what will happen if I take part?” renumber paragraphs as 1 - 4.</td>
</tr>
<tr>
<td>xii.</td>
<td>Consent Form</td>
<td>Consent forms add participant name and date to these forms in appendix F and G.</td>
</tr>
<tr>
<td>xiii.</td>
<td>Independent advisor</td>
<td>ok</td>
</tr>
<tr>
<td>xiv.</td>
<td>Feasibility</td>
<td>ok</td>
</tr>
<tr>
<td>xv.</td>
<td>Other issues</td>
<td>Appendix H paragraph starting “Whilst red flags …. ” Remove “is” after needed in last sentence of this paragraph.</td>
</tr>
</tbody>
</table>
Appendix F: Request letter 1 for ethical approval for minor amendments
2 Cranston Road
Lauder
TD2 6TU

2nd July 2012

Dr Derek Santos
DivREC Member
Division of Dietetics, Biological Sciences,
Physiotherapy, Podiatry and Radiography
Queen Margaret University
Edinburgh
EH21 6UU

Dear Dr Santos

Professional Doctorate Research Study - Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders:
Amendment to Recruitment Process

Further to my letter of 21/03/12 informing you of the minor changes as requested by the DivREC student project release form (16/3/12) for the above project, I would like to inform you of the following planned expansion to my recruitment process for the main Delphi study phase:

1. Approach individual health boards to recruit medical consultants.

2. Our local consultant neurologist has recommended expanding my recruitment call to include consultant neurosurgeons. My original inclusion criterion was limited to consultant neurologists.

3. Contact the Society of British Neurological Surgeons and Irish Institute of Clinical Neuroscience to request circulation of research call for participants to their respective membership.

I have included my amended Delphi study research call for medical consultants.

I hope the expanded recruitment process meets with your considered approval. The project methodology remains unchanged.

Yours sincerely

Colin Redmond
Prof Doc Student 09001905
Appendix G: Request letter 2 for ethical approval for minor amendments

2 Cranston Road
Lauder
TD2 6TU
16th October 2012

Dr Derek Santos
DivREC Member
Division of Dietetics, Biological Sciences, Physiotherapy, Podiatry and Radiography
Queen Margaret University
Edinburgh
EH21 6UU

Dear Dr Santos

Professional Doctorate Research Study - Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders: Amendment to Recruitment Process

Further to my letter of 21/03/12 and 02/07/2012 informing you of the minor changes as requested by the DivREC student project release form (16/3/12) for the above project, I would like to inform you of the following planned expansion to my recruitment process for the main Delphi study phase:

1. Approach individual health boards to recruit medical consultants (previously stated in letter 02/07/2012). Clarification - This will also require contacting individual consultants e.g. with a view to circulating research call for participants within their health boards (Scotland initially. North England if needed) or if identified through publications as having a specialist interest in the subject matter.

2. Letter dated 02/07/2012 stated: Contact the Society of British Neurological Surgeons and Irish Institute of Clinical Neuroscience to request circulation of research call for participants to their respective membership. This option has not been used to date; however, this strategy should be extended to include the Scottish Association of Neurological Sciences and the British Association for the Study of Headaches. These organisations include the same target professional groups of consultant neurosurgeons and consultant neurologists.

I hope the expanded recruitment process meets with your considered approval. The project methodology remains unchanged.

Yours sincerely

Colin Redmond
Prof Doc Student 09001905

Appendix H: Ethical approval letter to minor amendments

Tuesday, 23 October 2012

Dear Mr Redmond

I would like to inform you that the DivREG committee has approved the minor amendments to your original proposal outlined below in your letter.

Your sincerely

[Signature]

Dr Derek Santos
Senior Lecturer
Appendix I: Focus group recruitment call information

Dear Colleague
Would you like to help develop a red flag screening tool for musculoskeletal neck pain and headache?

I would like to invite you to participate in a focus group alongside other leading UK based physiotherapists. The group will consist of approximately 6 – 10 participants through video link or attendance (Edinburgh).

Provisional date/time: Wednesday 27th June 2012 1.45pm - 3.45pm.

Why?
• To promote early detection of potentially serious pathology mimicking as a cervical spine musculoskeletal disorder.
• To reduce the risk of adverse events, e.g. transient ischaemic attacks and stroke, occurring from physical treatments.
• Adverse events of this nature have been reported in international peer-reviewed literature.
• It is vitally important that any underlying serious complaints have been excluded in order to apply safe and effective physical treatment.

My name is Colin Redmond. My professional role is Principal Spinal Physiotherapist with NHS Borders and I am currently undertaking a Professional Doctorate programme with Queen Margaret University, Edinburgh. This research forms part of my Professional Doctorate studies. During the redevelopment of a regional spinal service it became apparent that some clinical indicators used by the physical therapy professions to identify potentially serious pathology presenting as, or alongside, a cervical spine musculoskeletal disorder were not reliable.

Whilst red flags for musculoskeletal low back pain have been developed and widely accepted, there is a need to review more specific red flag indicators or diagnostic screening for neck related pain or functional impairment. Your help is needed to enhance safe practice.

This research project combines a physiotherapy focus group and a Delphi survey (see information sheet) with medical experts (consultant neurologists) to develop an evidence-based screening tool for serious pathology in cervical spine musculoskeletal disorders. Some initial literature summaries prepared by me will be provided.

If you would like to become involved or receive a more detailed information sheet, please contact me: email 09001905@qmu.ac.uk or Colin.Redmond@borders.scot.nhs.uk or telephone: 01896 827004. Thank you.
Appendix J: Participant Information Sheet – Focus Group

Study Title

Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders

You are invited to take part in a research project that aims to improve physiotherapy red flag screening for serious pathology in cervical spine musculoskeletal disorders. Before you decide it is important for you to understand why the research is being undertaken and what it will involve. Please take time to read the following information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

What is the purpose of the study?
The study is being carried out to establish a red flag screening process for serious pathology presenting as, or alongside, a cervical spine musculoskeletal disorder, which can be used by physiotherapists, but additionally will inform chiropractic osteopathy, and general practitioners' practice. This could enhance early detection of potentially serious pathology and enable safer practice. Clinical experience supported by a literature review has identified a knowledge gap and inconsistencies in screening for neurological and neurovascular pathology. Red flag screening in low back pain has received significant attention by researchers; however specific questions and testing for the cervical spine remain inconsistent e.g. cervical arterial dissection has been reported following manipulation, but screening processes to reduce the potential of adverse incidents occurring remains unclear. Similarly, there is inconsistent evidence to support the diagnostic accuracy of clinical tests for signs of cord compression myelopathy. This study is looking to establish a screening process through the combination of literature review, focus group with leading physiotherapists, and a Delphi method with consultant neurologists that will be used to inform clinical practice and contribute to the evidence base for future research. Main findings from the literature review will be forwarded in advance to focus group participants. The combination of literature review and focus group discussion will be used to inform the Delphi phase. The Delphi technique is an iterative process used to collate expert opinion over a series of sequential rounds, which are interspersed by feedback from the previous round and is considered as a suitable method to gain a consensus where there is incomplete knowledge or uncertainty in clinical issues.

Why have you been selected?
You have been offered the opportunity to participate in this study as your expertise as a physiotherapist with relevant expertise is considered as highly valuable in contributing to the knowledge base that will inform clinical practice. Participants are being recruited from physiotherapists of: consultant level, specialist spinal roles, those with relevant peer-reviewed published papers, or MSc neuromusculoskeletal / manual therapy (post-graduate) level educators.

Do I have to take part?
No. It is up to you to decide whether or not you take part. However, in addition to help progress clinical practice, the study also offers an excellent professional development opportunity. Your clinical expertise is considered as very valuable to the study. Consent to participate in the study will be gained through email. If you decide to take part you are still free to withdraw at any time and without giving a reason.
What will happen to me if I take part?
By agreeing to take part in the study you will participate in a focus group with other UK based physiotherapists, however, you should not feel obliged to participate in all aspects of the discussion. The discussion will be conducted through a combination of video link or attendance at Queen Margaret University, Edinburgh. In preparation for the focus group you will receive a summary of the literature review that will part inform the discussion. The aim of the focus group is to identify key components for inclusion in a red flag screening process and discuss any main inconsistencies in the literature. Points for consideration may be: clinical features, risk factors, screening questions, and clinical tests related to screening for potentially serious neurological/neurovascular pathologies. The focus group discussion will be recorded. This information will be analysed by the principal investigator and combined with the evidence based literature review to develop a draft screening process to use in a Delphi phase of the study. The focus group is expected to last approximately 1 ½ - 2 hours and you are requested to ensure that you have relevant local authority / permission to participate in the study.

What are the risks of taking part?
There is no identifiable risk of taking part in this study.

What if something goes wrong?
This study is considered as low risk. Regardless of this, if you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, you may raise this concern directly with the principal investigator who will follow Queen Margaret University, Edinburgh complaint escalation procedure. Alternatively, you are free to contact the Independent Advisor.

Will my taking part in this study be kept confidential?
All information which is collected about you will be kept strictly confidential and managed in accordance with the Data Protection Act (1998). The group discussion will be recorded on an .mp4 file and downloaded and transferred from the secure JANET server (videolink) to a secure NHS encrypted information systems by an information systems expert from Queen Margaret University. Thereafter, the file will be handled by the principal investigator. This recording will be destroyed after being transcribed for analysis. You will then be assigned a study number which is linked to your name and stored in NHS encrypted information systems. Any information which leaves the NHS information system will have your name and contact details removed so that you cannot be recognised from it.

What will happen to the results of the research study?
The results of this study will be used to present a thesis that will be submitted as part fulfillment of a Professional Doctorate in Health and Social Sciences at Queen Margaret University, Edinburgh. The results will also be used to publish and present the findings of the research in a relevant journal and conference to inform clinical practice that promotes patient safety. However you will not be identified in any report or publication, unless you provide additional consent to do so.

The results will be submitted in a thesis format in June 2013 and you can obtain a summary from the principal investigator (details at the end of this sheet).

Who is organising and funding the research?
The study is being organised by the principal investigator who is completing the research as part of a higher academic degree as outlined above. Queen Margaret University, Edinburgh has approved the study and some time and financial support for this research has been obtained from NHS Borders and NHS Education for Scotland.
Who has reviewed the study?
The study protocol has been reviewed and conduct of the study has achieved Queen Margaret University, Edinburgh ethical approval. Recent changes to NHS Governance Arrangements for Research Ethics Committees states that additional NHS approval is not required. However, South East Scotland Research Ethics Service (NHS) has been informed of the study.

Contact for further information
If you would like any further information about this study please ask the Principal Investigator: Colin Redmond

Address: Physiotherapy Department
Borders General Hospital
Melrose
TD6 9BS

University email / work telephone: 09001905@qmu.ac.uk 01896 827004/826548

Alternative: Work email: Colin.Redmond@borders.scot.nhs.uk

If you have questions relating to any aspect of the study that you would like independent advice on, you will be able to speak to.

Independent Advisor: Professor James M Scobie
Director, CASL (Clinical Audiology, Speech and Language) Research Centre
Queen Margaret University
Edinburgh
EH21 6UU

Email JScobie@qmu.ac.uk
Telephone 0131 474 0000 (state name of person)

If you decide to take part in this study, please retain a copy of the information sheet for your own records and thank you for taking part in this project.
Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders

I have read and understood the information sheet and this consent form. I have had an opportunity to ask questions about my participation.

I understand that the focus group will be recorded.

I understand that I am under no obligation to take part in this study.

I understand that I have the right to withdraw from this study at any stage without giving any reason.

I have permission from my employer to participate in this study.

I agree to participate in this study and understand that returning this form to the researcher's university email address, using my personalised work email address will be considered as providing informed consent.

Name of participant:

Date:

Please copy/paste this completed form to your work email address and return to the researcher's university email address. Thank you.

Contact details of the researcher

Name of researcher: Colin Redmond

Address: Physiotherapy Department
Borders General Hospital
Melrose
TD6 9BS

University email / work telephone: 09001905@qmu.ac.uk 01896 827004/826548

Alternative work email: Colin.Redmond@borders.scot.nhs.uk
Appendix L: Focus group information pack (part-presented only)

Professional Doctorate in Health and Social Sciences

Research Project

Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders

Focus Group Information Pack

Wednesday 27th June 2012
13.45

Facilitator: Colin Redmond MSc MMACP MCSP
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<th>Page No.</th>
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<td>Introduction</td>
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</tr>
<tr>
<td>Technical glossary</td>
<td>4</td>
</tr>
<tr>
<td>1. Focus group outline construction plan</td>
<td>5</td>
</tr>
<tr>
<td>2. Literature review – grading evidence</td>
<td>6</td>
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<td>2.1. SIGN Guidelines</td>
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<tr>
<td>2.2. Statistical terminology</td>
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<tr>
<td>3. Current status on Red flags</td>
<td>8</td>
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Introduction
This information pack primarily consists of a range of literature summaries designed to inform participants in preparation for a focus group that forms part of this research project. The study design aims to review and develop our current red flag screening processes for potential serious pathology presenting as, or alongside, musculoskeletal cervical spine and headache presentations. There are examples of excellent clinical development, specifically, cervical arterial dysfunction (e.g. Taylor and Kerry 2010; Kerry et al 2008; Kerry and Taylor 2009; 2006; ongoing IFOMPT project). This project does not aim to replace such examples, but to contribute to their development, consider other areas of clinical uncertainty, and combine these clinical areas to sit within one cervical spine red flag screening system for ease of access. Due to the broad spectrum of pathologies that could potentially present alongside cervical spine disorders it is outwith the scope of this discussion group to review all areas. Therefore, the focus group discussion and information pack will primarily focus within cervical arterial dysfunction, headache, dizziness, and cervical myelopathy. Where applicable, other pathologies may be explored, and a later section identifies some additional considerations.

This document provides initial information on the focus group outline, grading quality of literature, current status on red flags, before progressing to section 4 that contains a number of problems/questions with accompanying literature for your consideration. I appreciate that you are very busy professionals; therefore, you are not expected to dissect all the literature. It is recommended that you initially read to page 13, and then consider the individual problems/questions contained within section 4. It is advisable not to attempt section 4 in one sitting, but to split your allocated time accordingly. The reader can then explore any statistics in more detail, as required, or simply consider how some of the information may be used in musculoskeletal clinical settings.

Thank you for agreeing to participate in the group discussion. As an experienced clinician, your expertise is considered as highly valuable. You will be familiar with some of the work contained within this document, but equally, I hope you have the benefit of gaining new knowledge within these subject areas. Similarly, if you have relevant articles to contribute to the project, this would also be greatly appreciated.
## Technical Glossary

### Abbreviations

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<td>AE</td>
<td>Adverse events</td>
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<td>CAD</td>
<td>Cervical Arterial Dissection</td>
</tr>
<tr>
<td>CADy</td>
<td>Cervical Arterial Dysfunction</td>
</tr>
<tr>
<td>ICA</td>
<td>Internal Carotid Artery</td>
</tr>
<tr>
<td>ICAD</td>
<td>Internal Carotid Artery Dissection</td>
</tr>
<tr>
<td>PI</td>
<td>Principal Investigator</td>
</tr>
<tr>
<td>sICAD</td>
<td>spontaneous Internal Carotid Artery Dissection</td>
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<tr>
<td>SCD</td>
<td>spontaneous Internal Carotid Artery Dissection</td>
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<tr>
<td>sVAD</td>
<td>spontaneous Vertebral Artery Dissection</td>
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<td>SVD</td>
<td>spontaneous Vertebral Artery Dissection</td>
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<td>VA</td>
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<td>VAD</td>
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<td>VBI</td>
<td>Vertebrobasilar Insufficiency</td>
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1. Focus group outline construction plan

**Focus group**
**Duration:** 1.5 – 2 hours

**Preliminaries**
- Introduction (facilitator)
- Discussion procedures
- Reminders:
  - Participants can withdraw at any time without reason
  - Discussion is recorded for post-analysis
- Outline problem / reason for group discussion
- Introductions (group)

**Topics for Discussion**
As part of the focus group preparations participants have received a series of literature summaries to help inform discussion. The summaries relate to clinical areas where literature on specific clinical topics is considered inconsistent to aspects of current clinical practice:
- What groups of serious clinical pathologies should be included within red flag screening for cervical spine musculoskeletal presentations?
- For each group - What main clinical indicators are suggestive of this clinical group?
- What questions should be asked to help identify or explore the presence of these pathologies?
- What clinical tests (not further investigations) should be utilised to help identify these pathologies?
- Do you have any suggested formats for the presentation of a red flag screening process / algorithm?
- Do you have any additional questions that you feel should be addressed by the expert medical panel?

**Delphi Survey – web-based survey**
The Delphi phase will be constructed following completion of the focus group. The anticipated format will consist of a draft clinical chart/algorithm accompanied by a series of open ended questions, similar to the above outline, to address any clinical areas that remain uncertain. The initial literature review has identified the areas of cervical arterial dissection/dysfunction, including associated symptoms of headache and dizziness, and cervical myelopathy where most clinical uncertainty remains.
2. Literature review – grading evidence

Where applicable, evidence has been graded (single reviewer) using the Scottish Intercollegiate Guidelines Network (SIGN) process for grading evidence.

2.1 SIGN Guidelines: Key to evidence statements and grades of recommendations adapted from http://www.sign.ac.uk/guidelines/fulltext/50/index.html

**LEVELS OF EVIDENCE**

1++. High quality meta-analyses, systematic reviews of RCTs, or RCTs with a very low risk of bias.
1+. Well conducted meta-analyses, systematic reviews, or RCTs with a low risk of bias.
1 -. Meta-analyses, systematic reviews, or RCTs with a high risk of bias.
2++. High quality systematic reviews of case control or cohort studies.
High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal.
2+. Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal.
2 -. Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal.
3. Non-analytic studies, e.g. case reports, case series.
4. Expert opinion.

**GRADES OF RECOMMENDATION**

Note: The grade of recommendation relates to the strength of the evidence on which the recommendation is based. It does not reflect the clinical importance of the recommendation.

A: At least one meta-analysis, systematic review, or RCT rated as 1++, and directly applicable to the target population; or
A body of evidence consisting principally of studies rated as 1+, directly applicable to the target population and demonstrating overall consistency of results.
B: A body of evidence including studies rated as 2++, directly applicable to the target population, and demonstrating overall consistency of results; or
Extrapolated evidence from studies rated as 1++ or 1+.
C: A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results; or extrapolated evidence from studies rated as 2++.
D: Evidence level 3 or 4; or extrapolated evidence from studies rated as 2+.

**GOOD PRACTICE POINTS**

✓ Recommended best practice based on the clinical experience of the guideline development group.

References not provided within summaries are located in the reference section.
2.2 Statistical terminology

Some key statistical terms have been provided as a refresher to assist evaluation of literature.

**Odds ratio (OR):** The odds of an event are the probability of occurrence over the probability of non-occurrence. OR is used to compare if the probability of an occurrence is the same for two groups. A value of 1 indicates that the occurrence is equally likely in both groups. An OR value >1 indicates that the event is more likely in the first group. A value of <1 indicates that that the event is less likely in the first group (Altman et al 2006; Munro 2001).

**Positive likelihood ratio (LR+):** The ratio of a positive test result of the people with the pathology to a positive test result in people without the pathology (Cook et al 2011).

**Negative likelihood ratio (LR-):** The ratio of a negative test result of the people with the pathology to a negative test result in people without the pathology (Cook et al 2011).

Jaeschke et al (1994 in Cook et al 2011) provides guidance: LR+ should be ≥5 to moderately influence post-test probability and clinical decision making. LR- should be ≤0.20. Small influences in post-test probability involve LR+ of ≥2.0 and LR- of ≤0.50.

**Sensitivity:** percentage of positive tests in individuals with the pathology. Tests with low sensitivity are not likely to identify the condition early in an examination (Cook et al 2011).

**Specificity:** percentage of a negative test result in individuals without the pathology (Cook et al 2011).

**Confidence intervals (95%CI):** Most studies estimate a quantity or risk. The CI is a range that is 95% confident it includes the true value. CI reveals the precision of an estimate. A wide CI points to lack of information, whether the difference is statistically significant or not, and is a warning against over interpreting results from small studies (Altman et al 2006).


3. Current status on ‘Red flags’

3.1 The Clinical Standards Advisory Group (1994 in Greenhalgh and Selfe 2006) red flag list (below) was developed for low back pain. In addition, Greenhalgh and Selfe (2010, 2006) have provided a weighted flag structure to assist gauging the clinician’s index of suspicion of potential serious pathology. This list does not provide sufficient information for the cervical spine:

1. Age of onset <20 or >55 years.
2. Violent trauma
3. Constant, progressive, non-mechanical pain.
4. Thoracic pain.
5. PMH of carcinoma
7. Drug abuse, HIV.
8. Systemically unwell.
9. Weight loss.
10. Persistent severe restriction of lumbar flexion.
11. Widespread neurology
12. Structural deformity.

With reference to specific red flags for the cervical spine, the health information website Patient.co.uk, which reports itself as “one of the most trusted medical resources in the UK, supplying evidence based information on a wide range of medical and health topics to patients and health professionals”, provides a list of red flags for neck pain. A section on ‘cervical radiculopathy’ cites the Clinical Knowledge Summaries (CKS: Jan 2009) as it’s source. CKS is a National Institute for Health and Clinical Excellence site (no longer maintained: http://www.cks.nhs.uk/neck_pain_cervical_radiculopathy. Follow link for red flags), and provides a number of references. Binder (2007) is the most relevant reference. This article provides a poorly referenced list of red flags.

Patient.co.uk provides the following red flag list, which the reviewer considers as a reasonable group (http://www.patient.co.uk/doctor/Cervical-Disc-Protrusion-and-Lesions.htm). However, are the tests and clinical indicators reliable or of sufficient detail to inform clinical practice?

Red flags for neck pain

* A serious underlying cause is more likely in people presenting with:
  - New symptoms before the age of 20 years or after the age of 55 years.
  - Weakness involving more than one myotome or loss of sensation involving more than one dermatome.
  - Intractable or increasing pain.

Red flags suggesting possible malignancy, infection or inflammation:
  - Fever
  - Unexplained loss of weight
  - History of inflammatory arthritis
  - History of malignancy, drug abuse, tuberculosis, AIDS, or other infection
  - Immunosuppression
- Pain that is increasing, unremitting and/or disturbs sleep
- Lymphadenopathy
- Exquisite localised tenderness over a vertebral body

Red flags suggesting myelopathy (compression of the spinal cord):
- Insidious progression
- Gait disturbance; clumsy or weak hands; loss of sexual/bladder/bowel function
- Lhermitte's sign (flexing the neck causes electric shock-like sensations that extend down the spine and shoot into the limbs)
- Upper motor Neuron signs in the lower limbs (Babinski's sign — up-going plantar reflex, hyperreflexia, clonus, spasticity)
- Lower motor Neuron signs in the upper limbs (atrophy, hyporeflexia)
- Variable sensory changes (loss of vibration and joint position sense more evident in the hands than in the feet)

Red flags suggesting severe trauma/skeletal injury:
- History of trauma
- Previous neck surgery
- Osteoporosis or risk factors for osteoporosis
- Increasing and/or unremitting pain

Red flags suggesting vascular insufficiency:
- Dizziness and blackouts (restriction of vertebral artery) on movement, especially on extension of the neck with upward gaze
- Dizziness, drop attacks

Current status on red flags for headaches (SIGN guidelines) is contained in section 4.

3.2 Cochrane library: 2 publications on red flag screening on lumbar spine. No reviews on cervical spine.

3.3 Traditional physiotherapy screening
3.3.1 ‘Classical’ cardinal signs and symptoms (Vascular and spinal cord)

Rivett et al (Australian guidelines - 2006 p.3) recommends:

“...In every patient presenting with upper quadrant dysfunction, questioning is specifically directed to determine the presence of dizziness which is the most common presenting symptom of VBI. If dizziness is present, other symptoms associated with VBI should be sought...”

No references are provided to support these cardinal signs and symptoms, which is a similar list referred to by Kerry and Taylor (2006 p.244-245) as “Classically, signs and symptoms related to hindbrain ischaemia are considered as the “5 Ds and 3Ns” of Coman (Coman 1986). This list is Dizziness (vertigo, giddiness, lightheadedness), Drop attacks, Diplopia, Dysarthria, Dysphagia (+ hoarseness/hiccups), Ataxia, Nausea, Nystagmus, Numbness (unilateral). Coman’s (1986) list of cardinal signs and symptoms is not referenced. However, unreasoned adherence to this list may result in an
incomplete understanding of the patient’s presentation (Taylor and Kerry 2010; Kerry and Taylor 2009; 2006; Kerry et al 2008), and in a commissioned report, Kerry et al (2007) state there is no support for Coman’s 5 Ds.

Patten (1998) suggests “differential diagnosis of brainstem disease is complicated by a specific feature of symptomatology – all common symptoms e.g. diplopia, dysarthria, vertigo, nausea and vomiting are by their very nature acute” p.171-2. This highlights the difficulty physiotherapists face in clinic when these questions are asked i.e. how relevant are they when we perform our screening, specifically relative to duration of onset and what pathology is indicated?

Thiel and Rix (2005) also produce a list using the “5D’s and 3N’s framework” whilst stating ‘adapted from Sturzenegger (1993) Saeed et al (2000)’. These references are omitted from Thiel and Rix’s (2005) reference list. However, these papers cited are small studies with patients presenting at neurological units with stroke. Other than one patient presenting with recurrent TIAs, signs and symptoms, such as the 5D’s and 3 N’s framework mainly formed part of the presenting features i.e. at development of stroke rather than early warning signs. Additionally, in referring to a variation of this framework Greenhalgh and Selfe (2006) cite Magarey et al (2004) who in-turn cite Grad and Baloh (1989 cited by Clendaniel 2000). Greenhalgh and Selfe (2006) also cite Grant (1994) regarding a variety of signs and symptoms resulting from vertebrobasilar dysfunction. Grant (1994) cites Williams and Wilson (1962). Therefore, it is questionable whether the 5D and 3N framework is an appropriate screening process in neck pain/headache presenting at musculoskeletal clinical setting. Taylor and Kerry (2010), Kerry and Taylor (2009; 2006) and Kerry et al (2008; 2007), are the only authors to question the 5Ds and 3Ns framework through a literature review approach.

The clinical features of myelopathy can overlap with a radiculopathy problem and spinal cord pathologies, such as multiple sclerosis and amyotrophic lateral sclerosis may have similar presenting signs and symptoms that may include upper and lower motor neuron signs, pain, paraesthesia, functional impairment (Cook et al 2007). This presents a significant challenge to practitioners to ensure correct differential diagnosis is obtained with subsequent selection of safe management strategies.
3.2 Problem: Terminology “CAD”
Medical literature and some physiotherapy articles e.g. Thomas et al (2011) use the acronym of “CAD” to represent cervical arterial dissection, which mainly occurs in a younger population. This pathology does not include atherosclerotic causes of stroke, although there can be some overlap. In contrast, Taylor and Kerry (2010), Kerry and Taylor (2009, 2006), and Kerry et al (2008) use the same acronym to represent cervical arterial dysfunction, which encompasses the anatomical and pathological spectrum of events. Such pathologies may be “…transient mechanical occlusions, dissections (intimal tearing), to frank atherosclerotic thromboembolic events leading to stroke” (Kerry and Taylor 2009 p.378). These pathologies may include “…the posterior system (vertebrobasilar) supplying blood to the hindbrain the anterior arterial system (internal carotid arteries) supplying blood to the cerebral hemispheres and eye…” (Kerry and Taylor 2009 p.378).

Identifying this distinction in terminology assists our evaluation of research. An example is Thomas et al (2011) who question Kerry et al’s (2008) recommendation of including a vascular risk assessment of patients prior to therapeutic management of neck dysfunction. The reviewer’s personal opinion is that this debate is arising due to considering two different interpretations of vascular pathology i.e. one is focusing on very specific problem (dissection), whereas Kerry et al’s (2008) emphasis is on the spectrum of vascular events. Therefore, for the purpose of this literature pack the following terms will be used:
Cervical arterial dissection (CAD)
Cervical arterial dysfunction (CADy)

From personal experience, the clinical reasoning or screening process should encompass the dysfunction approach, as clinicians’ primary interest at the initial stage is to identify risk or exclude a potentially serious vascular event occurring, regardless of the underlying pathology e.g. dissecting artery versus an atherosclerotic occlusion.

Additional: Kerry and Taylor (2006) p.249 “In addition to the early signs, it is important to be aware of signs and symptoms related to cerebral and retinal ischemia. It is unlikely that a patient with full stage cerebral ischemic stroke will present to the manual therapist, but the more subtle presentation of retinal ischemia might, which makes simple eye examination a key part of the assessment”. Cerebral or retinal ischemia includes ischemic stroke, TIA, or transient monocular blindness (Debette et al 2011). Thomas et al (2011) consider that more appropriate therapeutic risk management can
be applied if individuals with intrinsically enhanced risk of dissection can be more readily identified (refer to discussion point section 4.4.3. Risk factors: Cardiovascular).

3.3 Pain (Vascular)

Neck pain and/or headache symptoms are the most frequent local symptoms of CAD (Thomas et al 2011; Morelli et al 2008; Rigamonti et al 2008; Tobin and Flitman 2008; Hardmeier et al 2007a; Arnold et al 2006a; Taylor and Kerry 2006, 2005; Savitz and Caplan 2005; Silbert et al 1995), and additionally, can be the only presenting symptoms (Arnold et al 2006a; Guillon et al 1998; Biousse et al 1994; Biousse et al 1992). Furthermore, VAD has also been reported as presenting as a fifth cervical nerve root (C5) radiculopathy (Hardmeier et al 2007b). Note, Rivett et al (2006) stated that dizziness was the most frequent symptom. However, they also advise to check for presence of neck pain or headache.

For VBI, Rivett et al (2006 p.3) cite Haldeman et al (2002) and Krespi et al (2002) to add that pain is “Specifically, sudden, severe, sharp pain located in the ipsilateral postero-superior region of the neck and occiput and for which there is no past history should be regarded as suspicious”. This does not include ICAD. Acute onset neck pain/headache and has been described as “unlike any other” (Taylor and Kerry 2010; Kerry and Taylor 2009; 2006; Kerry et al 2008), but are these patterns, distributions and descriptions definitive in CAD or are they the severe end of the headache spectrum? This problem is explored in section 4. Neck pain and headache are symptoms that may present in patient presentations at musculoskeletal clinics without prior attendance at a medical practitioner, or could be the reason for referral from the medical practitioner.

3.4 Pain (cervicogenic headache)

Cervicogenic headache is considered a disorder that is manageable by the physical therapies (Jull 1997). This disorder has been recognised by the International Headache Society (IHS 2004). Part of the criteria referred to by the IHS is pain referred from a source in the neck and felt in one or more regions of the head and/or face. Differential diagnosis of cervicogenic headache, however, can be difficult to separate from other causes of headache unless additional features are presented. Some physiotherapy studies have attempted to address this differential diagnosis problem by comparing examination findings in headaches groups of cervicogenic and migraine with aura, and asymptomatic controls and have identified upper cervical spine mechanical dysfunction (Ogince et al 2007; Zito et al 2006). The most crucial differential diagnosis is headache
from CAD due to the heightened risk of adverse events with potentially near fatal consequences following manipulation (Bogduk and Govind 2009).

An additional problem with headache differentiation is that neurologists differ in their agreement of cervicogenic headache as having a nosological identity making the concept of cervicogenic headache controversial (Bogduk and Govind 2009; Zhou 2008; Leone et al 1998). This adds greater complexity to clinicians navigating their clinical reasoning processes when presented with patient complaining of neck pain and headache.

The publications in section 3 referring to signs and symptoms, such the ‘5Ds and 3Ns framework’, myelopathy and headache presentations highlights the difficulty therapists face in clinic when considering differential diagnosis. This creates the question of what subjective and objective procedures should musculoskeletal practitioners use to support awareness of detecting more subtle aspects of neurological change in order to prevent occurrences of an adverse event?

The following sections contain a number of clinical problems based on section 3. The problems focus on areas where the reviewer considers that most clinical uncertainty exists. These problems are cervical arterial dysfunction with associated symptoms of headache and dizziness, and cervical myelopathy. The problem areas are accompanied by literature summaries in various formats to help inform participants. The selection of literature is from the best identified literature following guidance from the SIGN methodology. Single-case studies are excluded within the main problems. Case series of minimum n= 5 have been selected, where literature is limited. Any review literature is a systematic process or accompanies another study (e.g Caplan et al 2005), unless otherwise indicated by appropriate SIGN grading.
4. Clinical problems with accompanying literature

**Problem 4.1:** What signs and symptoms indicate the presence of CADy? Studies appear to be based in neurological centres; therefore it is questionable as to how relevant the findings are to MSK clinics? Admission to neurological centres indicates significant progression of an arterial event. Taylor and Kerry (2010 masterclass) provide excellent information on CADy from a manual therapy perspective. However, the following studies highlight the difficulty in identifying features relevant to MSK clinics. Other than single case reports, there are no identifiable studies within MSK settings (excluding adverse events based reports).

<table>
<thead>
<tr>
<th>Study</th>
<th>Study design</th>
<th>Evid. level</th>
<th>Sample size</th>
<th>Population characteristics</th>
<th>Clinical presentation</th>
<th>statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Debette et al (21). 2011b. Differential features of carotid and vertebral artery dissections. The CADISP study. Neurology, 77: 1174-1181.</td>
<td>Large observational (20 centres, 9 countries 2004-2009)</td>
<td>2++</td>
<td>ICAD n=619 VAD n=327</td>
<td>ICAD: age 45.7±9.6 yrs Women n=245 (39.6%)  VAD: age 41.1±9.6 yrs Women n=245 (39.6%)</td>
<td>1. cervical pain 2. Headache 3. Cerebral ischemia a. Ischemic stroke b. TIA c. transient monocular blindness d. Subarachnoid haemorrhage</td>
<td>ICAD Vs VAD 1. ICAD n=231 (38.7%); VAD n=212 (66%) p=0.001; OR(95% CI) 0.36 (0.27 to 0.48). 2. ICAD n=405 (67.8%); VAD n=207 (64.5%) p=0.3; OR(95% CI) 1.36 (1.01 to 1.84). 3. ICAD n=453 (73.2%); VAD n=295 (90.2%) p=0.0001 OR(95% CI) 0.32 (0.21 to 0.49).</td>
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Reviewer comments: This report forms part of the large multicentre Cervical Artery Dissection and Ischemic Stroke Patients (CADISP) study. Excellent large study with this report presenting differential features of carotid and vertebral artery dissections. Unfortunately, does not offer specific detail on aspects of the pain presentation or ischemic signs. This study is presented to highlight the difficulty in establishing patterns of presenting features to assist MSK practitioners. Neck pain is more likely in VAD, headache more likely in ICAD, but does this matter from a clinical perspective i.e. can we differentiate between the two and would it alter your management?
<table>
<thead>
<tr>
<th>Study</th>
<th>Study design</th>
<th>Evid. level</th>
<th>Sample size</th>
<th>Population characteristics</th>
<th>Clinical presentation</th>
</tr>
</thead>
</table>

Authors’ general comments:“Considering (clinical recommendations e.g Rivett et al 2006; Kerry et al 2007), …headache not always present or severe in either VBAD or ICAD subjects, although was more common in VBAD (85%) and ICAD (75%) subjects than controls (51%)” p.355.(Reviewer comments: this latter point differs to Debette et al (2011) headache in ICAD > VAD). Similarly, neck pain and dizziness more likely in VBAD than ICAD subjects or controls. Dizziness present in only 52% VBAD cases, yet often stressed as primary clinical indicator of vertebrobasilar flow insufficiency. The presence or absence of nystagmus rarely recorded, although other visual disturbances were reported. Ataxia or balance problems were fairly common in VBAD group (67%). Recommend being aware of in assessment of neck pain or headache and perhaps consider formal testing of balance more routinely. Similarly findings for
ICAD suggest it may be appropriate to perform a focused cranial nerve examination (e.g. for facial palsy or ptosis) if specific symptoms are reported in the history or signs are evident on casual observation. (reviewer comments: cranial nerve testing recommended elsewhere (e.g. Taylor and Kerry 2010: masterclass article). Do participants or your departmental colleagues currently consider balance testing in neck pain/headache?

Limitations of retrospective studies: medical records not always detailed and it is acknowledged that negative responses to questions in the history may not always be recorded. Details of blood results and radiological imaging were sometimes limited.
Problem No. 4.4  Pre-manipulative screening, including provocative positional tests for vascular pathology, does not identify all patients at risk of an adverse event from therapeutic intervention. Therefore, other factors, such as identifying risk factors should be identified to assist in process. Unfortunately, there is inconsistency in the literature regarding what risk factors should be considered for subjective screening of CADy.

4.4.1 Theoretical modelling

Thomas et al (2011) consider that a consensus has not been achieved on definitive risk factors for CAD. Additionally, there is inconsistent evidence between studies investigating risk factors for CAD (Thomas et al 2011; Kerry et al 2008).

Taylor and Kerry (2010) consider CAD to be intrinsically linked to two inter-related principles of an underlying pathology (including atherosclerosis) and mechanical forces. Other features linking to underlying pathology or risk have been suggested as: genetic (e.g. connective tissue disorders, fibromuscular dysplasia (non-inflammatory disease of medium sized arteries), or family history) and environmental origins (e.g. major and minor trauma, infections, smoking, hypertension, oral contraceptives, iatrogenic causes such as, surgery or medical intervention) are also considered a risk (Debette and Leys 2009; Thanvi et al 2005).

Risk ratio – Kerry et al (2008) conclude that the actual risk cannot be determined from the available evidence and as a result, advise that clinicians should consider risk based on their clinical reasoning including assessment of the patient relative to their particular situation at a particular time

In an attempt to aid understanding and clinical reasoning of the pathogenesis of CAD Rubinstein et al (2006) present a theoretical model that outline four major risk categories of CAD: 1) genetic predisposition/underlying familial disorder; 2) environmental exposure e.g. infection, oral contraceptive; 3) trivial trauma e.g. normal neck movements, sports injury, neck manipulation; and 4) common risk factors associated with atherosclerosis. Rubinstein et al (2006) propose that a genetic predisposition must be present, plus an additional necessary trigger for a CAD event to occur. They argue that a CAD is highly unlikely in an otherwise healthy individual free from this combination of factors. In a thorough review focusing on sCAD Debette and Leys (2009) concluded that studies on genetic association with CAD have been underpowered.

The difficulties with studies investigating risk factors and presentations are that patients with local signs only (e.g. pain) or with minor cerebral or retinal ischemia changes may not be detected dependent on where and how they are managed (Debette et al 2011).

4.4.2 Manual therapies and minor trauma (note this is the subject of a current academic discussion in the BMJ)

Debette et al (2011a) observational study 2004-2009 20 centres in 9 countries observed 946 consecutive patients in with CAD (n=619 ICAD and n=327 VAD). The following were found to have experienced minor mechanical trauma defined as not requiring a medical visit attendance at a hospital, within the previous 1 month: ICAD n=177 (29.2%) and VAD (n=118 (36.5%) p=0.02 (adjusted for univariate) OR (95%
CI) 0.75 (0.56 to 1.007) p=0.05 (adjusted for age / gender). Major trauma defined as requiring a medical visit or attendance at a hospital did occur, but did not achieve significance (p=0.87 adjusted for age/gender). No specific detail of trauma type is provided. Thomas et al (2011) found a statistically significant association between minor mechanical trauma and CAD patients compared to a control group: 23 (64%) vs 3(7%) OR (95% CI) 26.67 (6.83 to 104.17) adjusted OR 25.29 (6.04 to 105.82) p<0.000. The minor trauma occurred within the previous 3-weeks and included manual therapy (chiropractic, osteopathy, physiotherapy, massage) to the neck; however, what type and extent of therapy was not recorded.

Miley et al (2008) conducted a detailed structured evidence based clinical neurologic practice review to address the question “Does cervical manipulative therapy cause vertebral arterial dissection and subsequent ischemic stroke?” and conclude there is weak to moderately strong evidence to support causation between CMT and VAD and associated stroke. Detailed search strategies are provided, which was reduced to 26 highest levels of evidence publications: 3 case-control studies, 8 prospective and retrospective case series, 4 illustrative case reports, 1 survey, 1 systematic review of observational research, 5 reviews, and 4 opinion and expert commentary pieces. A cross reference was conducted with this reviewer’s search results, which revealed the same main studies.

Ernst (2010) conducted a systematic review with no time limit to establish the numbers of fatalities following chiropractic intervention and reported a total of 26 deaths were published in the medical literature, but further states there is reason to believe that under-reporting is substantial and reliable incidence figures do not exist. Albuquerque et al (2011) reported a case-series of 13 patients (8 male, mean age 44, range 30-73 years) extracted from a prospective endovascular database who presented with craniocervical dissections following chiropractic manipulation. Symptoms of neurological deficit, head or neck pain, or both had atypical onset within hours or days of manipulation. In this case series 31% (n=4) were left permanently disabled or died as a result of their arterial injuries.

Haldeman et al (2002) concluded after reviewing 64-medical/legal (medical related compensation claims) cases that risk factors could not be identified and that dissection was an unpredictable event. Manipulation of the neck, however, is considered as having a strong association as a risk factor for CAD (Ernst 2007; Rubenstein et al 2005). Murphy (2010) offers a commentary to provide a balanced view of cervical manipulation and arterial dissections and suggests that the relationships is not causal, but that patients often seek chiropractic intervention for some initial symptoms of dissection in progress that may proceed to stroke following manipulation. Ernst (2010), however, reports that even taking this argument into consideration, causality is at least likely, which alongside the limited evidence to support the effectiveness of chiropractic manipulation this makes the risk-benefit balance for chiropractic manipulation less positive.

Rubinstein et al (2006) acknowledge that based on poor methodological processes identified in a systematic review (Rubinstein et al 2005), the true risk of CAD to the population remains unknown. Therefore, if manipulation could be a contributing factor, as opposed to a principal cause of CAD, the problem of identifying a young/middle aged person at risk from CAD still remains. This provides a challenging problem to clinicians – how do we identify these patients?

(Thesis note: due to the size of full information pack this is not included within thesis appendices)
Appendix M: Draft Clinical Chart

Electronic version - Embedded pdf.

Draft Clinical Chart

Hard copy – refer to CD inside rear cover.
Appendix N: Research call for Consultant Neurologists and Consultant Neurosurgeons

Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders

Dear Consultant

Would you like to help develop a red flag screening tool for musculoskeletal neck pain and headache?

Why?
- To promote early detection of potentially serious pathology mimicking or presenting alongside a cervical spine musculoskeletal disorder.
- To reduce the risk of delayed diagnosis, for example, cervical myelopathy, or adverse events, such as transient ischaemic attacks and stroke occurring from physical treatments (e.g. manipulation).
- Delayed diagnosis or adverse events of this nature have been reported in international peer-reviewed medical literature.
- It is vitally important that any underlying serious complaints have been excluded in order to apply safe and effective physical treatment.

Consultant neurologist and neurosurgeon expertise is needed to help address some uncertainty relating to clinical indicators and tests for serious pathology that may mimic or be a deteriorating cervical spine musculoskeletal problem.

My name is Colin Redmond. My professional role is Principal Spinal Physiotherapist with NHS Borders and I am currently undertaking a Professional Doctorate programme with Queen Margaret University, Edinburgh. This research forms part of my Professional Doctorate studies. During the redevelopment of a regional spinal service it became apparent that some clinical indicators used by the physical therapy professions to identify potentially serious pathology presenting as a musculoskeletal disorder were not reliable. Musculoskeletal practitioners (physiotherapists, chiropractors, osteopaths) provide assessment and treatment for pain and functional impairments relating to neck pain and headache. Your help is needed to enhance safe practice.

This research project combines a focus group with leading UK physiotherapists and a Delphi survey with medical experts (consultant neurologists/neurosurgeons) to develop an evidence-based screening process for serious pathology in cervical spine musculoskeletal disorders. A draft clinical screening chart will be provided for your comments alongside some open questions to address any remaining clinical uncertainties. There will be 3 rounds of on-line survey (October and December 2012 and January/February 2013) aimed at achieving a clinical consensus.

Your participation will be highly valued. If you would like to become involved or receive a more detailed information sheet, please contact me: email 09001905@qmu.ac.uk or Colin.Redmond@borders.scot.nhs.uk, or telephone: 01896 827004.

Thank you.
Appendix O: Participant Information Sheet – Delphi Survey

Study Title

Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders

You are invited to take part in a research project that aims to improve physiotherapy red flag screening for serious pathology in cervical spine musculoskeletal disorders. Before you decide it is important for you to understand why the research is being undertaken and what it will involve. Please take time to read the following information. Take time to decide whether or not you wish to take part.

Thank you for reading this.

What is the purpose of the study?
The study is being carried out to establish a red flag screening process for serious pathology presenting as, or alongside, a cervical spine musculoskeletal disorder, which can be used by physiotherapists, but additionally will inform chiropractic, osteopathy, and general practitioners’. This could enhance early detection of potentially serious pathology and enable safer practice. Clinical experience supported by a literature review has identified a knowledge gap and inconsistencies in screening for neurological and neurovascular pathology. Red flag screening in low back pain has received significant attention by researchers; however specific questions and testing for the cervical spine remain inconsistent e.g. cervical arterial dissection has been reported following manipulation, but screening processes to reduce the potential of adverse incidents occurring remains unclear. Similarly, there is inconsistent evidence to support the diagnostic accuracy of clinical tests for signs of cord compression myelopathy. This study is looking to establish a screening process through the combination of literature review, focus group with leading physiotherapists, and a Delphi method with consultant neurologists and consultant neurosurgeons that will be used to inform clinical practice and contribute to the evidence base for future research. The Delphi technique is considered as a suitable method to gain a consensus where there is incomplete knowledge or uncertainty in clinical issues.

Why have you been chosen?
You have been invited to participate in this study as your expertise as a consultant neurologist or consultant neurosurgeon is considered as highly valuable in contributing to the knowledge base that will inform clinical practice aimed at early detection of potentially serious neurological and neurovascular pathology presenting as, or alongside, a cervical spine musculoskeletal disorder.

Do I have to take part?
No. It is up to you to decide whether or not you take part. However, in addition to help progress clinical practice, the study also offers an excellent professional development opportunity. Your clinical expertise is considered as very valuable to the study. Informed consent will be considered as gained by your decision to access the web-based survey tool (http://www.survey.bris.ac.uk/), at the beginning of the study. If you decide to take part you are still free to withdraw at any time and without giving a reason.

What will happen to me if I take part?

1. By agreeing to take part in this study you will be forwarded a draft clinical chart and web link to round 1 of the Delphi survey. There will be a small number of
demographic questions to complete at the beginning. You will then be asked for your opinions on the presented information through open questions; however, you are free to omit items if you do not wish to answer. The draft red flag chart contains information on clinical features, risk factors, and clinical tests related to screening for potentially serious pathology, including neurological/neurovascular conditions. This information is collated from an evidence based literature review and physiotherapy focus group consisting of leading UK based physiotherapists (physiotherapy consultant level expertise, specialist spinal role, those with relevant peer-reviewed published papers, or MSc post-graduate level educators). Round 1 is anticipated to last approximately 15 minutes after initial viewing of the draft chart and will be complete when you press the ‘continue’ button on the main questions page.

2. The information is analysed in preparation for round 2.

3. Approximately 4-6-weeks from completing round 1 you will receive round 2 questionnaire via emailed web link. Round 2 will be a series of statements devised from the collective round 1 response that has been analysed into themes. You will be asked to give your level of agreement and disagreement based on a 5-point likert scale. There will also be an opportunity to provide added information if you feel it is appropriate or clarify a response.

4. Round 3 will follow a similar process to round 2 that will aim to achieve a consensus on the red flag screening process. Rounds 2 and 3 are anticipated to last approximately 10 minutes each.

What are the risks of taking part?
There is no identifiable risk of taking part in this study.

What if something goes wrong?
This study is considered as low risk. Regardless of this, if you wish to complain, or have any concerns about any aspect of the way you have been approached or treated during the course of this study, you may raise this concern directly with the Principal Investigator who will follow Queen Margaret University, Edinburgh complaint escalation procedure. Alternatively, you are free to contact the Independent Advisor.

Will my taking part in this study be kept confidential?
All information which is collected about you will be kept strictly confidential and managed in accordance with the Data Protection Act (1998). You will be assigned a study number which is linked to your name and stored in NHS encrypted information systems. Any information which leaves the NHS information system will have your name and contact details removed so that you cannot be recognised from it.

What will happen to the results of the research study?
The results of this study will be used to present a thesis that will be submitted as part fulfilment of a Professional Doctorate in Health and Social Sciences at Queen Margaret University, Edinburgh. The results will also be used to publish and present the findings of the research in a relevant journal and conference to inform clinical practice that promotes patient safety. However you will not be identified in any report or publication, unless you provide additional consent to do so.

The results will be submitted in a thesis format in June 2013 and you can obtain a summary from the Principal Investigator (details at the end of this sheet).
Who is organising and funding the research?
The study is being organised by the Principal Investigator who is completing the research as part of a higher academic degree as outlined above. Queen Margaret University, Edinburgh has approved the study and some time and financial support for this research has been obtained from NHS Borders and NHS Education for Scotland.

Who has reviewed the study?
The study protocol has been reviewed and conduct of the study has achieved Queen Margaret University, Edinburgh ethical approval. Recent changes to NHS Governance Arrangements for Research Ethics Committees states that additional NHS approval is not required. However, South East Scotland Research Ethics Service (NHS) has been informed of the study.

Contact for further information
Should you want any further information about this study please ask the Principal Investigator: Colin Redmond

Address: Physiotherapy Department
Borders General Hospital
Melrose
TD6 9BS

University email / work telephone: 09001905@qmu.ac.uk 01896 827004/826548

Alternative: Work email: Colin.Redmond@borders.scot.nhs.uk

If you have questions relating to any aspect of the study that you would like independent advice on, you will be able to speak to (to be inserted).

Independent Advisor: Professor James M Scobie
Director, CASL (Clinical Audiology, Speech and Language) Research Centre
Queen Margaret University
Edinburgh
EH21 6UU

Email JScobbie@qmu.ac.uk
Telephone 0131 474 0000 (state name of person)

If you decide to take part in this study, please retain a copy of the information sheet for your own records and thank you for taking part in this project.
Welcome

Welcome to the Delphi survey for the project 'Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders'.

The survey is completed anonymously, can be saved part way through and is anticipated to take around 15 minutes to complete after viewing the draft red flag chart.

Data Protection
All data collected in this survey will be held anonymously and securely. No personal data is asked for or retained.

Cookies, personal data stored by your Web browser, are not used in this survey.

Consent
I have read and understood the information sheet and have had an opportunity to ask questions about my participation.

I understand that I am under no obligation to take part in this study.

I understand that I have the right to withdraw from this study at any stage without giving any reason.

I agree to participate in this study and understand that by clicking the 'continue' button to the next page will be considered as providing informed consent.

Thank you for participating in this project.
Appendix Q: Round 1 Delphi Survey

The initial questions about you are mandatory (except 3a). The remaining questions are optional; however, your input to the 2 main questions (4 & 5) is considered as highly valuable, regardless of the extent of your response. All questions are contained on this page to allow ease of viewing between your responses.

Note that once you have clicked on the CONTINUE button your answers are submitted and you can not return to review or amend this page.

About you

1. Are you a consultant neurologist or consultant neurosurgeon?
   - ☐ neurologist  ☑ neurosurgeon

2. How many years have you been qualified as a consultant?
   - ☐ less than 5 years
   - ☑ 5-9 years
   - ☐ 10-19 years
   - ☐ 20 years or more

3. Which professional setting(s) do you work in?  
   (select all that apply)
   - NHS
   - Independent or private healthcare
   - Academia
   - Research
   - ☐ Other (please specify):

   a. If two or more settings are selected, which of these best represents your main work environment?  
   (Optional)
**Draft clinical chart - Feedback**

This section relates to your feedback on the draft chart. Your feedback will be used to inform the editing process to achieve a simpler and user-friendly chart with a specific focus on red flags.

4. Do you have any initial comments on the draft clinical chart as a tool to help identify red flags in neck pain and neck problems? See ‘More Info’.

Considerations may include your thoughts on the relevance of the sub-topics information, including tests or actions, and their inclusion/exclusion in the chart. *(Optional)*

*(Thesis note: Question 4 ‘More info’ appeared on clicking button)*

**Question 4 ‘More Info’** The chart intends to provide a ‘quick reference’ section with a specific list of key red flags. The more detailed background information is designed to provide context for these features that will assist Physiotherapists’ with their interpretation and subsequent clinical decision making.

**Developing list of red flags**

This section aims to develop a brief red flag list as previously outlined - *A starter list is provided below for your development:*

5. Please list the key red flag symptoms/signs that you consider to exist for neck pain or neck problems. Your list should include what the symptom/sign is likely to indicate e.g. cord compression.

**Starter list of red flags for your development** (copy & paste is available for editing purposes if required)

**Neck pain/stiffness/headache with brainstem symptoms/signs** - may be subtle and
**typically within 1 month onset:**
dizziness (typically episodic and between >1 min and <30 mins duration); slurred speech; double or loss of vision; drooping eyelid; pulsatile tinnitus or sudden loss of hearing; unilateral limb clumsiness or reduced balance; facial numbness or weakness; taste disturbance; nausea/vomiting; nystagmus.

**Headache (in presence of neck pain/stiffness) with features of:**
new onset (e.g. <1 month); change in usual pattern; changes with posture or exertion. Note if: cancer; age >50 yrs; or fever/infection present.

**Cord compression symptoms/signs (with or without neck pain/stiffness):**
early features: hand weakness or clumsiness; gait disturbance
progressive features: bilateral hand and/or feet pins/needles or numbness; upper or lower limbs spasticity or weakness; reflexes exaggerated or reduced; bladder/bowel disturbance (incontinence or retention); erectile dysfunction (rarely).

*(Optional)*

---

**Any other comments?**

6. Please add any further comments you may have about the red flag screening process that has not been covered above. *(Optional)*

---

*Thank you very much for completing this survey.*

Participants' responses will now be analysed and arranged into a series of themed short statements. These statements will be presented in round 2 using a 5-point likert scale format. This process aims to establish a consensus on the red flags to help develop the draft chart and improve our clinical decision making for earlier diagnosis of serious pathology.

*Thank you for your participation.*
Appendix R: Clinical scenario reminder for final round

- Patient attends non-medical musculoskeletal clinic (e.g. Physiotherapy - typically received by GP referral or by self referral with no previous GP contact).

- They complain of:
  - neck pain/stiffness (that may or may not have headache alongside their complaint); and / or upper limb dysfunction (e.g. pain, weakness, pins/needles/numbness, or reduced function - any combination of these). This occurs.

- When assessing the patient - Physiotherapist needs to expand their level of enquiry to screen for any underlying serious pathology that may be linked to their symptoms/signs. This enhanced enquiry may reveal additional symptoms/signs or other pathologies as outlined in the survey (developed from 1st round).

- With consultant input, we can improve this enhanced enquiry and understanding of how relevant these symptoms/signs and additional pathologies are, and when to raise our level of concern to seek additional medical opinion.

- Any consultant consensus on agreement / disagreement will help physios etc. achieve better focus on the relevant screening process and will part-inform our future development.
Welcome

Welcome to the Delphi survey 3rd (final) round for the project 'Red flag screening for serious pathology presenting in cervical spine musculoskeletal disorders'.

The survey is completed anonymously, can be saved part way through and is anticipated to take around 10-15 minutes to complete the questions.

The Delphi survey design is a re-run of the 2nd round with several minor modifications to help with clarity of information and is accompanied by the 2nd round results.

You are asked to simply re-rate your level of agreement/disagreement (5-point likert scale) on all clinical features (except Q10) for inclusion of the various components to guide clinical screening.

The additional comments option after each section has been retained should you wish to clarify or add any information.

Please note: All 2nd round additional comments have been saved to inform the full analysis, including the clinical groups listed on page 2.

Thank you for participating in the final phase of this project.

Red flag features

The clinical groups are presented in no particular order and may be combined at a later stage once clinical features are agreed.

1. Progressive pain
2. Cancer, infection, inflammatory arthritis/spondyloarthropathies, trauma (also risk of instability in this section)
3. Neurological deficit
4. Headache (may present alongside neck pain/stiffness)
5. Brainstem, cervical arterial, and cranial nerve dysfunctions

All groups are contained on this page to allow ease of viewing between your responses.

Note: Physiotherapists are increasingly aware to consider clusters of symptoms/signs indicating a raised index of suspicion for serious pathology, rather than acting prematurely on each individual feature in isolation.
Note: Once you have clicked on the CONTINUE button your answers are submitted and you are unable to return to review or amend this page.

Please remember to consider your level of agreement/disagreement for inclusion of the features in the list of red flags and to complete each question. Please select "No opinion" if there is uncertainty regarding level of agreement/disagreement.

1. Progressive Pain (Neck and/or radicular pain. May be accompanied by headache).

1. Progressive pain:

   a. May be in isolation (i.e. not accompanied or associated with others features e.g. history of cancer, trauma, presence of hard neurological signs, suspected atlanto-axial instability or infection).

      - Strongly agree
      - Agree
      - No opinion
      - Disagree
      - Strongly disagree

   b. May be associated with others features (e.g. history of cancer, trauma, presence of hard neurological signs, suspected atlanto-axial instability or infection).

      - Strongly agree
      - Agree
      - No opinion
      - Disagree
      - Strongly disagree

2. If pain is progressively worse (e.g. over past week).

   - Strongly agree
   - Agree
   - No opinion
   - Disagree
   - Strongly disagree

3. Pain not responding to simple measures (e.g. normal analgesics).

   - Strongly agree
   - Agree
   - No opinion
   - Disagree
   - Strongly disagree

4. Onset:

   a. May be a new 1st episode of acute/subacute pain onset (e.g. onset immediately following trauma or present for hours to 1-month for arterial dissection).

      - Strongly agree
      - Agree
      - No opinion
      - Disagree
      - Strongly disagree

   b. May be an acute/subacute aggravation on chronic pain.

      - Strongly agree
      - Agree
      - No opinion
      - Disagree
      - Strongly disagree

5. Description: Severe and/or unremitting.

Note caution: Description is not definitive - May be sharp or dull; localised or
diffuse/no specific distribution; moderate or severe.

<table>
<thead>
<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
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</table>

6. Severe pain on movement; reluctance to move; gross cervical spasm or torticollis (cancer, infection, atlanto-axial instability).

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<thead>
<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
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7. Disrupting sleep (does not ease with adjusting sleep position e.g. pillow).

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<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
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</table>

8. Any other comments on this section?

2. Cancer, infection, inflammatory arthritis/spondyloarthropathies, trauma

**Note:** Risk of upper cervical spine instability occurring as a progression of such pathologies

9. Cancer
Previous history of cancer; unexplained weight loss; lymphadenopathy.

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<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
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10. Age and risk (The consultant panel have provided 3 options):
What age group do you think should be used as an indicator of increased risk of serious pathology occurring naturally e.g. cancer? Additional consideration could be given to other pathologies e.g. cervical arterial dissection.

<table>
<thead>
<tr>
<th>Age greater than 40 years</th>
<th>Age greater than 50 years</th>
<th>No age group association (no age group is exempt)</th>
<th>Other (please specify):</th>
</tr>
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11. **Infection**
Malaise, fever, sweats, lethargy.

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<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
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12. **Tuberculosis, immunosuppression, drug abuse, HIV/AIDS, or other (significant) infection.**

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<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
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</table>

13. **Inflammatory arthritis/spondyloarthropathies**

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<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
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</table>

14. **Trauma (recent onset)**

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<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
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</thead>
</table>

15. **Any other comments on this section?**

3. **Neurological deficit (e.g. spinal cord compromise)**

16. **Quick guide**
Upper motor neuron symptoms/signs (in lower limbs more than upper limbs).
Lower motor neuron symptoms/signs (in upper limbs more than lower limbs).

<table>
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<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
</tr>
</thead>
</table>

17. **Hands: clumsy/loss of dexterity or weakness.**

<table>
<thead>
<tr>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
</tr>
</thead>
</table>

270
18. Weakness (widespread) of arms or legs.

19. Diffuse numbness or paraesthesia (pins/needles).

20. Loss of proprioception.


22. Hyperreflexia:
   (Increased/exaggerated reflexes in lower limbs more than upper limbs; Hoffman's reflex; finger flexion-extension jerks; clonus; myoclonus/spasticity in lower limbs > upper limbs; upgoing plantar response).

23. Gait disturbance
   e.g. stiff, slow, broad based.

24. Lower motor neuron symptoms/signs (in upper limb more than lower limb).
   Radicular pattern in particular dermatome: Numbness; paraesthesia (pins/needles);
25. Very late stage

- Strongly agree
- Agree
- No opinion
- Disagree
- Strongly disagree

a. Sphincter disturbance (bladder and/or bowel) disturbance (retention or incontinence).

- Strongly agree
- Agree
- No opinion
- Disagree
- Strongly disagree

b. Erectile dysfunction (rare occurrence).

- Strongly agree
- Agree
- No opinion
- Disagree
- Strongly disagree

26. General progressive neurological deficit

Any new progressive and/or unusual neurological symptoms/signs (irrespective of neck pain/stiffness).

- Strongly agree
- Agree
- No opinion
- Disagree
- Strongly disagree

27. Any other comments on this section?

4. Headache (accompanying neck pain/stiffness)

Headache may be reported at musculoskeletal clinics alongside neck pain/stiffness. Other possible sites of lesion should be considered e.g. Cervical arterial dissection/dysfunction; Chiari 1 malformation; raised intracranial pressure; instability (e.g. occipital neuralgia) if the following features present:

28. New onset (e.g. less than 1 month).

- Strongly agree
- Agree
- No opinion
- Disagree
- Strongly disagree

29. Severe and persistent.

Note caution: May have no definitive distribution (e.g. local (such as occipital), or diffuse), description (e.g. sharp or dull) or intensity (e.g. moderate or severe).
5. Brain stem, cervical arterial (occlusion/stenosis/dissection) and cranial nerve dysfunctions

Low level evidence suggests that subtle symptoms/signs of these other origins of pathology may present at musculoskeletal clinics alongside neck pain/headache before progressive deterioration.

*This section presents most clinical uncertainty for non-medical practitioners* see 'More Info'

33. Symptoms/signs outlined below should raise concern if: New or recent onset (e.g. within 1 month).

   **Caution** - symptoms/signs may be subtle if presenting at musculoskeletal clinics.

(Thesis note: Question 33 More info. The following text appears when button pressed)

Physiotherapists have traditionally used the acronym '5Ds And 3Ns' (Dizziness, diplopia, dysphagia, dysphasia, drop attacks, ataxia, nystagmus, numbness, nausea) for recognition of cervical arterial pathology. The 5Ds origin is an unreferenced book chapter (1986) and the A & 3Ns parts being added at later stages. There is limited research to support this acronym. However, it is possible that some features may present at musculoskeletal clinics (Physiotherapy, chiropractic, osteopathy).

In addition, observational studies of cervical arterial pathology are generally conducted at
neurological centres where the focus may not be on early features, making it more difficult to inform physiotherapy practice.

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<tr>
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<th>Strongly agree</th>
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<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
</tr>
</thead>
</table>

i. Dizziness (Central cause typically episodic and between >1min and <30mins duration. Not occurring in isolation).

ii. Slurred speech.

iii. Double or loss of vision.

iv. Drooping eyelid / Horner's sign.

v. Pulsatile tinnitus or sudden loss of hearing.

vi. Unilateral limb clumsiness or reduced balance.

vii. Facial numbness or weakness.

viii. Taste disturbance.

ix. Vomiting (Stronger indicator than nausea).

x. Nystagmus.

34. Any other comments on this section?
Lastly - Any other general comments?

35. Please add any further comments you may have about the red flag screening process that has not been covered above.
Thank you very much for completing the final survey of this project.

The consultant panel’s collective responses will now be analysed and re-presented in a similar format that accompanied this survey.

This Delphi study aims to establish a consensus on the red flags to help improve our clinical decision making for earlier diagnosis of serious pathology and to inform future projects.

Thank you for your highly valued support to this project.
Appendix T: Delphi survey Round 3 results in graph representation

Boxes shaded red indicate clinical indicators meeting consensus point 80%

Boxes shaded orange indicate clinical indicators achieving 70-79% based on minimum n=9 responses per indicator. This suggests borderline agreement.

---

**Section 1: 1. Progressive Pain (Neck and/or radicular pain. May be accompanied by headache).**

### 1. Progressive pain:

1.a. May be in isolation (i.e. not accompanied or associated with other features e.g. history of cancer, trauma, presence of hard neurological signs, suspected atlanto-axial instability or infection).

<table>
<thead>
<tr>
<th></th>
<th>Strongly agree</th>
<th>Agree</th>
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<th>Disagree</th>
<th>Strongly disagree</th>
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<tbody>
<tr>
<td></td>
<td>9.1%</td>
<td>45.5%</td>
<td>9.1%</td>
<td>36.4%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

1.b. May be associated with other features (e.g. history of cancer, trauma, presence of hard neurological signs, suspected atlanto-axial instability or infection).

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<thead>
<tr>
<th></th>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
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<tbody>
<tr>
<td></td>
<td>54.5%</td>
<td>36.4%</td>
<td>9.1%</td>
<td>0.0%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

---

2. If pain is progressively worse (e.g. over past week).

<table>
<thead>
<tr>
<th></th>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.0%</td>
<td>36.4%</td>
<td>18.2%</td>
<td>45.5%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

---

3. Pain not responding to simple measures (e.g. normal analgesics).

<table>
<thead>
<tr>
<th></th>
<th>Strongly agree</th>
<th>Agree</th>
<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.0%</td>
<td>27.3%</td>
<td>27.3%</td>
<td>36.4%</td>
<td>9.1%</td>
</tr>
</tbody>
</table>
### 4. Onset:

<table>
<thead>
<tr>
<th>4.a. May be a new 1st episode of acute/subacute pain onset (e.g. onset immediately following trauma or present for hours to 1-month for arterial dissection).</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Strongly agree:</strong></td>
</tr>
<tr>
<td><strong>Agree:</strong></td>
</tr>
<tr>
<td><strong>No opinion:</strong></td>
</tr>
<tr>
<td><strong>Disagree:</strong></td>
</tr>
<tr>
<td><strong>Strongly disagree:</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>4.b. May be an acute/subacute aggravation on chronic pain.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Strongly agree:</strong></td>
</tr>
<tr>
<td><strong>Agree:</strong></td>
</tr>
<tr>
<td><strong>No opinion:</strong></td>
</tr>
<tr>
<td><strong>Disagree:</strong></td>
</tr>
<tr>
<td><strong>Strongly disagree:</strong></td>
</tr>
</tbody>
</table>

### 5. Description: Severe and/or unremitting. Note caution: Description is not definitive - May be sharp or dull; localised or diffuse/no specific distribution; moderate or severe.

| **Strongly agree:** | 9.1% | 1 |
| **Agree:** | 54.5% | 6 |
| **No opinion:** | 9.1% | 1 |
| **Disagree:** | 27.3% | 3 |
| **Strongly disagree:** | 0.0% | 0 |

### 6. Severe pain on movement; reluctance to move; gross cervical spasm or torticollis (cancer, infection, atlanto-axial instability).

| **Strongly agree:** | 30.0% | 3 |
| **Agree:** | 50.0% | 5 |
| **No opinion:** | 10.0% | 1 |
| **Disagree:** | 10.0% | 1 |
| **Strongly disagree:** | 0.0% | 0 |

### 7. Disrupting sleep (does not ease with adjusting sleep position e.g. pillow).

| **Strongly agree:** | 9.1% | 1 |
| **Agree:** | 27.3% | 3 |
| **No opinion:** | 9.1% | 1 |
| **Disagree:** | 54.5% | 6 |
| **Strongly disagree:** | 0.0% | 0 |
### Section 2: Cancer, Infection, Inflammatory Arthritis/Spondyloarthropathies, Trauma

#### 9. Cancer

**Previous history of cancer; unexplained weight loss; lymphadenopathy.**

| Strongly agree: | 54.5% | 6 |
| Agree:          | 36.4% | 4 |
| No opinion:     | 0.0%  | 0 |
| Disagree:       | 9.1%  | 1 |
| Strongly disagree: | 0.0%  | 0 |

#### 10. Age and Risk

(The consultant panel have provided 3 options): What age group do you think should be used as an indicator of increased risk of serious pathology occurring naturally e.g. cancer? Additional consideration could be given to other pathologies e.g. cervical arterial dissection.

<table>
<thead>
<tr>
<th>Age group</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age greater than 40 years:</td>
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</tr>
<tr>
<td>Age greater than 50 years:</td>
<td>54.5%</td>
</tr>
<tr>
<td>No age group association (no age group is exempt):</td>
<td>45.5%</td>
</tr>
</tbody>
</table>

#### 11. Infection

Malaise, fever, sweats, lethargy.

| Strongly agree: | 27.3% | 3 |
| Agree:          | 54.5% | 6 |
| No opinion:     | 0.0%  | 0 |
| Disagree:       | 18.2% | 2 |
| Strongly disagree: | 0.0%  | 0 |

#### 12. Tuberculosis, Immunosuppression, Drug Abuse, HIV/AIDS, or Other (Significant) Infection.

| Strongly agree: | 18.2% | 2 |
| Agree:          | 72.7% | 8 |
| No opinion:     | 9.1%  | 1 |
| Disagree:       | 0.0%  | 0 |
| Strongly disagree: | 0.0%  | 0 |

#### 13. Inflammatory Arthritis/Spondyloarthropathies

| Strongly agree: | 10.0% | 1 |
| Agree:          | 40.0% | 4 |
| No opinion:     | 30.0% | 3 |
| Disagree:       | 20.0% | 2 |
| Strongly disagree: | 0.0%  | 0 |
### 14. Trauma (recent onset)

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strongly agree</td>
<td>45.5%</td>
</tr>
<tr>
<td>Agree</td>
<td>54.5%</td>
</tr>
<tr>
<td>No opinion</td>
<td>0.0%</td>
</tr>
<tr>
<td>Disagree</td>
<td>0.0%</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

### Section 3: 3. Neurological deficit (e.g. spinal cord compromise)

#### 16. Quick guide Upper motor neuron symptoms/signs (in lower limbs more than upper limbs). Lower motor neuron symptoms/signs (in upper limbs more than lower limbs).

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strongly agree</td>
<td>54.5%</td>
</tr>
<tr>
<td>Agree</td>
<td>45.5%</td>
</tr>
<tr>
<td>No opinion</td>
<td>0.0%</td>
</tr>
<tr>
<td>Disagree</td>
<td>0.0%</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

#### 17. Hands: clumsy/loss of dexterity or weakness.

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strongly agree</td>
<td>45.5%</td>
</tr>
<tr>
<td>Agree</td>
<td>54.5%</td>
</tr>
<tr>
<td>No opinion</td>
<td>0.0%</td>
</tr>
<tr>
<td>Disagree</td>
<td>0.0%</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

#### 18. Weakness (widespread) of arms or legs.

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strongly agree</td>
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<tr>
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<td>40.0%</td>
</tr>
<tr>
<td>No opinion</td>
<td>10.0%</td>
</tr>
<tr>
<td>Disagree</td>
<td>20.0%</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

#### 19. Diffuse numbness or paraesthesia (pins/needles).

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strongly agree</td>
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<tr>
<td>Agree</td>
<td>45.5%</td>
</tr>
<tr>
<td>No opinion</td>
<td>9.1%</td>
</tr>
<tr>
<td>Disagree</td>
<td>27.3%</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
</tr>
</tbody>
</table>
20. Loss of proprioception.

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
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<tr>
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<td>1</td>
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<table>
<thead>
<tr>
<th>Opinion</th>
<th>Percentage</th>
<th>Count</th>
</tr>
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<tbody>
<tr>
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<tr>
<td>Disagree</td>
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<td>0</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

22. Hyperreflexia: (Increased/exaggerated reflexes in lower limbs more than upper limbs; Hoffman's reflex; finger flexion-extension jerks; clonus; myoclonus/spasticity in lower limbs > upper limbs; upgoing plantar response).

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
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<td>0.0%</td>
<td>0</td>
</tr>
<tr>
<td>Disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

23. Gait disturbance e.g. stiff, slow, broad based.

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
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<td>0</td>
</tr>
<tr>
<td>Disagree</td>
<td>9.1%</td>
<td>1</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

24. Lower motor neuron symptoms/signs (in upper limb more than lower limb). Radicular pattern in particular dermatome: Numbness; paraesthesia (pins/needles); weakness; hyporeflexia (reduced reflexes) in particular dermatome.

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
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<tr>
<td>Disagree</td>
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<td>3</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
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</tr>
</tbody>
</table>
### 25. Very late stage

#### 25.a. Sphincter disturbance (bladder and/or bowel) disturbance (retention or incontinence).

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
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<tr>
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<td>2</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

#### 25.b. Erectile dysfunction (rare occurrence).

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
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<tr>
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<td>4</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

### 26. General progressive neurological deficit

Any new progressive and/or unusual neurological symptoms/signs (irrespective of neck pain/stiffness).

<table>
<thead>
<tr>
<th>Opinion</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td>Agree</td>
<td>36.4%</td>
<td>4</td>
</tr>
<tr>
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<td>9.1%</td>
<td>1</td>
</tr>
<tr>
<td>Disagree</td>
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<td>0</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>
### Section 4: 4. Headache (accompanying neck pain/stiffness)

#### 28. New onset (e.g. less than 1 month).

<table>
<thead>
<tr>
<th>Option</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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<td>3</td>
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<tr>
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<td>5</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

#### 29. Severe and persistent. **Note caution:** May have no definitive distribution (e.g. local (such as occipital), or diffuse), description (e.g. sharp or dull) or intensity (e.g. moderate or severe).

<table>
<thead>
<tr>
<th>Option</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strongly agree</td>
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<td>0</td>
</tr>
<tr>
<td>Agree</td>
<td>50.0%</td>
<td>5</td>
</tr>
<tr>
<td>No opinion</td>
<td>10.0%</td>
<td>1</td>
</tr>
<tr>
<td>Disagree</td>
<td>40.0%</td>
<td>4</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

#### 30. Headache changes:

**30.a.** In usual pattern.

<table>
<thead>
<tr>
<th>Option</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
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<td>2</td>
</tr>
<tr>
<td>Disagree</td>
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<td>6</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

**30.b.** With posture or brought on by exertion, cough, laugh or straining.

<table>
<thead>
<tr>
<th>Option</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>

#### 31. Sudden (unexplained) onset.

<table>
<thead>
<tr>
<th>Option</th>
<th>Percentage</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td>Agree</td>
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<tr>
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<tr>
<td>Disagree</td>
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<td>1</td>
</tr>
<tr>
<td>Strongly disagree</td>
<td>0.0%</td>
<td>0</td>
</tr>
</tbody>
</table>
Section 5: Brain stem, cervical arterial (occlusion/stenosis/dissection) and cranial nerve dysfunctions

33. Symptoms/signs outlined below should raise concern if: New or recent onset (e.g. within 1 month). Caution - symptoms/signs may be subtle if presenting at musculoskeletal clinics.

<table>
<thead>
<tr>
<th>Strongly agree:</th>
<th>Agree:</th>
<th>No opinion:</th>
<th>Disagree:</th>
<th>Strongly disagree:</th>
</tr>
</thead>
<tbody>
<tr>
<td>14.3%</td>
<td>42.9%</td>
<td>42.9%</td>
<td>0.0%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

33.a. Dizziness (Central cause typically episodic and between >1min and <30mins duration. Not occurring in isolation).

<table>
<thead>
<tr>
<th>Strongly agree:</th>
<th>Agree:</th>
<th>No opinion:</th>
<th>Disagree:</th>
<th>Strongly disagree:</th>
</tr>
</thead>
<tbody>
<tr>
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<td>40.0%</td>
<td>20.0%</td>
<td>40.0%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>

33.a.i. Slurred speech.

<table>
<thead>
<tr>
<th>Strongly agree:</th>
<th>Agree:</th>
<th>No opinion:</th>
<th>Disagree:</th>
<th>Strongly disagree:</th>
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<tbody>
<tr>
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<td>80.0%</td>
<td>0.0%</td>
<td>10.0%</td>
<td>0.0%</td>
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</tbody>
</table>

33.a.ii. Double or loss of vision.

<table>
<thead>
<tr>
<th>Strongly agree:</th>
<th>Agree:</th>
<th>No opinion:</th>
<th>Disagree:</th>
<th>Strongly disagree:</th>
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<td>60.0%</td>
<td>0.0%</td>
<td>10.0%</td>
<td>0.0%</td>
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</tbody>
</table>

33.a.iii. Drooping eyelid / Horner's sign.

<table>
<thead>
<tr>
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<th>Agree:</th>
<th>No opinion:</th>
<th>Disagree:</th>
<th>Strongly disagree:</th>
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<td>22.2%</td>
<td>11.1%</td>
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</tbody>
</table>

33.a.v. Pulsatile tinnitus or sudden loss of hearing.
<table>
<thead>
<tr>
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<th>No opinion</th>
<th>Disagree</th>
<th>Strongly disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>33.a.vi.</strong> Unilateral limb clumsiness or reduced balance.</td>
<td>30.0%</td>
<td>50.0%</td>
<td>10.0%</td>
<td>10.0%</td>
<td>0.0%</td>
</tr>
<tr>
<td><strong>33.a.vii.</strong> Facial numbness or weakness.</td>
<td>33.3%</td>
<td>66.7%</td>
<td>0.0%</td>
<td>0.0%</td>
<td>0.0%</td>
</tr>
<tr>
<td><strong>33.a.viii.</strong> Taste disturbance.</td>
<td>0.0%</td>
<td>70.0%</td>
<td>10.0%</td>
<td>20.0%</td>
<td>0.0%</td>
</tr>
<tr>
<td><strong>33.a.ix.</strong> Vomiting (Stronger indicator than nausea).</td>
<td>10.0%</td>
<td>50.0%</td>
<td>20.0%</td>
<td>20.0%</td>
<td>0.0%</td>
</tr>
<tr>
<td><strong>33.a.x.</strong> Nystagmus.</td>
<td>50.0%</td>
<td>40.0%</td>
<td>10.0%</td>
<td>0.0%</td>
<td>0.0%</td>
</tr>
</tbody>
</table>
### Appendix U: Descriptive statistics Delphi survey Round 3

**Descriptive Statistics Delphi Round 3**

<table>
<thead>
<tr>
<th>Clinical Indicator</th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progressive pain in isolation</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>3.27</td>
<td>1.104</td>
</tr>
<tr>
<td>Progressive pain associated with other features e.g. history of cancer</td>
<td>11</td>
<td>3</td>
<td>5</td>
<td>4.45</td>
<td>.688</td>
</tr>
<tr>
<td>Pain is progressively worse e.g. over past week</td>
<td>11</td>
<td>2</td>
<td>4</td>
<td>2.91</td>
<td>.944</td>
</tr>
<tr>
<td>Pain not responding to simple analgesia</td>
<td>11</td>
<td>1</td>
<td>4</td>
<td>2.73</td>
<td>1.009</td>
</tr>
<tr>
<td>Pain may be new 1st episode of acute/sub-acute onset</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>3.55</td>
<td>1.128</td>
</tr>
<tr>
<td>Pain may be an acute/subacute aggravation on chronic pain</td>
<td>11</td>
<td>1</td>
<td>4</td>
<td>2.73</td>
<td>1.104</td>
</tr>
<tr>
<td>Severe and/or unremitting pain</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>3.45</td>
<td>1.036</td>
</tr>
<tr>
<td>Severe pain on movement</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>4.00</td>
<td>.943</td>
</tr>
<tr>
<td>Pain disrupting sleep (does not ease with adjusting position)</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>2.91</td>
<td>1.136</td>
</tr>
</tbody>
</table>

5=Strongly Agree; 4= Agree; 3= No Opinion; 2=Disagree; 1=Strongly Disagree

<table>
<thead>
<tr>
<th>Clinical Indicator</th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer: Previous history; unexplained weight loss; lymphadenopathy</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>4.36</td>
<td>.924</td>
</tr>
<tr>
<td>Age as an indicator of increased risk of serious pathology occurring naturally</td>
<td>10</td>
<td>1.00</td>
<td>2.00</td>
<td>1.4000</td>
<td>.51640</td>
</tr>
<tr>
<td>Infection: Malaise, fever, sweats, lethargy</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>3.91</td>
<td>1.044</td>
</tr>
<tr>
<td>Tuberculosis, immunosuppression, drug abuse, HIV/AIDS, or other (significant) infection</td>
<td>11</td>
<td>3</td>
<td>5</td>
<td>4.09</td>
<td>.539</td>
</tr>
<tr>
<td>Inflammatory arthritis/spondyloarthropathies</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>3.40</td>
<td>.966</td>
</tr>
<tr>
<td>Trauma (recent onset)</td>
<td>10</td>
<td>4</td>
<td>5</td>
<td>4.40</td>
<td>.516</td>
</tr>
</tbody>
</table>

5=Strongly Agree; 4= Agree; 3= No Opinion; 2=Disagree; 1=Strongly Disagree
<table>
<thead>
<tr>
<th>Clinical Indicator</th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quick guide: UMN symptoms/signs in LL&gt;UL; LMN symptoms/signs in UL&gt;LL</td>
<td>11</td>
<td>4</td>
<td>5</td>
<td>4.55</td>
<td>.522</td>
</tr>
<tr>
<td>Hands: clumsy/loss of dexterity or weakness</td>
<td>11</td>
<td>4</td>
<td>5</td>
<td>4.45</td>
<td>.522</td>
</tr>
<tr>
<td>Weakness (widespread) of arms or legs</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>3.80</td>
<td>1.135</td>
</tr>
<tr>
<td>Diffuse numbness or paraesthesia</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>3.55</td>
<td>1.128</td>
</tr>
<tr>
<td>Loss of proprioception</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>4.00</td>
<td>.894</td>
</tr>
<tr>
<td>Lhermitte's phenomenon/sign</td>
<td>11</td>
<td>4</td>
<td>5</td>
<td>4.73</td>
<td>.467</td>
</tr>
<tr>
<td>Hyperreflexia</td>
<td>11</td>
<td>4</td>
<td>5</td>
<td>4.55</td>
<td>.522</td>
</tr>
<tr>
<td>Gait disturbance</td>
<td>11</td>
<td>2</td>
<td>5</td>
<td>4.18</td>
<td>.874</td>
</tr>
<tr>
<td>LMN symptoms/signs</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>3.60</td>
<td>1.174</td>
</tr>
<tr>
<td>Very late stage: Sphincter disturbance (bladder and/or bowel retention or incontinence)</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>4.10</td>
<td>1.197</td>
</tr>
<tr>
<td>Very late stage: Erectile dysfunction (rare occurrence)</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>3.40</td>
<td>1.265</td>
</tr>
<tr>
<td>General progressive neurological deficit</td>
<td>11</td>
<td>3</td>
<td>5</td>
<td>4.45</td>
<td>.688</td>
</tr>
</tbody>
</table>

5=Strongly Agree; 4=Agree; 3=No Opinion; 2=Disagree; 1=Strongly Disagree
Descriptive Statistics Delphi Round 3

<table>
<thead>
<tr>
<th>Clinical Indicator</th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>Std. Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache: new onset (e.g. less than 1-month)</td>
<td>10</td>
<td>2</td>
<td>4</td>
<td>2.70</td>
<td>.823</td>
</tr>
<tr>
<td>Headache: severe and persistent</td>
<td>10</td>
<td>2</td>
<td>4</td>
<td>3.10</td>
<td>.994</td>
</tr>
<tr>
<td>Headache: changes in usual pattern</td>
<td>10</td>
<td>2</td>
<td>4</td>
<td>2.60</td>
<td>.843</td>
</tr>
<tr>
<td>Headache: changes with posture or brought on by exertion, cough, laugh or straining</td>
<td>9</td>
<td>4</td>
<td>5</td>
<td>4.22</td>
<td>.441</td>
</tr>
<tr>
<td>Headache: sudden (unexpected) onset</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>4.10</td>
<td>.994</td>
</tr>
<tr>
<td>New or recent onset (e.g. within 1-month) of the following symptoms/signs</td>
<td>7</td>
<td>3</td>
<td>5</td>
<td>3.86</td>
<td>.900</td>
</tr>
<tr>
<td>Dizziness (central cause typically episodic and between &gt;1min and &lt;30min duration. Not occurring in isolation)</td>
<td>10</td>
<td>2</td>
<td>4</td>
<td>3.00</td>
<td>.943</td>
</tr>
<tr>
<td>Slurred speech</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>3.90</td>
<td>.738</td>
</tr>
<tr>
<td>Double or loss of vision</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>4.10</td>
<td>.876</td>
</tr>
<tr>
<td>Drooping eyelid / Horner's sign</td>
<td>9</td>
<td>3</td>
<td>5</td>
<td>4.56</td>
<td>.726</td>
</tr>
<tr>
<td>Pulstaile tinnitus or sudden loss of hearing</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>4.00</td>
<td>.943</td>
</tr>
<tr>
<td>Unilateral limb clumsiness or reduced balance</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>3.80</td>
<td>1.033</td>
</tr>
<tr>
<td>Facial numbness or weakness</td>
<td>9</td>
<td>4</td>
<td>5</td>
<td>4.33</td>
<td>.500</td>
</tr>
<tr>
<td>Taste disturbance</td>
<td>10</td>
<td>2</td>
<td>4</td>
<td>3.50</td>
<td>.850</td>
</tr>
<tr>
<td>Vomiting (stronger indicator than nausea)</td>
<td>10</td>
<td>2</td>
<td>5</td>
<td>3.50</td>
<td>.972</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>10</td>
<td>3</td>
<td>5</td>
<td>4.40</td>
<td>.699</td>
</tr>
</tbody>
</table>

5=Strongly Agree; 4= Agree; 3=No Opinion; 2=Disagree; 1=Strongly Disagree
## Appendix V: Raw data Delphi survey Round 2

### Delphi Round 2: Question numbers (1-33) and responses (1-5)

| P | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25a | 25b | 26 | 27 | 28 | 29 | 30 | 31 | 32 | 33a1 | 33a2 | 33a3 | 33a4 | 33a5 | 33a6 | 33a7 | 33a8 | 33a9 | 33a10 |
| 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 4 | 2 | 4 | 5 | 5 | 4 | 1 | 5 | 4 | 2 | 5 | 5 | 0 | 4 | 1 | 4 | 4 | 1 | 1 |
| 2 | 4 | 4 | 2 | 4 | 4 | 5 | 2 | 5 | 1 | 4 | 5 | 5 | 5 | 5 | 5 | 4 | 4 | 5 | 5 | 5 | 4 | 5 | 4 | 2 | 2 | 2 | 2 | 4 | 4 | 4 | 4 | 4 | 5 | 4 | 2 | 5 |
| 3 | 4 | 3 | 3 | 4 | 4 | 4 | 2 | 4 | 2 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 2 | 4 | 4 | 4 | 2 | 3 | 4 | 5 | 4 | 5 | 5 | 5 | 5 | 5 | 3 | 5 | 5 |
| 4 | 3 | 2 | 2 | 2 | 2 | 4 | 4 | 5 | 1 | 4 | 4 | 4 | 5 | 5 | 5 | 4 | 4 | 5 | 5 | 5 | 4 | 4 | 4 | 4 | 4 | 5 | 5 | 5 | 5 | 5 | 4 | 4 | 4 | 5 |
| 5 | 4 | 3 | 3 | 4 | 2 | 4 | 4 | 5 | 1 | 4 | 5 | 4 | 4 | 5 | 4 | 4 | 4 | 5 | 5 | 5 | 4 | 5 | 4 | 4 | 4 | 4 | 4 | 5 | 5 | 5 | 5 | 5 | 4 | 5 | 5 |
| 6 | 3 | 2 | 2 | 3 | 2 | 2 | 4 | 4 | 2 | 5 | 5 | 2 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 2 | 4 | 4 | 5 | 3 | 3 | 4 | 3 | 5 | 5 | 5 | 3 | 4 | 3 | 4 |
| 7 | 4 | 2 | 4 | 4 | 4 | 3 | 5 | 2 | 4 | 4 | 4 | 4 | 5 | 5 | 5 | 5 | 5 | 5 | 5 | 4 | 5 | 4 | 2 | 2 | 4 | 4 | 3 | 5 | 4 | 4 | 3 | 4 | 3 | 4 | 4 |
| 8 | 4 | 1 | 2 | 2 | 4 | 5 | 2 | 5 | 1 | 5 | 4 | 2 | 4 | 4 | 4 | 5 | 2 | 4 | 5 | 5 | 2 | 2 | 5 | 5 | 2 | 5 | 2 | 2 | 5 | 5 | 2 | 4 | 5 | 3 | 2 | 2 |
| 9 | 5 | 4 | 4 | 5 | 4 | 4 | 5 | 4 | 1 | 4 | 5 | 4 | 5 | 4 | 5 | 5 | 4 | 5 | 5 | 4 | 5 | 4 | 5 | 4 | 3 | 3 | 4 | 3 | 4 | 4 | 5 | 5 | 4 | 5 |
| 10 | 5 | 4 | 4 | 3 | 5 | 4 | 4 | 5 | 0 | 4 | 4 | 3 | 4 | 4 | 4 | 4 | 4 | 3 | 3 | 3 | 4 | 3 | 4 | 3 | 4 | 3 | 4 | 4 | 4 | 4 | 4 | 4 | 3 | 4 |
| 11 | 4 | 4 | 2 | 4 | 4 | 2 | 2 | 5 | 1 | 4 | 4 | 3 | 4 | 4 | 4 | 4 | 2 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 2 | 4 | 4 | 4 | 4 | 4 | 4 | 4 | 3 | 2 | 4 |

P = Participant numbers 1-11  
Responses: 5=Strongly Agree; 4= Agree; 3=No Opinion; 2=Disagree; 1=Strongly Disagree
Appendix W: Raw data Delphi survey Round 3

## Delphi Round 3: Question numbers (1-33) and responses (1-5)

| P  | 1a | 1b | 2  | 3  | 4a | 4b | 5  | 6  | 7  | 8  | 9  | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 | 19 | 20 | 21 | 22 | 23 | 24 | 25a | 25b | 26 | 27 | 28 | 29 | 30 | 31 | 32 | 33 | 33a1 | 33a2 | 33a3 | 33a4 | 33a5 | 33a6 | 33a7 | 33a8 | 33a9 | 33a10 |
|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|----|
| 1  | 4  | 5  | 2  | 1  | 2  | 1  | 4  | 2  | 5  | 1  | 4  | 5  | 5  | 4  | 5  | 5  | 5  | 3  | 5  | 5  | 5  | 4  | 4  | 4  | 4  | 4  | 5  | 2  | 2  | 4  | 2  | 4  | 4  | 5  | 5  | 5  | 5  | 2  | 4  | 5  |
| 2  | 5  | 5  | 4  | 4  | 5  | 4  | 5  | 5  | 5  | 2  | 5  | 2  | 5  | 4  | 5  | 5  | 5  | 5  | 5  | 5  | 5  | 4  | 5  | 2  | 5  | 2  | 4  | 2  | 4  | 4  | 2  | 2  | 2  | 4  | 4  | 5  | 2  | 4  | 4  | 5  |
| 3  | 2  | 4  | 2  | 2  | 2  | 2  | 4  | 4  | 4  | 2  | 4  | 4  | 4  | 5  | 5  | 4  | 4  | 4  | 5  | 4  | 4  | 2  | 4  | 4  | 2  | 2  | 2  | 4  | 5  | 2  | 4  | 4  | 5  | 4  | 4  | 4  | 4  | 2  | 4  |
| 4  | 4  | 4  | 4  | 2  | 4  | 2  | 4  | 5  | 2  | 5  | 1  | 5  | 5  | 5  | 5  | 4  | 5  | 4  | 5  | 5  | 5  | 4  | 5  | 4  | 4  | 2  | 5  | 5  | 4  | 4  | 5  | 5  | 5  | 5  | 4  | 4  | 4  | 5  | 5  |
| 5  | 2  | 4  | 2  | 4  | 4  | 2  | 2  | 2  | 2  | 3  | 4  | 1  | 2  | 4  | 3  | 4  | 5  | 5  | 2  | 2  | 3  | 3  | 5  | 5  | 5  | 2  | 2  | 2  | 2  | 2  | 2  | 2  | 3  | 3  | 4  | 5  | 3  | 4  | 4  | 4  | 4  | 5  |
| 6  | 4  | 5  | 3  | 2  | 4  | 4  | 4  | 5  | 2  | 4  | 2  | 5  | 3  | 4  | 5  | 4  | 4  | 5  | 5  | 4  | 4  | 2  | 5  | 5  | 5  | 2  | 4  | 3  | 4  | 5  | 5  | 2  | 4  | 5  | 5  | 5  | 4  | 5  | 4  | 3  | 3  |
| 7  | 2  | 5  | 4  | 3  | 5  | 4  | 4  | 4  | 5  | 5  | 1  | 4  | 4  | 3  | 5  | 4  | 4  | 3  | 2  | 2  | 4  | 5  | 4  | 4  | 4  | 5  | 3  | 4  | 2  | 4  | 4  | 4  | 4  | 5  | 3  | 4  | 2  | 4  | 4  | 4  | 5  |
| 8  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 2  | 5  | 1  | 4  | 4  | 2  | 4  | 4  | 2  | 2  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 5  | 3  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  |
| 9  | 2  | 5  | 3  | 3  | 3  | 3  | 4  | 4  | 4  | 4  | 5  | 1  | 4  | 4  | 3  | 4  | 5  | 5  | 5  | 4  | 4  | 5  | 5  | 5  | 5  | 5  | 5  | 5  | 3  | 3  | 3  | 3  | 5  | 5  | 3  | 4  | 4  | 4  | 5  | 4  | 5  | 5  | 3  | 3  |
| 10 | 4  | 3  | 2  | 3  | 2  | 2  | 3  | 3  | 4  | 2  | 2  | 2  | 2  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 2  | 2  | 2  | 4  | 2  | 2  | 4  | 3  | 3  | 2  | 4  | 4  | 4  | 2  | 2  | 2  | 2  | 2  | 4  |
| 11 | 3  | 5  | 2  | 2  | 4  | 2  | 2  | 4  | 2  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 5  | 5  | 2  | 4  | 3  | 2  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  | 4  |

P = Participant number  
5=Strongly Agree; 4= Agree; 3=No Opinion; 2=Disagree; 1=Strongly Disagree
Appendix X: Graphical representation of Kendall’s Coefficient of Concordance for Progressive Pain
Appendix Y: Graphical representation of Kendall's Coefficient of Concordance for Headache