June 2008 literature review cervical instability (full report)

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Background

The cervical spine has great mobility at the expense of mechanical stability along with a close neurophysiological connection to the vestibular and visual systems. Consequently it can be the source of a plethora of symptoms that would not arise from any other musculoskeletal region of the body (Kristjansson 2005).

Clinical cervical spine instability (CCSI) is controversial and difficult to diagnose. Within the literature, no clinical or diagnostic tests that yield valid and reliable results have been described to differentially diagnose this condition (Cook et al 2005, Richardson et al 1999). Cervical spine pain is a common musculoskeletal condition reportedly affecting 70% of people within their lifetime. Instability is one element of cervical pain and may contribute to the clinical presentation of various conditions, including cervicogenic headaches, chronic whiplash dysfunction, rheumatoid arthritis, osteoarthritis, and segmental degeneration. Situations involving trauma, genetic predisposition, disc degeneration, and surgery may compromise the stabilizing mechanisms of the cervical spine (Cook et al 2005).

There is a wide spectrum of conditions that come under the term ‘instability’ some of which can be serious and life threatening such as instability of the transverse ligament associated with rheumatoid arthritis, an inflammatory cause or downs syndrome, a congenital cause (Swinkles at al 1996). Kesson and Atkins (2005) describe the possibility of drop attacks being produced by more severe instability of the upper cervical segment which may be due to congenital ligamentous laxity of the atlanto-occipital joint, deformed odontoid peg, cervical spondylosis or spondylolysthesis.

According to Swinkles at al (1996) post whiplash patients with a minor non life threatening instability that results in persistent pain would be the other end of this spectrum. However in a literature review of the pathogenisis of upper cervical instability, Swinkles et al (1996) found much less information available regarding minor instabilities that may be responsible for chronic pain presentations commonly encountered by manual therapists.

Instability is described by Maitland (1986) as an intervertebral joint that has laxity of supportive ligaments permitting abnormal movement making it unstable. Maitland (1986) also refers to hypermobility in the spine as one or more intervertebral joints that are excessively mobile in relation to the neighbouring joints.

Radiographically appreciable cervical spine instability (RACSI) leading to compression of neural or vascular structures, pain, and neurological signs and symptoms reflects marked disruption of passive osseoligamentous anatomical constraints and hypermobility. Dysfunction of the active and neural subsystems is more appropriately described as an abnormality of movement rather than hypermobility and has been referred to as clinical cervical spine instability (CCSI), nonradiographic or minor cervical instability. It may demonstrate only subtle symptoms and clinical examination features and radiographic findings are frequently normal (Cook et al 2005).

The upper cervical spine differs functionally and anatomically to the rest of the cervical spine and these differences render the atlantoaxial joint vulnerable to subluxation. The bony structure facilitates mobility rather than stability 55% of cervical rotation taking place at the atlanto axial joint. The upper cervical spine ligaments (transverse ligament, alar ligaments and tectorial membrane) have an essential stabilising role (Swinkles et al 1996.)

Total range of motion (ROM) in the spine can be divided into two components, the neutral zone (NZ) and the elastic zone (EZ). Initially, minimal resistance to motion is encountered as the spine moves through the NZ, after this continuing movement is accompanied by higher levels of resistance, as the spine moves within its EZ. Spinal instability is associated with an increase in the neutral zone (NZ) of the spine. The fact that spinal fusion, an extreme treatment for instability has been shown to remove pain in patients with whiplash injury suggests that instability and pain are strongly related **(**Klein et al 2001).

Spinal ligaments therefore offer most restraint towards the end of the range of movement but they do not provide substantial support in neutral joint postures. This is dependent on adequate neuromuscular control, disruption of which is one concept of spinal instability (Richardson et al 1999). Spinal segments are highly flexible (Richardson et al 1999) or exhibit least stiffness (Hodges 2005) around the region of the neutral position. Motion occurs in the NZ against minimal internal resistance, the ligamentous structures only providing restraint in the EZ that limits end range of movement (Richardson et al 1999). Neutral zone size may increase in situations of clinical instability therefore requiring more muscle activity (Hodges 2005).

Clinical spinal instability is a significant decrease in the capacity of the stabilizing system of the spine to maintain the intervertebral neutral zones within the physiological limits (Niere and Torney 2004, Richardson et al 1999). Minor Cervical Instability (MCI) is an increase in the neutral zone associated with one or more segments within the cervical spine. This condition may be associated with a number of signs and symptoms which could include neurological dysfunction, and pain. Clinical features may be subtle potentially making it difficult to diagnose. It should not include severe incapacitating pain or symptoms indicative of spinal cord compression or vertebral artery dissection (Niere and Torney 2004).

Symptoms and Clinical findings

Niere and Torney (2004) and Cook et al (2005) surveyed physiotherapists with postgraduate qualifications in manipulative physiotherapy, experienced in the management of neck conditions on the importance of clinical findings in the diagnosis of MCI. The clinical findings considered important were;

- a history of major trauma
- unpredictable symptoms
- frequent acute attacks
- episodes with no precipitating cause
- apprehension when moving neck into extension or difficulty returning from extension
- sharp pain with sudden movements
- neck crepitus, catching, clicking, clunking or popping sensations

- locking or giving way
- feeling of a lump in the throat
- aberrant movement.
- motion not smooth throughout range (segmental hinging, pivoting or fulcruming)
- poor muscular control
- shoulder girdle weakness/atrophy
- feeling of instability, shaking, or lack of control
- feeling of 'heavy head', or 'head is dropping off'
- intolerance to prolonged static postures
- fatigue and inability to hold head up
- better with external support (hands or collar)
- frequent need for self-manipulation,
- signs of hypermobility on X-ray
- excessively free end-feel on passive motion testing
- young females with long thin necks
- poor response to previous treatment
- good response to stabilising treatment

Niere and Torney (2004) suggest that therapists treating patients with neck conditions should at least consider the possibility of MCI when presented with any of the above findings. They conclude no "gold standard" exists to positively identify MCI and there is little research regarding how reliably the factors identified as important can be assessed in clinical practice. They suggest a starting point would be to identify groups of clinical findings that reliably predict the outcome of specific treatment.

Mechanisms

Niere and Torney (2004), Swinkles et al (1996) and Richardson (1999) describe spinal stability being dependent on three interactive subsystems; passive, active and neural. Swinkles et al (1996) describes the passive subsystem consisting of vertebrae, intervertebral discs, ligaments, joint capsules and the passive properties of the spinal muscles. According to Jull et al (2005) the osseoligamentous system contributes 20% to cervical spine mechanical stability, 80% being provided by the surrounding neck musculature. Passive dysfunction of the stabilising system may be associated with hypermobility on X-ray, spondylolisthesis, history of repeated microtrauma, history of major trauma and traction spurs (Niere and Torney 2004).

The active subsystem consists of the spinal muscles and tendons. The neural subsystem consists of the force and motion transducers in the ligaments, tendons and muscles as well as the neural (motor and sensory) control centres. When any of these subsystems are subject to injury, degeneration or disease the other systems may compensate to maintain stability (Swinkles et al 1996).

Cervical instability may exist where there is pain and disability due to lack of control over neutral zone motion without compromise of vascular or neural structures (Niere and Torney 2004). It can also be associated with cervical myelopathy and radiculopathy (Cook et al 2005). In addition muscular atrophy may arise due to primary damage to muscular tissue, or as a result of disuse due to pain. This muscular atrophy may compromise function of the active subsystem of the cervical spine, resulting in pain, headache and muscle spasm (Niere and Torney 2004).

Panjabi et al (1998) carried out an experiment to quantify intervertebral mechanical flexibility changes of the cervical spine at all levels due to simulated whiplash trauma. Using fresh cadaveric human cervical spine specimens intervertebral flexibility tests were performed before and after whiplash trauma of increasing severity and the neutral zone (NZ) was found to be a more sensitive parameter than the range of movement (ROM) in determining the physical changes in the spine due to the trauma.

In a similar experiment Kettler et al (2002) investigated the importance of muscular control over unstable segments by testing whether mechanically simulated muscle forces of splenius capitits, semispinalis capitis and longus colli stabilised intact and injured cervical spine specimens. The experiment was performed on intact upper cervical spine cadaveric specimens and after unilateral and bilateral transection of the alar ligaments. Mechanically simulated cervical spine muscles were found to strongly stabilise intact and injured cervical spine specimens. As in the Panjabi et al (1998) experiment the injury-related increase of the neutral zone (NZ) was higher than that of the range of movement (ROM). NZ increasing with ligamentous injury much faster than ROM.

The physiotherapists surveyed by Cook et al (2005) reported neck symptoms secondary to instability to be better in unloaded positions such as lying down. The explanation is offered that this position may reduce intolerance to segmental physiological loading. Mid-postural position of the cervical spine displays the highest area of load sensitivity and hypothetically is the posture that requires the most dynamic control of the neutral zone and is therefore the position most prone to instability. Lincoln (2000) suggests pain at rest indicates inability to maintain a sufficient neutral zone (NZ) to prevent abnormal stresses on pain producing structures.

Long-term whiplash symptoms can be due to soft tissues which have been stretched beyond their elastic limit but not completely ruptured (Niere and Torney 2004.) Krakenes et al (2002 and 2003) report in a cohort of patients with chronic whiplash associated disorder (WAD) lesions in the upper cervical ligaments at the craniovertebral junction were visualised on MRI. This study indicated that the alar and transverse ligaments and the tectorial and posterior atlanto-occipital membranes can be damaged by whiplash injuries.

Relationships have been identified between headaches and instability within the upper cervical spine. Dysfunction of the deep neck flexors (longus colli and longus capitus muscles) has been identified in people with cervicogenic headache and whiplash. The coexistence of poor coordination and strength of the deep neck flexors and cervical spine instability may therefore contribute to cervicogenic symptoms such as headaches. Over activity of the upper trapezius muscle has also been identified in people with long-term, chronic instability-related conditions which is further evidence suggesting a distortion of motor control strategies Cook et al (2005).

In a case report of a patient with facial pain aggravated by active cervical spine movements Lincoln (2000) suggests pain felt in the head, throat and neck can be due to nociceptive information from the trigeminocervical nucleus which receives afferents from the trigeminal nerve, C1-3 spinal nerves and the VII, IX and X cranial nerves. Due to the convergence of primary afferent fibres onto common second-order neurones in the trigeminocervical nucleus, it is possible mechanical or chemical stimulation of cervical spine afferents may result in the pain being felt in the cervical spine receptive field, and/or the trigeminal receptive field.

Possible mechanisms suggested by Lincoln (2000) are mechanical deformation of the transverse ligament which is innervated by the sinuvertebral nerves arising from the ventral rami of the C1-3 spinal nerve roots or a cervical spine protruded posture at rest mechanically deforming C1 and C2 spinal nerves themselves.

Swinkles et.al (1996) describes the pharynx having lymphatic connections with the atlanto-axial region. Infections in the tonsils, middle ear, teeth and nose may drain into the cervical spine region and subsequent inflammation may lead to loosening of the transverse ligament attachments and cranio-vertebral hypermobility (Grisel's Syndrome). Grieve (1988) also describes this phenomenon in children and adolescents post upper respiratory tract infection.

The patient in the case report by Lincoln (2000) had been under the care of an ENT specialist for 2 years for persistent problems in the nasal area and reported a short-term aggravation of symptoms associated with the infuenza virus. A larger than normal amount of movement with less resistance appreciated by the therapist was found during the Sharp Purser test and Lincoln (2000) goes on to suggest this increased mobility could be due to chronic loosening of the transverse ligament over time secondary to chronic nasal inflammation.

Manual testing techniques

The clinical guidelines for the physiotherapy management of whiplash associated disorder (Moore et al 2005) recommend that joint integrity tests should only be applied by physiotherapists with specialist training in this area. The guidelines also list symptoms indicative of upper cervical instability that suggest a need for further medical investigation rather than physiotherapeutic tests for instability. These include inability to support the head, dysphagia, tongue parasthesia, metallic taste, face/lip/bilateral/quadrilateral limb parasthesia, nystagmus and gait disturbance.

Numerous clinical tests for cervical spine instability exist most examining the integrity of the tectorial membrane, alar and transverse ligaments (Cook et al 2005). Of these only the sharp pursers test has been validated and this was on a group of patients with rheumatoid disease (Swinkles at al 1996). According to Cook et al (2005) the Sharp-Purser test has been found to be a valid indicator for detection of radiographic instability however this method was not consensually chosen as an identifier for CCSI by the postgraduate physiotherapists experienced in manual therapy who participated in their survey.

The sharp pursers test is described by Lincoln (2000). At rest in sitting the therapist stands on the side of the patient's pain and glides the head posteriorly whilst stabilizing C2. This is similar to the headache SNAG technique described by Mulligan (1995) which was used as a treatment technique in the case study by Lincoln (2000). Nearly all manual instability testing methods require very skilled assessment, and have not been corroborated by simultaneous diagnostic measurement. Further difficulties arise due to passive joint assessment or palpatory tests traditionally having poor interrater reliability. (Cook et al 2005)

Piva et al (2006) carried out an assessment of inter rater reliability of passive intervertebral movement (PIM) testing to identify hypomobility in the cervical spine associated with neck dysfunction. Substantial to moderate reliability of occipital–atlas mobility was found. Poole et al (2004) looked at the consistency of assessing PIM from the occiput to T2 and found levels of agreement varied from poor to substantial. Smedmark et al (2000) reported fair to moderate levels of agreement for PIM testing in the cervical spine.

Kaale et al (2007) carried out a study comparing passive mobility testing of upper cervical spine ligaments and membranes with MRI findings in both whiplash patients and normals. Five different neck structures were considered; the left and right alar ligaments, the transverse ligament, the tectorial membrane and the posterior atlanto-occipital membrane. The clinical tests used were based on evaluation of passive intervertebral motion (PIM) performed through full range or until a muscular contraction occurred due to pain in the area. Illustrations and detailed descriptions of how the passive tests were performed are given in the article.

Loss of collagen integrity as judged by MRI was compared with degree of increased mobility as judged by the clinical examination. This was carried out in both whiplash patients and normals enabling exploration of the relationship between history of trauma, clinical examination of passive movement and MRI findings.

Good agreement was found between clinical examination using PIM testing and MRI in the assessment of abnormalities of ligaments and membranes in the upper cervical spine. This suggests PIM assessment techniques to be a useful clinical tool and that MRI findings are of clinical relevance. However the clinical tests used in this study were performed by a specialist manual therapist experienced in their application and the results from other studies of PIMs have shown varied reliability and validity. Additional studies are therefore needed to develop these clinical test procedures (Kaale et al 2007).

The value of MRI as a ‘gold standard’ against which to compare stability testing is questioned by Kongsted et al (2008) given that literature regarding the relationship between MRI findings and symptoms in patients with whiplash injury is contradictory. They looked at the predictive value of cervical MRI after whiplash injuries in 178 patients an average of 13 days after injury. The population was screened by standard procedures in the emergency unit and therefore represented patients considered to have soft tissue injuries. Findings of possible traumatic origin were observed in only seven participants suggesting that traumatic findings visible at standard cervical MRI are rare following whiplash injuries. However a tendency towards more severe neck pain and headache was observed in subjects with traumatic findings (Kongsted et al 2008).

In this study baseline MRI was performed on average 11 days and in 20 cases more than 3 weeks post accident. It is possible signs of minor tissue damage visible on MRI could have resolved before MRI examinations were performed. However the authors point out this may not have affected results as six of the seven cases with traumatic findings had MRI performed between 8 and 22 days after injury (Kongsted et al 2008).

Manual Therapy Treatment

Kesson and Atkins (2005) highlight any upper cervical segment instability associated with rheumatoid arthritis, trauma, downs syndrome along with radiographically appreciable cervical instability are absolute contraindications to orthopaedic medicine cervical mobilisation and manipulation techniques.

Maitland (1986) suggests patients with symptoms arising from a hypermobile or unstable joint can be treated with mobilising techniques. Once the joint has been made symptom free the patient must be shown exercises to strengthen the muscular support around the hypermobile or unstable segment. When pain is not easily relieved then stabilizing exercises should be added early in the treatment program and again if pain is aggravated by mobilising exercises then stabilising exercises are more appropriate.

In the case study by Lincoln (2000) the sustained natural apophyseal glide (SNAG) technique for headache described by Mulligan (1995) was found to be effective at relieving head, ear and facial pain. The explanation is offered that the technique realigns the atlas on the axis therefore decreasing stress on all surrounding pain-producing structures.

Exercise Programmes

While ligaments provide stability mostly at end range postures, muscles provide dynamic support around neutral and mid range postures where most functional daily tasks are performed. Muscles are therefore subject to constant internal and external forces and in the presence of injury or pathology to the ligamantous system their stabilising role becomes even more important (Jull et al 2005).

Studies using electromyography (EMG) have identified myoelectric manifestations of sternocleidomastoid (SCM) and anterior scalene (AS) muscle fatigue are greater in people with chronic neck pain compared to healthy controls. This could be attributed to differences in the recruited motor unit pool or a result of different control strategies employed by neck pain patients (Falla et al 2006)

In a study by (Klein et al 2001) the range of axial rotation of the cervical spine and the onset and activity of the sternocleidomastoid (SCM) muscles in healthy control subjects and in patients with chronic whiplash-associated disorder (WAD) was investigated. The hypothesis being that patients with WAD would have an earlier onset of neck muscle activity to compensate for ‘instability’.

The healthy controls reached a point in the range where SCM muscle activity rose steeply thought to be the EZ. Many of the patients did not reach this point, their movements remaining mostly within the NZ region of low muscle activity. The whiplash patients appeared either unable or unwilling to drive the cervical spine into the region of high muscle activity and therefore did not show the hypothesised earlier muscle activation (Klein et al 2001).

A suggested explanation of these findings is given. SCM spans many vertebrae, and when activated exerts both rotational and compressive forces along the axis of the spine. Axial rotation greater than 35° causes muscle and joint reaction forces to increase rapidly; applying an elevated force to an already damaged spine may cause pain, which in conjunction with increased tension of the passive tissues would cause the patients to avoid such movements. This characteristic may not be specific to whiplash patients and could be a manifestation of any painful neck disorder (Klein et al 2001).

Klein et al (2001) conclude that lack of early activation of SCM in this group of patients may be due to instability being a less important mechanism in these patients or that the deeper, short neck muscles are more important for spinal stability than the SCM.

Falla et al (2006) recommend general strengthening and endurance exercises for the cervical flexor muscles in the treatment of neck pain to improve the strength and reduce fatigability of neck muscles. Training the cervical flexors is described using a controlled head lift exercise, focusing on endurance by increasing the number of repetitions (Falla et al 2006)

According to Jull et al (2005) the more superficial multisegmental muscles are responsible for maintaining equilibrium of external forces so that load transmitted to the spinal segments can be efficiently controlled by the deep intersegmental muscle system. Neck pain patients have been shown to have disturbance in neck flexor synergy where impairment in the deep muscles important for segmental control is compensated for by increased activity in the superficial muscles. Therefore addressing strength alone in research and exercise programmes may be an oversimplification of the problems in the neuromuscular system.

Jull et al (2005) therefore recommend low load training of the cranio-cervical flexor muscles longus capitis and colli, rather than the superficial flexors, SCM and AS muscles (which flex the neck but not the head). It is suggested that craniocervical flexion (deep neck flexor) exercises relieve pain by improving coordination between layers of the cervical flexor muscles, and correcting aberrant patterns pf muscle activation responsible for overloading cervical structures and compromising joint stability. This may directly affect the pain system rather than increasing strength or fatigability of the superficial cervical flexor muscles. Detailed descriptions of how to analyse the aberrant movement patterns observed in patients are also given by Jull et al (2005).

Falla et al (2006) compared these two approaches in 58 female patients with chronic non-severe neck pain. At the 7th week follow-up assessment, the endurance-strength training group revealed a significant increase in MVC (maximum voluntary contraction force) and a reduction in myoelectric manifestations of superficial cervical flexor muscle fatigue as well as increasing cervical flexion strength. Physiological adaptation in the neuromuscular system and changes in control strategies are suggested meachanisms for the reduction in myoelectric manifestations of fatigue observed in the endurance training group in this study. Changes in electrophysiological parameters were not observed in the low load craniocervical flexor training group.

Both exercise groups reported a reduced average intensity of neck pain and reduced neck disability index score and there was no difference in pain or disability measures between the 2 groups at the end of training. This suggests improvements in strength and a reduction in muscle fatigue may be due to factors other than symptomatic improvement. (Falla et al 2006).

Lincoln (2000) suggests once vertebral alignment has been achieved using manual therapy techniques it is necessary to train the neuromuscular subsystem to maintain this position. Upper cervical spine flexion movement recruits superficial and deep neck flexor muscles. These muscles, particularly longus colli play a postural, supporting role enabling segmental stability. Lincoln (2000) suggests that the patient in their case study benefited from gentle chin retraction exercises in lying for two reasons. Firstly retracting the head in supine would also retract the atlas vertebra into alignment with the axis and secondly this movement may have been recruiting deep neck flexor muscles, resulting in improved segmental muscular control of the upper cervical region. This muscular activation has also been shown to improve proprioceptive function of the head and neck.

Proprioception

Proprioceptive reflexes of the neck originate from Mechanoreceptors in the cervical joints and muscles and are an important source of afferent information from the proprioceptive system to the postural control system. These afferents influence ocular control as well as proprioceptive and vestibular integration. The importance of cervical proprioception in the control of posture, spatial orientation and coordination of the eyes has been demonstrated in experimental studies and in studies with neck pain patients. Morphological changes have also been demonstrated in the muscle of neck pain patients that may influence the proprioceptive capabilities of the muscle. These include fatty infiltration and changes in muscle fibre type (Jull et al 2005).

Patients with whiplash associated disorder may demonstrate a deficit in their ability to reproduce a target position of the neck or to find a neutral position of the neck when compared to asymptomatic controls suggesting the importance of retraining proprioceptive function in these patients (Moore et al 2005).

Conclusion

Cervical instability is a broad term encompassing a wide range of symptom presentations and pathology. Minor cervical instability is a possible finding in many neck pain patients presenting for treatment. This review would suggest it is a multifaceted problem requiring careful assessment and application of a combination of treatment techniques. Establishing the most effective management approaches will require further research and debate.

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