REVIEW ARTICLE

Hyperextension soft tissue injuries of the cervical spine – a review

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The term “whiplash injury” was first used by Crowe in 1928 to describe a mechanism of injury in which acceleration forces produced cervical spine injury.¹ Although acceleration forces can produce a spectrum of injury, with vertebral column and spinal cord injury at its most severe end, over the years the term has become synonymous with the syndrome of soft tissue injury to the neck which in a proportion of patients is associated with prolonged pain and disability, often culminating in litigation for personal injury.

The potential for acceleration forces to produce hyperextension injury to the neck was first recognised in pilots being catapulted from aircraft carriers.² Modification of the pilot's seat was undertaken to limit hyperextension, and the injury effectively prevented. It was not until the emergence of the motor car as a popular method of urban transport that the association of rear end collisions, which accelerated the occupants of a vehicle, and soft tissue neck injury was widely recognised. Up to 62% of patients attending hospital after road traffic accidents complain of neck pain at some stage, though in many the onset of pain is delayed.³

While many patients make an uncomplicated recovery consistent with a simple musculo-ligamentous injury, in those cases in which there are prolonged symptoms and disability there has been much dispute over the interplay of injury, psychological factors, and attempts to manipulate personal injury claims. Early reports concluded that there was little to suggest an organic basis for the patients' continuing complaints. Gay and Abbott⁴ noted the protracted clinical course despite a low velocity impact, the preponderance of females, the absence of objective physical signs, and the association with symptoms such as anxiety, fatigue, and irritability. Liability for rear end collisions almost always lay with the insurers of the other vehicle, and in their series 66% of patients were pursuing claims for damages.

Gotten⁵ assessed the effect of completion of litigation on recovery and concluded that the injury was being used for personal gain. This report gained widespread acceptance and the persistence of symptoms after soft tissue injuries of the cervical spine was often thought to be an attempt by the patient to maximise personal injury damages. A further study reinforced this belief by correlating duration of symptoms with litigation and emotional factors.⁶

Opposing opinion developed from the work of McNab⁷ who identified features of the injury which were not consistent with litigation neurosis.⁸ Other injuries sustained in the same accident healed promptly and did not become the source of continuing complaint and in his series symptoms were not influenced by the outcome of litigation. He also noted that patients with forward or lateral flexion injuries rarely developed significant symptoms compared with those injured by hyperextension. The high incidence of neck pain after rear end impacts has been confirmed by other investigators.^{4 9 10}

Scepticism concerning the cause of prolonged symptoms remains widespread.¹¹ A survey published in 1994 showed that a large minority of doctors believed psychogenic and litigation factors were predominantly responsible for the condition and that symptoms resolved after settlement of personal injury claims.¹² Few subjects are as likely to provoke such vigorous debate and difference in opinion – especially with respect to the medicolegal assessment of personal injury – as the so called whiplash injury. There is a wealth of published reports which I shall review in this paper with respect to the pathogenesis, treatment, and prognosis of hyperextension soft tissue injuries of the cervical spine. The evidence for associated injuries to the brain, ear, eye, and temporomandibular joints is also examined and the question of whether this injury predisposes to premature degenerative change in the cervical spine is addressed.

Biomechanics and pathogenesis

The occupants of a vehicle struck in the rear are statistically more likely to develop a multiplicity of symptoms, including evidence of cranial nerve or brainstem dysfunction, than when other directions of impact are involved.¹⁰ Rotated or inclined head position and an unprepared occupant are also more likely to lead to the development of multiple symptoms.¹⁰

An understanding of the forces acting upon the cervical spine during a low velocity rear end collision has followed the work of Severy *et al*¹³ in 1955. Using accelerometers and cinematography in simulated rear end collisions to static vehicles with human volunteers or anthropomorphic mannikins as subjects, it was shown that hyperextension was the mechanism of injury, and not hyperflexion as previously postulated by Gay and Abbott.⁴ Head

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accelerations of up to 11.4 g were recorded at collision velocities of less than 20 mph. Maximum head extensions of 122 degrees were seen, the normal maximum being between 60 and 75 degrees. This therefore showed conclusively that significant forces were being generated in low speed impacts which produced cervical spine extension beyond the normal range of motion.

Animal experimentation with monkeys was used by McNab⁷ to determine the injuries produced by these forces. Muscle injuries varying from minor tears of the sternomastoid to major tears of the longus colli were seen in association with retropharyngeal haemorrhage, contusions of the oesophagus, and injuries to the cervical sympathetic chain. Some animals had tears of the anterior longitudinal ligament with separation of the intervertebral disc from the vertebra above. This specific injury has been demonstrated surgically^{14 15} and more recently by magnetic resonance imaging (MRI)¹⁶ after cervical spine hyperextension injury in humans.

Subsequently further work using monkeys¹⁷ found a high incidence of facet joint injury which was not apparent on x ray. Capsular tears with fissuring of the articular cartilage and subchondral bone fracture were found along with paraspinous muscle and ligamentous injury.

Necropsy examinations of the cervical spines of road traffic accident fatalities have shown a high incidence of injuries despite normal radiographs. Taylor and Kakulas¹⁸ showed a 96% incidence of vertebral disc injury and a 72% incidence of facet joint injury in the cervical spine at 43 necropsies. Examination of the spines of two patients who died 14 months

and 3.5 years after neck injury and who had persisting symptoms from their necks in the intervening period showed clefts in the intervertebral discs which had not been identified radiologically.

McNab⁷ examined the question of why hyperextension produced injury whereas forward and lateral flexion were rarely associated with significant problems. He suggests that in flexion injuries, movement was stopped within the anatomical range either by contact between the chin and the chest or between the ear and the shoulder. In extension, there is no bar to movement till the occiput reached the posterior chest wall, well beyond the normal range of motion.

The use of head restraints in cars to limit cervical hyperextension is potentially effective in preventing injury¹⁹ though one study found only a small effect which failed to reach statistical significance²⁰. It has been suggested that the design and adjustment of the restraint may often be at fault, as effective head restraints would conflict with comfort and vision requirements.²¹ The use of seat belts appears to increase the rate of neck injury.^{22 23}

Prognosis

The assessment of prognosis for personal injury claims is a common area of disagreement. Hyperextension soft tissue injury to the cervical spine is common. Up to 42.6%²⁴ of all road traffic accidents involve rear end impact, with about 20% of occupants sustaining some type of neck injury.²⁵ In American practice there are up to 1 000 000 injuries a year²⁶ with over 50% of those injured seeking legal advice.¹ These injuries now account for 34% of all compensation awards for road traffic accidents.²⁷

There is a vast literature on prognosis in patients with soft tissue hyperextension injury of the cervical spine. Comparison of these studies is hindered by differences in the populations studied, the methods used, and the outcomes measured. Various investigators have examined patients presenting to emergency departments, for specialist opinion or for medicolegal assessment. Outcome measures are subjective and do not allow comparison between series. A summary of the larger and more recent series is given in table 1. It appears that between 26% and 44% of patients with soft tissue cervical spine injury will develop significant long term problems. Final prognosis for recovery may be estimated at between two months after injury³¹ and at two to three years.^{29 32}

Prognostic factors

Attempts to predict prognosis on the presence of clinical or radiological factors present in the early stages after injury have been made by several investigators. Norris and Watt³⁶ grouped patients by the presence of symptoms, restricted neck movements, and neurological signs at presentation. At reassessment two years after injury 56% of those with symptoms only at presentation were symptom-free, whereas only 19% of the group with restricted

Table 1 Prognosis after hyperextension soft tissue injury of the cervical spine.

Authors	Population	Outcome
Balla ²⁴	All Motor Accident Board cases	At 6 months 74% normal activity
Livingston ²⁸	Primary care	Recovery 40% at 4 weeks 73% at 3 months 85% at 6 months
Deans <i>et al</i> ⁹	Emergency department cases	At 1 year 58% Symptomatic 36% Intermittent pain 6% Severe pain
Gargan and Bannister ²⁹	Emergency department cases	At 10-8 years 28% Intrusive symptoms 12% Severe symptoms
Hildingsson <i>et al</i> ³⁰	Emergency department cases	At 2 years 42% Recovery 14% Minor discomfort 44% Major complaints
Maimaris <i>et al</i> ³¹	Emergency department cases	At 2 years 66% Asymptomatic
Parmar and Raymakers ³²	Medicolegal	Significant pain 50% at 8 months 22% at 2 years 18% at 3 years 14% at 8 years
Robinson and Cassar-Pullicino ³³	Medicolegal	At 13.5 years 86% Persistent symptoms
McNab ⁷	Orthopaedic clinic	2 years after litigation settled 45% Persistent symptoms
Hohl ³⁴	Orthopaedic clinic	Greater than 5 years 43% persistent symptoms
Radanov <i>et al</i> ³⁵	Research clinic	Symptomatic patients 44% at 3 months 31% at 6 months 24% at 12 months

neck movements and 10% of the group with neurological signs had recovered.

Some investigators have been unable to find any useful predictors of outcome³⁰ while others have found various combinations of factors that predict prolonged symptoms (table 2). The influence of behavioural abnormalities on recovery has been the subject of much research. Farbmann⁶ suggested that emotional factors could be responsible for delaying recovery. More recent findings suggest that psychological factors at the time of injury do not influence the development and course of symptoms³⁸ and that improvement in these indices occurs following recovery from the somatic symptoms.⁴²

Gotten⁵ was first to suggest that this injury was being used by patients to maximise damages for personal injury. Although this may occur in individual cases, the majority of studies have failed to show that settlement of litigation has a beneficial effect on symptoms.^{7 32 36} The suggestion has been made that the public, through peer copying,⁴³ has come to expect treatment and compensation for soft tissue injuries of the cervical spine. However, public knowledge of the symptoms that may follow a hyperextension cervical spine injury, especially cognitive symptoms, and the impact speeds needed to produce injury, is poor.⁴⁴

Extracervical injury

CENTRAL NERVOUS SYSTEM

Patients commonly report symptoms such as irritability, emotional lability, insomnia, and difficulties with concentration after cervical spine hyperextension injury. Cerebral symptoms after a neck sprain have been taken as an indication of neurotic personality⁴⁵; however, experimental studies have shown that acceleration forces can produce significant intracranial injury in the absence of direct

head impact. Ommaya and colleagues,⁴⁶ using rhesus monkeys, were able to demonstrate macroscopic cerebral injury in 15 out of 19 animals concussed by the injury. Changes in intracranial pressure⁴⁷ and blood-brain barrier permeability⁴⁸ have also been shown in animals in laboratory simulations.

Evidence that humans may sustain brain injury during an acceleration injury is provided by neuropsychological tests which have consistently shown deficits in attention, concentration, and memory consistent with injury to frontobasal and upper brain stem structures.⁴⁹⁻⁵² These deficits have been found both early⁵⁰ and late⁴⁹ after the injury. Structural lesions in humans have, however, been more elusive. Examinations with MRI and brainstem auditory evoked potentials,^{49 50} sensitive investigations for brainstem lesions, have been normal. Until a structural abnormality is demonstrated, scepticism as to the true nature of these symptoms will continue.⁵³

TEMPORO-MANDIBULAR JOINTS

The occurrence of temporo-mandibular joint (TMJ) dysfunction with acceleration injuries of the cervical spine was first recognised in 1965⁵⁴; however, it is rarely acknowledged as being part of the syndrome of injury, and the temporo-mandibular joint is not often assessed in the early stages after presentation. The clinical picture is of TMJ pain with limitation of mouth opening and masticatory muscle tenderness.⁵⁵ Symptoms are often minor but 30% of patients would seek treatment for them.⁵⁵

The mechanism of injury is thought to be excessive mouth opening during maximum cervical spine hyperextension, with anterior subluxation of the mandibular condyles and injury to the intra-articular disc, ligaments, and synovium of the joint.⁵⁶ A recent study using MRI has shown an 88% incidence of TMJ abnormality in symptomatic patients, including intra-articular disc displacement, joint effusions, and oedema.⁵⁷ Specific early treatment, including exercises and intra-oral appliances, may help to minimise the morbidity of this component of the injury.⁵⁸

A routine assessment of the temporo-mandibular joint should be included in the examination of a patient following acceleration injury to the cervical spine, and referral to a maxillofacial specialist arranged for patients with persisting abnormalities.

THE EYE

The effects of acceleration injuries on the visual system have been recognised for many years but were not until recently documented objectively. Horner syndrome was the first reported ophthalmic manifestation of these injuries⁵⁹ but is rare, whereas oculomotor abnormalities are relatively common in symptomatic patients.⁶⁰

In a recent prospective ophthalmic assessment of 39 patients with soft tissue injuries to the cervical spine presenting to an accident and emergency department,⁶¹ 10

Table 2 Prognostic factors for continuing symptoms

	Associated with poor prognosis	No association with prognosis
Upper limb pain	Hohl ³⁴ Maimaris <i>et al</i> ³¹ Watkinson <i>et al</i> ³⁷ Radanov ³⁵	Parmar and Raymakers ³² Hildingsson <i>et al</i> ³⁰
Thoracolumbar pain	Maimaris <i>et al</i> ³¹ Watkinson <i>et al</i> ³⁷	
Age	Maimaris <i>et al</i> ³¹ Radanov <i>et al</i> ³⁸ Gargan and Bannister ²⁹ Miles <i>et al</i> ³⁹	
Psychosocial factors		Radanov <i>et al</i> ³⁸
Neurological signs	Hohl ³⁴ Maimaris <i>et al</i> ³¹ Norris and Watt ³⁶	Hildingsson <i>et al</i> ³⁰
Pre-existing cervical spondylosis	Maimaris <i>et al</i> ³¹ Norris and Watt ³⁶ Miles <i>et al</i> ³⁹ Watkinson <i>et al</i> ³⁷ Parmar and Raymakers ³²	Hildingsson <i>et al</i> ³⁰
Reversal of cervical lordosis	Norris and Watt ³⁶ Hohl ³⁴ Miles <i>et al</i> ³⁹	Hildingsson <i>et al</i> ³⁰
Legal action in progress	Gotten ⁵ Farbmann ⁶	McNab ⁷ Norris and Watt ³⁶ Parmar and Raymakers ³² Pennie and Agambar ⁴¹ Mendelson ⁴⁰
Initial pain intensity	Radanov ^{35 38}	

patients (26%) were found to have symptoms or signs of ophthalmic dysfunction. In six cases there was reduced convergence and accommodation with superior oblique paresis in two patients and reduced stereoacuity in one. In only one case with bilateral vitreous detachments did the abnormality directly affect the eye, the other nine having lesions which were probably at brainstem level. In all but two patients the abnormalities had resolved by nine months.

Ophthalmic symptoms after acceleration cervical spine injury merit further assessment and objective physical signs can be demonstrated to localise the lesion.

THE EAR

Complaints of dizziness and disorders of balance occur in as many as 23% of patients after acceleration injuries to the cervical spine.³⁰ As clinical examination is usually normal, the pathological basis for these symptoms is unclear and has variously been attributed to vertebral artery or inner ear injury or disturbance of the neck righting reflex induced by paraspinal muscle spasm.⁷

Objective evidence of vestibular involvement has been demonstrated by the correlation of symptoms with electronystagmograms.⁶²⁻⁶³ Togliola,⁶⁴ studying a group of 309 patients with dizziness after neck injury, found latent nystagmus in 29%, abnormal caloric tests in 57%, and abnormal rotatory tests in 51%.

Chester⁶⁵ postulated that the acceleration forces could cause significant damage to the delicate middle and inner ear structures. Using a series of tests on a group of 48 patients, faulty inner ear function leading to ineffective muscular control of balance and erect posture was demonstrated. Perilymph fistulae requiring surgery were found in seven patients. Other patients were treated with a combination of physiotherapy and vestibular suppressant medication.

Do hyperextension injuries lead to degenerative diseases?

The association of soft tissue cervical spine injury with the development of premature degenerative disease was first suggested by Hohl.³⁴ Reviewing patients with no radiological evidence of degenerative disease at the time of injury, he found spondylosis in 39% at greater than five years, compared with an expected incidence of 6%. These results should, however, be treated with caution as only 27% of the original group were successfully contacted for follow up. Twenty three percent of the patients with radiological degenerative changes at review were asymptomatic and the prognosis for recovery from the injury was not adversely affected by the subsequent development of degenerative changes.

Further evidence that the initial injury may cause a structural lesion predisposing to premature degenerative change is suggested by the finding of a high incidence of history of a previous acceleration injury among a group of patients undergoing anterior cervical discectomy.⁶⁶ Age at operation was also

significantly less for those patients who recalled a previous injury.

Two studies with late radiological follow up have failed to support Hohl's data and found no increase in degenerative changes in the cervical spine after acceleration injury.³²⁻³³ However, one study contained only 21 patients³³ while the other included 81 patients for radiological review³² but analysed them by separate age groups, thereby reducing the power to detect small changes in the whole group.

Treatment

There are few prospective trials of treatment of acute neck sprains. Historically, rest with a cervical collar, simple, compound, or anti-inflammatory analgesics, and physiotherapy have been prescribed empirically.

Mealy and colleagues,⁶⁷ in a randomised study of rest and use of a cervical collar against early active mobilisation, showed significant benefits in terms of pain and movement in the actively treated group. These findings were later confirmed; however, the benefit of early active mobilisation was just as great in a group given advice by a physiotherapist regarding home exercises as in a group attending for regular active outpatient physiotherapy.⁶⁸

Low energy, high frequency pulsed electromagnetic therapy has been shown to be effective in reducing pain and improving movement after hyperextension injury⁶⁹; however, traction has been condemned as traumatic, unscientific, and unproven.⁷⁰ Pennie and Agambar⁷¹ found no benefit from early traction compared with rest in a collar and analgesics.

The use of subcutaneous sterile water injections to tender and trigger points around the neck and shoulder following hyperextension injury has been shown to be of significant and persisting benefit in improving pain and movement compared with a control group given saline injections.⁷² The mechanism of this benefit is unknown; however, intra-cutaneous sterile water injections have been found to be of benefit in myofascial pain syndromes and it is speculated that there may be either a counterirritation effect or that hyperstimulation analgesia is induced.

A double blind controlled trial of facet joint injection with steroids compared with local anaesthetics showed good relief of symptoms in all patients in the short term but no long term benefit from intra-articular steroids.⁷³ A specific role for the third occipital nerve in patients with continuing head and neck pain after injury has been suggested by a double blind trial of diagnostic local anaesthetic block of the nerve.⁷⁴ Out of 100 patients, upper neck and head pain was relieved temporarily by the block in 27%. In a subgroup in whom headache was the predominant symptom, the success rate was 53%. A subsequent trial of percutaneous radiofrequency neurotomy of the third occipital nerve is in progress.

Surgery is generally undertaken for patients with persistent and disabling head and neck pain with a radiologically proven intervertebral disc lesion. It is therefore of limited

applicability. A study of 20 patients who underwent anterior cervical discectomy and fusion four years before showed good or fair results in 11 patients and poor relief of symptoms in the remaining nine.⁷⁵

In the face of a common problem with little effective treatment for patients with continuing symptoms, many people believe that costly investigation and unproven treatments should be abandoned and the patient encouraged to remain active and cope with their symptoms.⁷⁶

Summary

While a full understanding of continuing symptoms following a soft tissue hyperextension injury of the cervical spine remains elusive, recent research has shown that the explanation may lie with occult lesions beyond the musculoskeletal structures of the neck. The balance of the roles of injury, psychological factors, and the effects of litigation has shifted towards the former. However this injury would be unique if the latter two played only a minor role in determining recovery.

It seems likely that among the large numbers of patients presenting with symptoms after hyperextension soft tissue injuries, a proportion will have occult bone, joint, or intervertebral disc lesions. Improvements in medical imaging techniques may allow better definition of these specific injuries and the development of more appropriate treatment.

The search for a central nervous system lesion in humans continues and until this is demonstrated, many will dispute the existence of an organic brain syndrome. Evidence for significant injury to the temporomandibular joints, ear, and ophthalmic system has been found and this may be amenable to specialist intervention.

While there is little evidence for effective treatments of the established injury, reduction in related disability appears most likely to be achieved by prevention. Improvements in automobile design, with particular reference to head restraints, could limit the cost to society of this common and disabling injury.

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ACCIDENT & EMERGENCY STUDY DAY to be held in **Liverpool** on **12th March 1996**. This forms part of a week of Paediatric Events being hosted by the Royal Liverpool Childrens NHS Trust Alder Hey to celebrate the opening of its Education Centre. Topics to be covered at this Study Day will include **changing patterns of meningococcal disease, the limping child, the Alder Hey Trauma System, trauma in infants, chest x-rays in paediatric emergencies**. There will also be workshops on life support in serious illness and trauma. Further details/application form available from Postgraduate Office, Education Centre, Royal Liverpool Childrens NHS Trust Alder Hey, Eaton Road, Liverpool L12 2AP. Tel: 0151 252 5218 Fax: 0151 252 5103.