School of Physiotherapy

A Study of neck injury arising from motor vehicle accidents and its clinical management.

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ABSTRACT

The syndrome commonly referred to as "whiplash injury" resulting from motor vehicle accidents is complex and remains a challenge to clinicians, as is evidenced by the recent report of the Quebec task force on the "whiplash syndrome". The main objective of this prospective randomised study was to evaluate two conservative treatment regimens (early immobilisation-experimental group-1, early active mobilisation-experimental group-2) which are based on accepted physiological rationale and then to compare their effectiveness with existing treatment regimens that are commonly practiced (control group) in the management of "whiplash" type of injuries. To this stage, the current study is the only prospective randomised clinical trial of its type conducted with a sufficiently large sample size and over a long study period. The results of the current study clearly demonstrated that the subjects in the immobilised group recovered from their pain-related symptoms and returned to their normal duties sooner than those in the other two treatment groups. In addition to this, those subjects who received the immobilisation regimen did not show adverse effects on either the range of motion or the strength of the neck muscles. Thus, the immobilisation regimen was clearly shown to be the preferred option when compared to the other two treatment methodologies investigated in the current study.

Although the primary interest of the current study was to compare the efficacy of three different treatment regimens, a series of statistical analyses were performed to establish the prognostic significance of several factors associated with "whiplash" injury. This showed that factors such as gender, age, speed of the vehicles involved, paraesthesia, intensity of pain at the time of the initial examination, interscapular pain, blurred vision and

difficulty in focusing, all had prognostic value. Similarly, the type of collision, seating position, presence of headache within 24 hours post-injury, pre-existing degenerative changes in the cervical spine, loss of lordosis and litigation factors had no prognostic significance. Another major emphasis of the current study has been to concentrate on the pain-related symptoms of the neck which are of major concern to "whiplash" subjects and to those clinicians treating them. A paucity of such information is considered to be one of the most notable causes of difficulties encountered in the management of "whiplash" injuries.

As an adjunct to the main study, the morphology of the deep pre- and post-vertebral muscles of the neck region using embalmed cadavers and fresh post-mortem specimens was investigated, as the literature is deficient in this regard. Similarly, a longitudinal study of 45 subjects was also performed using Magnetic Resonance Imaging (MRI) technology. The longitudinal nature of the M.R.I study provided for the first time an account of the details associated with the progressive pathological changes that occurred in some disc lesions, at defined points in time following a MVA. The observations made from the adjunct studies help develop a better understanding of the pathoanatomy associated with the deep muscles of the neck region and the pathological changes that occur following a traumatic disc lesion as evidenced within 2 weeks, after 3 months and 12 months post- injury. On the basis of the observations made in the current study, a classification of the "whiplash" injury has been proposed for the consideration of clinicians. Similarly, the questionnaire used for data collection in the current study, can be readily modified and utilised in a clinical situation for establishing documentation, planning treatment strategies and for evaluation of the treatment outcomes of "whiplash" type of injuries.

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Gurumoorthy, D. (1992). An antomical study of the post-vertebral muscles of the cervical region and their clinical relevance in "whiplash" type of injuries. Australian Physiotherapy Association National Congress, Adelaide.

Gurumoorthy, D. (1993). Intervertebral disc lesions resulting from "whiplash" type of injury: An M.R.I. study. 8th Biennial Conference, Manipulative Physiotherapists Association of Australia. Perth.

Gurumoorthy, D. (1994). Intervertebral disc lesions resulting from "whiplash" type of injury: A longitudinal M.R.I. study. 4th International Congress of the Australian Physiotherapy Association, Bali, Indonesia.

Gurumoorthy, D. (1996). A study of "whiplash" injury and its clinical management. Australian Physiotherapy Association National Congress, Brisbane. (Paper accepted for oral presentation).

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CHAPTER ONE INTRODUCTION

1.1 Introduction

Soft tissue injuries to the neck as a result of motor vehicle accidents (MVA) are often complex and their management has become a challenge to clinicians. The nature and the magnitude of the injury to the cervical spine and its related structures are not always fully understood (MacNab, 1964; MacNab, 1971; MacNab, 1973; Huelke and Nusholtz, 1986; Bogduk, 1995; Spitzer, Skovron, Salmi, Cassidy, Duranceau, Suissa and Zeiss, 1995). One form of cervical injury is commonly known as "Whiplash", a term coined by Crowe in 1928, to graphically represent the forces imposed on the spine during acceleration/deceleration trauma. The diagnosis of "whiplash" excludes radiologically demonstrable fractures or dislocations of the cervical spine (States, Korn and Masengill,1970; Hohl, 1975; Hirsch, Hirsch, Hiramoto and Weiss, 1988; Spitzer et al., 1995). In many respects the symptoms contained in the "whiplash" syndrome complex are similar to the complex of musculo-skeletal symptoms known collectively by the descriptive term "low back pain". The notoriety as well as the socio-economical significance of the "whiplash" injury can be aptly summed up by the following statement:

> "The same patient may be viewed by different physicians as an individual with a significant alteration in functional capacity, or, alternatively, as an individual with a partially imaginary or magnified array of symptoms"
>
> (Hirsch et al., 1988, page, 791).

The "whiplash" syndrome itself has come to include an ever increasing list of symptoms. The syndrome remains difficult to properly comprehend and

continues to defy effective management (Winston, 1987; Barnsley Lord and Bogduk., 1994; Spitzer et al., 1995). However, its socio-economical implications are abundantly clear.

The clinical management of the effects of trauma to the neck still largely remains arbitrary, nonspecific, lacking in physiological rationale and often involves a protracted period of treatment (Barnsley et al., 1994; Spitzer et al., 1995). It is also evident that establishing an adequate prognosis is often difficult (Gotten, 1956; Hirsch et al., 1988; Spitzer et al., 1995). A search of the literature offers a possible explanation for this situation. There is a plethora of publications in respect of the pathomechanics, pathogenesis, pathology and symptomatology, whereas there are very few publications on the subject of clinical management of neck injuries arising from motor vehicle accidents (Mealy, Brennan and Fenelon, 1986; McKinney, Dornan and Ryan, 1989; Pennie and Agambar, 1990; Barnsley et al., 1994; Bogduk, 1995; Spitzer et al., 1995). It is generally considered that most of the methods of treatment which are currently offered to "whiplash" patients, during the acute stage, have not been subjected to scientific scrutiny by conducting appropriate clinical trials (Barnsley et al., 1994; Bogduk, 1995; Spitzer et al., 1995). There is also a surprising lack of knowledge of the detailed morphology and function of the constituent elements of the cervical spine. This knowledge is essential for effective clinical management of cervical conditions and thus warrants further investigation (Panjabi, Oxland, Takata, Goel, Duranceau and Krag, 1993).

MacNab (1964, 1971, 1973) has confirmed the relationship between the rear end motor vehicle collision and hyper-extension of the cervical spine, which has been generally regarded as the primary mechanism of "whiplash" injury (Hohl, 1975; Bogduk, 1986). However, there is adequate

information which might lend support to the concept that in addition to hyper-extension type of forces, there could be other mechanisms of injury (e.g side impact, roll-over) responsible for "whiplash" syndrome (Deans, McGalliard, and Rutherford, 1986). It appears that MacNab (1964, 1971, 1973) may have dismissed other possible injuring mechanisms during "whiplash" without adequate verification.

Even though "whiplash" injury has long been regarded as an exclusively soft tissue injury, recent histological studies have challenged such concept. The occurrence of zygapophyseal joint fractures in a "whiplash" type of neck injury has been proposed by Twomey and Taylor (1989, 1993), and the presence of radiologically occult fractures has been histologically demonstrated by these authors. The three column effect proposed by Loius (1985) lends support to the possibility of such fractures in both the anterior and the posterior elements of the motion segment.

There are numerous studies based on cadaveric impact experiments utilising laboratory dummies, and although many of these are quite sophisticated, the experiments are deficient in one critical factor, namely the active forces generated by muscles and ligaments on the moving spine in living people (Huelke and Nusholtz,1986). It is important to understand that the amount and type of muscle contraction, resulting as a consequence of MVA, would vary between live individuals and also would be related to other factors such as the velocity of the vehicle, body size and restraints, direction of the impact and most importantly the individuals reaction due to the awareness of the impending collision. The precise role of the pre-vertebral deep muscles and the post-vertebral deep muscles in this situation are not fully understood at present (Warwick and Williams, 1973). It is obvious that due to these short comings, the results of the simulated

experiments have limited value in fully understanding the pathology and also equally in formulating effective treatment methods.

1.2 Background to this study

Spinal injuries arising from motor vehicle accidents can be either the "whiplash type" of soft tissue lesion or a combination of soft tissue lesion and bony lesion such as a fracture of the vertebra and subluxation / dislocation of the motion segment. In the cervical spine, a bony lesion is invariably associated with severe soft tissue injuries (Clark, Gehweiler and Laib, 1979). Follow-up of the two groups of patients, i.e. the "whiplash " type of soft tissue injuries and bony lesions, has highlighted some interesting differences in the speed and in the extent of recovery (Batalin, 1988). Surprisingly some of the protracted symptoms reported by the soft-tissue "whiplash" patients are not found in the patient population treated for bony lesions (Batalin, 1988). A significant number of patients with the "whiplash type" of injuries continue to have symptoms for long periods of time and often have to modify their lifestyle at a high cost to themselves and their families (Gotten, 1956). On the other hand patients treated for bony lesions and associated soft tissue lesions usually recover relatively quickly and subsequently return to apparent normality (Batalin, 1988).

This difference may be attributed to the manner in which these two groups of patients are treated. Patients suffering from fractures and associated soft tissue lesions are treated by immobilisation over a specific period. Similarly, a patient who has sustained dislocation of a motion segment /s is reduced, maintained by traction and rested for a specific period, in order to promote repair of the soft tissues which in turn would contribute to mechanical stability of the affected motion segment /s. An appropriate duration of immobilisation is considered by many orthopaedic surgeons to

be critical to achieve better healing of soft tissues (Bedbrook, 1988). However, "whiplash type" of injuries are subjected to a variety of unproven treatment procedures, which may delay the adequate healing of soft tissues as well as radiologically occult fractures (if present) (Barnsley et al., 1994; Spitzer et al., 1995).

It could be postulated that patients with radiologically demonstrable fractures of the cervical vertebra resolve significantly better, because they are placed into a specific immobilisation regime. Therefore, it is possible that these patients have fewer long term complaints as opposed to the patient population suffering from the "whiplash type" of injuries, when the latter are treated by methods other than specific immobilisation. If this postulation was valid, it is reasonable to believe that if the "whiplash type" of soft tissue neck injuries were to be treated by immobilisation, as in the case of bony lesions, should also respond in a similar manner.

1.3 Summary

On the basis of the above information the following inferences can be made. Neck injuries resulting from the motor vehicle accidents are not necessarily confined to rear-end collision alone and the injury might consist of soft tissue as well as bony lesions. There is a paucity of information about the possible damage to relevant vertebral structures such as the intervertebral disc, capsule, the zygapophyseal joint articular surfaces, pre- and post-vertebral muscles and ligaments. This fundamental knowledge is essential for a better understanding of the pathologies involved. The extent and magnitude of such injuries remains to be verified and confirmed in order to improve the understanding of the pathologies of "whiplash". A search of the literature clearly demonstrates that clinical management of the syndrome is varied and nonspecific (Hirsch et al., 1988,

Barnsley et al, 1994; Spitzer et al., 1995). Similarly, the persistence of symptoms in many cases even after settlement of litigation confirms the ineffectiveness of much of the clinical management of the "whiplash type" of injuries (Gotten, 1956; Bedbrook, 1988). This highlights the pressing need for an investigation to determine the likely pathologies involved and an appropriate conservative treatment methodology.

1.4 Aims of this study

In the light of this information, it was decided that a prospective, randomised clinical trial, consisting of three groups of subjects, with an adequate sample size should be conducted in order to verify the effectiveness of treatment received in the form of early immobilization followed by a defined exercise programme (experimental group.1), defined active exercise programme (experimental group.2) and current method of treatment as prescribed by family doctors (control group). The main objective of this study is to evalute two conservative treatment regimens based on accepted physiological rationale and also to compare their effectiveness with existing methods of treatment which are commonly practiced.

1.5 Hypotheses

Patients with a "whiplash type" of neck injury as a result of motor vehicle accident and initially treated by immobilisation for a period of 4 weeks followed by a defined active exercise programme, when compared to those patients treated by a specified active physiotherapy (defined exercise programme) and to those patients treated by their family doctor in a non specified way will have:

Hypothesis 1

a significantly lower level of resting pain as measured at 4 weeks after the injury;

Hypothesis 2

a significantly lower level of pain during active movements of the neck at 12 weeks post-injury;

Hypothesis 3

no residual pain at 24 weeks post-injury;

Hypothesis 4

no significant difference in the range of motion at 12 weeks post-injury;

Hypothesis 5

no significant difference in muscle strength at 12 weeks post-injury;

Hypothesis 6

fewer working hours lost at 24 weeks post-injury.

CHAPTER TWO LITERATURE REVIEW

2.1 Introduction

The term "Whiplash" Injury was coined by Harold E Crowe in 1928, to graphically describe the mechanism of the injury to the neck arising from motor vehicle accidents (MVA) and the related complex of symptoms (Breck and Van Norman, 1971). A few other synonyms such as necklash injury, cantilever injury, hyper-extension injury, hyper-flexion injury, acceleration injury, deceleration injury and acceleration-extension syndrome have also been used in an effort to more accurately describe the mechanism of injury and the resulting cluster of symptoms. A review of the literature has shown that there is neither any unanimity in the definition of the term "Whiplash injury", nor in the collective signs and symptoms said to be related to this particular injury (Frankel, 1959; Ommaya, Fass and Yarnell, 1968; Paige, 1978; Bogduk, 1986b; Spitzer, et al., 1995). It is also evident that despite the knowledge that hundreds of papers concerning whiplash injury have been published, the clinical management of this syndrome still remains a matter of controversy (Huelke and Nusholtz, 1986, Bogduk, 1995; Riley, Long, Riley, 1995; Spitzer, et al., 1995).

2.2 Terminology

MacNab (1964) defined "whiplash" as an extension strain of the cervical spine produced by sudden acceleration as in a rear-end collision and suggested an alternative term, "acceleration-extension injury". However, States et al. (1970) considered that MacNab's definition of whiplash injury should be extended to include lateral flexion injuries as well. It has been also suggested that the term "cervical sprain" is an appropriate one to describe the injury and the resulting symptoms (Goff, Alden, Aldes, 1964).

Frankel (1971) preferred the term "deceleration injury", since he considered that the act of deceleration was the prime cause of the tissue damage. Hohl (1983) believed that since this syndrome is essentially due to damage to the cervical muscles, ligaments, intervertebral discs, blood vessels and nerves, it would be appropriate to use the term "soft tissue injury of the neck". Another aspect of criticism is that the term "whiplash" is indiscriminately used to refer to the neck injuries arising from all types of collisions and perhaps should be exclusively used only to refer to those injuries arising from rear-end collision and alternative terms, "neck sprain" or "the soft tissue injury" were recommended to refer to injuries arising from other types of collisions (Porter, 1989). Thus, it is clearly evident that several authorities have considered it essential that the terminology of the condition should reflect the pathomechanics rather than the pathology itself. However, it is apparent that it may not be always possible to establish a correlation between the pattern of injury and the impact in a motor vehicle accident (Burke, 1971; Gosch, Gooding and Schneider 1972; Tolonen, Santavirta, Kiviluoto and Lindqvist, 1986). For this reason, Jonsson (1992), proposed that any descriptive term which might imply a specific mechanism of injury should not be used and recommended that the term "whiplash-type" injury might be an appropriate one to describe the injuries of the cervical region sustained as a result of MVA.

In recent years the term "whiplash" which was originally used to graphically describe the manner in which the head was suddenly moved, has come to be used commonly as a diagnosis in itself and this practice has received wide criticism (Frankel, 1959; Bogduk, 1986b; Keith, 1986; Porter, 1989; States et al., 1970; Awerbuch, 1992). Fontanetta (1970) highlighted the difficulty faced by the clinicians when "whiplash" is used as a diagnostic terminology and stated that, "To the orthopaedic surgeon, 'whiplash'

connotes a lack of findings on physical and x-ray examination, an often profuse symptomatology, and prolonged disability" (page 2977). It is evident that the definition of the syndrome commonly referred to as "whiplash", remains as a matter of contention. Frankel (1959) vehemently argued that the "whiplash syndrome" consisted of a number of injuries from the inconsequential to serious injuries of the neck region and recommended that it should not be regarded as a simple muscle strain. However, thirty years later Pearce (1989) stated that, "Traumatic disc protrusions and damage to cord and nerve roots are usually pathologically and clinically distinctive, with segmental root or long tract signs. They should be excluded by definition, since they only serve to broaden the spectrum and muddy already turbid waters" (page.1329). It is obvious that the argument regarding the definition of the "whiplash" syndrome has not yet been resolved in the 66 years that have elapsed since Crowe first used the term.

2.3 Mechanism of injury

Similarly, the actual mechanism causing the "whiplash" injury remains a matter of contention (Bogduk, 1986b; Porter, 1989). It appears that clinicians seem to primarily rely on the accident details made available to them either by their patients or witnesses of the accident, in order to establish the mechanism of injury (Maimaris, Barnes and Allen, 1988). In this regard, several experimental studies have been conducted using both fresh human spines and laboratory animals so that the mechanism of the injury could be better understood (Roaf, 1960; MacNab, 1964; Martinez and Garcia, 1968; Gosch et al., 1972; Nusholtz, Huelke, Lux, Alem, and Montalvo, 1983). Similarly, information gathered during clinical examination as well as surgical findings in a few specialised medical centres and autopsy findings have been used to improve the understanding

of the mechanism of injury (Burke,.1971; Batalin, 1990; Chakera, 1990). In recent years several biomechanical studies using anthropometric dummies, human cadavers and mathematical models, have also been conducted in order to enhance the knowledge related to the mechanism of injury (Huelke and Nusholtz, 1986).

2.3.1 Collisions between motor vehicle accidents

Frankel (1959) and Jackson (1970) acknowledged that the neck injury might arise from the rear-end as well as lateral impact MVAs, whereas MacNab (1964, 1971), maintained that the rear-end type of collision is exclusively responsible for "whiplash" injuries. However, a retrospective study of "whiplash" injury has shown that only 46% of the subjects surveyed were involved in rear-end collisions, contradicting the findings of MacNab (Maimaris et al., 1988). It has also been claimed that most of the rear-end collisions that produce "whiplash" injury, occurred at low velocities at an average speed of 15 mph (La Rocca, 1978). In contrast, a more recent study showed that low velocity rear-end collision produced compressive and tensile forces sequentially directed along the axis of the cervical spine rather than resulting in hyperextension/hyperflexion of the cervical spine (McConnell, Howard, Guzman, Bomar, Raddin, Benedict, Smith, and Hatsell, 1993). Although, the rear-end type of collision is the single largest mechanism of injury, there are several other modes of impact such as head-on, frontal collision, roll over, concertina type (as happens in a pile up) which are also likely to result in "whiplash-type" of injury (Janes and Hooshmand, 1965; States et al., 1970; Frankel, 1971; du Toit, 1974; Hohl, 1983; Deans, McGalliard and Rutherford, 1986; Trinca, 1986, Deans, McGilliard, Kerr and Rutherford, 1987; Hodgson and Grundy, 1989). It has been shown that irrespective of the direction of the impact in a MVA, injury to the neck is likely to occur (Deans et al., 1987). Thus, while supporting the

findings of Deans et al. (1987), a report by Awerbuch (1992) drew attention to the persistent disability which may occur independent of the direction of impact. It is evident that mere knowledge of the direction of the impact alone may not be sufficient in establishing an estimate of the resultant forces which are responsible for the ultimate morbidity in the cervical region (Jonsson, 1992). There are several reports based on experimental, clinical, surgical and autopsy findings which would support the view expressed by Jonsson (1992) (Roaf,1960; Forsyth, 1964; Cheshire, 1969; Burke, 1971; Batalin, 1990).

In an experimental study, Roaf (1960) subjected fresh human spines to forces of different magnitude and directions such as flexion, extension, rotation, lateral flexion, compression and horizontal shear. He observed that it was not possible to rupture "normal" spinal ligaments by hyper-extension or hyper-flexion. The study showed that "pure" hyper-extension tend to cause a fracture of the neural arch, whereas a combination of rotation and extension often ruptured the anterior longitudinal ligament, while a combination of rotation and compression appeared to produce a variety of spinal injuries. Roaf (1960) concluded that, "The so-called hyper-extension injury is really a rotation injury" (p.823). Forsyth (1964) reported on a group of patients who have sustained hyper-extension injury of the cervical spine and yet presented with forward displacement of the vertebrae, giving a false impression that they had been subjected to a hyper-flexion type of injury. Two patients with similar pattern of injury, were surgically explored and the posterior longitudinal ligament was found to be intact (Burke, 1971). The complexity of interpretation of the mechanism of injury from plain radiographs has been frequently encountered at the Sir George Bedbrook Spinal Unit in Western Australia (Batalin, 1990). A review of the spinal injuries at this Unit has shown that contrary to the

generally held notion, the flexion- rotation type of violence (i.e unilateral facetal fracture-dislocation of the cervical spine), is not unique to the head-on type of MVA collisions. There have been frequent instances in which the rear-end collision has caused unilateral anterior dislocation of the inferior articular process and an associated fracture of the tip of the corresponding superior articular process of a motion segment.

States et al. (1970) believed that the severity of the injury is not only related to the direction of the impact but also to other factors such as the speed and the mass of the vehicles involved. A survey of road traffic accidents has clearly shown that the mass of the vehicles involved and the wearing of seat belts have an influence in the pattern of injury and showed that the incidence of the head injuries is significantly higher when the colliding vehicle is heavier than the vehicle of the injured person (Tolonen, Kiviluoto, Santavirta and Slatis, 1984). Similarly, the number of cervical spine injuries was found to be significantly higher where the vehicles had approximately the same weight (Tolonen et al., 1984).

Although the restraints such as the seat belt and the head rest play a major role in protecting the car occupants from fatal injuries, the wearing of a seat belt has significantly changed the pattern and the severity of the non-fatal injuries (Allen, Barnes and Bodiwala, 1985; Larder, Twiss and Mackay, 1985; Rutherford, 1985; Agran, Dunkel and Winn, 1987; Agran, Winn and Dunkel, 1989). States et al. (1970) have observed that the concussion of the brain resulting in loss of consciousness is prevalent among those occupants restrained by seat belts. A number of studies have shown that since the introduction of compulsory seat belt legislation, in some areas there has been a marked increase in the incidence of neck injuries among those car occupants so restrained (Allen et al., 1985; Rutherford, 1985;

Deans et al., 1987; Lestina, Williams, Lund, Zador and Kuhlmann, 1991). A retrospective survey of the autopsy records and hospital records of road accident victims, reported that deceleration injuries are significantly higher among those victims who were wearing seat belts (Tolonen et al., 1986). Thus, an autopsy study conducted by Skold and Voigt (1977) has described the different injury patterns as seen in victims wearing lap belt, shoulder belts and shoulder lap belts. It is evident from this study that the restraints themselves are responsible for some of the severe injuries to the throat, TMJ and even fractures of the upper cervical spine. Skold and Voigt (1977) found that, inadequate tensioning of the seat belt, either by wearing them too tightly or too slack, caused some of the severe neck injuries. Morris (1989) has also shown that lack of head rest contributed to a higher incidence of whiplash injury and that the resulting symptoms can persist for a prolonged period, while an inadequately positioned head rest may not save the car occupants from injury (Bogduk, 1986b; States et al., 1970). A head rest positioned too low could allow the head to roll over the restraint producing hyper-extension of the cervical spine (States et al., 1970).

2.3.2 Experimental studies

In order to enhance the understanding of the pathomechanics and pathoanatomy of "whiplash" injuries, several studies have been conducted using laboratory animals, anthropometric dummies, mathematical models, human volunteers, fresh and embalmed cadavers (Huelke and Nusholtz, 1986).

2.3.3 Animal models

MacNab (1964) studied the effect of sudden acceleration on the cervical spine by using anaesthetised monkeys, which were strapped in a supine position to a horizontal platform and dropped down a vertical track; thus,

simulating hyper-extension injury to the neck as in the rear-end collision. The pattern of the injuries induced by varying the magnitude of the forces was observed by altering the height from which the animals were dropped. MacNab (1964) found that the severity of the lesions was directly proportional to the magnitude of the force to which the animals were subjected. He reported that the injury to the muscles varied in severity, ranging from minor tears of the sternocleidomastoid muscle to partial avulsion of the longus colli muscle. He also noted that rupture of the anterior longitudinal ligament, separation of the intervertebral disc from its bony attachment and injury to the prevertebral muscles were consistently present. The retro-pharyngeal haematoma and injury to the cervical sympathetic nerves were found to be often associated with a torn longus colli muscle. Although this study has its limitations, McNab's (1964) findings are significant and most of his observations are still considered to be valid. MacNab (1964) believed that his experimental findings were clinically relevant, since he noted that eight patients who were operated by him within two years post-injury, each had ruptured anterior longitudinal ligaments and avulsed intervertebral discs, as seen in his previous experiment using monkeys. Thus, he was able to confirm that soft tissue lesions as seen in the experimental animals, can occur in humans due to rear-end collisions.

Similar studies have been conducted to study the effect of sudden accelerations on rabbits and Cebus monkeys, with the head and neck unrestrained (Martinez and Garcia, 1968). Although most of the observations are similar to those reported by MacNab (1964), frequent occurrence of retro-ocular haematoma, damage to the middle ear and a substantial number of brain haemorrhages are among some of the significant findings.

In an another study, fifty anaesthetised Rhesus monkeys were secured to a fibreglass chair without head restraint, in a sitting position and subjected to sudden acceleration forces (Ommaya et al., 1968). Unlike the study conducted by MacNab (1964), the experimental device was mounted horizontally in order to better replicate the conditions of rear-end collisions. The aim of this important study was to report the macroscopic and microscopic lesions in the brain and cervical cord of those monkeys subjected to such a "whiplash" injury. The authors monitored the animals for a period varying between one hour to one week post injury and then sacrificed them to study the lesions. They reported that experimental concussion and surface haemorrhage in parts of the brain were consistently present in the monkeys as a result of "whiplash" injury, while the brain stem and cervical cord showed surface haemorrhages in eight animals, while two of them were found to have haemorrhages in the upper cervical cord. The data obtained from this study enabled the authors to suggest that the levels of angular acceleration required to induce the cerebral concussion and brain injury in a human accident victim, may be in the range of 6000 to 7000 radians / sec^2. It is evident from the contribution made by Ommaya et al. (1968), that there is an adequate pathophysiological explanation for some of the bizarre symptoms presented by the patients affected by "whiplash" injury and therefore this study is a significant one.

Gosch et al. (1972) conducted a similar a study by using twenty eight Macaca mulatta monkeys, administered with a short acting anaesthetic agent with a view to observing the effects of forces of varying magnitude applied from different directions. The trauma was induced in three groups of animals, with the head and neck in flexion, extension and with the

cervical spine supported, in order to subject the spine to axial loading. The authors observed that:

1. the state of the muscle tone at the time of the injury affected the severity of the injury (i.e less severe injury when the muscles are in a state of relaxation);
2. in addition to flexion or extension, rotation of the cervical spine was an essential component, in addition to flexion or extension, in order to produce cervical dislocation;
3. the haemorrhagic necrosis was exclusively confined to the central portion of the cord substance, without any evidence of contusion on the external surface of the cord; and
4. disruption of the nervous tissue often occurred at the site of the fracture, extending up to one or two segments in the cephalo-caudal directions.

Thus, Gosch et al. (1972) have provided useful information related to the neural tissue involvement and in particular and have produced a possible pathophysiological explanation for the symptoms resembling "acute central cord injury syndrome" among "whiplash" patients.

2.3.4 Anthropometric studies

Severy, Matheson and Bechtol (1955) conducted experimental studies by using anthropometric dummies, in order to assess the characteristics of the forces encountered by the motor vehicle of the occupant and the neck region, during a simulated rear-end collisions at varying speeds. Detailed information such as the duration of the impact and the resultant acceleration and displacement-time for the head, neck and shoulder and the vehicles are available from this study. At an impact speed of 15 mph, the head was reported to accelerate with a force of 10 g. However, the speed of acceleration between the vehicle and the occupant's head and shoulder

was shown to be varied. The acceleration-time of the head and shoulders was found to be slower than the vehicle itself and this effect was considered to be due to the deformation of the body and the car seat. It has been shown that a translatory type of movement occurred in the neck, during most of the early motion in the neck in relation to the trunk, resulting in a shearing type of displacement of the cervical spine, followed by the rotation of the head in a backward direction. Therefore, it is possible that the structures of the cervical spine may be subjected to two different types of injury as a result of the rear-end collisions.

A recent sophisticated study using a physical model of the head neck and the upper-torso simulating the living human and including suitably pre-strained tissue replicas and transducers to measure various forces developed in the muscles, intervertebral disc pressure and intercranial pressure (Deng and Goldsmith, 1987a, Deng and Goldsmith, 1987b), has shown the following:

a. intradiscal pressure increases during flexion and decreases during extension;
b. the intervertebral disc pressure appeared to be higher in the cervical region when compared to the thoracic region;
c. the disc pressure between the C4-C5 vertebrae is the highest during flexion-extension type of whiplash sequence;
d. a similar peak pressure is present between the C3- C4 during simulated lateral impact;
e. the longus colli muscle is likely to be the most at risk in extension whiplash; whereas, the longus capitis muscle is vulnerable during a lateral impact.;
f. there is a reaction time delay in muscle contraction, in response to the sudden movement of the head and neck;

g. the time required for the head to reach the peak excursion is shorter than the time required by the muscle to develop a reflex contraction; and

h. during the frontal and rear-end collision simulations, the temporal lobe of the brain may be affected due to an increase in the intracranial pressure and similarly, the frontal lobes may be affected as a result of a side impact.

2.3.5 Mathematical models

Mathematical models used in various studies are essentially based on parametrized neck models in which the neck is modelled as a collection of masses, springs and dampers (Huelke and Nusholtz, 1986). However, such models can only predict motion of the neck together with forces and moments and unlike the cadaveric and anthropometric studies, the parametrized neck models cannot (without appropriate experimental data) provide information about the forces required to produce various injuries (Huelke and Nusholtz, 1986). Martinez and Garcia (1968) considered that most of the mathematical models have treated the head and the neck as a single unit pivoting around a fixed point at the base of the cervical spine, failing to take into account of the anatomical variations and kinematic characteristics of the upper and lower segments of the cervical spine and the cervico-thoracic junctional region. An improved mathematical model designed by Martinez and Garcia (1968) has shown that the rear-end collision is likely to produce high rotational acceleration of the occupant's head and translation accelerations larger than that experienced by the vehicle itself.

Schneider, Zernicke and Clark (1989) claimed that they have succeeded for the first time in designing a mathematical model to study the jaw-head-neck

dynamics during the rear-end collisions. A preliminary report of their initial computer simulations has also shown that a sudden hyper-extension of the neck caused opening of the jaw and that it is possible to predict the relative angle between the jaws (jaw-opening angle), temporomandibular joint (TMJ) torque, displacement and the linear acceleration of the jaw's centre of mass. It appeared that the peak jaw-opening angle and the peak values for the relative angular velocities of the jaw with respect to the head were larger with impact magnitudes of 6.71 ms^{1} (15 mph) when compared to 13.41 ms^{1} (30 mph). The study also showed that the jaw closed for a very short duration (0.75 s) during the initial period of the impact followed by opening of the jaw for the remaining impact duration. The linear acceleration of the jaw was higher with impact magnitude of 13.41 ms^{1} (30 mph) when compared to 6.71 ms^{1} (15 mph). Schneider et al. (1989) considered that when the jaw opens too fast or too wide, the speed and the amount of movement might adversely affect the lubrication of the TMJ by synovial fluid and cause high friction which could damage the disc, capsule or the ligaments. Similarly, compressive forces could also produce tearing or bruising of the disc or the articular surfaces.

2.3.6 Cadaveric studies

In a further attempt to simulate various situations that might arise as a result of rear-end, frontal, side impact and roll-over type of collisions, several experimental studies have been conducted using embalmed and nonembalmed human cadavers (Lange, 1971; Cromack and Zipperman, 1975; Patrick and Levine, 1975; Culver, Bender and Melvin, 1978; Nusholtz et al., 1983). Lange (1971) found that the torque exerted at the cervical spine and the resultant rotation between the head and the torso significantly influenced the nature and the severity of the injury to the cervical spine. He showed that the magnitude of the rotation and torque depend upon the

direction and degree of impact on the restraints such as the back rest, head rest and other internal parts of the motor vehicle, as well as the force generated by the seat belt.

A number of other cadaveric studies have been conducted to establish the morbidity that might be attributed to the wearing of the seat belt (Cromack and Zipperman, 1975; Patrick and Levine, 1975). It has been clearly shown that two-thirds of the specimens involved exhibited fracture of the cervical vertebrae. It appears that even at relatively low speeds (e.g 8 mph), fracture of the cervical vertebrae, subluxation, tearing of the anterior and posterior longitudinal ligaments, ligamentum flavum and disc rupture can occur and that such lesions were noticed predominantly at the levels of C5/C6, and C6/C7 (Clemens and Burrow, 1972; Jones et al., 1978). Despite the deployment of an air-bag, the driver of a car travelling at a speed of 30 mph, involved in a head-on collision, was reported to have sustained fracture of both pedicles, left lateral mass and also a fracture through the body of the C2 vertebra with associated anterior subluxation of the C2 over C3 vertebra (Traynelis and Gold, 1993). Clemens and Burrow (1972) reported that 90% of their cadavers had disc lesions and that the anterior longitudinal ligament was torn in 80% of the specimens, whereas the incidence of torn posterior longitudinal ligament and ligamentum flavum were 10% and that of fractures were in the order of 30%. Culver et al. (1978) and Nusholtz et al. (1986) conducted studies in which the crown of the head of unembalmed cadavers were impacted by a piston, simulating the roll-over effect and found that the fracture of the spinous processes was most common injury.

2.4 Spinal stability and related structures

A state of mechanical stability of the spinal segments is essential in order to effectively achieve the functional requirements of the spinal column as such

and this is achieved by a complex mechanism of integrated forces developed by the soft tissues and osseous elements in and around the spinal column (White, Southwick and Panjabi, 1976; Johnson and Wolf, 1983). A number of investigations have been conducted in order to identify the important anatomical structures which are essential for the mechanical integrity of the cervical spine. This knowledge is necessary for establishing differential diagnosis as well as to improve the efficacy of the treatment methods (Werne,1957; Roaf, 1960; Holdsworth, 1963; Bailey, 1964; Halliday, Sullivan, Hollinshead and Bahn, 1964; Munro, 1965; Bedbrook, 1969; Johnson, Crelin, White, Panjabi and Southwick, 1975; King, Prasad and Ewing, 1975; Panjabi, White and Johnson, 1975; White, Johnson, Panjabi and Southwick, 1975; Louis, 1985; Nolan and Sherk, 1988; Pal and Sherk, 1988; Crisco, Oda, Panjabi, Bueff, Dvorak and Grob, 1991; Panjabi, Dvorak, Crisco, Oda, Hilibrand and Grob, 1991a; Panjabi, Oda, Crisco, Oxland, Katz and Nolte, 1991b; Panjabi, Oxland, Takata, Goel, Duranceau and Krag, 1993).

2.4.1 Cranio-vertebral joints

There is a distinctive difference between the structures providing stability to the cranio-vertebral joints and rest of the motion segments in the cervical spine. Werne (1957) conducted a detailed and most significant anatomical study, using twenty four specimens, in order to determine the functional significance of the structures responsible for the integrity of the cranio-vertebral joints. He showed that:

a. the tectorial membrane is responsible for providing stability to the atlanto-occipital (C0 - C1) and atlanto-axial joints (C1 - C2) during extension and limits flexion in the atlanto-axial joint;

b. flexion movement in the atlanto-occipital joints is arrested by the skeletal contact between the anterior margin of the foramen

magnum and the odontoid process through an intervening bursa; and

c. the alar ligaments are responsible for limiting lateral flexion of the atlanto-occipital joint.

Werne (1957) demonstrated that when the head is tilted to one side (e.g to the right side), the alar ligament on the contra-lateral side (i.e left alar ligament) begins to tighten and the alar ligament on the ipsi-lateral side (i.e right alar ligament) becomes only taut at the limit of the lateral flexion. However, stability during a combined lateral flexion of the atlanto-occipital joints and rotation of the atlanto-axial joints, is achieved by the integrated restraining action of the alar ligaments and the tectorial membrane (Werne, 1957). Panjabi et al. (1991a) studied the role of the alar ligaments by sequential sectioning of this ligament and observed that the alar ligament together with the tectorial membrane, provided stability during flexion of the C0 - C1 - C2 joints. Similarly, he was able to specifically identify that the contralateral alar ligament in conjunction with the tectorial membrane is responsible for controlling the lateral flexion in the atlanto-occipital joints. The transverse ligament has been shown to limit the horizontal translation of the atlanto-axial joint along the sagittal (Werne, 1957; Fielding, Cochran, Van, Lawsing and Hohl, 1974).

It is also evident that the biomechanical role played by the capsular ligament of the atlanto-axial joints has not yet been clearly established (Crisco et al.,1991; Panjabi et al., 1991a). Crisco et al. (1991) carried out a study in which they transected the capsular ligament of the atlanto-axial joints and applied physiological torques in all three planes. Their findings has shown that the capsular ligament together with the alar ligaments, play a significant role in restraining the axial rotation of the atlanto-axial joints. However, in a similar study Panjabi et al. (1991b) reported that they were

unable to ascertain the stabilising role of the capsular ligament at the suboccipital region.

2.4.2 Lower cervical spine (C3-C7)

Even though the importance of the muscles in relation to the stability of the spine has been generally recognised, it appears that their role as stabilisers has not been adequately investigated (White et al., 1976; Nolan and Sherk, 1988; Panjabi, Abumi, Duranceau and Oxland, 1989). The deep intersegmental spinal muscles have been shown to play a significant role in providing mechanical stability to the spinal column (Nolan and Sherk, 1988; Panjabi et al., 1989). Bailey (1964) considered that a combination of dynamic forces exerted by the muscles in the cervical region and the static forces manifested by the intervertebral discs are the most important factors for the stability of the cervical spine. However, observations made by Munro (1961, 1965) and Bedbrook (1969) indicated that the stability of the cervical spine can be mainly attributed to the intervertebral discs, the anterior and posterior longitudinal ligaments. In an anatomical dissection study of fifteen fresh cervical spines, Johnson et al., (1975) observed that the anterior longitudinal ligament is a thin, translucent structure while the posterior longitudinal ligament, capsular ligament and the anulus fibrosus are the largest and most stable of all the soft tissue structures in the lower cervical spine, capable of stabilising the cervical spine. Even though the findings of Bedbrook (1969) indicated that the ligamentum flavum contributed towards the stability of the cervical spine, a recent study has shown that the tensile strength of this ligament is considerably reduced at the mid cervical region; thus, it may not have a significant role as a stabiliser (Myklebust, Pintar, Yoganandan, Cusick, Maiman, Myers and Sances, 1988).

In order to determine the role played by the ligaments and the zygapophyseal joints in providing stability to the cervical spine, White et al. (1975) conducted a detailed quantitative study using 17 motion segments from 8 human cervical spines. An axial load equivalent to 25% of the body weight was applied and the horizontal and angular displacement of the upper vertebra before and after transecting the ligaments was measured. The sequential transection of the ligaments was carried out in an anterior to posterior direction to measure the displacement during simulated flexion (n=5) and extension (n=4) and similarly in a posterior to anterior direction to document the displacement during flexion (n=4) and extension (n=4) moment. Failure of the anterior and posterior elements of the motion segment following the transection of the ligaments or the zygapophyseal joint has been reported for the first time. This significant study has shown the following:

1. the anterior ligament provided significant amount of stability during extension;
2. the posterior ligaments played a more effective role in limiting flexion, thereby contributing to the stability of the motion segment during flexion;
3. to remain stable under physiological load, the motion segment requires either all of its anterior elements and one additional structure belonging to the posterior elements or all of its posterior elements and an additional structure from the anterior elements; and
4. the zygapophyseal joints exert a significant influence in controlling the horizontal displacement during flexion of the motion segment and to a lesser extent in the angular displacement.

The significance of the interspinous and supraspinous ligaments and ligamentum nuchae remains unclear (Holdsworth, 1963; Halliday et al., 1964; Johnson et al., 1975). Holdsworth (1963) considered that the interspinous and supraspinous ligaments make an important contribution in providing stability to the cervical spine. However, other detailed anatomical studies have shown that these two structures are inconsistent in their presence and that the interspinous ligament has the least tensile strength of all the spinal ligaments in the cervical region (Halliday et al.,1964; Johnson et al., 1975; Myklebust et al.,1988). It seems unlikely that either the interspinous or the supraspinous ligaments have any significant biomechanical role.

The role of the osseous elements in providing stability to the cervical spine has also been extensively investigated (Fielding et al., 1974; White et al., 1975; Pal and Routal, 1986 ; Pal and Sherk, 1988). The odontoid process is a relatively strong structure capable of withstanding shear stresses up to 180 kiloponds and is essential for the stability of the atlanto-axial joint as well as the atlanto-occipital joints, due to the attachment of the alar ligaments to it (Fielding et al., 1974). The zygapophyseal joint structures, while exerting a significant influence in controlling the horizontal translation in a motion segment during flexion, play only a lesser role in controlling angular displacement (White et al., 1975). The shape of the vertebrae themselves is also regarded one of the important factors associated with the stability of the spine (Med, 1973). Louis (1985) studied the architecture of the vertebral column and demonstrated that the axial stability is maintained by two bony columns at the level of C1 - C2 vertebrae. Similarly, below the level of C2 vertebra, he showed that three bony columns viz an anterior column consisting of the bodies of the vertebrae together with the intervertebral disc and two posterior columns formed by the articular

structures on either side, are responsible for the axial stability of the spinal column. The triangular disposition of the elements in the anterior and posterior columns lead him to propose the "articular orthogonal triangulation" concept. This arrangement has been shown to be mechanically efficient in preserving the stability of the spine during axial loading (Louis, 1985; Pal and Routal, 1986; Pal and Sherk,1988). Thus, the body and bony articular structures together with the laminae of the vertebra are regarded as essential structures for the stability of the spinal column.

2.5 Kinematics

It has been stressed that a sound knowledge of kinematics is essential for a better understanding of the mechanism of "whiplash" injury as well as in the establishment of differential diagnosis and the formulation of appropriate treatment (Penning, 1978). The important factors which determine and influence the kinematics of a joint are the geometry of the articulating surfaces and the position and strength of the soft tissue structures (such as the ligaments and muscles) acting on the articular structures (Werne, 1957; Lysell, 1969; Panjabi et al., 1975; Jofe, White and Panjabi, 1983). Thus, a number of studies have investigated the geometry of the articular surfaces, the functional significance of the ligaments and the intervertebral disc (Werne, 1957; Ball and Meijers , 1964; Fielding , 1964; Hohl , 1964 ; Lysell, 1969; Med, 1973 ; White et al., 1975; Penning, 1978; Panjabi, Summers, Pelker, Vedeman, Friedlander and Southwick, 1986; Monu, Bohrer and Howard, 1987; Penning and Wilmink, 1987; Penning, 1988; Alund and Larsson , 1990; Milne, 1991). In recent years, the existence of discreet abnormal pattern of movements in the cervical spine has been recognised and has been shown to be clinically significant (Dimnet, Pasquet, Krag and Panjabi, 1982; Mayer, Herman, Pfaffenrath, Pollman and Auberger, 1985;

Amevo, April and Bogduk, 1992; van Mameren, 1988; van Mameren, Sanches, Beursgens and Drukker, 1992).

2.5.1 Normal Patterns of movements

Cranio-vertebral joints (C0-C1-C2)

The anatomical and radiographic investigation carried out by Werne (1957, 1958) is considered to be the authoritative report on the kinematics of the cranio-vertebral joints (Jofe et al., 1983). Werne (1957, 1958) has shown that the cranio-vertebral joints function as a single unit in order to facilitate three dimensional movement in the upper cervical spine. Flexion-extension and lateral flexion are the primary movements available in the atlanto-occipital joints under normal physiological conditions (Jofe et al., 1983) Similarly, axial rotation is the predominant movement in the atlanto-axial joints (Jofe et al., 1983). A number of studies have also indicated that flexion-extension and horizontal translation are part of a normal physiological movement pattern in the atlanto-axial joints (Fielding, 1964; Hohl, 1964; Swischuk, Hayden and Sawar, 1979; Bohrer, Kelin and Martin, 1985; Dvorak, Hayek and Zehnder, 1987; Monu et al., 1987). Werne (1957, 1958) showed that axial rotation cannot occur in the atlanto-occipital joints and has attributed this to the geometry of the articular surfaces of the atlanto-occipital joints. However whether or not the lateral flexion occurs in the atlanto-axial joints appears to be a matter of controversy. Werne (1957, 1958) considered that lateral flexion does not exist in the atlanto-axial joints, although a combined anatomical and cine-radiographic study of the atlanto-axial joints conducted by Hohl and Baker (1964), has shown that lateral bending and lateral translation do occur concomitantly under normal conditions. Werne (1957, 1958) has shown that a combined movement pattern of lateral flexion-axial rotation occurs in the cranio-vertebral joints (C0-C1-C2). He demonstrated that during the lateral flexion of the atlanto-

occipital joints, the alar ligaments are tightened up and that this action produced a coupled rotation in the atlanto-axial joints, in a direction opposite to the bending of the head. In a radiological study, another significant variation in the pattern of movement between the cranio-vertebral segment (C0-C2) and lower cervical spine (C3-C7) has been observed (Penning, 1978). He showed that the upper and lower cervical spine can move independent of each other, in opposite directions, since when the lower cervical spine moves into flexion, while the upper cervical spine is in extension and vice versa.

Lower Cervical Spine (C3-C7)

Flexion-extension, lateral flexion and axial rotation are the primary movements found in the lower cervical spine (Jofe et al., 1983). In addition, horizontal translation also accompanies the sagittal plane movements (Panjabi et al., 1975; Panjabi et al., 1991a). Fielding (1964) in his highly regarded study of the lower cervical spine using cineradiography, has shown that under normal conditions there is a gradual "intersegmental flow" of movement in the cervical spine. It is also evident that with the exception of the cranio-vertebral joints, a particular motion segment in the lower cervical spine does not move without a synchronous movement of the adjacent motion segments, so that the lower cervical spine functions as a single unit (Lysell, 1969; Penning, 1978). Fielding (1964) observed that the lateral flexion in the lower cervical spine (C3-C7) cannot occur without an accompanying axial rotation. Similarly, the rotation of the lower cervical spine (C3-C7) cannot occur without concomitant lateral flexion (Fielding, 1964). While confirming these observations, Lysell (1969) and Alund and Larsson (1990) have shown that the lower cervical spine exhibits a progressive variation in the combined movement, in a cranio-caudal direction. This combined movement pattern described by Fielding (1964)

could be explained by the observations made by Lysell (1969). The patterns of motion in the lower cervical spine are of two distinctively different types:

1. in the sagittal plane, the motion (eg. flexion-extension) takes place in such a way that the vertebrae move parallel to the plane thus, flexion-extension movement is confined to a single plane; and
2. during either lateral flexion or axial rotation, the motion of the vertebrae is not exclusively confined to a single plane (e.g. during lateral flexion, a particular vertebra will move primarily in the frontal plane and as the movement progresses, through the horizontal plane as well).

The shape and orientation of the articular surfaces and the shape of the intervertebral discs are primarily responsible for the above kinematic pattern (Lysell, 1969; Med, 1973; Panjabi et al., 1975). However, Panjabi et al. (1975) have shown that the shape of the articular surfaces in the lower cervical spine is the primary factor in restraining the "coupling effect" (combination two dissimilar movements viz sagittal rotation and a horizontal translatory movement) during flexion of the cervical spine. Similarly, the uncinate process is also considered to be responsible for the combined rotation and lateral flexion in the lower cervical spine (Penning and Wilmink, 1987). The extent of intersegmental movement in the lower cervical spine (C3-C7), during flexion-extension, lateral flexion and axial rotation vary according to the level of the spine. Lysell (1969) observed that while the largest amount of intersegmental movement (ISM) occurred in the middle part of the lower cervical spine during lateral flexion and axial rotation, ISM is evenly distributed throughout the lower cervical during flexion-extension. He also observed that during motion in the frontal and horizontal planes, the range of intersegmental motion is largest in the

middle part of the lower cervical spine whereas, during flexion-extension it is evenly distributed throughout the lower cervical spine. However, a radiological study of the normal range of motion between C2 - T1 vertebrae, among twenty normal individuals, conducted by Bhalla and Simmons (1969), demonstrated that the pattern of intersegmental movement at the levels of C6 - C7 and C7 - T1 differed considerably from that in the rest of the motion segments. The authors noted that during extension, there is a greater intersegmental movement at the levels of C6 - C7, C7 - T1 than flexion whereas, the ISM in rest of the motion segments is greater during flexion. Bhalla and Simmons (1969) also observed that the maximum range of total movement occurred at C4 - C5 levels, although recent radiological studies demonstrated a similar pattern at the level of C5 - C6 (Penning, 1978; Penning, 1988). The least amount of intersegmental motion has been shown to occur at the level of C2-C3 motion segment (Penning, 1978; Penning, 1988). Penning (1978) also demonstrated that the upper and lower parts of the cervical spine can move independent of each other, in opposite directions (e.g lower cervical spine moving into flexion while the upper cervical spine is in extension and vice versa).

Panjabi et al. (1986) observed that each translatory motion (often of significant magnitude) is associated with five coupled motions in the cervical spine. It is apparent from this study that small translatory movements can be readily initiated within the region described by Panjabi et al. (1986) as a "neutral zone", without the application of any appreciable external load. "The neutral zone is that zone within the range of motion in which the spine can be displaced with the application of a very small force or moment" (Panjabi et al., 1986; p.160). The authors observed that application of lateral and anterior / posterior shear forces produced an average of 1.5 mm displacement within the neutral zone. The point of origin

of the normal physiological movements in the X, Y, Z plane is indeterminant and considered to lie within the neutral zone. It was proposed by the authors that this neutral zone may be responsible for the inherent instability of the spinal column (Panjabi, Lydon, Vasavada, Grob, Crisco and Dvorak, 1994).

2.5.2 "Abnormal" patterns of movement

The clinical significance of the presence of an abnormal pattern of motion in the cervical spine has been realised and as a result an increasing number of anatomical and radiological studies have been conducted (Fielding, 1964; Hohl, 1964; Lysell, 1969; Dimnet et al., 1982; Mayer et al., 1985; Amevo et al., 1992;). When a particular motion segment looses its mobility either due to surgical fusion or spondylitic changes, an increased amount of intersegmental motion will occur in the adjacent motion segments (Ball and Meijers, 1964; Fielding,1964; Schoening and Hannan, 1964). However, Lysell (1969) stated that, "Contrary to most of the earlier investigations, there is no correlation between reducing range of motion and increasing degeneration. This applies to all directions of motion, to all interspaces, and also to the total motion of the specimens" (p.56). Thus a lateral translation of the atlas over the axis in excess of 4 mm is considered to pathologic (Hohl, 1964). Similarly a horizontal translation of the atlas over the axis, in the anterior direction, in excess of 5 mm, may be the result of either a disrupted transverse ligament or due to a fracture of the odontoid process (Fielding, 1964).

Recently, a number of radiological studies have investigated the clinical significance of the instantaneous axes or centre of rotation (IAR , ICR) (Dimnet et al., 1982; Mayer et al., 1985; van Mameren, 1988; Amevo, Worth, Bogduk, 1991; Amevo et al., 1992; van Mameren et al., 1992). Van

Mameren et al., (1992) defined the ICR as a "....parameter of quality of motion containing information on the rotary as well as the translatory component between two adjacent vertebrae" (p.467). It has been shown that it is possible to establish normal and abnormal patterns of movement in the cervical spine, by radiologically plotting and comparing the instantaneous axes or centre of rotation (IAR / ICR) of adjacent vertebrae. It is evident that there is a demonstrable correlation, between the abnormal range of distribution of IAR and the motion segment considered to be the source of a pain related symptom (Mayer et al., 1985; Amevo et al., 1992).

2.5.3 Normal range of motion

A considerable number of investigations have been undertaken to establish the range of normal physiological motion in the cervical spine and among them the observations made by Werne (1957) and Lysell (1969) are considered to be significant (Ball and Meijers, 1964; Fielding, 1964; Hohl, 1964 ; Penning, 1978; Panjabi et al., 1986; Dvorak et al., 1987; Penning and Wilmink; 1987; Penning, 1988; Alund and Larsson, 1990; Trott, Ruston, Pearcy and Fulton, 1991).

Werne (1957) reported that axial rotation does not exist in the atlanto-occipital joints, whereas Penning and Wilmink (1987) using computerised tomography, have demonstrated that a mean average of 1° (ranging from 2° - 5°) of axial rotation is present among healthy adults, during actively performed rotation to one side. There is a general agreement, that 55% of the axial rotation in the cervical spine occurs at the atlanto-axial level (Penning and Wilmink, 1987; Jofe et al, 1983). Although Werne (1957) reported that the mean value of axial rotation in the C1-C2 joints is 47°, Hohl (1964) demonstrated that up to 45° of axial rotation is available in this

joint. A recent investigation using C.T. scanner has shown that only 40.5.° of axial rotation is possible (Penning and Wilmink, 1987).

Werne (1957) showed that lateral flexion ranging from 4° - 13.5° with a mean average of 8° is present in the antlanto-occipital joint. He observed that individually, an average range of 13° of flexion - extension in the C0 - C1 joint and 10° in the C1 - C2 joint is present. According to Hohl (1964) approximately 15° of flexion and extension occur in both the C0 - C1 - C2 joints (Table 2.1).

Table 2.1 Range of motion in cranio-vertebral joints.

Investig-ators	Axial rotation			Lateral flexion			Flexion -extension		
	OC-C1	C1-C2	OC-C1-C2	OC-C1	C1-C2	OC-C1-C2	OC-C1	C1-C2	OC-C1-C2
Werne-(1957)	0°	47°	47°	4-13.5°	0°	-	13°	10°	23°
Hohl-(1964)	-	45°	45°	-	-	-	-	-	-
Penning& Wilmink (1987)	1°	40.5°	-	-	-	-	-	-	-
Penning-(1978)	-	-	35°	-	-	10°	30°	30°	-

Lysell (1969) found that a range of 45° of axial rotation is present in the lower cervical spine (C3-C7) with maximum axial rotation occurring between C3-C4 and gradually decreasing at the levels of C4-C5, and C5-C6 motion segments (Penning and Wilmink, 1987). The range of unilateral rotation between the skull and first thoracic vertebra is considered to be 72.2° (Penning and Wilmink, 1987). However, Alund and Larsson (1990) and Trott et al. (1991) showed that the mean average of axial rotation

between the skull and the C7 vertebra respectively are in the order of 76° and 74.8°. The range of motion can vary between right and left sides and difference ranging from 3.6° to 9° have been reported (Alund and Larsson, 1990; Trott et al., 1991).

Lysell reported that in the lower cervical spine, an average of 64° of total movement occurs in the of sagittal plane, consisting of 40° of flexion and 24° of extension. He observed that the C4-C5 and C5-C6 motion segments have the largest ranges of motion and these findings were later confirmed by Lind, Sihlbom, Nordwall and Malchau (1989). Alund and Larsson (1990) found that the mean average of flexion-extension in the whole of the cervical spine is 140°. According to Trott et al. (1991) a mean average range of 57.5° of flexion and 76.1° of extension is present over the entire cervical spine.

A detailed account of lateral flexion in the lower cervical spine has been presented by Lysell (1969). He concluded that between C2-T1, an average of 49° of lateral flexion with a concomitant axial rotation of 28° is present. He further described that the proportional relationship between the lateral flexion and combined rotation is unevenly distributed in the cervical spine. Lysell (1969) also observed that the ratio between the combined axial rotation and the lateral flexion between C2-C3 is respectively 3:2, whereas at the level of the C7-T1 is 1:7.5. According to Lysell (1969) the lateral flexion moment is evenly distributed intersegmentally in all levels, except C7-T1 where it shows a reduction. Alund and Larsson (1990) and Trott et al. (1991) respectively reported that the mean average of lateral flexion for the cervical spine as a whole is 45.1° and 47.2° (Table 2.2). However, the range of motion in the cervical spine in all directions except in flexion

generally decreased due to age related changes in the spine (Lind et al., 1989).

Table 2.2 Range of motion in the cervical spine.

Investig-ators	Flexion -extension		Lateral flexion		Axial rotation	
	OC-T1	C3-T1	OC-T1	C2-T1	OC-T1	C3-T1
Lysell-(1969)	40°	24°	-	49°	-	45°
Penning& Wilmink-(1987)	-	-	-	-	72.2°	-
Alund & Larsson (1991)	140°	-	45.1°	-	76°	-
Trott et al., (1991)	133.6°	-	47.2°	-	74.8°	-

A number of investigations have been performed to identify an instrument for its validity, reliability and simple to use in a clinical setting for documenting the range of motion of the cervical spine (Capuano-Pucci, Rheault, Aukai, Bracke, Day and Pastrick, 1991; Youdas, Carey and Garrett,1991; Rheault, Albright, Byers, Franta, Johnson, Skowronek and Dougherty,1992; Hole, Cook and Bolton, 1995). Thus, the Cervical Range of Motion Instrument (CROM) was shown to have a very high inter and intratester reliability (Capuano-Pucci, Rheault, Aukai, Bracke, Day and Pastrick, 1991; Youdas, Carey and Garrett,1991; Rheault, Albright, Byers, Franta, Johnson, Skowronek and Dougherty,1992; Hole, Cook and Bolton, 1995). A more recent report also supported the recommendations of the previous studies in this regard (Hole, Cook and Bolton, 1995). It is worth noting that in these reliability studies, Capuano-Pucci et al., (1991) used asymptomatic volunteers while Youdas et al., (1991) and Rheault et al., (1992) measured volunteers with neck symptoms who were treated as out-

patients (Table 2.3) and under both these circumstances the CROM was demonstrated to be a reliable instrument and also simple to use. A more recent report also supported the conclusions of previous studies (Hole, Cook and Bolton, 1995).

Table 2.3 **Intra class co-efficient of intra and inter tester reliability for CROM.**

Movement	Youdas et al.,(1991) ICC-Intra tester reliability	Youdas et al.,(1991) ICC-Inter tester reliability	Rheault et al., (1992) ICC-Inter tester reliability	Hole et al., (1995) ICC-Intra tester reliability
Flexion	0.95	0.86	0.76	0.96
Extension	0.90	0.86	0.98	0.96
Lt Lateral Flex	0.84	0.73	0.86	0.92
Rt Lateral Flex	0.92	0.88	0.87	0.92
Lt Rotation	0.90	0.82	0.82	0.92
Rt Rotation	0.93	0.92	0.81	0.92

2.6 Pathology

In 1964, MacNab stated that, "There is a remarkable paucity of information in the literature in regard to the underlying lesions resulting from extension-acceleration injuries of the cervical spine" (p.1798). It is interesting to note that nearly thirty years later, the controversy in nominating the structures which are affected as a result of "whiplash" injuries and the extent of morbidity has not been resolved (Bogduk, 1986b; Porter, 1989). It is evident that the "whiplash" mechanism can produce injury to the muscles, ligaments, bony elements, blood vessels and neural tissues situated in the neck region (Bogduk, 1986b). Some of these lesions are readily demonstrable and therefore generally acknowledged by clinicians. However, other lesions likely to arise from "whiplash" mechanism, such as injury to the spinal cord,

brain, vestibular apparatus and occult fracture of the vertebral structures have not yet been widely recognised by clinicians. A detailed knowledge of the structures likely to be involved in a "whiplash" injury is essential for an effective clinical management (Janes and Hooshmand,1965). A number of reports based on clinical investigations, post-operative observations, experimentally induced injuries, and post-mortem examination have enhanced the understanding of the pathology associated with "whiplash" type injury (Frankel, 1959; Roaf, 1960; Forsyth, 1964; Janes and Hooshmand, 1965; Cheshire, 1969; States et al., 1970; Burke, 1971; Penning, 1983; Batalin, 1990, Chakera, 1990; Davis, Teresi, Bradley, Ziemba and Bloze, 1991; Jonsson, Bring, Rauschning and Sahlstedt, 1991; Jonsson, Niklasson and Josefsson, 1991; Twomey and Taylor, 1991; Jonsson, Cesarini, Sahlstedt and Rauschning, 1994). It has been shown that the muscles in the pre- and post-vertebral regions, the interspinous ligament, ligamentum nuchae, anterior and posterior longitudinal ligaments and intervertebral disc are often damaged in such injuries (MacNab, 1964; MacNab, 1973; La Rocca, 1978; Gehwiler, Osborne, Becker, 1981; Scher, 1982; Rauschning, McAfee and Jonsson 1989; Davis et al., 1991; Jonsson, Bring et al., 1991; Jonsson et al., 1994). The "whiplash" mechanism has also been shown to cause fracture of the odontoid process, spinous process, pedicle, lamina, vertebral end plate, body of the vertebra, uncinate processes, and the articular surfaces (Roaf, 1960; Burke, 1971; Harris, 1978; Harris, Edeiken-Monroe and Kopaniky 1986; Clark, Igram, El-Khoury and Ehara, 1988; Batalin, 1990; Twomey and Taylor, 1991, Jonsson, Bring et al., 1991; Wong, Mack and Craigmile, 1991). In addition, avulsion of the ring apophyses of the cervical spine was also shown to occur as a result of "whiplash" (Jonsson et al., 1991). Similarly, injury to the brain, spinal cord, sympathetic nerves and peripheral as well as cranial nerves resulting from the "whiplash" mechanism have been clearly demonstrated (Torres and

Shapiro, 1961; Ommaya et al., 1968; Martinez and Gracia, 1968; Wickstrom and La Rocca, 1977). Dysfunction of the temporomandibular joint, injury to the vertebral artery and lesion of the vestibular apparatus have also been shown to occur as a result of "whiplash" type of injury (Frankel, 1971; Schneider et al.,1989).

2.6.1 Lesions of the intervertebral disc

Intervertebral disc lesions encountered as a result of "whiplash" type of injuries include bulging anulus fibrosus, protruding or free fragments of the nucleus material into the spinal canal, detachment of the anulus fibrosus together with their bony attachment and tearing of the peripheral anulus fibrosus from the vertebral body rim along a horizontal plane and adjacent to the end plates, the so called rim lesion (MacNab, 1964; Bedbrook, 1989; Davis et al., 1991; Jonsson, Bring et al., 1991; Twomey and Taylor, 1991; Jonsson et al., 1994). Even though MacNab (1964) has shown in his study that a significant number of experimental animals sustained IVD lesions in acceleration / deceleration studies, due to a lack of diagnostic technology, it was not possible until recently to demonstrate the entire spectrum of the IVD lesions which may be seen in "whiplash" patients. Therefore most of the available information regarding the IVD lesions are based on either clinical or surgical findings or following post-mortem examination reports. It is also evident that many clinicians had difficulty in identifying the component of the IVD responsible for the symptoms presented by their patients. Such a predicament can be demonstrated by the contradicting observations in which for instance, Janes and Hooshmand (1965) reported that the incidence of nucleus protrusion is not significant, while Gay and Abbott (1953) stated that 26% of their "whiplash" patients demonstrated disc herniation. However, available new information suggests that the nuclear material may not play a major role in compromising the contents of the

spinal canal in the cervical region. A recent histological study has shown that unlike the situation evident in the lumbar spine, age related changes to the upper cervical discs mean that the nucleus disappears in middle life and probably becomes incorporated into the anulus fibrosus (Twomey and Taylor, 1991). Therefore, contrary to generally held clinical views, it is more likely that bulging of the outer layers of the anulus fibrosus are responsible for the root and cord compressions in the cervical region, rather than extruded, denatured nuclear material (Twomey and Taylor, 1991).

A significant and detailed account of surgical findings reported by MacNab (1964) showed that the pattern of injury seen in the whiplash patients was remarkably similar to the lesions found in the experimental animals. His observations were based on eight patients who were operated within two years after the injury. In describing his findings graphically MacNab (1964) stated that,"....the remarkable finding was the manner in which the disc could be readily separated from the adjacent vertebral body on blunt dissection" (P 1799). A unique autopsy examination findings reported by Simeone and Goldberg (1968) of a MVA victim, who died within 24 hours post-injury due to cerebral thrombosis clearly supports the observations of MacNab (1964). Simeone and Goldberg (1968) demonstrated the presence of the IVD damage with associated separation of the thin rim of cortex from the inferior margin of the C-5 vertebra together with the rupture of the anterior longitudinal ligament.

The advent of the Magnetic Resonance Imaging technology has made it possible to evaluate the IVD lesions after "whiplash" and study the pathological changes at various stages (Figure 2.1, 2.2) (Davis et al.,1991; Jonsson, Cesarini, Nyman and Rauschning, 1992; Jonsson et al., 1994).

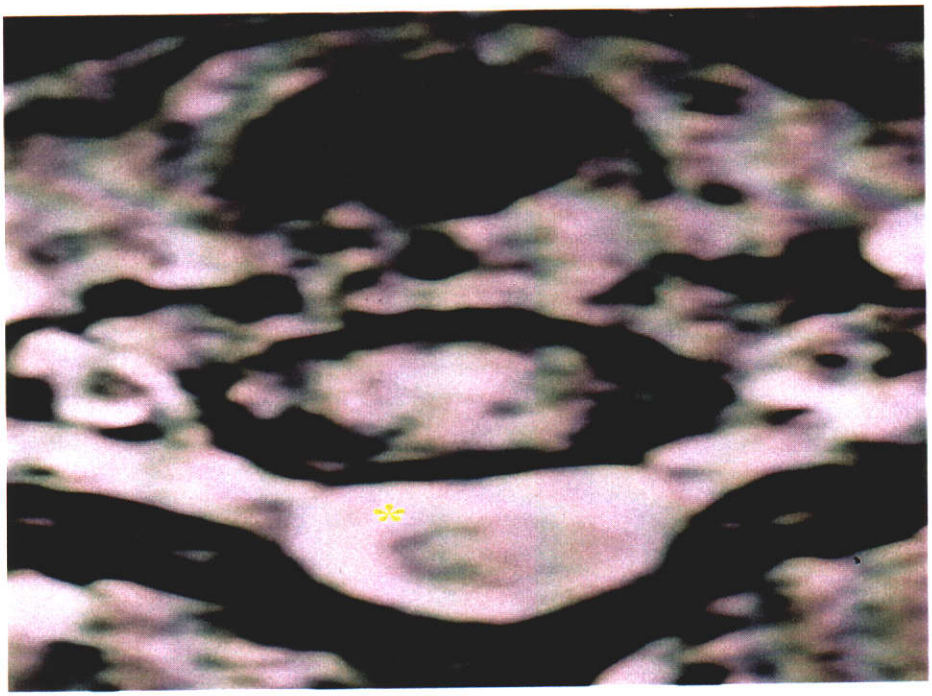

Figure 2.1 MRI showing normal arrangement of structures within the spinal canal.

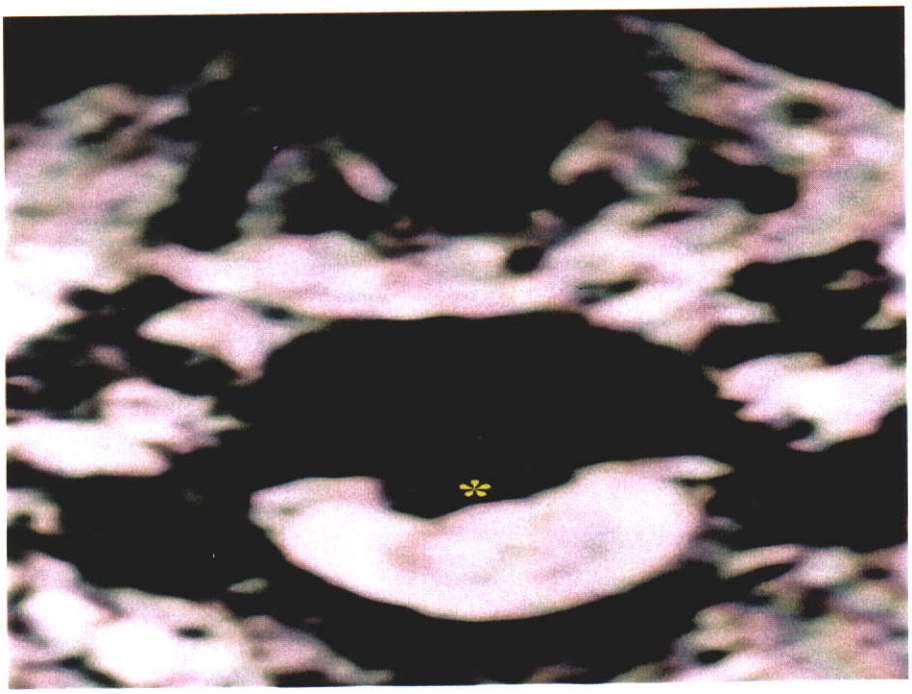

Figure 2.2 MRI of a "whiplash" patient demonstrating disc lesion causing indentation of the subarachnoid space.

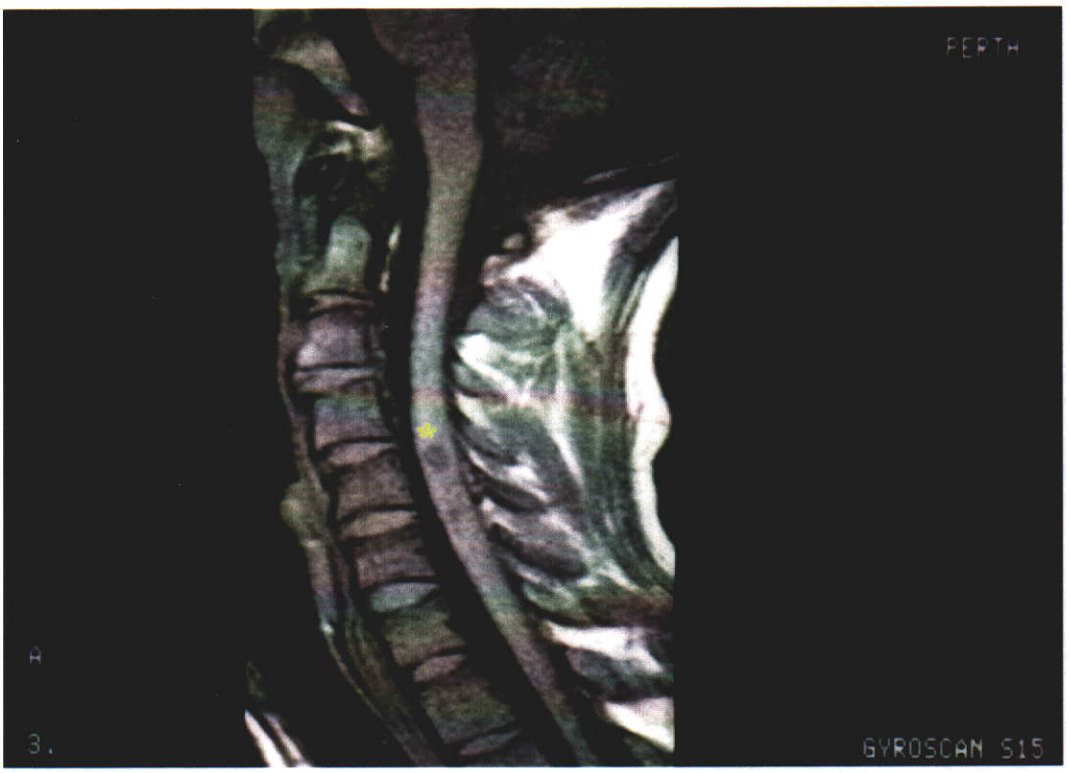

Figure 2.3 MRI of a "whiplash" patient demonstrating internal bleeding in the spinal cord.

While confirming the existence of anular bulge and anular tear, Davis et al. (1991) in their MRI study have also clearly demonstrated that horizontal avulsion of the vertebral end plate may accompany disruption of the IVD. They showed that five out of the total of nine patients in their study have had rim lesions and that four patients out of the nine had acute posterolateral disc herniations large enough to indent or displace the cord. More recent observations made by Jonsson et al. (1992, 1994) support the findings of Davis et al (1991). Thus, it is clear that the intervertebral disc lesion is more likely to occur as a result of a hyper-extension injury (Simeone and Goldberg, 1968; Davis et al.,1991; Jonsson et al., 1992, Jonsson et al., 1994)). There are several observations to suggest that the healing process of the disc lesion may require a prolonged period of time (MacNab, 1964; Bedbrook, 1989; Davis et al., 1991). A unique post-mortem examination of a "whiplash" patient who was suffering from chronic pain related symptoms, has clearly shown that the healing process of the injured IVD can remain incomplete even after five years post-injury (Bedbrook, 1989).

2.6.2 Lesions of the skeletal muscles

When a contracting muscle is rapidly lengthened, the length-tension ratio is adversely affected, resulting in varying degrees of micro trauma, rupture, haemorrhage and oedema in the muscles (Frankel, 1971). A number of authors have postulated various muscle lesions following trauma, on the basis of both the anatomical situation of the muscles and on observations made from experimental studies using animal and mathematical models (Pietrobono, 1956; MacNab, 1964; Hohl, 1975; Huelke and Nusholtz, 1986; Hirsch et al.,1988).

Even though there is a general agreement that the pre- and post-vertebral muscles are likely to be affected in a "whiplash" type of injury, it may not be

possible to specifically identify all of the injured muscles, especially the deeper pre- and post-vertebral muscles, during a physical examination (Jackson, 1970; MacNab, 1973; Frankel, 1971; Porter, 1989; Jonsson, Bring et al., 1991; Jonsson, 1992). The trapezius upper fibres and the sternomastoid muscles are considered to be the most frequently affected group of muscles in a "whiplash" injury and as a result of this local tenderness and muscle spasm are often reported by "whiplash" patients (Hohl, 1983; Frankel, 1971). A report based on post-mortem studies has clearly demonstrated injury to the post-vertebral muscles of the neck and the presence of haematoma in the substance of the muscles involved which is indicative of the severity of the injury (Jonsson, Bring et al., 1991; Jonsson, 1992). If there is rupture of the sternomastoid and anterior strap muscles, this can be readily recognised by demonstrable local swelling associated with haematoma formation (Frankel,1971). McNab (1964) in his study observed that a significant number of experimental animals exhibited damage to both the longus colli muscle and the muscle layer of the oesophagus, and believed that similar injury is likely to occur among "whiplash" patients. Until recently such lesions were difficult to demonstrate and hence the incidence of such an injury to the deep muscles has not been extensively documented. However, a recent MRI study of "whiplash' injury patients, demonstrated that only one out of the total nine patients examined exhibited focal haemorrhage in the substance of the longus colli muscle. Thus, even though the anatomical location of the longus colli muscle suggests that a higher proportion of "whiplash" patients are likely to show damage to the muscle, it appears that in reality this may not be a common injury. Similarly, observation at surgery also confirms the view that longus colli muscle is not usually implicated (Stokes, 1993). This is contrary to a generally held clinical view. In addition to the list of muscles mentioned, scaleni muscles are also said to be involved in the pathology associated

with "whiplash" injury (Pietrobono, 1956; Jackson, 1970). Surprisingly there is little information available with reference to the involvement of the sub-occipital muscles, levator scapulae, semispinalis capitus, splenius capitus and splenius cervicis muscles in the pathology of "whiplash". However, in an autopsy study, Jonsson (1992) has demonstrated rupture of the posterior neck muscles (possibly multifidus), in close vicinity of the zygapophyseal joints and distension or rupture of the joint capsules often associated with injuries and dislocation of the meniscoid inclusion of the facet joints.

2.6.3 Lesions of the ligaments

The rupture of inert structures such as the ligaments and the IVD has been attributed to a sudden unrestrained acceleration of the head and a noticeable failure of appropriate group of muscles to spontaneously contract without any time lag, so that the inert structures may be protected from becoming stretched beyond their physiological limit.(Frankel, 1971). Due to their deep anatomical location, it is difficult to identify injury sustained by ligaments in the cervical region and thus, current information in the issue is mainly based on surgical, experimental and post-mortem findings as well as from inferences made from radiological examinations (MacNab,1964; Marar, 1974; Scher, 1982; Jonsson, Bring et al., 1991, Rauschning, 1994). The anterior and posterior longitudinal ligaments, interspinous ligament and ligamentum nuchae have been shown to be damaged in some "whiplash" patients (Roaf, 1960; Beatson, 1963; MacNab, 1964; Janes and Hooshmand, 1965; Jackson, 1970; Davis et al., 1991). There is a diversity of opinion as to which ligaments are most likely to be damaged after a "whiplash" type of injury. The rupture of the interspinous ligament at the level of C6-C7 vertebrae, as demonstrated by the widening of the interspinous space in plain radiographs, is considered to occur more frequently than damage to any other ligaments in the cervical spine (Batalin,

1990, Chakera, 1990). The surgical findings of Janes and Hooshmand (1965) also indicate that the interspinous ligaments and the ligamentum nuchae are often ruptured as a result of "whiplash", and in addition, other observations show that rupture of the anterior longitudinal ligament is a relatively common occurrence in severe hyper-extension injuries (Simone and Goldberg, 1968; du Toit 1974; Scher, 1982; Harris et al.,1986; Swischunk,1988; Shifrin,1991). A recent MRI study has clearly demonstrated that when rupture of the anterior longitudinal ligament occurs, it may happen at a single or at multiple levels (Davis et al.,1991). However, Rauschning (1994) based on his extensive MRI study of "whiplash type" of patients observed that the frequency of a complete rupture of the anterior longitudinal ligament is considerably less when compared to the rupture of the "anterior longitudinal ligament- outer anular complex" (i.e the close attachment of the approximating part of the A.L.L to the outer most layer of the anualr fibres). Similarly, the rupture of the posterior longitudinal ligament is often seen as a result of a flexion-rotation type of violence which could eventually precipitate into subluxation of the zygapophyseal joints (Savini, Parisini and Cervellati, 1987; Swischuk, 1988). It is also evident from an autopsy study of the MVA victims that atlanto-occipital ligament rupture can also occur (Shkrum, Green and Nowak,1989; Jonsson, Bring et al., 1991). While damage to the alar ligament lesions among "whiplash" patients has not yet been clinically demonstrated, observation made during post-mortem examination of MVA victims indicate that such a lesion may occur in "whiplash" injury (Dvorak and Panjabi, 1987; Jonsson, Bring et al., 1991; Schonstrom, 1992). The alar ligament is maximally stretched when the cervical spine is rotated and flexed, as it happens in some rear-end collisions (Dvorak and Panjabi, 1987) and a recent post-mortem examination of the cervical spine of MVA victims has even demonstrated the existence of alar ligament lesion and associated fracture of its bony

attachment (Schonstrom, 1992). A post-mortem study of MVA victims highlighted an association between the severity of the ligamentous injury and the presence of skull fractures (Jonsson, Bring et al., 1991). It is interesting to note that the victims with skull fractures, were seen to have less severe ligamentous damage, whereas those victims with severe ligamentous lesions did not have skull fracture. The inference drawn from the observations of Jonsson, Bring et al. (1991) indicated that the skull fractures facilitate an absorption of some of the energy generated by the impact of the head against the interior of the motor vehicle, therefore the cervical spine is likely to experience a lesser force. It is worth noting that the above information may be clinically relevant.

It is also believed that a sudden excessive torsional force is likely to induce an injury to the soft tissue of the motion segment, resulting in a reduction of torque strength by 40% which in itself will contribute towards "spinal instability" (Farfan and Gracovetsky,1984). Post-traumatic progressive instability of the ligaments of the cervical spine is considered one of the complications following "whiplash" type of injury (Wilberger and Maroon, 1990). Pfeifer (1990) described a syndrome called "delayed instability of the cervical spine", in which patients who initially have had normal radiographs, developed instability due to the failure of the ligaments, up to three weeks after the injury, requiring surgical intervention to stabilise the spine.

2.6.4 Bony lesions

Fractures of the vertebral elements and or a combination of fracture / subluxation of the cervical vertebrae associated with a "whiplash" type injury have been extensively reported (Hohl, 1964; Burke, 1971; du Toit, 1974; Harris, 1978; Gehwiler et al., 1981; Scher, 1982; Penning, 1983; Harris et al., 1986; Clark et al.,1988, Swischuk, 1988; Batalin, 1990; Chakera, 1990;

Kastonas and Jakim, 1991; Jonsson, Bring et al, 1991; Merianos, Tsekouras and Koskinas, 1991; Twomey and Taylor, 1991a, Twomey and Taylor, 1991b; Taylor and Twomey, 1993). However, it may not be always possible to demonstrate bony lesions by routine radiological examination alone (Marar, 1974; Schonstrom, 1992; Twomey and Taylor, 1989; Twomey and Taylor, 1991a; Twomey and Taylor, 1991b). A recent histological study demonstrated that even though it is not always evident during routine radiological examination, micro-fractures can occur in the cervical vertebrae and facet joints of motor vehicle accident victims (Twomey and Taylor, 1991b; Taylor and Twomey, 1993).

Fracture of the spinous process of the C6 and C7 vertebrae is the most common bony lesion in a flexion-extension type of injury as in a rear-end collision (Harris, 1978; Scher, 1982; Penning, 1983; Harris et al., 1986). This may be due to sudden application of a tensile force to the bony attachments of the interspinous ligament and the ligamentum nuchae. When the flexion moment of the spine exceeds the normal physiological limits of the vertebrae, a crush wedge fracture, also known as a "Chance fracture" may occur (Chance, 1948). In the cervical spine, such a wedge fracture of the vertebral body associated with hyper-flexion type of injuries is usually seen at the C7 and T1 vertebral levels (Chakera, 1990). Another relatively common bone injury is a small avulsion of the anterior inferior edge of the vertebral body at the point of attachment of the anterior longitudinal ligament (du Toit, 1974). Similarly, a lamellar fracture of the atlas and pedicular fractures of the C5, C6 and C7, are often associated with high velocity impacts (Batalin, 1990). In addition to the above and as a result of high velocity impact, uni- and bi-facetal subluxation of the facet joints and motion segments, (without cord involvement), can also occur in

the lower cervical spine, usually also incorporating small fractures such as the tips of the articular process (Batalin, 1990).

Hangman's fracture (of the odontoid process), and fracture of the arch of the atlas and pedicle of the axis are bony lesions induced by a hyper-extension type of injury (Skold and Voigt,1977; Shkrum et al.,1989). Although it is less frequent, an avulsion fracture of the anterior tubercle of the atlas can also occur as a result of a hyper-extension type of injury (Kastonas and Jakim,1991). Although it is rare, Hohl (1964) has recorded lateral displacement of the atlas on the axis, with an intact odontoid peg, which resulted from a rear-end collision.

2.6.5 Lesions of the vertebral artery

Vertebral artery lesions arising from a "whiplash" accident are recognised clinical entities (Schneider and Crosby, 1959; Schneider and Schemm, 1961; Simeone and Goldberg, 1968, Toglia, 1976). The anatomical relationship of the vertebral artery to the unco-vertebral joint predisposes the vessel to injury under certain conditions. Thus, while the anteromedial part of the vertebral artery is in contact with the anterior third of the lateral aspect of the unco-vertebral joint from C2-C6, the posteromedial part of the vertebral artery is in close approximation with the foramen transversarium. In this position, large osteophyte formation at the unco-vertebral joint level or at the zygapophyseal joint may abut and potentially damage the vertebral artery and may even compress it especially at the extreme of flexion or extension movements of the cervical spine (Constantin and Lucretia, 1971).

The vertebral artery as it lies in the foramen transvarium at the level of the C6 vertebra, is also likely to be stretched across the bony fulcrum formed by the transverse process, when subjected to extreme force as encountered in

sudden, full range extension of the cervical spine. This mechanism has been shown to cause disruption of the vessel wall and initiate dissection of the vessel and release of a propagating thrombus (Simeone and Goldberg, 1968). A vascular spasm or compression of the vertebral artery thus induced may initiate a relative intracranial vascular insufficiency in the areas supplied by the basilar artery and its branches, as well as to the spinal cord in the cervical region supplied by the anterior spinal artery (Schneider and Schemm, 1961; Simeone and Goldberg, 1968). In their significant case report of a MVA victim who died within 24 hours after the accident, Simeone and Goldberg (1968) presented a graphical account of the signs and symptoms of a progressing thrombosis which developed as a result of bilateral vertebral artery injury. The autopsy finding showed that due to hyper-extension of the cervical spine, the left vertebral artery as it entered the foramen transversarium of the C6 was stretched across the bony fulcrum, thus disrupting the vessel wall and initiating a propagating thrombus. The thrombus extended into the posterior-inferior cerebellar arteries and completely occluded the basilar artery to its bifurcation, thereby resulting in necrotic softening of the posterior and inferior portion of the cerebellum.

2.6.6 Lesions of the central nervous system (CNS)

Most of the available information with regard to the lesions of the central nervous system are based on experimental and post-mortem studies (Schneider, Cherry and Pantek, 1954; Roaf, 1960; Ommaya et al.,1968; Gosch,1972). A detailed knowledge of the types and sites of lesions of the central nervous system is essential in order to help account for some of the bizarre symptoms reported by the "whiplash" patients, which are often dismissed by clinicians as being non-organic. The nature of the lesion and the CNS structures involved may vary depending upon the mechanism of

injury. A direct impact of the head or chin against the windscreen, steering wheel, dash board, the door or the head rest is one of the common causes of traumatic brain lesions after a "whiplash" accident (Krantz and Lowenhielm 1986). However, a non contact injury due to the rapid flexion-extension movement of the head relative to the torso, also has the potential to cause lesions such as brain stem contusion, high cervical cord transection, subdural or subarachnoid haemorrhage (Oppenheimer, 1968; Hadley, Sonntag, Rekate and Murphy, 1989; Shkrum et al.,1989). In addition, a variety of other lesions such as gross haemorrhage of the upper cervical cord and intracranial injuries such as brain laceration, cortical contusion, subarachnoidal and intracerebral haematoma have been shown to occur as a result of a "whiplash" type of injury (Ommaya et al.,1968; Krantz and Lowenhielm, 1986). Similarly, an angular acceleration type of injury may result in cortical and / or subcortical haemorrhages sometimes referred to as gliding contusions or rotational cerebral injuries (Krantz and Lowenhielm, 1986). The disruption of the parasagittal bridging vein due to shear forces is an additional factor sometimes responsible for the subdural haematoma. Less severe focal (contusion) and diffuse injuries (concussion) to the brain may also occur as a result of "whiplash" (Gennarelli, Spielman and Langfitt, 1982).

It is evident that the manifestation of a neurological deficit can exist even in the absence of a radiologically demonstrable bony lesion (Barnes, 1948; Schneider et al., 1954; Roaf, 1960; Goschet al.,1972). This may be due to the subluxation and spontaneous reduction of the zygapophyseal joints at the time of the injury, which may not be evident in subsequent radiological examination (Roaf,1960). Such a mechanism may be responsible for the lesions documented by Schneider et al. (1954) demonstrating the incidences of central cervical cord syndrome in which upper limb paralysis

have occurred, while the lower limb functions remained intact, in instances where the radiological findings were normal. The extension type of injuries have the potential to cause lesions of the long spinal tracts, manifesting as either an anterior or a posterior cord syndrome (Bedbrook, 1969).

The spinal cord, the intrinsic blood vessels and the plexuses of blood vessels surrounding the spinal cord are susceptible for compression forces due to a reduction in the size of the spinal canal brought about by a combination of several factors (Schneider and Schemm, 1961). It has been clearly shown that the cross section of the spinal cord decreases during flexion of the cervical spine and considerably increases during extension (Breig and El-Nadi, 1966). Under normal conditions, the spinal canal is capacious, therefore an increase in the size of the spinal cord as seen during extension of the cervical spine is unlikely to adversely affect the neural and vascular structures contained within this bony structure. However, the normal anatomical relationship between the contents of the spinal canal and the structures contained in it may be affected when disc bulging occurs or when free fragments of the IVD are present in the canal. A combination of the disc lesion caused by "whiplash" type of injury and pre-existing spondylitic changes and associated "buckling" of the ligamentum flavum, is likely to decrease the volume of the spinal canal even further (Schneider et al., 1954; Breig and El-Nadi, 1966; Batalin,1990). In this situation, as a result of a sudden hyper-extension of the cervical spine, the neural structures together with their blood vessels may be compressed between the bulging disc material and the "buckling" ligamentum flavum (Schneider et al., 1954; Burke, 1971). In this way, the spinal cord may be damaged as a result of a direct mechanical compromise or else experience partial or relative vascular insufficiency, resulting in a neurological deficit (Schneider and Schemm, 1961). It is also possible that such a mechanism

of injury may be responsible for the cord oedema demonstrated by Davis et al. (1991) amongst a significant percentage of hyper-extension injuries. When present, cord oedema can affect the micro-circulation in the spinal cord due to the thickening of the capillary walls and extravasation of blood and fluid from the vessels, eventually resulting in shrinking of the axons and eventual tissue necrosis (De La Torre, 1981; Hackney, Asato, Joseph, Carvlin, McGrath, Grossman, Kassab and De-Simone, 1986; Mirvis, Geisler, Jelinek, Joslyn and Gellad, 1988). A mechanical compromise of the epidural venous plexus may also lead to venous congestion and the eventual dilation of the non-compressed veins thus adversely affecting the neural tissues (Jansen, Bardosi, Hildebrandt and Lucke., 1989; Jayson, 1992, Jayson, 1993).

2.6.7 Lesions of the peripheral nerves

Lesions of the peripheral nerves among "whiplash" patients may be attributed to mechanical causes such as crushing, compression, and over stretching of the neural tissues (MacNab, 1964; Keith, 1986, Davis et al., 1991; Wetzel, 1992). An obstruction of the epidural veins is likely to result in venectasia, thus inducing a state of " varicose" veins in the epidural space (Jansen et al., 1989; Jayson, 1992). In this situation it has been shown that the densely formed, enlarged veins have the potential to compress the dorsal root ganglion as well as the nerve root by forming a tight cuff around the nerve root (Jansen et al., 1989). Jayson (1992) has also demonstrated that obstruction of the epidural veins can lead to proliferation of the fibrous tissues inside and around the nerve root resulting in eventual fibrosis of the neural structures. Schneider and Johnson (1971) believed that an extension force applied to the head from physical contact against the internal parts of a car, may be responsible for some of the cranial nerve injuries reported. Even though the incidence of cranial nerve injury is not

frequent, there are a number of case reports showing involvement of the trigeminal, abducent , vagus, hypoglossal and accessory nerves and even an unusual combination of bilateral abducens nerve palsy and trigeminal nerve injury resulting from a hyper-extension injury have been reported (Helliwell, Robertson, Todd, and Lobb, 1984; McGlone, Morton and Sloan., 1988; Shifrin,1991). However, the incidence of trigeminal nerve injury is the highest for all cranial nerves (Simeone and Goldberg, 1968; Hildingsson and Toolanen, 1990).

Recent detailed anatomical studies indicate that the 2nd cervical ganglion may be compressed between the posterior arch of the atlas and the superior articular process of the axis, during cervical extension combined with contralateral rotation of the suboccipital joints (Bogduk, 1981; Keith, 1986). Vital, Grenier, Dautheribes, Baspeyre, Lavgnolle and Senegas (1989) demonstrated that as a result of an acute flexion of the cervical spine, the posterior roots of the C2 nerve could be stretched over the denticulate ligament. Keith (1986) described an account of symptoms reported by thirteen patients involved in a "whiplash" type of injury where he considered that the second cervical nerve root and ganglion had sustained compression injury. It is evident from the reported outcome of the surgical division of the C2 nerve roots that Keith (1986) was able to retrospectively confirm his initial diagnosis.

In his experimental study of monkeys, MacNab (1964) observed that damage to the longus colli muscle was invariably associated with damage to the sympathetic nerves of the cervical region and believed that under similar circumstances, the sympathetic nerve to be similarly involved in human "whiplash" patients. The course and relationship of the sympathetic nerve to the vertebral artery is said to predispose the nerves to be stretched

and mechanically irritated by osteophytes present in the region of the uncinate processes at the levels of mid and lower segments of the cervical spine (Constantin and Lucretia, 1971; Xiuquing, Bo and Shizhen, 1988). Any instability of the cervical spine is also capable of compressing or stimulating the sympathetic nerve as well as its endings (Xiuquing et al., 1988).

2.6.8 Other injuries

Electronystagmography studies conducted by Rubin (1973) and Hinoki (1985) have confirmed the existence of vestibular apparatus lesions attributable to "whiplash" type injury. TMJ dysfunction related to "whiplash" injury has also been reported by several authors (Frankel, 1971; Scheneider et al, 1989; Heise, Laskin, and Gervin, 1992).

2.7 Symptoms and their causes

There is considerable disagreement among researchers regarding what is meant by the "whiplash syndrome" and the extent and validity of the symptoms which may arise from what is sometimes considered to be a relatively innocuous injury (Frankel, 1959; Pearce, 1989; Awerbuch, 1992). It is obvious from the literature that there is a well documented spectrum of symptoms resulting from the "whiplash" type of injuries, which often fail to correlate with the recognised patterns of symptoms as evidenced following trauma in other parts of the musculo-skeletal system. Although symptoms such as headache, neck pain and associated restriction of movements in the cervical spine, have been undisputedly attributed to the "whiplash" type of injury, some of the more unusual symptoms reported including vertigo, tinnitus, ocular problems, impaired memory, emotional instability, peripheral vascular disturbances, and attacks of paraesthesia in a non-anatomical pattern of distribution, are considered to be unusual and may receive scant

attention from clinicians (Miller, 1961a; Miller, 1961b; Miller, 1966; Pearce, 1989). However, as previously indicated (sections 2.6.6 and 2.6.7), a number of studies have shown that the cranial nerves, sympathetic plexus, parts of the brain, spinal cord, nerve root, spinal ganglion, blood vessels and auditory apparatus have been implicated as sources of some of the complex symptoms reported by the patients after "Whiplash" type of injury (Schneider and Schemm, 1961; Horwich and Kasner, 1962; Ommaya et al., 1968; du Toit, 1974; Berstad, Baerum, Lochen, Mogstad and Sjaastad, 1975; Toglia, 1976; Fisher, 1982; Merskey, 1984; Hinoki, 1985; Keith, 1986; Schwartz, Barth, Dane, Drenan, De Good and Rowlingson,1987; McGlone et al., 1988; Hadley et al.,1989; Hildingsson, Wenngren, Bring and Toolanen, 1989a; Tamura,1989; Chester, 1991; Shifrin, 1991; Radanov, Dvorak and Valach, 1992; Radanov, Di Stefano, Schnidrig and Sturzenegger, 1993a; Brown, 1995). Thus, the changes that follow an injury either to the peripheral nerve or the central nervous system itself may be associated with some of the chronic symptoms reported by "whiplash" patients (Wetzel, 1992). It is also evident that in spite of the vast array of current information on the topic, there has been a failure by some clinicians to acknowledge many of the previous studies.

2.7.1 Headache

Headache is one of the commonest symptoms among patients suffering from the effects of the "whiplash" type of injury and this symptom is often noticed within a few hours following the MVA (Berryman, 1956; Hohl, 1974; Hohl, 1975; Merskey 1984; Mark, 1990; Radanov et al., 1992; Radanov, Sturzenegger, Di Stefano, Schnidrig and Aljinovic, 1993b; Radanov, Sturzenegger and Di Stefano, 1995). Owing to the clinical significance of the "headache" associated with "whiplash", several investigators have studied the frequency and length of time taken for it to resolve (Hohl, 1974;

Hohl, 1975; Norris and Watt, 1983; Balla and Karnaghan 1987; Merskey 1984; Radanov et al., 1993b; Radanov et al., 1995). The information reported by Hohl (1974) and Balla and Karnaghan (1987) is based on extensive clinical observations and large series of patients and are thus worthy of close consideration. It is evident that within four weeks after the injury, two third of the "whiplash" patients are affected by headache (Hohl,1974; Balla and Karnaghan, 1987). In a prospective study of 180 subjects, Balla and Karnaghan (1987) showed that 82% of their subjects suffered from headache, an observation similar to that of Hohl (1974). The observations made in a more recent prospective study, which concentrated on headache among "whiplash" patients, while supporting previous reports noted that 76% of their subjects complained of headache, within 7 days post-injury (Radanov et al., 1995). This symptom may be manifested either as occipital or frontal headache or in a generalised and non-specific distribution. Thus, Balla and Karnaghan (1987) noted that 46% of "whiplash" subjects suffered from occipital headache while 34% complained from non-specific form of headache. It has also been clearly shown that headache is one of the predominant symptoms among chronic "whiplash" patients (Hohl,1974; Balla and Karnaghan, 1987; Pearce, 1989). Thus, in a retrospective analysis of 5000 "whiplash" patients, cited by Balla and Karnaghan (1987) found that 25% of their patients developed chronic disability due to headache, whereas Pearce (1989) observed that 43% of his patients suffering from protracted symptoms (that is six months post-injury) were shown to have headache. However, Radanov et al. (1995) observed that 86% of their subjects continued to be affected with chronic headache up to 2 years post-injury. Although headache is considered to be the most disabling and common symptom, it is evident that there is no adequate documentation in the literature of the distribution, severity,

description, time scale related to the onset, mechanism of injury and eventual progress of this symptom.

Several causes have been identified as being responsible for the onset of headache following a "whiplash" injury. Thus, cerebral concussion is considered to be one of the important causes of headache among patients suffering from cervical traumatic syndromes, while even a small amount of subarachnoid haemorrhage has been shown to produce headache (Goff et al., 1964; Ommaya et al., 1968). Injury to the muscles supplied by the C1 and C2 dorsal rami is also considered to be one of the causes of headache (Bogduk, 1982). Radanov et.al. (1992) considered that headache should be regarded as a part of the cervico-encephalic syndrome, indicating that the symptom is the result of an injury to the structures of the upper cervical spine and the contents of the cranium. In an earlier study, Torres and Shapiro (1961) have demonstrated EEG abnormalities among patients suffering from headache related to the "whiplash" type of injury. According to Seletz (1958), however, vasomotor disturbance resulting indirectly from the damage to the vertebral arteries and nerve fibres that accompany these arteries in their course through the foramina transversarium, is one of the important causes of headache experienced among patients suffering from "whiplash".

Various other cervical structures including the zygapophyseal joints, atlanto-occipital, ligaments of the cervical spine, anulus fibrosus of the intervertebral discs, periosteum of the vertebral bodies, the cervical muscles and their attachments, have all been shown to produce headache when adequately stimulated (Edmead, 1978, Bogduk, 1986a; Pfaffenrath, Dander, and Pollmann, 1987; Winston, 1987). Similarly, Janes and Hooshmand (1965) described injury to the atlanto-axial joints and its structures as an important

cause of headache. In extending Edmead's (1978) observation, Bogduk, Corrigan, Kelly, Schneider and Farr (1985), showed that sternomastoid, sub-occipital muscles and trapezius, were also capable of inducing cervical headache, when subjected to noxious stimuli. Observations made during the follow-up examinations of patients who have undergone spinal fusion procedures, confirm that the zygapophyseal joints and intervertebral discs at the level of C2-C3 and C3-C4 vertebrae, are also sources of headache (Batalin,1990). A unique post-mortem finding of a whiplash patient who suffered from headache over a period of four years, demonstrated that the healing process of the initial intervertebral disc injury remained incomplete even after several years, thereby suggesting that there may be a link between the original IVD injury and headache (Bedbrook, 1989). In addition, recent studies indicated that the nerve endings present in the outer third of the anulus fibrosus may also cause pain-related symptoms as a result of disc injury (Bogduk, Windsor and Inglis, 1988; Mendel, Wink and Zimny, 1992).

There are several studies which implicate the second cervical root, the greater occipital nerve and its ganglion as a major factors in occipital headache (Seletz, 1958; Edmeads, 1978; Keith, 1986; Vital et al., 1989). Thus, Seletz (1958) believed that a traction injury to the root of the second cervical nerve could be a major cause of the occipital headache. Similarly, the course of the greater occipital nerve suggests that it is possible for this nerve to be stretched over the inferior oblique muscle as a result of sudden forward flexion of the cervical spine (Vital et al.,1989). In an earlier study, Edmeads (1978) considered that the second cervical nerve sensory root or greater occipital nerve may be crushed between the atlas and the axis as the head is forcibly extended and rotated. He also considered that another likely cause for headache, is a possible compression of the sensory root of

the C2 nerve, by swollen muscles in a state of spasm. Similarly, Keith (1986) reporting on 13 patients involved in a motor vehicle accident who complained of headache confined to the upper neck and occipital region on one side, concluded that a crushing injury of the 2nd cervical nerve and / or its ganglion was the cause of the persisting occipital pain. An unpublished report based on surgical findings demonstrated that even though there was no evidence of a crush injury, the ganglion of the greater occipital nerve was often seen to be unusually "hyperaemic", suggesting an increased level of neuronal activity (Stokes, 1993). Stokes (1993) has also observed on several occasions that the venous plexus situated in the spinal canal, was grossly enlarged, and in this situation, there is a strong possibility for the greater occipital nerve root and the ganglion to be compressed by the "varicosed" veins, thus precipitating the chronic headache often seen among "whiplash" patients (Jansen et al., 1989; Jayson, 1993). The presence of the scar tissues in the greater occipital nerve at the level of the nuchal line, as routinely observed during surgical division of this nerve in order to relieve intractable headache among "whiplash" patients, suggests that the greater occipital nerve may be subjected to crush injury due to the impact of the head against the head rest (Stokes, 1993). Furthermore, recent studies showed that the incidence of headache resulting from "whiplash" injury was higher among those individuals who had a history of headache prior to their MVA and recommended that such patients be identified for receiving appropriate treatment at an early stage of their injury (Radanov et al., 1993b; Radanov et al., 1995).

Bogduk et al. (1985) showed that cervical headache may be referred to areas of the head such as the occiput which are innervated by cervical nerves or the areas innervated by the trigeminal nerve. In the latter example they considered that the possible neuroanatomical source of such

referral is likely to be the convergence of the cervical and trigeminal afferents on common neurones in the trigemino-cervical nucleus.

2.7.2 Neck pain

Pain in the cervical region is the most common early symptom following a "whiplash" type of injury (Gay and Abott, 1953; Breck and Van Norman, 1971; Frankel, 1971; Hohl, 1974; Hohl, 1975; Toglia, 1976; Hohl, 1983; Maimaris et al., 1988; Hildingsson, Hietala and Toolanen, 1989b; Porter, 1989; Hildingsson and Toolanen, 1990). During the acute stage regardless of the primary structures involved, neck pain is often associated with muscle spasm as evidenced in most subjects (Hohl, 1975). The incidence of neck pain reported in various studies ranged between 88% and 100% (Maimaris et al., 1988; Pearce, 1989; Hildingsson et al, 1989b; Hildingsson and Toolanen, 1990; Radanov et al., 1995). Similarly, it appears that a large number of "whiplash" patients continued to be affected with chronic neck pain up to 10 years after their injury (Watkinson et al., 1991). Deans et al. (1987) noted that 36% of their subjects complained of neck pain 12 months after the MVA. However, Maimaris et al. (1988) recorded that 35% of his patients considered that continuing neck pain was a major factor which interfered with their normal activities of daily living up to 2 years post-injury while Radanov et al. (1995) reported the incidence as 90% for a similar period. In another retrospective study also showed that 10 years after the MVA, 36% of the subjects complained of persistent neck pain. While a large number of muscles have been shown to be affected during the initial stage of the injury, the sternomastoid and trapezius muscles have most commonly been shown to be those responsible for protracted neck pain (Breck and Van Norman, 1971; Frankel, 1971, Hohl, 1983). Although the above symptoms are mostly associated with an over stretching or a sudden contraction of the respective muscles, it has been postulated that similar

symptoms may also be attributed to a traction injury sustained by the accessory nerve due to a "whiplash" injury (Seletz, 1958).

The capsular structure of the atlanto-occipital (AO) and atlanto-axial (AA) joints, when subjected to mechanical provocation, has been shown to cause headache and referred pain in the neck region (Dreyfuss, Michaelsen and Fletcher, 1994). In this study, the AO and AA joint capsule were distended by injecting radio opaque substance and the perceived sensation of pain reported by the subjects, demonstrated that the AO capsule may be relatively more sensitive to mechanical injury when compared to AA capsular structure under similar circumstances.

Injury to the anterior and posterior longitudinal ligaments of the cervical spine, as evidenced in the collateral ligaments of the peripheral joints, are likely sources of pain and there is a strong evidence to support this possibility. A histochemical study has shown that the posterior longitudinal ligament is capable of transmitting pain related afferent impulses (Korkala, Gronblad, Liesi and Karaharju, 1984). In this study the above investigators demonstrated the presence of nociceptive nerve endings and substance P, which is a polypeptide known to act as a nociceptive transmitter, in the posterior longitudinal ligament.

A recent histological study has lent support to the concept that the capsule of the zygapophyseal joints in the cervical spine, is another source of the neck pain (McLain,1994). In this study, the capsular structures are shown to contain Type-I, Type-III (mechanoreceptors) and unmyelinated nerves with fine terminals, considered to be nociceptive in function. McLain (1994) observed that the above specialised neural structures were consistently present in all the twenty one specimens examined and also noted that the

neural population was uniformly represented in both upper and lower cervical segments.

Recent studies indicated that pain-related symptoms including the neck, shoulder girdle and interscapular regions may be due to pathological changes associated with cervical zygapophyseal joints (Dwyer, Aprill, and Bogduk, 1990; Aprill, Dwyer, and Bogduk, 1990; Aprill and Bogduk, 1992). The authors also showed that the pain pattern related to a particular level of zygapophyseal joint was specific in its distribution and highly predictable. Furthermore, a subsequent prospective double blind, controlled study performing sequential nerve blocks, (by injecting short and long acting anaesthetic agents), concluded that the zygapophyseal joints of the cervical spine are one of the major sources of chronic neck pain following neck injury (Barnsley, Lord, Wallis and Bogduk, 1995). In this situation, the above authors observed that the pathological state of the zygapophyseal joint may exist either at a single level or at several spinal levels. Barnsley et al. (1995) further added that the most common motion segments thus affected were confined to C2-3 and C5-C6 (when present in a single level), whereas multi level lesions occurred in the C2-C3 and C5-C6, and C5-6-7 motion segments. Even though the above investigators confidently implicated the zygapophyseal joints as a significant source of chronic neck pain, the nature of the pathology in this situation is yet to be explained.

Pain related symptoms in the cervical spine may also arise as a result of abnormal movement in the motion segment, resulting in a clinical entity which is generally described as "spinal instability" (Farfan, 1977; Farfan and Gracovetsky,1984). Amevo et al. (1992) investigated the correlation between spinal instability and the neck pain experienced and suggested that a substantial proportion of patients with neck pain may have abnormal

movement in the motion segments of the cervical spine. An important factor which contributes for the instability of the motion segment is degeneration of I.V.D either due to age related degenerative changes or due to trauma (Osti, Vernon-Roberts and Fraser, 1990; Kaapa, Han, Holm, Peltonen, Takala and Vanharanta, 1995). Injury to the anulus fibrosus has been shown to cause a secondary cellular reaction in the nucleus pulposus, thus precipitating an early disc degeneration (Osti et al., 1990; Natarajan, Ke and Andersson, 1994; Kaapa et al.,1995).

Limitation of active and passive motion in the cervical spine is usually often associated with neck pain and interferes with many of the activities of daily living (Gay and Abott, 1953; Janes and Hooshmand, 1965; Breck and Van Norman, 1971; Frankel, 1971; Hohl, 1974; Hohl, 1975; Hohl, 1983; Toglia, 1976; Maimaris et al., 1988; Porter, 1989; Hildingsson and Toolanen, 1990). During the acute stage of the injury, it is evident that muscle spasm and pain play a major role in inhibiting active and passive movements in the cervical spine (Gay and Abott, 1953; Frankel, 1959; Janes and Hooshmand, 1965; Breck and Van Norman, 1971, Stevens, 1986). A number of authors have observed that pain in the interscapular region is also one of the commonest early symptoms reported by the "whiplash" patients (Hohl, 1975; Hohl and Hopp, 1978; Norris and Watt, 1983; Maimaris et al., 1988). Although the interscapular pain is considered to be of prognostic value, the correlation between the interscapular pain and its source has not been clearly established (Hohl and Hopp, 1978; Norris and Watt, 1983; Maimaris et al., 1988).

2.7.3 Shoulder girdle pain

Pain in the region of upper and middle fibres of trapezius muscle is one of the most intractable and often debilitating symptoms reported by "whiplash"

patients (Larsson, Alund, Cai and Oberg, 1994). In a recent study, the microcirculation in the trapezius muscles of twenty five "whiplash "patients (post-injury period ranging from 3 to 108 months) was measured using Lasser-doppler flowmetry. Dynamic recording of the microcirculation was obtained while the trapezius muscles were subjected to stepwise static loads and significant dysfunction in the regulation of the microcirculation was evident from this study. This circulatory disorder was attributed to a continuous flow of afferent impulses from the sensory nerve endings resulting in an interference with the production of chemicals responsible for vasodilatation (Larsson et al., 1994). It may be possible that there could be other reasons for the chronic pain in the trapezius muscles. However, the contribution made by Larsson et al. (1994) is significant, since for the first time a plausible explanation has been put forward to characterise the pathology of this poorly understood symptom, which often causes emotional distress to the patients when they encounter a sceptical medical specialist. A localised muscle fatigue and associated muscle tenderness after strong muscle contractions are common features even among asymptomatic individuals (Gogia and Sabbahi, 1991) and hence the development of chronic pain in a group muscles which has experienced trauma is not a surprise.

2.7.4 Neurological signs and symptoms

Symptoms such as pins and needles, distally radiating pain, altered sensation to light touch, sharp and blunt objects; altered thermal sensation, paraesthesia, hyperaesthesia, altered muscle power, Horner's syndrome and elicited signs like altered reflex responses in the upper and lower limbs, Babinski's reflex, the presence of altered vibration sensation are among the many neurological signs and symptoms which are known to occur as a result of a "whiplash" injury (Gay and Abbott, 1953; Haxton,

1954; Janes and Hooshmand, 1965; Reichl and Allen, 1987; Hildingsson and Toolanen, 1990). In all of these circumstances, the possible pathology might be related to an injury to the spinal cord, the peripheral nervous system, cranial nerves or the sympathetic nervous system (Gay and Abbott, 1953; Torres and Shapiro,1961; Janes and Hooshmand, 1965; Jackson, 1970; Berstad et al 1975; Merskey, 1984; Bogduk et al., 1985;.Hinoki,1985; Reichl and Allen, 1987; McGlone et al., 1988; Hildingsson et al., 1989a; Biby and Santora, 1990; Shifrin, 1991; Smith and Hodge, 1992).

In 1926, a cluster of symptoms were described and which later came to be known as the "Barre'-Lieou syndrome", consisting of vertigo, tinnitus and ocular problems (Wing and Hargrave-Wilson, 1974; Tamura, 1989, Burke, Orton, West, Strachan, Hockey and Ferguson, 1992). Patients suffering from Barre'-Lieou syndrome tend to develop secondary symptoms such as restlessness, irritability, and an inability to concentrate (Tamura,1989). Although there is a reluctance among the clinicians to recognise some of these symptoms, a number of studies have confirmed the existence of organic causes which may be responsible for "Barre'-Lieou" syndrome (Torres and Shapiro, 1961; Horwich and Kasner, 1962; Janes and Hooshmand, 1965; Breck and Van Norman, 1971; Berstad et al., 1975; Hinoki and Niki, 1975; Toglia, 1976; Merskey, 1984; Hinoki, 1985; Schwartz et al., 1987; Hildingsson et al.,1989a; Radanov et al., 1992; Smith and Hodge, 1992).

It is also evident that many "whiplash" subjects complain of symptoms commonly referred to as an ocular impairment. Some of the specific symptoms such as asthenopia, (i.e, weakness of accommodation) and convergence, pupillary changes, disturbance of ocular convergence, nystagmus, dilatation of retinal vessels and disturbed adaptation to light

have often been described (Horwich and Kasner, 1962; Merskey, 1984; Radanov et al., 1992). Blurred vision has also been widely reported as an important symptom (Janes and Hooshmand,1965; Breck and Van Norman, 1971; Toglia, 1976; Hohl, 1983; Hildingsson et al., 1989a). Photo sensitivity is also an infrequent symptom presented after a "whiplash" injury and when present, it affects either one or both eyes and is generally reported to be noticed a few days after the initial accident (Radanov et al.,1992).

Impairment of memory, particularly associated with recent events has also been well documented as a symptom following "whiplash" (Toglia, 1976; Fisher, 1982; Schwartz et al., 1987; Yarnell and Rossie, 1988; Radanov et al.,1992; Radanov et al., 1993a). Fisher (1982) used the term, "nonpermanent retrograde amnesia" to describe the memory impairment, since the problem usually resolved through the passage of time. However, a follow-up study of twenty seven patients twelve months and longer after the MVA showed that cognitive deficit, complaint of poor memory, concentration and attention deficits, associated with easy distractibility and difficulty in learning and retaining information were persistent (Yarnell and Rossie,1988). Even though, the electroencephalogram, C.T. scan and M.R.I. investigations were normal, the subjects demonstrated significantly abnormal results when tested by most sensitive neuro-psychological battery tests for the following:

a. sustained visual scanning and speed processing (abnormal findings-86%);
b. sustained concentration on mental arithmetic (abnormal findings-85%);
c. non-verbal abstract reasoning (abnormal findings-68%);
d. ten word memory test and delayed component (abnormal findings-86%); and

e. visual scanning and new learning skills, motor speed and nonverbal processing speed (abnormal findings-65%).

The interesting feature of the above tests is that the results were similar in characteristics when compared to patients with minor head injury with a history of "severe persisting symptoms and disability" associated with cerebral dysfunction (Yarnell and Rossie,1988). Recent studies lend further evidence to support the concept that the cognitive deficit demonstrated by some of the "whiplash" patients is a genuine symptom and this symptom may be attributed to a concussion type of injury to the brain as a result of "whiplash" injury (Radanov et al.,1992; Radanov, 1993a).

Jackson (1970), noticed that some of her patients complained of clumsiness while handling objects. According to Jackson (1970), clumsiness is considered to be one of the symptoms which arise due to subtle neurological changes affecting the proprioception in the upper extremities.

Thus, it is evident that a variety of neurological symptoms reported by "whiplash" patients have been recorded in the literature. Those neurological lesions which can be readily demonstrated during a routine clinical examination, have been clearly understood and recognised by clinicians. However, there are several less understood and complex neurological symptoms which may require sophisticated investigation methods and which are not readily available to a clinician. Therefore it is not surprising that some of the bizarre neurological symptoms may be labelled as inappropriate and due to non-organic causes.

2.7.5 Other symptoms

Tinnitus is a widely reported symptom which is related to disturbance in the auditory apparatus (Hinoki and Niki, 1975 ; Toglia, 1976; Hinoki, 1985). This symptom may be noticed a few hours after a MVA causing "whiplash" and may be present for several months. Retro-orbital pain, blurring of vision, diplopia and an enlarged pupil may also occur infrequently as a result of injury to the autonomic system in the neck or due to vascular impairment (Schneider and Crosby, 1959; Schneider and Schemm, 1961; Ommaya et al., 1968; Hohl, 1975; Brown, 1995), while pain in the temporomandibular joint as a result of "whiplash" type injury has also been documented (Scheneider et al.,1989, Heise et al.,1992).

2.7.6 Possible causes for the development of chronic pain

The emotional and litigation factors are often considered to be the causes for some of the chronic symptoms reported by "whiplash" patients (Gay and Abott, 1953; Gotten, 1956; Miller, 1961a; Miller, 1961b; Miller, 1966; Balla and Morattis, 1970; Breck and Van Norman, 1971; Balla, 1980; Balla, 1982; Awerbuch, 1992). However, factors that adversely affect the healing of injured soft tissues and certain changes that follow an injury to either the peripheral nerve or the central nervous system itself, may also lead to the development of chronic pain among "whiplash" patients (Gelberman, Menon, Gonsalves and Akeson, 1980; Nystrom and Holmlund, 1983; Korkalaet al., 1984; Lehto, Duance and Restall, 1985; Walter and Israel, 1987; Montgomery, 1989; McGuire, Degnan and Amundson, 1990; Rosomoff, Fishbain and Rosomoff, 1992; Wetzel, 1992). In this situation, irreversible physiological changes occur, causing aberrations in the modulation of afferent nociceptive in put thus, culminating in the development of central hypersensitivity within the spinal cord itself (Wall and Devor, 1981; Wall, 1991; La Rocca, 1992; Rosomoff et al., 1992;

Wetzel, 1992Konttinen, Kemppinen, Segerberg, Hukkanen, Rees, Santavirta, Sorsa, Pertovaara, and Polak, 1994).

2.7.7 Prognosis

The unpredictable course of the symptoms and the persistence of disability arising from injury, often cause difficulties in the clinical management of a "whiplash" injury and an early identification of prognostic factors are considered essential in order to avoid development of chronic symptoms (Watkinson, Gargan and Bannister, 1991; Carette, 1994). The inability to establish an accurate account of the structures involved in the accident and the lack of an accurate diagnosis are the main factors usually blamed for the difficulty in establishing prognosis (Janes and Hooshmand, 1965; Bogduk, 1986b; Spitzer et al.,1995). Although a number of clinical features are considered to be indicators of poor prognosis, the following account will highlight the noticeable lack of agreement among the investigators on this issue (Frankel, 1959; MacNab, 1964; Hohl, 1974; Greenfield and Ilfield, 1977; Hohl and Hopp, 1978; Hohl, 1983; Balla, 1982; Norris and Watt, 1983; Pearce, 1989; Mills and Horne, 1986; Gore, Sepic, Gardner and Murray., 1987; Maimaris et al., 1988; Miles, Maimaris, Finlay and Barnes, 1988; Radanov, Stefano, Schnidrig and Ballinari, 1991; Radanov et al, 1992; Radanov, Sturzenegger, Di Stefano and Schidrig, 1994b; Radanov et al, 1995).

Frankel (1959) observed that those patients with severe "whiplash" symptoms requiring prolonged bed rest during the acute stage of the injury will have a slow recovery, and that an accurate evaluation of the prognosis cannot be made in less than 12-18 months. In contrast, a report based on a retrospective study has shown that when older "whiplash" patients having degenerative changes in their cervical spine and continued to have any

one of the following factors 2 months after their MVA are likely to develop chronic symptoms (Maimaris et al., 1988). Thus, the factors identified by Maimaris et al. (1988) include occipital headache, interscapular pain, referred pain and objectively demonstrable neurological signs. Although Hildingsson and Toolanen (1990) appear to disagree, the presence of radiating pain, numbness in one or both limbs, severity of pain during the initial stages have all been regarded as early indicators of poor prognosis (Hohl, 1974, Greenfield and Ilfeld, 1977; Hohl, 1983). However, Gore et al (1987) observed that the severity of the symptoms as evidenced during the initial examination alone had prognostic significance. While agreeing with Hohl's findings, Pearce (1989) and Maimaris et al., (1988) have shown that there is a significant correlation between the presence of nerve root signs during the initial examination, and protracted symptoms. In addition, presence of paraesthesia during the initial examination has also been shown to have prognostic value (Watkinson et al., 1991). A more recent report while supporting previous studies also showed that initial radicular symptoms, higher intensity of neck pain and headache experienced by "whiplash" patients during the first few days following their MVA were indicative poor prognosis (Radanov et al, 1995). It is considered that patients complaining of interscapular pain tend to recover at a slower rate when compared to others (Greenfield and Ilfeld, 1977; Maimaris et al., 1988). An epidemiological survey indicated that the neck symptoms arising from whiplash injuries are mere manifestation of pre-existing neck complaints, thus suggesting that those whiplash patients with pre-existing neck pain are predisposed to have a protracted recovery period (Bovim, Schrader and Sand, 1994). Similarly, it was shown that individuals having a history of headache prior to MVA are more likely develop a chronic headache following "whiplash" type of injury (Radanov et al., 1992; Radanov et al, 1995).

Although Deans et al. (1986) and Hildingsson and Toolanen (1990) are in disagreement with Hohl (1974), more recent studies have shown that the rate and speed of recovery are likely to be affected by the age and gender of the people involved (Radanov et al., 1995; Spitzer et al., 1995). Thus, elderly "whiplash" patients often complain of protracted symptoms and tend to develop late degenerative changes in their cervical spine (Hohl, 1974; Gore et al, 1987). While female patients are considered to recover slowly, male patients seem to recover at a significantly faster rate and it is suggested that this difference may be due to morphological differences in the musculature of the neck region and the ability of the generally larger males to withstand higher loads when compared to many slighter females (Bovim et al., 1994). Presence of multiple symptoms at the time of the initial examination is also considered one of the indicators of poor prognosis (Watkinson et al., 1991; Radanov, 1995).

A number of radiological features such as pre-existing degenerative changes in the cervical spine and loss of cervical lordosis as evidenced during routine initial radiological examination of the cervical spine carried out immediately after the MVA, are considered to be useful in establishing the prognosis. However, there are several conflicting reports in this regard, indicating that further investigation may be necessary in order to identify reliable radiological features which may assist in establishing a prognosis among the "whiplash" patients. Hohl (1974) and Norris and Watt (1983) agreed that reversal of lordosis of the cervical spine as seen in the initial radiological examination, and pre-existing spondylitic changes are one of the significant indicators of poor prognosis, although other studies indicated that reversal of lordosis is of no clinical significance (Greenfield and Ilfeld, 1977; Gore, Sepic, Gardner and Murray, 1987; Hildingsson and

Toolanen, 1990). In a follow-up study, Miles, Maimaris, Findlay, and Burrow (1988) and Radanov et al. (1995) evaluated the correlation between radiological findings and the presenting symptoms recorded at the time of the initial examination and compared them with subsequent follow-up examination at a two year interval. Their findings suggested that the only radiological feature associated with poor prognosis is the presence of degenerative changes in the cervical spine prior to "whiplash"; thus, confirming the findings of Hohl (1974) and Norris and Watt (1983). Although the observations made by Maimaris et al., (1988) supported the observations of previous authors, Pearce (1989) refuted their claims and stated that, "Those with preceding spondylosis, have no greater propensity for these symptoms than those without antecedent radiological spondylosis" (p.1330).

It is considered by many that psychological and behavioural factors are related to illness behaviour and that these factors may influence prognosis in post-traumatic conditions such as a "whiplash" injury (Balla, 1980; Balla, 1982; Mills and Horne, 1986). A prospective study of "whiplash" conducted by Radanov et al. (1991) showed that psychological factors, negative affectivity and personality traits are not significant in predicting the prognosis thus, refuted the conclusion of previous authors. However, the authors have shown that initial neck pain intensity, injury-related cognitive impairment and age are significant factors in predicting illness behaviour after "whiplash" type of injury.

A noteworthy report by Hohl and Hopp (1978) based on a prospective long term follow-up study ranging from five to twenty years post injury, appears to be in contradiction with the views expressed by various authors, including the previously reported findings of Hohl himself. The authors stated that,

"There was no statistical effect on recovery or further degeneration of the following: age, sex, type of accident, amount of damage, litigation, settlement time and amount, severity of initial symptoms, repeat accidents, radiating pain or numbness, limited neck motion, neurologic loss, and the forward held head" (p.29). However, the authors found that the presence of interscapular pain and the reversal of cervical lordosis on initial x-ray, are associated with incomplete recovery. Hildingsson and Toolanen (1990) reported that the incidence of low back pain was higher among their subjects affected with persistent "whiplash" symptoms and their findings supported previous observations of Gay and Abbott (1953) and Hohl (1974). These studies indicated that low back pain experienced by "whiplash" patients following MVA may have a prognostic value. Awerbuch (1992) compared the "whiplash" injury patients within Australia and overseas and came to the conclusion that most often the "whiplash" syndrome is an illness reinforced by the society and influenced by the lawyers involved. The potential financial gain to be made through litigation and the psychological state of the "whiplash" patient are considered by many to adversely affect the outcome of the treatment (Gay and Abbott, 1953; Gotten, 1956; Miller, 1961a; Miller, 1961b; Miller, 1966; Balla and Moraitis, 1970; Breck and Van Norman, 1971; Balla, 1980; Balla, 1982). Hodge (1971) suggested that traumatic neurosis and secondary gain in terms of social and emotional aspects may also influence the prognosis. However, it has been shown that there is no correlation between the rate of progress and litigation in other studies (Depalma and Subin, 1965; Janes and Hooshmand, 1965; Hohl, 1974, Mendelson, 1982; Maimaris et al, 1988; Pearce, 1989). An authoritative report by MacNab (1964, 1971) totally refuted the claim that "traumatic neurosis" played a major role in determining the rate of progress. MacNab (1964) also dismissed the suggestion that the symptoms arising from "extension-acceleration" injuries

of the neck are the result of "litigation neurosis", since his observations showed that 45% of the patients remained symptomatic two or more years after the settlement of their litigation.

Although, MacNab (1964) and Deans et al. (1986) considered that the occupants of the motor vehicles hit from behind are likely to develop chronic symptoms from their "whiplash" injury, recent reports indicated that there was no correlation between the rear-end collision and poor prognosis (Maimaris et al., 1988; Hildingsson and Toolanen, 1990; Spitzer et al., 1995). Similarly, Maimaris et al. (1988) showed that seating position of the "whiplash" patients in their motor vehicle and head restraints had no effect on the recovery of individuals affected by "whiplash" injury. However, the individuals head remaining in either rotated or inclined position at the time of impact was shown to adversely affect the prognosis (Radanov et al., 1995; Sturzenegger, Di Stefano, Radanov, and Schnidrig, 1995; Sturzenegger, Radanov, and Di Stefano, 1995). Evans (1992) observations indicated that the speed of the vehicle involved in MVA had no prognostic value.

2.8 Treatment

It is evident that there is neither a universally accepted "standard" treatment nor a consensus on the management of the "whiplash" syndrome. (Breck and Van Norman, 1971; Pennie and Agambar, 1990; Barnsley et al., 1994; Riley et al., 1995; Spitzer et al., 1995). In an attempt to highlight the difficulties in formulating treatment methods, duToit (1974) stated that, "The occasional brilliant cure of neck-ache by a single manipulation, a solitary injection or a simple one level fusion must surely prove that the cause may be organic if it can be localised with precision" (p.9). The difficulty in choosing appropriate treatment measures stems from the predicament of establishing an accurate diagnosis (Hohl, 1975; Bogduk, 1986b, Bogduk,

1995, Riley et al., 1995; Spitzer et al., 1995). It is obvious that a wide variety of treatment methods have been proposed and practiced in managing the "whiplash" injuries. It is also apparent that until recently there has been no attempt to adequately evaluate the conservative management of "whiplash". The following account will demonstrate the diversity in treatment methods used by various clinicians over the years and also the conflicting rationale among the authorities concerned.

2.8.1 A survey of treatment methods

In 1927, Crowe considered that in general, the less the treatment, the better it is (cited in Breck and Van Norman, 1971). Similarly, Gay and Abott (1953) advised that bed rest during the first 24-72 hours and sedation are an essential part of the clinical management during the day time and were of the opinion that constant use of the cervical collar resulted in a worsening of the symptoms and atrophy of the cervical muscles and thus proposed that the collar only be worn intermittently. Several authors recommended the use of cervical traction as an effective method of treatment to relieve pain and prevent restriction of movements in the cervical joints (Gay and Abott, 1953; Seletz, 1958; Knott and Barufaldi 1961; Hohl, 1983). Seletz (1958) emphasised that cervical traction should be used without delay to prevent formation of adhesions and scarring of capsular ligaments in the zygapophyseal joints of the cervical spine while Gay and Abott (1953) suggested that heat and cervical traction should be applied in order to relieve muscle spasm and radicular pain. Gay and Abott (1953) also suggested that during the acute phase, the cervical traction should be progressively applied for two hours followed by two hours rest, using seven pounds of weight. The authors also believed that at later stages, cervical traction may be applied several times a day until the duration of one hour at a time is reached. While agreeing with this recommendation, Knott and

Barufaldi (1961) suggested that the cervical traction should be continued for "weeks or months" in conjunction with isometric exercises of the post-vertebral muscles and followed by relaxation techniques. While recommending that the cervical traction is by far the most effective conservative therapeutic measure to treat "whiplash" injury, Wiggins (cited in Breck and Van Norman, 1971) appears to contradict himself by recommending that an early surgical intervention (see below) may be beneficial to the "whiplash" patient. Although Hohl (1983) was in disagreement with regard to the application of cervical traction during the acute stage of "whiplash" injury, considered that traction may be appropriate after fibrous healing of soft tissues in order to restore mobility in the cervical joints.

A major report by Janes and Hooshmand (1965) is based on one of the largest and best documented series, consisting of 10,000 "whiplash" patients, who were investigated between 1956-63. They showed that 80% of their patients improved within the first year after the injury. Their recommendation is that the neck initially be immobilised in a Peterson collar for a period of six weeks with the periodical application of heat and muscle relaxants, while occasional injection of steroids into trigger points is also suggested. In reporting the efficacy of surgical procedures in 26 of their chronic patients, Jane and Hooshmand (1965) indicated that surgery was only performed as a last resort and approximately 28.3 months after the injury, whereas Wiggins (cited in Breck and Van Norman, 1971) believed in early surgical intervention and recommended that a cervico-brachial decompression procedure produced the best result. It is interesting to note that as a preoperative evaluation, Jane and Hooshmand (1965) immobilised their patients in a Peterson collar for 4-6 weeks and

noted that significant pain relief was reported, while their necks were immobilised and became symptomatic as soon as the collar was removed.

duToit (1974) considered that the "whiplash" patients should be given uninterrupted bed rest and clearly specified that the patients should lie flat on their back with one pillow under the head, until the acute symptoms subsided. He also recommended the administration of tranquillisers, anaesthetic blocks and the application of ice packs and cervical traction as an integral part of the early treatment regimen. He believed that congestion of the extensive venous plexus in the cervical spine may be responsible for the headache experienced by the "whiplash" patients and suggested that the subsequent headache could be relieved by the application of pressure to the back of the neck. du Toit (1974) also suggested that isometric exercises of the neck muscles should be commenced once the muscle tenderness has settled.

In a contrary view, Paige (1978) considered that passive mobilisation of the spine with active exercises to promote movements, massage directed towards the tightened soft tissue structures and a progressive exercise programme should be part of the clinical management of "whiplash" injuries. However, Sprague (1983) considered that it was inappropriate to apply either spinal mobilisation or manipulation procedures to treat "whiplash" injury.

Hohl (1975) suggested that the patients suffering from moderate to severe symptoms, required bed rest and sedation for a few days. He considered that heat packs and hot showers are more beneficial when compared to the application of either ultrasound or short-wave diathermy, which he considered prolonged the duration of the symptoms. Hohl (1975) believed

in isometric exercises and advocated an early return to activity and work in order to avoid injury induced anxiety and emotional instability. He also considered that there is no rationale for subjecting "whiplash" patients to cervical traction especially during the early stages (Hohl,1975; Hohl, 1983). He regarded discectomy and anterior spinal fusion as an option during the chronic stage of "whiplash" a few years after the injury.

In contrast, Porter (1989) preferred the prescription of non-steroid anti-inflammatory drugs and the use of a soft collar during the acute stage followed by physiotherapy, including isometric exercises of the post vertebral muscles.

Although the clinical management of the most common symptoms associated with "whiplash" injury have received considerable interest, there is little information on the treatment of symptoms such as retro-orbital pain, vertigo and some of the subjective symptoms presented by "whiplash" patients. There is a single report which claimed that the retro-orbital pain can be successfully treated by the application of short-wave diathermy and cervical traction (Harwich and Kasner, 1962). Similarly, it has also been shown that vertigo induced by over-excitation of the cervical proprioceptors in a "whiplash" patient, can be treated by infiltration of the deep neck muscles with local anaesthetics (Hinoki, 1985). It has been demonstrated that an increase in muscle tension, stress and fatigue reported by "whiplash" patients are secondary symptoms due to a peripheral vestibular deficit and it is recommended that these symptoms may be treated by a rehabilitation programme described by Shumway-Cook and Horak (1990), consisting of exercises in order to complement the natural compensatory mechanism of the central nervous system. Hinoki (1985) also observed that some of the subjective symptoms reported by the patients, as a result of

brain stem disturbance, disappeared by wearing a cervical collar while Janecki and Lipke (1978) cautioned that use of cervical collar over a long period may lead to disuse atrophy of the neck muscles. However, there is little information to support this widespread opinion by objectively measuring the changes in the strength of neck muscles (Highland, Dreinger, Vie, and Russell, 1992; Vernon, Aker, Aramenko, Battershill, Alepin and Penner, 1992). Furthermore, the cervical collar has been shown to be ineffective in restricting neck movements and hence the adverse effect attributed to the long term use of soft collar requires verification (Fisher, Bowar, Awad and Gullickson, 1977; Johnson, Hart, Simmons, Ramsby and Southwick, 1977; Johnson, Owen, Hart, and Callahan, 1981).

It is also apparent that in recent clinical trials a variety of methods have been used for documenting range of motion of the cervical spine. For instance, Mealy et al. (1986), McKinney et al. (1989) and Pennie and Agambar (1990) reported to have used goniometers to measure the cervical movements whose reliability and validity are not known. Similarly, Foley-Nolan, Moore, Codd, Barry, O'connor and Coughlan (1992) reported that in their study, the cervical range of movements were estimated and graded into four categories. Owing to these differences in their methods, it may not be possible to make considered comparison of the results of these studies. However, these authors have documented pain reported by their subjects, using visual analogue scale (VAS). The VAS procedure has been shown to be a reliable procedure and widely used for documenting pain (Chapman, Casey, Dubner, Foley, Gracely and Reading, 1985; Jensen, Karoly and Braver, 1986).

Even though there has been a change in recent years in the management of the "whiplash" injuries, it is apparent that treatment still remains non-

specific and often lacks a coherent rationale (Bogduk 1986b; Newman, 1990; Bogduk, 1995; Riley et al., 1995; Spitzer, et al., 1995). It is interesting to note that the duration for which it is suggested that a soft collar should be worn, varies between from minimum period of 2 weeks to up to one year (Gay and Abbott, 1953; Martin, 1959; Janes and Hooshmand, 1965; MacNab, 1964; Farbman, 1973; Hohl, 1975; Marshall, 1976; Balla, 1980; Norris and Watt, 1983; Mealy et al., 1986; McKinney et al., 1989). Similarly, the application of cervical traction remains a matter of controversy (Hohl, 1975; Hohl, 1983). In general the early management of the "whiplash" injury is based on a conservative approach to the problem and until recently no attempt has been made to evaluate the efficacies of the various conservative treatment methods typically used. However, a few notable studies have been conducted in more recent times and are considered below.

2.8.2 Clinical trials

A randomised single blind clinical trial conducted over a period of three months, compared the outcome of two conservative treatment methods (Mealy et al., 1986). One group of "whiplash" subjects were initially given a soft collar and advised to rest for 2 weeks, before commencing gradual mobilising exercises (Group 1). The second group of subjects received the application of ice in the first twenty four hours and after this period, passive mobilisation techniques as described by Maitland (1986) and an exercise programme was carried out. Local heat was applied after each treatment. The subjects were required to do a gentle home exercise programme every hour, within the limits of pain. The follow-up examinations were carried at 4 weeks and 8 weeks after the injury. The observations made by Mealy et al., (1986) indicated that the group 2 patients who were treated actively showed a significant reduction in pain, when compared to the group 1 who were

rested in a soft collar. Similarly, the range of motion in the cervical spine of the group treated actively, is significantly greater at 4 weeks and 8 weeks intervals, when compared to those treated by initial rest in soft collar. The authors concluded that early immobilisation is likely to result in persistent and prolonged pain and stiffness in the neck and advocated the early judicious use of mobilising exercises and activities in the treatment of "whiplash".

Pennie and Agambar (1990) considered that the active treatment regime advocated by Mealy et al. (1986) has not been adequately defined. In order to evaluate the efficacy of active treatment, Pennie and Agambar (1990) set up a clearly defined active treatment regime consisting of cervical traction, self care advice and a neck and shoulder home exercise programme. All patients in this group received treatment in the form of intermittent traction, twice a week for 10 minutes duration using a load of 5.4 kgs. The outcome of the group treated in this manner was compared with a second group of patients initially treated for a period of 2 weeks of rest in a soft collar or a moulded thermoplastic foam collar, followed by instruction as to an active exercise programme. In this way, 152 patients were followed-up over a period of 5 months post-injury. A comparison of the results at 6-8 weeks and at 5 months has shown that there was no significant difference between the outcome of the two groups

In a clinical trial conducted by McKinney et al. (1989), the subjects were randomised into three groups to receive the following treatment :

Group 1. Rest during the first 10-14 days followed by general advice about mobilisation.

Group 2. An "active physiotherapy" regimen.

Group.3 Received instruction in self-care management.

The "active physiotherapy" (Group.2) consisted of a combination of hot and cold applications, short-wave diathermy, hydrotherapy, active and passive repetitive movements using the principles of Maitland (1986) and McKenzie and were given three treatment sessions a week, of up to forty minutes a session. These patients were also instructed in postural awareness and required to do a home exercises programme. The subjects in Group.3 were provided with a verbal and reinforcing written instruction on postural correction, and instructions on the application of heat and muscle relaxation techniques. A demonstration of mobilising exercises was also given and the patients were required to undertake the exercises at home. When the subjects were examined at 4, 8, and 12 week intervals, it was observed that there was no significant difference in the outcome between the group-2 and group-3 subjects and that the subjects in group-1 significantly lagged behind other two groups. In conclusion McKinney et al. (1989) stated that, "....good advice and tailored practical instruction on early mobilisation, when given by a suitably experienced physiotherapist, is as effective as out-patient physiotherapy in reducing pain and increasing mobility...." (p.32). As a long term follow-up, 2 two years after the initial examination, all the subjects were requested to respond to a questionnaire. Based on the response of 167 subjects out of the original 247 subjects, McKinney (1989) observed that there was no significant difference in the percentage of symptomatic patients between group 1 and group 2, (46% and 44% respectively), whereas 23% subjects from group 3 still remained symptomatic. Therefore, McKinney (1989) concluded that in the long term, an early mobilisation programme is more likely to reduce the incidence of protracted symptoms and also recommended that soft collar should only be used for brief periods.

In a recent study, the application of pulsed low energy high frequency pulsed electromagnetic therapy (PEMT) was investigated (Foley-Nolan et al.,1992). In this double blind study, forty subjects were monitored for a period of three months in which the experimental group was subjected to electromagnetic therapy for eight hours a day for twelve weeks, whereas the control group was given a dummy electromagnetic therapy unit for a similar duration. Both group of subjects also received advise on a defined active mobilising exercise regimen. Those subjects deemed to be "unhappy with their progress" were allowed to receive a variety of physiotherapy modalities, twice weekly for six weeks. The patients were asked to document the level of pain using a visual analogue scale. The quantity of analgesic taken was also taken into account in assessing the pain level. In order to document the ROM, the patients used a predetermined grading system viz "full range", "two thirds normal" and "one third normal" and as a result of this the progress has been reported in a categorical manner viz "better", " much better" etc. The follow-up examinations were conducted at 2, 4, and 12 weeks and at the end of the study period (i.e 12 weeks), 85% of the subjects in the experimental group and 60% subjects from the control group were reported to be either "moderately better" or "much better" categories. The investigators concluded that PEMT is effective in treating acute and persistent neck ache. However, it appears that the statistical analysis has not taken into account of the effect of the compounding variables, therefore the validity of the conclusion drawn from this study is of questionable value.

It was evident that it may not be possible to verify some of the observations reported in these clinical trials. For instance, there were differences in the types of goniometers used by Mealy et al. (1986), McKinney et al., (1989) and Pennie and Agambar (1990), to measure the cervical range of motion.

In addition, the validity and reliability of these goniometers were not reported. Similarly, Foley-Nolan, Moore, Codd, Barry, O'connor and Coughlan (1992) reported that in their study, the cervical range of movements were estimated and graded into four categories. However, these authors have documented pain reported by their subjects, using visual analogue scale (VAS) (Chapman et al., 1985; Jensen, Karoly and Braver, 1986). Furthermore, there is little information available on the assessment of disability associated with "whiplash" injury, using a validated disability index similar to the one used to measure disabilities arising from low back pain (e.g Low Back Pain Index) (Vernon and Moir, 1991).

2.9 Consensus of opinions

The complexity of the "whiplash" type of injury and its adverse socio-economical impact has recently led to the establishment in Canada of a task force to analyse various aspects of the problems associated with this syndrome and to come up with clear directions for future research (Spitzer et al., 1995). This important document recently published by The Quebec Task Force on Whiplash-Associated Disorders is expected to have a considerable impact on the assessment and treatment of the "whiplash" syndrome. The Task Force performed an extensive critical review of the existing literature in the field and determined that only 294 publications out of 10,382 identified in the field were of scientific merit required consideration. Based on the information contained in the selected publications of merit and the derived "consensus findings", the panel of experts have made a number of recommendations to both clinicians and researchers in "whiplash". The Task Force considered that there is a compelling need to clearly define the syndrome and promoted a definition which included information concerning the mechanism of the injury, its nature and also acknowledged the wide variety of clinical presentations. In

addition, it was proposed that the term "Whiplash-Associated Disorders" (WAD) be routinely used to refer to this syndrome and compiled a list of classification of WAD which would readily convey the extent of the severity of the symptoms. In defining the WAD, the Task Force has also included neck symptoms arising from accidents such as "....diving or other mishaps..." (page.22S) as well as M.V.A. Therefore, it is open to question as to whether or not the Task Force has met its original mandate with their primary definition of what constitutes a "whiplash" injury (see Discussion).

A retrospective survey commissioned by the Task Force reported that the severity of the injury was positively correlated to the following factors viz:

1. female gender;
2. elderly people;
3. forces associated with the impact; and
4. failure to use an appropriate seat belt.

It is interesting to note that the rear-end type of collision appeared to cause less serious injury when compared to other forms of motor vehicle collisions.

The Task Force acknowledged the existence of the some of the bizarre symptoms often reported by WAD patients and agreed that these so called subjective symptoms should be taken into account at all stages of clinical management of the "whiplash" patients. Individuals with symptoms persisting for any longer than six months post-injury were classified as "chronic" patients and it was suggested that a history of residual disability and persisting symptoms after forty five days post injury are indicators of poor prognosis. It has been strongly recommended that such patients

considered to be in the risk category, should undergo a thorough investigation.

Commenting on the range of treatments administered, the Task Force was of the opinion that the use of soft collar and the concept of prescribing a period of rest have not been adequately verified and cautioned that such practices may inhibit the rate of recovery of "whiplash" patients. While they were critical of several previous studies it is matter of interest to note that the Task Force has stated that, "Based on limited evidence and reasoning by analogy, it is the Task Force consensus that the use of non-steroidal anti-inflammatory agents and analgesics, short-term manipulation and mobilisation by trained persons, and active exercises are useful in grade II and Grade III WAD...." (page 36S). It could be argued that by endorsing the use of spinal manipulation, the Task Force has committed a serious error of judgement. Noting from the citation of the literatures in their report, it appears that the Task Force has relied on the conclusions drawn from the clinical trial conducted by Cassidy, Lopes and Yong-Hing (1992) in which the efficacy of spinal manipulation was evaluated. According to the classification proposed by the Task Force, Grade II (presence of neck complaint and musculoskeletal signs viz decreased ROM and point of tenderness) and Grade III (presence of neck complaints and neurological signs viz decreased or absent deep tendon reflexes, weakness and sensory deficits) may be the result of severe soft tissue injury, IVD lesion and significant root lesion. In this situation a manipulative procedure described by Cassidy et al. (1992) as a, " high velocity, low amplitude thrust directed beyond the passive range of motion of the spine and associated with an audible crack caused by cavitation of the underlying facet joint" (page 571) is difficult to justify.

Secondly, the Task Force has clearly demonstrated a breach in their analysis of the literature based on their concept viz "results of Best Evidence Synthesis". Even though Cassidy, Lopes, and Yong-Hing (1993) concluded that a single manipulation was more effective than mobilisation, resulting in a reduction of neck pain, they subsequently admitted that the statistical analysis used to derive the results was inappropriate (Cassidy et al., 1993).

Thirdly, the subjects included in their study were reported to have been, ".....suffering from mechanical neck pain with radiation into the trapezius muscle" (page 571) which was directly not related to WAD. Therefore the expressed opinion by the Task Force regarding the use of spinal manipulation for the relief of pain and promoting early mobility in WAD should be treated with considerable caution.

Nevertheless, the Task Force has prepared an important and potentially influential document and has made several valid recommendations regarding:

1. the initial examination;
2. investigations;
3. clinical management;
4. follow-up examinations; and
5. a well defined protocol for researchers.

This will be considered later in the current thesis (see Discussion).

2.10 Summary

It is evident from the review of the literature that the "whiplash" syndrome is not yet fully understood and despite on-going research continues to present multifaceted challenges. The extent and magnitude of "whiplash" injury

remains to be verified and confirmed in order to improve the understanding of the pathologies of "whiplash". The clinical management of the effects of trauma to the neck still largely remains arbitrary, nonspecific, lacking in physiological rationale and often involves a protracted period of treatment. The pressing need to identify appropriate treatment regimen has been reiterated by several authorities and this has been addressed in this study.

CHAPTER THREE METHODOLOGY

3.1 Introduction

The principle aim of this project was to conduct a comprehensive randomised clinical trial, in order to evaluate the outcome of early immobilisation of the neck as opposed to other methods of treatment instituted to patients suffering from the effects of soft tissue injury to the neck, arising from motor vehicle accidents. The efficacy of a chosen treatment method will be generally determined by the time taken to achieve:

a. an acceptable (to the patient) reduction of pain;

b. the restoration of pain free full range of motion in respective joints;

c. full "normal" motor power; and

d. the ability of an individual to perform various activities of daily living, social and work related activities at a comparable level prior to the injury.

In accordance with this general concept, it was decided to measure:

a. the amount of pain as reported by the subjects;

b the range of motion in the cervical spine and its relationship to pain;

c the strength and the endurance of the pre and post vertebral neck muscles; and

d. the extent of incapacity to perform activities of daily living including social and work related activities.

3.2 Clinical trial

An approval of the ethics committee of each of Royal Perth Hospital and Curtin University of Technology was obtained prior to conducting the proposed clinical trial (Appendix 4).

3.2.1 Tenents of code

The following tenents of code of conduct were observed throughout the trial:

a. informed consent was obtained from each patient after they had been provided with the Patient Information Sheet;
b. every patient included in the clinical trial received treatment;
c. patient confidentiality was maintained at all times;
d. subjects were allowed to withdraw consent to proceed with the clinical trial at any time without prejudice to on-going clinical management;
e. methods used for the collection of data such as pain, range of motion, muscle strength, were safe and could not affect the subject's health nor clinical status;
f. all the subjects entered into the trial were under the care of an orthopaedic surgeon specialising in spinal injuries; and
g. the clinical trial was in accordance with the statement on Clinical Trials produced by the National Health & Medical Research Council of Australia.

3.2.2 Subjects

The sample size consisted of 220 subjects comprising of 145 females and 75 males who were residing in Perth metropolitan area and surrounding suburbs.

3.2.3 Inclusion criteria

Criteria for inclusion were patients of both genders involved in motor vehicle accidents with neck pain, who:

a presented to the Royal Perth Hospital Accident and Emergency Department and were subsequently discharged for follow-up; and

b were referred by general practitioners.

A retrospective survey of the records from Royal Perth Hospital accident and emergency department pertaining to the period between January, 1989 - June, 1989, was conducted in order to establish the number, gender and age of patients treated for "whiplash" type of injuries. It was determined from this survey that it would be impractical to recruit the required number of subjects according to specific gender and age characteristics within a time scale of two years. Hence, the selection of subjects was not based on either gender or age. During the period between August 1990 and June 1993, two hundred and forty five patients were referred from Royal Perth Hospital and of these 28 declined to participate in the clinical trial. During the same period three patients were referred by general practitioners.

3.2.4 Exclusion criteria

Criteria for exclusion were:

a. lack of informed consent from the patient;

b. patients with bony injury as seen on standard x-ray;

c. a history of previous neck injury within a 12 month period which required treatment;

d. those patients who have had previous direct exposure to workers compensation; and

e. patients not presenting for initial examination within 60 hours after the injury.

3.2.5 Withdrawal criteria

Criteria for withdrawal were when:

a. subjects withdrew their consent. This was without any prejudice to their on going treatment; and

b. subjects demonstrated non-compliance with the immobilisation regime and / or breach of randomisation by opting to select other forms of treatment.

3.2.6 Randomisation

All subjects were informed about the purpose of the clinical trial and then requested to read the Patient Information and Consent form (Appendix-4). Those subjects who had difficulty in understanding the contents of the information and consent form were assisted by their accompanying person. Prior to signing the consent form, each subject was given an opportunity to question the Investigator for further clarification. Subjects were randomly assigned into one of the three treatment groups by ballot.

3.2.7 Trial groups

Immobilised group:

The subjects were individually fitted with a Philadelphia collar (by a trained orthotist, at Royal Perth (Rehabilitation) Hospital, Shenton Park), for a period of 4 weeks and this was followed by a defined active physiotherapy regime (details in page 118).

Active exercise group:

The treatment consisted of application of local heat to neck region followed by a defined active physiotherapy regimen (details in page 120).

Control group: The subjects were returned to the care of their family doctors to receive treatment as directed by him / her.

3.3 Trial scheme

During the trial, data was collected in an initial examination and then in a series of subsequent examinations performed at 4 weeks, 6 weeks, 12 weeks, 24 weeks, and 52 weeks post-injury. The trial scheme is outlined in Figure 3.1.

3.4 Initial examination

At the commencement of the examination the investigator briefly explained the purpose of the examination in order to put the subjects at ease. A questionnaire with numbered subject identification was used for recording the subjective and objective findings and this was administered by the Investigator (Appendix-4). The initial examination of subjects (for the purpose of data collection) was carried out by the investigator, and this was followed by a medical examination from a spinal surgeon or his registrar. This dual procedure complied with medico-legal requirements and in addition enabled comparison of objective findings from the neurological portion of the examination.

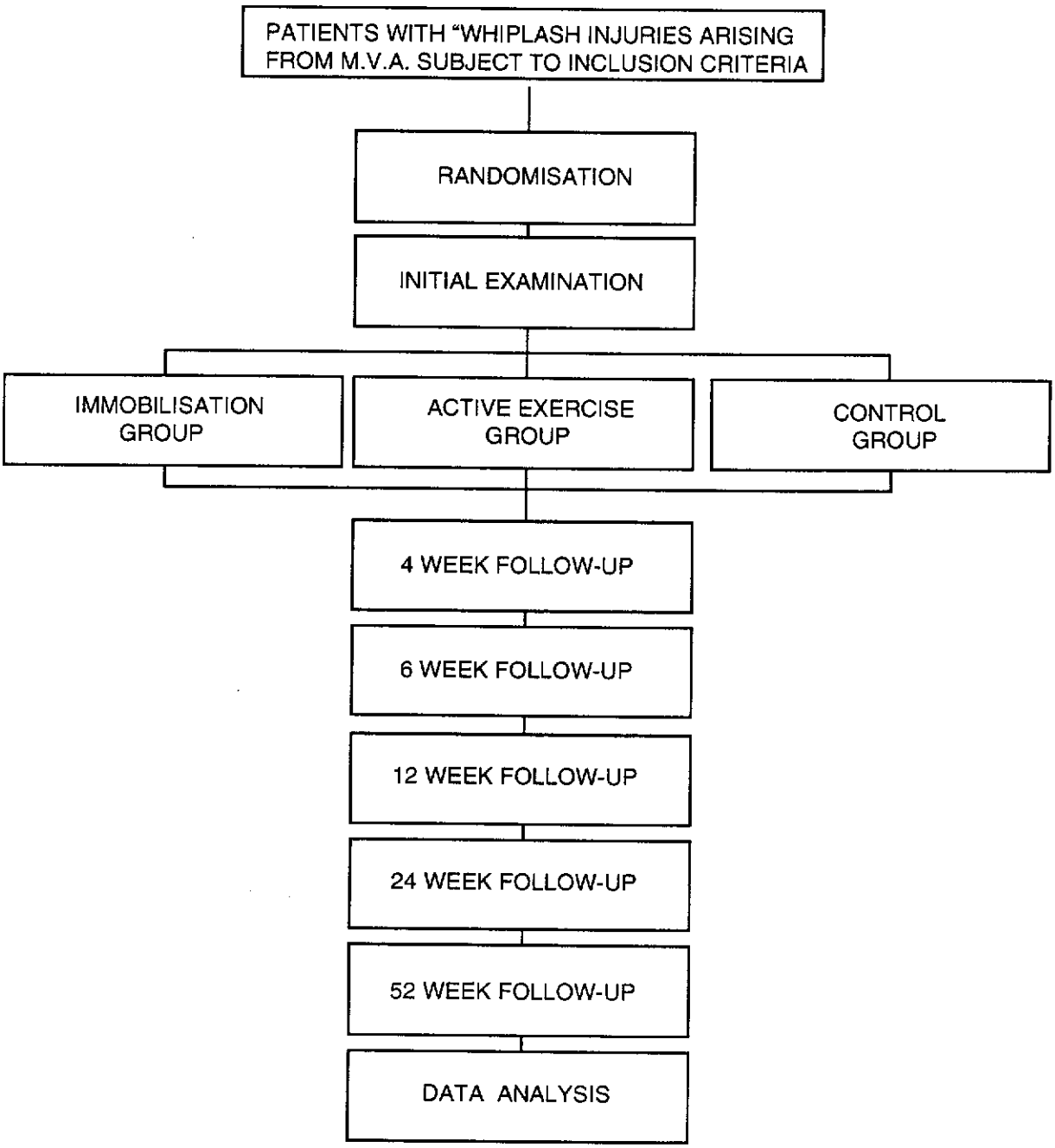

Figure 3.1 Flow chart showing clinical trial outline.

The initial examination comprised of components as described below, namely:

1. subjective examination; and
2. objective examination (physical examination).

Subjective examination comprised of the following:

1. demographic details;
2. information related to MVA.;
3 social history;
4. documentation pain including the area, intensity and description;
5. paraesthesia,
6. symptoms collectively known as "Barre'-Lieou syndrome";
7. information related to employment / home duties and activities of daily living;
8. medication taken; and
9. details of previous neck injury / neck symptoms.

Objective examination comprised of the following:

1. documentation of postural abnormality;
2. documentation of morphological features of the neck;
3 assessment of range of motion of the cervical spine and associated pain level;.
4. neurological examination to establish alteration in motor power, cutaneous sensation, vibration sensation, tendon reflexes, Horner's syndrome, proprioception and weakness of the recti muscles in the eye; and

5. soft tissue palpation.

3.4.1 Subjective examination

Information related to occupation, main hobbies, make, model and speed of the vehicles involved in the accident (as per the statement made to the police by the subjects); including where the subject was seated, presence and effectiveness of safety restraints, were obtained and documented. The type of collision was determined and recorded. The momentum of forces involved was estimated from the reported speed, direction, and size of the vehicles involved. The subject's report of awareness and possible reaction to the imminent accident was noted. The symptoms to be associated with neurological implications documented.

Pain and paraesthesia:

A body chart with coding specific to body region and structure was used to record the distribution, description, intensity and frequency of subject's pain and paraesthesia. Subjects were requested to describe any areas of pain and paraesthesia separately. Prior to commencement of this study a list of descriptors of type of pain and frequency of pain were compiled by the investigator following examination of "whiplash" patients presenting for treatment and discussion with a series of registered physiotherapists and medical practitioners. The descriptors thus obtained were trailed on 20 subjects and found to be adequate to enable subjects to describe their type and frequency of pain. In addition a numerical rating scale (NRS) was used to document the intensity of pain as perceived by the subjects. This system of pain measurement has been shown to be valid, reliable and convenient to administer either in written or verbal form (Jensen et al., 1986).

Each subject was given clear, simple instructions regarding the use of numerical rating scale and the following written instruction was repeated on each occasion, prior to the assessment of pain.

> "0 means no pain at all - 100 is equivalent to the severest pain you can imagine. Could you pick a number between 0-100 so that I can understand the severity of your pain."

It was emphasised to the subjects that they should choose a number which represented the intensity of pain most usually experienced by them.

The subjects were asked to indicate the painful areas, one at a time, using their index finger and draw a line over the body region. This information was recorded on the body chart using relevant code. This was followed by documentation of intensity (using NRS), nature and frequency of pain for that particular area of pain. Thus, the distribution, intensity, nature and frequency of the pain experienced by the subjects within 24 hours following the MVA and at the time of the initial examination were documented separately.

The distribution of paraesthesia experienced by the subjects following the MVA was obtained by asking them to indicate the body regions affected using their index finger and recorded on the body chart. This information was documented using specific codes which represented the relevant dermatome. The frequency of this symptom was also documented.

Activities of daily living (ADL):

Prior to commencement of this study, a list of ADL which were considered to aggravate pain related symptoms was compiled by the investigator

following examination of "whiplash" patients presenting for treatment. This list of ADL was trialed on 20 subjects and found to be comprehensive as a questionnaire for documenting ADL which were considered to cause discomfort by the subjects in the current study. Thus, the subjects were asked to choose from the questionnaire those activities which caused them discomfort.

Resumption of normal duties equivalent to pre-accident level:

In the case of those subjects who were employed, the normal number of hours worked per week prior to MVA was documented and used to monitor a subject's progress. In the event of subjects returning to work after a period of sick leave following their MVA, the time of return to work was recorded in weeks with reference to post-injury. Similarly, the number of hours worked after returning to their jobs was obtained and recorded as a percentage of that subject's normal working hours. In addition, it was also documented whether subjects were working in their usual job or whether work modifications had been instituted in order to minimise the severity of the symptoms. This information enabled a point in time to be used as identification for when subjects resumed work at a level equivalent to their pre-accident level.

In the case of female subjects who were not employed outside the home, their usual home duties were used to determine their rate of progress. This was estimated using various activities related to duties performed at home and expressed by the subjects in terms of percentage. Thus, a particular point in time of the study period at which the female subjects resumed home duties at a level equivalent to the pre-accident level (i.e 100%) was recorded.

Medication and previous neck injury:

Subject's medication such as anti-inflammatory preparation, analgesics and muscle relaxants was documented. Similarly details of previous neck injury was also recorded.

3.4.2 Objective examination

Prior to the objective examination, all subjects were adequately undressed. The presence of antalgic patterns in the neck, postural features such as increased lordosis, poked chin and bull neck deformities, morphological features viz thin neck, long neck, short neck and stout neck were identified and recorded.

Device used to measure range of motion of the cervical spine:

A Cervical Range of Motion goniometer (CROM) was used to measure active and passive range of flexion, extension, rotation and side flexion, of the cervical spine (Figures 3.2 and 3.3). This instrument consisted of two gravity assisted meters and a compass. The gravity assisted meter situated on the left side of the CROM (sagittal goniometer) measured flexion and extension of the cervical spine while the gravity assisted meter present in the front of the CROM (frontal goniometer) measured side flexion. Similarly, a small compass present in the CROM was used in conjunction with a magnetic yoke to document rotation of the cervical spine. A pilot study was carried out to verify the intratester reliability for the measurements obtained using the CROM and relevant details are reported in Appendix-I.

Figure 3.2 Cervical range of motion goniometer - side view.

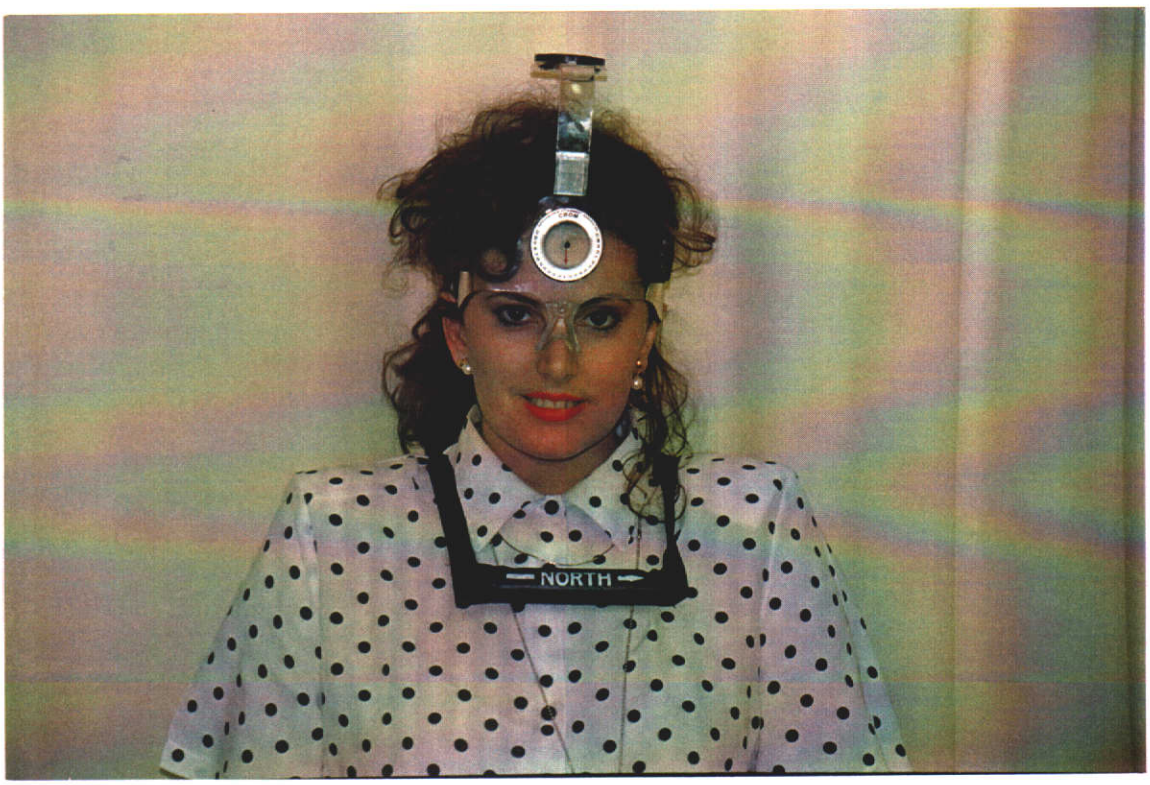

Figure 3.3 Cervical range of motion goniometer - front view.

Position of subjects:

A non-metallic straight back chair with arm rests was placed in such a way that the subjects sat facing in either a westerly or easterly direction, with the frontal plane of the body parallel to northerly / southerly direction. The subjects were seated (without leaning against the chair's back rest) in an erect posture, with their fore-arms resting on the arm rests and feet flat on the ground. The CROM was positioned on the forehead of the subjects such as when putting on a pair of reading glasses and secured with velcro straps. The magnetic yoke was placed on the subjects shoulders with the arrow pointing north. The dial of the compass was recalibrated, so that the figure "0" was in line with the tip of the magnetic needle.

Assessment of range of motion and associated pain:

A demonstration of each of the active movements was give to each subject highlighting the need for them to maintain a satisfactory posture during testing of various range of motion of the cervical spine. The numerical rating pain scale standard instruction was repeated prior to the examination.

Extension:

The investigator stood on the left side of the subjects and reminded them not to slump or move the body while testing. The investigator placed his right palm at the interscapular region and applied gentle pressure in order to stop the trunk from moving backward. The subjects were asked to nod their head backward and then continue to take it as far back as possible into extension. As soon as the subjects came to a halt the investigator took a reading from the sagittal meter. After returning their head to the neutral position, the investigator asked a standard question, "What stopped you

from moving any further?" If the subjects mentioned pain or discomfort being responsible for the limitation of the movement, the area of the pain was identified. On occasions where two or more areas were mentioned, the area reported to be the most painful was recorded. The description and the level of pain as perceived by the subjects were recorded. Passive extension of the cervical spine was carried out by repeating the procedure as mentioned for active ROM of extension and applying a gentle pressure over the forehead by the left hand of the investigator at the end of the available range. Whenever the pain was considered to be the limiting factor, the area, description and the level of pain were recorded.

Flexion:

The investigator gently restrained the trunk from moving forward, by applying the left palm over the upper part of the sternum, taking care not to limit the forward flexion of the cervical spine. To assume full flexion in the cervical spine, the subjects were first instructed to nod their head to make a double chin and then encouraged to move their head, "as in touching the upper chest with the chin". As soon as the subjects reached the end of the available range, the sagittal goniometer was read and a standard question," What stopped you from moving any further?" was asked. At this stage the subjects were asked to take their head back to the neutral position. If the limiting factor was attributed to either pain or discomfort, the relevant details, as described in the previous section, were recorded. This was followed by the recording of passive ROM of flexion. This was performed by repeating the procedure as described for active ROM of flexion and after reaching the end of range, a gentle pressure was applied over the occiput by the right hand of the investigator.

Rotation:

The subjects were asked to focus their eyes on to a horizontal line on the wall so that the head was not tipped laterally during rotation. Prior to the commencement of the rotation, the sagittal and the frontal goniometers were checked to make sure that the cervical spine was kept in a neutral position. In order to ensure that no shoulder rotation occurred, the left shoulder was lightly stabilised from the front, while the subjects turned their faces to the right side and vice versa. During this procedure, the subjects were constantly reminded to focus their eyes on the horizontal line on the wall. The active range of motion of rotation recorded by the compass and associated pain were documented as described in the previous section. The passive range of motion of rotation recording was performed by repeating the procedure as described for active rotation and after reaching the end range, a gentle pressure was applied over the ipsi-lateral side of the occiput and contra lateral side of the face. During this procedure the subjects were reminded to sit upright and not to move their bodies. The passive range of motion of rotation recorded by the compass and associated pain were documented as described in the previous section.

Lateral flexion:

The posture of the head was corrected so that the sagittal and frontal meters were remained at zero. A standard instruction, "Bend your neck to the (right / left) side as far as you can and take care not to move the body". During this procedure the investigator prevented the shoulders from being raised or lowered, by gently touching the respective shoulder. As soon as the movement was completed the investigator recorded the reading from the frontal meter. The active range of motion of lateral flexion and associated pain were documented as described in the previous section. The passive side flexion was performed by repeating the procedure as described for

active ROM and after reaching the end range, a gentle pressure was applied over the contra lateral side of the face. During this procedure the subjects were constantly reminded to sit upright and not to move their bodies. The passive range of motion of lateral flexion and associated pain were documented as described in the previous section.

Motor power:

The muscles of the left and right upper limb and shoulder girdle supplied by cervical 4th to 1st thoracic anterior rami were examined in accordance with the Medical Research Council's grading system.

The following muscles were examined:

Muscle	**Nerve Root**
Deltoid	C4 & 5
Biceps Brachii	C5 & 6
Triceps	C7 & 8
Long flexors of fingers & thumb	C8
Intrinsic muscles	T1

Cutaneous sensation:

The subjects were tested for changes in light touch, sharp and blunt sensation. Thermal sensation and hyperaesthesia were additionally checked when there was alteration in any of the other sensibilities. The 4th to 8th cervical and 1st thoracic dermatomes were also tested and documented either normal or altered using specific codes. The following procedures were used to test the cutaneous sensation of left and right side of the subjects.

Light touch:

A demonstration of the testing procedure was given by stroking the skin over the interscapular region and the face. A standard instruction was repeated at the commencement of the examination as follows, "Please say 'Yes' if you can feel the cotton wool. If you thought it felt different say 'Stop". Prior to testing, subjects were asked to close their eyes and the cotton wool was stroked at random, over the dermatomes supplied by the cervical roots 4, 5, 6, 7, 8 and the 1st thoracic root. If the patient showed any sign of hesitancy in responding to the light touch or said "Stop", the examination was repeated over that area at random, till the finding was confirmed and recorded as normal or altered.

Sharp / blunt sensation:

A sterile needle and the end of a paper clip were used to test sharp / blunt sensations. A demonstration was carried out by touching the skin over the interscapular region and the subjects were asked to say, " Sharp" or "Blunt", to establish that they were able to differentiate between the sensibilities. At the time of the testing, subjects remained eyes closed and the investigator randomly touched the skin either with the needle or the blunt end of the paper clip. Thus, the dermatomes supplied by the cervical roots 4, 5, 6, 7, 8, and the 1st thoracic root were tested and the findings were recorded as normal or altered.

Thermal sensation:

Test tubes filled with hot and cold water were used to test hot / cold sensation. A demonstration was carried by randomly placing the test tubes on the skin over the interscapular region and the subjects were asked to say, "Hot" or "Cold" in order to establish that they were able to differentiate between the two. During the testing the subjects remained with their eyes

closed and the investigator randomly placed the test tubes over the dermatomes supplied by the cervical roots 4, 5, 6, 7, 8 and the 1st thoracic root and the findings were recorded as normal or altered.

Hyperaesthesia:

The body segment which was reported to be hypersensitive during the subjective examination was tested by gently striking with a linen cloth while the subjects remained with their eyes closed. This procedure was repeated randomly and only the abnormal finding was recorded.

Vibration sensation:

A tuning fork with a frequency of 256 cycles per second was used to test vibration sensation. A demonstration was carried out by placing the vibrating tuning fork on the sternum of the subject. A standard instruction was repeated prior to the testing as follows, "Please keep your eyes closed and say "Yes" when you feel the vibration". The vibrating tuning fork was placed from distal to proximal direction on the right 5th and 1st metacarpal bones, head of the ulna, radial styloid process, olecranon, medial epicondyle of the humerus, tip of the acromion process, and the mandible. A similar procedure was repeated on the left side and the findings were recorded as normal or altered.

Reflex testing:

The subjects were seated on an examination couch and the following reflexes were tested:

Reflex	**Root level**
Biceps Brachii	C5-6
Brachio Radialis	C6-7
Triceps	C7-8

Quadriceps Tendon	L3-4
Tendo Achilles	S1-2

Subject's responses were recorded as left or right side and full, depressed, or absent in comparison to the contra lateral side. In the event of muted response, reinforcement technique was applied to observe the difference.

Horner's Syndrome:

All subjects were examined for the presence of the following signs indicative of Horner's syndrome:

Ptosis: Drooping of the upper eye lid.

Miosis: Normal reaction present but constricted when compared to the other side. Reaction to light was checked by shining a torch light across the eye moving from lateral to medial side.

Anhidrosis: Decreased sweating over the face was checked by gently stroking by a plastic object such as a Biro, over the face. Absence of sweat and associated sebum will render the skin dry and hence the drag of the Biro will differ when compared to a normal skin.

Enophthalmos: In-drawing of the orbit.

Proprioception in the upper limbs and weakness of the recti muscles in the eye were examined as part of the general examination. However, the findings were only documented if they were considered to be clinically significant.

Soft tissue palpation:

Palpation of the cervical, upper and middle thoracic regions was performed with subjects in sitting and palpation of the lower dorsal and lumbar spine

was carried out with the subjects standing. Gentle pressure was first applied together with the pads of the index and middle fingers and then through the pads of the middle and ring fingers, over an area which was pain free, to create a shallow dimple under the pads of the fingers. This procedure was repeated three times, as an exercise to apply uniform amount of pressure during the actual examination. The standard NRS instruction was repeated. Palpation examination was done always in a sequence commencing with a known pain free area, followed by less painful, moderately painful and finally the most painful areas as indicated in the body chart which was obtained during the subjective examination. After the completion of palpation of a chosen structure, the subjects were asked to express the intensity of the pain / discomfort in a Numerical Rating Scale and recorded. In the case of the sternomastoid and the paravertebral structures of the cervical and upper thoracic regions, care was taken to identify the levels, as shown in the body chart. Whenever a subject, as a result of palpation, complained of either referred pain or aggravation of a particular symptom, an accurate account of the reported signs were recorded. The following structures were examined.

Muscle attachments at the nuchal line (Area 5 & 6): The investigator stood on the left side of the subjects . While restraining their head by placing the left palm over the forehead, palpated the nuchal line from a lateral to medial direction.

Paravertebral muscles (Area 10 & 11): The starting position was similar to the one described above. Palpation was carried out from the level of the seventh cervical vertebra, towards the nuchal line.

Articular pillar (Area 8 & 9): Soft tissue structures around the posterior aspect of the cervical articular pillar were palpated in sitting position as described above. The investigator stood on the respective side during this examination.

Ligamentum nuchae (Area 45): The starting position was similar to the one described above. The cervical spine was slightly guided into flexion, to the extent where it was either possible to palpate most of the spinous processes of the cervical vertebrae between second and the seventh or the ligamentum nuchae appeared to be taut. Palpation was carried out from the spinous process of the first thoracic vertebra in a cephalo-caudal direction

Cervico-thoracic junction (Area 12): The investigator stood on the left side of the subjects and palpated around and in between the spinous processes of the seventh cervical and the first thoracic vertebrae.

Trapezius upper fibres (Areas 31 & 32): The investigator stood behind the subjects and palpated the suprascapular area, including the postero-lateral border of the clavicle and traced the trapezius upper fibres along the angle of the neck towards their attachment in the cervical spine.

Trapezius middle fibres and the conjoint fibrous attachment at the site of the spine of the scapula (Areas 21 & 22): The investigator stood behind the subjects and palpated the area slightly above the root of the spine and the entire length of the upper border up to the medial border of the acromion

Levator scapulae: The Investigator stood behind the subjects and identified the superior angle of the scapula, the site of the insertion of the above

muscle and slightly medially traced along the course of the muscle, in a rostral direction up to the level of the spinous process of the seventh cervical vertebra.

Upper dorsal region (Areas 13 & 14): The investigator stood on the left side of the subjects and identified the spinous processes of the upper five thoracic vertebrae. Using the right index and the middle fingers, the investigator palpated the tendinous digitations of the semispinalis and spinalis capitis muscles, which were situated approximately 10mm -15mm lateral to the spinous processes of the above vertebrae.

Sternomastoid muscle: This important muscle had to be examined and reported as three distinct areas namely, upper end denoting the site of insertion together with the tendinous structure of the sternomastoid, mid belly, and the lower end meaning the fibrous origin of the sternomastoid. The investigator stood on the respective side of the subjects for examining this muscle. In addition to the palpable tenderness / discomfort, presence of haematoma and swelling were also recorded.

Temporomandibular joints (Area 35 & 36): The subjects were asked to keep the mouth closed without clenching the teeth. The investigator stood in front of the subjects and palpated the extra-articular joint structures of the above joint.

Platysma (Area 29 & 30): The investigator stood on the left side of the subjects and passively guided the upper cervical spine into slight extension, so that the chin was not obstructing the palpation of the pre-trachial region. Care was taken not to elicit pain either in the sternomastoid muscles or in the post-vertebral region. The occiput was supported by the

right hand and by using the left index and middle fingers the medial border of the left and right sternomastoid muscles were identified. A part of the Platysma confined between the medial borders of the sternomastoid muscles along the length of the trachea, was palpated.

3.5 Radiology

Plain radiographs of the cervical spine to examine the sub-occipital joints through open mouth, antero-posterior, lateral, oblique and swimmers in order to adequately examine the C7 and T1 vertebrae, were routinely performed at the Accident and Emergency Department. As part of the initial examination at the trial Centre, functional views of the cervical spine were also obtained. In the presence of clinical indication such as severe headache, intense pain, marked limitation of movements with associated antalgic posture, presence of intense paraesthesia and pain on axial loading of the spine, a C.A.T Scan of the cervical spine was also performed. These detailed radiological examinations were authorised by either the orthopaedic surgeon or his registrar. The standard radiological examinations were reported by the radiologist and cross checked by the orthopaedic surgeon or his registrar. A further review of the radiographs and CAT Scans were carried out by a senior radiologist specialising in the field of spinal trauma. These films were indefinitely preserved for long term follow-up. The following information was documented from standard x-rays:

Lordosis:	Normal / Altered
Alignment of the vertebrae (lateral & Antero-posterior films):	Normal / Altered
Widening of the Interspinous space:	If present the levels were noted
Instability:	If present the levels were noted
Limitation of Flexion:	If present the levels were noted
Pre-vertebral swelling:	If present the levels were noted

Degenerative changes: If present the levels were noted

Radiological findings such as presence of cervical rib, congenital anomaly, evidence of old trauma and details of the degenerative changes, were separately recorded.

3.6 Magnetic resonance imaging

As an adjunct study, routine MR Imaging of the subjects was organised and approval was given by the radiology department to investigate a maximum of fifty subjects. Forty subjects were involved who were considered to fit the "severe" category and five subjects from the "moderate" category as defined below. The subjects were imaged within two weeks after their injury. Using a Philips Gyroscan (1.5 T). 4-5mm sagittal spine echo T1 and T2 weighted images and 3mm axial gradient echo "T2" weighted images were obtained covering from the mid pons to D2 levels. Four subjects reported to have had significant amount of disc lesion were also imaged 3 months and 12 months post injury. The following definition was used to identify the "severe" and "moderate" categories:

Severe category: (A patient must display two or more positive findings described in the following sections.)

Section1: Altered Neurology

Motor deficit

Sensory deficit

Altered reflexes

Distally radiating pain

Paraesthesia - following an anatomical pattern

Retro-orbital pain

Blurred vision

Positive Horner's syndrome

Section2: Visible swelling in the sterno-mastoid muscle

Section3: Restriction of movements (75% +) in the cervical spine due to soft pain in the pre/post vertebral region measuring 70NRS or more.

Section 4: Radiology (cervical spine)

Pre-vertebral swelling

Marked spondylitic changes in 1 motion segment

Moderate spondylitic changes 2 or more motion segments.

Presence of congenital anomaly

Interspinous space widening

Signs of instability

Moderate category:

Neurologically intact

No visible swelling in the sternomastoid muscle

Active movements limited up to 50 % as a result of soft tissue pain measuring 40NRS-50NRS.

Palpable soft tissue pain measuring between 30NRS-50NRSin a pain scale

Activities of Daily Living - performed to a level of 75NRS

3.7 Follow-up examinations

Follow-up examinations were carried out at 4 weeks, 6 weeks, 12 weeks, 24 weeks, and 52 weeks intervals from the date of first examination. The

purpose was to document the changes in the information recorded during the initial examination and subsequently used to establish the progress of the subjects.

3.7.1 Subjective examination

The information obtained during the follow-up examinations were to the initial examination except the demographic and accident details. In addition, information related to treatment received by the control group between examinations was documented. In the experimental groups information deemed to be in breach of the trial were monitored and whenever this occurred the patients were excluded from the trial.

3.7.2 Objective examination

Range of motion in the cervical spine and associated pain were assessed and documented as it was carried out in the initial examination. Neurological examinations were performed if there were any impairment had existed in the previous examination. Isometric testing of the pre- and post-vertebral muscles of the neck were also documented as described below.

3.8. Muscle strength testing

Isometric muscle testing of the pre- and post-vertebral muscles of the cervical region was carried out at the 6th,12th, 24th, 52nd week follow-up examination. Using a specially designed electronic system, the peak force generated by the above muscles was recorded. The description of the tools used and an account of the pilot study which was conducted to test the reliability of the device, have been described elsewhere (Appendix-II).

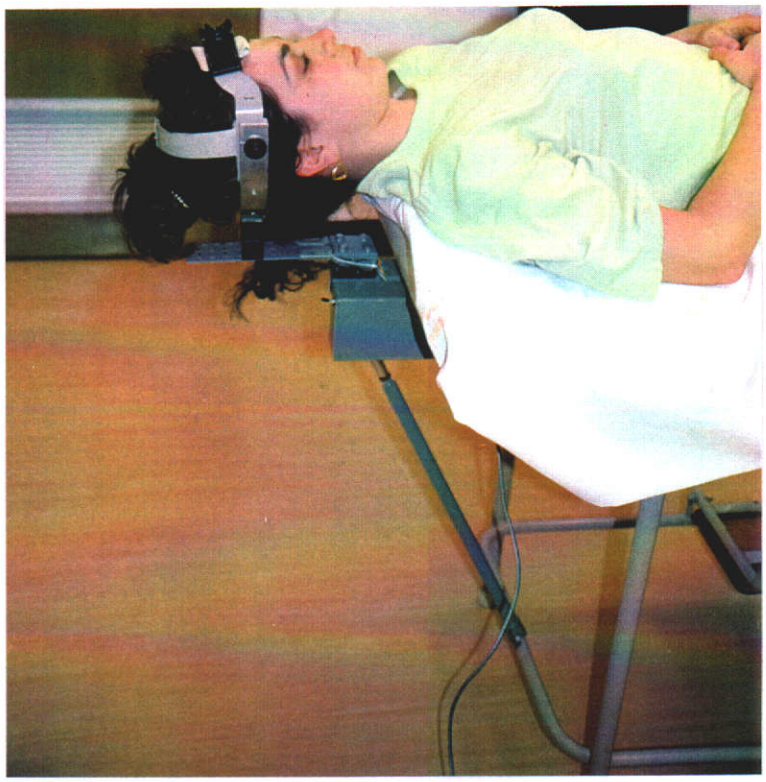

Figure 3.4 Strain gauge used for testing the peak force generated by neck muscles.

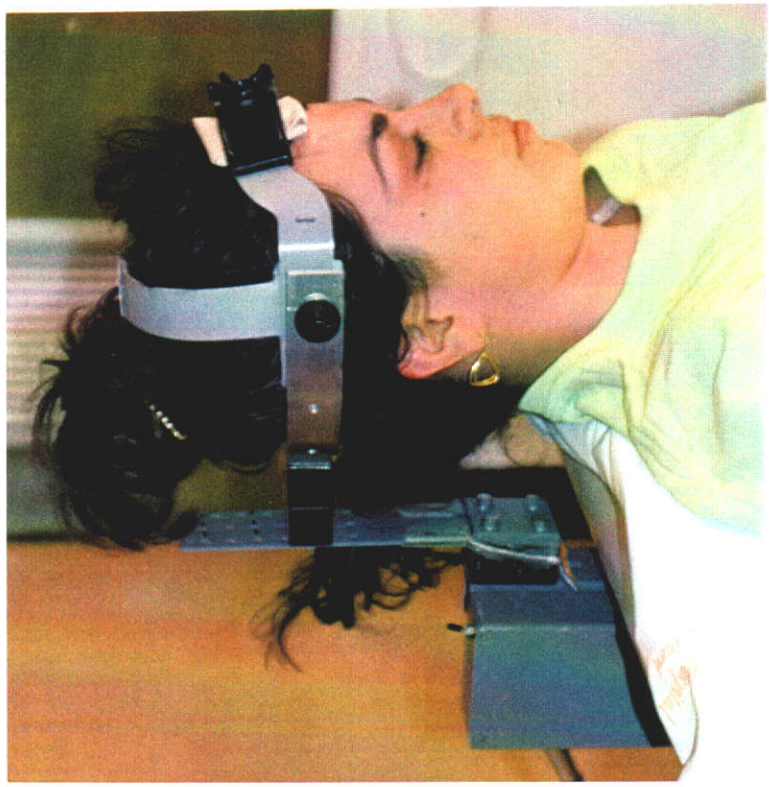

Figure 3.5 A close up of strain gauge used for testing the peak force generated by neck muscles.

The subjects were given a brief explanation about the testing and then were assisted to lie supine on the plinth, with their head resting on the strain gauge (Figure 3.4). The investigator adjusted the position of the subject, so that the bony prominence found on the superior aspect of the acromion processes were in level the edge of the plinth. A screw mechanism incorporated in the strut attached to the plinth was adjusted to bring the crown of the head to come into contact with the restraining plastic strap fitted to the free end of the strain gauge beam (Figure 3.5). At this stage, the fixating mechanism situated in the under surface of the plinth was tightened, in order to secure the strain gauge beam. A soft pad was placed on the forehead of the subject to avoid discomfort that might be caused by the pressure exerted by the plastic head strap. At the commencement of the testing, the subjects were asked to fold their arms across their chest as a precaution against arching their back as reinforcement during post-vertebral muscle testing.

3.8.1 Post-vertebral muscles testing

The following standard instruction regarding the test procedures was repeated to each subject:

> "This test will be carried out in two stages. During the first stage, you have to push your head against the strain gauge as hard as you can and while doing so, do not arch your back or dig your heels. Push hard and then hold it until I say, 'Let go' If it hurts a lot, you can let it go without waiting for me to say so. You will start pushing when I say, 'Push now' ".

A countdown of five seconds was given prior to giving the command "Push now'. Similarly, an instruction "Let go" was given at the end of ten seconds and the at this stage the pen recorder was switched off. An electronic digital

timer was used for the study purposes. The peak force was recorded by noting the digital display in the pen recorder and cross checked with the readings obtained from the pen recorder tracing paper.

3.8.2 Pre-vertebral muscles testing

A rest period of five minutes was allowed in between the pre and post vertebral muscle tests. The forehead strap was checked to make sure, it was well secured. The following standard instruction was repeated:

> "This time you will lift your head up as hard as you can towards the ceiling and hold it until I say, 'Let go'. If it hurts too much let it go gently without waiting for me to say so. You will lift your head when I say, 'Lift now' ".

The pen recorder was switched on just before the instruction was given. A countdown of five seconds was given to commence the lift and after ten seconds an instruction "Let go" was given. At the end of the testing the pen recorder was switched off once again. While the isometric contraction was in progress, the investigator noted down the peak force exerted as displayed by the pen recorder and later verified it with the pen recording. On completion of the isometric testing the forehead straps were undone and the subject was allowed to sit up after resting for a few minutes. Soon after the testing, often the subjects either reported sudden onset of pain-related symptoms or an aggravation of existing pain. This information together with the severity and the duration taken for the symptoms to subside were obtained and recorded.

Treatment protocol

Immobilised group:

The subjects in the immobilised group were fitted with a Philadelphia collar following their initial examination. (The Philadelphia collar is a Plastizote

appliance made with moulded chin and occipital supports, extended down to the upper part of the thorax. The front and back pieces of the collar are reinforced by two longitudinal plastic strips and secured to the neck by adjustable velcro straps. The collar came in different sizes so that individuals neck with varying morphological features can be adequately immobilised. Philadelphia collar is widely used at Sir George Bedbrook Spinal Unit for treating neck injuries associated with uncomplicated bony lesions. The subjects were allowed to remove the collar for showering, general cleansing, dressing, while applying local heat and in the case of men prior to shaving. Subjects were allowed to apply local heat to their neck including shoulder girdle region (e.g hot pack, hot water bottle, hot towel) for a duration of 15 minutes 3 times a day.

After the completion of the assessment at 4-weeks post-injury, the subjects were given a demonstration which was followed by an instruction on active mobilising exercises for the neck and isometric strengthening exercise for the post-vertebral muscles of the neck. The active mobilising exercises consisted of flexion, extension, right side flexion, left side flexion, right rotation and left rotation, repeated 15 times in each direction. These active movements were carried out three times a day within the limits of pain. The isometric strengthening exercise for the post-vertebral muscle was performed in supine lying position, with one pillow under the head. The subjects were asked to gently press the back of their head into the pillow, without tucking the chin, hold the contraction for three seconds and then to relax. The subjects were allowed to have short rest in between the isometric contractions and a clear instruction was given to stop the exercise if there was an increase in their pain level. This exercise was repeated for 15 times, twice a day. This programme was performed until the follow-up examination at week-6 post-injury. Thereafter, the subjects were instructed

to increase the number of repetition of the active range of motion exercises and the isometric strengthening exercise to 25 times, 3 sessions a day.

Active exercise group:

The subjects in the active exercise group were also allowed to apply local heat to their neck and shoulder girdle region as in the case of the immobilised group. The subjects in the active exercise group were shown and instructed on range of motion and isometric strengthening exercises as described in the preceding section for the immobilised group. The exercises were commenced from the day of initial examination and the subjects were instructed to repeat them within the limits of their pain. The subjects were instructed to repeat the range of motion exercises 15 times, 3 times a day. Similarly, the subjects were asked to do the isometric exercise 15 times, twice a day. This programme was performed until the follow-up examination at week-6 post-injury. Thereafter, the subjects were instructed to increase the number of repetition of the active range of motion exercises and the isometric strengthening exercise to 25 times 3 sessions a day.

The subjects were allowed to choose a physiotherapist for supervising the respective exercise programme and an information sheet was sent to clinicians through the subjects, in order to inform them about the treatment regimen. Whenever the subjects either wished to withdraw from the clinical trial or a breach of the above protocol occurred, it was duly noted and the subject was excluded from the study from that point in time of the trial period. Three subjects from the immobilised group withdrew from the study after the 6 week follow-up examination and two subjects withdrew after their follow-up examination at week-4 and one subject was excluded from the study for breach of protocol after week 12 follow-up examination. Four subjects withdrew from the study after the follow-up examination at week 4.

The rest of the subjects who had dropped out of the study were either symptom free or experienced difficulty in finding time to attend the follow-up appointments.

3.9 Statistical analysis

Statistical analyses except for the reliability studies were performed on a main frame VAX computer, under VMS operating system using SPSS (Release 4.1) programme. Statistical analyses for establishing intra tester reliability for the measuring instruments were performed on a Macintosh computer using Statview (Abacus concepts 1992) programme. An alpha level of <0.05 was chosen as a criterion of significance for all the analyses.

3.9.1 Reliability tests

Intra tester reliability for the measuring instruments was calculated using Repeated measures of ANOVA and derived intra class correlation coefficients (Hass, 1991; Portney and Watkins, 1993).

3.9.2 Demographic details

Descriptive statistics are provided for the age, gender, occupation, types of collisions, types of vehicles, speed involved, restraints, past history of "whiplash" type of injury, and litigation status.

3.9.3 Perceived pain

An initial analysis of the histograms of the means demonstrated a skewed distribution for the total sample as a whole and also in the cases of random groups (Figure A.III.1). For this reason the raw data was transformed using natural logarithm in order to remove the skewness and to achieve a normal distribution. Thus, the derived log transformed data has been used through

out the statistical analysis (explanation for the Transformation is presented in the Results Chapter under the relevant section- see page 173).

The histograms of "raw data" and "log transformed" data are presented for a comparison (Figure A.III.1, A.III.2, Pages 350-351). In the text section, geometric mean and geometric standard deviations have been reported for the study population and respective treatment groups. In addition to this the arithmetic mean (mean) has also been presented in relevant tables. It should be noted that the arithmetic mean has only been reported as a matter of interest and was not used for statistical analysis. Descriptive statistics are provided for the total sample and treatment groups for each of the time periods. In addition, the frequency of the symptomatic body areas are also provided for the total sample and the treatment groups for each of the time periods.

Pearson correlation coefficient was used to establish the strength of association between the total number of symptomatic areas and the total sum of pain score reported by the subjects at the initial examination.

A one-way ANOVA test was performed for each of the time periods to test the null hypothesis that there was no statistically significant difference between the treatment groups. When the respective p value was equal to or less than .05, demonstrating significant differences between the groups, *post hoc* multiple comparison testing using the Scheffe's procedure was performed to identify the treatment groups with statistically significant differences.

A repeated measures ANOVA (ANOVA-RM) was performed to test the between subjects effects. In addition to this, an ANOVA-RM was also

performed using the follow-up time periods as a covariate, to verify whether the subjects could have got better with or without any form of treatment, through passage of the time alone.

Prognostic variables

A statistically significant difference between the treatment groups as evidenced from the ANOVA results of the initial examination may be due to the presence of one or more confounding variables in the treatment groups. Hence it was decided to perform a series of analysis of covariance (ANCOVA) for the continuous variables and a series of 2-way ANOVA for the categorical variables, to determine the effect of the following covariates on pain and the interaction with the treatment factors:

1. gender;
2. age;
3. occupation;
4. types of collision;
5. speed of the vehicles involved;
6. seating position;
7. presence of headache within 24 hours after the collision;
8. the severity of the pain related symptom as reported at the base level examination;
9. presence of paraesthesia;
10. litigation;
11. loss of cervical lordosis
12. pre existing degeneration of the I.V.D in the cervical spine; and
13. interscapular pain.

Multiple Regression using change scores

The results of the one-way ANOVA corresponding to the initial examination indicated that statistically significant differences existed between the treatment groups, indicating that the randomisation was not effective and that this might have resulted in selection bias. In order to test whether the above situation has affected the inferences obtained from ANOVAs, further test was performed using multiple regression analyses. The procedure is explained in the Results Chapter under the relevant section and this departure from convention is for the convenience of the reader (see page 201).

Logistic regression

Logistic regression (Forward stepwise) was performed in order to identify the significant predictor variable and associated odds ratio. The following predictor variables included in the regression model were either identified as significant covariates from the preceding analyses or mentioned in the literature as having prognostic value:

1. age;
2. gender;
3. speed of the vehicles;
4. baseline pain;
5. radom groups
6. immobilisation regimen;
7. active exercise regimen;
8. presence or absence of cervical lordosis;
9. disc degeneration;
10. presence of interscapular pain;
11. blurred vision;
12. litigation

13. difficulty in focusing; and
14. paraesthesia.

The justification for performing logistic regression is presented in the Result Chapter under the relevant section .

Survival analysis

Survival analysis was performed using Lee-Desu statistics to test the probability that ST (survival time) = Grp A = B = C. The resulting survival tables are presented.

3.9.4 Strength of the pre-and post-vertebral muscles

The descriptive data for the pre and post vertebral peak muscle force for each of the time periods from week-6 are provided for the total sample and also for the treatment groups. Series of one-way ANOVAs were performed in order to establish statistically significant differences between groups and when present *post hoc* multiple comparison test (Scheffe's procedure) was used to identify the groups that significantly differed.

Series of ANCOVAs were performed to establish the effect of pain at the corresponding time period on the respective peak force generated.

3.9.5 Range of motion of the cervical spine

The descriptive data are presented for the active flexion, extension, right side flexion, left side flexion, right rotation and left rotation for each of the time periods by treatment groups as well as the total sample.

Pearson correlation coefficient was obtained in order to establish the strength of association between the active and passive range of motion for each of the time periods. A similar test was also used to determine the

strength of correlation between right and left active side flexion and right and left active rotation.

Series of one-way ANOVAs were performed in order to determine whether there was a statistically significant difference between groups and when present *post hoc* multiple comparison test (Scheffe's procedure) was used to identify the group that significantly differed. Thus, the differences in active flexion, extension, right side flexion and right rotation were analysed for each of the time periods.

Series of ANCOVAs were performed to determine the significance of age and pain factors as covariates and also their interaction effects.

Multiple regression analysis

The ANOVA results of the initial examination indicated that there was a statistically significant difference between groups for each of the movements that were analysed. It was evident that the randomisation has failed to formulate treatment groups with similar values; thus, introducing a selection bias. This situation was addressed by creating a new variable using change scores method. The new variable was used as an independent variable and the following predictor variables were used as independent variables:

1. age;
2. level of pain for the corresponding time period; and
3. treatment factors.

The justification for the procedure used is described in the Result Chapter under the relevant section.

3.9.6 Return to work

Survival Analysis

Survival analysis was performed using Lee-Desu statistics to determine the differences between the treatment groups and relevant life tables are presented. The event of interest was the time period at which the subjects returned to 100% of their normal work level which corresponded to their respective pre-accident level.

CHAPTER FOUR DESCRIPTIVE ANATOMICAL STUDY OF THE DEEP MUSCLES OF THE CERVICAL REGION

4.1 Introduction

The main functions of the human spine include the axial support of the body, the facilitation of purposeful complex motions, shock absorption and protection of the neural elements. All of these functions of the spine can only be achieved by the integration of the mechanical forces contributed by the bony architecture, the ligaments of the spine together with the structures of the intervertebral discs and complex extrinsic forces provided by the skeletal muscles, adjacent to the axial skeleton. An accurate account of the morphological information of the muscles in the cervical region, is essential for a proper understanding of soft tissue injuries of the neck and thus facilitate appropriate treatment. Derived mathematical models of the cervical region used in anthropometric and biomechanical studies also require precise morphological details of these muscles, if they are to produce models which would simulate reality (Huelke and Nusholtz, 1986; Deng and Goldsmith, 1987a; Deng and Goldsmith,1987b; Nolan and Sherk, 1988; Panjabi et al., 1993).

A preliminary dissection study of the cervical region which was undertaken to attain familiarisation of anatomical structures, highlighted a large number of deficiencies and inaccuracies found in the literature, in respect of the morphological details and functional role of the prevertebral and post vertebral deep muscles (Gray, 1858; Sharpey et al., 1878; Fountain, Minear and Allison, 1966; Sinclair, 1981; Vitti, Fujiwara, Basmajian and Lida, 1973; Warwick and Williams, 1973; Johnson et al., 1975; Last, 1978; Williams et al., 1989). From this information, it was evident that available biomechanical information which is based on existing anatomical details

and widely used in the study of "whiplash" type of injuries may be flawed. Similarly, the deficient anatomical description and thus the inferred functions of these deep muscles are also likely to mislead clinicians from gaining appropriate information with reference to pathology and might also adversely affect the possible treatment of soft tissue injuries of the cervical region. Thus, a detailed dissection study was deemed necessary in order to establish an accurate account of morphological details of longus capitis, rectus capitis anterior, longus colli, multifidus which is also sometimes referred as rotatores and semispinalis capitis muscles. As a result of this, a descriptive histological study of the longus capitis and rectus capitis anterior muscles was also subsequently carried out.

4.2 Method

Nine embalmed cadavers and twenty five fresh post-mortem cervical spines with partially resected basiocciput removed during autopsy, were used for this dissection study.

4.2.1 Longus capitis

The embalmed cadavers were dissected to expose the prevertebral deep muscles. In order to trace the rostral attachment of the longus capitis and longus colli muscles and explore rectus capitis anterior et lateralis muscles, the mandible was disarticulated and the contents of the buccal cavity were removed. It was essential to resect the face of the cadavers by means of a hand saw. The caudal attachments of longus capitis and longus colli muscles were traced by exploring the thoracic cavity. The sternum was split and the contents of the thoracic cavity were removed. The bony attachments at the thoracic level were identified by counting the ribs. The fascia covering the longus capitis was removed in order to verify its medial, lateral, and dorsal attachments along its entire course, and its length was measured, while the width of the muscle was measured by using a calliper.

Three of the nine cadavers were prepared in the manner described above and the fascia covering the longus capitis muscles was left undisturbed until a motion study of the cranio-vertebral joints was done. Ten of the twenty five fresh specimens were also used in the motion study. In the case of the embalmed cadavers, flexion-extension of the atlanto-occipital joint and rotation of the atlanto-axial joints were performed by moving the head, whereas in the fresh specimens, the basal part of the skull was moved to produce the above movements in the respective joints. The mechanical behaviour of the longus capitis in terms of tightening and slackening was observed and recorded.

4.2.2 Longus colli

A similar study was carried out on the same cadaveric preparations and twenty five post-mortem spines which were used to study longus capitis muscles. The longus capitis muscles were transversely cut at the levels of C3-4 motion segment and the rostral segment was reflected in order to expose the tendinous attachment of superior oblique part of the longus colli muscles from either sides. Similarly, the caudal segments of the longus capitis muscles were reflected so that the attachments, size and course of the vertical and inferior oblique parts of the longus colli muscles could be clearly observed. A motion study was carried out in the fresh spines with particular reference to the tendon of the superior parts of the longus colli muscle during extension of the atlanto-occipital joints and rotation of the atlanto-axial joints. The above movements were simulated by moving the basal part of the skull.

4.2.3 Rectus capitis anterior

After the completion of preceding studies of the longus capitis and longus colli muscles, the same cadaveric preparations and fresh spines were used to document the size, attachments and relationship of rectus capitis

anterior. This muscle were viewed by cutting and reflecting the longus capitis muscles as described previously. Due to the proximity of the rectus capitis anterior to longus capitis muscles at the level of atlanto-occipital joints, extreme care was necessary to separate and reflect the rostral segment of longus capitis muscle so that the cranial attachment could be clearly studied. The length and width of the rectus capitis anterior muscles of the cadaveric preparations were documented by using a calliper.

4.2.4 Semispinalis capitis

The structure, course, attachments and relationship of semispinalis capitis was documented by dissecting nine embalmed cadavers. The skin and fascia from the posterior aspect of the neck and thoracic regions were removed to expose trapezius and splenius capitis muscles which were carefully resected to expose semispinalis capitis muscle. The caudal attachments of semispinalis capitis muscle in the thoracic region and its medial and lateral attachments in the cervical region were carefully scrutinised. The relationship of semispinalis capitis muscle to greater occipital nerve and in particular the manner in which the nerve made its passage through this muscle was also documented.

4.2.5 Multifidus (rotatores)

The structure, course, attachments and the relationship of the multifidus was studied in nine embalmed cadavers after documenting morphological details of semispinalis capitis muscle. In addition, twenty five fresh spines were also used to document similar information. This muscle was dissected in layers so that the course and attachments could be accurately recorded. The site of origin and its relationship to the articular capsule of the zygapophyseal joints were also recorded.

4.2.6 Capsular structures

As a part of a wider study, the macroscopic appearance of the articular capsular structures of the zygapophyseal joints of the cervical spine were documented by dissecting nine embalmed cadavers and twenty five fresh spines. Similarly, capsular structure of the atlanto-axial and atlanto-occipital joints were also recorded as found in three cadaveric preparations and twenty five fresh post-mortem spines.

4.2.7 Silver stain preparation - modified Bielschoschowsky's method (Beech and Davenport, 1933; Wilson, 1991)

Longus capitis and rectus capitis anterior muscles were removed from three post-mortem spines and immediately secured to a glass slide with strings, so that the muscles will be kept fully stretched while fixation in 10% marbled formaldehyde for a period of 12 weeks. The mounts were appropriately marked in order to correctly identify the caudal, rostral ends and ventral, dorsal surfaces of these muscles. While the specimens were fixed, several practice sessions of the following procedures were carried out using longus colli muscles removed from Quokka (Setonix Brachyurus) which were sacrificed for other experiments. Similarly, three longus capitis and two rectus capitis anterior muscles were also used during a pilot study. After a period of 12 weeks in formaldehyde, remaining three longus capitis muscle was cut into three segments measuring approximately 25 mm in length, marked for later orientation of sections and stored. However, owing to the size of the rectus capitis anterior muscles, it was possible to section remaining four specimens without having to cut into smaller segments. The Leitz quadrant freezing microtome was used to obtain 10 micron sections of the rectus capitis anterior and longus capitis muscles. This was done by placing the respective muscles on the jig so that the sectioning was always carried out from ventral to dorsal surface of the muscle. In order to maintain the identification of the specimens, only one muscle or segment of the

muscle (e.g middle segment of longus capitis) was sectioned and processed. Thus, three sections obtained from the ventral and dorsal aspects of the respective specimens were used for histological preparations as described below.

All the glass utensils used were soaked in a plastic container filled with bromic acid for a period of 24 hours, then thoroughly rinsed several times in distilled water and allowed to drain. Throughout this procedure, it was necessary to use a new, clean glass stirring rod whenever the preparations were transferred from one container to the next in order to avoid chemical contamination. The sections were transferred into a beaker containing pyridene which was used as a cleansing agent to remove fatty deposits in the muscle and allowed to stand on an agitator till the sections appeared transparent. At this stage the sections were transferred into a beaker containing distilled water using a glass stirring rod and thoroughly washed to remove pyridene. This process was completed by changing the distilled water four times. The sections were placed on a clean blotting paper to remove the water and then left in a beaker containing 5% silver nitrate ($AgNo_3$) solution for five minutes. Thereafter the specimens were placed on a clean blotting paper to remove excess $AgNo_3$ and transferred into culture dishes respectively containing 10% and 2% formaldehyde mixed with a grain of NaCl for a duration of 4 seconds in each dish. Then the sections were left in a beaker containing 2% formaldehyde mixed with a grain of NaCl for a duration of 30 minutes and then transferred into a bath of ammoniacal silver solution prepared by adding a few drops of 30% ammonium hydroxide into $AgNo_3$ preparation for a duration of 4 seconds. The next transfer into 2% formaldehyde solution was a critical one. The sections were carefully transferred from the ammoniated $AgNo_3$ preparation one at a time into the 2% formaldehyde solution for a few seconds and

quickly removed as soon as the specimen turned into a golden brown colour. The next stage was a rinse using 5% sodium thiosulpahate solution for 5 seconds so that excessive $AgNo_3$ could be removed. During the final stage the sections were washed in distilled water and viewed under a microscope. Those specimens which have turned out to dark and unsuitable for viewing were discarded and suitably stained sections were dehydrated and mounted using routine procedures (Wilson, 1991).

4.3 Results

4.3.1 Longus capitis

This muscle was found to be consistently present in all the nine embalmed cadavers and twenty five fresh specimens, situated ventral to the longus colli muscles on either side of the vertebral column. The longus capitis appeared as a strong fleshy muscle, presenting its maximum girth as it traversed over the third cervical vertebra (Figure 4.1). The average length of this muscle was 180 mm, an average width of 16 mm and with a cross section of 8 mm at the level of the third cervical vertebra. The origin could be traced as a thin tendinous slip arising from the anterior surfaces of the T2, T1, vertebrae (Figure 4.2) and increasing in its size by thick tendinous slips received from the transverse process of the C7 vertebra. At the level of the C6 vertebra the tendinous appearance was lost as it assumed a spindle shape and a glistening appearance. There was no anchorage on the dorso-medial side of this muscle, however a thick fibrous sheath of fascia from the level of the 6th vertebra up to the site of rostral attachment invests the muscle. The lateral border of the muscle was firmly anchored by means of strong, short, thin fibrous slips to the anterior tubercle and the adjacent transverse process. This arrangement was invariably present on the transverse processes of the C6, C5, C4, and C3 vertebrae.

Figure 4.1 Longus capitis and Longus colli.

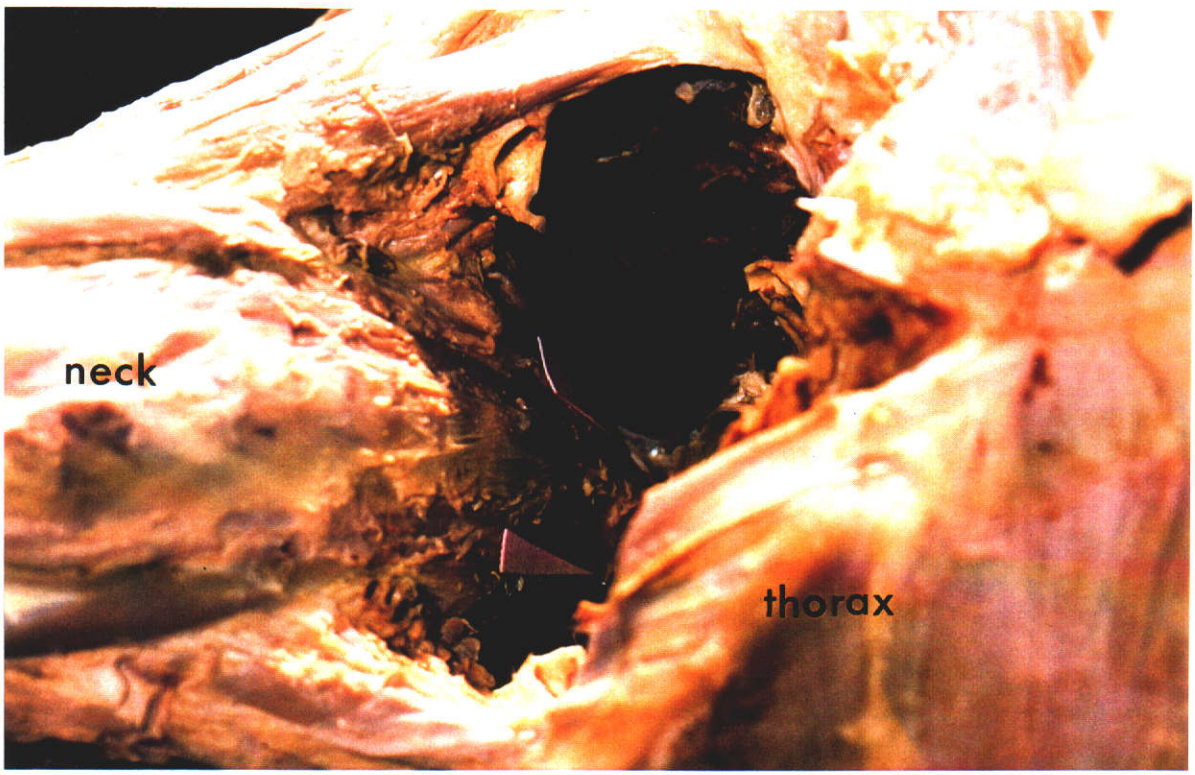

Figure 4.2 Origin of longus capitis muscle displayed. The caudal attachment of the longus capitis traced into the thoracic cavity.

As the muscle traversed the lateral mass of the axis, it was closely related to the rectus capitis anterior muscle, which sometimes fused with the longus capitis as they approached the site of insertion. Unlike other muscles, the longus capitis did not transform into a typical tendinous structure. This muscle neither lost its muscular appearance nor its size as it found it's insertion slightly antero-laterally to the articular condyles of the basal part of the skull. The average width of the muscle at this site was 10mm. The muscle tightened during extension of the atlanto-occipital joints and slackened during flexion movement. During rotation of the atlanto-axial joints, the longus capitis muscle of the contralateral side tightened while the ipsilateral muscle slackened.

4.3.2 Longus colli

Longus colli was consistently present in all the specimens and situated dorsal to the longus capitis. This is the deepest muscle found in the prevertebral region of the cervical spine, and is made up of a number of fusiform shaped digitations stretching between two or three motion segments (Figure 4.1). Unlike the situation reported in standard text books, the superior oblique part was well developed when compared to the vertical and inferior oblique parts of the muscle (Warwick and Williams, 1973; Williams et.al., 1989). A similar comparison between the vertical and inferior oblique parts showed that the vertical part was well developed and differentiated from the superior oblique part of this muscle. On the contrary in all the specimens, the inferior oblique part was clearly defined and also appeared to consist of thicker muscle digitations when compared to the vertical part and this observation was in contradiction with the literature (Warwick and Williams, 1973; Williams et.al., 1989). The digitations forming the superior oblique part were attached to the anterior aspect of the transverse processes and the anterior tubercle of C5, C4 and C3 vertebrae

and converged to form a very short conjoint tendon measuring between 4mm-7mm in length. As this tendon gained its rostral attachment around the tubercle situated in the anterior arch of the atlas, it also fused with the capsule of the atlanto-axial joint; thus, reinforcing part of the anterior aspect of the capsular structure. Simulated extension/ flexion of the atlanto-occipital joints effectively produced tightening and slackening of the conjoint tendon and this was due to the manner in which the tendon fused with the anterior aspect of the atlanto-occipital joints capsular structure.

4.3.3 Rectus capitis anterior

The rectus capitis anterior muscle was consistently present in all the three cadaveric preparations and fresh post-mortem spines, whereas the rectus capitis lateralis was present in one of the three cadaveric preparations and ten of the twenty five fresh spines. The rectus capitis anterior measured 20mm-25mm in length and 4mm-6mm in width, thin and membranous in appearance. The caudal end of the rectus capitis anterior is attached to the lateral aspect of the lateral mass and an adjoining area in the anterior aspect and superior border of the transverse process. The rostral end more or less fused with the ventral surface of the longus capitis muscle (Figure 4.3) and gained its attachment to the inferior surface of the basilar part of the occipital bone. Macroscopic examination of the caudal and rostral ends failed to demonstrate features of a typical muscle tendon. Moreover, the size and location of this muscle with respect to the atlanto-occipital joint indicated that the rectus capitis anterior was unsuited to play any mechanical role as described in the literature (Gray, 1858; Sharpey et al., 1878; Sinclair, 1981; Warwick and Williams, 1973; Last, 1978; Williams et al., 1989). However, the histological study of the rectus capitis anterior indicated that this muscle is more likely to contribute as an integral part of the proprioceptor mechanism operating in the cranio-vertebral region.

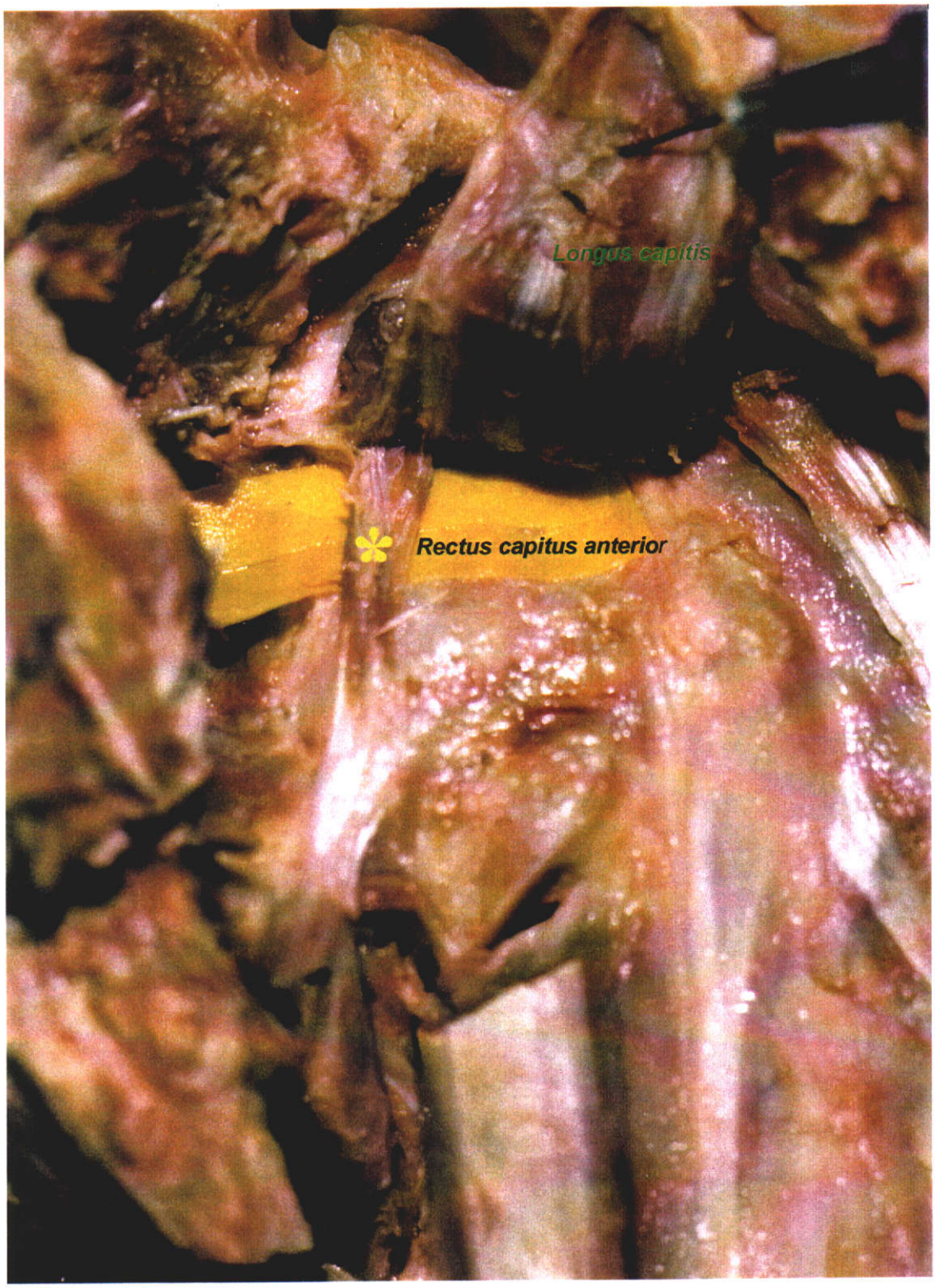

Figure 4.3 Rectus capitis anterior muscle.

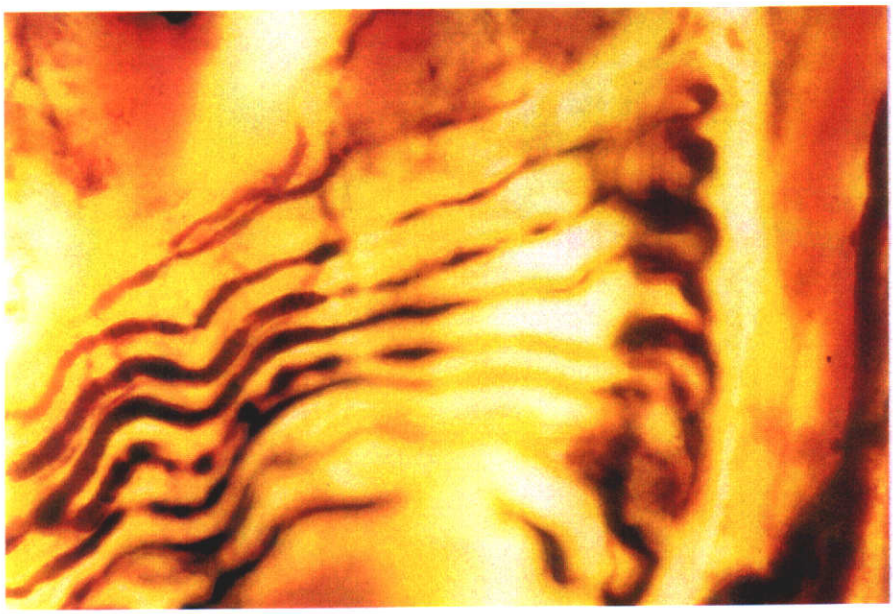

Figure 4.4 Silver stain histological preparation of rectus capitis anterior muscle showing nerve fibres.

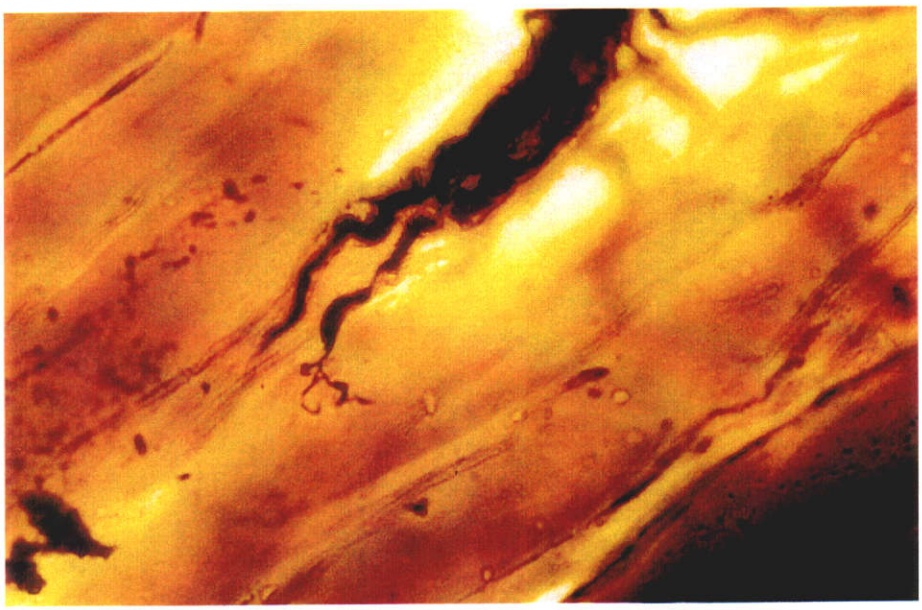

Figure 4.5 Silver stain histological preparation of longus capitis muscle showing nerve fibres.

This postulation can be further supported by the following observation made from the histological study (see below). The histological study of the rectus capitis anterior and longus capitis muscles, using silver staining method, clearly demonstrated that the size and proportion of nerve fibres present in both muscles. An estimation of the number and type of nerve fibres among these two muscles showed that the rectus capitis anterior which is considerably smaller in size when compared to the longus capitis received disproportionately large number of myelinated nerve fibres and also a higher proportion of free nerve endings (Figure 4.4, 4.5).

4.3.4 Semispinalis capitis

The size and length of the semispinalis capitis muscle clearly showed that this is the strongest muscle among the post-vertebral group of muscles. The semispinalis capitis gained its rostral attachment by a fleshy tendinous structure, to the medial half of an area situated between the superior and inferior nuchal lines. The caudal attachments consisted of a number of small but strong, flat fibro-tendinous structures which were predominantly attached to either sides of the rib angles rather than the transverse processes of the thoracic vertebrae as mentioned in the literature (Gray, 1858; Sharpey et al., 1878; Sinclair, 1981; Warwick and Williams, 1973; Last, 1978; Williams et al., 1989) (Figure 4.6). A detailed examination showed that the tendinous structures attached to 1st, 2nd and 3rd ribs derived from the deeper (ventral) part of this muscle, whereas the tendinous slips from the middle and superficial part of the semispinalis capitis muscle found their attachments primarily to an area medial to the rib angles of the 4th, 5th, 6th and 7th ribs and only a very few slips fused around the posterior aspect of the transverse processes of corresponding thoracic vertebrae.

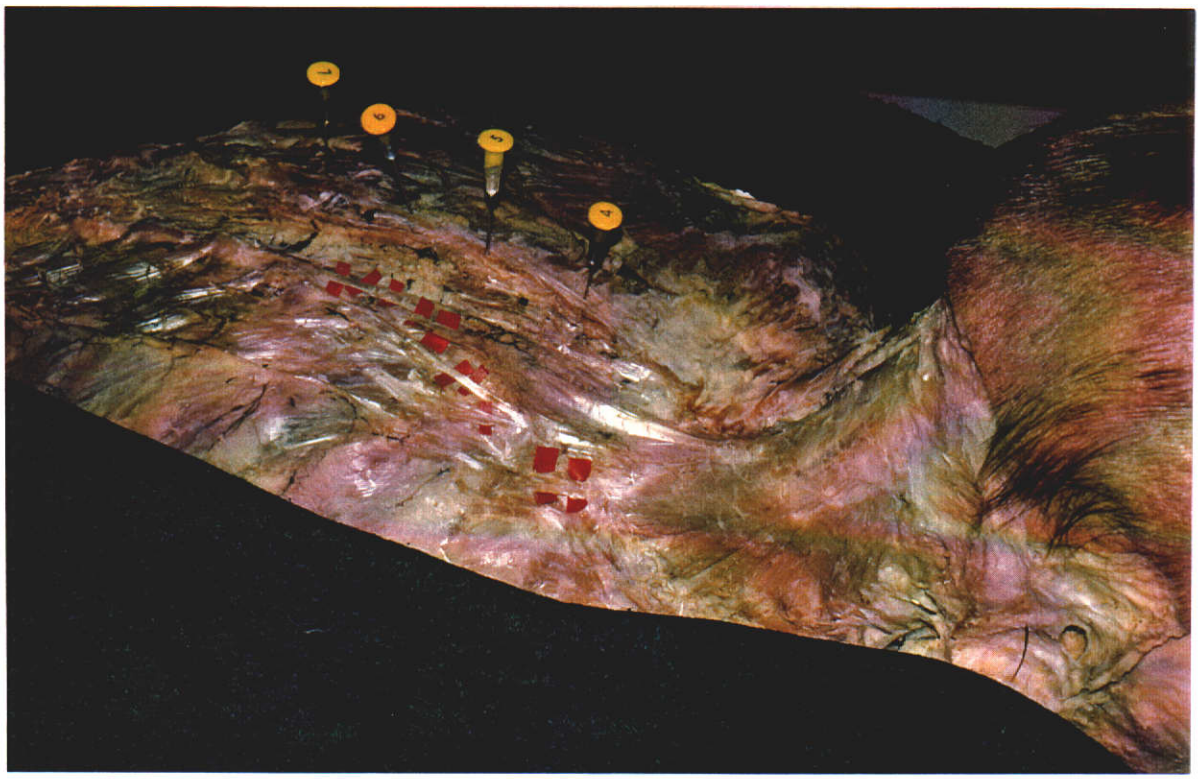

Figure 4.6 **Caudal tendinous attachments of semispinalis capitis muscle.**

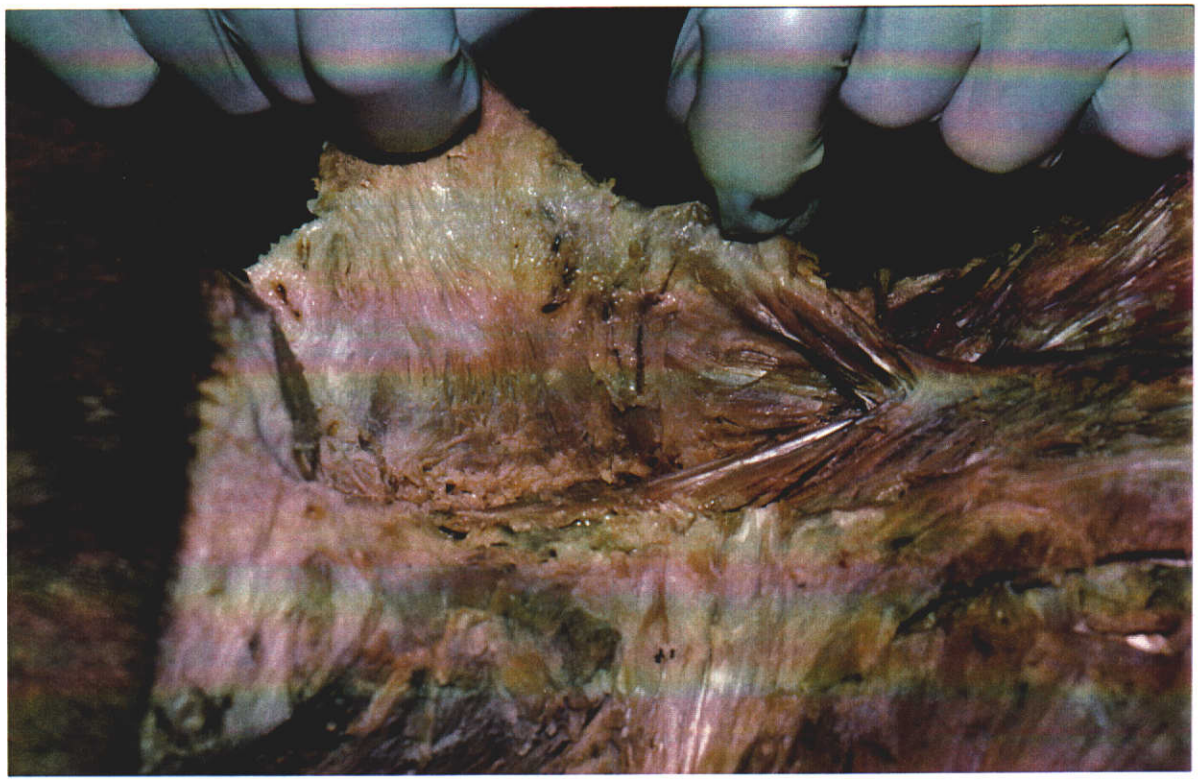

Figure 4.7 Medial border of the semispinalis capitis muscle in the cervical region.

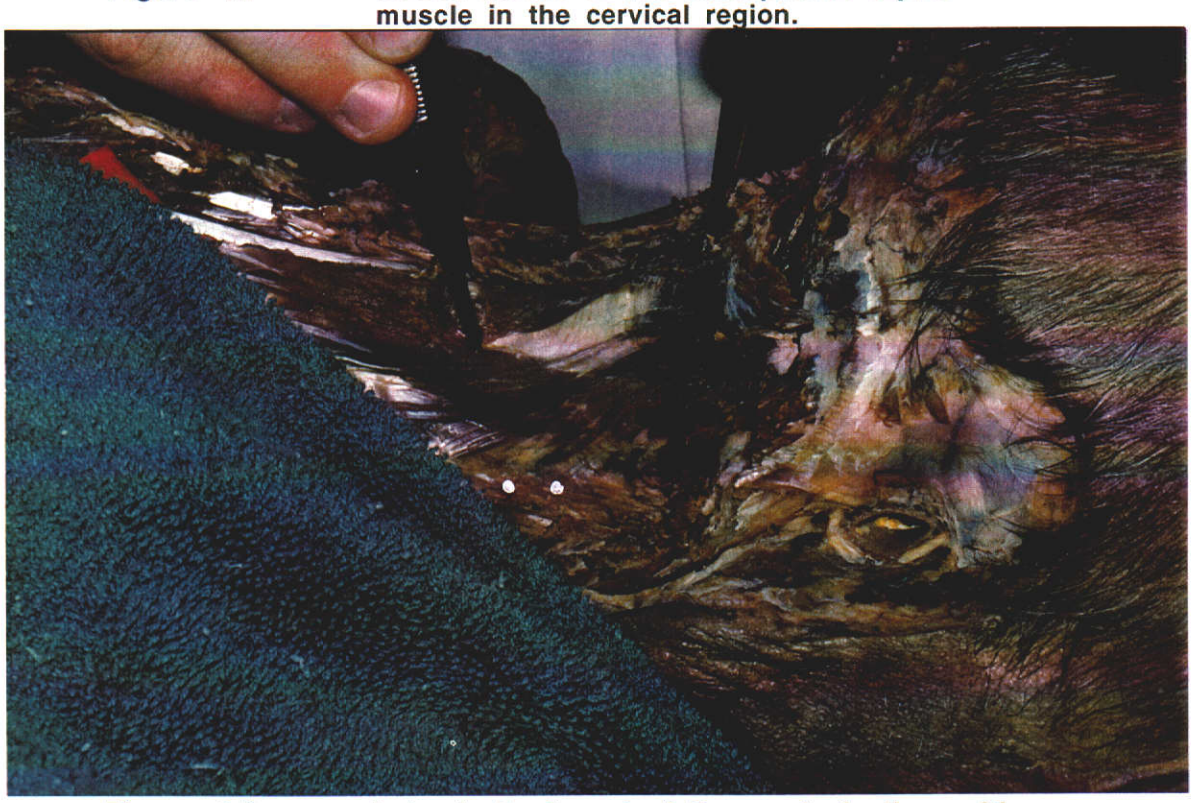

Figure 4.8 Lateral attachment of the semispinalis capitis muscle in the cervical region, by fibrous slips to the posterior aspect of the transverse processes of the cervical vertebrae.

As the tendons passed rostrally, at the level of C6-T2 vertebrae, transformed into a fusiform shaped muscular structure which significantly increased in its size between C2-C5 vertebrae. Even though the semispinalis capitis failed to have any direct bony attachment, at the level of C3-C7 vertebrae, the medial border of this muscle fused with the ligamentum nuchae by means of a thick fibrous structure, information which is not available in the literature (Gray, 1858; Sharpey et al., 1878; Sinclair, 1981; Warwick and Williams, 1973; Last, 1978; Williams et.al., 1989) (Figure 4.7). Similarly, the lateral border of the muscle was anchored by thin fibrous slips to the tips and posterior aspect of the transverse processes of the C3-C7 vertebrae (Figure 4.8). A detailed description of the caudal, medial and lateral attachments of semispinalis capitis is also not available in the literature. After emerging under the inferior border of the obliquus capitis inferior muscle, the greater occipital nerve pierced the ventral surface of the semispinalis capitis; thus, forming a tunnel through the substance of this muscle for its passage and made its exit on the dorsal surface, very close to the medial border of the muscle at a point slightly rostral to the spinous process of the second cervical vertebra.

4.3.5 Multifidus (rotatores)

A detailed study of the muscle was performed by dissecting the nine embalmed cadavers and twenty five post-mortem specimens, in order to establish the bony attachments and orientation of this muscle. The multifidus group of muscles were the deepest placed muscle of considerable size in the post vertebral region of the cervical spine. In all the specimens, the muscle was consistently present. The course and attachments of different layers of the muscle has not been adequately described in the literature (Last, 1978; Warwick and Williams, 1973; Williams et.al., 1989).

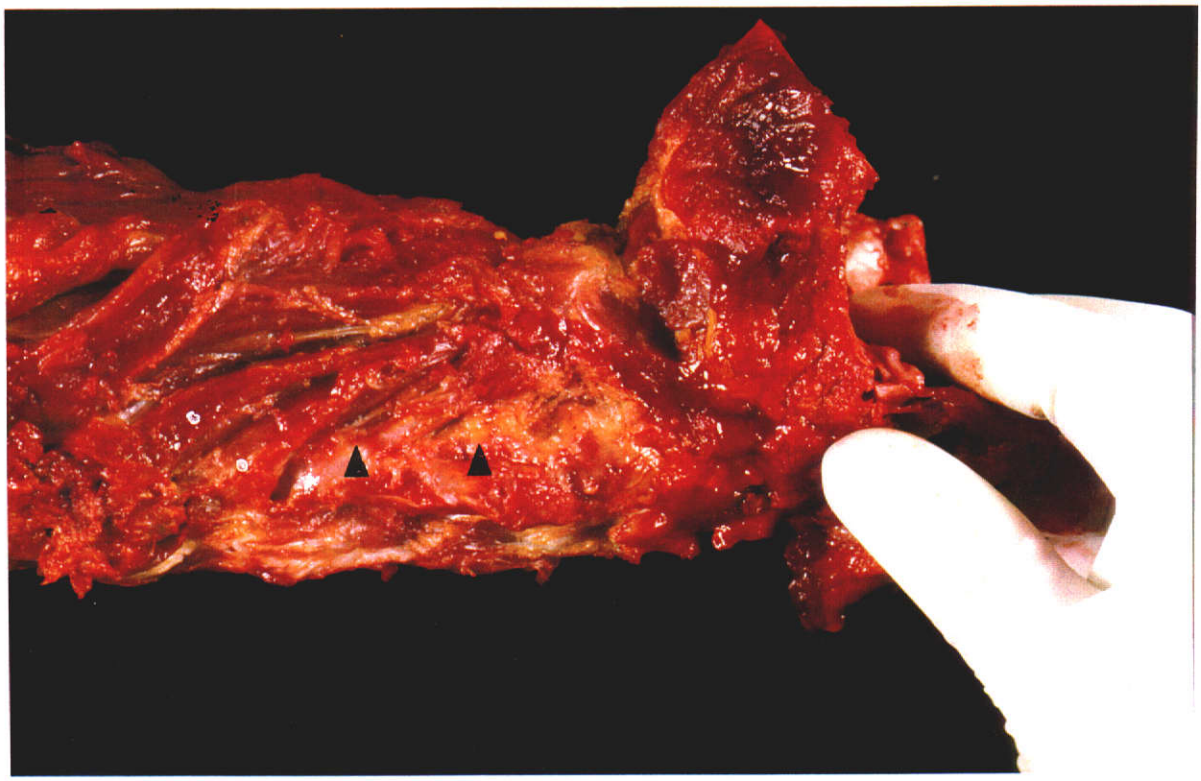

Figure 4.9 **The superficial and deep fibres of multifidus muscle.**

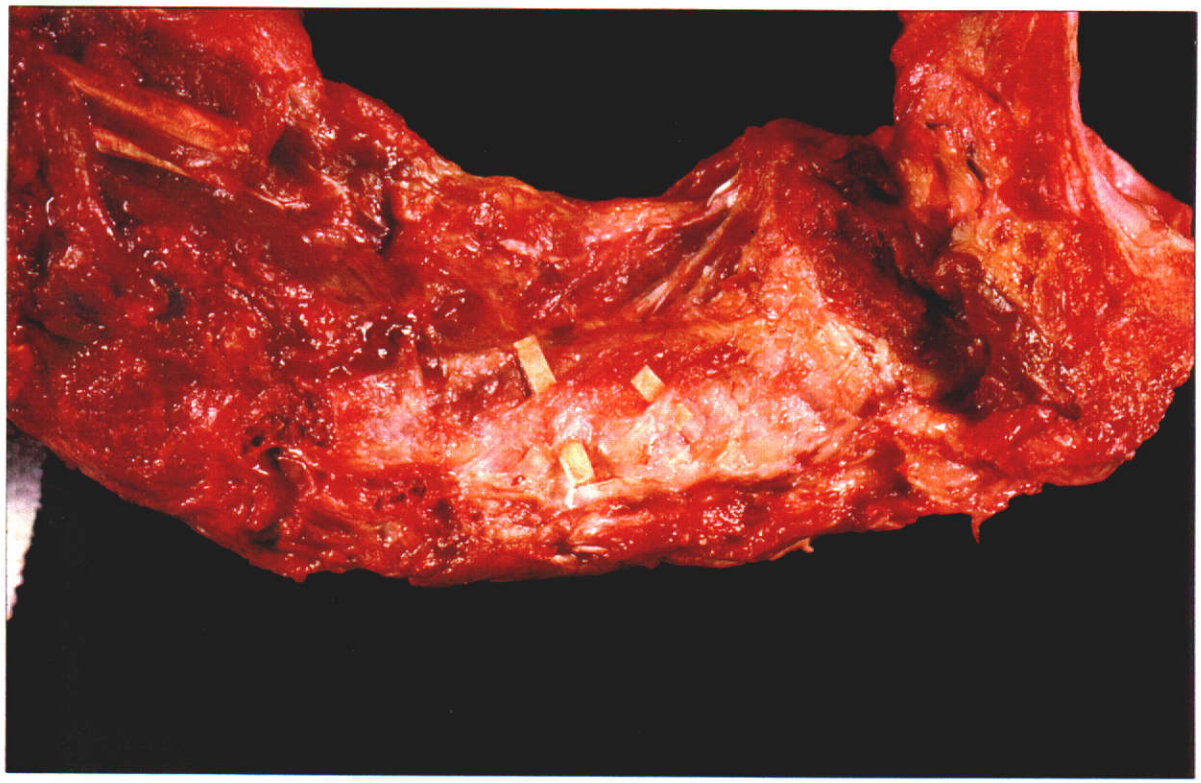

Figure 4.10 Tendinous origin of the multifidus muscle fusing with the posterolateral aspect of the articular capsule of the zygapophyseal joint.

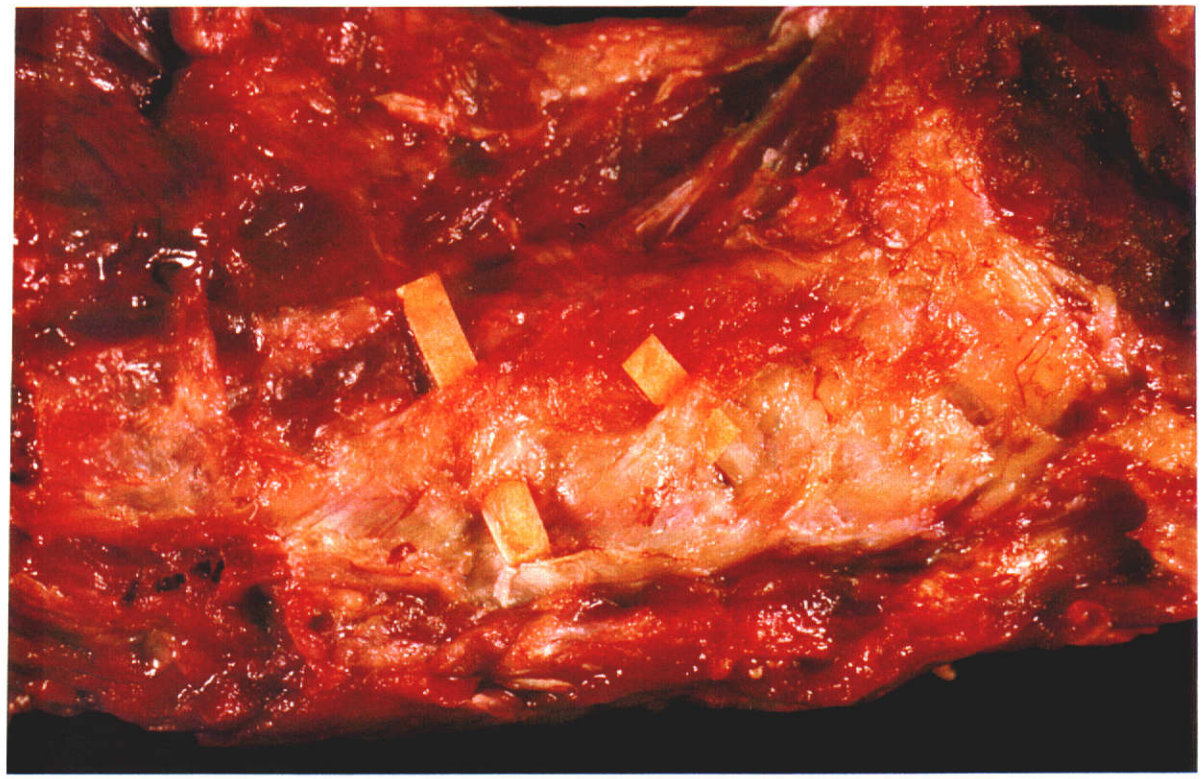

Figure 4.11 A close up view of the tendinous origin of the multifidus muscle fusing with the posterolateral aspect of the articular capsule of the zygapophyseal joint.

Two layers of fibres were easily distinguished, viz superficial fibres which were crossing more than one motion segment (Figure 4.9), and the deeper fibres which were inserted in the lamina of the proximal vertebra. The site of origin was confined to the articular processes of the 7th, 6th, 5th and 4th vertebrae. In four of the twenty five fresh specimens it was possible to identify a short, stumpy origin from the articular processes of the 3rd cervical vertebra. In all cases the thick tendinous origin fused with the posterolateral aspect of the articular capsule of the zygapophyseal joint (Figure 4.10, 4.11). This observation has clinical importance and has not been reported in the literature. The superficial fibres were orientated in a rostral direction, running medially towards the spinous process of the proximal vertebra. In all the specimens examined, the superficial fibres arising from the 7th cervical vertebra, were invariably inserted into the spinous process and lamina of the 6th vertebra. In fourteen of the twenty five fresh specimens, the superficial fibres arising from the 6th cervical vertebra were inserted in a similar manner to the 5th cervical vertebra. However, with the rest of the specimen, the upper most fibres of the superficial fibres joined with those arising from the 5th and 4th cervical vertebrae thus, forming a conjoint tendon inserted into the spinous process of the 2nd cervical vertebra. As these superficial fibres traversed proximally, the deeper fibres found their attachment in the dorsal surface of the laminae of the proximal vertebrae.

4.3.6 Capsular structures

The capsule of the zygapophyseal joints in the cervical region appeared very thin, in contradiction to the observations of Johnson et al. (1975). Unlike its description in the literature, the capsule of the above joints were neither loosely arranged nor voluminous (Williams et al, 1989).

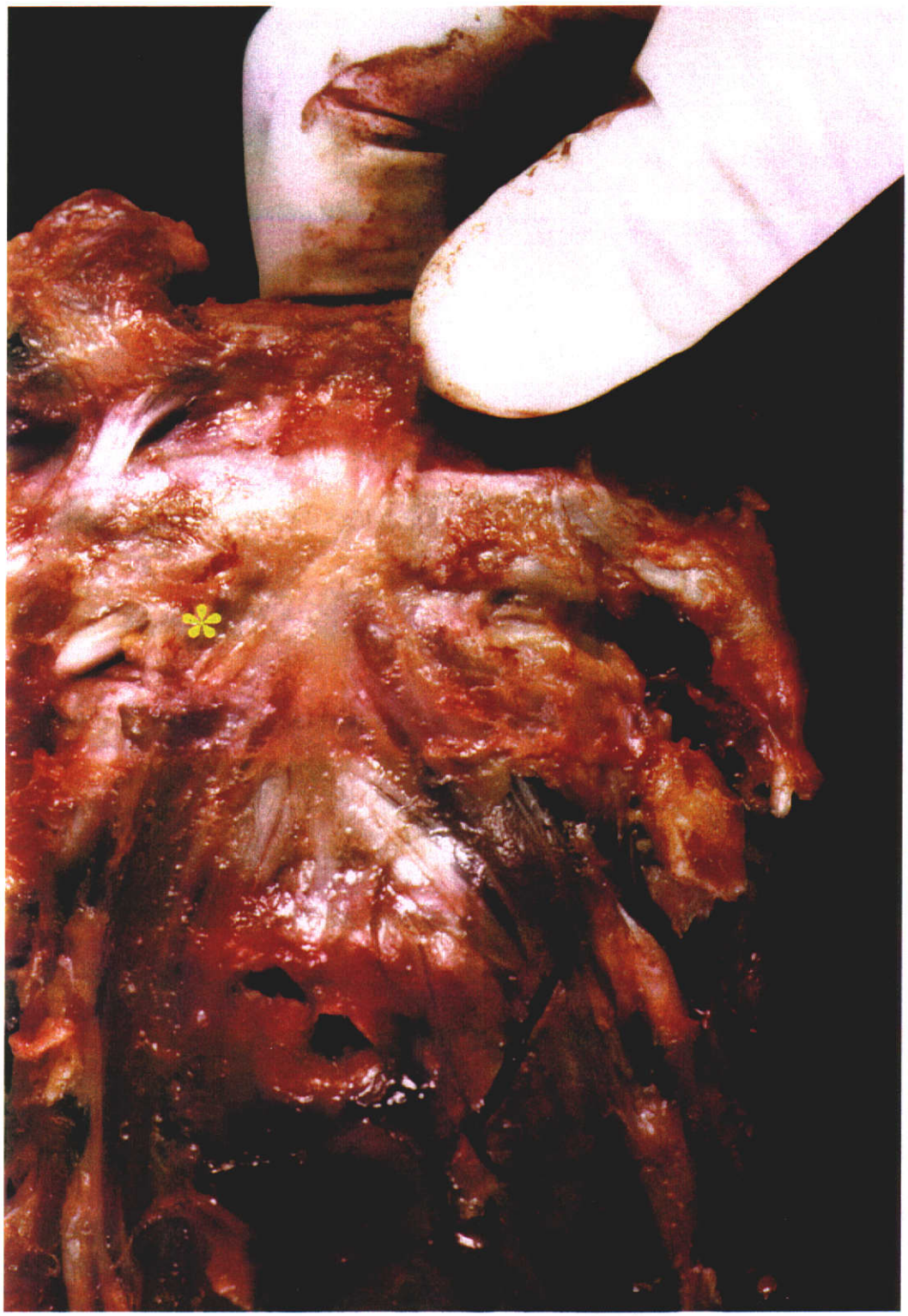

Figure 4.12 Thin articular capsule of the atlanto-axial joints.

In all the specimens, the capsule was reinforced dorsally and laterally by the tendinous origin as it is found in the rotator cuff of the gleno-humeral joint, whereas the posteromedial aspect of the capsule appeared thin and failed to receive any contribution from the multifidus.

Anteriorly, the articular capsule of the atlanto-axial joints and the atlanto-occipital joint were extremely thin and this observation is in contradiction with the literature (Johnson et al., 1975) (Figure 4.12). In the case of the atlanto-occipital joint, the ventral portion of the articular capsule received reinforcement from the conjoint tendon of the superior oblique fibres of the longus colli. The tendons from either sides of the spine united to be inserted over and around the tubercle present in the anterior surface of the arch of the atlas and from there on continued proximally. The central part of the articular capsule was thickened by this prolongation. When the atanto-occipital joint was passively extended, it was possible to see the increase in tension being transmitted to the conjoint tendon of the longus colli through the thickened portion of the capsule. The anterior surface of the atlanto-axial articular capsule was also found to be thin and weak in appearance. This information has not been reported before.

4.4 Discussion

A search of the literature has shown that the morphology of the longus capitis differed considerably from the observation made in this study (Gray, 1858; Sharpey et al., 1878; Sinclair, 1981; Warwick and Williams, 1973; Last, 1978; Williams et al., 1989). Surprisingly, during the last 137 years since the first edition of the text book, currently published as Gray's Anatomy (Figure 4.13, 4.14), there has been very little change in the morphological description as well as the action of this muscle.

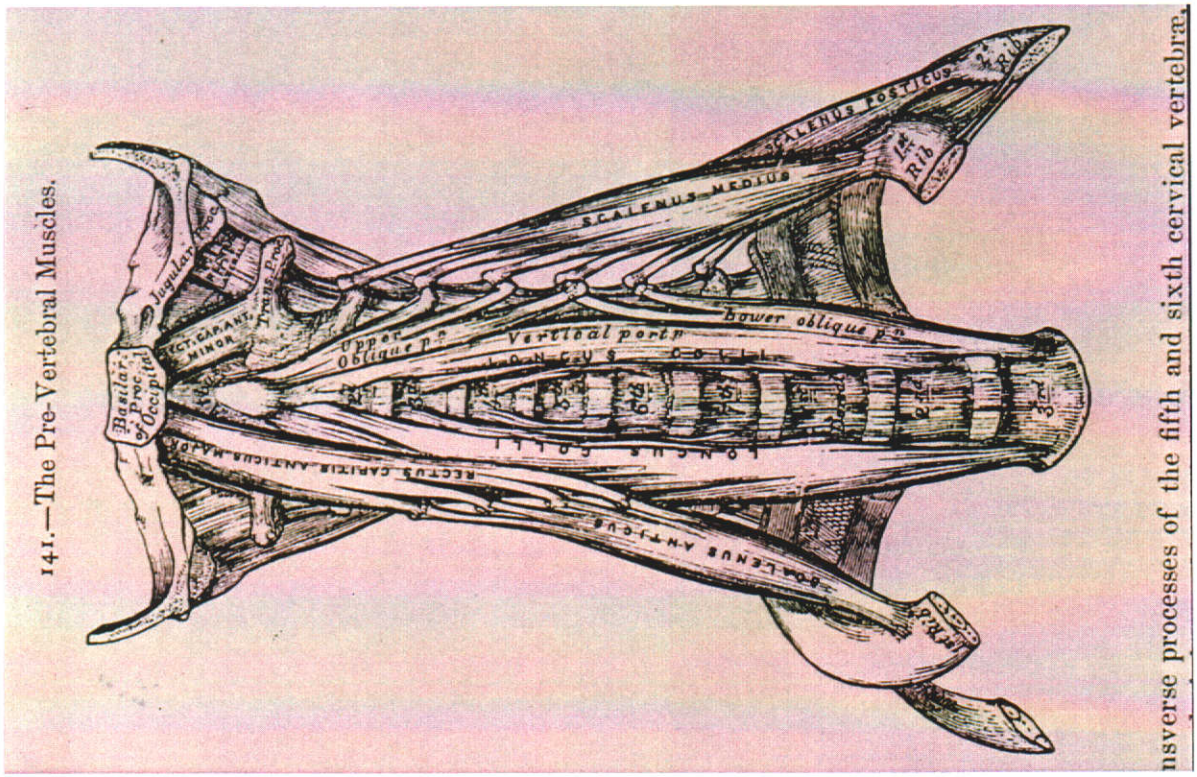

Figure 4.13 **Diagram showing longus capitis muscle (labelled as Rectus capitis anticus) by Henry Gray (1858).**

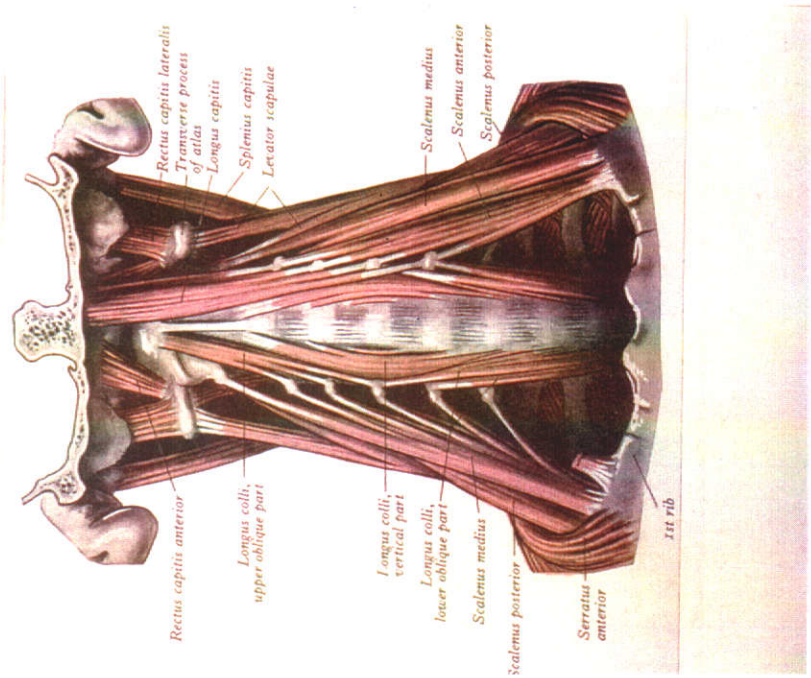

Figure 4.14 **Diagram showing longus capitis muscle in Gray's Anatomy 35th Edition.**

This has largely gone unnoticed until this report. According to Cunningham's text Book of Anatomy, the longus capitis is a thin muscle which, " Travels from the transverse processes of the middle third or fourth cervical vertebrae to the basilar part of the occipital bone lateral to the pharyngeal tubercle" (Sinclair, 1981; Page 281). Last (1978) described the longus capitis muscle as a weak muscle. According to the literature, the longus capitis, sometimes referred as the rectus capitis anticus major, is considered to flex the head (Gray, 1858; Sharpey et al., 1878; Sinclair, 1981; Warwick and Williams, 1973; Last, 1978; Williams et al., 1989). It is said that the size of a muscle is an indicator of its strength which in turn reflects its functional significance (Adams, 1975). Many of the morphological details reported from this study have clearly shown that by virtue of the thickness, length and bony attachments the longus capitis is the strongest prevertebral deep muscle in the cervical region. It is worth noting that there has been no record of either verification of the action of this muscle as mentioned in the literature, or any postulation as to its functional role. On the contrary, longus colli muscle, formed by a thin group of fasciculi, has been investigated (Fountain et.al., 1966; Vitti et.al., 1973). Owing to its proximity to the vertebral column, the longus colli is generally regarded as an important muscle and this could partly explain why it has received attention in comparison to that of the longus capitis. The location of the longus capitis, its site of attachment and eventual lever arm in relation to the instantaneous axis of rotation of the atlanto-occipital joint, suggest that this muscle cannot be a prime mover. There is enough evidence to support an entirely new and significant functional role for this paired muscle. The orientation and the attachments of the longus capitis is well suited to facilitate the kinematics of the cranio-vertebral joints and to play an important biomechanical role in the cervical spine. Werne (1957) suggested that the cranio-vertebral joints should be regarded as a single

functional unit in terms of kinematics. These joints are the single most important section of the spine in achieving movements through the cardinal planes, in order to derive the maximum benefit from the ocular, auditory and proprioceptor organs contained in the skull and the upper spine. As in all synovial joints, the cranio-vertebral joints must have conjunct rotation associated with their "physiological" movements (Warwick and Williams, 1973). This inevitable movement of conjunct rotation is brought about by a combination of the geometry of the articular surfaces, static stabilisers such as the ligaments and capsular structures and the dynamic muscular structures (Warwick and Williams, 1973; Williams et al., 1989). The longus capitis is well placed to fulfil this role in the cranio-vertebral joints. A motion study conducted as part of the dissection study has shown that during rotation of the atlanto-axial joints, the longus capitis muscle of the contralateral side tightened while the ipsilateral muscle slackened. Even though the alar ligaments and the transverse ligament act as static stabilisers, there is no prevertebral muscle other than longus capitis, capable of acting as a dynamic stabiliser. In the atlanto-occipital joint, this muscle is capable of playing a similar role during flexion and extension movements. The anterior part of the capsular structures of the cranio-vertebral joints do not have sufficient strength to withstand the shear forces to which they are constantly subjected to. It is apparent that by it's synergic action, the longus capitis is capable of providing mechanical protection to these capsular structures.

The cervical spine is constantly subjected to axial loading of varying magnitudes (Panjabi et.al., 1989). If the spinal column is considered as a curved beam, then it would be subjected to resultant forces as seen in similar mechanical structures. When a curved beam is subjected to axial loading, the concave surface will be subjected to compressive forces,

whereas the convex surface will experience tensile forces. Among these two forces, the tensile force is regarded as the most destructive in nature (Pauwels, 1980). As it is seen in structural engineering designs, throughout the animal kingdom, the skeletal system has appropriate counter measures to prevent any damage to the bony architecture from such tensile forces. As an example in the human skeleton, the tensor fascia lata together with the ilio-tibial band provides such a protective mechanism against the tensile forces created on the lateral surface of the shaft of femur, when the femur is subjected to axial loading (Pauwels, 1980).

The cervical spine is subjected to considerable axial loading which is a product of the weight of the head, the forces transmitted to the cervical spine through the attachments of the shoulder girdle muscles and the pre- and post-vertebral deep muscles of the neck region. The passive structures such as the anterior longitudinal ligament and the anulus fibrosi in the anterior part of the motion segment alone can not provide adequate counterforce to the tensile force generated in the anterior part of the cervical spine. More over passive structures such as ligaments, capsules and peripheral anulus fibrosi are incapable of intstantly responding to a sudden increase in the magnitude of either the tensile or compressive forces. Therefore it is imperative that an inherently fast acting strong dynamic structure which runs the entire length of the cervical spine, must exist in the prevertebral region of the cervical spine to fulfil this role. This study has clearly shown that the longus capitis is the only structure capable of providing such an important mechanical role. The longus colli will also assist the longus capitis muscle in this situation.

The multifidus group of muscles are said to be capable of producing extension, lateral flexion and rotation of the cervical spine in conjunction

with, respectively the interspinales and the intertransversarii (Warwick and Williams, 1973). A detailed observation made in this dissection study suggests that the bony attachments and available lever arm render the multifidus incapable of producing any of these actions. Moreover the interspinales and intertransversarii are inconstant in their presence, thin and weak muscle slips of no consequence in contributing any muscle power. The morphology of the multifidus muscle strongly suggests that it is a synergist muscle and particularly well placed to control the translatory type of movements found in the zygapophyseal joints of the cervical spine. Contradictory to the generally held view that the zygapophyseal joints of the cervical spine are all inclined in an angle of 45°, there is a considerable variation between individual vertebrae. The inclination of the articular surfaces gradually increase from rostral to caudal direction (Milne, 1991) thus, the upper motion segments are likely to have a greater degree of translation. The manner in which the multifidus gains attachment to the spinous process of the 2nd and 4th cervical vertebrae and the laminae of the 3rd, 4th, 5th cervical vertebrae may not be a coincidence. It is also evident that the reinforcement offered to the weak capsular structure of the zygapophyseal joints of the cervical spine, is an important factor in preventing damage to the capsules. Owing to the unique morphological arrangement of the multifidus muscle and its role in the kinematics of the cervical spine, it is susceptible to injury when subjected to both low velocity and high velocity forces. High velocity injuries sustained in the cervical region are capable of damaging the longus capitis muscle, seen as a prevertebral swelling in a plain x-ray film. Sudden acceleration of the head as evidenced in rear-end collisions may cause trauma to the tendinous origin of the semispinalis capitis muscle, causing pain related symptoms and this could explain interscapular pain. Similarly, when the semispinalis capitis is in a state of muscle spasm induced by trauma, may also

strangulate the greater occipital nerve and cause occipital headache (Figure 4.15).

It is also evident that the researchers in the field of biomechanics and anthropometric study have largely relied on data derived from the existing information readily available in the anatomical literature (Hodgson and Thomas, 1980; Huelke and Nusholtz,1986; Deng and Goldsmith, 1987a; Deng and Goldsmith, 1987b). However, if valid mathematical modelling of the region is to be accomplished, then an accurate account of anatomical details of the spine and its related structures become absolutely necessary. Hence, it could be argued that many of the reported biomechanical ideas based on inaccurate anatomical details, are unlikely to be valid (Figure 4.16).

4.5 Summary

Many observations made during this dissection study have highlighted significant inaccuracies which are present in the literature. An important functional role has been put forward with regard to the longus capitis and multifidus muscles in the cervical region. The detailed morphological information presented from this study will assist in improving the mathematical models used in the field of bioengineering and anthropometric studies as well as assisting radiologist using Magnetic Resonance Imaging techniques. The clinical relevance of the above anatomical structures fully described in this part of the thesis will be highlighted in the discussion chapter.

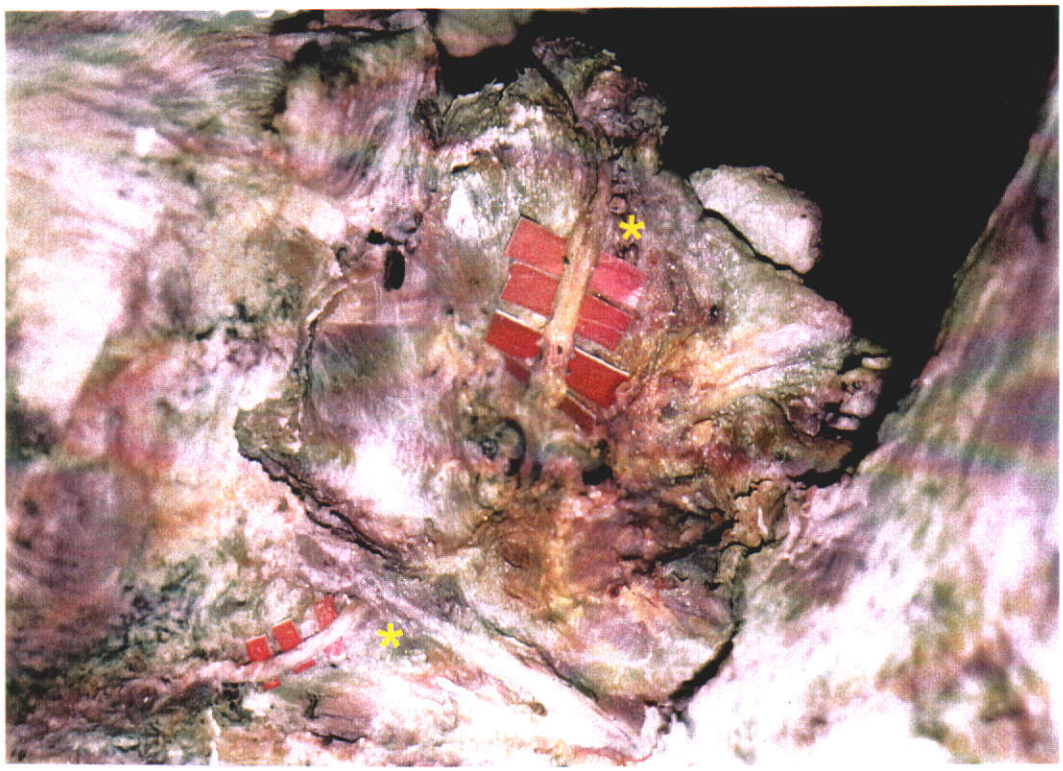

Figure 4.15 Greater occipital nerve entering and exiting through the substance of semispinalis capitis muscle.

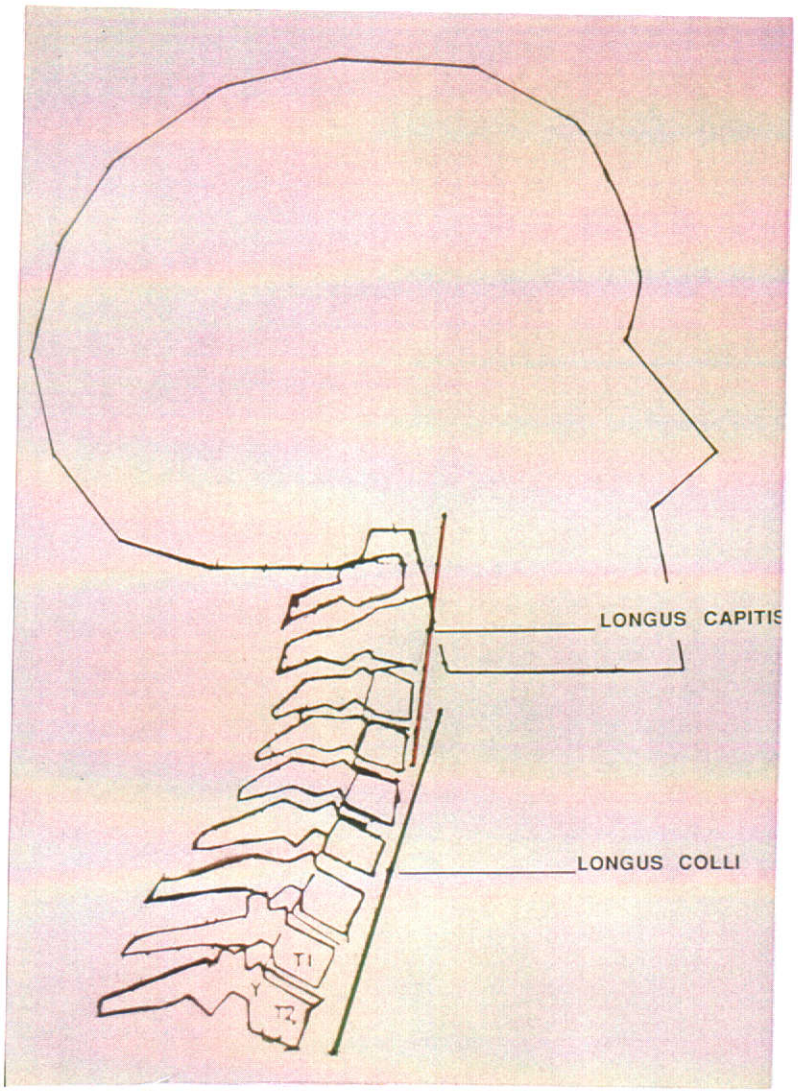

Figure 4.16 **The numerical model of head/neck/upper-torso used by Deng and Goldsmith (1987b, page 488). Original diagram modified to highlight the inaccuracy in the model with reference to longus capitis and longus colli muscles.**

CHAPTER FIVE RESULTS

5.1 Introduction

Series of data analysis were performed to test the hypotheses of this study and the results are presented under the following headings:

5.2 demographic details;

5.3 descriptive statistics related to subjective and objective findings;

Inferential statistics

5.4 pain;

5.5 range of motion;

5.6 muscle strength;

5.7 return to work; and

5.8 prognostic factors.

In addition to standard descriptive data, in a few instances a more detailed account of information pertaining to the experimental and control groups are also provided. The significance of this information will be discussed in relevant sections. A number of independent variables and related descriptive data have been included for the following reasons:

1. to establish the prognostic value;
2. to verify previous reports; and
3. to describe potential confounding factors.

5.2.1 Age

The minimum and maximum age of the study population were respectively 13 and 75 years. The sample mean was 32.69 with a standard deviation of 12.39. The number of subjects in each of the age categories viz "up to 30

years", "31 to 40 years", "41 to 50 years" and "51 years and above" constituting the study population as a whole and the experimental groups viz immobilisation group (group-A), active exercise group (group-B) and the control group (group-C) is provided in Figure 5.2.1. A breakdown of similar frequencies for each of the follow-up time periods (i.e 4 weeks, 6 weeks, 12 weeks, 24 weeks and 52 weeks) are expressed in the form of percentages as shown in the Table 5.2.1.

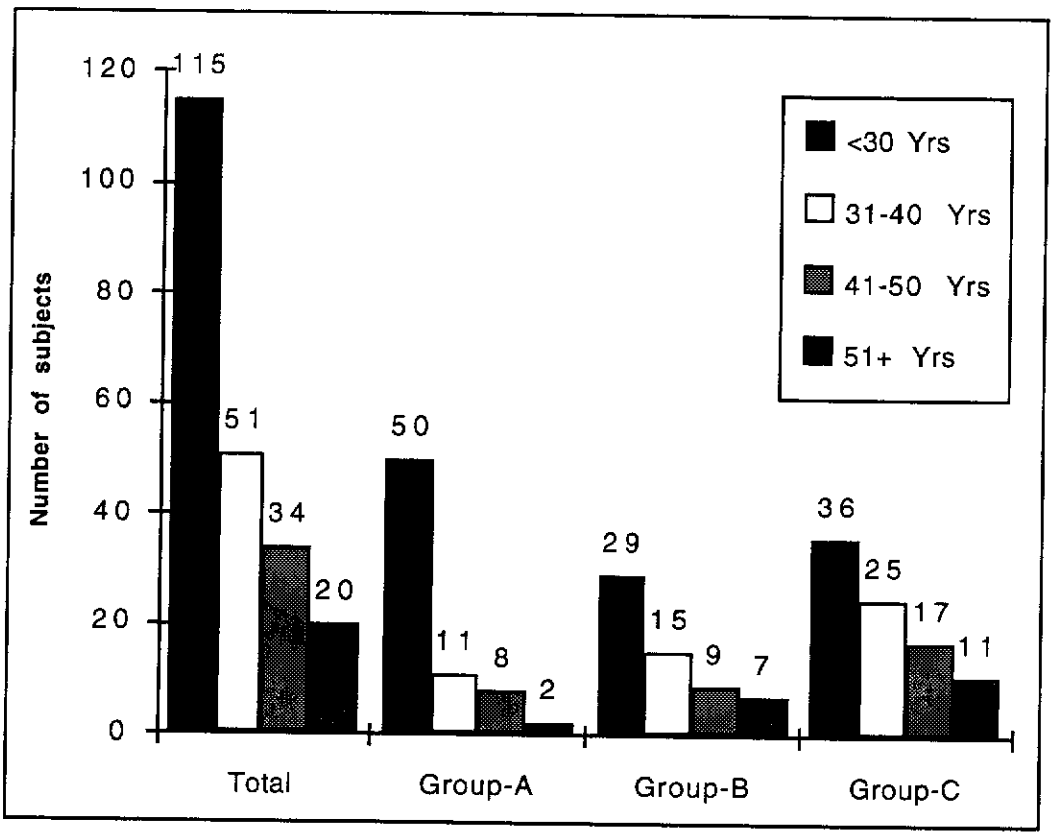

Figure 5.2.1 **Histogram showing the frequency distribution of the total sample (n=220) and individual random groups: Immobilised (Group A, n=71), Active exercise (Group B, n=60), Control group (Group C, n=89) by their age categories. (Time period = Initial examination).**

Among the total study population, 52.3% were in the "up to 30 years" age category followed by "31-40 years" (23.2%), "41-50 years" (15.5%) and "51 years and above" (9%) categories. The characteristics of the age distribution between the active exercise group and the control group were

some what similar, whereas the immobilisation group differed considerably when compared to the rest of the groups with more subjects in the under 30 years group (Table 5.2.1). A similar pattern was observed at each of the follow-up examination time periods (Table 5.2.1).

Table 5.2.1 **The percentage of age categories in immobilised (Gp-A), active exercise (Gp-B) and control group (Gp-C).**

Time periods	Groups	n	<30 yrs	31-40 yrs	41-50 yrs	51+ yrs
Initial examination	A	71	70.4%	15.5%	11.3%	2.8%
	B	60	48.3%	25.0%	15.0%	11.7%
	C	89	40.4%	28.1%	19.1%	12.4%
4 weeks	A	59	76.3%	13.6%	10.1%	0%
	B	44	43.2%	27.3%	13.6%	15.9%
	C	59	35.6%	28.8%	20.3%	15.3%
6 weeks	A	60	70.0%	16.7%	10.0%	3.3%
	B	37	37.8%	27.0%	16.2%	18.9%
	C	54	37.0%	25.9%	18.5%	18.5%
12 weeks	A	57	71.9%	14.0%	10.5%	3.5%
	B	42	42.9%	23.8%	16.7%	16.7%
	C	50	34.0%	30.0%	22.0%	14.0%
24 weeks	A	48	70.8%	12.5%	14.6%	2.1%
	B	29	27.6%	27.6%	20.7%	24.1%
	C	45	33.3%	31.1%	17.8%	17.8%
52 weeks	A	42	66.7%	14.3%	16.7%	2.4%
	B	28	28.6%	25.0%	25.0%	21.4%
	C	45	35.6%	31.1%	20.0%	13.3%

5.2.2 Gender

The study population was made up of 145 female and 75 male subjects (Figure 5.2.2). The immobilised group consisted of 46 females and 25 males, whereas 40 females and 20 males were present in the active exercise group. The control group had 59 females and 30 males. At the

initial examination, the overall ratio between female and male subjects in this study was 2:1 and a similar pattern was evident in all three randomised groups. A graphical comparison of the frequency of female and male subjects of experimental groups viz immobilisation group (group-A), active exercise group (group-B) and the control group (group-C) is provided in Figure 5.2.2 and Figure 5.2.3.

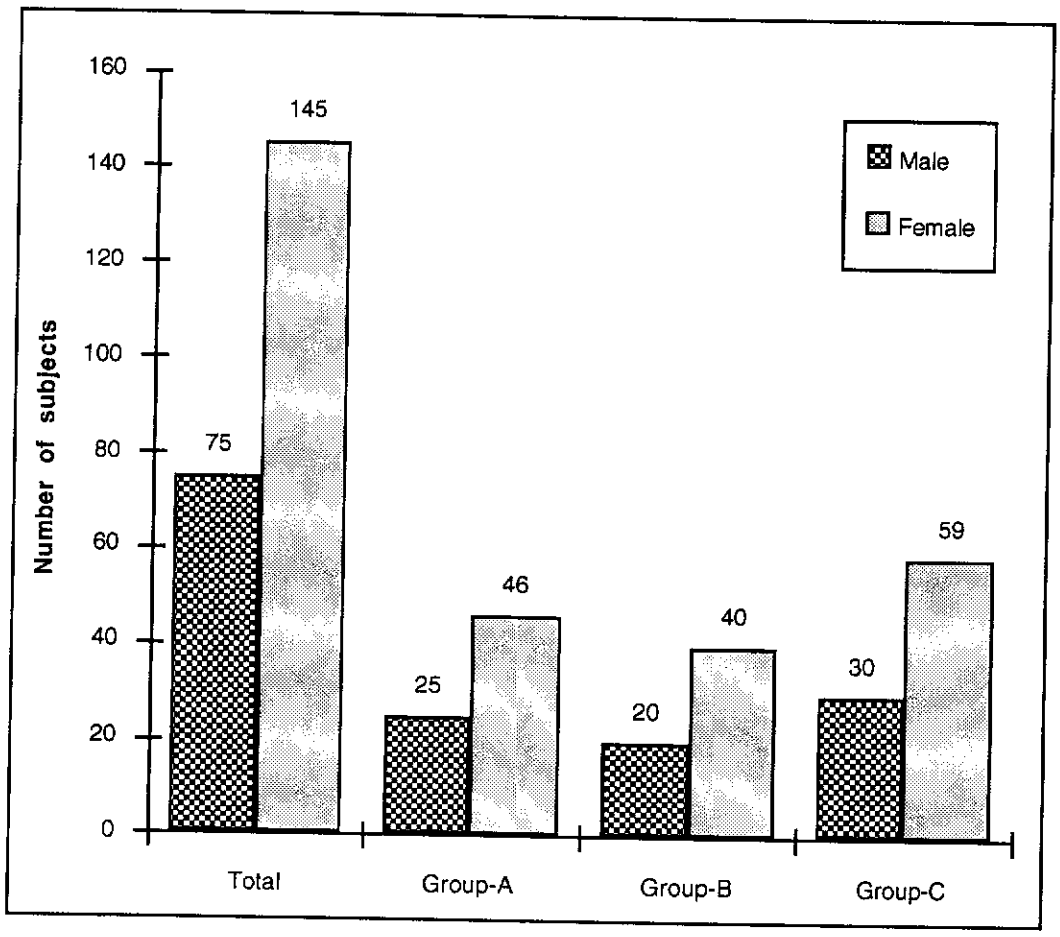

Figure 5.2.2 **Histograms showing the frequency distribution of male and female subjects in immobilised group (group-A), active exercise group (group-B), and control group (group-C) (Time period = Initial examination).**

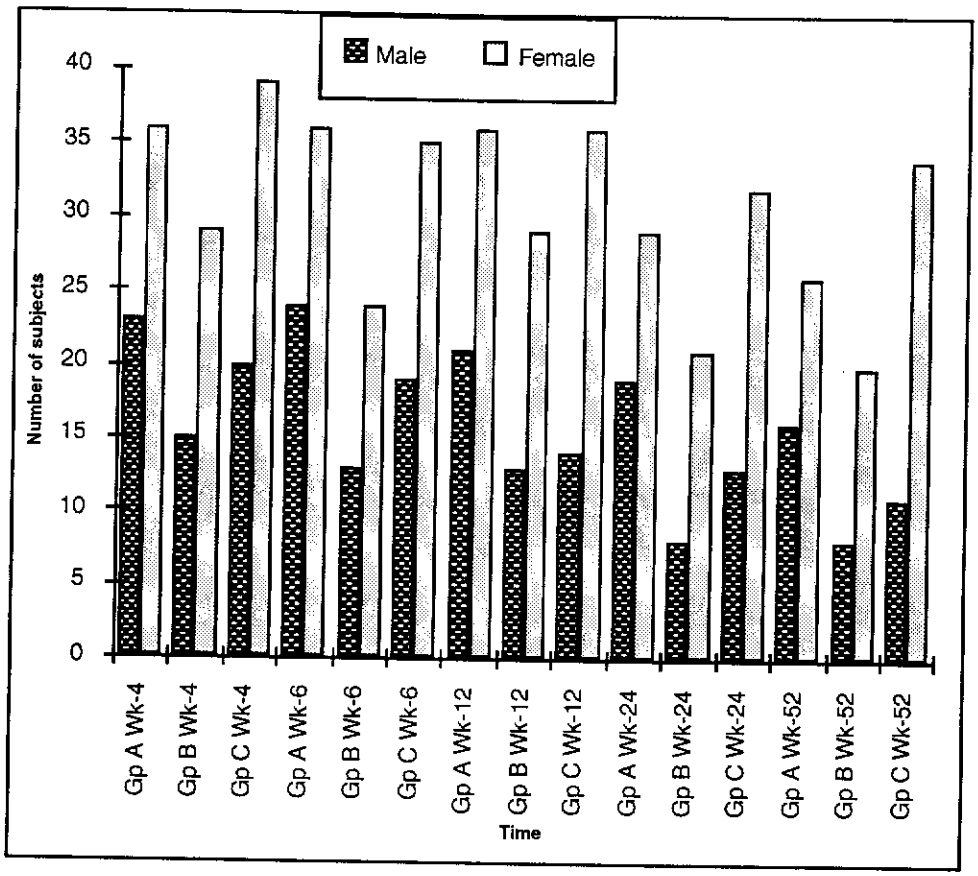

Figure 5.2.3 **Histograms showing a comparison of male and female subjects in each of the treatment groups during the study (Gp A = immobilised group; Gp B = active exercise group; Gp C = control group).**

5.2.3 Occupation

The sample consisted of 18 unemployed subjects, 26 females reported to be on home duties, 25 full time students, 3 part time students, 2 retired pensioners and the rest engaged in a variety of occupations (146). The frequencies of most of the occupations were only one or two in number and the type of occupations with a frequency of three and above are presented in the Table 5.2.2.

Table 5.2.2 **The most common type of occupations in a descending order.**

TYPES OF OCCUPATION	FREQUENCIES	PERCENTAGE
RECEPTIONIST/TELEPHONIST	8	3.6%
TRAFFIC POLICE OFFICER	7	3.2%
STOREMAN	6	2.7%
COMPUTER OPERATOR	6	2.7%
SECRETARY	6	2.7%
CLERK	5	2.3%
TELEPHONIST	4	1.8%
BANK TELLER	4	1.8%
SHOP ASSISTANT	4	1.8%
LABOURER	4	1.8%
SALES REP	3	1.4%
CATERING SUPERVISOR	3	1.4%
WARD ASSISTANT	3	1.4%
NURSE	3	1.4%

5.2.4 Types of collisions

Even though there were several types of collisions, the rear-end (57) and frontal (52) types were more prevalent when compared to the others. The second largest incidence included pile-up (30), side impact (25), head on collision (24) and roll-over (7). There were other categories in which only a section of the subject's vehicle sustained the impact. Figure 5.2.4 illustrates the above information.

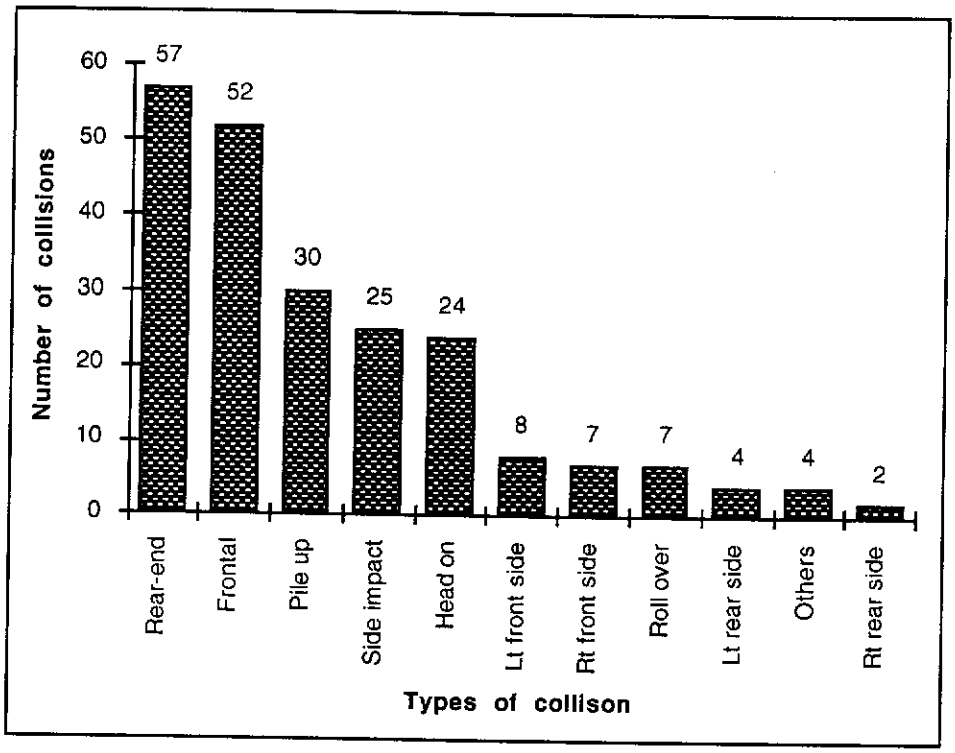

Figure 5.2.4 **Histograms showing the frequencies for the types of collision (n=220).**

5.2.5 Types of vehicles

The type of vehicles involved and the estimated speed at the time of collision (i.e mass and velocity) has a strong association with the resulting momentum and the severity of the injury. Hence the following details have been presented. The analysis showed that at the time of the impact, the subjects were either driving or seated in 16 types of vehicles ranging from small sedan to medium sized truck. When the vehicles were ranked according to the frequencies, 72 (32.7%) subjects were in medium sized sedans, followed by big sedans (38, i.e 17.3%), medium sized hatch back (34, i.e 15.5%) and small hatch backs (24, i.e 10.9%). The frequencies of the remainder of the motor vehicle types were ranging between 16 and 1 (Table 5.2.3). A similar analysis of the other vehicles / objects involved in the accidents showed that the highest frequencies were attributed to big sedans (64, i.e 29.1%) followed by medium sized sedans (36, i.e 16.4%)

and medium sized hatch backs (19, i.e 8.6%). In 21 (9.5%) instances the subject's vehicles collided with either trees or lamp posts and in the case of one subject, a large kangaroo was involved. The remaining vehicles are presented in the Table 5.2.3.

Table 5.2.3 Frequency distributions of types of vehicles / objects involved.

Type of vehicle / object	Subject's	Other
Small sedan	16	8
Small hatch	24	14
Medium sedan	72	36
Medium hatch	34	19
Medium station wagon	15	9
Big sedan	38	64
Big station wagon	4	5
Mini van	1	3
Mini bus	1	4
UTE	1	10
Small truck	1	3
Medium truck	2	4
4 WD	4	10
Motor bike	3	0
Bus	0	3
Large truck	0	6
Tree, Post	-	21
Kangaroo	-	1

5.2.6 Speed of the vehicles involved

The estimated speed of the subject's vehicles ranged from 0 kph to 100 kph with a mean of 29.7 kph and a standard deviation of 27.5. In the case of the other vehicles involved the estimated minimum and maximum of speeds were respectively 0 kph and 100 kph and the mean and standard deviation were 44.2 kph and 28.04. It was noted that 75 (34.1%) subjects were the occupants of stationary vehicles, whereas 39 subjects (17.7%) were in a vehicle travelling at a speed of 60 kph. The next highest frequency was for those subjects seated in a vehicle moving at a speed of 50 kph which was 25 (17.7 %). The remaining subjects fell into groups with a frequency of 11

and less (Table 5.2.4). In the "other vehicles" category, 54 (24.5 %) vehicles were reported to be travelling at a speed of 60 kph followed by 25 vehicles (11.4 %) in the group of 50 kph and 70 kph of speed. The frequency distributions for the rest of the vehicles are listed in Table 5.2.4.

Table 5.2.4 Frequency distribution of speed of the vehicles involved in the accident.

Speed in kph	Subject's vehicle	%	Other vehicle	%
0-4	75	34.1	15	16.8
5-9	11	5.0	6	2.7
10-14	11	5.0	7	3.2
15-19	3	1.4	4	1.8
20-24	4	1.8	9	4.1
25-29	3	1.4	0	0
30-34	10	4.5	16	7.3
40-44	8	3.6	6	2.7
45-49	9	4.1	1	0.5
50-54	25	11.4	25	11.4
55-59	2	0.9	1	0.5
60-64	39	17.7	54	24.5
65-69	10	4.5	3	1.4
70-74	5	2.3	25	11.4
75-79	1	0.5	1	0.5
80-89	2	0.9	20	9.1
90-99	0	0	4	1.8
100+	2	0.9	1	0.5

One hundred and ninety seven subjects encountered high momentum forces associated with their motor vehicle accident, while the remaining 23 were considered to be in the low momentum category.

5.2.7 Subject's seating position

At the time of the impact, 165 of the 220 subjects were in the driving seat, 42 passengers were seated in the passenger's front seat with 7 subjects in

the left rear seat, 4 subjects in the right rear seat while the remaining 2 were seated in the middle rear seat of their respective vehicles (Table 5.2.5).

Table 5.2.5 Seating positions of the subjects a the time of impact.

Seating positions	Number of subjects	%
Driver's seat	165	75.0
Passenger-Front	42	19.1
Rear seat -Left	7	3.2
Rear seat-Right	4	1.8
Rear seat-Middle	2	0.9

5.2.8 Seat belt and head rest

Two hundred and fifteen subjects were wearing their seat belt at the time of the collision, of which 211 were lap / sash type and the remaining 4 were lap type seat belts. One hundred and ninety eight of the 220 subjects reported that their vehicles were fitted with head rests and among this group only 193 had the benefit of adequately placed head rests which were capable of providing effective protection from hyper-extension of the cervical spine.

5.2.9 Past history of "whiplash" type of injury

Only 6 of the 220 subjects reported to have had a previous "whiplash" type of injury and 2 subjects from this group of 6 sustained their injuries 4 years prior to the current injury and claimed to have been symptom free a few months after their initial MVA. Similarly, small groups of subjects consisting of 2, 1 and 1 in numbers reported to have been treated for "whiplash" injury, sustained respectively 7 years, 6 years and 19 years prior to their current injury.

5.2.10 Litigation

The subjects were grouped into three categories viz:

1. claimants against Motor vehicle insurance trust;
2. claimants under worker's compensation insurance;
3. non-compensable.

Even though the categories 1 and 2 were grouped separately, based on the classification of insurance claims which existed at the time of the accidents, for analytical purposes the categories 1 and 2 were in effect treated as the one group and nominated as the compensable injury group. The study population consisted of 74.1% of the subjects in the compensable injury group, whereas the remaining 25.9% of the subjects were in the non-compensable category (Table 5.2.6).

Table 5.2.6 Frequency distribution of compensable and non-compensable accidents.

Accident Status	No of Subjects	Percentage
Motor Vehicle Insurance Trust	107	48.6 %
Worker's Compensation Insurance	56	25.5 %
Non-compensable	57	25.9 %

5.3 Signs and symptoms of injury

In this section, the descriptive and where ever appropriate, relevant inferential statistics for the following are presented:

1. paraesthesia;
2. neurological examinations;
3. eye symptoms;
4. clumsiness;
5. radiological findings;
6. magnetic resonance imaging;
7. intensity of "Perceived pain" reported by the subjects;
8. pre- and post-vertebral muscle peak strength;
9. range of motion of the cervical spine;

10. return to work; and

12. other matters of interest.

Paraesthesia:

At the time of the initial examination, 48 of the total 220 subjects complained of paraesthesia and the highest reported incidences were related to the tips of the digits in the left and right hands (29 and 24 subjects respectively). Thereafter, C-8 left (5-subjects) and C-8 right (4-subjects) dermatomes were most affected. There was a single incidence of paraesthesia in the region of the left side of the face supplied by the trigeminal nerve. It was also interesting to note that L5 (5-subjects) and S1(3-subject) dermatomes were also reported to be associated with paraesthesia. The frequency for the remaining dermatomes are listed in the Table A.III.3.1. The results showed that two thirds of the subjects in the current study affected with paraesthesia recovered within four weeks after their injury (Table A.III.3.1). The only subject who complained of paraesthesia over the trigeminal nerve distribution during the initial examination, was free of this symptom after 6 weeks following MVA. The follow-up examinations at week-6 and week-12 post-injury showed that 13 and 11 subjects (respectively), continued to be affected with paraesthesia. Similarly, the results indicated that 7 and 8 subjects continued to experience paraesthesia as evidenced at the follow-up examinations performed at 24 and 52 weeks post-injury (respectively). The frequencies of this symptom for the three treatment groups are listed in the Tables A.III.3.2 to A.III.3.4.

"Bizarre" symptoms:

Among the subjective symptoms the incidence of nausea was the largest (24.1%) and thereafter followed by difficulty in focusing (17.3%), blurred vision (16.8%), and retro-orbital pain (13.2%). At the end of the study period, the blurred vision and difficulty in focusing persisted among 6% and

5.2% of the subjects. During the follow-up examination at week-4, 16% of the subjects complained of difficulty in concentrating in the task that was performed and similarly 10.5% of the subjects described that they noticed their hand dexterity were affected (clumsiness). The frequency distribution for these symptoms corresponding to each of the time periods are presented in the Tables A.III.3.5 to A.III.3.15.

Neurological examination findings:

The neurological examination conducted during the initial examination demonstrated that 18 of 220 subjects have had altered sensation and subsequent follow-up examinations showed them to have reached normality. The type of sensory deficits recorded during the initial examination are listed in the Table 5. 3.1. The motor power, pupil reaction to light, and vibratory sensation were in tact in all the subjects. The tendon reflexes were normal in all subjects except two who have had depressed biceps tendon reflex and subsequent examination at week 4 demonstrated that these subjects had recovered to normality. None of the subjects had signs indicative of positive Horner's syndrome.

Table 5.3.1 **Frequencies for neurological examination findings with reference to initial examination for the total population and treatment groups (n = number of subjects with respective findings- Codes listed below the table).**

Dermatomes-Total sample	Initial examination	Dermatomesim mobilised group	Base-line	Dermatomes Active Exercise group	Base-line	Dermatomes-Control group	Base-line
C-2-Right	0 (n=220)	C-2-Right	0 (n=71)	C-2-Right	0 (n=60)	C-2-Right	0 (n=89)
C-2-Left	0 (n=220)	C-2-Left	0 (n=71)	C-2-Left	0 (n=60)	C-2-Left	0 (n=89)
C-3-Right	0(n=220)	C-3-Right	0(n=71)	C-3-Right	0(n=60)	C-3-Right	0(n=89)
C-3-Left	0 (n=220)	C-3-Left	0 (n=71)	C-3-Left	0 (n=60)	C-3-Left	0 (n=89)
C-4-Right	3 (n=1)	C-4-Right	3 (n=1)	C-4-Right	0 (n=60)	C-4-Right	0 (n=89)
C-4-Left	4 (n=1)	C-4-Left	4 (n=1)	C-4-Left	0 (n=60)	C-4-Left	0 (n=89)
C-5-Right	3 (n=1)	C-5-Right	3 (n=1)	C-5-Right	0 (n=60)	C-5-Right	0 (n=89)
C-5-Left	5 (n=3)	C-5-Left	5 (n=2)	C-5-Left	0 (n=60)	C-5-Left	5 (n=1)
C-6-Right	3 (n=1)	C-6-Right	3 (n=1)	C-6-Right	0 (n=60)	C-6-Right	0 (n=89)
C-6-Left	5 (n=2)	C-6-Left	5 (n=1)	C-6-Left	0 (n=60)	C-6-Left	5 (n=1)
C-7-Right	0 (n=220)	C-7-Right	0 (n=71)	C-7-Right	0 (n=60)	C-7-Right	0 (n=89)
C-7-Left	5 (n=2)	C-7-Left	5 (n=1)	C-7-Left	0 (n=60)	C-7-Left	5 (n=1)
C-8-Right	1 (n=1)	C-8-Right	1 (n=1)	C-8-Right	0 (n=60)	C-8-Right	0 (n=89)
C-8-Left	1 (n=1) 5 (n=1)	C-8-Left	1 (n=1)	C-8-Left	0 (n=60)	C-8-Left	5 (n=1)
T-1-Right	1 (n=1)	T-1-Right	1 (n=1)	T-1-Right	0 (n=60)	T-1-Right	0 (n=89)
T-1-Left	1 (n=1) 2 (n=1) 5 (n=1)	T-1-Left	1 (n=1) 2 (n=1)	T-1-Left	0 (n=60)	T-1-Left	5 (n=1)

Codes: 0= Normal 1= Hypothesia 2= Hyperalgesia 3= light touch- altered
4= Hot / cold- altered 5= Altered sensation to sharp / blunt sensation testing

Radiology:

The radiographic examinations of the subjects cervical spine demonstrated that 33.2% of the subjects had loss of cervical lordosis and the functional views showed that 45.7% of the subjects had limitation of flexion movement in their necks. The I.V.D degeneration was present in 14.5% of the study population. It was also evident that 7.5% of the subjects had widening of the intervertebral body space and instability of the motion segment was reported to be present among 3% of the subjects. Similarly, the normal alignment of the cervical spine was affected among 17 subjects. There was a single instance of pre-vertebral swelling. It should be noted that 21 subjects with mild symptoms declined to have functional views; thus, it was not possible to establish those information obtained from functional views of the cervical spine. The frequencies for the above radiological finding are listed in the Table 5. 3.2 and 5.3.3. A single instance of gas formation in the I.V.D was noted in a radiograph taken within 8 hours post injury (Figure 5.3.1, 5.3.2).

Table 5.3.2 **Frequencies for abnormal radiological findings for the total sample.**

Radiological findings-	Number of subjects affected	%
Loss of Lordosis	73	33.2
Normal Alignment affected	17	8.5
Widening of inter-spinal space	15	7.5
Evidence of instability	6	3.0
Presence of flexion Limitation	91	45.7
Presence of pre-vertebral swelling	1	0.5
Presence of disc degeneration	32	14.5

Table 5.3.3 **Frequencies for abnormal radiological findings for the treatment groups.**

Radiological findings - by group	Immobilised group	Active Exercise group	Control group
Loss of Lordosis	30	10	33
Normal Alignment affected	9	3	5
Widening of inter-spinal space	6	5	4
Instability	2	1	3
Flexion Limitation	37	18	36
Pre-vertebral swelling	1	0	0
Disc degeneration	7	6	19

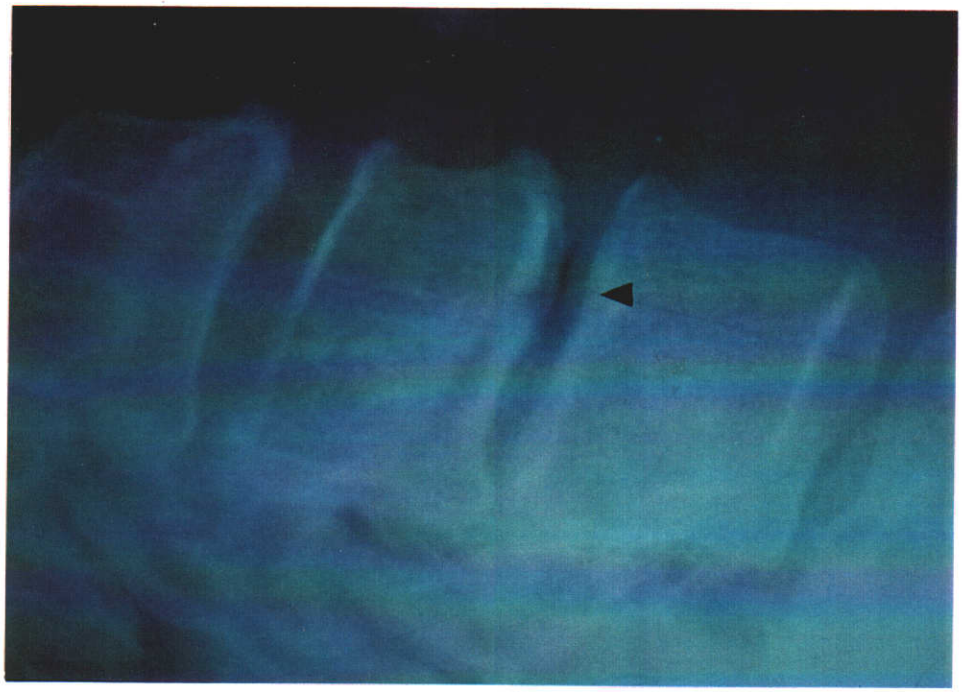

Figure 5.3.1 Radiograph of a subject taken within 8 hours post-injury showing gas formation in the intervertebral disc.

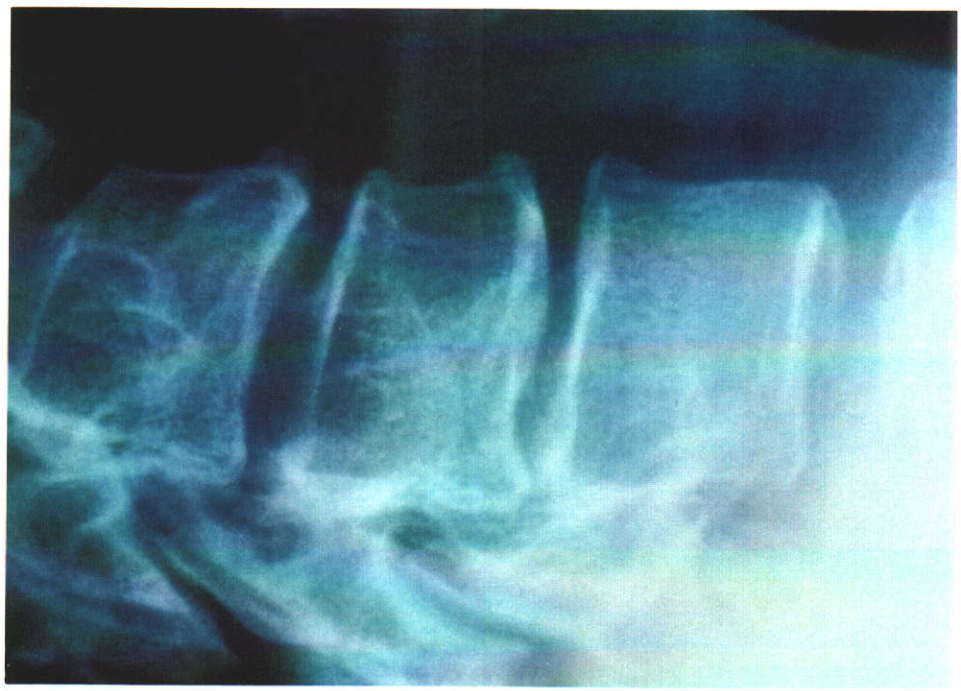

Figure 5.3.2 Radiograph of the same subject taken 24 hours post-injury showing absence of gas in the intervertebral disc.

5.4 The intensity of Perceived pain level reported by the subjects

An initial analysis of the histograms of the means demonstrated a skewed distribution for the total sample as a whole and also in the cases of random groups (Figure A.III.1). For this reason the raw data had to be transformed using natural logarithm in order to remove the skewness and to achieve a normal distribution. Thus, the derived log transformed data has been used through out the statistical analysis.

5.4.1 Transformation

The validity of the inferences derived from parametric statistical procedures, such as t-tests, linear regression and analysis of variance is dependent up on a few prescribed assumptions in relation to the homogeneity of variance and its normality (Battacharyya and Johnson, 1977; Snedecor and Cochran, 1980; Bourke, Dlay, and McGilvary, 1985; Armitage and Berry, 1987; Altman, 1991; Portney and Watkins, 1993). It is not uncommon to encounter a situation in which the data collected may indicate that there has been violation of the assumption with reference to the normality of the population. A deviation from normal distribution may also be due to atypical extreme values exhibited by the outliers resulting in a skewed distribution. In such circumstances, "data transformation" is performed by the application of appropriate mathematical procedures to convert the original variable (X) into a new variable (X') (Armitage and Berry, 1987). The transformation of the original variable X into X', in effect will only change the scale of the measurement (eg. converting Fahrenheit into Centigrade) and does not alter the inherent value of the original data (Portney and Watkins, 1993). There are several approaches to data transformation and the results presented in this study were based on a procedure known as "log transformation" using the natural log. The antilog of the mean of the

transformed variables is referred as "geometric mean", whereas the mean obtained from the raw data is "arithmetic mean" which is commonly referred as "mean". The geometric mean is always less than the arithmetic mean and this characteristic of the geometric mean facilitates to stabilise the variance and brings about a normal distribution to the distribution which is otherwise a log normal distribution (Battacharyya and Johnson, 1977; Snedecor and Cochran, 1980; Bourke, Dlay, and McGilvary, 1985; Armitage and Berry, 1987; Altman, 1991; Portney and Watkins, 1993). Prior to the transformation, for mathematical reasons it was necessary to change the raw pain score 0 as 1. Thus, in this study the validity of the statistical inferences has been ensured using "log transformation".

The histograms of "raw data" and "log transformed" data are presented for a comparison (Figures A.III.1, Fig.A.III.2). In the text section, geometric means and geometric standard deviations have been reported for the study population and respective treatment groups. In addition to this the arithmetic mean (mean) has also been presented in relevant tables. It should be noted that the arithmetic mean has only been reported as a matter of interest and was not used for statistical analysis.

5.4.2 Descriptive statistics

The geometric mean of the "perceived pain" intensity within 24 hours after the MVA was 441.42 with a geometric standard deviation of 1.97, whereas at the time of the initial examination the geometric mean and the geometric standard deviation were respectively 424.11 and 2.09. The maximum and minimum of the pain intensity within 24 hours were respectively 30 and 1670 and similarly for the Initial examination were 30 and 1652. From this it was evident that any change that might have occurred in the intensity of pain between these two time periods is very small.

The geometric mean, geometric standard deviation, minima and maxima for the rest of the time periods are presented in the Table 5.4.1. A comparison of the geometric means showed that there was a noticeable decrease at four weeks after the injury which is indicative of a reduction of pain level among some of the subjects. At the same time period (i.e 4 weeks), the pain level ranged from 0 to 1326 which in turn suggested that a few subjects were totally pain free. This trend was noticed progressively up to the end-point (Table 5.4.1). Conversely, the geometric standard deviation for the succeeding time scales from 4 weeks onwards, was greater than the previous time scale. The geometric standard deviation characterises the variability or dispersion of scores around the mean and in this context demonstrating that the variability between the subjects was gradually increasing (i.e reflecting the progress or lack of it); thus, describing the rate of progress between follow-up time periods. The confidence interval, lower and upper limits for the total sample are presented in Table 5.4.2.

Table 5.4.1 **Descriptive statistics of the study population with reference to time periods(Transformed data).**

Time scale	n	Geometric Mean	Geometric S.D	Arithmetic mean	Minimum	Maximum
Within 24 hours	220	441.42	1.97	537.12	30	1670.0
Initial examination	220	423.69	2.09	528.07	30	1652.0
4-Weeks	162	207.68	3.68	333.01	1	1326.0
6-Weeks	151	113.41	6.20	257.28	1	1097.0
12-Weeks	149	59.38	9.32	202.04	1	1023.0
24-Weeks	122	37.56	11.91	173.41	1	1188.0
52-Weeks	115	25.71	13.64	154.16	1	728.0

Table 5.4.2 **Confidence intervals for the study population with reference to time periods (Transformed data).**

Time scale-	n	Geometric Mean	Geometric S.D	Geometric lower limit	Geometric upper limit
Within 24 hours	220	441.42	1.97	403.59	482.75
Initial examination	220	423.69	2.09	384.27	467.15
4-Weeks	162	207.68	3.68	169.95	253.79
6-Weeks	151	113.41	6.20	84.77	151.73
12-Weeks	149	59.38	9.32	41.49	84.98
24-Weeks	122	37.56	11.91	24.20	58.29
52-Weeks	115	25.71	13.64	15.90	41.57

The descriptive statistics for the random groups are presented in the Tables A.III.4.1 to A.III.4.6. A comparison of the geometric means of the random groups corresponding to the initial examination showed that the immobilised group (548.95) was greater than the rest of the groups and similarly the geometric mean of the control group (412.82) was greater than the active exercise group (324.1). It was also evident from a comparison of the geometric means and respective standard deviations that a larger proportion of the subjects in the immobilised group have reported a higher intensity of pain. The tabulated descriptive statistics showed a very interesting change occurring at 4 weeks post injury (Tables A.III.4.1, A.III.4.3 and A.III.4.5). The geometric mean of the immobilised group considerably decreased from 548.95 to 134.56 showing a change of 75.5% from the initial examination, whereas the active exercise and control groups exhibited a change of 34.9% and 23.2% respectively. The above changes were also reflected in the range of minima and maxima for each of the groups. A similar pattern was evidenced at successive time periods. There

were differences between the female and male subjects and the geometric means for the gender groups with respect to the relevant follow-up time periods are presented in the Table A.III.4.7. An examination of the geometric means for the male and female subjects indicated that the geometric means for the latter were consistently larger than the former.

5.4.3 Areas of pain

Total sample:

Information related to areas of pain (areas that were most affected) is of considerable interest to clinicians. Therefore a comprehensive frequency distribution of areas of pain as shown in the body chart and the percentage of subjects in the entire sample and individual random groups have been reported in the Tables 5.4.3, A.III.4.8 to A.III.4.10. The details contained in these tables do not convey any information with reference to the intensity of pain. The areas in the initial examination (IE) are not ranked and this break of convention is for the convenience of comparing left and right sides. There was no noticeable difference in the percentage of subjects for each of the areas reported to be painful within 24 hours after the MVA and during the initial examination. The areas 11 (back of the neck-right side) and 10 (back of the neck- left side) shown in the body chart were reported to be painful by 83.2% and 82.7% of the subjects followed by trapezius middle fibres-right (70.5%) and trapezius middle fibres-left (64.1%). The frontal headache (right fore-head-60.5% and left fore-head-58.6%) and trapezius upper fibres (right and left sides 50.5% respectively) were ranked next. The occipital headache and interscapular pain were reported by 29.5% of the subjects. The pre-vertebral region was reported to be painful by 25% of the subjects and succeeded by upper part of the right and left sternomastoid muscle (19.5% and 19.1% respectively).

Table 5.4.3 **The Frequency distribution of the areas of pain for the study population (IE= initial examination).**

AREA OF PAIN-t	% IE	% 4WKS	% 6WKS	% 12WKS	% 24WKS	% 52 WKS
Headache-(RT-Fore head)	60.5	48.7	40.8	32.3	33.7	36.1
Headache-(LT-Forehead)	58.6	51.3	40.8	43.6	37.6	34.9
Headache-(RT-occipital)	29.5	29.7	21.1	18.5	15.8	26.5
Headache-(LT-occipital)	29.5	31.0	21.8	22.3	21.8	25.3
Back of the neck -Left	82.7	69.6	50.7	51.5	41.6	39.8
Back of the neck-Right	83.2	70.3	56.3	49.2	43.6	44.6
Trapezius-Rt-middle fibres	70.5	57.0	45.1	33.1	32.7	28.9
Trapezius-Lt-middle fibres	64.1	55.7	45.8	36.2	31.7	32.5
Trapezius-Rt-upper fibres	50.5	39.9	37.3	28.5	27.7	30.1
Trapezius-Lt-upper fibres	50.5	44.9	42.3	33.1	24.8	24.1
Pre-vertebral region	25.0	1.3	1.4	0.0	0.0	0.0
Retro-orbital-Rt	18.2	13.3	8.5	11.5	5.9	8.4
Retro-orbital -Lt	16.8	10.8	8.5	11.5	8.9	4.8
Sternomastoid-Rt upper	19.5	8.9	4.9	4.6	4.0	2.4
Sternomastoid-Rt middle	16.8	6.3	4.2	3.8	2.0	1.2
Sternomastoid-Rt lower	16.4	7.6	3.5	3.8	2.0	1.2
Sternomastoid-Lt upper	19.1	6.3	4.2	2.3	1.0	0.0
Sternomastoid-Lt middle	15.9	6.3	5.6	2.3	1.0	0.0
Sternomastoid-Lt lower	15.5	7.0	4.9	2.3	1.0	0.0
Interscapular-Rt	30.9	25.9	15.5	16.9	5.0	3.6
Interscapular-Lt	28.2	24.1	15.5	16.9	5.0	2.4
Nuchal attachment-Rt	17.7	8.9	4.2	4.6	2.0	2.4
Nuchal attachment-Lt	15.9	5.7	4.2	5.4	2.0	2.4
TM Joint-Rt	4.5	1.3	1.4	3.8	2.0	2.4
TM Joint-Lt	0.5	0.6	3.5	3.1	0.0	1.2
C7-T1 Junction	15.9	17.1	19.0	15.4	7.9	16.9

A significant decrease was noticed at the time of 4-weeks follow-up examination among the subjects reported to have had pain in the pre-vertebral region and the sternomastoid muscles. The follow-up examination at 12-weeks demonstrated that the pain in the pre-vertebral region was resolved in all subjects. A comparison between the initial and 52-week follow-up examinations showed a marked reduction in the percentage of areas reported to be painful at the end of the study. A reduction in the areas with pain related symptoms was noticed by a proportion of 49% in the posterior aspect of the neck (areas 10 & 11 as shown in body chart), 46% trapezius upper fibres and 54% middle fibres. A reduction of 86% and above was noted among the subjects presented with pain in the nuchal line, sternomastoid muscles and interscapular region. While the proportion of frontal headache was reported to have reduced by 40%, a significant percentage of subjects continued to report the presence of occipital headache, thus, the proportion of reduction in occipital headache was 12%. Unlike other symptomatic areas, at the end of the study period, the cervico-thoracic junction was the only area which has shown a marked reversal of the trend when compared to the initial percentage. The initial examination recorded that 15.9% of the subjects were affected, whereas 16.9% reported pain in the cervico-thoracic junction at the end point. However, the follow-up examination conducted at the 12th and 24th weeks showed a reduction in the proportion of subjects, respectively by (7.9%) when compared to the initial time period which was 15.9%.

A similar frequency distribution of the areas of pain for each of the random groups are presented in Tables A.III.4.8 to A.III.4.10.

Immobilised group:

Among the immobilised group (Table A.III.4.8), the highest incidence of pain was reported at the initial examination was in the posterior aspect of the neck (right and left sides-87.3%) followed by trapezius middle fibres right and left side (77.5%, 70.4% respectively), headache (forehead-59.15%), trapezius upper fibres (56.3%), pre-vertebral region (32.4%), interscapular region (28.2%) and occipital headache (28.9%). In addition to the above areas, the incidence of pain in the upper part of the sternomastoid (tendinous part) muscles on the left and right side (25.4% and 23.9% respectively) were reported more than the middle and lower parts of the same muscle on left and right sides (21.5%,.21.5%; 21.1%,19.7% respectively). The retro-orbital pain was more prevalent in the right side (18.5%) than the left side (16.9%).and the reverse was the case in T.M. joints (left = 8.5%, right=7.0%).

None of the subjects experienced pain in the pre-vertebral region by 4 weeks after the MVA and a similar observations were made in the case of T.M. joints. A comparison of the initial examination and the follow-up examination at week-52, demonstrated that there was a considerable difference between these two time periods. There was a significant reduction in the percentage of subjects complaining of occipital headache (I.E=28.9%, 52 wks=4.5%) and pain in the posterior aspect of the neck (back of the neck right and left; I.E=87.3%, 52 wks= 4.5%). A similar reduction was also evident in the areas associated with pain related symptoms viz trapezius upper and middle fibres. A steady decline was evident in the proportion of the subjects with pain related symptoms in the cervico-thoracic junction. At the time of the initial examination 14.1% of the subjects complained of pain in the cervico-thoracic junction and thereafter showed a steady reduction up to the follow-up examination at 24 weeks

post injury when 3.1% of the subjects reported pain, and this trend was reversed at the time follow-up examination week-52 in which 9.1% of the subjects in this group presented with pain. However, the proportion of subjects with pain in the cervico-thoracic junction was less than the initial examination (Figure A.III.3). In summary, the proportions in reduction of the painful areas in the body based on a comparison of baseline and follow-up examination at week-52 were readily noticeable in the following areas:

Areas	Proportion of reduction
Posterior aspect of the neck	94.8%
Trapezius upper fibres right and left	90.8%
Trapezius middle fibres right and left	89.7%
Occipital Headache	86.0%
Headache (forehead)	69.2%

A graphical presentation is provided in Figures A.III.3, A.III.5, A.III.7 to A.III.23 in order to clearly demonstrate the pattern of recovery as evidenced over different time periods and this will also illustrate the differences between the three treatment groups.

Active exercise group:

The frequency distributions of areas of pain for the active exercise group subjects are listed in the Table A.III.4.9. The initial examination showed

that there were close similarities to the immobilised group in the ranking of the areas of pain.

The pre-vertebral region was not reported to be painful from the time of the follow-up examination performed at 4 weeks post injury and throughout the full study period. A comparison of the observations made at the initial examination and end point examinations showed that the number of body areas affected were higher on the right side of the body as opposed to corresponding areas on the left side. The cervico-thoracic joint continued to be a matter of interest throughout the study period. At the initial examination, 20% of the population reported pain associated with cervico-thoracic junction and this was reduced to 17.1% at 4 weeks post-injury, only to rise again to the same level of initial assessment when examined at 6 weeks post injury (20%). Subsequent examinations at 12 and 24 weeks showed that 16.7% and 11.1% of the subjects reported that the cervico-thoracic junction was painful at the respective time periods. However, as previously noted in the immobilised group, the follow-up examination of the subjects in the active exercise group at week 52, showed that the incidence of pain in the C.T junction increased to 17.4% (Figure A.III.3). At the end of the study period, none of the subjects in the active exercise group experienced pain related symptoms associated with the left sternomastoid muscle, interscapular region, nuchal line and left retro-orbital pain. In summary, based on a comparison of observations made between the initial examination and at the end of the study period, the proportion of changes that reflected the rate of reduction of pain in the areas, associated with pain related symptoms are listed below:

Areas	Proportion of reduction
Sternomastoid (upper-right)	65.6%
Trapezius middle fibres right and left	49.4%
Posterior aspect of the neck (areas 10 & 11 as shown in the body chart)	47.4%
Occipital Headache	44.0%
Sternomastoid middle-right	36.7%
Headache- forehead	36.6%
Trapezius upper fibres right and left	19.3%

A graphical presentation is provided in Figures A.III.3, A.III.5, A.III.7 to A.III.23 in order to clearly demonstrate the pattern of recovery as evidenced over different time periods and this will also illustrate the differences between the three treatment groups.

Control group:

In Table A.III.4.10 similar frequency distribution of percentage of area of pain for the control group are presented and it was evident from the information contained in this table that the highest incidences of pain related symptoms were confined to the posterior aspect of the neck, followed by trapezius middle fibres, frontal headache and pain in the upper trapezius fibres; occipital headache ranked next succeeded by pain in the pre-vertebral region, the upper part of the sternomastoid muscle, retro-orbital area and cervico-thoracic junction.

The follow-up observations showed that in the control group, the pre-vertebral pain ceased to be a problem by 12 weeks post injury. A comparison of the observations made during the initial examination and the final follow-up examination, showed that a large percentage of subjects

continued to report pain mainly in the posterior aspect of the neck, then in the left trapezius middle fibres, frontal headache and occipital headache (Figures A.III.8, A.III.9, A.III.11, A.III.12, A.III.5, A.III.7). The second highest incidence of pain related symptom was attributed to the trapezius upper fibres on the right and left sides (Figures A.III.12, A.III.13). The percentage of reduction in the case of retro-orbital pain was not significant when compared to the initial examination and at the end of the study period. The percentage of subjects reported to have had pain in the cervico-thoracic junction were successively higher than the initial examination (14.6%) at 4th and 6th week follow-up examinations (15.3%, 26.4% respectively) followed by a reduction on consecutive follow-up observations at 12th and 24th weeks (19.1%, 9.5% respectively). However, as evidenced at the end of the study period , there was a marked increase in the percentage of subjects associated with pain in the cervico-thoracic junction (21.1%). A similar feature was evident among those subjects affected with occipital headache in which 32.6% of the subjects reported during the initial examination as having had right sided occipital headache, whereas the follow-up examination at week 52 noted that 44.7% of the population were now affected by occipital headache. The control group was the only group in which subjects complained of bilateral T.M joint pain and the over all proportion of changes between the initial and final follow-up examinations were noticeably lower when compared to the remainder of the groups (Figures A.III.22, A.III.23).

In summary, the proportions of reduction of pain in the number of the body areas related to pain symptoms, based on a comparison of initial and follow-up examinations at week-52 are as listed below :

Areas	Proportion of reduction
Sternomastoid middle-right	100.0%
Sternomastoid upper-right	87.1%
Interscapular regions	77.3%
Nuchal line	52.8%
Trapezius middle fibres right and left	33.1%
Trapezius upper fibres right and left	30.0%
Headache- forehead	26.2%
Posterior aspect of the neck	21.4%

A graphical presentation is provided in Figures A.III.3, A.III.5, A.III.7 to A.III.23 in order to clearly demonstrate the pattern of recovery as evidenced over different time periods and this will also illustrate the differences between the three treatment groups.

5.4.4 Correlation between the number of body areas reported to be painful and the cumulative pain score

There is no information on the association between the perceived pain level and the number of areas reported to be painful by a patient. It is conceivable to assume that if a subject were to have a greater number of painful areas, that person is more likely to either experience or report a proportionally higher level of pain. Conversely it can also be said that subjects with lesser number of areas of pain are likely to consider themselves suffering from lower level of pain. Thus, there is a possibility that the reported pain level might have been influenced due to the reasons out lined above. Hence it was considered that it was important to establish the association between the above variables by performing Pearson product correlation coefficient. The correlation coefficients presented in the Table 5.4.4 showed that the Pearson's r for each of the time periods was highly correlated in a positive direction. The inference from the tabulated results was that the total number of painful areas did not have an additive effect over the level of perceived pain reported by the subjects.

Table 5.4.4 **Pearson Product-Moment correlation coefficient: The total sum of pain level vs number of painful areas reported for each of the time periods.**

Time periods	Correlation Coefficients
24 hours post injury	0.84
Base-line examination	0.84
Follow-up examination-Week-4	0.78
Follow-up examination-Week-6	0.79
Follow-up examination-Week-12	0.81
Follow-up examination-Week-24	0.81
Follow-up examination-Week-52	0.86

5.5 Differences between groups (ANOVA)

A one-way ANOVA test was performed for each of the time periods to test the null hypothesis that there was no statistically significant difference between the treatment groups. Furthermore, when the respective p value was equal to or less than .05 (demonstrating significant differences between the groups) *post hoc* multiple comparison testing using the Scheffe's procedure was performed to identify the treatment groups with significant differences. The summary of ANOVA results and the Scheffe's procedure are provided for each of the time periods. The geometric means derived from the Scheffe's procedure are tabulated and identified to demonstrate significant differences when present.

An analysis of variance (ANOVA) was performed, using the level pain as noted during the initial examination, to compare the experimental and control groups in order to verify the effectiveness of the randomisation. It was evident from the p value that there was a significant difference between the groups (F= 8.9262 ; p= .0002) (Table 5.5.1). Subsequent multiple comparisons testing using the Scheffe procedure, demonstrated that the immobilised group was significantly different to the active exercise and control groups as evidenced by a consideration of resulting geometric mean for each of the three groups (Table 5.5.3). The above results showed

that the randomisation procedure may not have been effective and this may be due to the presence of various confounding factors which will be discussed elsewhere (see Chapter-6 Discussion page 251).

Series of ANOVA were performed for each of the time intervals in order to compare the differences between the three treatment groups and also the Scheffe's procedure was used to identify the groups which were significantly different (Table 5.5.1, 5.5.3). The ANOVA results showed that there was a significant difference between the groups at the 4 week follow-up examination ($F=10.8281$; $p=.0014$). The Scheffe's procedure identified that there was a significant difference between the control group and the immobilised group, whereas the difference between the active exercise group and the control groups was not significant (Table 5.5.4). The geometric mean for each of the three groups are presented in the Table 5.5.4. Similar statistically significant differences were observed at the 6th week follow-up examination, between the three treatment groups ($F=16.4454$; $p=.0000$) while the Scheffe procedure demonstrated that the control and active exercise groups were significantly different from the immobilised group (Table 5.5.5). The respective geometric means are provided in the Table 5.5.5. One way ANOVA results for the 12th week follow-up examination also showed statistically significant differences between the treatment groups ($F=15.8961$; $p=.0000$) and it was evident from the Scheffe's procedure that the differences between the immobilised group and the remaining two groups were due to statistically significant reduction in the level of pain reported by the subjects in the immobilised group. The geometric mean for each of the three groups are listed in the Table 5.5.6 for comparison.

From the ANOVA results of week-24 follow-up examination, it was evident that statistically significant differences between the three treatment groups

continued to exist at 24 weeks post injury (F=18.8794; p=.0000). The Scheffe's procedure demonstrated that the subjects in the immobilised group were improving significantly when compared to the other groups and this difference was characterised by the smaller value of the geometric mean of the immobilised group, when compared to other groups (Table 5.5.7). A statistically significant difference between the groups was observed at 52 weeks post injury (F=29.3141; p=.0000) and the Scheffe procedure again identified that the immobilised group was significantly better when compared to the active exercise group and the control group. The geometric means for each of the three groups are listed in the Table 5.5.8.

Table 5.5.1 Summary of ANOVA results for the treatment groups.

Time periods	Source	D.F	SS	MS	F	p
24 HRS	Between groups	2	6.42	3.21	7.39	0.0008*
	Within groups	217	94.27	0.43		
Initial exam	Between groups	2	9.10	4.55	8.92	0.0002*
	Within groups	217	110.62	0.51		
4-Weeks	Between groups	2	21.66	10.83	6.86	0.0014*
	Within groups	159	251.14	1.58		
6-weeks	Between groups	2	90.80	45.40	16.44	0.0000*
	Within groups	148	408.59	2.77		
12-Weeks	Between groups	2	131.82	65.90	15.89	0.0000*
	Within groups	146	605.34	4.146		
24-Weeks	Between groups	2	178.81	89.40	18.88	0.0000*
	Within groups	119	563.52	4.74		
52-Weeks	Between groups	2	268.28	134.14	29.31	0.0000*
	Within groups	112	495.30	4.42		

*** = Statistically significant**

Table 5.5.2 **Summary of Scheffe procedure to demonstrate the treatment group with significant difference- Time period - 24 hours post injury.**

Groups-& Geometric mean	Group-B	Group-C	Group-A
Group-B- 354			
Group-C- 428			
Group-A- **550**	*		

* = Statistically significant
Group B= Active exercise group
Group A= Immobilised group
Group C= Control group

Table 5.5.3 **Summary of Scheffe's procedure to demonstrate the treatment group with significant difference- Time period -Initial examination.**

GROUPS-& Geometric Mean	Group-B	Group-C	Group-A
Group-B- 324			
Group-C- 412			
Group-A- **550**	*	*	

*= Statistically significant
Group 2= Active exercise group
Group 1= Immobilised group
Group 3= Control group

Table 5.5.4 **Summary of Scheffe's procedure to demonstrate the treatment group with significant difference- Time period -week-4 post injury.**

Groups & Geometric Mean	Group-A	Group-B	Group-C
Group-A- 134			
Group-B- 211			
Group-C- **317**	*		

* = Statistically significant
Group 2= Active exercise group
Group 1= Immobilised group
Group 3= Control group

Table 5.5.5 **Summary of Scheffe procedure to demonstrate the treatment group with significant difference- Time period -week-6 post injury.**

Groups- & Geometric Mean	Group-A	Group-B	Group-C
Group-A- 45			
Group-B- 147	*		
Group-C- **262**	*		

* = Statistically significant
Group 2= Active exercise group
Group 1= Immobilised group
Group 3= Control group

Table 5.5.6 **Summary of Scheffe procedure to demonstrate the treatment group with significant difference- Time period -week-12 post injury.**

Groups- & Geometric Mean	Group-A	Group-B	Group-C
Group-A- 19			
Group-B- **80**	*		
Group-C- **169**	*		

*= Statistically significant
Group 2= Active exercise group
Group 1= Immobilised group
Group 3= Control group

Table 5.5.7 **Summary of Scheffe procedure to demonstrate the treatment group with significant difference- Time period -week-24 post injury.**

Groups & Geometric Mean	Group-A	Group-B	Group-C
Group-A- 8			
Group-B- **98**	*		
Group-C- **100**	*		

*= Statistically significant
Group 2= Active exercise group
Group 1= Immobilised group
Group 3= Control group

Table 5.5.8 **Summary of Scheffe procedure to demonstrate the treatment group with significant difference- Time period -week-52 post injury.**

Groups & Geometric Mean	Group-A	Group-B	Group-C
Group-A 4			
Group-B 54	*		
Group-C 112	*		

*= Statistically significant Group 1= Immobilised group
Group 2= Active exercise group Group 3= Control group

The above results clearly demonstrated that the subjects in the immobilised group were consistently different from the active exercise and control groups, at each level of the follow-up time periods. It was also evident that throughout the study period, that the immobilised group showed a marked reduction in the geometric mean; thus, providing an estimate of the degree of progress experienced by the subjects in this group as opposed to remainder of the treatment groups.

A repeated measures ANOVA (ANOVA-RM) was performed to test the between subjects effects and this showed a statistically significant difference between treatment groups (F=18.03; p=.0000). In addition to this, an ANOVA-RM was performed using the follow-up time periods as a covariate, to verify whether the subjects could have got better with or without any form of treatment, through passage of the time alone. The analysis showed that the covariate "time" was statistically significant (F=57.27; p=.000), thus, showing that given sufficient time, the subjects will get better. However, it was evident from an interaction effect analysis using covariates time and treatment factors that a significant interaction was present between the time factor and the treatment factors (F=8.56; p=.0000) which clearly demonstrated that not only the time factor is responsible for

the recovery but also the treatment factors made a significant contribution in this regard i.e the rate of recovery differs between treatment groups.

5.5.1 Prognostic variables

A statistically significant difference between the treatment groups as evidenced from the ANOVA results of the initial examination may be due to the presence of one or more confounding variables in the treatment groups. Hence it was decided to perform a series of analysis of covariance (ANCOVA) and two-way ANOVA to determine the effect of the following covariates on pain and the interaction with the treatment factors:

1. gender;
2. age;
3. occupation;
4. types of collision;
5. speed of the vehicles involved;
6. seating position;
7. presence of headache within 24 hours after the collision;
8. the severity of the pain related symptom as reported at the base level examination;
9. presence of paraesthesia;
10. litigation;
11. loss of cervical lordosis;
12. pre-existing degeneration of the I.V.D in the cervical spine; and
13. interscapular pain.

A series of ANCOVA and 2- way ANOVAs was performed to determine the effect of several covariates and their interaction effect with the treatment factors was also analysed.

Effect of age as a factor influencing the recovery:

A series of ANCOVAs were performed using the age of the subjects as a covariate, for each of the time intervals. The results of the ANCOVAs presented in the Table A.III.5.1 showed that the age of the subjects had a significant effect on the pain, during all the time periods except the initial examination (F=.384; P=.536). It is reasonable to make an inference that irrespective of the age of the subjects, there was no difference in the severity of the pain related symptoms soon after the MVA. However, this was not the case during subsequent time periods in which the respective p values were statistically significant (Table A.III.5.1). It was evident from the results of the ANCOVA that there were significant differences in the level of pain between the subjects that can be attributed to the age effect (Figure A.III.24) The scattergrams showed that the subjects in the age group <30 years reported to have had significantly lower level of pain which in turn signifies the relative rate of progress when compared to other age groups. It should be noted that the treatment factors were also included along with age factor in the respective ANCOVAs and in spite of controlling for the age effect the treatment effects were statistically significant for all of the time periods demonstrating that the treatment factors are also responsible for the reduction of the pain (Table A.III.5.1).

Effect of gender as a factor influencing the recovery:

A series of two-way ANOVAs were performed, to analyse the effect of gender as a prognostic factor, for each of the time intervals and the results are presented in the Table A.III.5.2. It was evident that irrespective of the treatment received, a statistically significant difference in pain level attributable to the gender difference existed among the subjects. The above significant difference between groups was present for each of the time periods from the initial examination up to the follow-up examination at 24th week(Table A.III.5.2). However, this gender effect disappeared at the

52 week follow-up examination (F=0.030; p=.864), whereas the treatment effects were statistically significant throughout the study period. The interaction effect between the treatment and gender factors were tested by performing a two way interaction analysis for each of the follow-up examinations in order to determine to what extent the gender factor influenced the treatment factors. The tabulated p values for the interaction effect for each of the follow-up examinations were larger than .05. Therefore, an interaction was unlikely to exist between the treatment and gender factors and the change in the pain level is likely to be attributable to both treatment factors and gender factors acting independently.

Occupation:

The effect of the occupation on the level of pain was tested by performing a series of ANOVAs for each of the follow-up examination time periods, using the reported occupation as a covariate. The results of the respective ANOVA presented in the Table A.III.5.3 showed that the p value for each of the time periods were larger than .05 which in turn indicated that occupation is an unlikely factor to have an effect on the pain level. Similarly, a two way interaction analysis also resulted in a p value larger than .05; thus, confirming an absence of interaction effect (Table A.III.5.3). However, the statistically non-significant p value may be attributed to smaller number of subjects in some of the occupational categories and this situation was rectified by grouping the subjects into four occupational categories viz:

1. manual workers;
2. non-manual workers;
3. house wives; and
4. unemployed.

A series of two-way ANOVAs were performed using the four grouped occupational category and the tabulated results (Table A.III.5.4) showed that there were statistically significant differences in certain grouped occupational categories as evidenced at the 4th and 12th follow-up examinations (F=4.147; p=.007 and F=3.289; p=.023 respectively). It was evident that during the first 12 weeks certain occupations may have a significant effect on the pain level. The absence of statistically significant interaction effect for any of the follow-up examination time periods, demonstrate that the occupational categories had not affected the treatment effect in any specific manner and the extent of recovery was independently attributable to both the respective treatment effect and the occupational category.

Types of collisions:

In order to verify the prognostic value of different types of collisions such as rear-end collision etc, a series of two-way ANOVAs were performed for each of the follow-up time periods and the results are presented in the Table A.III.5.5. A statistically significant effect for the collision factor was evident at the 6th and 12th week follow-up examinations (F=2.704; p=.003; F=2.090; p=.017 respectively) indicating that there were significant differences in the level of pain experienced by the subjects according to the type of collision encountered by them. However, the statistically significant differences were not consistently present at all levels of the study period, demonstrating that the type of collisions may not be an important factor in determining the progress of the subjects (Table A.III.5.5). This observation was further strengthened by p values larger than .05 for interaction effects in all instances of the follow-up examinations with an exception of the 4th week (F=2.173; p=.012). It is conceivable that the above inferences might have been due to smaller sample size in some of the groups (Figure 5.2.4)

and in order to correct this situation, a further series of two-way ANOVAs were performed by grouping the types of collisions into 5 larger categories viz:

1. rear-end collisions;
2. front-end collisions;
3. side impacts;
4. roll overs; and
5. others.

The p value for the grouped collision factors (newly created covariates) was larger than .05 for all the time periods except week-24 follow-up examination (p=.046) and the derived p value for the interaction effect was larger than .05 for all of the time periods (Table A.III.5.6). The inference drawn from the two-way ANOVA results for grouped collision categories (Table A.III.5.6) was in agreement with the earlier observations in that there was no prognostic value attached to the types of collisions. Thus, it is reasonable to conclude that there is not sufficient evidence to support the hypothesis that the recovery of the subjects is related to the type of collision encountered by them.

Seating Positions:

A series of two-way ANOVAs were performed using the subjects seating positions as a covariate so that the influence of this factor over the pain level can be established. The respective summary of the results are presented in the Table A.III.5.7 and it was evident that the effect of the seating position was statistically significant (F=3.034; p=.02) in relation to the 24th week follow-up examination and had not affected the pain level for the remainder of the time periods. A two way analysis of interaction effect between the treatment factors and seating position also showed p values which were larger than .05 for each of the time periods; thus, indicating that

the seating position of the subjects at the time of the MVA is unlikely to have any influence on the progress of the "whiplash" patients. However, the number of subjects seated in the rear seats were relatively fewer when compared to the front passenger and driver categories (Table 5.2.5) and possibly might have affected the statistical power. This factor was addressed by creating two categories of seating positions viz, driver and passenger and a series of ANCOVAs were performed. The results were similar to that of the preceding ANCOVA showing a statistically significant effect at the 24th week follow-up period (F=8.678; p=.004) and had no statistically significant interaction effect for any one of the time periods (Table A.III.5.8). Hence, it can be concluded that the seating position of the subjects at the time of the impact is unlikely to have any effect over the recovery of the "whiplash" subjects and the recovery is more likely to be attributed to the type of treatment received by them.

Speed of the vehicles involved:

The effect of the speed of the vehicles involved over the pain level is matter of considerable interest to the clinicians. This confounding variable was used as a covariate and a series of ANCOVAs were performed for each of the time periods. The results are summarised in the Tables A.III.5.9 and Table A.III.5.10 for the speed of the subject's vehicles and other vehicles/ objects involved respectively. The ANCOVA results presented in the Table A.III.5.9 demonstrated that the p value for the initial examination and the 4th week follow-up examinations (F=1.954; p=.164 and F=1.12.0; p=.291; respectively) were not statistically significant meaning that the speed of the subject's vehicles had no association with the severity of the pain reported by the subjects for the respective time periods. In contrast to this, a statistically significant effect attributable to the speed of the subject's vehicle was evident for each of the remainder of the time periods (i.e 6th, 12th, 24th and 52 week follow-up examinations), thus, demonstrating that

the differences in pain level reported by the subjects is more likely attributable to the speed of the subject's vehicles.

The effect of the speed of the other vehicles / objects involved in the MVA was analysed by performing a series of ANCOVAs for each of the time periods and the results are presented in Table A.III.5.10. The tabulated results with p values smaller than .05 clearly showed that the speed of the other vehicles / objects was a significant covariate for each of the time periods. The results showed that speed of the other vehicle / object is relatively more likely to influence the level pain when compared to the speed of the subject's vehicle.

The prognostic value of level pain at the time of initial examination:

A series of ANCOVAs were performed using the geometric mean of the pain scale reported at the initial examination as a covariate to verify whether the severity of the pain experienced by the subjects during the acute phase of the injury can predict the pattern of recovery for a given subject. The results of the ANCOVAs are summarised in the Table A.III.5.11 and the tabulated p values are statistically significant for each of the time periods. It was evident from these consistent occurrences of p values which are smaller than .05, that the level of pain at the time of the initial examination is a significant covariate and also highly likely to have the attributes of a prognostic factor.

Prognostic value of the headache factor:

A series of two-way ANOVAs were performed using the presence of headache (i.e occipital and frontal headache grouped as a single factor) within 24 hours after the MVA as a covariate and its effect for each of the follow-up time periods are reported in the Table A.III.5.12. The

corresponding p value for the 4th, 6th, 12th and 52 week follow-up examinations were larger than .05 and therefore the covariate was deemed to be non significant, whereas the covariate had a statistically significant effect at the 24th week follow-up examination (F=9.442; p=.003). However, occurrences of consistent non significant p value for all the follow-up time periods with the exception of 24th week follow-up examination showed that the presence of headache within 24 hours after the MVA is highly unlikely to have prognostic value in predicting the progress of "whiplash" patients.

Paraesthesia:

A series of 2-way ANOVAs were performed for each of the time periods to establish the effect of the paraesthesia as a covariate and also its interaction effect over the treatment outcome. The results of the ANOVA indicated that the p values for each of the time periods, except for week-6, were smaller than .05, thus, indicating that paraesthesia was a significant covariate (Table.A.III.5.13). The consistency of association between the paraesthesia and pain between the treatment groups demonstrates the underlying severity of the injury to which the respective subjects were exposed to. However, there was no significant interaction between the treatment regimens and paraesthesia as to inform which of the three treatment regimen is suitable to obtain a better treatment outcome.

Loss of cervical lordosis and presence of I.V.D degeneration:

The significance of the spondylitic changes and "loss of cervical lordosis" as covariates were tested by performing a series of 2-way ANOVAs for each of the time periods. The results showed that the spondylitic changes is not a significant covariate, whereas statistically significant association between the pain and loss of cervical lordosis was present at the initial examination and the follow-up examination conducted at week-12 (F=5.8,

p=.017; F=4.56, p=.035; respectively). A lack of consistent significant p value indicated that loss of lordosis may not have prognostic value. The ANOVA results for the above covariates are presented in the (Tables A.III.5.14, A.III.5.15).

Interscapular pain:

The prognostic value of the interscapular pain was tested by performing a series of 2-way ANOVAs for each of the time periods. The ANOVA results showed that the interscapular pain was a significant covariate for each of the time periods (Table A.III.5.16). The association between the interscapular pain and the treatment groups may be attributed to the severity of the injury. The interaction effect was not statistically significant. The above results indicate that the subjects complaining of interscapular pain are more likely to have a greater level of pain when compared to a subject without interscapular pain.

Effect of litigation on pain:

The significance of litigation as a covariate and its effect on the outcome of treatment was tested by performing a 2-way ANOVA and the results showed that the litigation was a significant covariate at the initial examination and also at the final follow-up examination conducted in week-52 (Table A.III.5.17). Even after controlling for the effects of litigation, the differences between the treatment groups remained statistically significant for the corresponding time periods. There was no interaction effect for any of the time periods. The inference was that the differences between treatment groups were not entirely attributable to the influence of the litigation factor and that the treatment outcome has not been significantly affected by the legal status of the accident. A *post hoc* multiple comparison testing using the Scheffe's procedure identified that at initial examination, the geometric mean of the compensable subjects from the immobilised

group was significantly higher than that of the non-compensable subjects from the active exercise group (Table A.III.5.18). A similar comparison for the follow-up examination at week-52, showed that the compensable subjects from the active exercise and the control groups significantly had a higher geometric mean when compared to the subjects both in compensable as well as non-compensable subjects in the immobilised group. The Scheffe's procedure also identified that the compensable subjects from the control group had a significantly higher geometric mean when compared to the non-compensable subjects from the control group (Table A.III.5.19).

Visual dysfunction:

Series of 2- way ANOVAS were performed to establish the significance of difficulty in focusing and blurred vision as covariates (Tables A.III.5.20, A.III.5.21). In the case of difficulty in focusing the p values for the initial examination and the follow-up examinations at week-12, week-24 and week-52 were smaller than .05, indicating that blurred vision was a significant covariate. The presence of blurred vision was demonstrated to be a significant covariate for each of the time periods (p= .05), signifying that the blurred vision had a stronger association to pain when compared to the effect of difficulty in focusing. However, the above covariates had no interaction effect.

5.5.2 Changed scores

From the ANOVA of the initial examination pain (Table 5.5.1) and corresponding Scheffe's procedure (Table 5.5.3) it was clearly evident that the pain level between treatment groups were not equal. The Scheffe's procedure identified that the geometric mean of the immobilised group was the highest of the three groups being significantly different when compared to the active exercise and the control groups. Hence, it was obvious that

the randomisation may not have been effective in formulating groups with equal amount of pain level. In spite of the noted statistically significant difference at the initial examination, further ANOVAs and Scheffe's procedure for the subsequent follow-up time periods up to the end of the study, showed that the immobilised group demonstrated a significant reduction in the pain level. It could be argued that as noticed in the case of the immobilised group, the proportion of change from a larger number as noted at the initial examination to a small number at subsequent time periods is likely to contribute towards significant differences in favour of the immobilised group, as evidenced from the ANOVAs. In order to determine whether a situation as stated above might have occurred, a new dependent variable was created by subtracting the pain level for the corresponding time period from the base-line pain level viz "change in base-line pain" (also known as change score). The use of change score is recommended for removing stable individual differences between subjects which in turn would increase the power of the statistical procedure (Norman, 1989). In addition, the change score can also be used to correct the differences that existed between randomised groups at the initial examination itself (Norman, 1989). Thus, having removed the selection bias introduced by the randomisation, a multiple regression was performed for each of the time periods, to analyse the relationship between the dependent variable (change scores) and a set of predictor variables which were shown to have significant effect on the treatment groups viz:

1. age;
2. pain at initial examination;
3. treatment factor- active exercise regimen; and
4. treatment factor- immobilisation regimen.

The result of the multiple regression for week-4 showed that the p value for the F statistics was smaller than .05, thus, demonstrating that the variance attributable to the regression was significant (Table 5.5.9)

Table 5.5.9 **Summary of Multiple regression statistics for change scores and predictor variables -week-4.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	28.7	7.18	10.29	0.0000*
Residual	75	52.31	0.697		

*** = Statistically significant**

Table 5.5.10 **Prediction of change in pain in relation to the initial examination (progress) at week-4, from Age, initial examination pain level and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.01	0.01	-0.08	-0.08	0.43
Initial examination pain level	0.25	0.14	0.17	1.75	0.08
Active exercise regimen	0.01	0.25	0.05	0.05	0.96
Immobilisation regimen	1.03	0.23	0.50	4.45	0.0000*

*** = Statistically significant**

The regression coefficient (B) for each of the independent variables (X) describe the weight contributed by the respective variable to the explanation of the dependent variable (Y). The standardised regression coefficients are indicative of the relative importance of a specific variable within the regression equation. As it can be seen from the summary of the results in the Table 5.5.10, the immobilisation regimen was statistically significant and also relatively an important predictor variable. Multiple regression analysis for the rest of the follow-up time periods consistently showed that the p value for the immobilisation regimen was less than .05,

thus, indicating that the above variable was a relatively important predictor variable (Tables 5.5.11 to 5.5.18). Therefore, it can be concluded that earlier ANOVA results were not affected by the selection bias.

Table 5.5.11 **Summary of Multiple regression statistics for change scores and predictor variables-week-6.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	61.9	15.47	9.29	0.0000*
Residual	75	124.9	1.67		

Table 5.5.12 **Prediction of change in pain in relation to the initial examination (progress) at week-6, from Age, initial examination pain level and type of treatments.**

Variable wk-6	B	SEB	Standardised coeff	T	p
Age	0.007	0.01	0.06	0.56	0.58
Initial examination pain level	0.13	0.22	0.06	0.59	0.56
Active exercise regimen	0.25	0.39	0.07	0.63	0.53
Immobilisation regimen	1.90	0.36	0.61	5.33	0.0000*

Table 5.5.13 **Summary of Multiple regression statistics for change scores and predictor variables -week-12.**

Source of variance-wk12	df	Ss	Ms	F	p
Attributable to regression	4	112.07	28.01	10.72	0.0000*
Residual	75	195.93	2.61		

Table 5.5.14 **Prediction of change in pain in relation to the initial examination (progress) at week-12, from Age, initial examination pain level and type of treatments.**

Variable wk-12	B	SEB	Standardised coeff	T	p
Age	-0.001	0.02	-0.01	-0.11	0.91
Initial examination pain level	-0.65	0.28	-0.23	-2.36	0.02*
Active exercise regimen	-0.41	0.49	-0.09	-0.85	0.39
Immobilisation regimen	2.23	0.44	0.55	4.90	0.0000*

*** = Statistically significant**

Table 5.5.15 **Summary of Multiple regression statistics for change scores and predictor variables -week-24.**

Source of variance-wk24	df	Ss	Ms	F	p
Attributable to regression	4	204.27	51.07	13.26	0.0000*
Residual	75	288.87	3.85		

Table 5.5.16 **Prediction of change in pain in relation to the initial examination (progress) at week-24, from Age, initial examination pain level and type of treatments.**

Variable wk-24	B	SEB	Standardised coeff	T	p
Age	0.01	0.02	0.03	0.30	0.76
Initial examination pain level	-0.26	0.34	-0.07	-0.79	0.43
Active exercise regimen	-0.34	0.59	-0.06	-0.60	0.56
Immobilisation regimen	3.23	0.54	0.64	5.93	0.0000*

Table 5.5.17 **Summary of Multiple regression statistics for change scores and predictor variables -week-52.**

Source of variance-wk52	df	Ss	Ms	F	p
Attributable to regression	4	189.52	47.38	10.7	0.0000*
Residual	75	332.13	4.43		

Table 5.5.18 **Prediction of change in pain in relation to the initial examination (progress) at week-52, from Age, initial examination pain level and type of treatments.**

Variable wk-52	B	SEB	Standardised coeff	T	p
Age	-0.03	0.02	-0.18	-1.80	0.08
Initial examination pain level	-0.04	0.36	-0.01	-0.10	0.92
Active exercise regimen	0.23	0.63	0.04	0.36	0.72
Immobilisation regimen	2.72	0.58	0.52	4.7	0.0000*

*** = Statistically significant**

Even though the multiple regression analyses showed that the derived p value for the immobilisation regimen was consistently significant for each of the time periods, the histograms of change scores showed the existence of

a bi-modal distribution (Figure A.III.25). It was evident from these histograms that one of the assumptions in respect of the linearity has been violated. In general it is a normal practice in a situation like this, to apply relevant mathematical procedures in order to remove the skewness and transform the data into a normal distribution. However, transformation procedure can not be applied when a bi-modal distribution is present and also it should be noted that the present set of data has already been log transformed. The histogram of the change score clearly show that the bi-modal distribution is attributable to the pattern of resolution of the pain experienced by the subjects. As the pain level decreased, a group of patients were moving towards the lower end of the scale, whereas those subjects with a higher pain level were on the other end of the pain scale, thus, a bi-modal distribution was clearly emerging. It was also evident from the histograms of the change scores that the point of separation for the bi-modal distributions, occurred consistently around the log pain 4 (Figure A.III.25). This location (log pain 4) in the histogram appeared to be the point at which those subjects who were either getting better or not getting better can be clearly identified. Based on this empirical observation, two new dichotomous categorical variables "getting better" and "not getting better" were created and the following covariates viz:

1. age;
2. gender;
3. speed of the vehicles;
4. initial examination pain level;
5. random groups;
6. immobilisation regimen;
7. active exercise regimen;
8. presence or absence of cervical lordosis;
9. disc degeneration;

10. presence of interscapular pain;
11. blurred vision;
12. litigation;
13. difficulty in focusing; and
14. paraesthesia.

were used as dependent variables to perform a series of logistic regressions for each of the time periods. The predictor variables used in the logistic regressions included those either identified as significant covariates from the preceding statistical analyses or mentioned in the literature as having prognostic value. The regression coefficient for the respective predictor variable denotes how the odds of "getting better" will alter in relation to any changes in the predictor variables. The coefficient is further converted as odds ratio which is a mathematical expression of the estimate of the recovery for those subjects in the experimental groups (i.e immobilisation and active exercise groups) when compared to the control group. The summary of the logistic regression analysis comparing the immobilised group vs control group and active exercise group vs control group are presented in the Table 5.5.19 and Table 5.5.20 respectively.

The immobilisation group was statistically significant consistently except week 4, whereas the other predictor variables were occasionally significant during the study period. It was evident from the results with reference to the follow-up examination at week 4, that none of the predictor variables were statistically significant. The results showed that the initial examination pain level was a significant predictor variable at week-6, week-12 and week-24 (p=.0387, .0166, .0282, respectively). The speed of the vehicles and gender were also identified as statistically significant predictor variables at week-12 (p=.0314, .0035, respectively). The immobilised group was not statistically significant at week 4 (p= 397), whereas it was the only predictor

variable which was statistically significant for the remainder of the follow-up time periods and also showed an odds ratio which was greater than 1 for the respective time periods (Table 5.5.19). It was also evident from the odds ratio that at 6 weeks post injury, the probability of getting better by being in the immobilised group was 8.39 times greater than being in the control group. The tabulated odds ratio consistently showed that the above advantage continued to exist for the immobilised group for subsequent time periods (Table 5.5.20). A similar comparison between the active exercise group and the control group demonstrated that the odds of getting better by being in the active exercise was not different to that of the subjects in the control group (Table 5.5.20).

The regression model's ability to predict the rate of recovery during the early stages was 0% and from week 12 on wards the rate of prediction noticeably improved to 30.43% to reach the level of 75.76% at the end of the study period.

Table 5.5.19 **Summary for Logistic regression analysis (Immobilised group vs control group)- Using listed covariates as predictor variables.**

Weeks	Total number recover-ed	ß Reg coeff	Wald	p	Odds Ratio	% prediction of recovery
6	13	2.13	10.23	0.0014*	8.39	0.00
12	16	1.45	9.90	0.0016*	4.26	30.43
24	12	2.09	18.60	0.0000*	8.11	52.00
52	8	1.78	25.79	0.0000*	5.93	75.76

*** = Statistically significant**

Table 5.5.20 **Summary for Logistic regression analysis (active exercise group vs control group)- using listed covariates as predictor variables.**

Weeks	Total number recovered	ß Reg coeff	Wald	p	Odds Ratio	% prediction of recovery
6	13	-0.81	1.02	0.31	0.44	0.00
12	16	-0.29	0.38	0.53	0.74	30.43
24	12	-1.01	3.10	0.0779	0.36	52.00
52	8	-0.599	1.99	0.1581	0.55	75.76

*** = Statistically significant**

5.6 Survival analysis of pain

In a longitudinal clinical trial, the follow-up time may not be long enough, for some of the subjects, to observe the event of interest (e.g in this instance, reaching "0" pain level). Since a clinical trial can not be conducted for an indefinite duration, the survival analysis procedure is generally performed to determine the cumulative proportion of survival at the end point of the study and used as a measure of comparison of survival rate between treatment groups (Armitage and Berry, 1987). In addition, the median survival time is also taken into consideration to demonstrate the differences between the treatment groups. The derived life table is characterised by its ability to convey both quantitative and qualitative information for all the subjects included in the study with reference to the events of clinical interest at a given time period. The null hypothesis states that ST (survival time) = Grp A = B = C.

The summary of the Survival analysis and the relevant life table for each of the treatment groups are presented in Tables 5.6.1 to 5.6.4. In addition to the actual number as well as the proportion of the subjects reaching 0 pain level (terminal event), the time scale at which this event occurred (interval

start time), and the proportion of the subjects experiencing pain above the level of 0, at a given time period (proportion of survival) are also presented (Table 5.6.1; 5.6.2; 5.6.3). The cumulative proportion of survival for a given time interval denotes the over all percentage of subjects who have reported pain level higher than 0, with reference to a given time period from the start of the study. The hazard rate is the function of the time period and the covariate (i.e treatment regimen),.thus, representing the dependence on the variables for each subject as well as on time intervals (Armitage and Berry, 1987). In essence, the hazard rate represents the probability at a particular point in time at which the event of interest (i.e reaching pain level 0) will occur. Thus, the hazard rate reflects the differences between groups. The derived median survival time represents the differences between the groups for achieving "0" pain level. The cumulative proportion of survival as noted at the end of the study period and the median survival time are the two important statistics presented in this section to describe the differences between the treatment groups.

The Table 5.6.1 showed that the cumulative proportion of the subjects who have survived at the end of the study period was 0.1794. The inference was that 18% of the subjects from the immobilised group were still experiencing a pain level that was above 0. The median survival time for the immobilised group was 25.23 weeks and this represents the average time period in which most of the changes occurred, thus, distinguishing those subjects who were reaching 0 pain level and others reporting pain at a level above 0. Similarly, the cumulative proportion of the subjects reported to have had pain level above 0 at the end of the study period for the active exercise group, was 0.58 meaning 58% of the subjects reported pain level that was above 0 (Table 5.6.2). The median survival time for the active exercise group was 52.00+ weeks which meant that a 50% of the

subjects were still having pain level greater than 0. The cumulative proportion of the survival at the end of the study period presented in the Table 5.6.3 demonstrated that the experience of the control group was also similar to that of the active exercise group in that the cumulative proportion of the surviving subjects was 0.6 (i.e 60% of the subjects reported pain level greater than 0) with a median survival time of 52.00+ weeks. The survival experience of the subjects for each of the treatment groups is graphically presented in Figure 5.6.1.

A comparison of survival experience using the Lee-Desu statistic showed that there was a statistically significant difference between the treatment groups (F=21.597, D.F=2, p=.0000*) which means that the observed differences between the treatment groups may not have been purely due to chance alone (Table 5.6.4).

Table 5.6.1 Life table for the immobilised group - Survival variables: Study time -Time in study.

n Interval Start	n Entering this interval	n Withdrawn during interval	n Exposed to risk	n Terminal events	Propn terminating	prop surviving	Cumul propo-rtion surviv-al at end	proba-bility Densi-ty	Haz-ard rate	SE of cuml surviving	SE of prob Dens-ity	SE of hazard rate
0	71	0	0.0	0	0.00	1.00	1.00	0.00	0.00	0.00	0.00	0.00
4	71	10	66.0	2	0.03	0.97	0.97	0.02	0.02	0.02	0.01	0.01
6	59	2	58.0	9	0.16	0.85	0.82	0.08	0.08	0.05	0.02	0.02
12	48	3	46.5	9	0.19	0.81	0.66	0.08	0.11	0.06	0.02	0.04
24	36	1	35.5	14	0.39	0.61	0.40	0.13	0.25	0.07	0.03	0.06
52	21	13	14.5	8	0.55	0.45	0.18	-	-	0.06	-	-

The Median Survival Time for these data is 25.23 weeks.

Table 5.6.2 **Life table for the active exercise group - Survival variables: Study time -Time in study treatment groups .**

n Interval Start time-	n Entering this interval	n With-drawn dur-ing inter-val	n Expo-sed to risk	n Termi -nal event s	Propn termi-nating	prop survi-ving	Cumul propo-rtion survi-val at end	proba-bility Densi-ty	Haz-ard rate	SE of cuml survi-ving	SE of prob Dens-ity	SE of hazard rate
0	60	0	60.0	0	0.00	1.00	1.00	0.00	0.00	0.00	0.00	0.00
4	60	16	52.0	2	0.39	0.96	0.96	0.02	0.02	0.03	0.01	0.01
6	42	5	39.5	2	0.05	0.95	0.91	0.02	0.02	0.04	0.01	0.02
12	35	2	34.0	4	0.12	0.88	0.80	0.05	0.06	0.06	0.03	0.03
24	29	6	26.0	3	0.12	0.88	0.71	0.05	0.06	0.08	0.03	0.04
52 +	20	18	11.0	2	0.18	0.82	0.58	-	-	0.10	-	-

The Median Survival Time for these data is 52 + weeks.

Table 5.6.3 **Life table for the control group survival variables: Study time -Time in study treatment groups.**

n Interval Start time-	n Entering this interval	n With-drawn dur-ing inter-val	n Expo-sed to risk	n Term -inal eve-nts	Propn termi-nating	prop survi-ving	Cumul propo-rtion survi-val at end	proba-bility Densi-ty	Haz-ard rate	SE of cuml survi-ving	SE of prob Dens-ity	SE of hazard rate
0	89	0	89.0	0	0.00	1.00	1.00	0.00	0.00	0.00	0.00	0.00
4	89	29	74.5	1	0.01	0.99	0.99	0.01	0.01	0.01	0.01	0.01
6	59	9	54.5	1	0.02	0.98	0.97	0.01	0.01	0.02	0.01	0.01
12	49	9	44.5	3	0.07	0.93	0.90	0.03	0.03	0.04	0.01	0.02
24	37	6	34.0	5	0.15	0.90	0.80	0.07	0.08	0.07	0.03	0.04
52 +	26	23	14.5	3	0.21	0.79	0.60	-	-	0.10	-	-

The Median Survival Time for these data is 52 + weeks.

Table 5.6.4 **Comparison of survival experience using the Lee-Desu Statistic- Survival variable: Study time -Time in study Treatment groups.**

Treatment Group	n	Uncensored	Censored	% Censored	Geometric Mean score
Immobilised grp	71	42	29	40.85	-30.09
Active exercise grp	60	13	47	78.33	8.90
Control grp	89	13	76	85.39	18.00

(F=21.597, D.F-2, p=.0000*) * = **Statistically significant**

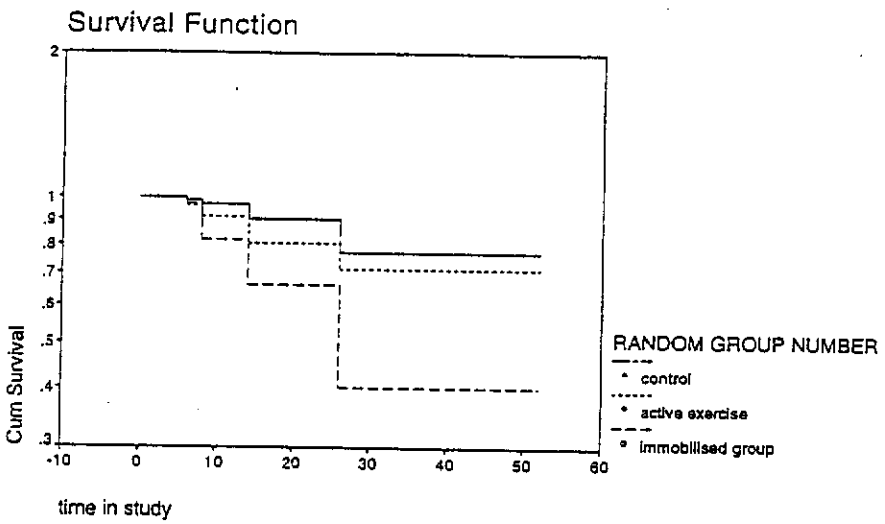

Figure 5.6.1 **The survival curves to illustrate the differences between treatment groups in reaching "0" pain level.**

5.7 Strength of the pre-and post-vertebral neck muscles

5.7.1 Descriptive statistics

The descriptive statistics for the pre- and post-vertebral neck muscles for each of the time periods are presented in the Tables 5.7.1 to 5.7.4. The number of subjects who were tested at the 4th week follow-up examination was relatively smaller when compared to the successive examinations and due to this reason relevant ANOVA and ANCOVA results corresponding to the 6th, 12th, 24th and 52 week follow-up examinations are considered in this section. The mean value of the peak force generated during the isometric contraction of the pre-vertebral muscles showed a gradual increase by small increments from the 6th week follow-up examination (6.331 kgs) up to the final follow-up examination (7.653 kgs) (Table 5.7.1). Similarly, the differences between the standard deviation for the corresponding time periods were also marginal. In the case of the post-vertebral neck muscles, the mean of the peak force generated for the follow-up examination at week 6 was 7.823 kgs and there after showed a slight increase at week-12 and week 24 (8.195 kgs; 9.182 kgs, respectively), whereas at the final follow-up examination the mean value showed a slight fall (9.040 kgs). The standard deviations for the peak force of the post-vertebral for the respective time periods showed a slight variation (Table 5.7.3). The mean, upper and lower confidence limits (95%) for the pre- and post-vertebral muscle peak force are provided in the Tables (Table 5.7.2, 5.7.4).

Table 5.7.1 **Descriptive statistics for the study population with reference to the pre-vertebral peak force (kgs).**

Time scale	n	Mean	S.D	Minimum	Maximum
4-Weeks	18	4.33	4.28	0	19
6-Weeks	130	6.33	3.12	0	18
12-Weeks	123	6.79	3.15	1	17
24-Weeks	99	7.35	3.57	1	18
52-Weeks	101	7.65	3.57	2	20

Table 5.7.2 **Mean, upper and lower 95% confidence limits of the mean for the pre-vertebral peak force (kgs) with reference to respective time periods.**

Time scale	n	Mean	95% Upper limit	95% Lower limit
4-Weeks	18	4.33	6.46	2.20
6-Weeks	130	6.33	6.87	5.79
12-Weeks	123	6.79	7.35	6.24
24-Weeks	99	7.35	8.07	6.64
52-Weeks	101	7.65	8.36	6.94

Table 5.7.3 **Descriptive statistics for the study population with reference to the post-vertebral peak force-(kgs).**

Time scale	n	Mean	S.D	Minimum	Maximum
4-Weeks	15	7.00	3.91	3	16
6-Weeks	130	7.82	3.87	0	18
12-Weeks	123	8.19	3.52	2	18
24-Weeks	99	9.18	3.99	2	20
52-Weeks	101	9.04	4.00	1	19

Table 5.7.4 **Mean, upper and lower 95% confidence limits of the mean for the post-vertebral peak force (kgs) with reference to respective time periods.**

Time scale	n	Mean	95% Upper limit	95% Lower limit
4-Weeks	15	7.00	9.15	4.85
6-Weeks	130	7.82	8.49	7.16
12-Weeks	123	8.19	8.81	7.57
24-Weeks	99	9.18	9.98	8.38
52-Weeks	101	9.04	9.84	8.24

5.7.2 Differences between groups for pre-vertebral muscle peak force

A one-way ANOVA test was performed for each of the time periods to test the null hypothesis that there was no statistically significant difference in muscle strength, between the treatment groups. When the respective p value was equal to or less than .05, demonstrating statistically significant differences between the groups, *post hoc* multiple comparison testing using the Scheffe's procedure was performed to identify the treatment groups with significant differences. The summary of ANOVA results and the Scheffe's procedure are provided for each of the time periods. The mean derived from the Scheffe's procedure are tabulated and identified to demonstrate significant differences when present.

The ANOVA results for the pre-vertebral muscles presented in the Table 5.7.5 showed that the p value for the 6th week follow-up examination was statistically significant (F=3.6071; p=.0299), whereas p values for the successive follow-up examinations were larger than .05. These results demonstrated that there were statistically significant differences between the treatment groups at 6 weeks and thereafter the differences between groups were not statistically significant.

Table 5.7.5 Summary of ANOVA results for the pre-vertebral muscles peak force.

Time periods	Source	D.F	SS	MS	F	P
Week-4	Between groups	2	21.63	10.81	0.56	0.5835
	Within groups	15	290.38	19.36		
Week-6	Between groups	2	67.55	33.78	3.61	0.0299*
	Within groups	127	1189.22	9.36		
Week-12	Between groups	2	20.22	10.11	1.01	0.3644
	Within groups	120	1191.70	9.94		
Week-24	Between groups	2	67.17	33.58	2.73	0.0701
	Within groups	96	1179.46	12.29		
Week-52	Between groups	2	61.82	30.91	2.50	0.0875
	Within groups	98	1213.10	12.38		

*** = Statistically significant**

The Scheffe procedure identified that the immobilised group significantly differed from the active exercise and control groups and the tabulated mean of the peak force generated clearly showed that the subjects in the immobilised group were able to generate a significantly stronger muscle contraction (7.2157) when compared to the control group (5.6304). The smaller difference between the immobilised group and the active exercise group (Mean = 5.94) was not significant (Table 5.7.6).

Table 5.7.6 **Summary of Scheffe procedure to demonstrate the treatment group with significant difference in peak force (pre-vertebral muscles)- Time period -week-6 post injury.**

Mean(kgs)	Group	Grp-3	Grp-2	Grp-1
5.6	Grp-3			
5.9	Grp-2			
7.2	Grp-1	*		

* = Statistically significant
Group 1= Immobilised group
Group 2= Active exercise group
Group 3= Control group

The observed differences in the peak force may not necessarily mean that the subjects in the immobilised group have gained strength as a result of the treatment received. A plausible explanation for this observation is that the subjects in the immobilised group were able to produce a stronger contraction only because they had less pain during the manoeuvre. This explanation was put to test by performing an ANCOVA using the pain score for the relevant time period as a covariate and the results are presented in the Table 5.7.7. The tabulated p values for each of the corresponding follow-up examinations were smaller than .05 for the covariate pain and larger than .05 for the association between treatment group and pre-vertebral isometric strength, thus demonstrating that the pain factor has been a significant covariate and that the earlier explanation in relation to the effect of the pain over isometric contraction appears to be valid.

Table 5.7.7 **Summary of ANCOVA results for the pre-vertebral muscles using pain level for the corresponding time period as a covariate.**

Time Period-	Source of variation	SS	DF	MS	F	p
week-4	Treatment	5.53	2	2.77	0.150	0.862
	Pain -4wks	36.05	1	36.05	1.955	0.190
	Residual	202.81	11	18.44		
week-6	Treatment gps	12.21	2	6.11	0.702	0.497
	Pain-6wks	149.39	1	149.39	17.187	0.000*
	Residual	1095.18	126	8.69		
week-12	Treatment gps	6.05	2	3.02	0.345	0.709
	Pain -12wks	162.40	1	162.41	18.522	0.000*
	Residual	1043.46	119	8.77		
week-24	Treatment gps	13.09	2	6.54	0.556	0.575
	Pain 24wks	115.59	1	115.59	9.823	0.002*
	Residual	1117.95	95	11.77		
week-52	Treatment gps	13.07	2	6.53	0.549	0.579
	Pain 52wks	108.01	1	108.01	9.080	0.003*
	Residual	1153.79	97	11.89		

*** = Statistically significant**

5.7.3 Differences between groups for post-vertebral muscle peak force

A similar analysis was performed to examine the between group differences for the post-vertebral muscle peak force and the results of the ANOVAs are summarised in the Table 5.7.8. In this instance the p values were statistically significant for the 6th week and 52 week follow-up examinations (F=4.72, p=.0105; F=5.51, p=.0054 respectively), showing that there were significant differences between the treatment groups for the respective time periods. A subsequent Scheffe's procedure identified that for the corresponding time periods, the mean value of the peak force generated by the subjects in the immobilised group was greater than the active exercise and the control groups (Table 5.7.9; 5.7.10). The Scheffe's procedure also showed that the difference between the immobilised and the control groups were statistically significant at week 6 and week 52 post

injury. Based on similar argument put forward in the case of the pre-vertebral muscles, a series of ANCOVAs were conducted using pain score for the corresponding time periods and the results shown in the Table 5.7.11 demonstrated that the pain was a significant covariate. When the effect of the covariate "pain" was statistically controlled, the significant between group differences demonstrated by the ANOVA no longer existed. Thus, the between group differences can be attributed to the level of pain experienced by the respective subject and this in turn reflected the efficacy of the treatment received by the subjects in the respective groups.

However, the main interest attached to the above analyses is to test the stated hypothesis that the subjects immobilised in a Philadelphia collar for four weeks are unlikely to develop muscle weakness due to disuse and the statistical analyses are in favour of the above hypothesis. The results of the proceeding analyses support the stated hypothesis.

Table 5.7.8 **Summary of ANOVA results for the post-vertebral muscles peak force.**

Time periods-	Source	DF	SS	MS	F	p
Week-4	Between groups	2	4.50	2.25	0.12	0.8803
	Within groups	12	209.50	17.46		
Week-6	Between groups	2	133.30	66.67	4.72	0.0105*
	Within groups	127	1793.58	14.12		
Week-12	Between groups	2	11.87	5.93	0.48	0.6224
	Within groups	120	1495.45	12.46		
Week-24	Between groups	2	79.36	39.67	2.57	0.0820
	Within groups	96	1483.37	15.45		
Week	Between groups	2	162.15	81.07	5.51	0.0054*
	Within groups	98				

*** = Statistically significant**

Table 5.7.9 **Summary of Scheffe procedure to demonstrate the treatment group with significant difference in peak force (post-vertebral muscles)- Time period -week-6 post-injury.**

Mean (kgs)	Group	Grp-3	Grp-2	Grp-1
6.91	Grp-3			
7.15	Grp-2			
9.08	Grp-1	*		

Table 5.7.10 **Summary of Scheffe procedure to demonstrate the treatment group with significant difference in peak force (post-vertebral muscles)- Time period -week-52 post-injury.**

Mean (kgs)-	Group	Grp-3	Grp-2	Grp-1
8.0	Grp-3			
8.21	Grp-2			
10.70	Grp-1	*		

*= Statistically significant
Group 1= Immobilised group
Group 2= Active exercise group
Group 3= Control group

Table 5.7.11 **Summary of ANCOVA results for the post-vertebral muscles using pain level for the corresponding time period as a covariate.**

Time Period-	Source of variation	SS	DF	MS	F	p
Week-4	Treatment	8.98	2	4.49	0.27	0.768
	Pain -4wks	22.44	1	22.44	1.35	0.270
	Residual	182.58	11	16.59		
Week-6	Treatment gps	29.65	2	14.63	1.13	0.326
	Pain-6wks	269.38	1	269.38	20.85	0.000*
	Residual	1628.28	126	12.92		
Week-12	Treatment gps	24.43	2	12.21	1.15	0.318
	Pain -12wks	226.94	1	226.94	21.50	0.000*
	Residual	1255.94	119	10.55		
Week-24	Treatment gps	5.54	2	2.77	0.20	0.816
	Pain 24wks	269.71	1	269.71	19.90	0.000*
	Residual	1287.48	95	13.55		
Week-52	Treatment gps	1.89	2	0.95	0.074	0.929
	Pain 24wks	356.04	1	356.04	27.72	0.000*
	Residual	1245.90	97	12.84		

* = Statistically significant

5.8 The range of motion of the cervical spine

5.8.1 Descriptive statistics

The mean values, standard deviations, minima, maxima, upper and lower confidence limits for the active range of motion of flexion, extension, side flexions and rotations for each of the time periods pertaining to the total sample are presented in the Table A.III.8.1 to A.III.8.2 and similar information for the respective treatment groups are summarised in the Tables A.III.8.3 to A.III.8.8.

The tabulated mean values of the range of motion for the total study population showed that noticeable changes were occurring at 6 weeks post injury, for each of the active movements and this trend was better demonstrated by a comparison of respective mean for each of the treatment groups.

A comparison of the mean obtained from the initial examination, for all the movements showed that the subjects in the active exercise group were able to move their necks relatively more than the subjects in the other treatment groups (Act ex grp > immob grp > cont grp). A similar comparison of the mean for the week-4 follow-up examination demonstrated that the subjects in the active exercise group had a greater range of flexion and extension when compared to the remainder of the groups (act ex grp > Immob grp > cont grp). Similarly, the range of motion for the right and left side flexion and right rotation were higher in the active exercise group followed by immobilised and control groups (act ex grp> Immob grp > cont grp). The mean of the left rotation were similar for the immobilised and active exercise groups while the control group recorded a relatively lower mean value(Tables A.III.8.3, A.III.8.5, A.III.8.7). The mean values of the range of motion derived from the week-6, 12, 24 and 52 follow-up

examination for each of the active movements, clearly showed that the subjects in the immobilised group were progressively showing a greater ability to move their necks in all directions when compared to the active exercise group which in turn was trailed by the control group.

5.8.2 Correlation between active vs passive Range of Motion and right vs left sides

A comparison between the active range of motion and corresponding passive range of motion was performed in order to establish the strength of correlation between the above variables and the results are presented in the Table A.III.8.9. The respective p values were statistically significant and the tabulated correlation coefficients demonstrate a very strong association between the active and passive range of motion for flexion, extension, side flexions and rotations (Portney and Watkins, 1993). A similar comparison between the right and left side flexion and right and left rotation, demonstrated a strong association between right and left side flexions and right and left rotations (Table A.III.8.10). Therefore, it was decided only to perform a series of ANOVA for each of the time periods, using the measurements obtained from active flexion, extension, right side flexion and right rotation in order to minimise the number of tests of hypothesis conducted.

5.8.3 Differences in the Range of Motion between groups

A one-way ANOVA test was performed for each of the time periods to test the null hypothesis that there was no statistically significant difference in the range of motion between the treatment groups. When the respective p value was equal to or less than .05, demonstrating significant differences between the groups, *post hoc* multiple comparison testing using the Scheffe's procedure was performed to identify the treatment groups with significant differences. The summary of ANOVA results and the Scheffe's procedure are provided for each of the time periods. The means derived

from the Scheffe's procedure are tabulated and identified to demonstrate significant differences when present.

The tabulated ANOVA results showed that there were statistically significant differences between the groups for all of the movements tested at the initial examination (Table 5.8.1) and the *post hoc* multiple comparison test viz the Scheffe's procedure demonstrated that the immobilised group was significantly different from the active exercise group (Table 5.8.2). A comparison of the mean of the range of motion for flexion, extension, right side flexion and right rotation showed that the subjects in the immobilised group (25.99°, 24.26°, 22.39°, 42.75°, respectively) were smaller than the active exercise group (31.75°, 30.50°, 27.41°, 54.91°, respectively).

Table 5.8.1 **ANOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine (Initial examination).**

Movements	Source of variation	SS	DF	MS	F	P
Flexion	Groups	1167.82	2	583.90	4.43	0.013*
	Residual	28615.52	217	131.87		
Extension	Groups	1325.82	2	662.91	5.41	0.005*
	Residual	26593.61	217	122.55		
Side flexion-right	Groups	884.27	2	442.14	5.07	0.007*
	Residual	18916.98	217	87.18		
Rotation-right	Groups	4853.61	2	2426.87	8.90	0.000*
	Residual	59125.82	217	272.47		

*** = Statistically significant**

Table 5.8.2 **Summary of Scheffe's procedure for ANOVAs -initial examination.**

Mean -	Grps Flex	Grp-1	Grp-2	Grp-3
25.99	Grp-1			
29.91	Grp-3			
31.75	Grp-2	*		

Mean	Grps Ext	Grp-1	Grp-2	Grp-3
24.30	Grp-1			
25.95	Grp-3			
30.50	Grp-2	*		

Mean -	Grps Sd flx	Grp-1	Grp-2	Grp-3
22.39	Grp-1			
23.59	Grp-3			
27.41	Grp-2	*		

Mean	Grps Rot	Grp-1	Grp-2	Grp-3
42.75	Grp-1			
49.15	Grp-3			
54.91	Grp-2	*		

* = Statistically significant Group 1= Immobilised group
Group 2= Active exercise group Group 3= Control group

Similarly, the ANOVAs results for the 4th week follow-up examination presented in Tables 5.8.3 also demonstrated statistically significant differences for all the movements but this time the control group was showing significant differences when compared to the remainder of the groups. The Scheffe procedure showed that the mean for each of the movements of the control group was significantly lower than the active exercise and immobilised groups (Table 5.8.4).

Table 5.8.3 **ANOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine-Week-4.**

Movements	Source of variation	SS	DF	MS	F	P
Flexion	Groups	1740.31	2	870.15	10.59	0.000*
	Residual	13069.72	159	82.20		
Extension	Groups	1722.39	2	861.19	9.99	0 .000*
	Residual	13712.17	159	86.24		
Side flexion -right	Groups	1054.01	2	527.00	8.19	0.000*
	Residual	10220.84	159	62.28		
Rotation-right	Groups	3015.91	2	1507.95	9.66	0.000*
	Residual	24814.48	159	156.07		

*= Statistically significant

Table 5.8.4 **Summary of Scheffe's procedure for ANOVAs -Week-4.**

Mean	Grps Flex	Grp-3	Grp-1	Grp-2
33.31	Grp-3			
39.83	Grp-1	*		
40.45	Grp-2	*		

Mean -Wk-4-	Grps Ext	Grp-3	Grp-1	Grp-2
31.02	Grp-3			
37.46	Grp-1	*		
38.18	Grp-2	*		

Mean	Grps Sd flx	Grp-3	Grp-2	Grp-1
26.61	Grp-3			
31.59	Grp-2	*		
32.11	Grp-1	*		

Mean -Wk-4-	Grps Rot	Grp-3	Grp-1	Grp-2
54.83	Grp-3			
63.55	Grp-1	*		
64.09	Grp-2	*		

*= Statistically significant
Group 2= Active exercise group
Group 1= Immobilised group
Group 3= Control group

The ANOVAs corresponding to the 6th week follow-up examination showed that statistically significant differences were present for each of the movements (Table 5.8.5) and the Scheffe's procedure identified that the flexion, extension, right side flexion and right rotation (mean values- 43.66°, 41.66°, 35.17°, 68.33°, respectively, Table 5.8.6) of the subjects in the immobilised group were significantly more than that of the control group (37.22°, 34.81°, 29.04°, 61.02°, respectively). It was evident at this stage that the application of Philadelphia collar. during the acute stage of the injury, has not adversely affected the neck movements of the subjects in the immobilised group.

Table 5.8.5 **ANOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine.-Week-6.**

Movements	Source of variation	SS	DF	MS	F	p
Flexion	Groups	1190.88	2	595.44	7.92	0.001*
	Residual	11126.67	148	75.18		
Extension	Groups	1337.04	2	668.52	8.68	0.000*
	Residual	13399.05	148	77.02		
Side flexion-right	Groups	1070.43	2	535.22	10.94	0.000*
	Residual	7243.34	148	48.94		
Rotation-right	Groups	1533.33	2	766.67	7.01	0.001*
	Residual	16191.50	148	109.40		

Table 5.8.6 **Summary of Scheffe's procedure for ANOVAs -Week-6.**

Mean -Wk-6	Grps Flex	Grp-3	Grp-1	Grp-2
37.22	Grp-3			
40.0	Grp-2			
43.66	Grp-1	*		

Mean -Wk-6	Grps Ext	Gr-3	Grp-2	Grp-1
34.81	Grp-3			
38.10	Grp-2			
41.66	Grp-1	*		

Mean -Wk-6	Grps Sd flx	Grp-3	Grp-2	Grp-1
29.04	Grp-3			
32.57	Grp-2			
35.17	Grp-1	*		

Mean -Wk-6	Grps Rot	Grp-3	Grp-2	Grp-1
61.02	Grp-3			
65.54	Grp-2			
68.33	Grp-1	*		

* = Statistically significant
Group 1= Immobilised group
Group 2= Active exercise group
Group 3= Control group

The tabulated ANOVA results for the 12th week follow-up examination were statistically significant (Table 5.8.7) and the Scheffe's procedure (Table 5.8.8) identified that the active flexion, right side flexion and right rotation were significantly different between immobilised (44.56°, 36.22°, 71.4°, respectively) and control groups (39.8°, 31.9°, 64°, respectively), whereas in the case of extension the control group (36.5°) was significantly different to the immobilised and active exercise groups (42.19°, 41.19°, respectively). It was evident from these results that the use of the Philadelphia collar has not resulted in a reduction of the range of motion of the cervical spine.

Table 5.8.7 **ANOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine- Follow-up week-12.**

Movements	Source of variation	SS	DF	MS	F	P
Flexion	Groups	604.02	2	302.01	5.11	0.007*
	Residual	8622.15	146	59.06		
Extension	Groups	946.15	2	473.07	7.35	0.001*
	Residual	9403.85	146	64.41		
Side flexion-right	Groups	511.07	2	255.54	5.95	0.003*
	Residual	6272.82	146	42.97		
Rotation-right	Groups	1485.48	2	742.74	7.51	0.001*
	Residual	14440.69	146	98.91		

***= Statistically significant**

Table 5.8.8 **Summary of Scheffe's procedure for ANOVAs -Week-12.**

Mean -Wk-12	Grps Flex	Grp-3	Grp-2	Grp-1
39.8	Grp-3			
42.26	Grp-2			
44.56	Grp-1	*		

Mean -Wk-12	Grps Ext	Grp-3	Grp-2	Grp-1
36.50	Grp-3			
41.19	Grp-2	*		
42.19	Grp-1	*		

Mean -Wk-12	Grps Sd flx	Grp-3	Grp-2	Grp-1
31.9	Grp-3			
33.57	Grp-2			
36.22	Grp-1	*		

Mean -Wk-12	Grps Rot	Grp-3	Grp-2	Grp-1
64.00	Grp-3			
67.02	Grp-2			
71.40	Grp-1	*		

*= Statistically significant

The ANOVA results for the 24th week follow-up examination also showed statistically significant differences between groups for each of the movements analysed (Table 5.8.9) and the Scheffe's (Table 5.8.10) procedure demonstrated that the differences were significant for flexion, extension and right rotation between immobilised group (45.52°, 42.91°, 72.08°, respectively) and control group (40.77°, 39.31°, 67.86°, respectively). In the case of right side flexion, the immobilised group (37.6°) was significantly different to the active exercise group (34.13°) and the control group (32.44°). The Scheffe's procedure clearly demonstrated that the subjects in the immobilised group were not adversely affected by the use of Philadelphia collar.

Table 5.8.9 **ANOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine.-Week-24.**

Movements	Source of variation	SS	DF	MS	F	P
Flexion	Groups	537.08	2	268.54	6.67	0.002*
	Residual	4790.79	119	40.26		
Extension	Groups	796.86	2	398.43	8.54	0.000*
	Residual	5552.32	119	46.66		
Side flexion-right	Groups	639.11	2	319.56	10.46	0.000*
	Residual	3634.04	119	30.54		
Rotation-right	Groups	1046.18	2	523.09	6.40	0.002*
	Residual	9724.23	119	81.72		

* = Statistically significant

Table 5.8.10 **Summary of Scheffe's procedure for ANOVAs -Wk-24.**

Mean -Wk-24	Grps Flex	Grp-3	Grp-2	Grp-1		Mean -Wk-24	Grps Ext	Grp-3	Grp-2	Grp-1
40.77	Grp-3					37.11	Grp-3			
42.41	Grp-2					39.31	Grp-2			
45.52	Grp-1	*				42.91	Grp-1	*		

Mean -Wk-24	Grps Sd flx	Grp-3	Grp-2	Grp-1		Mean -Wk-24	Grps Rot	Grp-3	Grp-2	Grp-1
32.44	Grp-3					65.44	Grp-3			
34.13	Grp-2					67.86	Grp-2			
37.60	Grp-1	*	*			72.08	Grp-1	*		

* = Statistically significant
Group 1= Immobilised group
Group 2= Active exercise group
Group 3= Control group

The ANOVA results for the week-52 follow-up examination were statistically significant for each of the movements analysed, thus showing there were significant differences between groups (Table 5.8.11) and the Scheffe's procedure (Table 5.8.12) identified that the differences for the extension and right side flexion existed between the immobilised group (47.5°, 37.62°, respectively) and the control group (41°, 31.55°, respectively). It was also evident from the Scheffe's procedure that the extension and right rotation were significantly different between the immobilised group (44.64°, 73.45°, respectively) and the remainder of the groups (active ex grp-39.64°, 67.86°, respectively; cont grp- 37.33°, 66.44°, respectively). The above results demonstrated that the statistically significant differences evidenced from the preceding analyses were in favour of the immobilised group and that the use of Philadelphia collar has no adverse effect on the subjects ability to move their necks.

Table 5.8.11 **ANOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine.-Week-52.**

Movements	Source of variation	SS	DF	MS	F	P
Flexion	Groups	929.60	2	464.80	8.91	0.000*
	Residual	5845.18	112	52.19		
Extension	Groups	1192.19	2	596.09		0.000*
	Residual	6346.07	112	56.66		
Side flexion-right	Groups	559.69	2	279.84	9.98	0.000*
	Residual	3139.44	112	28.03		
Rotation-right	Groups	1149.14	2	574.57	8.17	0.000*
	Residual	7876.94	112	70.33		

*** = Statistically significant**

Table 5.8.12 **Summary of Scheffe's procedure for ANOVAs -Week-52.**

Mean	Grps Flex	Grp-3	Grp-2	Grp-1		Mean -Wk-52	Grps Ext	Grp-3	Grp-2	Grp-1
41	Grp-3					37.33	Grp-3			
43.39	Grp-2					39.64	Grp-2			
47.5	Grp-1	*				44.64	Grp-1	*	*	

Mean	Grps Sd flx	Grp-3	Grp-2	Grp-1		Mean -Wk-52	Grps Rot	Grp-3	Grp-2	Grp-1
32.55	Grp-3					66.44	Grp-3			
34.64	Grp-2					67.86	Grp-2			
37.62	Grp-1	*				73.45	Grp-1	*	*	

* = Statistically significant Group 1= Immobilised group
Group 2= Active exercise group Group 3= Control group

5.8.4 The effect of age and pain as covariates

The factors that might contribute to a reduction in active range of motion is of clinical interest. Hence, a series of ANCOVAs was performed for each of the time periods, using age and level of pain for the corresponding time period, to determine the effect of the above factors on the range of motion of the cervical spine and the summary of the results are presented.

The age was not a significant covariate for the initial examination time period, whereas the pain factor was statistically significant for each of the movements analysed, denoted by p values smaller than .05, (Table A.III.8.11). It was evident that when the pain factor was taken into account any differences that might have existed between groups disappeared, thus, demonstrating that the limitation of flexion, extension and right side flexion was attributable to the pain factor and not due to the age related differences. The ANCOVAs for the remainder of the time periods demonstrated that the age and level of pain for the corresponding time periods were significant covariates (Table A.III.8.12 to A.III.8.16) indicating that any differences that might have existed between the treatment groups, in the range of motion for each of the movements analysed, are more likely attributable to the differences in age and also the level of pain experienced by the subjects.

5.8.5 Changed scores

From the ANOVA results of the initial examination (Table 5.8.1) and corresponding Scheffe's procedure (Table 5.8.2), it was clearly evident that the range of motion of active flexion, extension, right side flexion and right rotation, between treatment groups were not equal. The Scheffe's procedure identified that the mean of the immobilised group was the least of the three groups, thus, significantly different when compared to the active exercise group. Hence, it was obvious that the randomisation may not have been effective in formulating groups with equal amount of range of motion. In spite of the noted statistically significant difference at the initial examination, further ANOVAs and Scheffe's procedure for the subsequent follow-up time periods up to the end of the study, showed that the immobilised group demonstrated a significant increase in the range of motion. It could be argued that as noticed in the case of the immobilised group, the proportion of change from a smaller range as noted at the initial

examination to a greater range of motion at subsequent time periods, is likely to contribute towards significant differences in favour of the immobilised group, as evidenced from the ANOVAs. In order to determine whether a situation as stated above might have occurred, a new dependent variable was created by subtracting the initial examination range of motion of the respective movements from the corresponding time period viz "change in initial examination range of motion" (also known as change score). The use of change score is recommended for removing stable individual differences between subjects which in turn would increase the power of the statistical procedure (Norman, 1989). In addition, the change score can also be used to correct the differences that existed between randomised groups at the initial examination itself (Norman, 1989). Thus, having removed the selection bias introduced by the randomisation, a multiple regression was performed for each of the time periods, to analyse the relationship between the dependent variable (change scores) and a set of predictor variables which were shown to have significant effect on the treatment groups viz:

1. age;
2. pain at initial examination;
3. treatment factor- active exercise regimen; and
4. treatment factor- immobilisation regimen.

The result of the corresponding multiple regression of the flexion, extension, right side flexion and right rotation at each of the time periods showed that the p value for the F statistics was smaller than .05, thus demonstrating that the variance attributable to the regression was significant (Table A.III.8.17 to A.III.8.56). The regression coefficient (B) for each of the independent variables (X) describe the weight contributed by the respective variable to the explanation of the dependent variable (Y).

The standardised regression coefficients are indicative of the relative importance of a specific variable within the regression equation.

As it can be seen from the summary of the results in the respective tables, the immobilisation regimen was statistically significant and also relatively an important predictor variable. Multiple regression analysis for each of the follow-up time periods to the respective movements, except for right side flexion at week- 52, consistently showed that of all the predictor variables, the immobilisation regimen had a relatively higher standardised coefficient which was statistically significant ($p = < .05$) thus, indicating that the above variable was a relatively important predictor variable (Table A.III.8.17 to A.III.8.56).

The age of the subjects was also identified as a significant predictor variable in the case of extension, right side flexion and right rotation at week-4. The results demonstrated that the age of the subjects had an effect on the range of motion of active extension at each time periods and the respective p value which was smaller than .05, indicated that in addition to the immobilisation regimen, the age of the subjects had a significant predictive role. The active exercise regimen was identified on one occasion at week-52 as a significant predictor variable in relation to right rotation and such an isolated occurrence of significance in multiple analysis should be disregarded. It is evident from these results that it can be concluded that earlier ANOVA results were not affected by the selection bias.

5.9 Return to work

5.9.1 Survival analysis

The summary of the Survival analysis and the relevant life table for each of the treatment groups are presented in Tables 5.9.1 to 5.9.4. In addition to

the actual number as well as the proportion of the subjects returning to their respective work 100% which is equivalent to the pre-accident level (*terminal event*), the time scale at which this event occurred (*interval start time*) and the proportion of the subjects who have not fully returned to their respective employment equivalent to pre-accident level, at a given time period (*proportion of survival*) can be obtained from the life tables. The cumulative proportion of survival for a given time interval denotes the over all percentage of the subjects who have not fully returned to their respective employment, with reference to a given time period from the start of the study. The hazard rate is the function of the time period and the covariate (i.e treatment regimen) thus, representing the dependence on the variables for each subject as well as on time intervals. The derived median survival times reflect the differences between the groups for achieving 100% of their respective pre-accident level of employment. The cumulative proportion of survival as noted at the end of the study period and the median survival time are the two important statistics presented in this section to describe the differences between the treatment groups.

The life table for the immobilised group showed that the cumulative proportion of the subjects who have survived at the end of the study period was 0.21 (Table 5.9.1). The inference was that 21% of the subjects from the immobilised group were working at a level less than 100% when compared to pre-accident level. The median survival time for the immobilised group was 7.65 weeks and this represents the average time period in which most of the changes were occurring thus, distinguishing those subjects who have returned to the same level of employment which was equivalent to the pre-accident level and others working less than their normal level. Similarly, the cumulative proportion of the subjects from the active exercise group, reported to be working less than their pre-accident level (i.e subjects survived at the end of the study period), was 0.30

meaning that 30 % of the subjects were working less than 100% of their pre-accident level of their respective employment (Table 5.9.2). The median survival time for the active exercise group was 10.2 weeks which meant that a considerable proportion of the subjects were still working less number of hours when compared to their pre-accident level of employment. The experience of the subjects in the control group was also similar to that of the active exercise group in that the cumulative proportion of the surviving subjects was 0.37 (i.e 37 % of the subjects have not returned to their respective employment at a level equivalent prior to the accident) with a median survival time of 20.4 weeks (Table 5.9.3).

The survival experience of the subjects for each of the treatment groups is graphically presented in Figure 5.9.1. A comparison of the survival experience using the Wilcoxon statistic showed that the observed differences between the treatment groups were not statistically significant. (F=3.237, D.F=2, p=.1982; Table 5.9.4). Even though there was no statistically significant differences between the treatment groups, the median survival time for the immobilised group (7.65 weeks) is relatively lower when compared to the active exercise and control groups (10.2 weeks, 20.4 weeks, respectively). It was clearly evident that 50% of the subjects from the immobilised group have returned, within a relatively shorter period, to their respective work in all respects equivalent to the pre-accident level. In contrast, the remainder of the groups were clearly lagging behind the immobilised group. The results support the hypothesis that the subjects in the immobilised group will return to their respective employment sooner than the remainder of the groups.

Table 5. 9.1 **Life table for the immobilised group survival variables: Study time -Time in study.**

n Inter-val Start time-	n Ente-ring this inter-val	n Withd-rawn dur-ing inter-val	n Expo-sed to risk	n Term-inal even-ts	Propn termi-nating	prop survi -ving	Cumu-lative prop survi-val at end	proba-bility Densi-ty	Hazard rate	SE of cumi-lative survi-ving	SE of prob Densi-ty	SE of haza-rd rate
0	71	0	71	10	0.14	0.86	0.86	0.07	0.08	0.04	0.02	0.02
2	61	0	61	7	0.11	0.89	0.76	0.05	0.06	0.05	0.02	0.02
4	54	10	49	11	0.22	0.78	0.59	0.09	0.12	0.06	0.02	0.04
6	33	1	32.5	6	0.18	0.80	0.48	0.05	0.10	0.06	0.02	0.04
8	26	0	26	1	0.04	0.96	0.47	0.01	0.02	0.06	0.01	0.02
10	25	0	25	1	0.04	0.96	0.44	0.01	0.02	0.06	0.01	0.02
12	24	2	23	2	0.09	0.91	0.41	0.02	0.05	0.06	0.01	0.03
14	20	0	20	2	0.10	0.90	0.36	0.02	0.05	0.06	0.01	0.04
16	18	0	18	0	0.00	1.00	0.36	0.00	0.00	0.06	0.00	0.00
18	18	0	18	0	0.00	1.00	0.36	0.00	0.00	0.06	0.00	0.00
20	18	0	18	2	0.11	0.89	0.32	0.02	0.06	0.06	0.01	0.04
22	16	0	16	1	0.06	0.94	0.30	0.01	0.03	0.06	0.01	0.03
24	15	2	14	0	0.00	1.00	0.30	0.00	0.00	0.06	0.00	0.00
26	13	0	13	0	0.00	1.00	0.30	0.00	0.00	0.06	0.00	0.00
28	13	0	13	0	0.00	1.00	0.30	0.00	0.00	0.06	0.00	0.00
30	13	0	13	0	0.00	1.00	0.30	0.00	0.00	0.06	0.00	0.00
32	13	0	13	1	0.08	0.92	0.28	0.01	0.04	0.06	0.01	0.04
34	12	0	12	1	0.08	0.92	0.26	0.01	0.04	0.06	0.01	0.04
36	11	0	11	0	0.00	1.00	0.26	0.00	0.00	0.06	0.00	0.00
38	11	0	11	0	0.00	1.00	0.26	0.00	0.00	0.06	0.00	0.00
40	11	0	11	2	0.18	0.82	0.21	0.02	0.10	0.06	0.02	0.07
42	9	0	9	0	0.00	1.00	0.21	0.00	0.00	0.06	0.00	0.00
44	9	0	9	0	0.00	1.00	0.21	0.00	0.00	0.06	0.00	0.00
46	9	0	9	0	0.00	1.00	0.21	0.00	0.00	0.06	0.00	0.00
48	9	0	9	0	0.00	1.00	0.21	0.00	0.00	0.06	0.00	0.00
50	9	0	9	0	0.00	1.00	0.21	0.00	0.00	0.06	0.00	0.00
52	9	9	4.5	0	0.00	1.00	0.21	0.00	0.00	0.06	0.00	0.00

Median Survival Time- 7.65 weeks

Table 5.9.2 **Life table for the active exercise group survival variables: Study time -Time in study.**

n Interval Start time-	n Entering this interval	n Withd -rawn during interval	n Exposed to risk	n Terminal events	Propn terminating	prop surviving	Cumulative prop survival at end	probability Density	Hazard rate	SE of cumilative surviving	SE of prob Density	SE of hazard rate
0.	60	0	60	10	0.17	0.83	0.83	0.08	0.09	0.05	0.02	0.03
2	50	0	50	10	0.20	0.80	0.67	0.08	0.11	0.06	0.02	0.03
4	40	13	33.5	4	0.12	0.88	0.59	0.04	0.06	0.07	0.02	0.03
6	23	2	22	2	0.09	0.90	0.53	0.03	0.05	0.07	0.02	0.31
8	19	0	19	0	0.00	1.00	0.53	0.00	0.00	0.07	0.00	0.00
10	19	0	19	2	0.11	0.89	0.48	0.03	0.06	0.07	0.02	0.04
12	17	0	17	1	0.06	0.94	0.45	0.01	0.03	0.07	0.01	0.03
14	16	0	16	0	0.00	1.00	0.45	0.00	0.00	0.07	0.00	0.00
16	16	0	16	0	0.00	1.00	0.45	0.00	0.00	0.07	0.00	0.00
18	16	0	16	1	0.06	0.94	0.42	0.01	0.03	0.07	0.01	0.03
20	15	0	15	1	0.70	0.93	0.39	0.01	0.03	0.07	0.01	0.03
22	14	0	14	0	0.00	1.00	0.39	0.00	0.00	0.07	0.00	0.00
24	14	3	12.5	2	0.16	0.84	0.33	0.03	0.09	0.07	0.02	0.06
26	9	0	9	0	0.00	1.00	0.33	0.00	0.00	0.07	0.00	0.00
28	9	0	9	0	0.00	1.00	0.34	0.00	0.00	0.07	0.00	0.00
30	9	0	9	0	0.00	1.00	0.34	0.00	0.00	0.07	0.00	0.00
32	9	0	9	1	0.11	0.89	0.30	0.02	0.06	0.07	0.02	0.06
34	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
36	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
38	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
40	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
42	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
44	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
46	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
48	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
50	8	0	8	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00
52	8	8	4	0	0.00	1.00	0.30	0.00	0.00	0.07	0.00	0.00

Median Survival Time- 10.2 weeks

Table 5.9.3 **Life table for the control group survival variables: Study time -Time in study.**

n Interval Start time-gp C WORK	n Entering this interval	n Withdrawn during interval	n Exposed to risk	n Terminal events	Propn terminating	prop surviving	Cumulative prop survival at end	probability Density	Hazard rate	SE of cumilative surviving	SE of prob Density	SE of hazard rate
0	89	0	89	9	0.10	0.89	0.89	0.05	0.05	0.03	0.02	0.02
2	80	0	80	12	0.15	0.85	0.76	0.07	0.08	0.05	0.02	0.02
4	68	24	56	3	0.05	0.95	0.72	0.02	0.03	0.05	0.01	0.02
6	41	6	38	3	0.08	0.92	0.07	0.03	0.04	0.06	0.01	0.02
8	32	0	32	0	0.00	1.00	0.67	0.00	0.00	-0.06	0.00	0.00
10	32	0	32	3	0.09	0.90	0.60	0.03	0.05	0.06	0.02	0.03
12	29	6	26	4	0.15	0.85	0.51	0.05	0.08	0.70	0.02	0.04
14	19	0	19	0	0.00	1.00	0.51	0.00	0.00	0.07	0.00	0.00
16	19	0	19	0	0.00	1.00	0.51	0.00	0.00	0.07	0.00	0.00
18	19	0	19	0	0.00	1.00	0.51	0.00	0.00	0.07	0.00	0.00
20	19	0	19	2	0.11	0.89	0.46	0.03	0.06	0.07	0.02	0.04
22	17	0	17	0	0.00	1.00	0.46	0.00	0.00	0.07	0.00	0.00
24	17	1	16.5	2	0.12	0.88	0.40	0.03	0.06	0.07	0.02	0.05
26	14	0	14	0	0.00	1.00	0.40	0.00	0.00	0.07	0.00	0.00
28	14	0	14	0	0.00	1.00	0.40	0.00	0.00	0.07	0.00	0.00
30	14	0	14	0	0.00	1.00	0.40	0.00	0.00	0.07	0.00	0.00
32	14	0	14	1	0.07	0.93	0.37	0.01	0.04	0.07	0.01	0.04
34	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
36	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
38	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
40	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
42	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
44	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
46	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
48	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
50	13	0	13	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00
52	13	13	6.5	0	0.00	1.00	0.37	0.00	0.00	0.07	0.00	0.00

Median Survival Time- 20.4 weeks

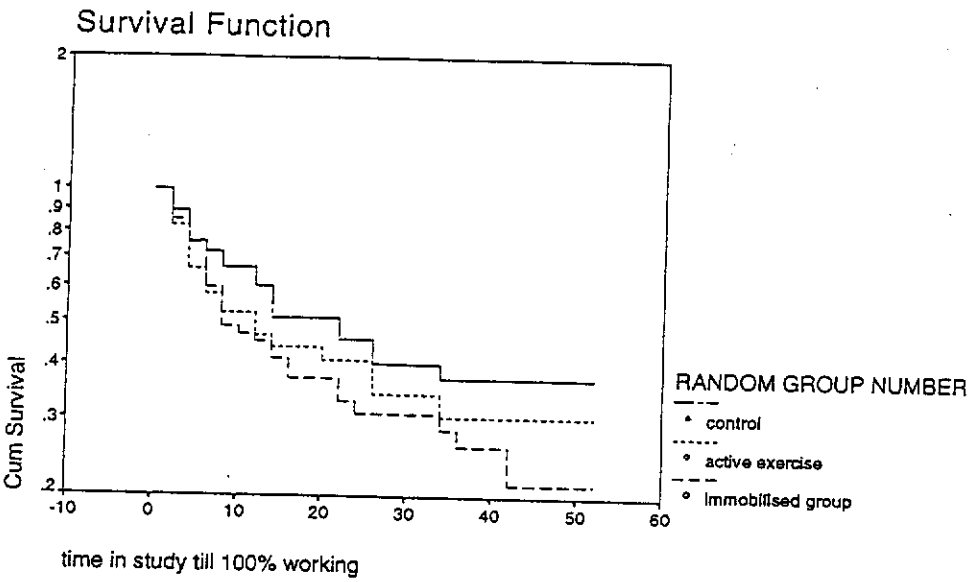

Figure 5.9.1 **The survival curves to illustrate the differences between treatment groups in relation to the subjects returning to their normal duties to a level equivalent (100%) to pre-accident level.**

Table 5.9.4 **Comparison of survival experience using the Wilcoxon Statistic- Survival variable: Study time -Time in study till 100% working Treatment groups.**

Treatment Group	n	Uncensored	Censored	% Censored	Mean score
Immobilised grp	71	47	24	33.80	-8.62
Active exercise grp	60	34	26	43.33	-13.62
Control grp	89	39	50	56.18	16.05

(F=3.24, D.F-2, p=.1982)

5.10 Matters of clinical interest

5.10.1 Results of the magnetic resonance imaging

The MRI performed within two weeks post-injury, demonstrated that soft tissue lesions were present among 10 out of 45 subjects examined soon after the injury and among them, with the exception one subject, the rest of the nine have had their lesions confined to a single motion segment (Table 5.10.1). It was also evident that the highest incidence of lesion occurred at the level of C5-6 followed by C4-5, whereas C3-4 and C6-7 levels were least affected. Even though there were four incidences of disruption at the junction of the anterior longitudinal ligament and the outer anular fibrosus (A.L.L-anular fibrosus complex), it was interesting to note that the posterior longitudinal ligament was intact in all subjects. Similarly, disc herniation causing indentation of the subarachnoid space and mild cord compression was evident among four subjects, although there was not a single incidence of compression or displacement of nerve root. A "Rim lesion" was clearly evident in one subject who demonstrated multilevel lesions of adjacent motion segments (Figure 5.10.1 to 5.10.3). The T-2 weighted images showed that three more subjects had high signals in the I.V.D which is indicative of injury and associated inflammatory reaction. One of the 45 subjects was shown to have pre-vertebral fluid collection (Figure 5.10.4, 5.10.5).

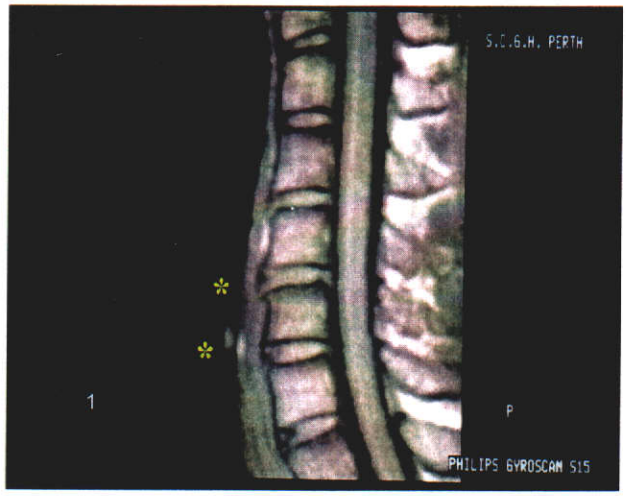

Figure 5.10.1 MRI performed within two weeks post-injury showing multi level disruption of ALL-anular fibrosus complex.

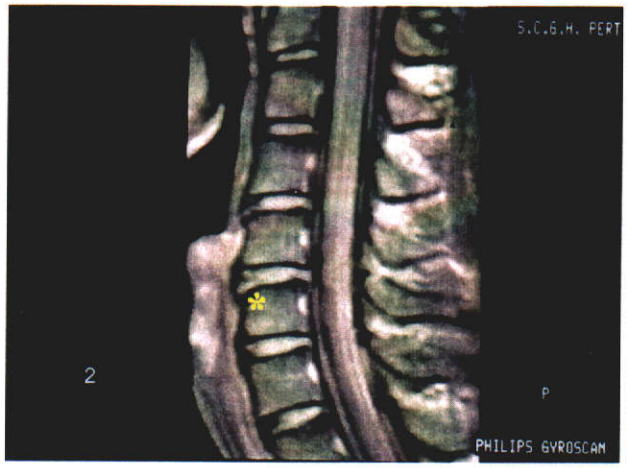

Figure 5.10.2 MRI performed after three months post-injury showing evidence of rim lesion.

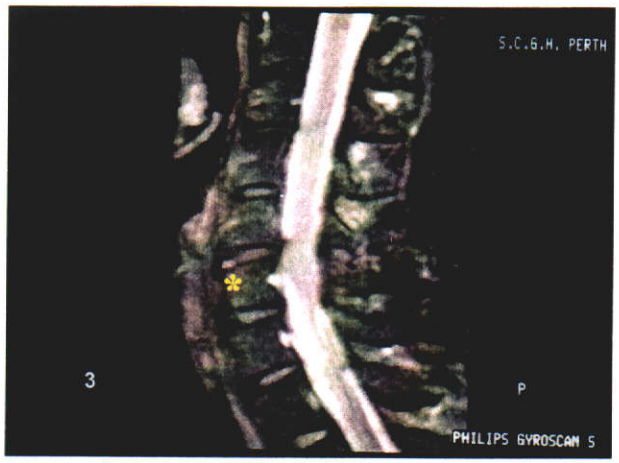

Figure 5.10.3 MRI performed after twelve months post-injury showing progressive degenerative change in the IVD.

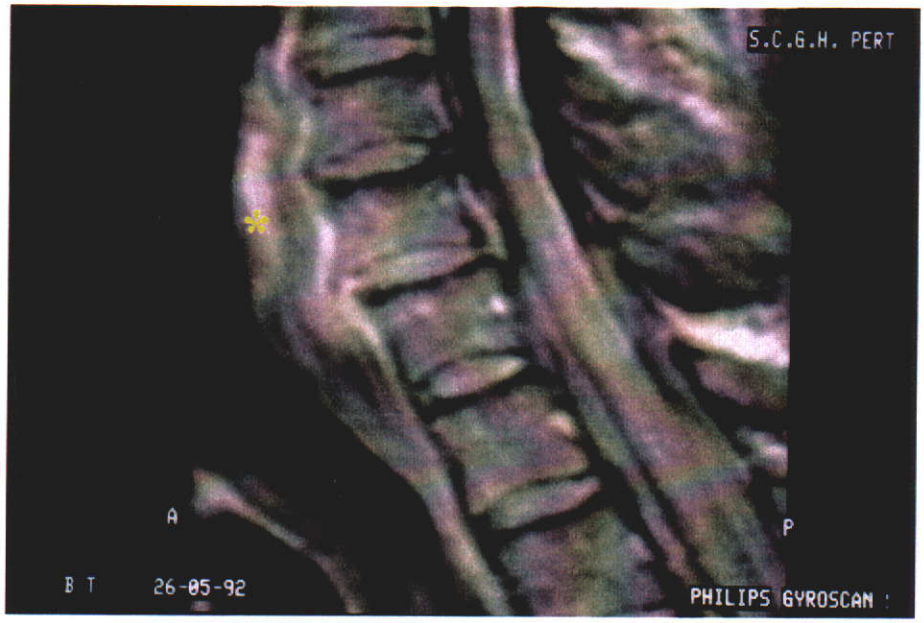

Figure 5.10.4 MRI performed within two weeks post-injury showing pre-vertebral swelling

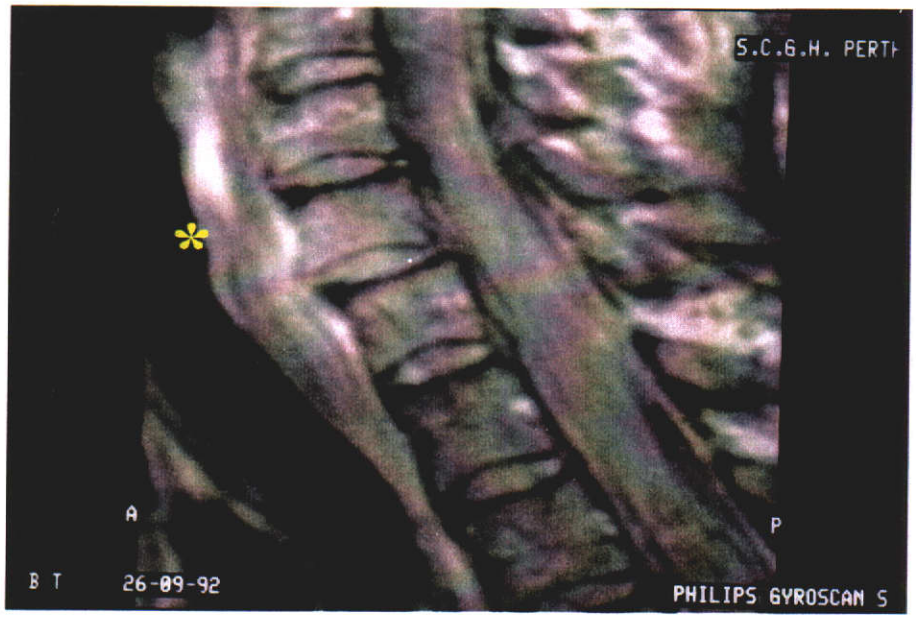

Figure 5.10.5 MRI performed after three months post-injury showing resolution of pre-vertebral swelling

The results of the follow-up M.R.I performed three months post-injury showed that the pre-vertebral fluid collection which was present in one of the subjects has resolved. Similarly, the lesion at the A.L.L-anular fibrosus complex noted among four subjects, appeared to have healed. The I.V.D lesions among four subjects noted in the first M.R.I, demonstrated a clear evidence of progressive changes and well defined cleft formation at the attachment of anular fibrosus and the end plate (rim lesion). A final follow-up M.R.Imaging of these subjects performed 12 months post-injury confirmed the presence of well defined rim-lesion suggesting that the healing of the I.V.D have not occurred.

Table 5.10.1 Abnormal magnetic resonance imaging findings.

No	Subject	Disc lesion	Anterior longitudinal ligament-anular fibre complex lesion	Level	Description
1	122	Yes	No	C5-6	Central disc herniation
2	146	Yes	No	C4-5	Broad based disc bulge indenting the ventral subarachnoid space
3	142	Yes	Yes	C4-5-6	C4-5-6 ALL-ANF complex disruption & C5-6 rim lesion
4	163	Yes	Yes	C6-7	ALL-ANF complex disruption
5	169	Yes	Yes	C6-7	Disruption of ALL-ANF complex;- disc material extending into the right C6-7 foramen; & pre-vertebral fluid collection
6	174	Yes	Yes	C5-6	Disruption of ALL-ANF complex; & anterior disc lesion
7	206	Yes	No	C3-4	Mild bulging indenting the anterior subarachnoid space
8	211	Yes	No	C5-6	Central broad based disc protrusion abutting the cord
9	213	Yes	No	C4-5	Minimal bulging
10	224	Yes	No	C3-4	Central disc protrusion abutting the cord

5.10.2 Low back pain

The frequency of low back pain as reported by the subjects presented for each of the time periods showed that in addition to "whiplash" type of

injuries, the subjects were also affected by low back pain. At the initial examination 36.4% of the subjects reported back pain and at the week- 4 follow-up examination showed an increase in the percentage of subjects with back pain (42.2%). The subsequent follow-up examinations at week-6, week-12, week-24 and week-52 showed that more than one quarter of the subjects continued to report that low back pain persisted (38.05%, 37.3%, 32.4%, 26.1%, respectively). The pain level reported by the subjects and corresponding frequencies are presented for each of the time periods (Table A.III.10.1).

5.10.3 Activities of Daily living (A.D.L):

The frequencies for A.D.L which were reported to be affected as a result of the MVA by the subjects are presented in Tables A.III.10.2 to A.III.10.6.

5.10.4 Headache

The association between headache and predictor variables viz:

1. speed of the subject's vehicle;
2. speed of the other vehicles / objects;
3. speed and types of collisions; and
4. types of collisions.

were analysed using a logistic regression model and the results showed that none of the predictor variables included in the analysis were statistically significant (Table A.III.10.7).

5.11 Summary

Series of data analyses were performed to test the hypotheses of this study and the following is a summary of the results for the relevant hypothesis.

5.11.1 Pain

A series of ANOVAs was performed for each of the time periods and the results showed that there was a statistically significant difference for the respective time periods. The *post hoc* multiple comparison test (Scheffe's procedure) for each of the time periods identified the treatment groups that were significantly different and the difference was characterised by the derived geometric mean for the respective treatment group.

At the initial examination the pain level for the immobilised group was significantly different when compared to the active exercise group. Although there was a difference between the immobilised and the control groups, it was not large enough to be statistically significant. In effect, the immobilised group had a higher level of pain to start with and yet demonstrated a reduction in pain that was consistent for each of the time period as well as statistically significant. In spite of randomisation, there were differences in the level of pain between groups and the observed differences may be attributed to various confounding variables. Further analyses were performed using the following covariates:

1. age;
2. gender ;
3. speed of the subject's vehicle;
4. speed of the other vehicle / object;
5. seating position;
6. type of collision;
7. baseline pain;
8. headache;

9. occupation;
10. paraesthesia;
11. blurred vision;
12. difficulty in focusing;
13. interscapular pain;
14. loss of cervical lordosis; and
15. degeneration of the I.V.D.

The effect of the covariates and their influence over the treatment outcome were tested. While none of the covariates had an interaction effect, there was a strong association between the pain and the following covariates:

1. age;
2. gender;
3. speed involved;
4. initial pain;
5. paraesthesia;
6. blurred vision;
7. difficulty in focusing;
8. loss of cervical lordosis; and
9. degeneration of the I.V.D.

A logistic regression was performed for each of the time periods using the above covariates which were shown to be significantly associated with the pain, as predictor variables, in order to identify the significant predictor variables and also to estimate the odds ratio for reaching a pain level of 0. The regression model determined that of all the predictor variables included in the model, the immobilisation regimen was the most consistent and significant predictor variable. In addition, it was also evident that the odds of reaching 0 pain level was considerably higher for those subjects in the immobilisation regimen when compared to the remainder of the treatment groups.

While the survival analysis graphically illustrates the proportion of changes occurring at a particular time scale, the accompanying life table clearly summarises the experience of the subjects in different treatment groups. The above results demonstrated the advantage of receiving treatment as defined in the immobilisation regimen and also support the hypothesis that the subjects in the immobilised group will recover quicker when compared to the other two groups.

5.11.2 Neck muscles strength

Similarly the results of the ANOVAs and relevant *post hoc* multiple comparison testing showed that the subjects in the immobilised group were not disadvantaged, by being in the Philadelphia collar, due to disuse atrophy of the neck muscles. The ANCOVAs indicated that observed statistically significant differences between groups were the result of the pain level experienced by the subjects. It was evident that a reduction in the pain level eventuated in stronger muscle contraction, which in turn indicated the advantage gained by the subjects by being in the immobilised group. The results of the statistical tests support the hypothesis that immobilisation of the neck in a Philadelphia collar for a period of 4 weeks is unlikely to produce muscle weakness in the long term.

5.11.3 Range of motion

The results of the ANOVAs indicated that the at the initial examination, there was a statistically significant difference between groups and the *post hoc* multiple comparison test showed that the immobilised had a significantly lesser range of motion. However, the respective ANOVA results for subsequent time periods clearly demonstrated that the immobilised group had significantly greater range of motion in the cervical spine. ANCOVA results showed that the age and level of pain for the corresponding time periods had an association with the range of motion,

thus, indicating that the above variables were significant covariates. The focus of the analysis is to determine whether the immobilisation has adversely affected the range of motion in the long term. The results of the statistical tests clearly lend support to the hypothesis that the use of Philadelphia collar for a period of 4 weeks is unlikely to contribute to a reduction in the range of motion in the cervical spine at 12 weeks post-injury.

5.11.4 Return to work

The survival analysis estimated that the return to work at 100% level (equivalent to the pre-accident) was achieved by the immobilised group sooner than the remainder of the groups. Similarly, the proportion of the subjects in the immobilised group returning to work at 100% level of their pre-accident level was also higher than the remainder of the groups. However, the observed differences were not statistically significant.

In a summary, all of the statistical analyses performed consistently demonstrated that the subjects in the immobilised group returned to normality sooner than those subjects in the active exercise and the control groups and that time taken by the immobilised group to achieve normality was statistically significant. Similarly, the respective analyses demonstrated that the immobilisation of the neck neither resulted in weakness of the neck muscle nor a reduction in the mobility of the cervical spine. The results also demonstrated that a relatively larger proportion of the subjects from the immobilised group returned to their respective work at a level that was 100% equivalent to the pre-accident level. It was evident that patients affected by "whiplash" type of injury and treated as per the immobilisation regimen are more likely to get better within a relatively shorter time scale when compared to those patients treated as indicated for the active exercise and the control groups.

CHAPTER SIX DISCUSSION

Whiplash an "Emotional term, common in the vocabulary frequently used by the attorneys, a term of derision by comedians, and sometimes the object of scorn by physicians" (Hirsch et al., 1988).

6.1 Introduction

The syndrome commonly referred as "whiplash injury" resulting from motor vehicle accidents is complex and remains a challenge to clinicians (MacNab, 1973; Hohl, 1975; Bogduk, 1986; Hildingsson et al., 1989; Radanov et al., 1991; Spitzer et al., 1995). The chronic nature of this syndrome and its adverse socio-economical impact has led recently to the establishment in Canada of a task force (Quebec Task Force) to analyse various aspects of the problems associated with "whiplash" syndrome and come up with clear directions for future research. The report was compiled by the Quebec Task Force and promulgated in the journal Spine (Supplement 24 S,1995) is primarily based on a critical review of the literature. Hence their recommendations in many respects reflect the observations made by several key authorities in the field of "whiplash" injury (Spitzer et al., 1995). The complexity associated with the clinical management of "whiplash" injury has been attributed to several factors (Spitzer et al., 1995). The most important of these include the lack of a clear understanding of the structures which are affected, the magnitude of the injury and the associated pathological changes (Frankel, 1953; Janes and Hooshmand, 1975; Bogduk, 1986; Pearce, 1989; Watkinson et al., 1991). In addition, the secondary financial gain often made from a "whiplash" injury has also been considered an important factor, responsible for the often poor

treatment outcome (Miller, 1961 and 1966; Awerbuch, 1992). There is a consensus that most of the treatment methods which are currently used in the management of "whiplash" injury are of questionable value and lacking in physiological rationale (Bogduk, 1995; Spitzer et al., 1995). Thus, there is a compelling need to evaluate the efficacy of the treatment methods that are widely practiced at present in the management of "whiplash" injury and the Quebec Task Force has made several recommendations in this regard.

It is a coincidence that the current study has adopted several key recommendations put forward by the Quebec Task Force.

6.1.1 The current study

The main objective of this prospective randomised study was to evaluate two conservative treatment regimens (early immobilisation-experimental group-1, early active mobilisation- experimental group-2) which are based on accepted physiological rationale and then to compare the effectiveness of their methodologies with existing treatment regimens that are commonly practiced (control group) in the management of "whiplash" type of injuries. To this stage, the current study is the only prospective randomised clinical trial of its type known to the investigator conducted with a sufficiently large sample size and over a long study period. The follow-up time interval (4, 6, 12, 24 and 52 weeks post-injury) adopted in the current study enabled adequate documentation of the pattern of progress of the subjects. This time interval was justified by similarities to the standard practice observed by orthopaedic surgeons in the management of uncomplicated bony lesions of the cervical spine and associated soft tissue lesions (Batalin, 1989). The results of the current study demonstrated that the subjects in the immobilised group recovered from their pain related symptoms and returned to their normal duties sooner than those in the other two treatment

groups. In addition, the results showed that those subjects who received the immobilisation regimen did not demonstrate adverse effects in either range of motion or strength of the neck muscles. Thus, the immobilisation regimen was shown to be the preferred option compared with the remainder of the treatment methodologies adopted in the current study.

Although the primary aim of the current study was to compare the efficacy of three different treatment regimens, the prognostic significance of several factors associated with "whiplash" injury was also established. It should be noted that the Quebec Task Force emphasised the importance of establishing prognostic factors associated with "whiplash" injury and nominated this issue as the first priority in a list of, "High Priority Questions-Research To Be Initiated in the Near Term" (Spitzer et al., 1995, page 41.S). Another major emphasis of the current study was to concentrate on the symptoms which are of major concern to "whiplash" subjects and to those clinicians treating them. Most of these symptoms involved pain, in particular relating to the head and neck. A paucity of such information is considered to be one of the most notable causes of difficulties encountered in the management of "whiplash" injuries (Frankel, 1953; Janes and Hooshmand, 1975; Pearce, 1989; Watkinson et al., 1991). As an adjunct to the main study, it was considered essential to update the morphology of the deep pre- and post-vertebral muscles of the neck region using embalmed cadavers and fresh post-mortem specimens as the literature was deficient in this regard (Gray,1858; Sharpey et.al,1878; Fountain et.al,1966; Romanes,1972; Vitti et.al,1973; Warwick and Williams,1973; Johnson et.al,1975; Last,1978; Williams et.al,1989). Similarly, a longitudinal study of 45 subjects was also performed using Magnetic Resonance Imaging technology as an additional extension of the main study. The longitudinal nature of the M.R.I study provided for the first time an account of details associated with progressive pathological changes that occurred in some

disc lesions, at defined points in time following an MVA. The observations made from the adjunct studies in the current research help to clarify the overall position and thus assist in developing a better understanding of the pathoanatomy associated with deep muscles of the neck region and pathological changes that occur following a traumatic disc lesion as evidenced within 2 weeks, after 3 months and 12 months post- injury. On the basis of the observations made in the current study, a classification of the "whiplash" injury has been proposed for the consideration of clinicians. Similarly, the questionnaire that was used for data collection in the current study can be readily modified for use in the clinical setting for documentation of patients condition, planning treatment strategies and for the evaluation of treatment outcome in "whiplash" type of injuries.

This discussion considers the information obtained under the following headings:

1. pain;
2. range of motion;
3. muscle strength;
4. return to work;
5. prognostic value of the factors identified;
6. observations of secondary interest; and
7. clinical implications.

6.2 An explanation for the differences observed between the treatment groups at the initial examination

The analyses of the data collected at the initial examination of the current study showed that there were statistically significant differences between the treatment groups. An explanation is offered for this situation in the following section so that the validity of the results of this study is understood

It was evident from the results of the ANOVA of the initial examination (page 188) that statistically significant differences existed in the level of pain between treatment groups, indicating that the randomisation procedure had not been effective as was anticipated. A subsequent *post hoc* multiple comparison test identified that these differences were due to a higher pain level for the immobilised group, when compared to the active exercise and the control groups. It is not unusual to come across such situations in clinical trials of a similar nature and it should be noted, that in the current study, the randomisation procedure was not designed to formulate treatment groups matched on the basis of pain. Although it would have been desirable, it is impractical to adopt such measures owing to the underlying medico-legal implications associated with "whiplash" injury. In addition, there are several confounding variables which are likely to influence the severity of the symptoms (Deans et al., 1986; Hildingsson and Toolanen, 1990; Ryan et al., 1993; Radanov et al., 1995). Thus, it could be argued that the effect of the confounding variables cannot be controlled by the randomisation procedure. However, in the current study, appropriate statistical methods were applied to correct the situation so that the observed results were valid.

6.3 Perceived pain

In this section the rate of progress with respect to pain as evidenced between the treatment groups is considered. At the initial examination, the intensity of pain for the immobilised group was significantly higher when compared to the active exercise group. Although there was also a difference between the immobilised and the control groups, it was not large enough to be statistically significant. Thus, the immobilised group had a higher level of pain to start with and yet subsequently demonstrated a greater reduction in pain than the other two groups. This was consistent for

each of the examination time periods and was statistically significant. Thus, there was evidence indicating that the immobilisation regimen offered the most effective form of treatment when compared to the other two methodologies. In addition, it was found in the immobilised group that the pain level declined at a higher rate during the study period and this requires a detailed consideration.

The strength of the treatment effect attributable to the respective treatment regimens adopted in the current study is highlighted by the observed reduction in pain level during the follow-up assessments. A comparison of the geometric mean of the pain for the respective treatment groups between the initial examination and the follow-up examination at week 4 showed that there was a considerable reduction in the level of pain among the subjects in the immobilised group (Table 5.5.1, 5.5.4; page 188, 189). In stark contrast, a comparison of the geometric means of pain for corresponding observation periods showed that the rate of progress among those subjects from the active exercise group and the control group was relatively slower than the immobilised group (Table 5.5.4; page 189). In addition, comparison of the upper and the lower limits of the confidence interval for each of the treatment groups similarly indicated that those subjects in the immobilised group had a marked reduction in the range of severity of their pain, while the subjects in the other two groups had a relatively larger upper and lower limits.

In the current study, the differences in the rate of recovery between the treatment groups were so large that it was statistically possible to predict the "odds of getting better" for the immobilised group, as early as six weeks after the injury. Although there were differences between the treatment groups in the rate of progress, as observed at six weeks and twelve weeks

post-injury, a large number of subjects in each of the groups were getting better by twelve weeks after the MVA. However, it was apparent from the respective confidence interval that irrespective of the treatment regimens, a relatively small proportion of subjects were experiencing severe pain from their "whiplash" injury.

Observations made three months after the "whiplash" injury indicated that the rate of progress was consistent during the rest of the study period for the immobilisation group, while in contrast the subjects in the active exercise group demonstrated fluctuations in their progress between three and six months following the MVA. A similar fluctuation was evidenced in the case of the control group, six months after the MVA. These fluctuations may have been due to some of the subjects in the active exercise and the control groups having either a relapse or aggravation of their symptoms at that time and these characteristics of recovery also bear witness to the efficacy of the respective treatment methodologies which were used in the current study.

The statistical methods used in the current study demonstrated both qualitative and quantitative changes in pain-related symptom between the three treatment groups and also showed that irrespective of the treatment methodologies a number of subjects still experienced severe pain for up to twelve months after the MVA. These distinct differences in the rate of recovery between the groups and the development of chronic symptoms may be attributed to the severity of the injury sustained by the soft tissue and the neural structures of the cervical region following the MVA. In a situation such as this, the evolution of the pathological changes associated with these injured structures is not homogenous and the implications of this are considered in some detail in the following section.

6.3.1 An explanation for the presence of chronic pain

The general pattern of recovery indicated that the majority of subjects who recovered from their pain during the early period, might have had minor trauma to the soft tissues, which would usually heal under ideal conditions within six weeks of the injury. It should be noted that the ideal conditions for soft tissue healing include a rich blood supply and avoidance of excessive movement in order to eliminate the effect of adverse tensile forces acting on the healing tissues (Walter and Israel, 1987). It is therefore not surprising that these were the conditions experienced by the subjects in the immobilised group during the first four weeks after their MVA and thus their improvement was considerable. In contrast, the tendinous structures and ligaments which do not have as rich vascularity as the skeletal muscles are likely to take relatively longer to recover and achieve complete resolution (Walter and Israel, 1987). Although the time scale for resolution of injury for the intervertebral disc (which is an avascular structure), has not been established, the result of MRI performed at three and twelve months after the MVA demonstrated that progressive degenerative changes were occurring within those discs that had been damaged in the MVA. Furthermore, there are instances in which incomplete healing of intervertebral disc following "whiplash" injury have been observed for up to five years post-injury (MacNab, 1964; Bedbrook, 1989). Thus, it is conceivable that some of those subjects who took longer than 12 weeks to recover may have had moderate to severe injury to their discs and in addition, the treatment methodology in itself might have also contributed to the prolongation of their pain . The nascent nerve fibres that are present in granulation tissues have been also considered to be responsible for some of the pain experienced by patients following soft tissue injury (Cyriax, 1978; La Rocca, 1992). Under these circumstances the newly formed neural tissue is excited by the slightest mechanical or chemical stimuli and

continuously send afferent nociceptive afferent impulses into the central nervous system (La Rocca, 1992).

Chronic pain may also be due to the changes that follow an injury either to the peripheral nerve or the central nervous system itself (Wall,1991; Rosomoff et al., 1992; Wetzel, 1992). The peripheral nerve injury can occur in a variety of possibilities ranging from compression of the nerve root to micro-trauma of the smaller nerve fibres which abundantly innervate the entire soft tissue structures that constitute a motion segment and the muscles in the pre and post-vertebral regions. As a result of the damage to the neural tissues, irreversible physiological changes occur, causing aberrations in the modulation of afferent nociceptive input (Wall and Devor, 1981; Wall,1991; Rosomoff et al., 1992; Wetzel, 1992). Thus, it has been considered that a barrage of chronic afferent nociceptive input to the CNS may at the same time be accompanied by a reduction in the normal pain inhibitory mechanism, culminating in the development of central hypersensitivity within the spinal cord itself (Wall, 1991). Recent studies have shown that in chronic pain problems, reverberating circuits are set up within the central nervous system resulting in the subject continuing to experience pain as coming from the initially affected area, even after the symptoms from that area have subsided (Konttenan et al., 1994). This "cascade of physiological changes" which have involved the central nervous system has been considered to be responsible for such "phantom pain" and for many referred pain syndromes that remain long after the initial injury has resolved (La Rocca, 1992). In a situation like this, treating the original site of the injury may not help, as even the slightest mechanical or bio-chemical stimuli may produce a disproportionate sensation of pain (Wetzel, 1992). Although it is logical to consider that these complex physiological changes may be responsible for the development chronic

pain in some of the subjects in the current study, it is also possible that a few of the subjects may have other problems which are yet to be determined.

6.4 Common symptoms

Another major emphasis of this study was to concentrate on the symptoms which are of major concern to "whiplash" subjects and to those clinicians treating them. Most of these symptoms concern pain particularly of the head and neck. A paucity of information regarding the evolution of pathology and the rate of progress is considered to be one of the most notable causes of the difficulties encountered in the management of "whiplash" injuries (Frankel, 1953; Janes and Hooshmand, 1975; Pearce, 1989; Watkinson et al., 1991).

6.5 Headache

Headache is one of the commonest and most persistent symptoms among many patients affected by "whiplash" type of injuries (Hohl, 1974; Hohl, 1975; Norris and Watt, 1983; Balla and Karnaghan, 1987; Merskey, 1984; Maimaris et al., 1988; Hildingsson and Toolanen, 1990; Radanov et al., 1993b; Radanov et al., 1995). The presence of headache during the acute stage of "whiplash" injury has been shown to have prognostic value (Maimaris et al., 1988). In the current study headache has been grouped into two categories, viz frontal headache (forehead) and occipital headache and each considered individually. The overall incidence of frontal and occipital headache associated with "whiplash" (as reported in the literature), is provided in the relevant sections as a matter of interest only. It should be noted that a comparison of the observations made in the current study and the information available in the literature regarding frontal and occipital headache may not be appropriate for the following reasons. There were

wide variations among the studies with reference to the time scale at which the initial examination was conducted following MVA. As an example, in the current study, the initial examination was performed within 60 hours following the injury, whereas Norris and Watt (1983) allowed themselves a period of up to seven days for documenting the initial examination of their subjects following MVA. In the case of Hildingsson and Toolanen (1990) the time of initial examination was somewhat similar to that of the current study. However, these authors reported the headache symptom as a single category, unlike the situation in the current study in which distinctions were made between the incidence of frontal headache and occipital headache. In spite of the variations in the data, the current study supported the conclusions of those studies which nominated headache as a major problem among "whiplash" patients.

6.5.1 Frontal headache

The frequency of frontal headache as recorded at each of the designated examination periods and the pattern of recovery for the total sample are considered in this section and thereafter the differences observed between the treatment groups discussed. The proportion of changes evidenced between the follow-up examinations was an indication of whether or not there had been progress in the frontal headache symptom experienced by the subjects. This information may be useful in making predictions for those individuals affected with frontal headache resulting from "whiplash" type of injury.

The incidence of right and left sided frontal headache was recorded separately for each of the observation periods, as there were instances where subjects only reported unilateral headache. However, since the differences between right and left sided frontal headache were relatively small, the average figure is taken when discussing the results. The initial

examination showed that 60% of all subjects in the current study reported frontal headache (Table 5.4.3). Similarly, Hohl (1974) reported that two thirds of his "whiplash" patients complained of headache, supported by a more recent prospective study of "whiplash" reporting that 57% of the subjects complained of the frontal headache symptom (Radanov, et al., 1993).

A month after the injury, the follow-up examination demonstrated that 50% of subjects assessed reported that frontal headache continued to be a problem and when compared to the initial examination, it was evident that not many subjects recovered during the first month following their injury. The subsequent follow-up examinations at week 6 and week 12 indicated that this incidence had declined and that 40.8% and 38% (respectively) of subjects were still affected with frontal headache. A trend in the pattern of the recovery was evident at three months after the injury, indicating that the rate of progress had not markedly increased during the second and third month following injury (Figure A.III.4). In addition, subsequent observations at week 24 and week 52 demonstrated that the pattern of recovery noted by the end of the third month may be of some significance in predicting the eventual progress, since 36% of all subjects examined continued to complain of frontal headache. Thus, these the current study showed that there was little change in the pattern of progress between the third and twelfth month following MVA. It was evident that most subjects who remained symptomatic at the end of third month following MVA were likely to develop a long-term problem (i.e one that existed at 12 months) associated with frontal headache. In addition, a comparison between the initial examination and the final examination showed that the incidence of frontal headache during the study period declined from 60% to 36%,

indicating that a large number of the subjects who remained in the study were affected with a chronic problem associated with this symptom.

On the basis of the reported observations, certain general conclusions may be drawn with reference to the "whiplash" patients. A large proportion of the "whiplash" patients are likely to be affected with frontal headache following MVA and more than half of those affected patients may not recover completely from this symptom within one year post-injury. Those patients who are still affected with frontal headache three months after their injury are more likely to develop a chronic problem associated with this symptom.

6.5.2 Differences observed between the treatment groups affected with frontal headache

In the following section the difference between the treatment groups with reference to frontal headache at various times during the study period is considered. A graphic comparison of the frequencies of symptoms is presented as Appendix III. 5 to illustrate the natural progression and associated characteristics with reference to the treatment regimens.

The characteristics of the progression of subjects affected with frontal headache among the three treatment groups demonstrated a gradual reduction in the incidence of frontal headache up to the follow-up examination at week 12 (FigureA.III.5). However, the immobilised group demonstrated a considerable decline in the percentage of subjects affected with frontal headache during the first four weeks and this trend continued until week 12. Thereafter, distinct differences started to emerge between the groups. The general trend up to week 12 reflected the reduction in the percentage of all subjects with frontal headache symptoms in all three groups. The proportion of change in the active exercise group was

relatively smaller when compared to the immobilised group, although greater than with the control group.

It was interesting to note that both the immobilised and the active exercise groups showed an increase in the percentage of subjects with frontal headache during the period between third month and sixth month after sustaining their "whiplash" injury. This trend indicated that some of the subjects experienced a relapse in their frontal headache symptom during this period. A continuation of this trend into the second half of the study period was observed only in the case of active exercise group. In contrast, the control group continued to progress without experiencing a relapse in frontal headache symptom, but the progress was characterised by a relatively smaller amount of change between the observation periods. The overall changes were more noticeable in the case of the immobilised group reflecting the apparent effectiveness of this treatment regimen, in so far as frontal headache is concerned. However, a comparison of the changes between the initial and final follow-up examination at week 52, demonstrated that a large number of the subjects in all treatment groups continued to report frontal headache as a continuing problem. The observed differences in the rate of decline of the symptom, between the treatment groups, were an indication of the efficacy of the respective treatment regimens. An explanation for the observed differences is presented in the following section.

Several causes have been identified as being responsible for the onset of headache following "whiplash" type of injury (Seletz, 1958; Torres and Shapiro, 1961; Goff et. al., 1964; Janes and Hooshmand, 1965; Ommaya et al., 1968; Edmead, 1978; Bogduk, 1982; Keith, 1986; Vital et al., 1989; Bedbrook, 1989; Radanov et al.,1993b; Stokes, 1993; Radanov et al.,1995). They include cerebral concussion, injury to the neural tissues and the

motion segments of the cervical spine. The spinal instability due to disruption of the structures which control the normal kinematics of the motion segments in the cervical spine has also been ascribed as one of the causes of headache symptoms among "whiplash" patients (Janes and Hooshmand, 1965; Bedbrook, 1989). Accordingly headache among "whiplash" patients has been described as manifestation of either neural irritation or referred pain from any one of the causes mentioned above (Janes and Hooshmand, 1965; Bogduk, 1982; Mark, 1990; Stokes, 1993). In addition, pre-traumatic headache has also been shown to have a significant association with headache symptom resulting from "whiplash" type of injuries (Radanov et al.,1993b).

A specific time scale required for the resolution of headache symptoms attributable to traumatised neural and vascular structures is not available. However, in the event of spinal instability and intervertebral disc lesions, "whiplash" patients are reported to experience the headache symptom up to five years after the event (Janes and Hooshmand, 1965; Bedbrook, 1989). Clinical observations have indicated that in general, pain related symptoms resulting from sprains and similar minor trauma to the muscles and ligaments usually resolve within 12 weeks (Janes and Hooshmand, 1965). On this basis, it is reasonable to propose that the manifestation of either referred pain or local pain attributable to minor trauma to the muscles and ligaments of the cervical region is also likely to resolve within a similar time scale (Janes and Hooshmand, 1965).

In the light of the above information, it is logical to propose that the observed reduction in the incidence of headache related symptoms during the early part of the study can be largely attributed to the resolution of minor injury to the soft tissues of the neck. It is also likely that those subjects who

developed chronic symptoms associated with headache, may have had a more severe form of injury to any one of the pain sensitive structures previously described, or that there has been central nervous system excitation which manifests as "phantom pain" (Konttinen et al., 1994). This phenomenon has been described in section 6.3.1 in some detail. The observed differences evidenced between the groups in the rate of progress indicated the contribution made by the respective treatment regimen in facilitating the resolution of injury to the muscles and ligaments. The merits of the individual treatment regimens are considered in detail later in this chapter.

It was interesting to note that there was an abrupt change in the pattern of recovery at 12 weeks after the injury, indicating that some of the subjects in both the immobilised and the active exercise groups had a sudden relapse of headache symptoms (Figure A.III.5). It was evident that the median duration for returning to normal duties at a level equivalent to the pre-accident level was 8 weeks for the immobilised group and 10 weeks in the case of active exercise group (page 233). Some of those subjects who have returned to their normal duties might conceivably have aggravated and subsequently reported their relapse of headache symptom at the follow-up examination at week 12. A similarity between the current study and that of Balla and Karnaghan (1987) was evident in this regard. In their retrospective study, Balla and Karnaghan (1987) observed that 55% of their subjects indicated that activities and neck movements were the major causes of aggravation of their headache even after 6 months post-injury. It is also conceivable to suggest that those subjects who have had a relapse might have an under lying pathology other than just minor trauma to the soft tissues involved. The observations made during the current study lends support to this explanation. A similar relapse of the headache symptom was reported by those subjects who were diagnosed as having disc lesions

and disruption of the outer anular fibres anterior longitudinal ligament complex.

6.5.3 Occipital headache

The natural evolution of occipital headache evidenced in the current presented graphically in Appendix III (Figure A.III.6) showed that 30% of all subjects reported occipital headache during the initial examination, as one of their symptoms which contrasted to the study by Balla and Karnaghan (1987) who reported that 46% of their subjects experienced occipital headache. It should be noted that the initial examination in the current study was performed within 60 hours following the MVA, while Balla and Karnaghan (1987) allowed their subjects up to 4 weeks before entering their study and the observed variation between the studies may also be due to this difference in the time scale. at which the initial examinations were performed following MVA. For this reason, some caution should be exercised in making a direct comparison between the observations made by these studies.

The subsequent follow-up examinations in the current study indicated that the rate of recovery from the occipital headache symptom was relatively lower when compared to the rate of recovery from frontal headache. Not many subjects recovered from their occipital headache during the course of the study. A comparison between the initial and the final assessments showed that the incidence of occipital headache during the study period declined from 30% to 26% and this indicated that a considerable number of the subjects who remained in the study were affected with chronic problem associated with this symptom.

6.5.4 Differences observed between the treatment groups affected with occipital headache

A comparison between the treatment groups demonstrated that the immobilisation regimen was preferable over the other treatment methodologies for occipital headache was concerned (Tables A.III.4.8-A.III.4.10; Figure A.III.7). Since during the first four weeks following the MVA, the percentage of subjects in both the active exercise and the control groups who were affected by occipital headache increased slightly, in stark contrast to the reduction in the occipital headache symptom within the immobilised group. This contrast might have been due to the continuing aggravation of the traumatised joints and soft tissues of the cervical region in these groups who were allowed mobility.

Those subjects who were part of the immobilisation regimen showed a continued decline in occipital headache throughout the first three months after injury. Although this trend changed abruptly during the follow-up examinations at week 12 and week 24, further progress was reported during the second half of the study period. The sudden change in the pattern of recover at week 12, indicated that some of the subjects in the immobilised group experienced a relapse in their occipital headache symptom. This pattern was similar to that seen for frontal headache, and thus cannot be dismissed as an aberration in the pattern of recovery. The possible explanation for the relapse may be associated with the subjects returning to their normal work related duties, (as discussed previously).

A rapid recovery was observed among the active exercise group during the period between the 4th and 6th week following injury indicating that a significant proportion of subjects showed an early decline in occipital headache. However this was followed by a pattern of aggravation of the

symptom during the period between weeks 6 and 12 after injury. Similar to the situation observed with the immobilised group, the second half of the study period was characterised by the progressive reduction in symptoms. However, the percentage of recovery in the active exercise group lagged behind that of the immobilised group.

The relapse evident in the active exercise group may be attributed to two factors. The subjects in this group would have commenced isometric strengthening exercise soon after the follow-up examination at week 4, in order to strengthen their posterior neck muscles. It was generally reported by the subjects in this group that the isometric exercise induced pain in the sub occipital region which lasted in some cases for several hours following the exercise period. A similar adverse reaction was also reported frequently following muscle strength testing procedure using the strain gauge. Thus, the traumatised soft tissues in the posterior region of neck, which were still in their early stage of recovery, may have been aggravated by the isometric exercise, which may have induced a relapse of occipital headache. The second factor may have been the resumption of normal duties by some of the subjects in the active exercise group. It should be noted that fifty percent of the subjects in the active exercise group returned to their normal duties around 10 weeks after the MVA (median time as indicated by the respective survival analysis). It is not uncommon for "whiplash" patients to experience a relapse in their symptoms after resuming their normal work duties. It is conceivable that one of the factors mentioned above, or their combined influence, may have been responsible for the relapse experienced during the period between the 6th and 12th week after sustaining their "whiplash" injury.

The rate of progress in the control group was considerably lower than the other two groups. The control group subjects began to show recovery of their occipital headache from the 4th week following MVA and this trend continued up to 6 months from the time of initial assessment. Thereafter, unlike the other two groups, there was an abrupt change in the course of recovery indicating a worsening of the symptom so that the number of the subjects affected with occipital headache increased significantly (Tables A.III.4.8 to A.III.4.10, Figure A.III.7). The pattern of aggravation evidenced in the control group six months after the injury may be associated with the subjects resuming their normal working duties in full around this time. It should be noted that the median time for retuning to normal duties for the control group was 20 weeks.

6.5.5 Summary

It was apparent in the current study that headache was one of the most important of the chronic symptoms among the "whiplash" patients and that the rate of recovery of those subjects affected with occipital headache was considerably slower when compared to those subjects with the frontal type of headache. It was also evident that a large proportion of "whiplash" patients affected with occipital headache are likely to develop a chronic problem associated with this symptom. The results indicated that the subjects receiving treatment in the form of the immobilisation regimen, demonstrated a faster rate of recovery from frontal and occipital headache symptoms. In addition, it can also be concluded that those "whiplash" patients who were affected with occipital headache and treated in accordance with the immobilisation regimen were less likely to develop chronic occipital headache.

6.6 Neck pain

Pain in the posterior aspect of the neck was reported by 83% of all subjects at the time of the initial examination and remained the single most commonly reported symptom.

There is a general agreement in the literature that neck pain is the most debilitating of all the physical symptoms among "whiplash" patients (Gay and Abott,1953; Frankel,1959; James,1965; Breck and Norman, 1971; Hohl,1974; Hohl, 1975; Hohl,1983; Toglia,1976; Maimaris et al., 1988; Porter,1989; Hildingsson and Toolanen, 1990). In spite of the clinical importance attached to neck pain, there is a paucity of information related to the natural history of its progress that would reflect the characteristics of resolution over a given time period. In general, injury to the muscles, ligaments, intervertebral disc, and the joint structures in the neck region are shown to be the primary sources of neck pain among "whiplash" patients (Janes and Hooshmand 1965; MacNab, 1971 and 1973; Barnsley et al., 1995). However, there is a lack information regarding which of the above mentioned structures is most often responsible for the neck pain experienced by many "whiplash" patients.

The association between neck pain reported by the "whiplash" patients and the superficial muscles of the neck region can be established during the course of physical examination, whereas invasive diagnostic procedures are required for establishing a similar association in the case of the other structures which might be responsible for the pain. It was not within the scope of the current study to undertake such invasive diagnostic procedures and only the information documented on the basis of physical examination of the subjects affected with neck pain is discussed. In this regard, the following superficial muscles were identified as being

associated with neck pain among the study subjects and the pattern of their recovery is considered in this section:

1. semispinalis and splenius capitis muscles;
2. trapezius muscle; and
3. sternomastoid muscle.

6.6.1 Semispinalis capitis and splenius capitis muscles

The initial examination showed that 83% of the total sample complained of pain in the posterior aspect of the neck on either side of the spinous processes (Appendix-4, body chart areas 10 & 11). During data collection appropriate care and attention was given to ensure that the painful area of the body was clearly identified and documented on the body chart. Thus, when the posterior aspect of the neck was nominated by the subjects as being one of the symptomatic areas the author was able, by palpation, to locate the tenderness within the prominent superficial muscles in the para vertebral region. Nuchal line tenderness was predominantly indicative of the involvement of the proximal attachment of the semispinalis capitis and to a lesser extent to the splenius capitis muscles. Similarly, the interscapular pain was found to be related to the distal tendinous attachments of the semispinalis capitis muscles. The matters related to the nuchal line tenderness and the interscapular pain are discussed elsewhere in this chapter.

In general, the follow-up examinations of all those subjects affected with neck pain indicated that the rate of recovery was slow and only a small proportion of those subjects who continued to be affected with neck pain fully recovered 6 months after injury. A comparison of the frequencies associated with neck pain indicated that the rate of recovery was noticeably higher between the 4th and 6th week after injury followed by the

period between the 12th and 24th week after injury. The final assessment performed at week 52 following injury, showed that 45% of the subjects still continued to have neck pain which indicated that a large proportion of "whiplash" patients are likely to develop chronic neck pain following MVA.

6.6.2 Differences observed between groups

A comparison between the treatment groups as observed during the study period is graphically presented in figure A.III.8 and A.III.9, in order to illustrate the pattern of recovery. The observed variations in the pattern of recovery reflected the efficacy of the respective treatment regimen. The rate of recovery as observed at defined time periods during the current study and the proportion of the subjects who continued to report neck pain at the end of the study period, demonstrated that the immobilisation regimen was considerably better than the other regimens in treating neck pain. It is likely that the early immobilisation of the injured soft tissues might have provided an ideal situation for better healing and thus averted pathophysiological changes that lead to sensitisation of nerve fibers and development of chronic pain (Walter and Israel, 1987; Rosomoff et al., 1992; Wetzel, 1992; Konttenan et al., 1994). It was also evident that the rate of progress between the right and left sides of the neck was varied. It is possible that these differences between the sides may be either due to the mechanism of accident, or to the differences in the dominance of the upper extremities. The active exercise regimen appeared to have aggravated the neck pain during the first four weeks after injury and this factor is a likely explanation for an increase in the incidence of neck pain on the left side evidenced in this group. The effectiveness of the immobilisation regimen was apparent even at the early stage of the study. A significant proportion of the subjects in the immobilisation group recovered from their neck pain within 6 weeks after injury. Although there was slight reduction in the rate of progress in

the immobilised group between the 6th and 12th week, the remainder of the study period was characterised by a considerable reduction in the proportion of the subjects affected with neck pain. In stark contrast, there were significant fluctuations in the rate of recovery among those subjects in the active exercise and the control groups who were so affected. This trend indicated that some of those subjects who recovered from neck pain symptoms experienced a relapse, possibly due to an increase in the level of activities associated with their normal duties. In addition to this, the relapse might also have reflected the attributes of the respective treatment regimen.

6.6.3 Trapezius muscle (upper and middle fibres)

In the current study, the incidence of pain in the lateral aspect of the neck also ranked high among the list of all pain related symptoms. The upper and middle fibres of the left and right trapezius muscles were identified by palpation to be the muscles in this region as one of the sources responsible for this symptom (Appendix-4 Body chart- areas 31,32, 21 & 22). There was little information in the literature pertaining to the involvement of trapezius muscle as a source of neck pain, apart from one recent study describing the association of pain with trapezius muscle (Larsson et al., 1994). In this work, Larsson et al. (1994) were concerned as to the cause of the pain in the trapezius muscle, whereas the observations made in the current study indicated that in addition to being a possible source of neck pain, the upper and middle trapezius fibres appeared to be also associated with restriction of the lateral flexion and rotation movements of the cervical spine. Therefore, a detailed account of the incidence of pain related symptom associated with trapezius muscle and the characteristics of the recovery pattern as evidenced in each of the treatment groups are described in some detail.

6.6.4 Trapezius middle fibres

The initial examination indicated that 67% of the total study population reported pain related symptoms associated with the middle fibres of the trapezius muscle. Similarly, at the end of the study period at 52 weeks after the injury, 31% of all the subjects examined continued to report this symptom. The assessment performed at each of the time periods demonstrated that the incidence of pain attributable to the middle fibres of the trapezius muscle, was slightly different between the left and right side of those subjects affected with this symptom. A steady reduction in the incidence of this symptom was noticed during the first three months after the injury and thereafter the rate of resolution slowed considerably. The observed trend indicated that evidence of chronicity associated with pain arising from middle fibres of trapezius muscle may be identified at a relatively early stage of the injury (i.e 12 weeks following MVA).

6.6.5 Differences observed between treatment groups

A comparison of the treatment groups showed distinct differences in the pattern of recovery in relation to the middle fibres of the trapezius muscle (Figure A.III.10 and A.III.11). The subjects in the immobilised group recovered much quicker than the remainder of the groups and at the end of the study period, only a relatively small proportion of the subjects remained symptomatic. In contrast, the active exercise group and the control group were second and third respectively in terms of the rate of recovery. As a consequence, by the end of the current study, a larger proportion of the subjects examined both in the active exercise and the control groups, continued to be affected with pain related to the middle fibres of the trapezius muscle.

The effectiveness of the immobilisation regimen was most obvious as early as 4 weeks after the injury. Three quarters of those subjects in the immobilised group affected with this symptom recovered within 12 weeks post-injury, whereas a relatively small proportion of those subjects from the other groups with similar symptom were fully recovered within 12 weeks after injury. Some of the subjects in the active exercise group showed a relapse in their symptom during the period between week 6 and week 12 and a similar pattern was also observed in the control group. after week 12 post-injury. The observations indicated that in all three treatment groups, a relatively small percentage of the subjects affected with this symptom were fully recovered six months after their injury. Therefore, it is more likely that those subjects presenting with pain related symptoms associated with trapezius middle fibres at this point in time, may become chronic. The other interesting feature indicated that slight differences were evident in the rate of recovery between the left and right side of the neck. The following reduction was obtained by comparing the initial and final examination results and are an indication of the effectiveness of the treatment regimens:

1. immobilised group 90%;
2. active exercise group 50%; and
3. control group 33%.

6.6.6 The upper trapezius muscle fibres

In the initial examination, 51% of subjects in the study reported pain-related symptoms associated with the upper fibres of the trapezius muscle. At the end of the study period 30% and 24% of subjects examined continued to report pain in the right and left upper fibres of the trapezius muscle. The amount of pain experienced in the upper fibres of trapezius was less in comparison to the pain experienced in the middle fibres of the trapezius muscle. The rate of recovery during the first 12 weeks post-injury was

slightly slower on the left side of the neck. However, subsequent follow-up examinations demonstrated an abrupt change in the recovery pattern of the right side, characterised by an increase in the percentage of the subjects so affected. In general the characteristics of progress of this symptom was similar to the pattern observed in the case of pain associated with the middle fibres of trapezius. The differences evidenced between the groups are presented in the following section.

6.6.7 Differences between groups

Comparison of the treatment groups showed considerable differences in the pattern of recovery with respect to pain experienced in the upper fibres of the trapezius muscle (Figure A.III.12 and A.III.13). It was evident that the immobilisation regimen was again the most effective of the three treatment methodologies in relation to these pain symptoms. Approximately two thirds of subjects in the immobilised group so affected recovered fully within 12 weeks after the injury and only a relatively small percentage of the subjects examined at the end of the study period continued to be troubled by this symptom (Tables A.III.4.8 to A.III.4.10). This was in stark contrast to the pattern of recovery seen in the other groups. In general, there was a decline in the rate of recovery six months after the injury among all the treatment groups. This pattern was similar to those observations made in connection with the muscles discussed in the preceding sections.

It was interesting to note that during the first four weeks following the MVA, the active exercise group was the only group which showed an increase in the percentage of subjects complaining of pain from upper fibres of the trapezius muscle (Figure A.III.12, A.III.13). This trend may have been due to the effect of the active exercises undertaken by the subjects in this group as a part of the treatment regimen. However, it took until 6 weeks after their

initial injury for those subjects in the active exercise group to notice a reduction in pain in their upper fibres of trapezius. An increase in the incidence of those affected with pain in the upper fibres of the trapezius muscle was also noticed in the control group between week 4 and 6 post-injury and this pattern of aggravation is likely to be due to the type of treatment received. It should be noted that the subjects in the control group received a variety of treatments which included manipulation of the cervical spine, cervical traction, chiropractic and spinal mobilisation. The fluctuations in the rate of recovery observed in both the active exercise group after 12 weeks post injury and in the control group after 6 months post injury, are likely to be due to an increase in the level of activities performed by those subjects. This aspect has been discussed in some detail in the preceding sections.

The other interesting feature related to the pattern of recovery was the marked differences which were evident among those subjects in the active exercise group, from week 12 post injury, in the rate of progress regarding pain between the right and left upper and middle fibres of the trapezius muscle. In general, the pattern of progress after six months post-injury among all three treatment groups indicated that those subjects who continued to experience pain from the upper fibres of trapezius, were likely to develop a chronic problem. The following reductions were obtained by comparing the initial and final observations and are an indication of the effectiveness of the three treatment regimens:

1. immobilised group 91%;
2. active exercise group 19.3%; and
3. control group 30%.

6.6.8 Summary

The initial assessment showed that the incidence of pain associated with the middle fibres of the trapezius muscle was more prevalent when compared to pain from upper fibres of the trapezius muscle. However at the end of the study period there was no significant difference between these two regions. It was evident that of the three treatment methodologies, the immobilisation regimen was the most effective for treating pain symptoms arising from these regions. Although the active exercise regimen appeared to be the least effective in reducing pain associated with the upper fibres of the trapezius muscle, it was only moderately effective in treating pain arising from the middle fibres of trapezius. In contrast, the control group consistently demonstrated a poor rate of recovery. The observations made in the current study show that those subjects who continued to experience pain from the upper and middle fibres of the trapezius muscle, until six months after the MVA, were likely to develop a chronic problem. The observation also highlighted the differences in the rate of recovery from the pain symptom between the right and left side of the upper and middle fibres of the trapezius muscle. It is also likely that "whiplash" patients may experience a relapse in pain from the upper and middle fibres of the trapezius muscle after resuming their normal work duties.

6.7 Interscapular pain

A number of authors have observed that pain in the interscapular region is also one of the commonest early symptoms reported by the "whiplash" patients (Hohl,1975; Hohl and Hopp,1978; Hohl,1983; Norris and Watt, 1983; Maimaris et al.,1988). Although interscapular pain is considered to be of prognostic value, the correlation between the interscapular pain and its source has not been clearly established (Hohl and Hopp,1978; Hohl,1983; Norris and Watt, 1983; Maimaris et al.,1988). In spite of the

relative interest attached to this particular problem, information regarding the frequency, the natural course of progress and associated characteristics are not available.

In the current study, the initial assessment showed that 30% of the total population were initially affected with pain in the interscapular region while at the end of the study period only 3% of the subjects examined reported that interscapular pain was a continuing problem. The pattern of progress showed that a considerable percentage of those affected with this symptom recovered within 6 weeks of the MVA (Tables A.III.4.8 to A.III.4.10). A slight increase in the incidence of this symptom as evidenced between the examinations at week 6 and week 12 indicated that some of the patients either had a relapse or developed late onset of interscapular pain. Thereafter there was a slow reduction in the number of subjects affected with interscapular pain.

6.7.1 Differences between the treatment groups

There were distinct differences between the three treatment groups (Figure A.III.14). Although subjects in the immobilised group affected with interscapular pain showed marked progress within the first 4 weeks following the MVA, there was an increase in the number of subjects with this symptom during the period between the 4th and 12th week. This reversal in improvement between the 4th and 6th follow-up examinations may have been related to the commencement of isometric exercises soon after the follow-up examination at week 4, performed in order to strengthen the post vertebral neck muscles. In addition, an increase in the level of activities undertaken by those subjects at that time may also account for the sudden increase in the incidence of interscapular pain between week 6 and week 12 after injury. A similar relapse was also evidenced both in the active exercise and the control groups between week 6 and week 12 after

the MVA. At the end of six months after MVA, the recovery was complete among both the immobilised and active exercise groups, whereas a small percentage of subjects in the control group remained symptomatic 52 weeks after injury. These observations demonstrated that the "whiplash" patients affected with interscapular pain are very likely to fully recover from such pain within six months of injury. It was also evident that the immobilisation regimen and the active exercise regimen were similarly effective in achieving full recovery from interscapular pain.

6.8 Pain in the region of nuchal line

There is little information in the literature regarding the incidence of pain in the region of nuchal line. It is generally considered that there is an association between this pain symptom and the presence of occipital headache among "whiplash" patients (Hohl, 1974; du Toit, 1974). In the current study, 17% of all the subjects examined during the initial assessment reported pain in the vicinity of the nuchal line and during the course of the physical examination, a careful palpation of the tendinous structures attached in the region of the nuchal line, elicited intense occipital headache. A large number of the subjects affected with pain in the area of nuchal line fully recovered within 4 weeks after injury. Thereafter the progress was characterised by a slow reduction in the number of subjects affected with this symptom which continued up to week 24 after injury. At the end of the study period 3% of the subjects examined were complaining of a continuing problem associated with pain along the nuchal line.

6.8.1 Differences between the treatment groups

The differences between the groups were more obvious between the control group and the remainder of the treatment groups (Figure A.III.15). The rate of progress among the subjects in the control group affected with

this pain was considerably slower when compared with the immobilised and active exercise groups. All those subjects in the immobilised group affected with pain along the nuchal line were fully recovered within six months of the initial injury, while those subjects with a similar pain problem in the active exercise group recovered before the end of the study. In contrast, the final examination at week 52 demonstrated that a few subjects in the control group continued to experience pain along the nuchal line (Tables A.III.4.8 to A.III.4.10). It was evident from the pattern of recovery that those subjects who continued to experience this pain even after six months post-injury, were likely to remain symptomatic up to 52 weeks post-injury. A comparison of nuchal line pain frequencies as evidenced at the initial and final examination indicated a considerable reduction in the number of the subjects and also that it is unlikely for an association to exist between pain along the nuchal line and occipital headache.

6.9 Pain in the cervico-thoracic junction (C.T. junction)

The pattern of recovery evidenced in the current study is interesting in that this was the only symptom in which all three treatment groups experienced deterioration during the second half of the study period (Figure A.III.3). The characteristics of the pain-related symptom associated with cervico-thoracic junction is not available in the literature. The proportion of reduction between the initial examination and the final follow-up examinations for each of the treatment groups, indicated that a relatively large proportion of the injured population were likely to develop chronic symptoms associated with pain in the C.T. junction region. A comparison between the treatment groups showed that the immobilisation regimen offered the best result, whereas the control group was characterised by marked fluctuations in response. The rate of recovery was at its best for all the groups between the third and six months after sustaining the injury. As

in previous observations, the individuals who are likely to develop chronic symptom associated with the C.T junction may be identified at six months post-injury.

6.10 Symptoms associated with temporomandibular Joint (T.M.J) pain

Several reports claim a strong association between the "whiplash" type of injuries and T.M.J pain (Heise et al., 1992). The incidence of T.M.J pain among "whiplash" patients is a matter of contention and for that reason the following account is presented. In the current study, initial examination showed that 4.5% of the total sample was affected with T.M.J symptoms (Table.5.4.3). At the end of the study period 2.4% of those subjects still remained symptomatic. Thus, the observations made in the current study supported Heise et al., (1992) who concluded that a relatively small number of individuals are affected with TMJ problem following "whiplash" type of injury. The observed differences between the treatment groups demonstrated wide fluctuations in the rate of progress (Figure A.III.22, A.III.23). However, noticeable progress was again observed among the immobilised group. Initially recovery was noticed for all the groups, during the first four weeks post-injury and thereafter a pattern of aggravation was evident among the active exercise and the control groups. It was apparent that most of the subjects with continuing T.M.J pain went on to develop chronic problem approximately six months after the MVA. It should be noted that none of the subjects were treated specifically for the T.M.J problem. The observed progress or lack of it may either be incidental or associated with the natural course of events that occurs during soft tissue healing.

6.11 Pain symptoms associated with sternomastoid muscle

Although the experimental studies and anecdotal evidences indicated that the sternomastoid muscles are predisposed to be injured as a result of "whiplash" mechanism, there is little information on the incidence and the pattern of progress associated with pain symptoms from this muscle (MacNab, 1973; Frankel, 1971). In the current study, 20% of the subjects examined at the time of the initial examination after the MVA were affected with pain in the sternomastoid muscle, while 2% of the subjects in the study at 12 months post-injury, continued to experience this pain symptom. The incidence of pain associated with the right sternomastoid muscle was slightly higher than its left counter part. A similar difference was observed between the different parts of this muscle which showed that the incidence of pain in the tendinous upper part was slightly greater than in the middle and the lower parts of the sternomastoid muscle. These differences may be an indication of the severity of the trauma sustained by these muscles and as a consequence the rate of recovery from pain symptoms associated with the right sternomastoid was slightly slower than the left. In addition, the rate of recovery as evidenced during the study period showed that a considerable number of subjects recovered completely from pain associated with this muscle by 6 weeks post-injury and thereafter the rate of progress declined significantly. The final examination at the end of the study period showed that 2% of those subjects assessed continued to have pain in the upper part and 1% of the subjects developed chronic pain in the middle and lower parts of the right sternomastoid muscle.

6.11.1 Differences between the treatment groups

Distinct differences were observed between the treatment groups which showed that the subjects treated by the immobilisation regimen achieved a sustainable recovery when compared to those subjects treated by the other

two regimens (Figures A.III.16 to A.III.21). It appeared that a small number of those subjects from the active exercise group, had relapse six months post-injury in their pain coming from the right sternomastoid muscle and developed a chronic problem (Table A.III.4.9). The pattern of recovery among the control group indicated a slower rate of progress when compared to the active exercise group. A small number of the subjects from the immobilised group also appeared to have a relapse during the period between week 12 and week 24 after the MVA (Table A.III.4.8). The observations demonstrated that the chronic pain symptom associated with sternomastoid muscle was more prevalent among those subjects who were encouraged to exercise during the acute stage of "whiplash" injury and that those subjects who continued to be affected with pain from the muscle, six months after the MVA are likely to develop a chronic problem.

6.12 Pain in the pre-tracheal region

The pain in the pre-tracheal region reported by 25% of all subjects examined at the time of the initial assessment was invariably associated with swallowing food or drinking fluids. The follow-up examinations performed at week 4 and week 6 showed that 1% of the examined during the respective periods had a continuing pain symptom in this region and the subsequent follow-up examinations showed that there was no incidence of relapse. A comparison between the treatment groups indicated that all of the subjects in the immobilised and the controls group had recovered completely from this symptom within 4 weeks after the MVA, while 3% of the subjects in the control group continued to experience pain in the pre-trachial region up to 6 weeks post-injury. On the basis this observation it can be concluded that a complete resolution of the pain symptom from the pre-trachial region is likely to within 6 weeks following the MVA.

6.13 Strength of the pre- and post-vertebral neck muscles

There is a lack of information regarding the adverse effect on the strength of the neck muscles from the long term use of the cervical collar. Although there is little or no evidence to indicate an association between disuse atrophy of the neck muscles and prolonged use of the cervical collar, there is a widespread opinion which does not favour immobilisation of the neck after acute injury (Martin, 1959; Farbman, 1973; Janecki and Lipke 1978). This question was addressed in the current study and is discussed in the following section.

Since most of the subjects complained of severe pain in the neck during the strength test manoeuvre and failed to complete the tests, it was not possible to establish the differences in the muscle strength between the three treatment groups at four weeks after the MVA. However, the tests performed at six weeks post-injury, clearly demonstrated that the subjects in the immobilised group were able to generate a stronger muscle contraction than those in the other two groups. Although there was difference between the immobilised and the active exercise groups, a statistically significant difference was only present between the immobilised and the control groups. This interesting observation which was contrary to the generally held opinion and thus needs further consideration.

The observed differences in the peak force generated by the treatment groups do not mean necessarily that subjects in the active exercise and the control groups had developed weakness in the neck muscles, nor that the subjects in the immobilised group had gained more strength as a result of the treatment received. In this situation, the ability to generate a peak force during muscle contraction was affected by the level of pain experienced by

an individual. An examination of the relevant geometric means of the pain experienced for the corresponding time period, would indicate that the subjects in the immobilised group had relatively less pain compared with those in the active exercise and the control groups (Table 5.5.5). Therefore, the subjects with the least pain level were able to produce a stronger muscle contraction than those subjects in the other two groups. This argument was further verified by appropriate statistical tests and found to be valid (pages 218, 220).

This finding can be explained in terms of function and structure of the neck muscles. Large powerful muscles such as deltoid, the gluteal muscles and quadriceps femoris are well known for their rapidity of atrophy within a relatively short duration of inactivity, whereas smaller muscles do not appear to weaken in a similar manner (Cyriax, 1978). Clinical observations showed that those patients who have had either subluxation or fracture in the cervical spine and who are treated by immobilisation of their neck for up to 3 months (and some instances even longer), do not appear to show any adverse effect from disuse atrophy of the neck muscles (Batalin, 1990). The study results clearly demonstrated that the "whiplash" patients who are treated by an immobilisation regimen as prescribed in the current study, are unlikely to be disadvantaged as a result of disuse atrophy of their neck muscles.

6.14 The effect of immobilisation on range of motion of the cervical spine

The long term immobilisation of the neck is considered to result in irreversible changes in the neck musculature and associated collagenous tissues, thereby causing a reduction in the range of motion of the cervical spine (Frankel, 1959; Martin, 1959; Farbman, 1973). However, it appears

that the concern is based largely on subjective opinion and is lacking in scientific evidence to support such claims. This important issue was addressed in the current study, thus the effect of the immobilisation of the neck on the range of motion at various point in time of the study period was evaluated.

The initial examination of the range of motion of the cervical spine demonstrated that the subjects in the immobilised group had less movement (statistically significant) in all directions when compared to the active exercise and the control groups (Table 5.8.2). In stark contrast, at 4 weeks after the MVA, the immobilised group showed a considerable increase in the range of motion in all directions when compared to the other two treatment groups and this progress was consistently maintained throughout the whole study period. It should be noted that subjects in the active exercise group showed an advantage in the range of motion over the other two groups at the commencement of the current study and yet lagged behind the immobilised group, as evidenced at all of the follow-up examinations except that performed at week-4 post-injury. Although this result may be contrary to generally held clinical views, a consideration of the underlying factors help clarify the situation.

The only difference between the immobilised and the active exercise treatment groups occurred during the first 4 weeks following the MVA consisted of the use of the Philadelphia collar by the immobilised group. Thereafter both of these groups performed similar mobilising exercise programme. In this situation, the general physical therapy expectation would be that the active exercise group would show a relatively larger gain in range of motion, throughout the study period, when compared to the group which had been immobilised for the same period of time. It is

commonly believed that active exercises will prevent joint stiffness by limiting either the formation of adhesions or scar tissue and by the maintenance of compliance in the soft tissues surrounding synovial joints. However, the current results indicated that joint stiffness did not appear to have occurred in the immobilised group during the early stages of "whiplash" and thus alternative explanations are required. The statistical analyses showed that the pain factor and the age of the subjects had a significant effect on the range of motion (Tables A.III.8.12 to A.III.8.16). A comparison of the geometric mean of the pain for the respective treatment groups at a corresponding point in time, confirmed the existence of a strong association between the pain factor and the changes in the range of motion. Although the age of the subjects was also shown to have an effect on the range of motion, this association was not as strong as the pain factor. Therefore, the immobilised group which showed the largest reduction in the level of pain experienced, demonstrated the most noticeable increase in the range of motion of the cervical spine. Thus, the results of the current study clearly show that the immobilisation regimen had no adverse effect on the range of motion of the cervical spine.

There is general view held by many clinicians, that the "whiplash" injury is likely to result in the development of adhesions and scar formation in the capsule of the zygapophyseal joints (Hohl, 1983; Mealy et al., 1986; McKinney et al., 1989). This issue requires clarification since clinicians often justify the use of certain physical treatment modalities for the management of "whiplash" type of injuries. These include cervical traction and spinal mobilisation procedures, and are performed under the assumption that they prevent pathoanatomical changes from occurring within around the joint (Mealy et al., 1986; McKinney et al., 1989). In general, the formation of adhesions in synovial joints mainly occur following

trauma which is associated with considerable effusion in and around a joint. The subsequent organisation of serous exudate is primarily responsible for the adhesions which develop in the soft tissues around the joint. In a recent histological study Twomey and Taylor (1991) observed that the capsule of the cervical zygapophyseal joints is not structurally well developed and the observations made in the current dissection study also showed that the cervical facets joint capsules are thin and weak structures. Thus, inflammatory resulting from injury to the cervical spine, instead being contained within a complete fibrous joint capsule would more readily disperse into the neighbouring tissue spaces enhanced by the influence of gravity. This offers a plausible explanation as to why soft tissue adhesions are less likely to occur around the zygapophyseal joints of the cervical spine and thus the use of procedures such as movement, manipulation and traction may not be necessary, nor can they be justified on the basis of either maintaining or improving the range of motion in the cervical spine.

6.15 Resumption of full normal duties

The result of the current study demonstrated that 50% of the subjects from the immobilised group returned to their normal duties at a level equivalent to the pre-accident level at 7.7 weeks post-injury while the subjects from the active exercise and the control groups have taken 11.2 weeks and 20.4 weeks respectively. Similarly, at the end of the study period fewer subjects in the immobilised group have not returned to their normal level of duties when compared to those in the other two treatment groups (Tables 5.9.1 to 5.9.3). Although there was no statistically significant difference between the groups in the number of subjects returning to their pre-accident level, a relatively shorter median survival time for the immobilised group clearly demonstrated the advantage over the other two treatment groups and in socio-economic terms these differences are quite significant.

6.16 Prognostic indicators

Although several factors associated with "whiplash" type of injuries have been reported to have prognostic significance, their validity requires verification and the following account is based on the analyses of the current study.

The rate and speed of recovery has been shown to be affected by the age and gender of the people involved (Hohl, 1974;). The results of the present study confirmed that there was a statistically significant difference between the female and male subjects in the level of pain, which indicated that the female population affected by "whiplash" type of injury is likely to respond more slowly than the males (Hohl, 1974; Bovim et al., 1994). Although the current study demonstrated that the age factor was also a statistically significant prognostic variable, the sample size was not large enough to identify the exact age group involved. However, the scatter graph showed that the subjects below the age of 30 years recovered quicker than the remainder (page 362). In respect of occupational categories, the statistical analyses failed to demonstrate a consistent statistical significance during the current study and therefore it is not possible to confirm the prognostic value of the occupational categories considered in the present study. Although Dean et al. (1986) considered that the "whiplash" injuries arising specifically from rear-end collisions were associated with poor prognosis, recent studies have clearly shown that the rear-end collision had no prognostic value (Maimaris, et al., 1988; Hildingsson and Toolanen, 1990; Spitzer et al., 1995). The results of the current study while confirming the lack of prognostic significance attached to the rear-end collision, also supported Maimaris et al.(1988) who observed that neither the type of collisions in itself, nor the seating position of the individual, had any

prognostic value in predicting the progress of the "whiplash" patients. However, the current study showed that the speed of the vehicles involved in the MVA is a significant prognostic indicator. There is no information available regarding the onset of headache within 24 hours after the MVA and its predictive value on the progress of the "Whiplash" injury and the results of the current study also showed that this symptom had no predictive value. The presence of paraesthesia was shown to have prognostic significance and this finding is in agreement with the reports of previous study (Watkinson et al., 1991).

The loss of lordosis of the cervical spine and presence of spondylitic changes as evidenced in the radiological examination of the cervical spine are considered by several authors to have prognostic value. (Hohl, 1974; Norris and Watt, 1983; Miles et al., 1988; Porter, 1989, Radanov et al., 1995). However, Maimaris et al.(1988), Pearce (1989), and Hildingsson et al.(1990) disputed the prognostic value of some or all of these radiological features and the results of the current study clearly showed that the loss of lordosis and the presence of pre-existing spondylitis changes do not have any prognostic value. The analyses showed that the presence of interscapular pain did have prognostic value and this is in agreement with other reported observations (Greenfield and Ilfed, 1977; Hohl and Hopp, 1978; Norris and Watt,1983; Maimaris et al.,1989). In addition, the current study also demonstrated that the severity of pain experienced within 60 hours of the MVA, had prognostic significance. This observation supported the conclusions of several studies which indicated that initial severity of pain reported by "whiplash" patients had prognostic value (Deans et al, 1986; Deans et al., 1987; Gore et al, 1987; Ryan et al., 1993; Radanov et al., 1995). Ocular symptoms, such as difficulty in focusing and blurred vision are the result of lesions in the cranial nerves and brain stem and

considered invariably associated with poor prognosis (Hildingsson et al., 1989). The current study also showed that presence of blurred vision and difficulty in focusing were indicative of poor prognosis. There is disagreement in the literature regarding the effect of litigation on the recovery of those affected with "whiplash" type of injuries. The potential financial gain to be made through litigation and psychological state of the "whiplash" patients are considered to be significant factors which adversely affect the outcome of the treatment. (Gay and Abott, 1953; Gotten, 1956; Miller, 1961; Miller, 1966; Balla, 1970; Breck and Van Norman, 1971; Awerbuch, 1992). On the contrary, MacNab (1964, 1971) refuted the claim that the litigation played a major role in the progress of "whiplash" patients and several authors are in agreement with his observations (Depalma and Subin, 1965; Janes and Hooshmand, 1965; Hohl and Hopp, 1978; Mendelson, 1982; Maimaris et al., 1988; Pearce, 1989; Newman, 1990). However the results of the current study failed to demonstrate a consistent association between the litigation status and the pattern of progress. At the time of the initial and final examinations, statistically significant differences were observed in the level of reported pain between the compensable and non-compensable subjects, while the follow-up examinations performed in between these measurement periods failed to show such differences. For these reasons, it was concluded that there was no consistent evidence to show that the litigation factor had an adverse effect on the progress of "whiplash" patients.

6.17 Observations of secondary interest

Although it may not be relevant to the objective of the current study, It is considered that the following observations may be of interest to both clinicians and future investigators.

At the time of the initial examination a number of subjects reported that they were affected by the so called "bizarre" symptoms of dizziness, ocular dysfunction including photo sensitivity, impairment of memory, clumsiness and mood swings. These symptoms have been cited as the basis for "whiplash" patients being challenged by clinical specialists who often dismiss the existence of such symptoms for lack of demonstrable organic causes. On the contrary, there is abundant information which demonstrates that injury to the vertebral artery, subtle organic cerebral dysfunction and lesions in the brain stem are responsible for these symptoms in "whiplash" patients (Horwich and Kasner, 1962; Krantz and Lowenhielm, 1986; Yarnell and Rossie, 1988; Hildingsson et al., 1989; Burke et al; 1992). Furthermore, the presence of these symptoms among such patients is considered as an indication of the severity of the injury sustained by the individual (MacNab, 1964; Ommaya et al., 1968; Shifrin, 1991; Radanov et al., 1995). For instance the presence of ocular symptoms have been shown to have prognostic value and yet it appears that clinicians and medical specialists have been largely unaware of the underlying importance attached to these symptoms (Hildingsson et al., 1989).

Those "whiplash" subjects who complained of impairment in their memory and clumsiness in either one or both hands have often considered to be in an emotional state causing considerable strain at work and among family members (Yarnell and Rossie, 1988; Radanov et al., 1992). The observations made in the current study indicated that such problems may last in a majority of the subjects up to six months post-injury. It is strongly recommended that due consideration be given to investigate the so called "bizarre" symptoms, so that an appropriate explanation and counselling may be offered to ameliorate the problem.

A number of subjects complained of paraesthesia in the tips of their digits on both hands and similar presentations are often dismissed by the clinical specialists on the basis that they are inappropriate, non-specific and non-anatomical in distribution (Table A.III.3.1). However, it has been clearly shown that lesions affecting the upper spinal cord can often lead to "false localisation" and other sensory disturbances (Smith and Hodge, 1992). Therefore it may be appropriate to give proper consideration to this type of atypical presentation.

The incidence of low back pain among "whiplash" patients has also been widely reported by and yet there is reluctance in the part of the physicians working for the compensable bodies and medical specialists to acknowledge the concomitant injury to the lower back (Gay and Abott, 1953; Hohl, 1974; Hildingsson and Toolanen, 1990; Watkinson et al., 1991). The results of the current study indicated that a large number of the subjects were affected with low back pain and the frequency of this symptom as evidenced during the study period are presented elsewhere (Table A.III.10.1). It is also worth noting that there were instances in which the subjects concerned fully recovered from their neck symptoms, whereas they continued to experience low back pain for which eventually had to undergo surgical treatment such as discectomy.

Similarly, 4 of the 220 subjects from the current study were diagnosed as having rotator cuff lesion as result of the MVA and two had rotator cuff repair surgery. The interesting feature among these subjects was the manner in which their shoulder symptoms mimicked as those of nerve root compromise.

The observations made from the MRI study are unique as there is no information presently available which is based on a longitudinal study of the intervertebral disc lesion and the lesions of the anterior longitudinal ligament which arise from "whiplash" type of injury. The MRI demonstrated the progressive nature of the disc degeneration as early as three months following MVA and the scan performed twelve months after the injury further showed advanced degenerative changes in the I.V.D. In addition, it was evident that the anterior longitudinal ligament was more prone to injury as a result of "Whiplash". None of the subjects examined by MRI showed compression of the nerve root as a result of disc lesion although there were instances in which a disc bulge abutted the subarachnoid space without compromising the spinal cord. This type of lesion may either interfere with the vascular supply to the sections of the spinal cord or cause venous congestion within the spinal canal and this situation is likely to be one of the causes for headache reported by "whiplash" patients (Jayson, 1993).

During the course of the follow-up examinations, the subjects were asked to list problems in relation to their activities of daily living. In this way, an unexpected social and emotional problem was identified. A number of subjects in the current study reported that their sex life has been greatly affected due to their neck pain and in some instances blamed the back pain for the problem. It was evident that in instances the personal relationships had been significantly affected leading to their break down. This aspect of problem affecting the personal life of "whiplash" patients has not yet been widely reported and warrants further study by an investigator from an appropriate investigator.

6.18 Clinical implications

The results of the current study conclusively demonstrated that early immobilisation followed by a defined active exercise programme is by far the best physical therapy approach to the treatment of "whiplash" type of injuries. These observations are in stark contrast to the results of the previous authors (Mealy et al., 1986; McKinney et al., 1989; Pennie and Agambar, 1990). The observed differences may be attributed to the methodologies adopted in these studies. It is apparent from the previous studies that use of soft collars is not an effective method of immobilising the neck and also that the duration of immobilisation is critical in achieving an optimum soft tissue healing. Therefore, it is not surprising that those subjects who were immobilised in a soft collar for 2 weeks, failed to show the positive results and lead to the conclusion that inactivity during the early stage of "whiplash" injury is not conducive for a good recovery. However, it can be argued that the better method of immobilisation used in the current study largely contributed to the noticeable advantage gained by those subjects in the immobilised group. In this regard, the factors that adversely affect wound healing include poor blood supply and excessive movement during the acute stage of healing thus causing persistent break down of newly laid granulation tissues (Walter and Israel, 1989; Montgomery, 1989). Furthermore, it has been demonstrated that in the case of skeletal muscle injury, that early immobilisation promote an accelerated formation of Type I collagen and thus reduced the risk of wound break down (Lehto et al., 1985). Subsequently, mobilisation is recommended to improve the growth and orientation of regenerating muscle fibres and the reabsorption of the scar tissues (Lehto et al., 1985). The importance of approximation of the wound edges in order to protect the newly laid tissues from adverse tensile forces is even greater in the case of injury to tendinous structures which have a slower rate of wound healing due to their poor blood supply

(Gelberman et al., 1980; Nystrom and Holmlund, 1983; Korkala et al., 1984; Montgomery, 1989).

In "whiplash" type of injury, lesion of the ligaments, tendinous structures and the skeletal muscles are common. It was apparent from the dissection study that the multifidus muscle in the cervical region is prone to injury in "whiplash". The reason being this muscle is the primary soft tissue structure which controls the horizontal translation movement in the zygapophyseal joints. In an extreme situation the horizontal translation may cause major injury to the multifidus and the weak capsular structure it reinforces. Similarly, the tendinous attachments of semispinalis capitis lateral to the articular surface of the zygapophyseal joint, along the nuchal line and in the interscapular region are also likely to be injured. The proximal attachment of the upper fibres of trapezius muscle is another structure which is often affected in a "whiplash" type of injury. Thus, it is evident that the immobilisation procedure adopted in the current study has provided the proper situation for better wound healing by preventing excessive movement. Furthermore, the subsequent active exercise programme (after 4 weeks), has helped in restoring the biomechanical characteristics of the soft tissues. It should be noted that patients with uncomplicated fractures in the cervical region or after reduction of subluxation of the zygapophyseal joints, are often treated by immobilisation of their neck, without adversely affecting the biomechanical properties of the soft tissues. By promoting better healing, the immobilisation regimen also would have averted the pathophysiological changes which contribute for the development of "Phantom pain".

6.18.1 Classification of "whiplash" type of Injuries

On the basis of the clinical observations and the results of the current study, the following classification of "whiplash" injury is recommended for the use of clinicians:

First Degree	Pain level <30 in a numerical rating scale or VAS
	Full range of motion in the cervical spine
Second Degree	Pain level 30-60 in a numerical rating scale or VAS
	Range of motion in the cervical spine reduced by one thirds of normal level
Third Degree	Pain level 60> in a numerical rating scale or VAS
	Range of motion in the cervical spine reduced by half of the normal level
	Presence of paraesthesia in the fore-limbs, demonstrable neurological signs, presence of blurred vision and difficulty in focusing
Fourth Degree	Pain level 60> in a numerical rating scale or VAS
	Range of motion in the cervical spine reduced by half of the normal level
	Presence of paraesthesia in the fore-limbs, demonstrable neurological signs, presence of blurred vision and difficulty in focusing
	Radiological examination showing widening of inter body distance, widening of inter spinous space
	MRI findings demonstrating soft tissue lesions

6.19 Recommendations

Although the observed results were quite unambiguous in favour of immobilisation in the early treatment of "whiplash" type of injury, further investigation with even a larger sample size and over an extended study period perhaps up to two years, may provide additional information. Such a study would provide an opportunity to more clearly consider some of the chronic problems identified in this study. Although a double blind study

would have been desirable, it would require substantial funding and full co-operation from both the insurers and the patients concerned. The efficacy of vertebral mobilisation techniques may also be worth investigating and in a future study the control group may be replaced by vertebral mobilisation regimen. It is also recommended that in any future study selection criteria is formulated so that carefully identified subjects may be examined by specialists in the field of ophthalmology and neurology as a part of the initial examination. It is also stressed that for obvious reasons that clinicians refrain from applying "high velocity low amplitude" manipulative procedures as a form of treating "whiplash" type of injuries. Similarly, the use of cervical traction is also not recommended. The prevalence of low back pain among "whiplash" patients also calls for an investigation.

CHAPTER SEVEN CONCLUSION

"Despite the frustratingly negative examination, the patient's aches and pains respond to the Brownian movements of routine medical therapy: pulls, pills, and the passage of time. The impression is gained, however, that the symptoms are worse after every visit to the lawyer's office" (MacNab 1973).

The main objective of this prospective randomised study was to evaluate two conservative treatment regimens (early immobilisation-experimental group-1, early active mobilisation- experimental group-2) which are based on accepted although differing physiological rationale and then to compare their effectiveness with those of existing treatment regimens that are commonly practiced (control group), in the management of "whiplash" type of injuries. To this stage, the current study is the only prospective randomised clinical trial conducted with a sufficiently large sample size and over a long study period.

The results of the current study demonstrated that the subjects in the immobilised group recovered from their pain-related symptoms and returned to their normal duties sooner than those in the other two treatment groups. In addition to this, the results clearly showed that those subjects who were immobilised for 4 weeks, did not show any adverse effect on either the range of motion or the strength of their neck muscles. Thus, the immobilisation regimen was clearly shown to be the preferred option, when compared to the remainder of the treatment methodologies adopted in the current study.

The results of the current study identified a number of prognostic indicators. It was evident that younger age group recovered sooner from their "whiplash" symptoms. The results also indicated that female patients are likely to recover slower than males and that there is no correlation between the type of collision and the rate of recovery. Similarly, the seating position of the "whiplash" patients had no predictive value on the severity of the injury. However, the severity of the pain experienced by the patients within 60 hours post-injury does has predictive value. Presence of paraesthesia, difficulty in focusing, blurred vision and interscapular pain were demonstrated to be indicators of poor prognosis while the loss of cervical lordosis and pre-existing spondylitic changes had no prognostic value. The speed of the vehicles had an association with the rate of recovery. The study results did not provide conclusive evidence to support the contention that the litigation factor had an adverse effect on the progress of "whiplash" patients. For the first time this study has reported information regarding the evolution of pain related symptoms such as headache, neck pain, pain arising from trapezius and sternomastoid muscles, interscapular pain, pain in the pre-tracheal and the cervico-thoracic regions and pain in the TM joints. This information may be useful to clinicians in the management of the "whiplash" type of injury. On the basis of the observations made in the current study, a classification of the "whiplash" injury has been proposed for the clinical consideration.

As an adjunct to the main study, the morphology of the deep pre- and post-vertebral muscles of the neck region were established using embalmed cadavers and fresh post-mortem specimens, as the literature was considered deficient in this regard . In addition, a histological study of the longus capitis and rectus capitis anterior was performed in order to elucidate the functional significance of the latter. The histological study

showed that the rectus capitis anterior muscle has a rich innervation which are disproportionate to its size and this observation suggested that this quite small muscle may be well suited to play a proprioceptive role rather than have a mechanical function.

A longitudinal study of 45 subjects was also performed using Magnetic Resonance Imaging technology as a further part of the total study. The longitudinal nature of the M.R.I study provided for the first time an account of the progressive pathological changes that occurred in some cervical disc lesions, at defined points in time following an MVA. The observations made from the adjunct studies help clarify the overall position and thus assist in a better understanding of the pathoanatomy associated with deep muscles of the neck region and pathological changes that occur following a traumatic disc lesion as evidenced within 2 weeks, after 3 months and 12 months post- injury.

Fundamentally the current study has clearly shown the need for a critical period of immobilisation subsequent to an acute "whiplash" type of injury.

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APPENDIX-I RELIABILITY STUDY OF CROM GONIOMETER

A.I.1 Introduction

A pilot trial was conducted in order to determine the intratester reliability for the measurements of active and passive flexion, extension, rotation and side flexion range of motion in the cervical spine, documented by means of CROM goniometer.

A.I.2 Methodology

A.I.2.1 Subjects

Twenty eight volunteers without neck symptoms were recruited from two hospitals and a physiotherapy private practice. The subjects consisted of fifteen females and thirteen males aged between 22 and 58 years. The subjects were either staff members or relatives of patients who agreed to participate in this pilot trial.

A.I.2.2 Procedure

The seating arrangement and positioning of the CROM were similar to the main study, as described in methodology chapter. The measurement of active and passive range of motion of flexion, extension, rotation (right & left) and side flexion (right & left) was repeated four weeks after the initial recording and during this interval the medical status of the subjects remained unaltered.

A.I.2.3 Extension

The investigator stood on the left side of the subjects and reminded the subjects not to slump or to move their body while testing. At this stage the investigator placed his right palm at the interscapular region and applied gentle pressure in order to prevent the trunk from moving backward. The subjects were asked to nod the head backward and then continue to take it as far back as possible into extension and remain still until the reading was

noted from the sagittal goniometer. After returning their head to the neutral position, one minute rest was allowed followed immediately by the recording of the passive ROM of extension. This was performed by repeating the procedure as mentioned for active ROM and applying a gentle pressure over the fore head by the left hand of the investigator.

A.I.2.4 Flexion

The starting position was similar to the one described above. The investigator gently restrained the trunk from moving forward, by applying the left palm over the upper part of the subject's sternum, taking care not to limit the forward flexion of the cervical spine. To assume full flexion in the cervical spine, the subjects were first instructed to nod the head to make a double chin and then encouraged to move the head, as far as possible "as in touching the upper chest with the chin". As soon as the end of the available range was reached, the subjects were asked to remain still until the goniometer was read. After returning the head to the neutral position the subjects were allowed to rest for one minute and then this was followed by the recording of passive ROM of flexion. This was performed by repeating the procedure as described for active ROM and after reaching the end of range, a gentle pressure was applied over the occiput by the right hand of the investigator.

A.I.2.5 Rotation

The starting position was similar to that described in the previous section. The subjects were asked to focus their eyes on to a horizontal line on the wall so that the head was not tipped laterally during rotation. Prior to the commencement of the rotation movement, using the sagittal and the frontal goniometers, the cervical spine was kept in a neutral position. In order to ensure that no shoulder rotation occurred, the left shoulder was lightly stabilised from the front, while the subjects turned their face to the right side and vice versa. During this procedure, the subjects were constantly

reminded to focus their eyes on to a horizontal line on the walls. The active and passive range of motion were recorded using the compass. As soon as the end of the available range was reached, the subjects were asked to remain still until the compass reading was noted and were then asked to return their heads to neutral position. The passive ROM reading was carried after a rest period of one minute. This was performed by repeating the procedure as described for active ROM and after reaching the end of range, a gentle pressure was applied over the ipsi-lateral side of the occiput and contra lateral side of the face. During this procedure the subjects were constantly reminded to sit upright and not to move their bodies.

A.I.2.6 Lateral Flexion

There was no change to the original starting position. In order to avoid unwanted movement in the cervical spine during the testing procedure, the subjects were instructed to focus on a point in front of them on the wall. The posture of the head was corrected so that the sagittal and frontal meters were remained at zero. A standard instruction, "Bend your neck to the (right / left) side as far as you can and take care not to move the body". During this procedure the investigator prevented the shoulders from being raised or lowered, by gently touching the respective shoulder. As soon as the movement was completed the subjects were asked to remain still and the investigator recorded the reading from the frontal meter. Thus, the right and left side flexion were recorded. A similar procedure was repeated to record passive ROM after a rest period of one minute. This was performed by repeating the procedure as described for active ROM and after reaching the end of range, a gentle pressure was applied over the contra lateral side of the face. During this procedure the subjects were constantly reminded to sit upright and not to move their bodies.

A.I.2.7 Statistical analysis

An alpha level of <0.05 was chosen as a criterion of significance for the analyses, in order to establish the intratester reliability of the measurements of movements in the cervical spine, obtained using CROM goniometer. A one way repeated measures analyses of variance (ANOVA) was performed for each of the movement, to calculate intraclass correlation coefficients (ICC) in order to establish the intratester reliability of the measurements obtained in the current pilot study.

A.I.2.8 Result

The ICCs for intratester reliability of the measurements obtained using CROM device are presented in the table A.I.1.

Table A.I.1 **Intraclass correlation coefficients (ICC) for intratester reliability of the CROM (n=28).**

Direction of movement	I C C
Active Flexion	.96
Passive Flexion	.98
Active Extension	.98
Passive Extension	.98
Active Rotation Right	.97
Passive Rotation Right	.97
Active Rotation Left	.96
Passive Rotation Left	.97
Active Side Flexion Right	.98
Passive Side Flexion Right	.97
Active Side Flexion Left	.97
Passive Side Flexion Left	.97

A.I.2.9 Discussion

ICC is the preferred statistics for establishing the intra and intertester reliability of the investigators performing experimental studies (Hass, 1991). Youdas et al. (1991) characterised the ICC values as: .90 to .99, high reliability; .80 to .89, good reliability; .70 to .79 fair reliability; and .69 and below poor reliability. On the basis of this guideline, it was shown that the ICC values for all the neck movements measured in the current pilot study were highly reliable. Thus, it was evident that the measurements of neck movements obtained using the CROM device are highly reproducible when repeated by the same investigator. Therefore, it can be concluded that the CROM device is a reliable instrument for documenting the active and passive cervical range of motion in the current study.

APPENDIX-II RELIABILITY STUDY OF CERVICAL MUSCLE STRENGTH TESTING DEVICE (C.M.S.T.D)

A.II.1 Introduction

In order to measure the strength of the pre- and post-vertebral neck muscles, in an out patient department where the clinical trial was conducted, an electronic muscle strength testing device was constructed and a pilot trial was conducted in order to establish the intratester reliability of the C.M.S.T.D. It should be noted that at the commencement of this study, a similar device capable of being used as a clinical tool was not available (Highland et al., 1992; Vernon et al., 1992).

A.II.2 Description of C.M.S.T D

An electronic strain gauge capable of converting mechanical deformation generated in a steel strain gauge, into a measurable electrical energy was fabricated by the design engineering department, Curtin University of Technology (Figure 3.4, 3.5). A steel beam which could withstand a force of 100 kgs was used to construct a force platform. The force platform comprised of four load cells, two on each side of the steel beam to generate electrical potential. A padded head rest and an adjustable head band were incorporated to the force platform, so that the subject did not experience discomfort during the test. A head (vertex) restraint was also incorporated and this arrangement ensured that the subject's head was securely fixed against the platform during all testing manoeuvres. The electrical potential generated by the load cells was amplified and the resulting voltage readings were obtained in a graphic form, via a pen recorder (Yokogawa, model 4151-300-71/BU) using its absolute reading facility. The range of measurements documented by the pen recorder was defined by choosing appropriate linear scaling select codes. Thus, the pen recorder was calibrated and tested to

record a maximum of 25 kgs and a minimum of 0 kg to denote the resting position. The strain gauge was fitted to a designated examination couch (Figure 3.4). An electronic timer was used during the study to monitor the "hold period".

A.II.3 Methodology

A.II.3.1 Subjects

Thirty four volunteers without any history of neck or back symptoms either as a result of trauma or other musculo-skeletal disorders, during a period of twelve months prior to the testing, were recruited from a hospital and a physiotherapy private practice. The subjects were staff members and either friends or relatives of the patients. The sample consisted of 18 females and 16 males, aged between 17-56 years.

A.II.3.2 Procedure

Prior to the test, the subjects were given an explanation as to the purpose of the pilot study. The subjects were requested to loosen their garments so that the clothing did not cause restriction during the test manoeuvres. The strain gauge was connected to the pen recorder and the calibration was checked and adjusted if required. In an unloaded situation, the accuracy of the calibration was confirmed by the following observations:

1. pen recorder digital read out should display "0"; and
2. the tracing pen should be tracking on a grid line which was defined to be "0" in value.

The investigator, in addition to explaining the test procedures to the subjects, demonstrated the manoeuvres prior to the commencement of the test. This arrangement facilitated a better understanding of the test procedures without the subjects needing trial attempts which were likely to result in bias due to a "learning" effect. While the subjects were in supine lying, the investigator assisted the subjects so that they were comfortably positioned on the head

rest. The investigator then adjusted the subjects, so that the bony prominence located on the superior aspect of the acromion process was level with the edge of the plinth. A screw mechanism incorporated in the strut attached to the plinth, was adjusted in order to bring the crown of the head in contact with the restraining plastic strap fitted to the free end of the strain gauge beam. At this stage, the strain gauge beam was secured by tightening the fixating mechanism situated on the under surface of the plinth. A soft pad was placed on the forehead of the subjects to reduce discomfort. At the commencement of the testing, the subjects were asked to fold their arms across their chest and to keep the hips and knees flexed as in crook-lying position. This starting position prevented the subjects from arching their back as reinforcement during the post-vertebral neck muscle testing. A written instruction which was relevant to the test sequence, viz pre-vertebral or post-vertebral muscle test, was then read. In the case of post-vertebral muscle testing the instruction was,

> "At the count of five you will be given a command 'push your head down'. You must push your head down as hard as you can and hold it until I say 'let go'. Remember not to arch your back while pushing your head down".

A similar instruction was given prior to pre-vertebral muscle testing which said,

> "At the count of five you will be given a command 'lift your head'. You must lift your head as hard as you can and hold it until I say 'let go' ".

An electronic timer was used to time the duration of the test (hold period) which was ten seconds. A duration of ten seconds "hold period" was derived from a preliminary trial by testing five male volunteers. It was observed that all of the five volunteers were only able to generate and maintain the peak

force for a few seconds and that the whole process was completed within ten seconds. Since the male volunteers were physically stronger than the female sample population included in this reliability study, it was decided to administer a "hold period" of ten seconds for both genders.

A.II.3.3 Post-vertebral muscle testing

Prior to reading the written instruction, the strain gauge and the pen recorder were switched on . The standard instruction, was read and the electronic timer was switched on to coincide with the command "push your head down". The subject was instructed to "let go" at the end of ten seconds. The peak force was read from the digital display of the pen recorder and later verified with the pen recording as shown on the tracing paper. Time taken to achieve peak force and the duration for which the peak force maintained were also recorded by observing the timer. The subjects were allowed to rest for five minutes and was followed by the pre-vertebral muscle testing.

A.II.3.4 Pre-vertebral muscle testing

A relevant instruction was read and the procedure described as above followed for testing of the pre-vertebral muscle strength.

A.II.3.5 Re-testing

This was carried out between four to six weeks from the first test and the volunteers were asked to report if they have developed any neck or back symptoms during that period.

A.II.4 Statistical analysis

An alpha level of <0.05 was chosen as a criterion of significance for the analyses, in order to establish the reliability of the measurements obtained using the cervical muscle strength testing device. A one way repeated measures analyses of variance (ANOVA) was performed to calculate

intraclass correlation coefficients (ICC) in order to establish the intratester reliability of the measurements obtained from these two tests.

A.II.4.1 Result

The obtained ICCs for the pre- and post-vertebral muscle tests are .98 and .97, respectively.

A.II.4.2 Discussion

ICC is the preferred statistics for establishing the intra and intertester reliability of the investigators performing experimental studies (Hass, 1991). Youdas et al. (1991) characterised the ICC values as: .90 to .99, high reliability; .80 to .89, good reliability; .70 to .79 fair reliability; and .69 and below poor reliability. On the basis of this guideline, the ICC values for intrarater reliability obtained in the pilot study were shown to be highly reliable. Thus, it was evident that the measurements obtained using the C.M.S.T. device are highly reproducible when repeated by the same investigator. Therefore, it can be concluded that the C.M.S.T. device is a reliable instrument for documenting the force generated by the pre- and post-vertebral neck muscles in the current study.

APPENDIX-III RESULTS

Table A.III.3.1 **Paraesthesia - Frequency for dermatomes involved for each of the time periods (Total sample) Frq=Frequency; %= percentage of subjects; IE= initial examination.**

Dermatomes	Frq IE n=48	% IE	Frq wk4 n=15	% wk4	Frq wk6 n=13	% wk6	Frq wk12 n=11	% wk12	Frq wk24 n=7	% wk24	Frq wk52 n=8	% wk52
Tips of fingers-Lt	28	58.3	6	40.0	5	38.5	2	18.2	3	42.9	2	25.0
Tips of fingers-Rt	25	52.0	4	26.7	3	23.1	4	36.4	2	28.6	1	12.5
C 8 Lt	5	10.4	6	40.0	6	46.2	1	9.1	1	14.3	-	-
C 8 Rt	4	8.3	3	20.0	2	15.4	2	18.2	-	-	2	25.0
C 7 Rt	4	8.3	1	6.7	2	15.4	2	18.2	2	28.6	1	12.5
T1 Lt	4	8.3	3	20.0	5	38.5	1	9.1	1	14.3	-	-
T1 Rt	3	6.3	2	13.3	1	7.7	2	18.2	-	-	1	12.5
C7 -Lt	3	6.3	3	20.0	1	7.7	3	27.3	2	28.6	2	25.0
L5 -Rt	3	6.3	2	13.3	-	-	-	-	-	-	-	-
C 6 - Rt	2	4.2	2	13.3	2	15.4	1	9.1	1	14.3	1	12.5
L5 -Lt	2	4.2	1	6.7	-	-	-	-	-	-	1	12.5
S1-Lt	2	4.2	-	-	-	-	-	-	-	-	1	12.5
C 5- Rt	1	2.1	1	6.7	-	-	-	-	-	-	-	-
C 6-Lt	1	2.1	3	20.0	1	7.7	2	18.2	1	14.3	2	25.0
S 1-Rt	1	2.1	1	6.7	-	-	-	-	-	--	-	-
Trigeminal -Lt	1	2.1	-	-	1	7.7	-	-	--	-	-	-

Table A.III.3.2 **Paraesthesia - Frequency for dermatomes involved for each of the time periods (Immobilised group) Frq = Frequency; %= percentage of subjects; IE = initial examination.**

Dermatomes	Frq (IE) n=18	% (IE)	Frq wk4 n=4	% wk4	Frq wk6 n=4	% wk6	Frq wk12 n=3	% wk12	Frq wk24 n=1	% wk24	Frq wk52 n=0	% wk52
Tips of fingers-Lt	10	55.6	-	-	1	25	1	33.3	0	0	-	-
Tips of fingers-Rt	10	55.6	2	50	2	50	1	33.3	1	100	-	-
C 8 Rt	3	16.7	1	25	-	-	-	-	-	-	-	-
C 7 Rt	3	16.7	-	-	-	-	-	-	-	-	-	-
C 8 Lt	2	11.1	1	25	1	25	1	33.3	-	-	-	-
C 7Lt	2	11.1	-	-	-	-	1	33.3	-	-	-	-
T1 Rt	2	11.1	1	25	-	-	-	-	-	-	-	-
T1 Lt	1	5.6	-	-	1	25	1	33.3	-	-	-	-
C 6_Lt	1	5.6	1	25	-	-	-	-	-	-	-	-
L5 -Lt	1	5.6	-	-	-	-	-	-	-	-	-	-
S1-Lt	1	5.6	-	-	-	-	-	-	-	-	-	-
Trigeminal -Lt	1	5.6	-	-	1	25	-	-	-	-	-	-

Table A.III.3.3 **Paraesthesia - Frequency for dermatomes involved for each of the time periods (Active exercise group) Frq=Frequency; %= percentage of subjects; IE= initial examination.**

Derma-tomes	Frq (IE) n=6	% (IE)	Frq wk4 n=1	% wk4	Frq wk6 n=1	% wk6	Frq wk1 2 n=2	% wk1 2	Frq wk2 4 n=3	% wk2 4	Frq wk5 2 n=2	% wk5 2
L-5 _Lt	1	16.7	-	-	-	-	-	-	-	-	-	-
L5- Rt	1	16.7	-	-	-	-	-	-	-	-	-	-
Tips of fingers Lt	5	83.3	1	100	-	-	-	-	1	33.3	-	-
Tipsof Fingers - Rt	3	50.7	1	100	-	-	1	50	1	33.3	-	-
C6 Rt	-	-	-	-	1	100	1	50	1	33.3	-	-
C7 Rt			-	-	1	100	1	50	2	66.7	1	50
T1 -Lt	-	-	-	-	-	-	-	-	1	33.3	-	-
C8-Lt	-	-	-	-	-	-	-	-	1	33.3	-	-
C7 Rt	-	-		-	-	-	-	-	-	-	2	100
T1 Rt	-	-	-	-	-	-	-	-	-	-	1	50

Table A.III.3.4 **Paraesthesia - Frequency for dermatomes involved for each of the time periods (Control group) Frq=Frequency; %= percentage of subjects; IE = initial examination.**

Derma-tomes	Frq (IE) n=24	% (IE)	Frq wk4 n=10	% wk4	Frq wk6 n=8	% wk6	Frq wk1 2 n=6	% wk1 2	Frq wk2 4 n=3	% wk2 4	Frq wk5 2 n=6	% wk5 2
Tips of fingers Lt	13	54.2	3	30	3	37.5	1	16.7	1	33.3	2	33.3
Tips of fingers Rt	11	50.0	3	30	2	25.0	2	33.3	1	33.3	1	16.7
T1- Lt	3	12.5	3	30	4	50.0	-	-	-	-	-	-
C8 -Lt	3	12.5	5	50	5	62.5	-	-	-	-	-	-
T1-Rt	1	4.2	1	10	1	12.5	2	33.3	-	-	-	-
C6 -Rt	1	4.2	2	20	1	12.5	-	-	-	-	1	16.7
C7 -Rt	1	4.2	1	10	1	12.5	1	16.7	-	-	-	-
C8 -Rt	1	4.2	2	20	2	25.0	2	33.3	-	-	-	-
C7 -Lt	1	4.2	3	30	1	12.5	2	33.3	2	66.7	2	33.3
C5 -Rt	1	4.2	1	10	-	-	-	-	-	-	-	-
S1-Rt	1	4.2	-	-	-	-	-	-	-	-	1	16.7
S1-Lt	1	4.2	1	10	-	-	-	-	-	-		
L5- Rt	1	4.2	2	20	-	-	-	-	-	-	-	-
C6 -Lt	-	-	2	20	1	12.5	2	33.3	1	33.3	2	33.3
L5- Lt	-	-	1	10	-	-	-	-	-	-	1	16.7

Table A.III.3.5 **Nausea- The frequency for the total sample as evidenced during the study period.**

Time period	Frequency	%
Initial examination	53	24.1
Week-4	22	13.6
Week-6	14	9.3
Week-12	9	6.0
Week-24	8	6.6
Week-52	3	2.6

Table A.III.3.6 **Blurred vision-The frequency for the total sample as evidenced during the study period.**

Time period	Bilateral	%	Right	%	Left	%
Initial examination	37	16.8	1	0.5	1	0.5
Week-4	23	14.2	3	1.9	2	1.2
Week-6	14	9.3	4	2.6	1	0.7
Week-12	17	11.4	1	0.7	-	-
Week-24	7	5.7	0	0.0	1	0.8
Week-52	7	6.0	2	1.7	0	0.0

Table A.III.3.7 **Double vision- The frequency for the total sample as evidenced during the study period.**

Time period	Bilteral	%	Right	%	Left	%
Initial examination	9	4.1	0	0.0	1.0	0.5
Week-4	2	1.2	1	0.6	1.0	0.6
Week-6	1	0.7	0	0.0	1.0	0.7
Week-12	2	1.3	0	0.0	0.0	0.0
Week-24	1	0.8	0	0.0	0.0	0.0
Week-52	0	0.0	0	0.0	0.0	0.0

Table A.III.3.8 **Black spots in the visusal field- The frequency for the total sample as evidenced during the study period.**

Time period	Bilteral	%	Right	%	Left	%
Initial examination	17	7.7	3	1.4	0	0.0
Week-4	16	9.9	2	1.2	2	1.2
Week-6	13	8.6	3	2.0	2	1.3
Week-12	9	6.0	1	0.7	1	0.7
Week-24	9	7.4	1	0.8	1	0.8
Week-52	6	5.2	1	0.9	0	0.0

Table A.III.3.9 **Difficulty in focusing- The frequency for the total sample as evidenced during the study period.**

Time period	Bilteral	%	Right	%	Left	%
Initial examination	38	17.3	0	0.0	2	0.9
Week-4	17	10.5	2	1.2	2	1.2
Week-6	13	8.6	1	0.7	1	0.7
Week--12	20	13.4	1	0.7	0	0.0
Week-24	9	7.4	1	0.8	0	0.0
Week-52	6	5.2	2	1.7	0	0.0

Table A.III.3.10 **Dizziness- The frequency for the total sample as evidenced during the study period.**

Time period	FREQUENCY	%
Initial examination	30	13.6
Week-4	29	17.9
Week-6	17	11.3
Week--12	10	6.7
Week-24	8	6.6
Week-52	4	3.4

Table A.III.3.11 **Loss of balance- The frequency for the total sample as evidenced during the study period.**

Time period	BILATERAL	%	RIGHT	%	LEFT	%
Initial examination	15	6.8	2	0.9	0	0
Week-4	14	8.6	0	0.0	0	0
Week-6	9	6.0	0	0.0	0	0
Week--12	7	4.7	1	0.7	0	0
Week-24	3	2.5	0	0.0	0	0
Week-52	1	0.9	0	0.0	0	0

Table A.III.3.12 **Tinnitus- The frequency for the total sample as evidenced during the study period.**

Time period	BILATERAL	%	RIGHT	%	LEFT	%
Initial examination	19	8.6	1	0.5	1	0.5
Week-4	17	10.5	8	4.9	5	3.1
Week-6	13	8.6	5	3.3	1	0.7
Week--12	8	5.4	4	2.7	6	4.0
Week-24	3	2.5	3	2.5	5	4.1
Week-52	8	6.9	4	3.4	1	0.9

Table A.III.3.13 **Retro-orbital pain- The frequency for the total sample as evidenced during the study period.**

Time period	BILATERAL	%	RIGHT	%	LEFT	%
Initial examination	29	13.2	8	3.6	2	0.9
Week-4	18	11.1	3	1.9	1	0.6
Week-6	14	9.3	1	0.7	2	1.3
Week--12	16	10.7	3	2.0	3	2.0
Week-24	6	4.9	2	1.6	4	3.3
Week-52	5	4.3	4	3.4	1	0.9

Table A.III.3.14 **Lack of concentration- The frequency for the total sample as evidenced during the study period.**

Time period	Frequency	%
Initial examination		
Week-4	26	16.0
Week-6	19	12.6
Week--12	22	14.8
Week-24	8	6.6
Week-52	7	6.0

Table A.III.3.15 **Clumsiness- The frequency for the total sample as evidenced during the study period.**

Time period	BILATERAL	%	RIGHT	%	LEFT	%
Initial examination	-	-	-	-	-	-
Week-4	17	10.5	7	4.3	1	0.6
Week-6	14	9.3	4	2.6	2	1.3
Week--12	14	9.4	3	2.0	2	1.3
Week-24	9	7.4	3	2.5	4	3.3
Week-52	7	6.0	3	2.6	0	0

Table A.III.4.1 **Descriptive statistics for the immobilised group with reference to time periods (Transformed data).**

Time scale-	n	Geometric mean	Geometric S.D	Arithmetic mean	Mlinimum	Maximum
Within 24 hours	71	551.70	1.66	625.99	150.0	1669.0
Initial examination	71	548.95	1.66	308.80	140.0	1652.0
4-weeks	59	134.56	3.36	161.61	1.0	672.0
6-weeks	60	45.33	8.22	151.68	1.0	863.0
12-weeks	57	19.09	9.12	87.25	1.0	633.0
24-weeks	48	8.36	10.89	60.13	1.0	361.0
52-weeks	42	3.51	7.06	24.50	1.0	180.0

Table A.III.4.2 **Geometric mean, upper and lower 95% confidence limit for the immobilised group with reference to time periods (Transformed data).**

Time scale-	n	Geometric mean	Geometric S.D	Geometric lower limit	Geometric upper limit
Within 24 hours	71	551.70	1.66	488.80	622.69
Initial examination	71	548.95	1.66	486.48	619.44
4-weeks	59	134.56	3.36	97.84	185.05
6-weeks	60	45.33	8.22	26.31	78.10
12-weeks	57	19.09	9.12	10.56	34.50
24-weeks	48	8.36	10.89	4.16	16.77
52-weeks	42	3.51	7.06	1.91	6.45

Table A.III.4.3 **Descriptive statistics for the active exercise group with reference to time periods (Transformed data).**

Time scale-	n	Geometric Mean	Geometric S.D	Arithmetic Mean	Minimum	Maximum
Within 24 hours	60	355.67	2.13	448.50	30.0	1367.0
Initial examination	60	324.08	2.28	422.58	40.0	1367.0
4-weeks	44	210.89	4.28	346.64	1.0	1002.0
6-weeks	37	148.12	4.54	258.64	1.0	889.0
12-weeks	42	79.76	8.31	240.52	1.0	1023.0
24-weeks	29	98.30	7.30	224.10	1.0	889.0
52-weeks	28	53.41	9.84	183.0	1.0	728.0

Table A.III.4.4 **Geometric mean, upper and lower 95% confidence limit for the active exercise group with reference to time periods (Transformed data).**

Time scale-	n	Geometric mean	Geometric S.D	Geometric lower limit	Geometric upper limit
Within 24 hours	60	355.67	2.13	292.45	432.56
Initial examination	60	324.08	2.28	258.08	406.97
4-weeks	44	210.89	4.28	135.41	328.22
6-weeks	37	148.12	4.54	89.16	246.06
12-weeks	42	79.76	8.31	40.55	156.87
24-weeks	29	98.30	7.30	46.21	209.12
52-weeks	28	53.41	9.84	22.00	129.66

Table A.III.4.5 **Descriptive statistics of the control group with reference to time periods (Transformed data).**

Time scale	n	Geometric mean	Geometric S.D	Arithmetic mean	Minimum	Maximum
Within 24 hours	89	427.09	1.99	525.98	60.0	1669.0
Initial examination	89	412.82	2.17	525.73	30.0	1635.0
4-weeks	59	317.03	3.12	449.93	1.0	1326.0
6-weeks	54	261.65	3.01	374.93	1.0	1096.0
12-weeks	50	169.02	5.38	300.58	1.0	796.0
24-weeks	45	100.48	7.78	261.53	1.0	1187.0
52-weeks	45	101.49	9.10	255.17	1.0	602.0

Table A.III.4.6 **Geometric mean, upper and lower 95% confidence limit for the control group with reference to time periods (Transformed data).**

Time scale	n	Geometric mean	Geometric S.D	Geometric lower Limit	Geometric upper Limit
Within 24 hours	89	427.09	1.99	368.82	494.58
Initial examination	89	412.82	2.17	350.42	486.32
4-weeks	59	317.03	3.12	234.94	427.81
6-weeks	54	261.65	3.01	193.29	354.18
12-weeks	50	169.02	5.38	104.51	273.34
24-weeks	45	100.48	7.78	54.15	186.46
52-weeks	45	101.49	9.10	52.56	195.96

Table A.III.4.7 **Geometric mean and geometric standard deviation for male and female subjects within treatment groups by time periods (IE= initial examination).**

Time period	Group	Gender	Geometric Mean	Geometric Std Dev	n
IE	Immobilised group		6.31	0.51	71
		Female	6.34	0.51	46
		Male	6.1	0.51	25
	Active exercise group		5.8	0.82	60
		Female	5.9	0.79	40
		Male	5.6	0.89	20
	Control group		6.02	0.77	89
		Female	6.21	0.67	59
		Male	5.67	0.83	30
Week-4	Immobilised group		4.9	1.2	59
		Female	4.9	1.1	36
		Male	4.9	1.4	23
	Active exercise group		5.4	1.5	44
		Female	5.53	1.4	29
		Male	4.99	1.62	15
	Control group		5.8	1.14	59
		Female	6.1	0.68	39
		Male	5.1	1.5	20
Week-6	Immobilised group		3.8	2.12	60
		Female	4.03	2.0	36
		Male	3.48	2.3	24
	Active exercise group		4.99	1.5	37
		Female	5.14	1.4	24
		Male	4.74	1.74	13
	Control group		5.57	1.1	54
		Female	5.8	0.83	35
		Male	5.14	1.41	19
Week-12	Immobilised group		2.94	2.2	57
		Female	3.24	2.12	36
		Male	2.5	2.3	21
	Active exercise group		4.4	2.17	42
		Female	4.74	1.9	29
		Male	3.6	2.6	13
	Control group		5.13	1.7	50
		Female	5.54	1.2	36
		Male	4.06	2.25	14
Week-24	Immobilised group		2.12	2.4	48
		Female	2.6	2.5	29
		Male	1.3	2.07	19
	Active exercise group		4.58	1.98	29
		Female	4.59	2.04	21
		Male	4.56	1.94	8
	Control group		4.6	2.05	45
		Female	4.84	2.07	32
		Male	4.04	1.94	13
Week-52	Immobilised group		1.26	1.95	42
		Female	1.4	2.06	26
		Male	0.96	1.8	16
	Active exercise group		3.98	2.29	28
		Female	3.8	2.4	20
		Male	4.5	2.1	8
	Control group		4.61	2.2	45
		Female	4.67	2.3	34
		Male	4.9	1.7	11

Table A.III.4.8 **The frequency distribution of the areas of pain for the immobilised group. (IE= Initial examination).**

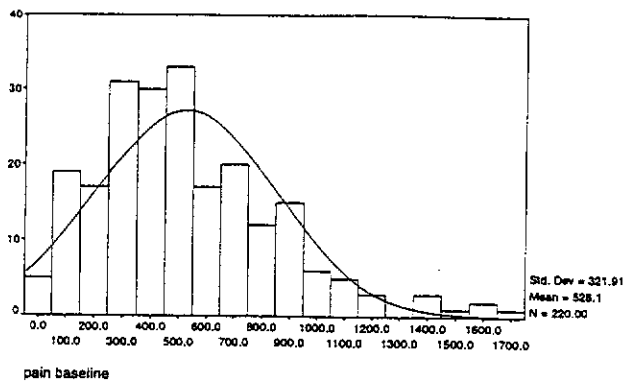
Area of pain	% IE	% Week-4	% Week-6	% Week-12	% Week-24	% Week-52
	n=71	n=59	n=60	n=57	n=48	n=42
Headache-(Rt-Forehead)	57.7	31.6	27.8	17.0	25.0	18.2
Headache-(Lt-Forehead)	60.6	36.8	25.9	19.1	18.8	18.2
Headache-(Rt-Occipital)	28.2	22.8	18.5	8.5	12.5	4.5
Headache-(Lt-Occipital)	29.6	28.1	18.5	10.6	15.6	4.5
Back Of The Neck -Left	87.3	61.4	38.9	40.4	21.9	4.5
Back Of The Neck-Right	87.3	63.2	42.6	29.8	18.8	4.5
Trapezius-Rt-Middle Fibres	77.5	50.9	37.0	10.6	9.4	4.5
Trapezius-Lt-Middle Fibres	70.4	47.4	33.3	17.0	15.6	9.1
Trapezius-Rt-Upper Fibres	56.3	35.1	20.4	19.1	9.4	4.5
Trapezius-Lt-Upper Fibres	56.3	42.1	29.6	25.5	12.5	4.5
Pre-Vertebral Region	32.4	0.0	0.0	0.0	0.0	0.0
Retro-Orbital-Rt	18.3	7.0	5.6	6.4	6.3	4.5
Retro-Orbital -Lt	16.9	3.5	5.6	6.4	9.4	0.0
Sternomastoid-Rt Upper	23.9	5.3	1.9	0.0	0.0	0.0
Sternomastoid-Rt Middle	21.1	1.8	1.9	0.0	0.0	0.0
Sternomastoid-Rt Lower	19.7	3.5	1.9	0.0	2.0	0.0
Sternomastoid-Lt Upper	25.4	7.0	5.6	0.0	1.0	0.0
Sternomastoid-Lt Middle	21.5	3.5	5.6	0.0	1.0	0.0
Sternomastoid-Lt Lower	21.5	3.5	5.6	0.0	1.0	0.0
Interscapular-Rt	28.2	15.8	16.7	17.0	0.0	0.0.
Interscapular-Lt	28.2	15.8	18.5	19.1	0.0	0.0
Nuchal Attachment-Rt	19.7	5.3	1.9	2.1	0.0	0.0
Nuchal Attachment-Lt	21.1	7.0	1.9	2.1	0.0	0.0
Tm.J Joint-Rt	7.0	0.0	0.0	0.0	2.0	0.0
T.M.J.-Joint-Lt	8.5	0.0	0.0	0.0	0	0.0
C7-T1 Junction	14.1	12.3	11.1	10.6	3.1	9.1

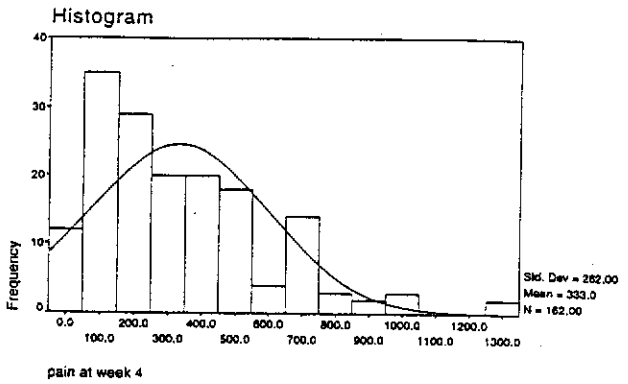
Table A.III.4.9 **The frequency distribution of the areas of pain for the active exercise group (IE= initial examination).**

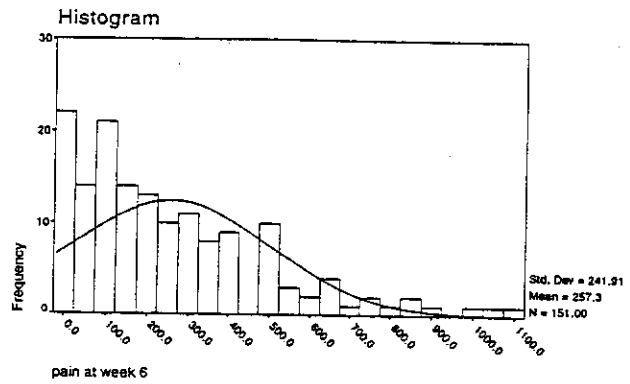
Area of pain	% IE n=60	% Week-4 n=44	% Week-6 n=37	% Week-12 n=42	% Week-24 n=29	% Week-52 n=28
Headache-(Rt-Forehead)	58.3	54.8	45.7	27.8	29.6	39.1
Headache-(Lt-Forehead)	58.3	57.1	45.7	36.1	37.0	34.8
Headache-(Rt-Occipital)	26.7	26.2	8.6	30.6	25.9	17.4
Headache-(Lt-Occipital)	28.3	31.0	14.3	36.1	25.9	13.0
Back Of The Neck -Left	78.3	81.0	65.7	50.0	48.1	39.1
Back Of The Neck-Right	80.0	69.0	60.0	47.2	55.6	43.5
Trapezius-Rt-Middle Fibres	61.7	52.4	48.6	50.0	44.4	30.4
Trapezius-Lt-Middle Fibres	58.3	57.1	48.6	52.8	33.3	30.4
Trapezius-Rt-Upper Fibres	45.0	47.6	48.6	25.0	44.4	43.5
Trapezius-Lt-Upper Fibres	46.7	54.8	54.3	36.1	33.8	30.4
Pre-Vertebral Region	19.3	0.0	0.0	0.0	0.0	0.0
Retro-Orbital-Rt	20.0	14.3	8.6	11.1	0.0	4.3
Retro-Orbital -Lt	21.7	11.9	8.6	13.9	0.0	0.0
Sternomastoid-Rt Upper	13.3	11.9	8.6	5.6	3.7	4.3
Sternomastoid-Rt Middle	11.7	7.1	5.7	5.6	0.0	4.3
Sternomastoid-Rt Lower	11.7	7.1	5.7	5.6	0.0	4.3
Sternomastoid-Lt Upper	11.7	0.0	2.9	2.8	0.0	0.0
Sternomastoid-Lt Middle	11.7	2.4	2.9	2.8	0.0	0.0
Sternomastoid-Lt Lower	10.0	4.8	2.9	2.8	0.0	0.0
Interscapular-Rt	36.7	28.6	11.4	11.1	0.0	0.0
Interscapular-Lt	26.7	19.0	8.6	11.1	0.0	0.0
Nuchal Attachment-Rt	21.7	9.5	5.7	5.6	3.7	0.0
Nuchal Attachment-Lt	20.0	4.8	5.7	8.3	0.0	0.0
Tm.J Joint-Rt	5.0	2.4	2.9	5.6	3.7	4.3
T.M.J.-Joint-Lt	3.3	0.6	2.9	2.8	0.0	0.0
C7-T1 Junction	20.0	17.1	20.0	16.7	11.1	17.4

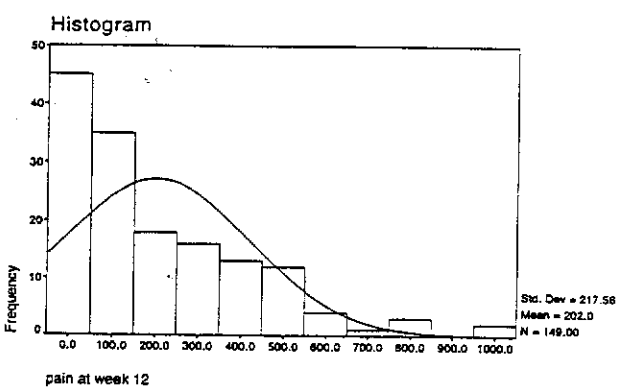
Table A.III.4.10 The frequency distribution of the areas of pain for the control group (IE= initial examination).

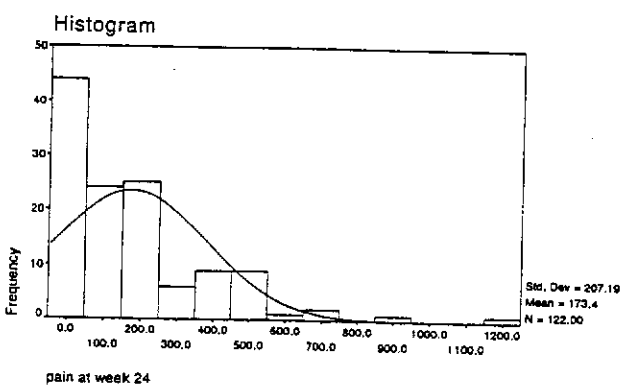
Area of pain	% IE n= 89	% Week-4 n=59	% Week-6 n=54	% Week-12 n=45	% Week-24 n=45	% Week-52 n=45
Headache-(Rt-Forehead)	64.0	61.0	50.9	51.1	42.9	44.7
Headache-(Lt-Forehead)	57.3	61.0	52.8	48.9	52.4	44.7
Headache-(Rt-Occipital)	32.6	39.0	32.1	19.1	11.9	44.7
Headache-(Lt-Occipital)	30.3	39.0	30.2	23.4	23.8	44.7
Back Of The Neck -Left	82.0	69.5	52.8	63.8	52.4	60.5
Back Of The Neck-Right	82.0	78.0	67.9	70.2	54.8	68.4
Trapezius-Rt-Middle Fibres	70.8	66.1	50.9	42.6	42.9	42.1
Trapezius-Lt-Middle Fibres	62.9	62.7	56.6	42.6	42.9	47.4
Trapezius-Rt-Upper Fibres	49.4	39.0	47.2	40.4	31.0	36.8
Trapezius-Lt-Upper Fibres	48.3	40.7	47.2	38.3	28.6	31.6
Pre-Vertebral Region	23.1	3.4	3.8	0.0	0.0	0.0
Retro-Orbital-Rt	16.9	18.6	11.3	17.0	9.5	13.2
Retro-Orbital -Lt	13.5	16.9	11.3	14.9	14.3	10.5
Sternomastoid-Rt Upper	20.2	10.2	5.7	8.5	7.1	2.6
Sternomastoid-Rt Middle	16.9	10.2	5.7	6.4	4.8	0.0
Sternomastoid-Rt Lower	16.9	11.9	3.8	6.4	4.8	0.0
Sternomastoid-Lt Upper	19.1	10.2	3.8	4.3	2.4	0.0
Sternomastoid-Lt Middle	14.6	11.9	7.5	4.3	2.4	0.0
Sternomastoid-Lt Lower	14.6	11.9	5.7	4.3	2.4	0.0
Interscapular-Rt	29.2	33.9	17.0	21.3	11.9	7.9
Interscapular-Lt	29.2	35.9	17.0	19.1	11.9	5.3
Nuchal Attachment-Rt	13.5	11.9	5.7	6.4	2.4	5.3
Nuchal Attachment-Lt	9.0	5.1	5.7	6.4	4.8	5.3
Tm.J Joint-Rt	2.2	1.7	1.9	6.4	2.4	2.6
T.M.J.-Joint-Lt	3.4	1.7	1.9	6.4	0.0	2.6
C7-T1 Junction	14.6	15.3	26.4	19.1	9.5	21.1











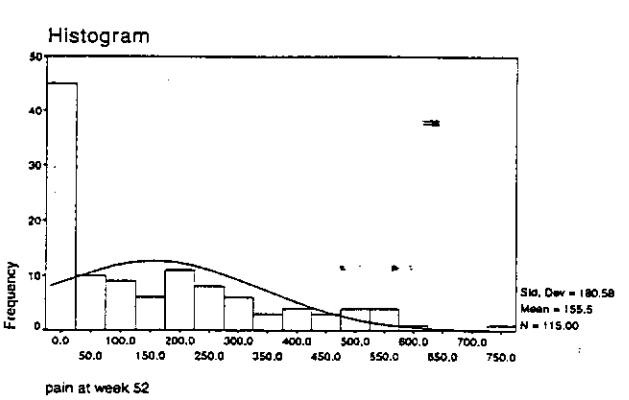
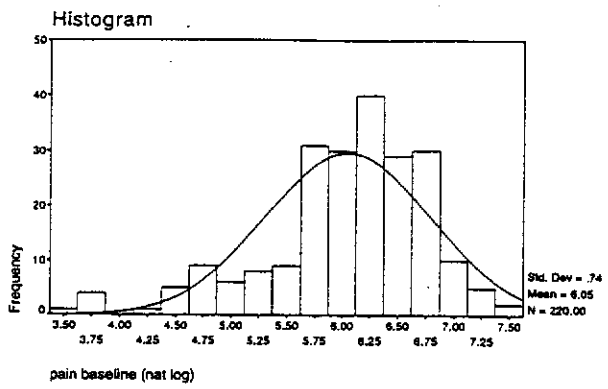
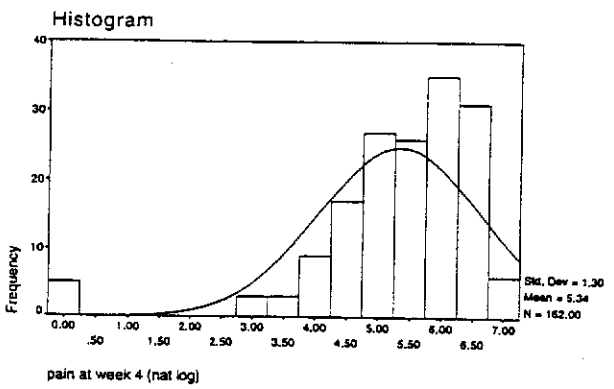
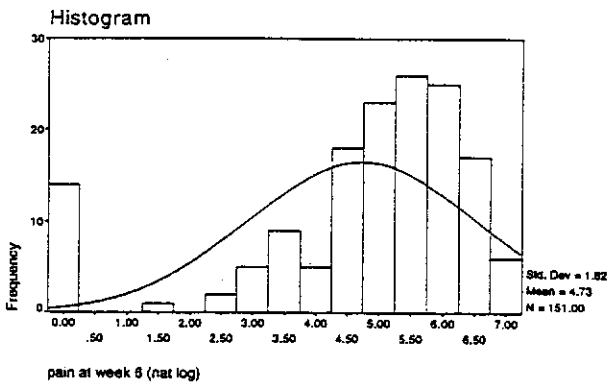
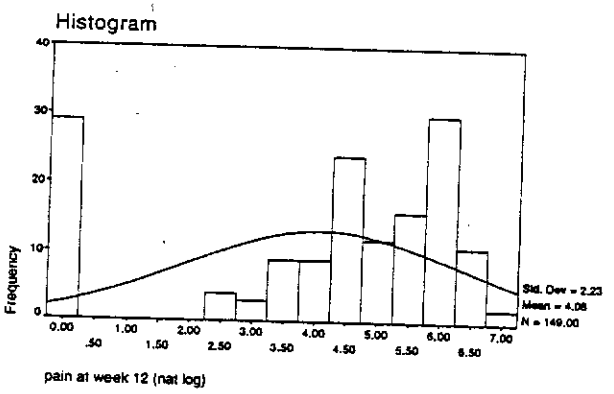


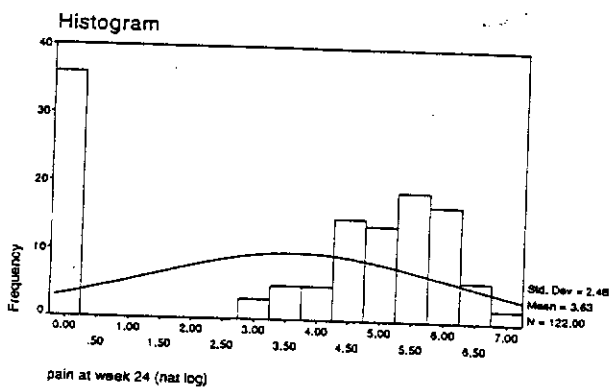
Figure A.III.1 **Histograms of the raw data to show the skewed distribution.**











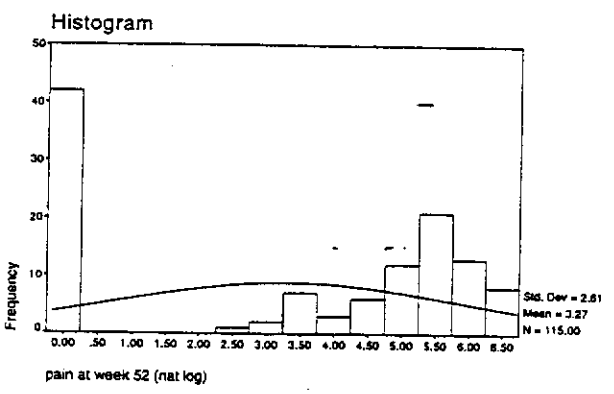


Figure A.III.2 **Histograms of the log transfomed data to show normalised distribution.**

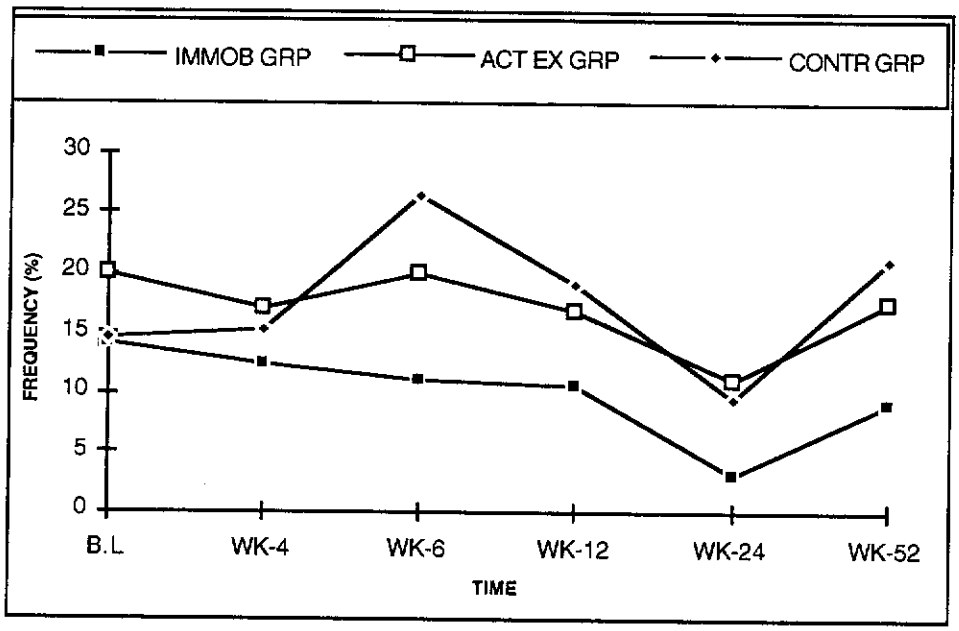


Figure A.III.3 **CERVICO-THORACIC JUNCTION-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

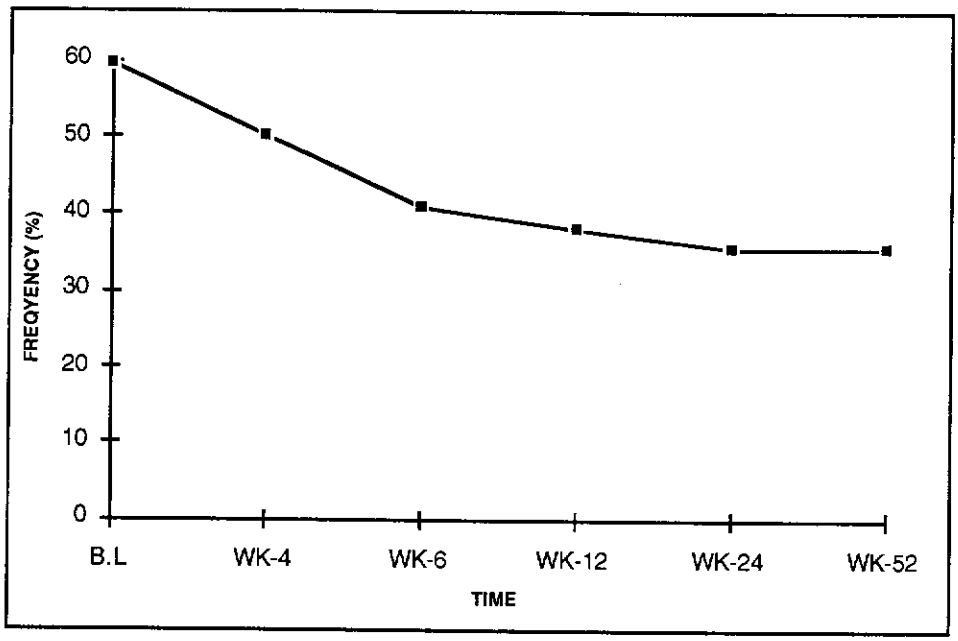


Figure A.III.4 **HEADACHE(Forehead)- (total sample)- Line graphs to illustrate the recovery pattern as evidenced during the follow-up examinations. (B.L = initial examination).**

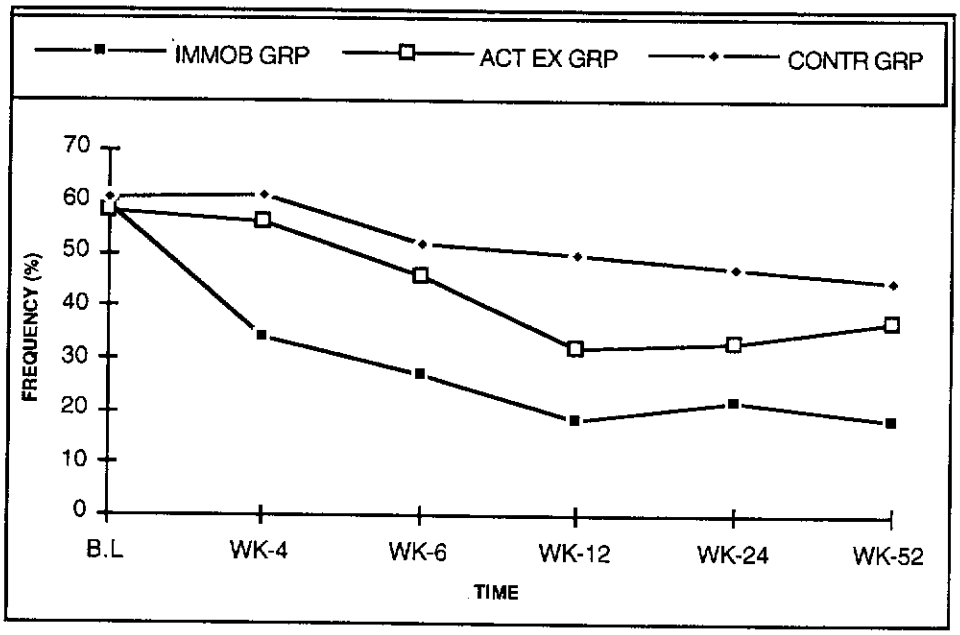


Figure A.III.5 **HEADACHE (Forehead)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

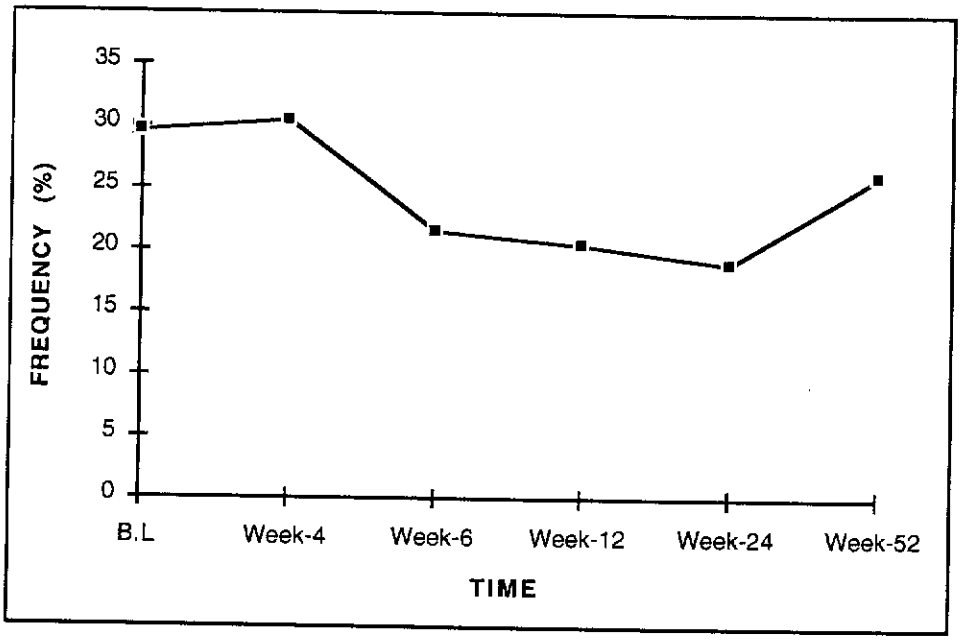


Figure A.III.6 **OCCIPITAL HEADACHE (total sample) -Line graphs to illustrate the variations in the recovery pattern as evidenced during the follow-up examinations. (B.L = Intial examination).**

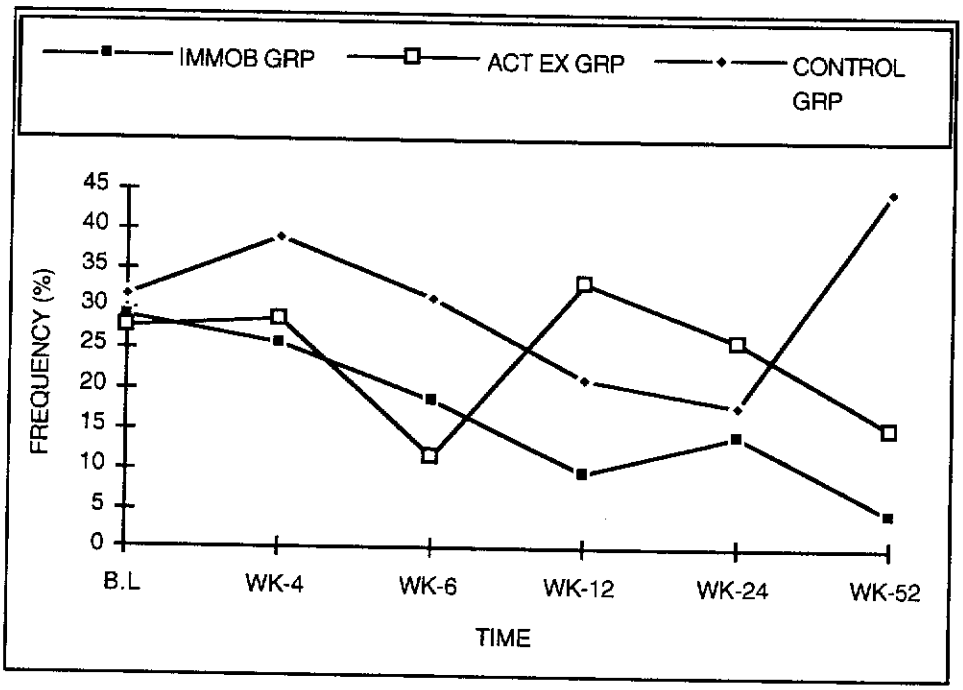


Figure A.III.7 **OCCIPITAL HEADACHE -Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

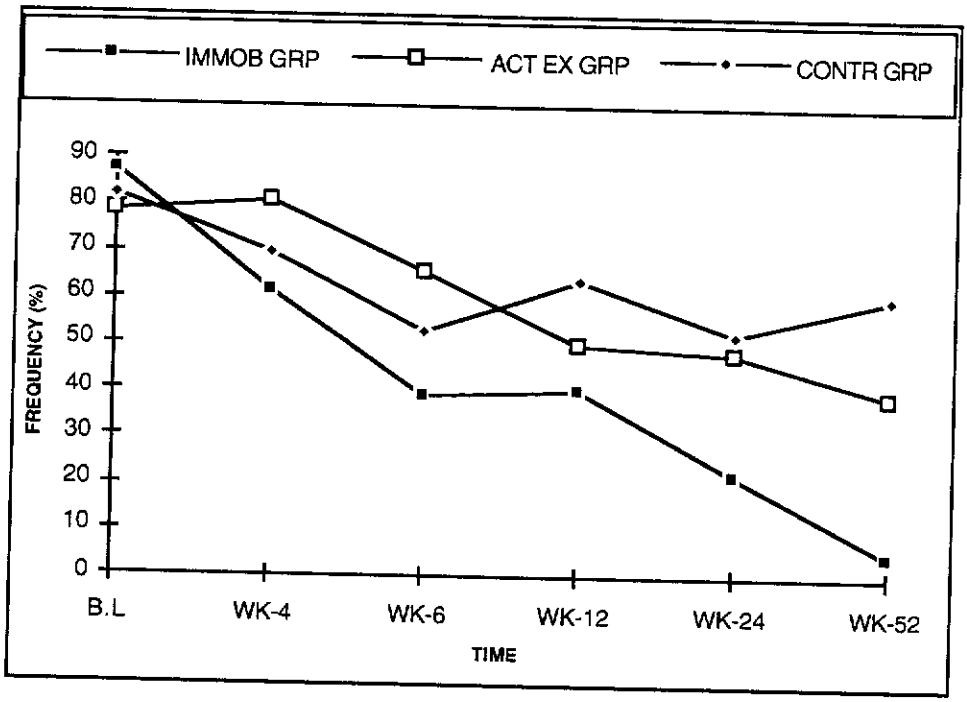


Figure A.III.8 **POSTERIOR ASPECT OF THE NECK (left- area-10)- Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

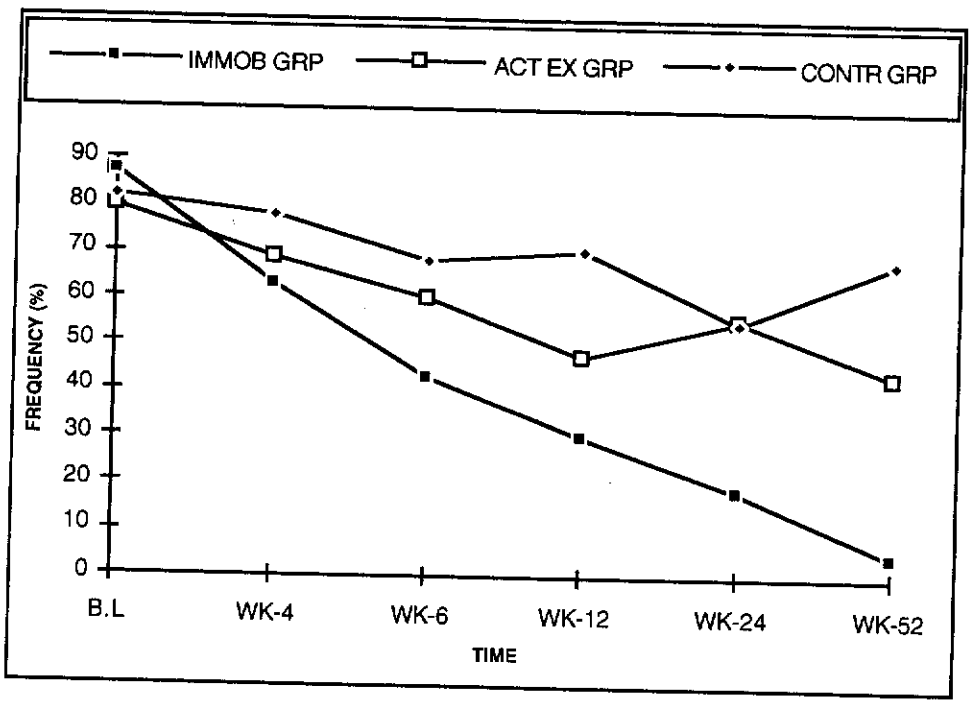


Figure A.III.9 **POSTERIOR ASPECT OF THE NECK (right-area-11)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

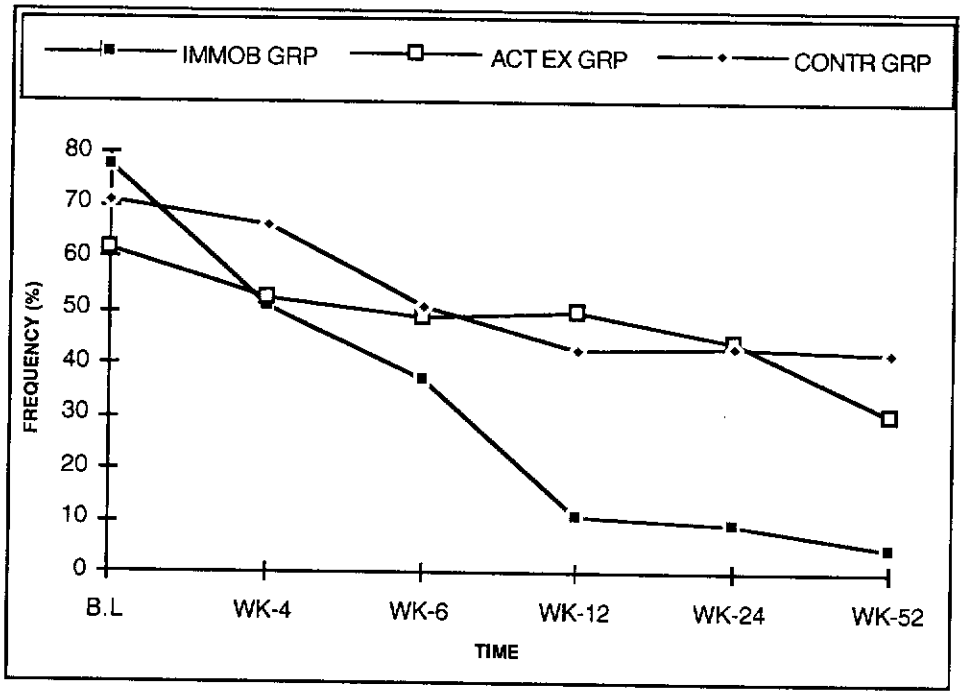


Figure A.III.10 **THE MIDDLE FIBRES OF TRAPEZIUS (right)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

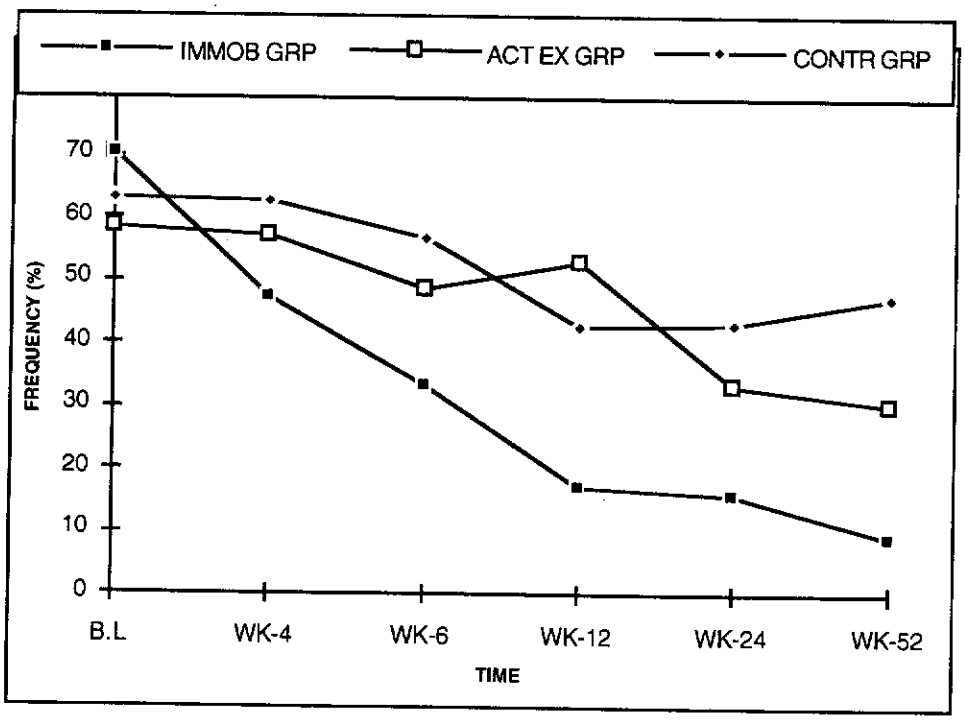


Figure A.III.11 **THE MIDDLE FIBRES OF TRAPEZIUS (left)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

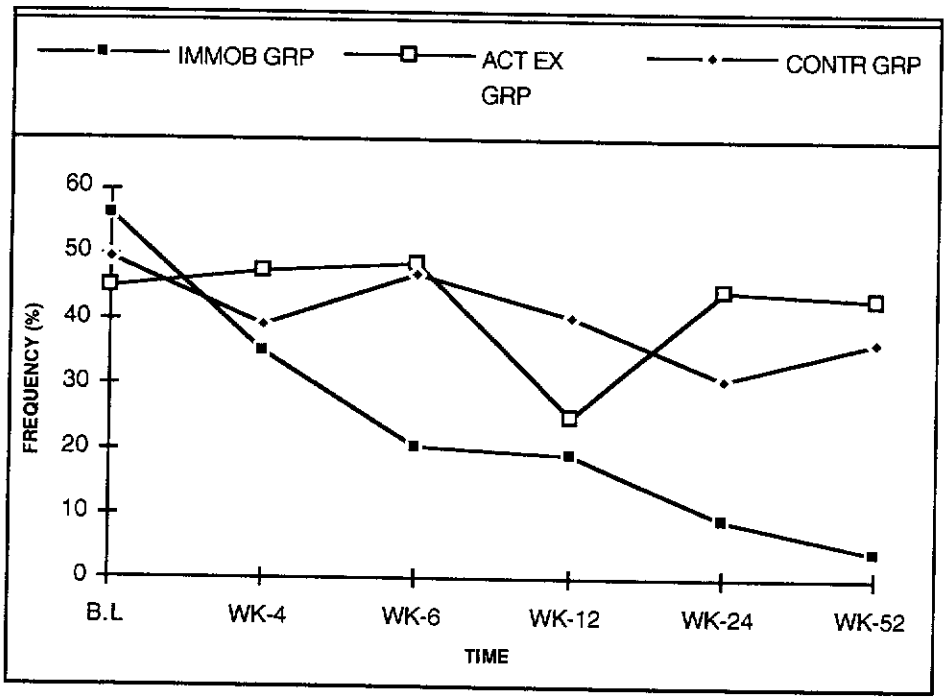


Figure A.III.12 **THE UPPER FIBRES OF TRAPEZIUS (right)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

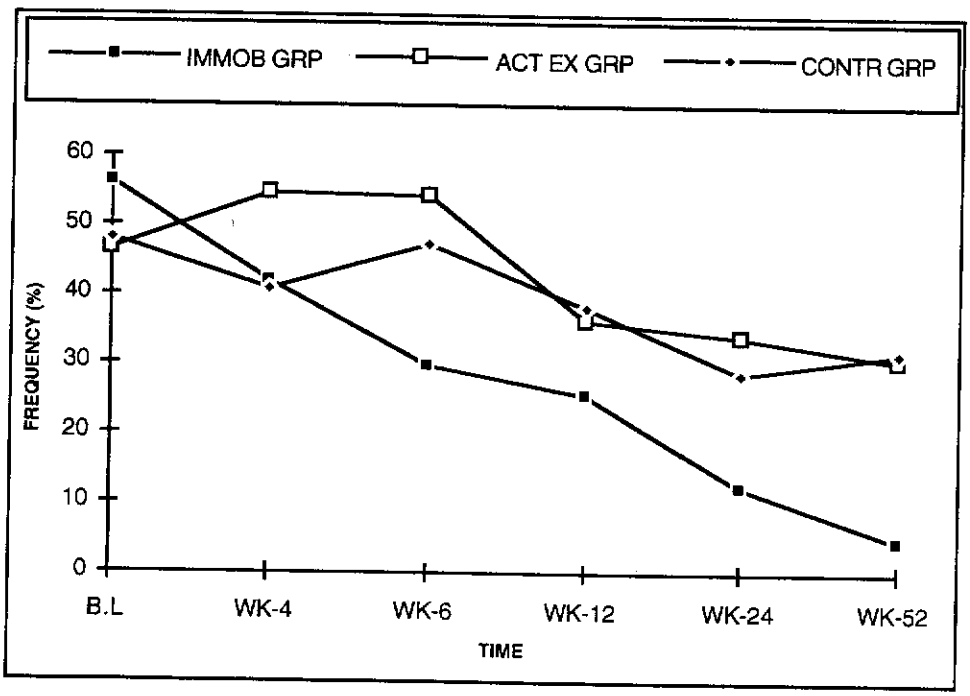


Figure A.III.13 **THE UPPER FIBRES OF TRAPEZIUS (left)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

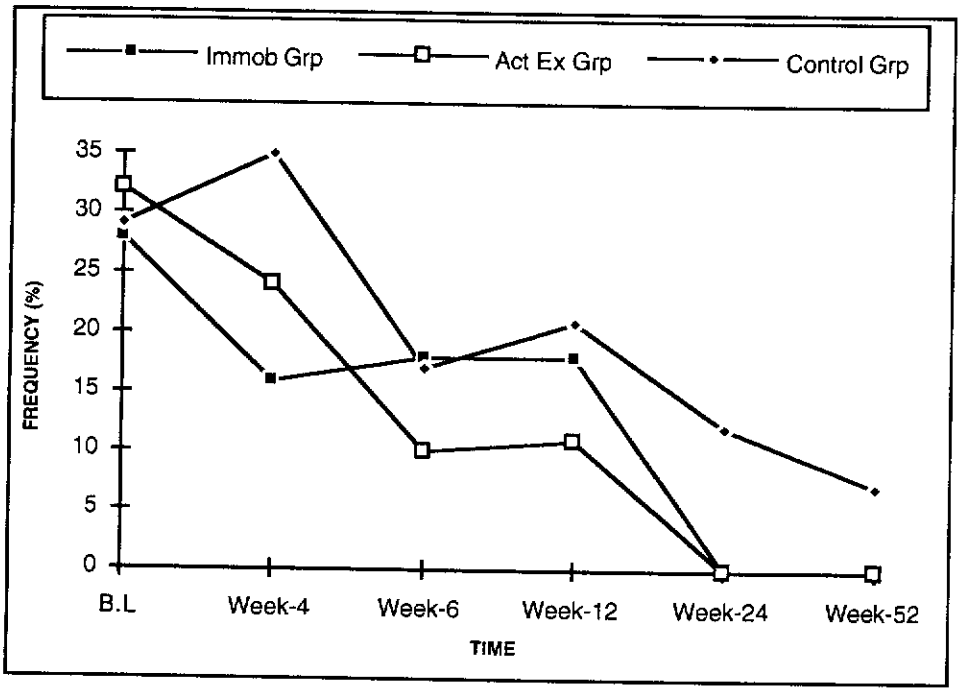


Figure A.III.14 **PAIN IN THE INTERSCAPULAR REGION-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

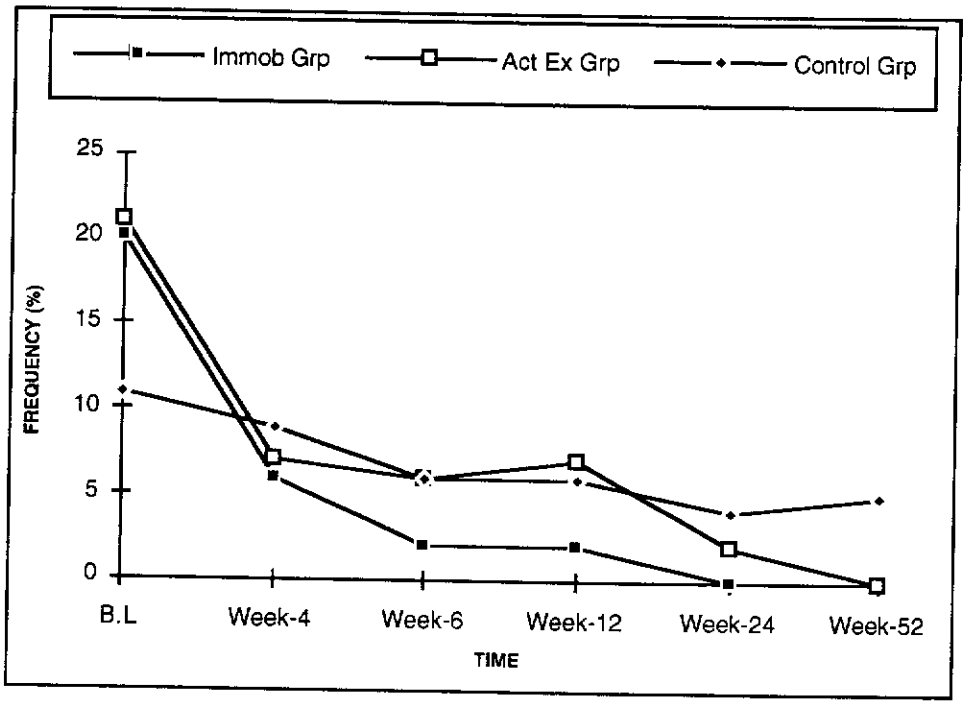


Figure A.III.15 **PAIN ALONG THE NUCHAL LINE-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

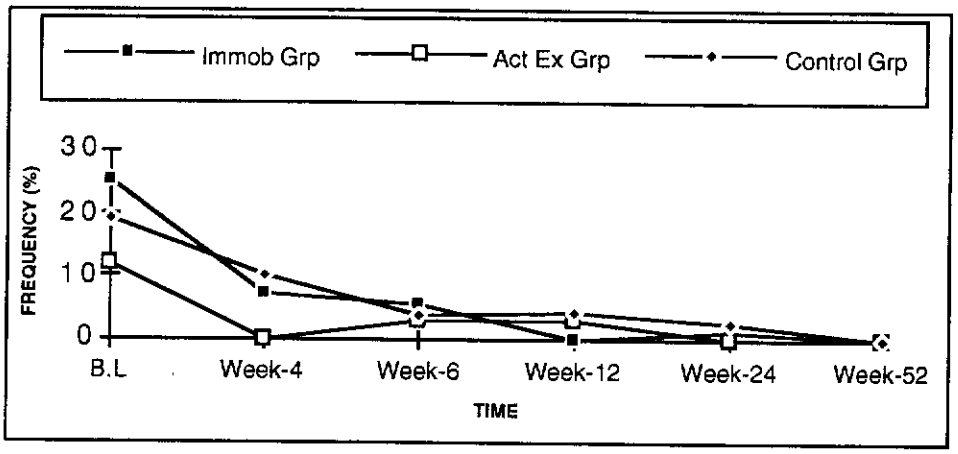


Figure A.III.16 **UPPER PART OF STERNOMASTOID** (left)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).

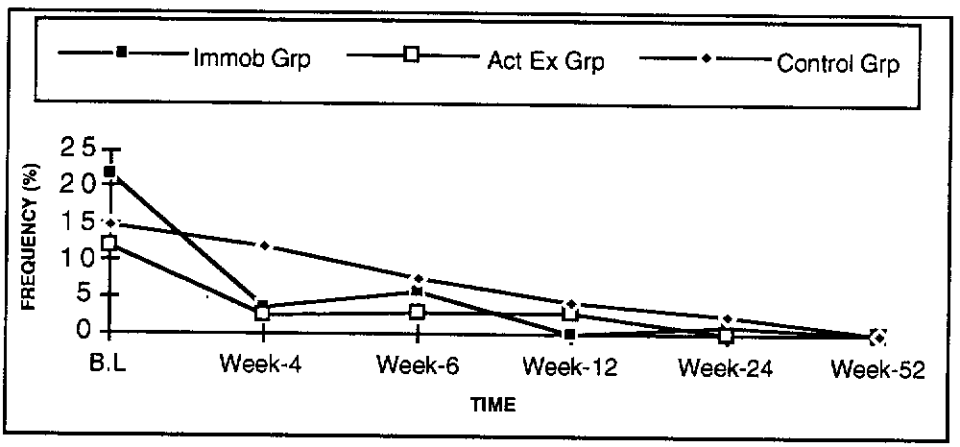


Figure A.III.17 **MIDDLE PART OF STERNOMASTOID** (left)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).

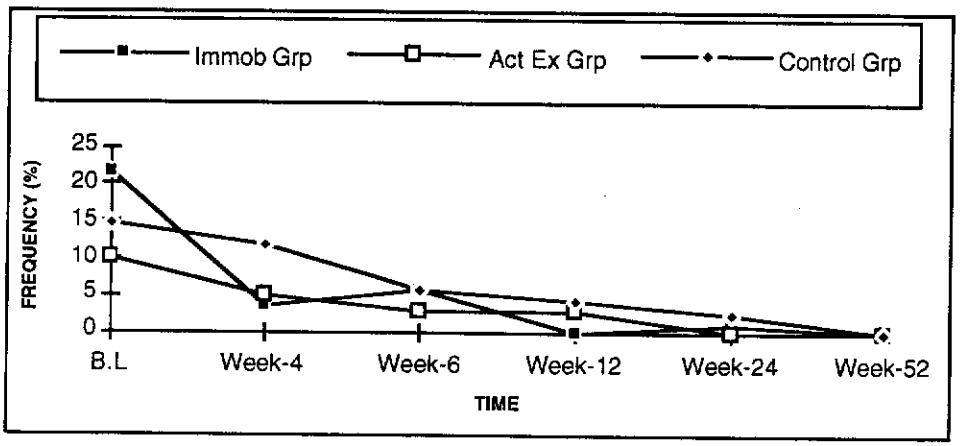


Figure A.III.18 **LOWER PART OF STERNOMASTOID** (left)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).

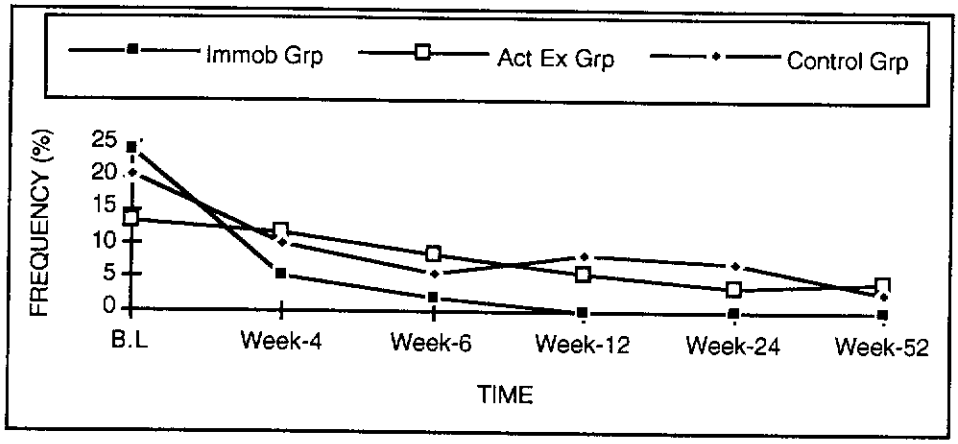


Figure A.III.19 **UPPER PART OF STERNOMASTOID (right)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

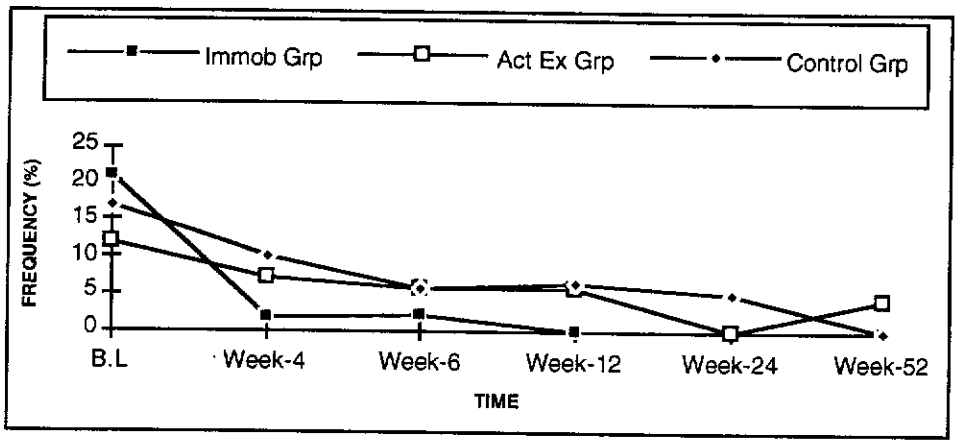


Figure A.III.20 **MIDDLE PART OF STERNOMASTOID (right)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

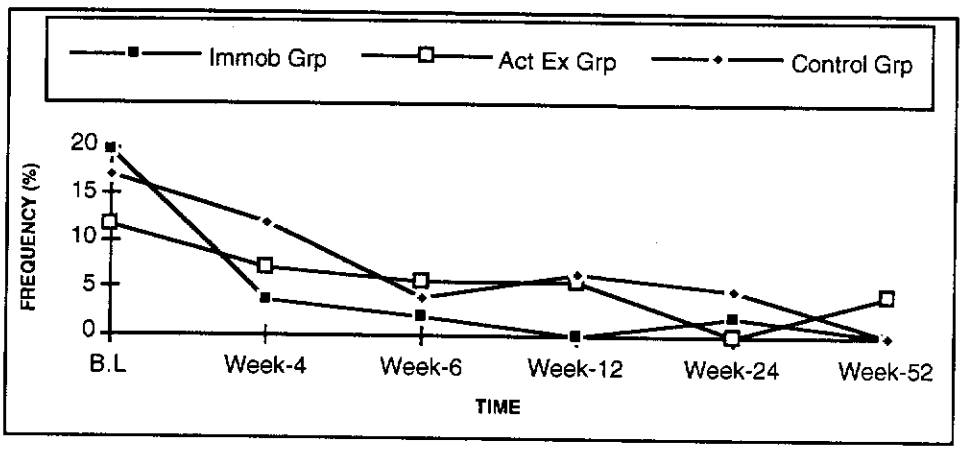


Figure A.III.21 **LOWER PART OF STERNOMASTOID (right)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

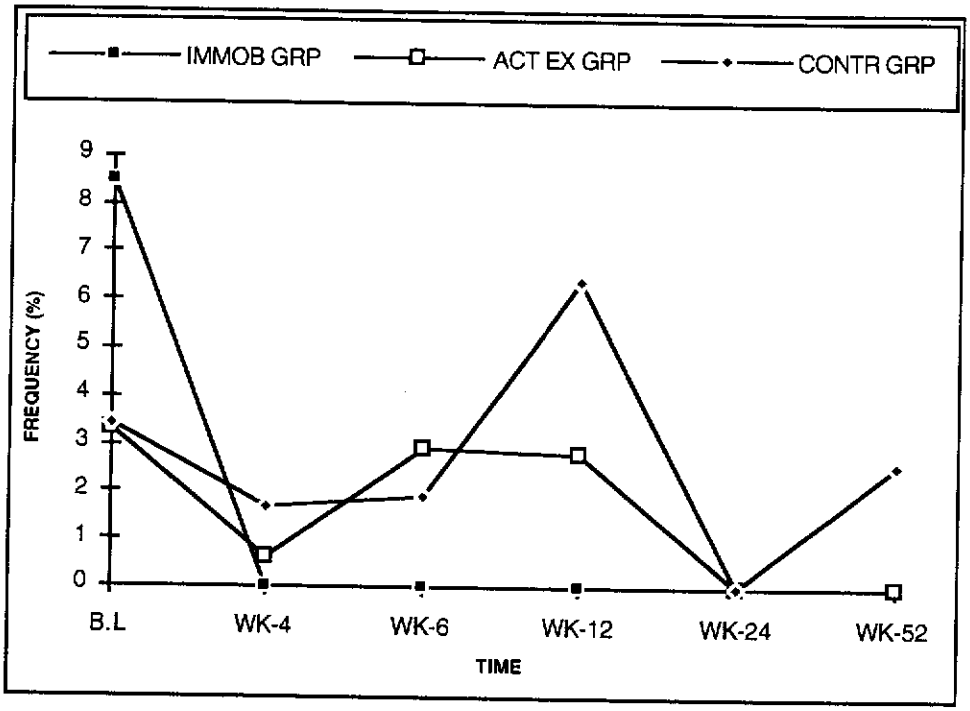


Figure A.III.22 **PAIN IN THE TM JOINT (left)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**

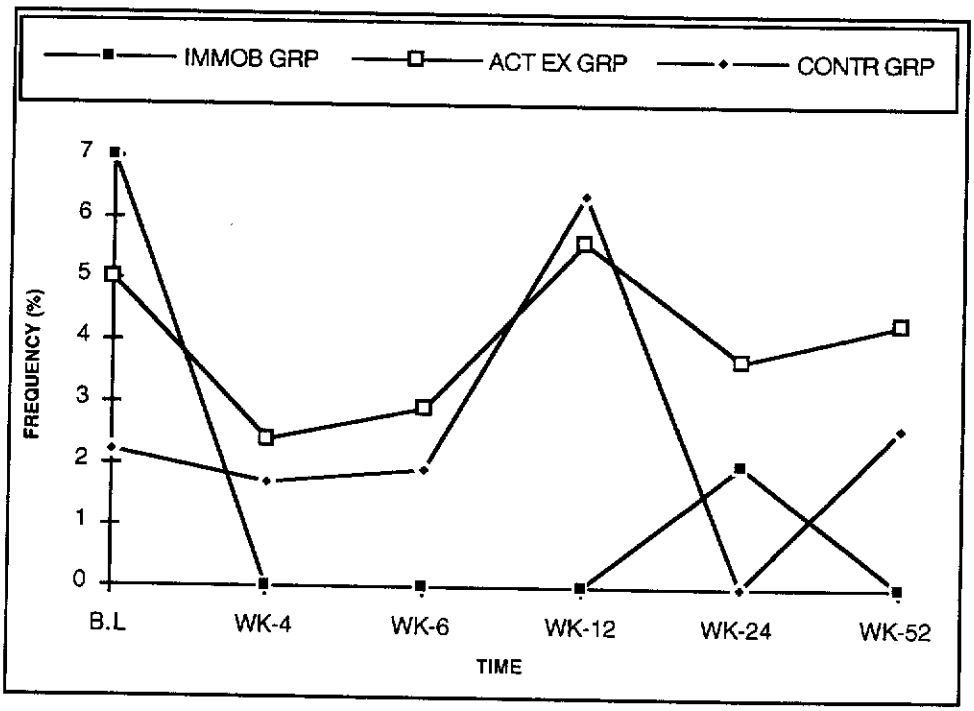
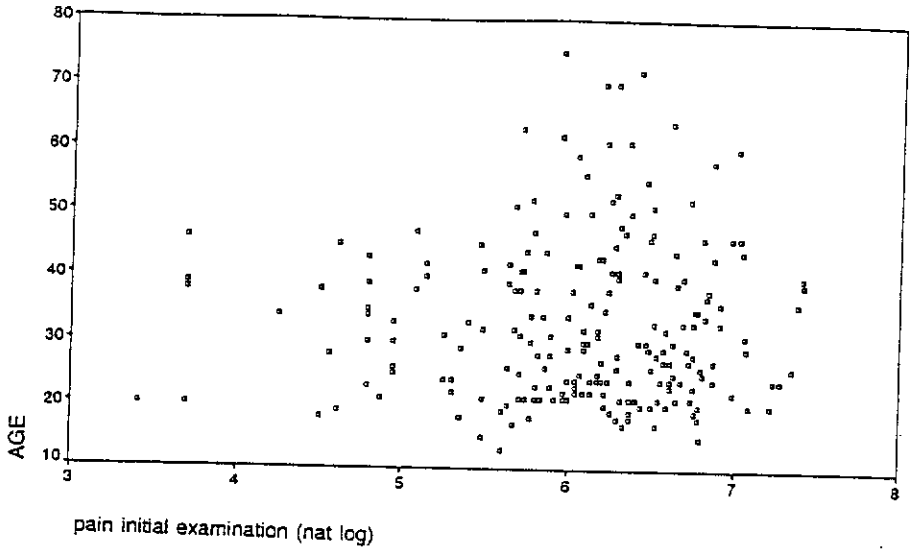


Figure A.III.23 **PAIN IN THE TM JOINT (right)-Line graphs to illustrate the variations in the recovery pattern between the treatment groups as evidenced during the follow-up examinations. (B.L = initial examination).**



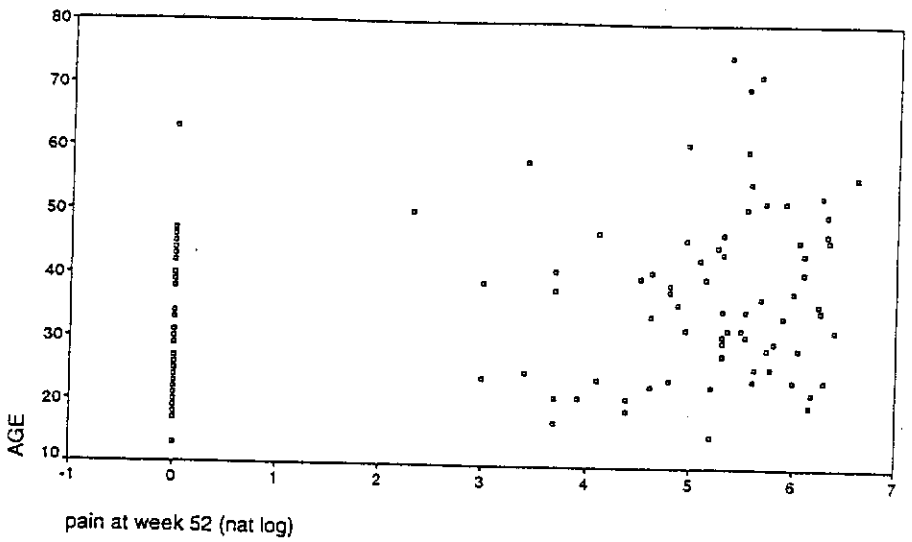
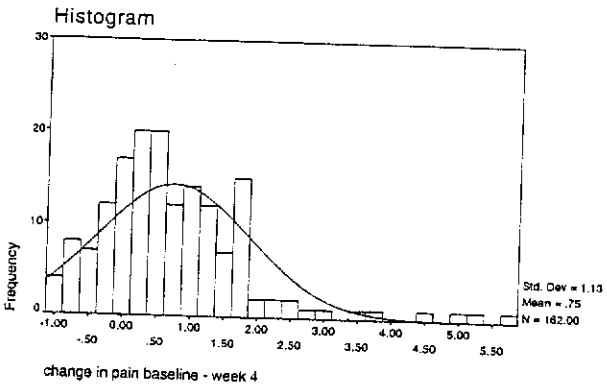
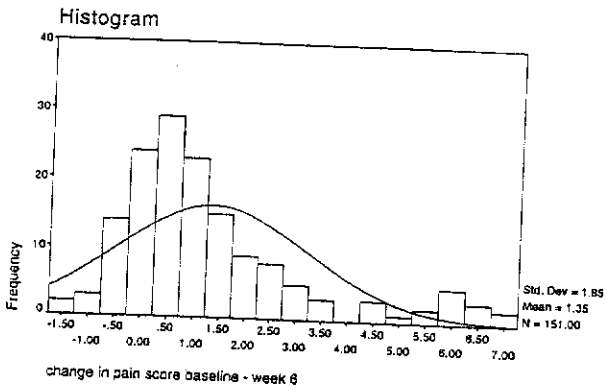
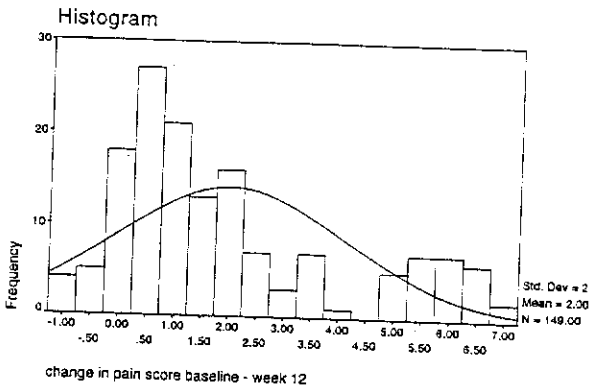
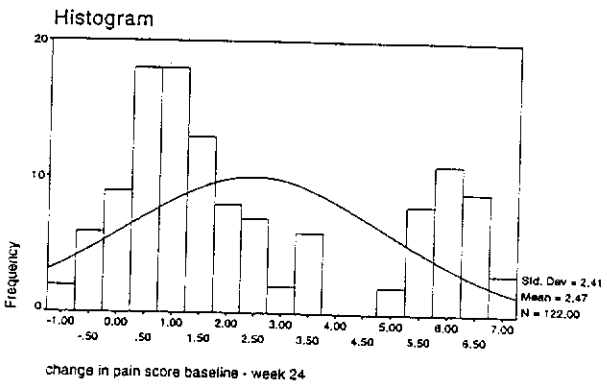


Figure A.III.24 Scatergrams to show the association between age factor and intensity of pain.









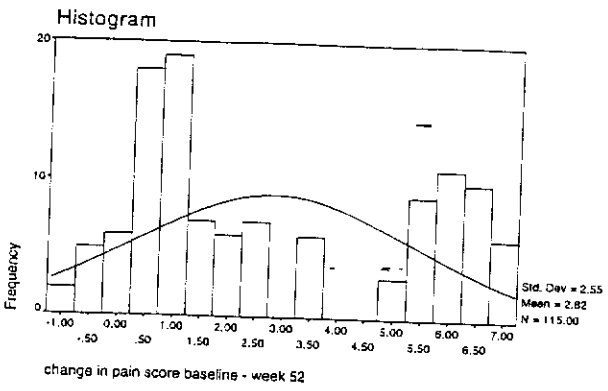


Figure A.III.25 **Histograms to show the bi-model distribution of changed scores.**

Table A.III.5.1 Summary of ANCOVA results for the effect of age.

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment gps	9.99	2	4.99	9.85	.000*
	Age	0.20	1	0.20	0.38	.536
	Residual	109.54	216	0.55		
4 weeks	Treatment gps	12.06	2	6.03	3.90	.022*
	Age	16.31	1	16.31	10.54	.001*
	Residual	244.42	158	1.55		
6 weeks	Treatment gps	64.34	2	32.17	11.92	.000*
	Age	38.22	1	38.22	14.16	.000*
	Residual	396.83	147	2.70		
12 weeks	Treatment gps	86.74	2	43.37	10.93	.000*
	Age	75.09	1	75.09	18.93	.000*
	Residual	575.32	145	3.97		
24 weeks	Treatment gps	134.03	2	67.01	14.16	.000*
	Age	49.89	1	49.89	10.54	.002*
	Residual	558.44	118	4.73		
52 weeks	Treatment gps	212.19	2	106.10	24.90	.000*
	Age	89.72	1	89.72	21.10	.000*
	Residual	472.80	111	4.26		

* = **Statistically significant**

Table A.III.5.2 **Summary of Two-way ANOVA results for the effect of gender.**

Time Period	Source of variation	SS	DF	MS	F	p
Initial exam-ination	Treatment	9.31	2	4.65	9.58	.000*
	Gender	4.68	1	4.68	9.63	.002*
	Treatment x Gender	1.96	2	0.98	2.02	.135
	Residual	103.98	214	0.49		
4 weeks	Treatment	20.41	2	10.20	6.77	.002*
	Gender	8.25	1	8.25	5.48	.021*
	Treatment x Gender	7.70	2	3.85	2.55	.081
	Residual	235.19	156	1.51		
6 weeks	Treatment	87.68	2	43.84	15.99	.000*
	Gender	10.87	1	10.87	3.97	.048*
	Treatment x Gender	0.34	2	0.17	0.06	.939
	Residual	397.38	145	2.74		
12 weeks	Treatment	119.42	2	59.77	15.17	.000*
	Gender	39.62	1	39.62	10.06	.002*
	Treatment x Gender	2.74	2	1.37	0.35	.706
	Residual	562.98	143	3.94		
24 weeks	Treatment	163.18	2	81.59	17.58	.000*
	Gender	18.91	1	18.91	4.07	.046*
	Treatment x Gender	6.12	2	3.06	0.66	.519
	Residual	538.49	116	4.64		
52 weeks	Treatment	276.16	2	138.08	30.77	.000*
	Gender	0.11	1	0.11	0.02	.874
	Treatment x Gender	5.98	2	2.99	0.66	.516
	Residual	489.20	109	4.49		

* = **Statisically significant**

Table A.III.5.3 **Summary of Two-way ANOVA results for the effect of different types of occupations.**

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment gps	7.34	2	3.67	9.68	.000*
	Occupation	55.79	84	0.66	1.75	.004*
	Treatment X occupation	17.64	35	0.50	1.33	.140
	Residual	37.19	98	0.38		
4 weeks	Treatment gps	11.18	2	5.59	3.06	.054
	Occupation	114.05	71	1.61	0.88	.699
	Treatment X occupation	27.57	28	0.98	0.54	.962
	Residual	109.52	60	1.82		
6 weeks	Treatment gps	37.37	2	18.69	5.46	.007*
	Occupation	178.58	72	2.48	0.72	.9
	Treatment X occupation	41.93	21	1.99	0.58	.912
	Residual	188.09	55	3.42		
12 weeks	Treatment gps	36.38	2	18.19	3.95	.025*
	Occupation	278.99	69	4.04	0.88	.698
	Treatment X occupation	72.93	22	3.32	0.72	.800
	Residual	253.42	55	4.61		
24 weeks	Treatment gps	68.85	2	34.42	6.94	.003*
	Occupation	298.56	60	4.98	1.00	.502
	Treatment X occupation	61.74	18	3.43	0.69	.799
	Residual	203.00	41	4.96		
52 weeks	Treatment gps	50.76	2	25.38	4.42	.019*
	Occupation	187.67	57	3.3	0.57	.97
	Treatment X occupation	100.90	19	5.31	0.92	.560
	Residual	206.69	36	5.74		

* = **Statistically significant**

Table A.III.5.4 Summary of Two-way ANOVA results for the effect of occupational categories.

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment gps	10.17	2	5.08	10.52	.000*
	Occupation -GP	7.09	3	2.36	4.89	.003*
	Treatment X occupation	3.04	6	0.51	1.05	.394
	Residual	100.48	208	0.48		
4 weeks	Treatment gps	13.06	2	6.53	4.35	.015
	Occupation -GP	18.68	3	6.23	4.15	.007*
	Treatment X occupation	7.22	6	1.20	0.80	.570
	Residual	225.24	150	1.50		
6 weeks	Treatment gps	71.17	2	35.58	12.99	.000*
	Occupation -GP	15.34	3	5.11	1.87	.138
	Treatment X occupation	12.43	6	2.07	0.76	.606
	Residual	380.83	139	2.74		
12 weeks	Treatment gps	97.69	2	48.84	12.07	.000*
	Occupation -GP	39.92	3	13.31	3.29	.023*
	Treatment X occupation	11.22	6	1.87	0.46	.835
	Residual	554.21	137	4.04		
24 weeks	Treatment gps	153.69	2	76.88	16.80	.000*
	Occupation -GP	17.79	3	5.93	1.30	.279
	Treatment X occupation	42.61	6	7.10	1.55	.168
	Residual	503.12	110	4.57		
52 weeks	Treatment gps	270.61	2	135.30	30.74	.000*
	Occupation -GP	16.02	3	5.34	1.20	.31
	Treatment X occupation	25.90	6	4.30	0.98	.441
	Residual	453.30	103	4.40		

* = **Statistically significant**

Table A.III.5.5 **Summary of Two way ANOVA results for the effect of types of collision.**

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment effect	8.56	2	4.28	8.91	.000*
	Collision	8.11	14	0.58	1.21	.274
	Treatment X collision	13.18	17	0.78	1.61	.064
	Residual	89.33	186	0.48		
4 weeks	Treatment effect	17.78	2	8.89	6.12	.003*
	Collision	15.25	13	1.17	0.81	.651
	Treatment X collision	44.10	14	3.16	2.17	.012*
	Residual	191.70	132	1.45		
6 weeks	Treatment effect	57.48	2	28.74	11.55	.000*
	Collision	80.72	12	6.73	2.70	.003*
	Treatment X collision	19.39	12	1.62	0.65	.796
	Residual	308.48	124	2.49		
12 weeks	Treatment effect	91.14	2	45.57	11.96	.000*
	Collision	111.47	14	7.96	2.09	.017*
	Treatment X collision	48.20	15	3.21	0.84	.628
	Residual	445.67	117	3.81		
24 weeks	Treatment effect	133.40	2	66.70	14.37	.000*
	Collision	86.84	13	6.68	1.44	.157
	Treatment X collision	44.94	13	3.46	0.75	.715
	Residual	431.75	93	4.64		
52 weeks	Treatment effect	242.60	2	121.30	27.50	.000*
	Collision	67.90	13	5.20	1.10	0.307
	Treatment X collision	47.55	13	3.37	0.83	.629
	Residual	379.86	86	4.42		

* = **Statistically significant**

Table A.III.5.6 Summary Two way ANOVA results for the effect of collision (grouped).

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment gps	9.50	2	4.75	9.40	.000*
	Collision gp	3.39	4	0.09	1.68	.156
	Treatment X Collision gp	2.64	6	0.44	0.87	.517
	Residual	104.59	207	0.51		
4 weeks	Treatment gps	17.32	2	8.66	5.29	.006*
	Collision gp	0.76	4	0.19	0.12	.977
	Treatment X Collision gp	3.16	4	0.79	0.48	.748
	Residual	247.21	151	1.64		
6 weeks	Treatment gps	73.00	2	36.50	13.34	.000*
	Collision gp	18.78	4	4.69	1.72	.150
	Treatment X Collision gp	9.41	5	1.88	0.69	.634
	Residual	380.41	139	2.74		
12 weeks	Treatment gps	102.94	2	51.47	12.57	.000*
	Collision gp	40.80	4	10.20	2.49	.046*
	Treatment X Collision gp	3.62	5	0.72	0.18	.971
	Residual	560.92	137	4.09		
24 weeks	Treatment gps	148.08	2	74.04	15.52	.000*
	Collision gp	21.79	4	5.45	1.14	.341
	Treatment X Collision gp	12.07	4	3.02	0.63	.641
	Residual	529.67	111	4.77		
52 weeks	Treatment gps	252.02	2	126.01	26.74	.000*
	Collision gp	18.29	4	4.57	0.97	.427
	Treatment X Collision gp	3.99	4	0.99	0.21	.931
	Residual	494.79	104	4.71		

*** = Statistically significant**

Table A.III.5.7 **Summary of Two way ANOVA results for the effect of seating positions.**

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment groups	8.92	2	4.46	8.66	.000*
	Seating position	1.83	4	0.46	0.89	.472
	Treatment X seating	2.66	7	0.38	0.74	.639
	Residual	106.12	206	0.52		
4 weeks	Treatment groups	21.51	2	10.76	6.51	.002*
	Seating position	0.29	4	0.07	0.04	.996
	Treatment X seating	3.08	5	0.62	0.37	.867
	Residual	247.77	150	1.65		
6 weeks	Treatment groups	91.32	2	45.66	17.04	.000*
	Seating position	8.48	4	2.12	0.79	.533
	Treatment X seating	27.54	5	5.51	2.06	.075
	Residual	372.58	139	2.68		
12 weeks	Treatment groups	123.87	2	61.93	15.03	.000*
	Seating position	9.22	4	2.31	0.56	.692
	Treatment X seating	31.69	5	6.34	1.54	.182
	Residual	564.43	137	4.12		
24 weeks	Treatment groups	175.44	2	87.72	19.33	.000*
	Seating position	55.07	4	13.77	3.03	.020*
	Treatment X seating	4.74	4	1.19	0.26	.902
	Residual	503.71	111	4.54		
52 weeks	Treatment groups	269.80	2	134.93	30.10	.000*
	Seating position	15.35	4	3.83	0.86	.492
	Treatment X seating	10.27	3	3.40	0.77	.516
	Residual	469.64	105	4.47		

*** = Statistically significant**

Table A.III.5.8 **Summary Two way ANOVA results for the effect of seating positions (grouped).**

Time Period	Source of variation	SS	DF	MS	F	p
Initial exam-ination	Treatment gps	8.88	2	4.44	8.70	.000*
	Seating gp	0.59	1	0.59	1.16	.283
	Treatment X Seating	0.71	2	0.36	0.70	.499
	Residual	109.32	214	0.51		
4 weeks	Treatment gps	21.65	2	10.83	6.80	.001*
	Seating gp	0.12	1	0.12	0.08	.783
	Treatment X Seating	2.50	2	1.25	0.79	.458
	Residual	248.51	156	1.59		
6 weeks	Treatment gps	91.02	2	45.51	16.42	.000*
	Seating gp	3.87	1	3.87	1.40	.239
	Treatment X Seating	2.94	2	1.47	0.53	.590
	Residual	401.78	145	2.77		
12 weeks	Treatment gps	131.11	2	65.55	15.64	.000*
	Seating gp	0.07	1	0.07	0.02	.899
	Treatment X Seating	5.89	2	2.94	0.70	.497
	Residual	599.39	143	4.19		
24 weeks	Treatment gps	186.70	2	93.35	20.72	.000*
	Seating gp	39.091	1	39.09	8.68	.004*
	Treatment X Seating	1.87	2	0.94	0.21	.813
	Residual	522.560	116	4.51		
52 weeks	Treatment gps	289.40	2	144.68	33.40	.000*
	Seating gp	11.45	1	11.45	2.60	.107
	Treatment X Seating	11.62	2	5.81	1.34	.266
	Residual	472.19	109	4.3O		

Table A.III.5.9 **Summary of ANCOVA results for the effect of speed of subject's vehicle.**

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment gps	10.30	2	5.15	10.25	.0000*
	Speed	0.98	1	0.98	1.95	.164
	Residual	108.44	216	0.50		
4 weeks	Treatment gps	20.22	2	10.11	6.37	.002*
	Speed	1.78	1	1.78	1.12	.291
	Residual	250.79	158	1.59		
6 weeks	Treatment gps	80.77	2	40.39	14.80	.000*
	Speed	17.37	1	17.37	6.36	.013*
	Residual	401.26	147	2.73		
12 weeks	Treatment gps	108.54	2	54.27	13.41	.000*
	Speed	41.60	1	41.60	10.28	.002*
	Residual	587.02	145	4.05		
24 weeks	Treatment gps	158.27	2	79.14	16.71	.000*
	Speed	25.30	1	25.30	5.34	.023*
	Residual	558.76	118	4.74		
52 weeks	Treatment gps	258.60	2	129.30	29.03	.000*
	Speed	21.58	1	21.58	4.80	.03*
	Residual	494.50	111	4.50		

* = **Statistically significant**

Table A.III.5.10 **Summary of ANCOVA results for the effect of the speed of the other vehicles / objects.**

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment gps	11.34	2	5.67	11.62	.000*
	Speed-	3.05	1	3.05	6.25	.013*
	Residual	105.33	216	0.49		
4 weeks	Treatment gps	15.49	2	7.75	4.98	.008*
	Speed	11.28	1	11.28	7.24	.008*
	Residual	246.02	158	1.56		
6 weeks	Treatment gps	64.56	2	32.28	12.15	.000*
	Speed	44.26	1	44.26	16.66	.000*
	Residual	390.58	147	2.66		
12 weeks	Treatment gps	98.84	2	49.42	12.27	.000*
	Speed	54.39	1	54.39	13.51	.000*
	Residual	583.92	145	4.03		
24 weeks	Treatment gps	141.61	2	70.81	15.04	.000*
	Speed	45.26	1	45.26	9.62	.002*
	Residual	555.46	118	4.71		
52 weeks	Treatment gps	257.30	2	128.70	28.90	.000*
	Speed	22.40	1	22.40	5.01	.027*
	Residual	494.99	111	4.46		

* = **Statistically significant**

Table A.III.5.11 **Summary of ANCOVA results for the effect of the severity of the pain (IE = initial examination).**

Time Period	Source of variation	SS	DF	MS	F	p
4 weeks	Treatment gps	41.77	2	20.88	20.05	.000*
	IE.Pain	66.42	1	66.42	63.76	.000*
	Residual	164.60	158	1.04		
6 weeks	Treatment gps	120.36	2	60.18	24.21	.000*
	IE.Pain	13.59	1	13.59	5.47	.021*
	Residual	365.45	147	2.49		
12 weeks	Treatment gps	197.44	2	98.72	31.61	.000*
	IE.Pain	86.92	1	86.92	27.84	.000*
	Residual	452.80	145	3.12		
24 weeks	Treatment gps	251.54	2	125.77	33.02	.000*
	IE.Pain	41.32	1	41.32	10.85	.001*
	Residual	449.47	118	3.81		
52 -weeks	Treatment gps	312.73	2	156.37	40.97	.000*
	IE.Pain	38.33	1	38.33	10.04	.002*
	Residual	423.67	111	3.82		

* = **Statistically significant**

Table A.III.5.12 **Summary Two way ANOVA results for the prognostic value of headache when present within 24 hours after the MVA.**

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment gps	8.57	2	4.29	8.79	.000*
	Headache	5.40	1	5.40	11.06	.001*
	Treatment X Headache	1.21	2	0.61	1.24	.291
	Residual	103.91	213	0.49		
4 weeks	Treatment gps	22.45	2	11.22	7.02	.001*
	Headache	1.32	1	1.32	0.83	.365
	Treatment X Headache	1.27	2	0.64	0.40	.672
	Residual	247.72	155	1.60		
6 weeks	Treatment gps	96.84	2	48.42	17.97	.000*
	Headache	5.94	1	5.94	2.20	.140
	Treatment X Headache	7.34	2	3.67	1.36	.260
	Residual	388.11	144	2.70		
12 weeks	Treatment gps	137.95	2	68.98	16.62	.000*
	Headache	4.78	1	4.78	1.15	.285
	Treatment X Headache	2.639	2	1.32	0.32	.728
	Residual	589.48	142	4.15		
24 weeks	Treatment gps	182.87	2	91.43	21.01	.000*
	Headache	41.09	1	41.09	9.44	.003*
	Treatment X Headache	19.48	2	9.74	2.24	.111
	Residual	500.46	115	4.35		
52 weeks	Treatment gps	271.85	2	135.95	30.27	.000*
	Headache	2.25	1	2.25	0.50	.481
	Treatment X Headache	3.60	2	1.81	0.40	.67
	Residual	489.40	109	4.49		

* = **Statistically significant**

Table A.III.5.13 Summary of Two-way ANOVA results for the effect of paraesthesia.

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment groups	7.29	2	3.60	7.50	.001*
	Paraesthesia	5.50	1	5.50	11.30	.001*
	Residual	104.28	214	0.49		
4 weeks	Treatment groups	15.98	2	7.99	5.26	.006*
	Paraesthesia	13.52	1	13.52	8.91	.003*
	Treatment x Paraesthesia	0.803	2	0.40	0.27	.768
	Residual	236.80	156	1.50		
12 weeks	Treatment groups	119.87	2	59.90	14.90	.000*
	Paraesthesia	27.03	1	27.03	6.73	.010*
	Treatment x Paraesthesia	3.70	2	1.90	0.46	.63
	Residual	574.60	143	4.02		
24 weeks	Treatment groups	158.46	2	79.20	17.30	.000*
	Paraesthesia	29.30	1	29.30	6.40	.013*
	Treatment x Paraesthesia	3.69	2	1.80	0.40	.669
	Residual	530.60	116	4.57		
52 weeks	Treatment groups	235.20	2	117.60	27.31	.000*
	Paraesthesia	19.20	1	19.20	4.46	.037*
	Treatment x Paraesthesia	2.44	1	2.44	0.57	.453
	Residual	473.61	110	4.31		

Week-6Group B contained one subject.
Computer out put:At least one variable is a constant. Therefore the list containing the constant could not be processed.
* = Statistically significant

Table A.III.5.14 Summary of 2-way ANOVA results for the effect of loss of lordosis and its interaction with treatment factor.

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment groups	7.08	2	3.50	7.10	.001*
	Lordosis	2.87	1	2.87	5.80	.017*
	Residual	105.40	213	0.50		
4 weeks	Treatment groups	21.50	2	10.75	6.70	.002*
	Lordosis	2.15	1	2.15	1.30	.248
	Treatment x Lordosis	0.70	2	0.35	0.22	.8
	Residual	247.85	155	1.59		
6 weeks	Treatment groups	92.39	2	46.19	16.45	.000*
	Lordosis	4.22	1	4.22	1.50	.222
	Treatment x Lordosis	0.12	2	0.06	0.02	.978
	Residual	404.18	144	2.81		
12 weeks	Treatment groups	143.17	2	71.59	17.40	.000*
	Lordosis	18.73	1	18.73	4.56	.035*
	Treatment x Lordosis	0.65	2	0.32	0.08	.924
	Residual	583.87	142	4.11		
24 weeks	Treatment groups	187.80	2	93.88	19.78	.000*
	Lordosis	4.40	1	4.40	0.93	.338
	Treatment x Lordosis	8.17	2	4.10	0.86	.43
	Residual	545.70	115	4.75		
52 weeks	Treatment groups	269.40	2	134.70	30.70	.000*
	Lordosis	0.14	1	0.14	0.03	.86
	Treatment x Lordosis	19.89	2	9.90	2.30	.1
	Residual	473.60	108	4.30		

*** = Statistically significant**

Table A.III.5.15 Summary of 2-way ANOVA results for the effect of disc degeneration and its interaction with treatment facor.

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment groups	9.15	2	4.60	9.10	.000*
	Disc degeneration	0.22	1	0.22	0.43	.514
	Residual	107.70	214	0.50		
4 weeks	Treatment groups	19.50	2	9.76	6.16	.003*
	Disc degeneration	0.25	1	0.25	0.16	.69
	Treatment x Disc degeneration	3.70	2	1.87	1.18	.31
	Residual	247.15	156	1.58		
6 weeks	Treatment groups	90.96	2	45.48	16.30	.000*
	Disc degeneration	0.88	1	0.88	0.315	.576
	Treatment x Disc degeneration	2.56	2	1.28	0.46	.63
	Residual	405.16	145	2.80		
12 weeks	Treatment groups	121.10	2	60.56	14.66	.000*
	Disc degeneration	0.75	1	0.75	0.18	.67
	Treatment x Disc degeneration	13.79	2	6.89	1.67	.192
	Residual	590.80	143	0.41		
24 weeks	Treatment groups	169.20	2	84.60	17.59	.000*
	Disc degeneration	0.04	1	0.04	0.009	.925
	Treatment x Disc degeneration	5.79	2	2.89	0.603	.549
	Residual	557.69	116	4.80		
52 weeks	Treatment groups	257.45	2	128.7	29.48	.000*
	Disc degeneration	1.49	1	1.49	0.34	.56
	Treatment x Disc degeneration	17.83	2	8.90	2.04	.135
	Residual	475.93	109	4.40		

*** = Statistically significant**

Table A.III.5.16 Summary of Two-way ANOVA results for the effect of interscapular pain (ISP).

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment groups	7.60	2	3.80	8.60	.000*
	Interscapular pain	14.60	1	14.60	33.30	.000*
	Treatment x ISP	2.14	2	1.70	2.40	.089
	Residual	93.86	214	0.44		
4 weeks	Treatment groups	6.59	2	3.27	2.45	.089
	Interscapular pain	41.89	1	41.89	31.40	.000*
	Treatment x ISP	1.34	2	0.67	0.50	.607
	Residual	207.90	161	1.69		
6 weeks	Treatment groups	76.10	2	38.01	15.00	.000*
	Interscapular pain	34.27	1	34.27	13.54	.000*
	Treatment x ISP	7.27	2	3.60	1.40	.241
	Residual	367.05	145	2.50		
12 weeks	Treatment groups	94.90	2	47.40	13.10	.000*
	Interscapular pain	81.10	1	81.10	22.47	.000*
	Treatment x ISP	8.08	2	4.04	1.10	.33
	Residual	516.10	143	3.60		
24 weeks	Treatment groups	135.20	2	67.60	15.30	.000*
	Interscapular pain	46.40	1	46.40	10.50	.002*
	Treatment x ISP	5.09	2	2.50	0.58	.56
	Residual	511.97	116	4.40		
52 weeks	Treatment groups	235.20	2	117.6	27.40	.000*
	Interscapular pain	18.10	1	18.10	4.20	.042*
	Treatment x ISP	9.80	2	4.90	1.10	.321
	Residual	467.30	109	4.29		

*** = Statistically significant**

Table A.III.5.17 Summary of 2-way ANOVA results for the effect of compensable and noncompensable status and its interaction with treatment factor.

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment groups	8.25	2	4.12	8.34	.000*
	Litigation	3.10	1	3.10	6.30	.013*
	Residual	84.99	172	0.49		
4 weeks	Treatment groups	13.90	2	6.90	4.80	.009*
	Litigation	0.27	1	0.27	0.19	.667
	Treatment x Litigation	0.78	2	0.39	0.27	.763
	Residual	182.50	127	1.44		
6 weeks	Treatment groups	84.02	2	42.01	15.10	.000*
	Litigation	0.74	1	0.74	0.27	.607
	Treatment x Litigation	5.00	2	2.50	0.90	.407
	Residual	327.60	118	2.78		
12 weeks	Treatment groups	108.60	2	54.30	12.40	.000*
	Litigation	2.90	1	2.99	0.68	.41
	Treatment x Litigation	1.50	2	0.76	0.17	.84
	Residual	510.70	117	4.36		
24 weeks	Treatment groups	146.90	2	73.40	16.80	.000*
	Litigation	16.20	1	16.20	3.70	.057
	Treatment x Litigation	5.82	2	2.90	0.66	.517
	Residual					
52 weeks	Treatment groups	290.57	2	125.76	33.10	.000*
	Litigation	34.79	1	34.79	9.16	.003*
	Treatment x Litigation	22.30	2	11.15	2.94	.06
	Residual	345.70	91	3.80		

*** = Statistically significant.**

Table A.III.5.18 Summary of Scheffe's procedure with reference to the effect of litigation of initial examination (IE).

Geometric mean - IE	Group	Group-1	Group-2	Group-3	Group-4	Group-5	Group-6
179.5	Grp-4 - Active exercise(non-compensable)						
181.3	Grp-6 - Control group (non-compensable)						
372.4	Grp-3 - Active exercise (compensable)						
424.1	Grp-5 - Control group (compensable)						
578.2	Grp-2 - Immobilised (non-compensable)						
584.1	Grp-1 - Immobilised (compensable)				*		

**** = Statisically significant***

Table A.III.5.19 Summary of Scheffe's procedure with reference to the effect of litigation at Week-52 follow -up examination.

Geometric mean - Wk 52	Group	Group-2	Group-6	Group-1	Group-4	Group-3	Group-5
1.0	Grp-2 - Immobilised (non-compensable)						
1.0	Grp-6 -Control group (non-compensable)						
3.9	Grp-1 - Immobilised (compensable)						
24.3	Grp-4 - Active exercise(non-compensable)						
62.2	Grp-3 - Active exercise (compensable)	*		*			
167.3	Grp-5 - Control group (compensable)	*	*	*			

**** = Statisically significant***

Table A.III.5.20 Summary of 2-way ANOVA results for the effect of "Difficulty in focusing" and its interaction with treatment factors.

Time Period	Source of variation	S S	DF	MS	F	p
Initial exam-ination	Treatment groups	6.38	2	3.19	6.47	.002*
	Difficulty in Focusing	4.81	1	4.81	9.77	.002*
	Treatment X Dif Focusing	1.26	2	0.63	1.28	.281
	Residual	104.46	212	0.49		
4 weeks	Treatment groups	16.76	2	8.38	5.25	.006*
	Difficulty in Focusing	2.87	1	2.87	1.80	.182
	Treatment X Dif Focusing	2.08	2	1.04	0.65	.522
	Residual	242.46	152	1.59		
6 weeks	Treatment groups	86.40	2	43.20	15.72	.000*
	Difficulty in Focusing	7.53	1	7.53	2.74	.100
	Treatment X Dif Focusing	4.18	1	4.18	1.52	.219
	Residual	395.81	144	2.75		
12 weeks	Treatment groups	118.21	2	59.10	14.85	.000*
	Difficulty in Focusing	27.18	1	27.18	6.83	.01*
	Treatment X Dif Focusing	8.77	2	4.38	1.10	.335
	Residual	565.37	142	3.98		
24 weeks	Treatment groups	151.55	2	75.77	16.23	.000*
	Difficulty in Focusing	21.25	1	21.25	4.55	.035*
	Treatment X Dif Focusing	3.69	2	1.85	0.40	.67
	Residual	536.84	115	4.67		
52 weeks	Treatment groups	267.70	2	133.86	30.87	.000*
	Difficulty in Focusing	25.33	1	25.33	5.80	.017*
	Treatment X Dif Focusing	3.52	2	1.76	0.40	.667
	Residual	463.94	107	4.34		

* = **Statistically significant**

Table A.III.5.21 Summary of 2-way ANOVA results for the effect of "Blurred vision" and its interaction with treatment factors.

Time Period	Source of variation	SS	DF	MS	F	p
Initial examination	Treatment groups	7.71	2	3.90	7.70	.001*
	Blurred vision	2.40	1	2.40	4.86	.029*
	Treatment X Blurred vision	1.89	2	0.95	1.90	.154
	Residual	106.23	212	0.50		
4 weeks	Treatment groups	12.45	2	6.20	4.00	.02*
	Blurred vision	12.10	1	12.10	7.90	.006*
	Treatment X Blurred vision	1.05	2	0.52	0.34	.7
	Residual	233.10	151	1.50		
6 weeks	Treatment groups	82.30	2	41.20	15.20	.000*
	Blurred vision	15.10	1	15.10	5.60	.02*
	Treatment X Blurred vision	0.76	2	0.38	0.14	.87
	Residual	379.50	140	2.70		
12 weeks	Treatment groups	114.61	2	57.30	14.70	.000*
	Blurred vision	40.40	1	40.40	10.30	.002*
	Treatment X Blurred vision	5.80	2	2.90	0.70	.48
	Residual	555.12	142	3.90		
24 weeks	Treatment groups	166.66	2	83.30	18.00	.000*
	Blurred vision	27.40	1	27.40	5.90	.016*
	Treatment X Blurred vision	2.10	2	1.06	0.20	.8
	Residual	531.70	115	4.60		
52 weeks	Treatment groups	245.38	2	122.69	27.87	.000*
	Blurred vision	18.67	1	18.67	4.20	.042*
	Treatment X Blurred vision	1.23	2	0.62	0.14	.87
	Residual	470.90	107	4.40		

* = **Statisically significant**

Table A.III.8.1 **Descriptive statistics of the active range of motion of the cervical spine for the total sample. (IE = Initial examination, Rt=Right, Lt=Left).**

Time period	Movement	n	Mean	S.D	Minimum	Maximum
IE	Flexion-Active	220	29.15	11.66	5	50
Week-4	Flexion	162	37.62	9.59	10	60
Week-6	Flexion	151	40.46	9.06	10	60
Week-12	Flexion	149	42.32	7.89	10	60
Week-24	Flexion	122	43.03	6.64	20	60
Week-52	Flexion	115	43.96	7.71	20	60
IE	Extension	220	26.66	11.29	5	50
Week-4	Extension	162	35.31	9.79	10	60
Week-6	Extension	151	38.34	9.22	10	65
Week-12	Extension	149	40.00	8.36	10	55
Week-24	Extension	122	39.92	7.24	10	60
Week-52	Extension	115	40.57	8.13	10	60
IE	Side Flexion-Rt	220	24.25	9.51	5	45
Week-4	Side Flexion-Rt	162	29.97	8.37	5	50
Week-6	Side Flexion-Rt	151	32.34	7.45	10	45
Week-12	Side Flexion-Rt	149	34.03	6.77	10	45
Week-24	Side Flexion-Rt	122	34.88	5.94	20	45
Week-52	Side Flexion-Rt	115	34.91	5.69	20	45
IE	Side Flexion-Lt	220	24.91	9.84	5	40
Week-4	Side Flexion-Lt	162	30.49	7.77	10	50
Week-6	Side Flexion-Lt	151	32.65	7.18	10	45
Week-12	Side Flexion-Lt	149	34.19	6.50	10	45
Week-24	Side Flexion-Lt	122	35.00	5.46	15	45
Week-52	Side Flexion-Lt	115	35.22	6.12	15	50
IE	Rotation -Rt	220	48.66	17.09	10	80
Week-4	Rotation-Rt	162	60.53	13.15	20	85
Week-6	Rotation-Rt	151	65.03	10.87	30	35
Week-12	Rotation-Rt	149	67.88	10.37	30	85
Week-24	Rotation-Rt	122	68.63	9.44	40	85
Week-52	Rotation-Rt	115	69.35	8.89	40	85
IE	Rotation-Lt	220	50.05	17.09	10	80
Week-4	Rotation-Lt	162	60.89	13.35	20	85
Week-6	Rotation-Lt	151	65.43	11.62	20	85
Week-12	Rotation-Lt	149	68.05	10.63	20	85
Week-24	Rotation-Lt	122	68.93	8.82	40	85
Week-52	Rotation-Lt	115	69.35	9.19	30	85

Table A.III.8.2 **Confidence interval for the range of motion -Total sample. (IE = Initial examination, Rt=Right, Lt=Left).**

Time period	Movement	n	S.D	Mean	Upper limit	Lower limit
IE	Flexion-Active	220	11.66	29.15	30.69	27.60
Week-4	Flexion	162	9.59	37.62	39.10	36.15
Week-6	Flexion	151	9.06	40.46	41.90	39.02
Week-12	Flexion	149	7.89	42.32	43.58	41.05
Week-24	Flexion	122	6.64	43.03	44.30	41.76
Week-52	Flexion	115	7.71	43.96	45.39	42.51
IE	Extension	220	11.29	26.66	28.15	25.17
Week-4	Extension	162	9.79	35.31	36.81	33.80
Week-6	Extension	151	9.22	38.34	39.81	36.87
Week-12	Extension	149	8.36	40.00	41.34	38.66
Week-24	Extension	122	7.24	39.92	41.20	38.63
Week-52	Extension	115	8.13	40.57	42.08	39.04
IE	Side Flexion-Rt	220	9.51	24.25	25.50	22.99
Week-4	SideFlexion-Rt	162	8.37	29.97	31.26	28.68
Week-6	Side Flexion-Rt	151	7.45	32.34	33.52	31.11
Week-12	Side Flexion-Rt	149	6.77	34.03	35.11	32.94
Week-24	Side Flexion-Rt	122	5.94	34.88	35.93	33.82
Week-52	Side Flexion-Rt	115	5.69	34.91	35.97	33.85
IE	Side Flexion-Lt	220	9.84	24.91	26.21	23.61
Week-4	Side Flexion-Lt	162	7.77	30.49	31.69	29.30
Week-6	Side Flexion-Lt	151	7.18	32.65	33.80	31.50
Week-12	Side Flexion-Lt	149	6.50	34.19	35.24	33.15
Week-24	Side Flexion-Lt	122	5.46	35.00	35.97	34.03
Week-52	Side Flexion-Ltt	115	6.12	35.22	36.36	34.07
IE	Rotation -Rt	220	17.09	48.66	50.91	46.40
Week-4	Rotation-Rt	162	13.15	60.53	62.55	58.50
Week-6	Rotation-Rt	151	10.87	65.03	66.77	63.29
Week-12	Rotation-Rt	149	10.37	67.88	69.54	66.21
Week-24	Rotation-Rt	122	9.44	68.63	70.30	66.96
Week-52	Rotation-Rt	115	8.89	69.35	71.76	67.03
IE	Rotation-Lt	220	17.09	50.05	52.65	47.44
Week-4	Rotation-Lt	162	13.35	60.89	62.95	58.83
Week-6	Rotation-Lt	151	11.62	65.43	67.28	63.58
Week-12	Rotation-Lt	149	10.63	68.05	69.76	66.35
Week-24	Rotation-Lt	122	8.82	68.93	70.49	66.37
Week-52	Rotation-Lt	115	9.18	69.35	71.06	76.63

Table A.III.8.3 **Descriptive statistics of the active range of motion of the cervical spine for the immobilised group. (IE = Initial examination, Rt=Right, Lt=Left).**

Time period-A	Movement	n	Mean	S.D	Minimum	Maximum
IE	Flexion-Active	71	25.99	12.00	5	50
Week-4	Flexion	59	39.83	8.51	20	60
Week-6	Flexion	60	43.67	7.75	10	60
Week-12	Flexion	57	44.56	6.89	30	60
Week-24	Flexion	48	45.52	5.67	30	60
Week-52	Flexion	42	47.50	5.87	35	60
IE	Extension	71	24.29	11.81	5	45
Week-4	Extension	59	37.46	8.27	20	60
Week-6	Extension	60	41.67	8.32	10	65
Week-12	Extension	57	42.19	7.19	20	55
Week-24	Extension	48	42.92	5.63	30	60
Week-52	Extension	42	44.64	5.67	35	60
IE	Side Flexion-Rt	71	22.39	9.71	5	45
Week-4	Side Flexion-Rt	59	32.12	7.32	15	50
Week-6	Side Flexion-Rt	60	35.17	6.24	15	45
Week-12	Side Flexion-Rt	57	36.23	5.61	20	45
Week-24	Side Flexion-Rt	48	37.60	4.12	30	45
Week-52	Side Flexion-Rt	42	37.62	3.70	30	45
IE	Side Flexion-Lt	71	22.68	10.41	5	40
Week-4	Side Flexion-Lt	59	32.88	6.84	20	50
Week-6	Side Flexion-Lt	60	35.50	6.07	20	45
Week-12	Side Flexion-Ltt	57	36.10	5.67	20	45
Week-24	Side Flexion-Lt	48	37.08	3.97	30	45
Week-52	Side Flexion-Lt	42	38.21	4.11	35	50
IE	Rotation -Rt	71	42.75	17.21	10	75
Week-4	Rotation-Rt	59	63.56	10.13	30	80
Week-6	Rotation-Rt	60	68.33	9.51	40	85
Week-12	Rotation-Rt	57	71.40	8.06	50	85
Week-24	Rotation-Rt	48	72.08	6.83	50	80
Week-52	Rotation-Rt	42	73.45	6.19	50	85
IE	Rotation-Lt	71	44.29	17.95	10	80
Week-4	Rotation-Lt	59	64.07	8.63	40	80
Week-6	Rotation-Lt	60	68.67	10.97	40	85
Week-12	Rotation-Lt	57	71.49	7.62	50	85
Week-24	Rotation-Lt	48	72.19	6.35	50	80
Week-52	Rotation-Lt	42	73.33	5.59	50	85

Table A.III.8.4 **Mean, standard deviation, upper and lower 95% confidence limits of the mean for the range of motion-Immobilised group. (IE = Initial examination, Rt=Right, Lt=Left).**

Time period	Movement	n	S.D	Mean	95% Upper limit	95% Lower limit
IE	Flexion-Active	71	12.00	25.97	28.83	23.13
Week-4	Flexion	59	8.51	39.83	42.07	37.59
Week-6	Flexion	60	7.75	43.67	48.19	39.14
Week-12	Flexion	57	6.89	44.56	46.40	42.71
Week-24	Flexion	48	5.67	45.52	47.17	43.87
Week-52	Flexion	42	5.87	47.50	49.33	45.66
IE	Extension	71	11.81	24.29	27.09	21.49
Week-4	Extension	59	8.27	37.46	39.63	35.28
Week-6	Extension	60	8.32	41.67	43.84	39.49
Week-12	Extension	57	7.19	42.19	44.11	40.27
Week-24	Extension	48	5.63	42.92	44.56	40.55
Week-52	Extension	42	5.67	44.64	46.41	42.87
IE	Side Flexion-Rt	71	9.71	22.39	24.69	20.69
Week-4	Side Flexion-Rt	59	7.32	32.12	34.04	30.19
Week-6	Side Flexion -Rt	60	6.24	35.17	36.79	33.53
Week-12	Side Flexion-Rt	57	5.61	36.23	37.73	34.72
Week-24	Side Flexion-Rt	48	4.12	37.60	38.81	36.40
Week-52	Side Flexion-Rt	42	3.70	37.62	38.78	36.46
IE	Side Flexion-Lt	71	10.41	22.68	25.14	20.20
Week-4	Side Flexion-Lt	59	6.84	32.88	34.67	31.08
Week-6	Side Flexion-Lt	60	6.09	35.50	37.08	33.91
Week-12	Side Flexion-Lt	57	5.67	36.14	37.65	34.62
Week-24	Side Flexion-Lt	48	3.97	37.08	38.24	35.92
Week-52	Side Flexion-Lt	42	4.11	38.21	39.49	36.93
IE	Rotation -Rt	71	17.21	42.75	46.83	38.66
Week-4	Rotation-Rt	59	10.13	63.56	66.22	60.89
Week-6	Rotation-Rt	60	9.51	68.33	70.81	65.85
Week-12	Rotation-Rt	57	8.06	71.40	73.88	68.92
Week-24	Rotation-Rt	48	6.83	72.08	74.07	70.09
Week-52	Rotation-Rt	42	6.19	73.45	75.38	71.51
IE	Rotation-Lt	71	17.95	44.29	45.23	43.35
Week-4	Rotation-Lt	59	8.63	64.07	66.34	61.79
Week-6	Rotation-Lt	60	10.97	68.67	71.53	65.80
Week-12	Rotation-Lt	57	7.62	71.49	73.53	69.45
Week-24	Rotation-Lt	48	6.35	72.19	74.04	70.33
Week-52	Rotation-Lt	42	5.59	73.33	75.18	71.48

Table A.III.8.5 **Descriptive statistics of the active range of motion of the cervical spine for the active exercise group. (IE = Initial examination, Rt=Right, Lt=Left).**

Time period	Movement	n	Mean	S.D	Minimum	Maximum
IE	Flexion-Active	60	31.75	11.00	10	50
Week-4	Flexion	44	40.46	9.00	20	55
Week-6	Flexion	37	40.00	8.49	20	55
Week-12	Flexion	42	42.26	7.34	20	50
Week-24	Flexion	29	42.14	7.02	30	50
Week-52	Flexion	28	43.39	8.28	20	60
IE	Extension	60	30.50	10.40	10	50
Week-4	Extension	44	38.18	8.43	20	50
Week-6	Extension	37	38.11	8.45	20	50
Week-12	Extension	42	41.19	7.39	20	50
Week-24	Extension	29	39.31	6.37	30	50
Week-52	Extension	28	39.64	9.02	25	60
IE	Side Flexion-Rt	60	27.42	9.04	10	40
Week-4	Side Flexion-Rt	44	31.59	7.98	10	40
Week-6	Side Flexion-Rt	37	32.57	7.32	15	40
Week-12	Side Flexion-Rt	42	33.57	7.01	15	40
Week-24	Side Flexion-Rt	29	34.14	5.01	25	40
Week-52	Side Flexion-Rt	28	34.64	7.06	20	45
IE	Side Flexion-Lt	60	29.08	8.85	10	40
Week-4	Side Flexion-Lt	44	32.52	7.35	20	45
Week-6	Side Flexion-Lt	37	32.03	6.39	15	40
Week-12	Side Flexion-Lt	42	33.93	7.03	15	45
Week-24	Side Flexion-Lt	29	34.83	4.53	25	40
Week-52	Side Flexion-Lt	28	35.18	6.16	20	45
IE	Rotation -Rt	60	54.92	16.35	10	80
Week-4	Rotation-Rt	44	64.09	11.48	40	85
Week-6	Rotation-Rt	37	65.54	11.35	40	85
Week-12	Rotation-Rt	42	67.02	10.06	40	85
Week-24	Rotation-Rt	29	67.86	9.89	40	85
Week-52	Rotation-Rt	28	67.86	10.40	45	85
IE	Rotation-Lt	60	57.58	14.59	20	80
Week-4	Rotation-Lt	44	64.21	12.25	30	85
Week-6	Rotation-Lt	37	64.87	11.64	40	85
Week-12	Rotation-Lt	42	66.79	10.29	40	85
Week-24	Rotation-Lt	29	67.76	8.19	50	85
Week-52	Rotation-Lt	28	68.21	9.55	50	85

Table A.III.8.6 **Mean, standard deviation, upper and lower 95% confidence limits of the mean for the range of motion - Active exercise group. (IE = Initial examination, Rt=Right, Lt=Left).**

Time period	Movement	n	S.D	Mean	95% Upper limit	95% Lower limit
IE	Flexion-Active	60	11.01	31.75	34.62	28.87
Week-4	Flexion	44	9.08	40.46	43.22	37.69
Week-6	Flexion	37	8.49	40.00	42.85	37.15
Week-12	Flexion	42	7.34	42.26	44.55	39.97
Week-24	Flexion	29	7.02	42.14	44.81	39.47
Week-52	Flexion	28	8.28	43.39	46.60	40.18
IE	Extension	60	10.40	30.5	33.21	27.78
Week-4	Extension	44	8.43	38.18	40.75	35.61
Week-6	Extension	37	8.45	38.11	40.94	35.27
Week-12	Extension	42	7.39	41.19	43.49	38.88
Week-24	Extension	29	6.37	39.31	41.73	36.89
Week-52	Extension	28	9.02	39.64	43.14	36.14
IE	Side Flexion-Rt	60	9.04	27.42	29.78	25.05
Week-4	Side Flexion-Rt	44	7.98	31.59	34.02	29.16
Week-6	Side Flexion-Rt	37	7.32	32.57	35.02	30.11
Week-12	Side Flexion-Rt	42	7.01	33.57	35.78	31.36
Week-24	Side Flexion-Rt	29	5.01	34.14	36.04	32.23
Week-52	Side Flexion-Rt	28	7.06	34.64	37.38	31.9
IE	Side Flexion-Lt	60	8.85	29.08	31.39	26.77
Week-4	Side Flexion-Lt	44	7.35	32.52	34.76	30.28
Week-6	Side Flexion-Lt	37	6.39	32.03	34.17	29.88
Week-12	Side Flexion-Lt	42	7.03	33.93	36.12	31.74
Week-24	Side Flexion-Lt	29	4.53	34.83	36.55	33.10
Week-52	Side Flexion-Lt	28	6.16	35.18	37.57	32.79
IE	Rotation -Rt	60	16.35	54.92	59.19	50.65
Week-4	Rotation-Rt	44	11.48	64.09	67.90	60.59
Week-6	Rotation-Rt	37	11.35	65.54	69.35	61.73
Week-12	Rotation-Rt	42	10.06	67.02	70.16	63.88
Week-24	Rotation-Rt	29	9.89	67.86	71.62	64.09
Week-52	Rotation-Rt	28	10.40	67.86	71.89	63.82
IE	Rotation-Lt	60	14.59	57.58	61.39	53.77
Week-4	Rotation-Lt	44	12.25	64.21	67.93	60.47
Week-6	Rotation-Lt	37	11.65	64.87	68.77	60.96
Week-12	Rotation-Lt	42	10.29	66.79	69.99	63.58
Week-24	Rotation-Lt	29	8.19	67.76	70.87	64.64
Week-52	Rotation-Lt	28	9.55	68.21	71.92	64.51

Table A.III.8.7 **Descriptive statistics of the active range of motion of the cervical spine for the control group. (IE = Initial exmination, Rt=Right, Lt=Left).**

Time period-C	Movement	n	Mean	S.D	Minimum	Maximum
IE	Flexion-Active	89	29.91	11.38	5	50
Week-4	Flexion	59	33.31	9.59	10	50
Week-6	Flexion	54	37.22	9.69	10	55
Week-12	Flexion	50	39.80	8.75	10	55
Week-24	Flexion	45	40.78	6.57	20	55
Week-52	Flexion	45	41.00	7.66	20	55
IE	Extension	89	25.96	10.89	5	45
Week-4	Extension	59	31.02	10.74	10	50
Week-6	Extension	54	34.82	9.47	10	50
Week-12	Extension	50	36.50	9.33	10	55
Week-24	Extension	45	37.11	8.15	10	50
Week-52	Extension	45	37.33	8.02	10	50
IE	Side flexion-Rt	89	23.59	9.23	5	40
Week-4	Side flexion-Rt	59	26.61	8.68	5	40
Week-6	Side flexion-Rt	54	29.04	7.54	10	40
Week-12	Side flexion-Rt	50	31.99	7.14	10	40
Week-24	Side flexion-Rt	45	32.44	6.96	20	40
Week-52	Side flexion-Rt	45	32.56	5.29	20	40
IE	Side flexion-Lt	89	23.88	9.22	5	40
Week-4	Side flexion-Lt	59	26.86	7.71	10	40
Week-6	Side flexion-Lt	54	29.91	7.74	10	40
Week-12	Side flexion-Lt	50	32.20	6.40	10	40
Week-24	Side flexion-Lt	45	32.89	6.53	15	40
Week-52	Side flexion-Lt	45	32.44	6.36	15	40
IE	Rotation -Rt	89	49.16	16.03	10	75
Week-4	Rotation-Rt	59	54.83	15.09	20	80
Week-6	Rotation-Rt	54	61.02	10.83	30	85
Week-12	Rotation-Rt	50	64.00	11.65	30	85
Week-24	Rotation-Rt	45	65.44	20.44	40	80
Week-52	Rotation-Rt	45	66.44	8.77	40	85
IE	Rotation-Lt	89	49.55	16.19	10	75
Week-4	Rotation-Lt	59	55.25	16.07	20	80
Week-6	Rotation-Lt	54	62.22	11.56	30	85
Week-12	Rotation-Lt	50	65.20	12.78	20	85
Week-24	Rotation-Lt	45	66.22	10.39	40	80
Week-52	Rotation-Lt	45	66.33	10.41	30	85

Table A.III.8.8 **Mean, standard deviation, upper and lower 95% confidence limits of the mean for the range of motion - Control group. (IE = Initial examination, Rt=Right, Lt=Left).**

Time period-c	Movement	n	S.D	Mean	95% Upper limit	95% Lower limit
IE	Flexion-Active	89	11.38	29.91	32.29	27.20
Week-4	Flexion	59	9.59	33.31	35.83	30.78
Week-6	Flexion	54	9.69	37.20	39.89	34.55
Week-12	Flexion	50	8.75	39.80	42.3	37.30
Week-24	Flexion	45	6.57	40.78	42.76	38.80
Week-52	Flexion	45	7.66	41.00	43.31	38.69
IE	Extension	89	10.89	25.96	28.24	25.67
Week-4	Extension	59	10.74	31.02	33.84	28.19
Week-6	Extension	54	9.47	34.82	37.42	32.21
Week-12	Extension	50	9.33	36.50	39.16	33.83
Week-24	Extension	45	8.15	37.11	39.57	34.65
Week-52	Extension	45	8.02	37.33	39.75	34.92
IE	Side flexion-Rt	89	9.23	23.59	25.53	21.66
Week-4	Side flexion-Rt	59	8.68	26.61	28.89	24.32
Week-6	Side flexion-Rt	54	7.54	29.04	31.11	26.96
Week-12	Side flexion-Rt	50	7.14	31.99	34.03	29.95
Week-24	Side flexion-Rt	45	6.96	32.44	34.54	30.35
Week-52	Side flexion-Rt	45	5.29	32.56	34.15	30.96
IE	Side flexion-Lt	89	9.22	23.88	25.81	21.94
Week-4	Side flexion-Lt	59	7.71	26.86	28.89	24.83
Week-6	Side flexion-Lt	54	7.74	29.91	32.03	27.78
Week-12	Side flexion-Lt	50	6.40	32.20	34.03	30.37
Week-24	Side flexion-Lt	45	6.53	32.89	34.85	30.92
Week-52	Side flexion-Lt	45	6.36	32.44	34.36	30.53
IE	Rotation -Rt	89	16.03	49.16	52.52	45.79
Week-4	Rotation-Rt	59	15.09	54.83	58.80	50.86
Week-6	Rotation-Rt	54	10.83	61.02	64.00	58.04
Week-12	Rotation-Rt	50	11.65	64.00	67.33	60.67
Week-24	Rotation-Rt	45	10.44	65.44	68.59	62.29
Week-52	Rotation-Rt	45	8.77	66.44	69.08	63.8
IE	Rotation-Lt	89	16.18	49.55	52.95	46.15
Week-4	Rotation-Lt	59	16.07	55.25	59.48	51.03
Week-6	Rotation-Lt	54	11.56	62.22	65.40	59.04
Week-12	Rotation-Lt	50	12.78	65.20	68.85	61.55
Week-24	Rotation-Lt	45	10.39	66.22	69.35	63.09
Week-52	Rotation-Lt	45	10.41	66.33	69.47	63.2

Table A.III.8.9 **A comparison between active and passive range of motions for all the movements in the cervical spine (IE = Initial examination).**

Movemets	IE n=220	4th-week n=162	6th-week n=151	12th-week n=149	24th-week n=122	52-week n=116
Flexion Active / Passive	r=.93 p=.000*	r=.89 p=.000*	r=.90 p=.000*	r=.92 p=.000*	r=.83 p=.000*	r=.94 p=.000*
Extension Active / Passive	r=.91 p=.000*	r=.91 p=.000*	r=.95 p=.000*	r=.93 p=.000*	r=.94 p=.000*	r=.97 p=.000*
Side Flexion-Rt Active / Passive	r=.97 p=.000*	r=.94 p=.000*	r=.92 p=.000*	r=.94 p=.000*	r=.97 p=.000*	r=.94 p=.000*
Side Flexion-Lt Active / Passive	r=.95 p=.000*	r=.93 p=.000*	r=.93 p=.000*	r=.94 p=.000*	r=.95 p=.000*	r=.94 p=.000*
Rotation-Rt Active / Passive	r=.92 p=.000*	r=.87 p=.000*	r=.93 p=.000*	r=.91 p=.000*	r=.93 p=.000*	r=.84 p=.000*
Rotation-Lt Active / Passive	r=.94 p=.000*	r=.84 p=.000*	r=.82 p=.000*	r=.94 p=.000*	r=.83 p=.000*	r=.89 p=.000*

* = Statistically significant

Table A.III.8.10 **A comparison of range of motion between the right and left sides for rotations and side flexions of the cervical spine (IE = Initial examination).**

Movements	IE n=220	4th-week n=162	6th-week n=151	12th-week n=149	24th-week n=122	52-week n=116
Side Flexion- Active Right / Left	r=.87 p=.000*	r=.89 p=.000*	r=.89 p=.000*	r=.89 p=.000*	r=.87 p=.000*	r=.86 p=.000*
Side Flexion Passive Right / Left	r=.87 p=.000*	r=.86 p=.000*	r=.91 p=.000*	r=.84 p=.000*	r=.82 p=.000*	r=.76 p=.000*
Rotation-Active Right / Left	r=.84 p=.000*	r=.84 p=.000*	r=.77 p=.000*	r=.88 p=.000*	r=.89 p=.000*	r=.79 p=.000*
Rotation-Passive Right / Left	r=.84 p=.000*	r=.72 p=.000*	r=.72 p=.000*	r=.83 p=.000*	r=.82 p=.000*	r=.84 p=.000*

* = Statistically significant

Table A.III.8.11 ANCOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine, using age and pain as covariates -initial examination.

Movements	Source of variation	SS	DF	MS	F	p
Flexion	Treatment	245.32	2	122.66	1.17	.311
	Age	88.09	1	88.09	0.84	.360
	Pain	6895.42	1	6895.42	65.95	.000*
	Residual	22480.14	215	104.56		
Extension	Treatment	314.54	2	157.27	1.60	.204
	Age	94.54	1	94.54	0.96	.328
	Pain	6416.19	1	6416.19	1.60	.000*
	Residual	21146.49	215	98.36		
Side flexion-right	Treatment	147.92	2	73.96	1.20	.304
	Age	75.91	1	75.91	1.23	.269
	Pain	6221.84	1	6221.84	100.66.	.000*
	Residual	13289.78	215	61.81		
Rotation-right	Treatment	1262.65	2	631.32	3.07	.049*
	Age	34.14	1	34.14	0.17	.684
	Pain	18313.87	1	18313.87 2.00	88.93	.000*
	Residual	44274.95	215	205.93		

* = **Statistically significant.**

Table A.III.8.12 **ANCOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine, using age and pain as covariates -Week-4.**

Movements	Source of variation	SS	DF	MS	F	p
Flexion	Treatment	781.37	2	390.68	6.35	.002*
	Age	763.26	1	763.26	12.41	.001*
	Pain	2653.66	1	2653.66	43.16	.000*
	Residual	9654.13	157	61.49		
Extension	Treatment	782.55	2	391.27	5.91	.003*
	Age	437.84	1	437.84	6.61	.011*
	Pain	2997.24	1	2997.24	45.24	.000*
	Residual	10402.59	157	66.26		
Side flexion-right	Treatment	336.22	2	168.11	3.64	.028*
	Age	1384.12	1	1384.12	29.98	.000*
	Pain	1404.61	1	1404.61	30.4	.000*
	Residual	7247.25	157	46.16		
Rotation-right	Treatment	1386.04	2	693.02	5.7	.004*
	Age	3056.86	1	3056.86	25.2	.000*
	Pain	2559.28	1	2559.28	21.1	.000*
	Residual	19016.10	157	121.12		

* = **Statistically significant**

Table A.III.8.13 **ANCOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine,using age and pain as covariates -Week-6.**

Movements	Source of variation	SS	DF	MS	F	p
Flexion	Treatment	77.52	2	38.76	0.70	.496
	Age	1633.38	1	1633.38	29.72	.000*
	Pain	1419.12	1	1419.12	25.80	.000*
	Residual	8023.70	146	54.96		
Extension	Treatment	103.54	2	51.77	0.97	.383
	Age	1631.76	1	1631.76	30.46	.000*
	Pain	1849.55	1	1849.55	34.52	.000*
	Residual	7821.81	146	53.57		
Side flexion-right	Treatment	164.27	2	82.13	2.52	.084
	Age	1441.15	1	1441.15	44.18	.000*
	Pain	1017.06	1	1017.06	31.18	.000*
	Residual	4762.16	146	32.62		
Rotation-right	Treatment	231.08	2	115.54	1.57	.212
	Age	2050.69	1	2050.69	27.83	.000*
	Pain	2834.66	1	2834.66	38.47	.000*
	Residual	10758.90	146	73.69		

*** = Statistically significant**

Table A.III.8.14 **ANCOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine, using age and pain as covariates -Week-12.**

Movements	Source of variation	SS	DF	MS	F	P
Flexion	Treatment	46.41	2	23.20	0.52	.598
	Age	1183.90	1	1183.90	26.28	.000*
	Pain	667.89	1	667.89	14.83	.000*
	Residual	6485.94	144	45.04		
Extension	Treatment	303.41	2	151.71	3.21	.043*
	Age	1575.88	1	1575.88	33.37	.000*
	Pain	678.98	1	678.98	14.38	.000*
	Residual	6800.78	144	47.23		
Side flexion-right	Treatment	13.02	2	6.51	0.22	.803
	Age	1277.33	1	1277.33	43.06	.000*
	Pain	471.90	1	471.90	15.91	.000*
	Residual	4271.44	144	29.66		
Rotation-right	Treatment	65.70	2	32.85	0.50	.607
	Age	3316.09	1	3316.09	50.62	.000*
	Pain	1189.04	1	1189.04	18.15	.000*
	Residual	9433.04	144	65.51		

Table A.III.8.15 **ANCOVA summary for the active movements- flexion, extension, right side flexion and right rotation of the cervical spine, using age and pain as covariates -Week-24**

Movements	Source of variation	SS	DF	MS	F	P
Flexion	Treatment	74.12	2	37.06	1.28	.280
	Age	811.31	1	811.31	28.14	.000*
	Pain	585.14	1	585.14	20.29	.000*
	Residual	3373.71	117	28.84		
Extension	Treatment	116.62	2	58.31	1.71	.185
	Age	757.53	1	757.53	22.21	.000*
	Pain	904.32	1	904.32	26.51	.000*
	Residual	3990.91	117	34.11		
Side flexion-right	Treatment	86.23	2	43.11	2.08	.130
	Age	938.38	1	938.38	45.24	.000*
	Pain	389.78	1	389.78	18.79	.000*
	Residual	2426.90	117	20.74		
Rotation-right	Treatment	224.23	2	112.12	2.28	.107
	Age	2029.32	1	2029.32	41.18	.000*
	Pain	1519.89	1	1519.89	30.85	.000*
	Residual	5765.03	117	49.27		

Table A.III.8.16 **ANCOVA summary for the active movements-flexion, extension, right side flexion and right rotation of the cervical spine -Week-52.**

Movements (wk-52)	Source of variation	SS	DF	MS	F	P
Flexion	Treatment	145.41	2	72.70	1.66	.194
	Age	656.74	1	656.74	15.04	.000*
	Pain	549.42	1	549.42	12.58	.001*
	Residual	4802.87	110	43.66		
Extension	Treatment	84.02	2	42.01	0.96	.384
	Age	626.32	1	626.32	14.39	.000*
	Pain	1152.80	1	1152.80	26.48	.000*
	Residual	4788.04	110	43.53		
Side flexion-right	Treatment	69.02	2	43.51	1.89	.156
	Age	585.68	1	585.68	32.03	.000*
	Pain	483.56	1	483.56	26.45	.000*
	Residual	2011.18	110	18.28		
Rotation-right	Treatment	44.87	2	22.43	0.45	.639
	Age	1511.49	1	1511.49	30.32	.000*
	Pain	822.68	1	822.68	16.51	.000*
	Residual	5483.00	110	49.84		

Table A.III.8.17. Multiple regression statistics for active flexion (ROM) and predictor variables- Week-4.

Source of variance-	df	Ss	Ms	F	p
Attributable to regression	4	2845.5	711.37	6.81	.0000*
Residual	157	16394.2	104.40		

Table A.III.8.18 Prediction of change in active flexion (ROM) in relation to the level of pain at week-4, from age, and type of treatments.

Variable	B	SEB	Standardised coeff	T	p
Age	0.09	0.07	0.12	1.46	.1460
Active exercise regimen	3.94	2.05	0.16	1.91	.0573
Pain	0.62	0.65	0.07	0.94	.3468
Immobilisation regimen	7.58	2.07	0.33	3.69	.0003*

Table A.III.8.19 Multiple regression statistics for active extension (ROM) and predictor variables-Week-4.

Source of variance- wk4- Extension	df	Ss	Ms	F	p
Attributable to regression	4	3844.6	961.16	9.21	.0000*
Residual	157	16380.7	104.33		

Table A.III.8.20 Prediction of change in active extension (ROM) in relation to the level of pain at week-4, from age, and type of treatments.

Variable	B	SEB	Standardised coeff	T	p
Age	0.18	0.07	-0.20	-2.60	.0101*
Active exercise regimen	0.88	2.05	0.04	0.43	.6663
Pain	-1.10	0.65	0.13	-1.70	.0836
Immobilisation regimen	6.15	2.05	0.27	2.99	.0032*

Table A.III.8.21 Multiple regression statistics for right side flexion extension (ROM) and predictor variables- Week-4.

Source of variance-	df	Ss	Ms	F	p
Attributable to regression	4	263.1	657.76	8.65	.0000*
Residual	157	11930.1			

*** = Statistically significant**

Table A.III.8.22 **Prediction of change in active right side flexion (ROM) in relation to the level of pain at week-4, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	0.15	0.05	-0.21	-2.70	.0086*
Active exercise regimen	0.25	1.75	0.01	0.14	.8884
Pain	0.19	0.55	0.03	0.34	.7278
Immobilisation regimen	5.84	1.75	0.29	3.30	.0011*

Table A.III.8.23 **Multiple regression statistics for active right rotation (ROM) and predictor variables -Week-4.**

Source of variance-	df	Ss	Ms	F	p
Attributable to regression	4	9236.61	2309.2	11.19	.0000*
Residual	157	32375.88	206.2		

Table A.III.8.24 **Prediction of change in active right rotation (ROM) in relation to the level of pain at week-4, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	0.19	0.09	-0.15	-2.05	.0417*
Active exercise regimen	0.55	2.88	0.02	0.19	.84
Pain	0.34	0.92	0.03	0.38	.71
Immobilisation regimen	13.50	2.88	0.40	4.60	.0000*

Table A.III.8.25 **Multiple regression statistics for active flexion (ROM) and predictor variables-Week-6.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	4341.70	1085.40	9.01	.0000*
Residual	146	17576.90	120.40		

Table A.III.8.26 **Prediction of change in active flexion (ROM) in relation to the level of pain at week-6, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	0.13	0.07	0.14	-1.80	.0734
Active exercise regimen	1.58	2.36	0.06	0.67	.5046
Pain	-0.19	0.55	-0.02	-0.36	.7233
Immobilisation regimen	9.52	2.31	0.38	4.10	.0001*

*** = Statistically significant**

Table A.III.8.27 **Multiple regression statistics for active extension (ROM) and predictor variables-Week-6.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	6086.20	1521.6	12.41	.0000*
Residual	146	17894.60	122.6		

* = Statistically significant

Table A.III.8.28 **Prediction of change in active extension (ROM) in relation to the level of pain at week-6, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.29	0.07	-0.31	-4.03	.0001*
Active exercise regimen	-1.00	2.38	-0.03	-0.42	.67
Pain	-0.35	0.55	-0.05	-0.63	.5311
Immobilisation regimen	6.85	2.33	0.27	2.90	.0038*

* = Statistically significant

Table A.III.8.29 **Multiple regression statistics for active right side flexion (ROM) and predictor variables-Week-6.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	3572.1	893.1	9.8	.0000*
Residual	146	133314.9	91.2		

* = Statistically significant

Table A.III.8.30 **Prediction of change in active right side flexion (ROM) in relation to the level of pain at week-6, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.14	0.06	-0.17	-2.20	.0280*
Active exercise regimen	-2.27	2.05	-9.243	0.00	.9999
Pain	-0.86	0.48	-0.14	-1.80	.0737
Immobilisation regimen	6.07	2.01	0.28	3.01	.0030*

* = Statistically significant

Table A.III.8.31 **Multiple regression statistics for active right rotation (ROM) and predictor variables-Week-6.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	9393.80	2348.45	11.07	.0000*
Residual	146	30952.90	212.00		

* = Statistically significant

Table A.III.8.32 **Prediction of change in active right rotation (ROM) in relation to the level of pain at week-6, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.17	0.09	-0.13	-1.70	.0858
Active exercise regimen	-1.67	3.13	-0.44	-0.53	.5951
Pain	-0.23	0.73	0.03	-0.31	.7509
Immobilisation regimen	12.98	3.07	0.388	4.22	.0000*

* = Statistically significant

Table A.III.8.33 **Multiple regression statistics for active flexion (ROM) and predictor variables-Week-12.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	3141.9	785.5	7.7	.0000*
Residual	144	14655.2	101.8		

* = Statistically significant

Table A.III.8.34 **Prediction of change in active flexion (ROM) in relation to the level of pain at week-12, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.06	0.07	0.07	-0.85	.3993
Active exercise regimen	0.14	2.13	0.05	0.06	.94
Pain	0.06	0.42	0.01	0.15	.8808
Immobilisation regimen	9.04	2.18	0.40	4.10	.0001*

* = Statistically significant

Table A.III.8.35 **Multiple regression statistics for active extension (ROM) and predictor variables-Week-12.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	4043.3	1010.82	8.14	.000*
Residual	144	17864.4	124.06		

* = Statistically significant

Table A.III.8.36 **Prediction of change in active extension (ROM) in relation to the level of pain at week-12, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.26	0.08	-0.27	-3.35	.0010*
Active exercise regimen	-1.29	2.36	-0.05	-0.60	.5829
Pain	-0.09	0.46	-0.02	-0.20	.8391
Immobilisation regimen	5.64	2.41	0.23	2.30	.0208*

* = Statistically significant

Table A.III.8.37 **Multiple regression statistics for active right side flexion (ROM) and predictor variables-Week-12.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	2830.5	707.6	9.61	.0000*
Residual	144	10602.3	73.6		

* = Statistically significant

Table A.III.8.38 **Prediction of change in active right side flexion (ROM) in relation to the level of pain at week-12, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-.011	0.1	-0.15	-1.90	.0573
Active exercise regimen	-2.90	1.8	-0.13	-1.60	.1119
Pain	-0.32	0.4	-0.08	-0.89	.37
Immobilisation regimen	5.20	1.9	0.27	2.80	.0056*

* = Statistically significant

Table A.III.8.39 **Multiple regression statistics for active right rotation (ROM) and predictor variables-Week-12.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	11056.4	2764.11	12.40	.0000*
Residual	144	32048.3	222.56		

* = Statistically significant

Table A.III.8.40 **Prediction of change in active right rotation (ROM) in relation to the level of pain at week-12, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.21	0.10	-0.16	-.2.11	.0361
Active exercise regimen	-5.82	3.15	-0.15	-1.84	.0672
Pain	0.42	0.62	0.05	0.68	.4975
Immobilisation regimen	12.6	3.23	0.36	3.90	.0001*

* = Statistically significant

Table A.III.8.41 **Multiple regression statistics for active flexion (ROM) and predictor variables- week-24.**

Source of variance-	df	Ss	Ms	F	p
Attributable to regression	4	2505.9	626.47	4.84	.0012*
Residual	117	15139.3	129.39		

* = Statistically significant

Table A.III.8.42 **Prediction of change in active fleixon (ROM) in relation to the level of pain at week-24, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	0.01	0.08	0.01	0.129	.8974
Active exercise regimen	1.12	2.71	0.04	0.414	.68
Pain	0.23	0.48	0.04	0.47	.63
Immobilisation regimen	10.29	2.73	0.41	3.77	.0003*

*** = Statistically significant**

Table A.III.8.43 **Multiple regression statistics for active extension (ROM) and predictor variables- week-24.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	3946.9	986.7	7.73	.0000*
Residual	117	14942.4	127.7		

*** = Statistically significant**

Table A.III.8.44 **Prediction of change in active extension (ROM) in relation to the level of pain at week-24, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.18	0.08	-0.19	-0.22	.0297*
Active exercise regimen	-2.00	2.69	-0.07	-0.75	.4557
Pain	0.33	0.48	0.07	0.70	.4881
Immobilisation regimen	8.65	2.71	0.34	3.20	.0018*

*** = Statistically significant**

Table A.III.8.45 **Multiple regression statistics for active right side flexion (ROM) and predictor variables- week-24.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	2397.1	599.30	7.14	.0000*
Residual	117	9818.7	83.90		

*** = Statistically significant**

Table A.III.8.46 **Prediction of change in active right side flexion (ROM) in relation to the level of pain at week-24, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.05	-0.07	-0.07	-0.74	.4629
Active exercise regimen	-2.15	2.18	-0.09	-0.98	.3277
Pain	0.08	0.39	0.02	0.21	.8350
Immobilisation regimen	7.7	2.2	0.37	3.49	.0007*

*** = Statistically significant**

Table A.III.8.47 Multiple regression statistics for active right rotation (ROM) and predictor variables- week-24

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	7162.7	1790.68	7.69	.0000*
Residual	117	27234.4	232.80		

* = Statistically significant

Table A.III.8.48 Prediction of change in active right rotation (ROM) in relation to the level of pain at week-24, from Age, and type of treatments.

Variable	B	SEB	Standardised coeff	T	p
Age	-0.04	0.11	-0.04	-0.41	.69
Active exercise regimen	-5.38	3.64	-0.13	-1.47	.1418
Pain	0.35	0.65	0.05	0.54	.5905
Immobilisation regimen	13.28	3.69	0.39	3.60	.0004*

* = Statistically significant

Table A.III.8.49 Multiple regression statistics for active flexion (ROM) and predictor variables- week-52.

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	1753.8	438.5	3.56	.0000*
Residual	110	13551.1	123.2		

* = Statistically significant

Table A.III.8.50 Prediction of change in active flexion (ROM) in relation to the level of pain at week-52, from age, and type of treatments.

Variable	B	SEB	Standardised coeff	T	p
Age	0.07	0.09	0.08	0.8	.44
Active exercise regimen	0.37	2.71	0.01	0.13	.8913
Pain	-0.85	0.51	-0.19	-1.65	.1000
Immobilisation regimen	5.19	2.96	0.21	1.76	.82

* = Statistically significant

Table A.III.8.51 Multiple regression statistics for active extension (ROM) and predictor variables- week-52.

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	2920.70	730.18	5.43	.0005*
Residual	110	14778.40	134.35		

* = Statistically significant

Table A.III.8.52 **Prediction of change in active extension (ROM) in relation to the level of pain at week-52, from age, and type of treatments.**

Variable	B	SEB	Standardised coeff	T	p
Age	-0.18	0.09	-0.19	-2.09	.0392*
Active exercise regimen	-4.55	2.83	-0.16	-1.60	.1117
Pain	-0.29	0.53	-0.06	-0.54	.5899
Immobilisation regimen	3.92	3.09	0.15	1.27	.0000*

*** = Statistically significant**

Table A.III.8.53 **Multiple regression statistics for active right side flexion (ROM) and predictor variables- week-52.**

Source of variance	df	Ss	Ms	F	p
Attributable to regression	4	1179.60	294.90	4.61	.0018*
Residual	110	7043.49	64.03		

*** = Statistically significant**

Table A.III.8.54 **Prediction of change in active right side flexion (ROM) in relation to the level of pain at week-52, from age, and type of treatments.**

Variable 52 sd fl	B	SEB	Standardised coeff	T	p
Age	-0.02	0.06	-0.03	-0.30	.7633
Active exercise regimen	-3.44	1.96	-0.17	-1.76	.08
Pain	-0.21	0.37	-0.07	-0.58	.5626
Immobilisation regimen	3.80	2.13	0.22	1.80	.0756

*** = Statistically significant**

Table A.III.8.55 **Multiple regression statistics for active right rotation (ROM) and predictor variables- week-52.**

Source of variance- wk 52 rot	df	Ss	Ms	F	p
Attributable to regression	4	5018.10	1254.53	5.64	.0004*
Residual	110	24461.46	222.38		

*** = Statistically significant**

Table A.III.8.56 **Prediction of change in active right rotation (ROM) in relation to the level of pain at week-52, from age, and type of treatments.**

Variable 52 rot	B	SEB	Standardised coeff	T	p
Age	0.04	0.11	0.04	0.38	.7037
Active exercise regimen	-7.56	3.64	-20.00	-2.07	.0406*
Pain	-0.54	0.69	-0.09	-0.79	.4305
Immobilisation regimen	8.03	3.97	0.24	2.02	.0457*

*** = Statistically significant**

Table A.III.10.1 Number of subjects reporting back pain and frequency of pain scale for each of the time periods.

Time period	Pain scale	Frequency Right side	%	Frequency Left side	%
Initial Examination n=220	0	139	63.2	140	63.6
	20	4	1.8	5	2.3
	30	5	2.3	4	1.8
	40	11	5.0	10	4.5
	50	22	10	23	10.5
	60	20	9.1	20	9.1
	70	8	3.6	7	3.2
	80	11	5.0	11	5.0
Week-4- n=162	0	93	57.4	95	58.6
	20	2	1.2	1	0.6
	30	4	2.5	4	2.5
	35	0	0	1	0.6
	40	11	6.8	12	7.4
	50	20	12.3	19	11.7
	60	24	14.8	23	14.2
	70	6	3.7	5	3.1
	80	2	1.2	2	1.2
Week-6- n=151	0	94	62.3	93	61.6
	20	1	0.7	1	0.7
	30	7	4.6	8	5.3
	40	12	7.9	11	7.3
	50	15	9.9	14	9.3
	60	19	12.6	22	14.6
	70	3	2.0	2	1.3
Week-12- n=149	0	93	62.4	94	63.1
	10	1	0.7	-	-
	20	-	-	1	0.7
	30	11	7.4	8	5.4
	40	11	7.4	9	6.0
	50	19	12.8	23	15.4
	60	11	7.4	12	8.1
	70	2	1.3	1	0.7
	80	1	0.7	1	0.7
Week-24- n=122	0	83	68.0	82	67.2
	20	3	2.5	3	2.5
	30	3	2.5	3	2.5
	40	9	7.4	9	7.4
	50	10	8.2	10	8.2
	60	13	10.7	14	11.5
	80	1	0.8	1	0.8
Week-52- n=115	0	85	73.9	85	73.9
	30	1	0.9	1	0.9
	40	8	7.0	8	7.0
	50	8	7.0	8	7.0
	60	10	8.7	10	8.7
	70	2	1.7	2	1.7
	80	1	0.9	1	0.9

Table A.III.10.2 Frequency of activities of daily living causing discomfort -Week-4.

Activities - (n= 155)	FREQUENCY	%
Turning head	103	66.5
General Home duties	98	63.2
Lifting arms above the head	87	56.1
Driving car	72	46.5
Prolonged standing (LBP)	50	32.3
Lifting pots and pans	47	30.3
Prolonged bending forward	42	27.1
Making bed	41	26.5
Carrying weights up to -5 kgs	39	25.2
Vacuuming carpet	30	19.4
Washing hair	29	18.7
Hanging clothes	28	18.1
Reading longer than 5-15 minutes	17	11.0
Ironing	18	11.6
Reading longer than15-30 minutes	14	9.0
Sexual intercourse	13	8.4
Totally unable to dirve a car	13	8.4

Table A.III.10.3 Frequency of activities of daily living causing discomfort -Week-6.

Activity - (n=139)	Frequency	%
General Home duties	90	64.7
Turning head	72	51.8
Lifting arms above the head	65	46.8
Driving car	64	46.0
Prolonged standing (LBP)	47	33.8
Prolonged bending forward	39	28.1
Carrying weights up to -5 kgs	35	25.2
Lifting pots and pans	34	24.5
Gardening	29	20.9
Vacuuming carpert	28	20.1
Carrying washing	25	18.0
Hanging clothes	19	13.7
Sexual intercourse	18	12.9
Washing hair	16	11.5
Ironing	16	11.5
Reading longer than15-30minutes	16	11.5
Sporting activites	13	9.4
Reading longer than 5-15 minutes	12	8.6

Table A.III.10.4 Frequency of activities of daily living causing discomfort -Week-12.

Activities- (n=123)	FREQUENCY	%
General Home duties	66	53.7
Prologed standing (LBP)	60	48.8
Turning head	56	45.5
Prologed bending forward	55	44.7
Lifting arms above the head	48	39.0
Driving car	43	35.0
Gardening	28	22.8
Lifting pots & pans	26	21.1
Carrying weights up to -5 kgs	24	19.5
Carrying washing	21	17.1
Vacuuming carpet	20	16.3
Hanging clothes	19	15.4
Sexual intercourse	14	11.4
Reading longer than15-30 minutes	12	9.8
Sporting activities	11	8.9
Washing hair	11	8.9
Reading longer than 1/2- 1 hour	10	8.1
Ironing	10	8.1
Carrying loads up to 10 kgs	9	7.3
Chewing	9	7.3
Writing longer than 15 minutes	9	7.3

Table A.III.10.5 Frequency of activities of daily living causing discomfort-Week-24.

ACTIVITY- (n= 98)	FREQUENCY	%.
General Home duties	47	48.0
Prolonged standing (LBP)	40	40.8
Prolonged bending forward	39	39.8
Turning head	38	38.8
Driving car	33	33.7
Lifting arms above the head	28	28.6
Vacuuming carpet	21	21.4
Gardinging	17	17.3
Lifting pots & pans	17	17.3
Carrying loads up to 10 kgs	16	16.3
Carrying washing	14	14.3
Sporting activities	12	12.2
Washing hair	10	10.2
Repeated lifting of loads 10 kgs+	10	10.2
Hanging clothes	10	10.2
Writing longer than15- 30 minutes	9	9.2
Reading longer than 1/2 - 1hour	9	9.2
Reading longer than15-30 minutes	8	8.2
Sexual intercourse	7	7.1
Ironing	7	7.1

Table A.III.10.6 Frequency of activities of daily living causing discomfort -Week-52.

ACTIVITY- (n= 82)	FREQUENCY	%.
General Home duties	44	53.7
Turning head	40	48.8
Prolonged standing (LBP)	38	46.3
Prolonged bending forward	28	34.1
Lifting arm above head	28	34.1
Driving car	28	34.1
Lifting pots & pans	18	22.0
Carrying loads up to 5 kgs	22	26.8
Carrying washing	14	17.1
Vacuuming carpet	12	14.6
Gardening	12	14.6
Sporting activites	10	12.2
Hanging clothes	9	11.0
Ironing	9	11.0
Gardning	7	8.5
Washing hair	7	8.5
Repeated liftting 10 kgs+	7	8.5
Sexual intercourse	5	6.1

Table A.III.10.7 Results of Multiple Regression model to establish the association between headache and predictor variables.

Headache vs Covariate	Reg coeff	Wald	p	Odds Ratio	% prediction of headache
Headache vs Own speed	-.0053	.656	.4180	.9947	83.56
Headache vs Other speed	.0085	1.7524	.1856	1.008	83.56
Headache vs Own speed	-.0036	.1753	.68	.99	83.25
and type of collision grouped	.0157	.002	.96	1.01	83.25
Headache vs Other speed	.0071	.8351	.3618	1	83.25
and type of collision grouped	.0096	.0009	.98	1	83.25
Headache vs Side impact	.08	.02	.89	1.1	85.56
Headache vs Front end collision	.44	.66	.42	1	85.36
Headache vs Rear end collision	.19	.12	.73	1.2	85.36

APPENDIX-IV CLINICAL TRIAL DOCUMENTS

SOFT TISSUE NECK INJURY CLINIC
ROYAL PERTH(REHAB) HOSPITAL
SHENTON PARK

Trial No...../Random gp.....

☐☐ 1-2

☐☐☐☐☐☐☐☐ 3-10

☐☐☐ 11-13

☐ 14

G.P.....
.....
.....
Phone:.....

NAME STICKER

DATE...../...../.....

15-20

☐☐☐ 21 - 23

☐☐☐ 24 - 26

Phone: Work.....Home.....

D.O.B / /
27-32

GENDER

M / F
33 / 34

OCCUPATION (21-23) .....

D.O.A...../...../.....

35-40

HOBBY(24-26) .....

TIME OF ACCIDENT

.....

41-44

1.Approximate speed of vehicle in K.P.H:

Self..... 45 - 47

Other..... 48 - 50

2.Make & Model of Vehilcles:

Self..... 51 - 52

Other..... 53 - 54

3.Momentum Low/High (=1/2) 55

4. **1**.Driver **2**.Passenger **Front** **3**.Passenger **Rear RT**
4.Passenger **Rear Lt** **5**.Passenger **Rear Middle**

56

5. Head rest - Yes /No 57

6.Height of Head rest: (Please use the diagrams to answer the following question)

Adequate (diag.1)

Not adequate(diag.2) 58

7.At the time of the accident you were

wearing a SASH / LAP belt (1)

wearing a LAP belt (2)

Not wearing a seat belt (3) 59

8.Type of collision: Rear End (1)

Head on (2)

Side (3)

Pile up (4)

Frontal (5)

Others (6) ☐☐ 60 - 65

Specify:.....

.....

9.Were you aware of the impending accident?

yes /no ☐ 66

10.Were you unconscious after the accident?

yes /no ☐ 67

11.If yes for how long? Mins ☐☐ 68 - 69

12. Within 24 hrs after the accident did you experience any of the following?

Nauseayes /no ☐ 70

Head ache yes /no ☐ 71

Blured vision yes /no ☐ 72

Double vision yes /no ☐ 73

Black spots in the visual field

yes /no ☐ 74

Difficulty in focusing your eyes

yes /no ☐ 75

Pain behind the eyes

yes /no ☐ 76

Dizziness yes/ no ☐ 77

Specify:

.....

.....

.....

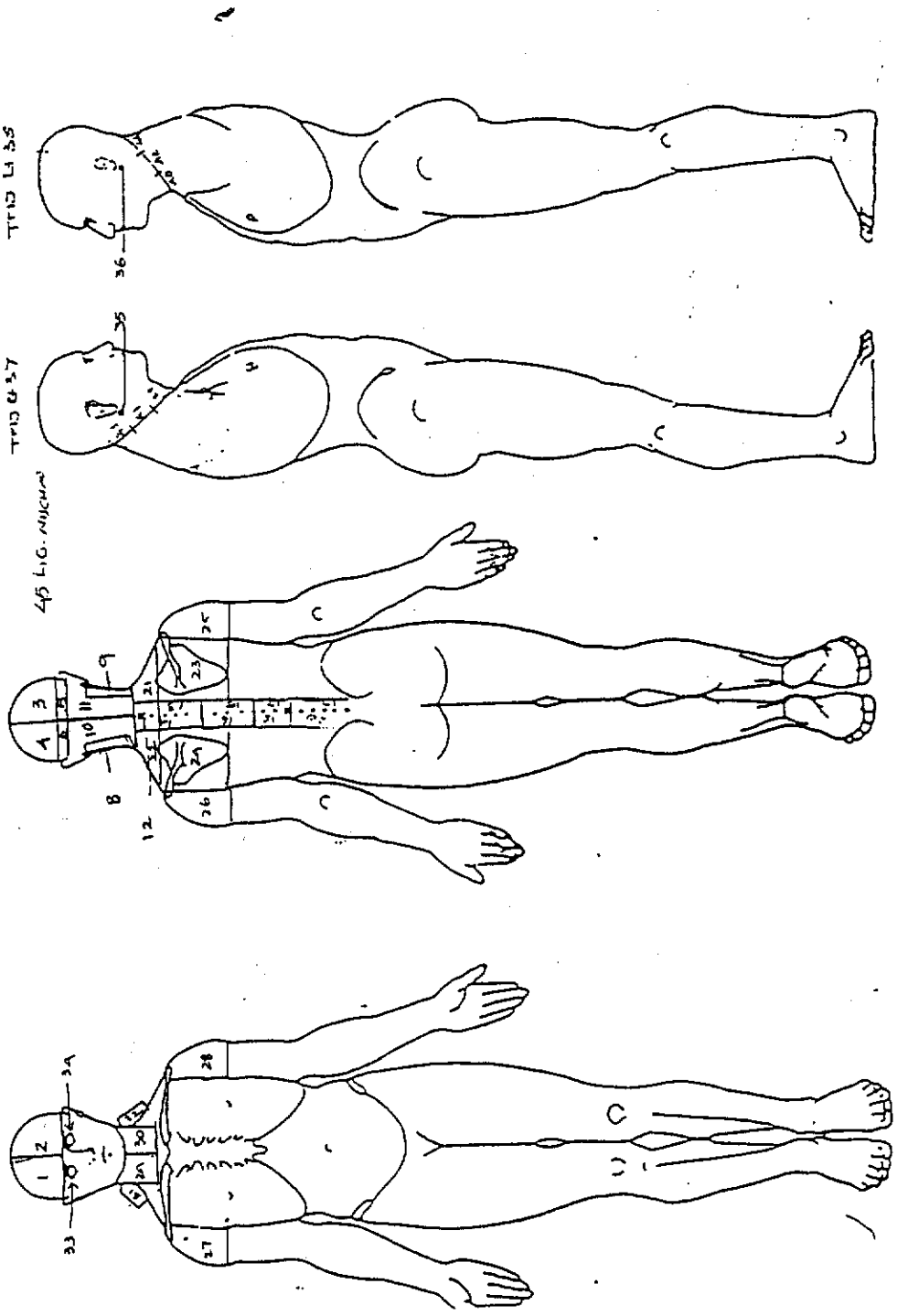
.....

.....

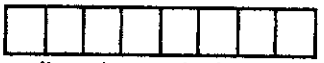
.....

Yes=1, No=2, Yes(Right)=3, Yes(Left)=4

13. Please use the body diagram (Chart 1.) to show pain or pins and needles sensation or numbness that you noticed within 24 hours after the accident:



 1-2

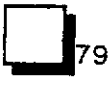
 3-10

14. Please use the following ***expressions*** to describe the painthat you ***experienced(chart 1)*** and how ***severe*** it was at that time.
(Please read explanation of 0 - 100 pain scale from the chart).

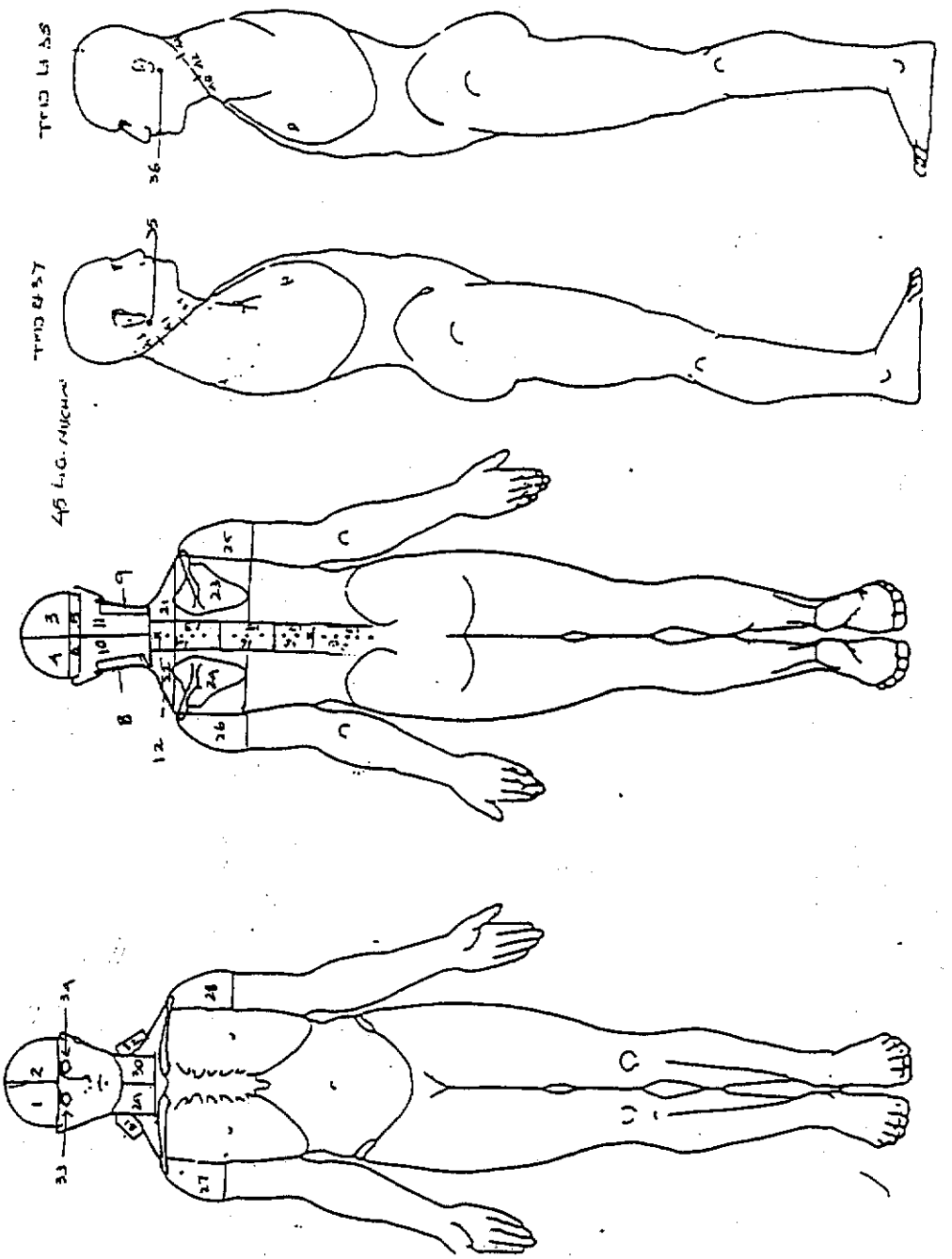
1.Dull ache ***2.Deep ache*** ***3.Sharp pain***

4.Throbbing pain ***5.Stabbing pain*** ***6.Tooth ache***

7.Burning pain ***8 .Stretching pain***

AREA		DESCRIPTION OF PAIN		INTENSITY	
	11 - 12		13		14 - 16
	17 - 18		19		20 - 22
	23 - 24		25		26 - 28
	29 - 30		31		32 - 34
	35 - 36		37		38 - 40
	41 - 42		43		44 - 46
	47 - 48		49		50 - 52
	53 - 54		55		56 - 58
	59 - 60		61		62 - 64
	65 - 66		67		68 - 70
	71 - 72		73		74 - 76
	77 - 78		79		80 - 82

15.CURRENT SYMPTOMS: Please use the body diagram (Chart 2) to show either pain or pins and needles sensation or numbnessthat you might have at present.



1-2

3-10

16. Please use the following expressions to describe the pain experienced by you (Chart 2) and how severe it is (Please read the explanation of 0-100 pain scale from the chart)

1. Dull ache ***2.Deep ache*** ***3.Sharp pain***

4.Throbbing pain ***5.Stabbing pain*** ***6.Tooth ache***

7.Burning pain ***8.Stretching pain***

AREA	DESCRIPTION OF PAIN	INTENSITY
11 - 12	13	14 - 16
17 - 18	19	20 - 22
23 - 24	25	26 - 28
29 - 30	31	32 - 34
35 - 36	37	38 - 40
41 - 42	43	44 - 46
47 - 48	49	50 - 52
53 - 54	55	56 - 58
59 - 60	61	62 - 64
65 - 66	67	68 - 70
71 - 72	73	74 - 76
77 - 78	79	80.-.82


1-2


3-10

17. Please indicate how often you have been noticing the pain and pins and needles sensation that you have shownin the body diagram .2 ?

(Please choose one of the following expressions to answer this question)

1. All the time ***2.Most of the time***

3.Now and then ***4.After moving the part***

5.Occasionally ***6.All the time made worse by movement***

7.Bending the head forward for a long time(15 minutes +)

AREA of pain	***Frequency***	***AREA ofP&N***	***Frequency***
1 1- 12	13	14- 15	16
17- 18	19	20 - 21	22
23 - 24	25	26 - 27	28
29 - 30	31	3 2 - 33	34
35 - 36	37	38 - 39	40
41 - 42	43	44 - 45	46
47 - 48	49	50 - 51	52
53 - 54	55	56 - 57	58
59 - 60	61	62 - 63	64
55 - 56	57	58 - 59	60
71 - 72	73	74 - 75	76
77 - 78	79	80 - 81	82

Others specify:

.....

.....

.....

☐☐ 1-2

☐☐☐☐☐☐☐☐ 3-10

18. Are you suffering from any of the following:

Nausea Yes / No ☐ 11

Blured vision Yes / No ☐ 12

Double vision Yes/ No ☐ 13

Black spots in the visual field

Yes / No ☐ 14

Difficulty in focusing your eyes

Yes / No ☐ 15

Dizziness Yes / No ☐ 16

Loss of balance Yes / No ☐ 17

Ringing in the ear Yes / No ☐ 18

Pain behind the eyes Yes/ No ☐ 19

Specify:

.....

.....

.....

Yes=1, No=2, Yes(Right)=3, Yes(Left)=4

19. Do any of the following increase your pain or pins and needles sensation?

	AREA OF PAIN		PINS&NEEDLES	
LOOKING UP	☐☐☐☐☐	20 - 24	☐	25
LOOKING DOWN	☐☐☐☐☐	26 - 30	☐	31
TURNING HEAD-RT	☐☐☐☐☐	32 - 36	☐	37
TURNING HEAD-LT	☐☐☐☐☐	38 - 42	☐	43

20. What do you do to ease your pain?

Resting in collor ☐ 44

Pain killers ☐ 45

Lying in bed ☐ 46

Other ☐ 47

SPECIFY.....

21. Is your sleep affected? y / n ☐ 48

22. Are you RIGHT HANDED (1) OR *LEFT HANDED (2)* ? ☐ 49

23. Are you working in your usual job now? ☐ 50

Yes=1, No=2

24. Indicate those activities you are totally unable to do now or having difficulty due to pain

1.....	☐	☐ 51 - 52
2.....	☐	☐ 53 -54
3.....	☐	☐ 55 - 56
4.....	☐	☐ 57 - 58
5.....	☐	☐ 59 - 60
6.....	☐	☐ 61 - 62
7.....	☐	☐ 63 - 64
8.....	☐	☐ 65 - 66
9.....	☐	☐ 67.- 68
10.....	☐	☐ 69 - 70

25. Medication:

1.....	☐ 71
2.....	☐ 72
3.....	☐ 73
4.....	☐ 74

1-2

3-10

26.Observations:

1.THIN NECK 2.LONG NECK

3.SHORT NECK 4.STOUT NECK 17-18

OTHERS .....

.....

27.Postural Abnormality:

1.Poked chin & Bull Neck 2.Lordotic

3.Antalgic Pattern 19-20

Specify .....

.....

28.Passive Movement Testing of OC- C1

(in sitting position)

Remarks.....

.....

Pain Score in NRS(0 - 100) 21-23

29. Active Movement Testing of OC-C1

(in sitting position)

Remarks.....

.....

Pain Score in NRS 24-26

1-2

3-10

30. *RANGE OF MOTION*

MOVEMENT	ROM	AREA OF PAIN	NRS	DESCRIPTION
FLEXION	ACT. 11	12-13	14-16	17
	PAS. 18	19-20	21-23	24
EXTENSION	ACT 25	26-27	28-30	31
	PAS 32	33-34	35-37	38
SIDE FLEXION RT	ACT 39	40-41	42-44	45
	PAS. 46	47-48	49-51	52
SIDE FLEXION LT	ACT 53	54-55	56-58	59
	PAS 60	61-62	63-65	66
ROTATION RT	ACT 67	68-69	70-72	73
	PAS 74	75-76	77-79	80

MOVEMENT	ROM	AREA OF PAIN	NRS	DESCRIPTION
ACT				
ROTATION LT	81	82-83	84-86	87
PAS				
	88	... 89-90	91-93	94

1-2

3-10

31 **NEUROLOGY**

LEVEL	MOTOR POWER		SENSORY CHANGES	
	RT	LT	RT	LT
C2	11	12	13	14
C3	15	16	17	18
C4	19	20	21	22
C5	23	24	25	26
C6	27	28	29	30
C7	31	32	33	34
C8	35	36	37	38
T1	39	40	41	42

KEY FOR SENSORY TESTS:

0=NORMAL 1=HYPOSTHESIA 2=PARAESTHESIA 3=ANAESTHESIA TO LIGHT TOUCH

4=ANAESTHESIA TO TEMPERATURE 5=SHARP / BLUNT ALTERED 6=HYPERAESTHESIA

32. VIBRATORY SENSATION

	ALTERED Y/N RT	LT	
Jaw	☐	☐	43 - 44
Acromion	☐	☐	45 - 46
Med epicondyle	☐	☐	47 - 48
Olecronon	☐	☐	49 - 50
RadialStyloid	☐	☐	51 - 52
Ulnar Head	☐	☐	53 - 54
1st MC Head	☐	☐	55 - 56
5th MC Head	☐	☐	57 - 58

33.Reflexes: (FULL=3 DEPRESSED=2 ABSENT=0)

	RIGHT	LEFT	
Biceps	☐	☐	59 - 60
Triceps	☐	☐	61 .- 62
Brach Rad	☐	☐	63 - 64

34.Horner's (+ve= 1) (- ve = 2)

☐ ☐ 65 - 66

35.Light reflex (+ve= 1) (- ve = 2)

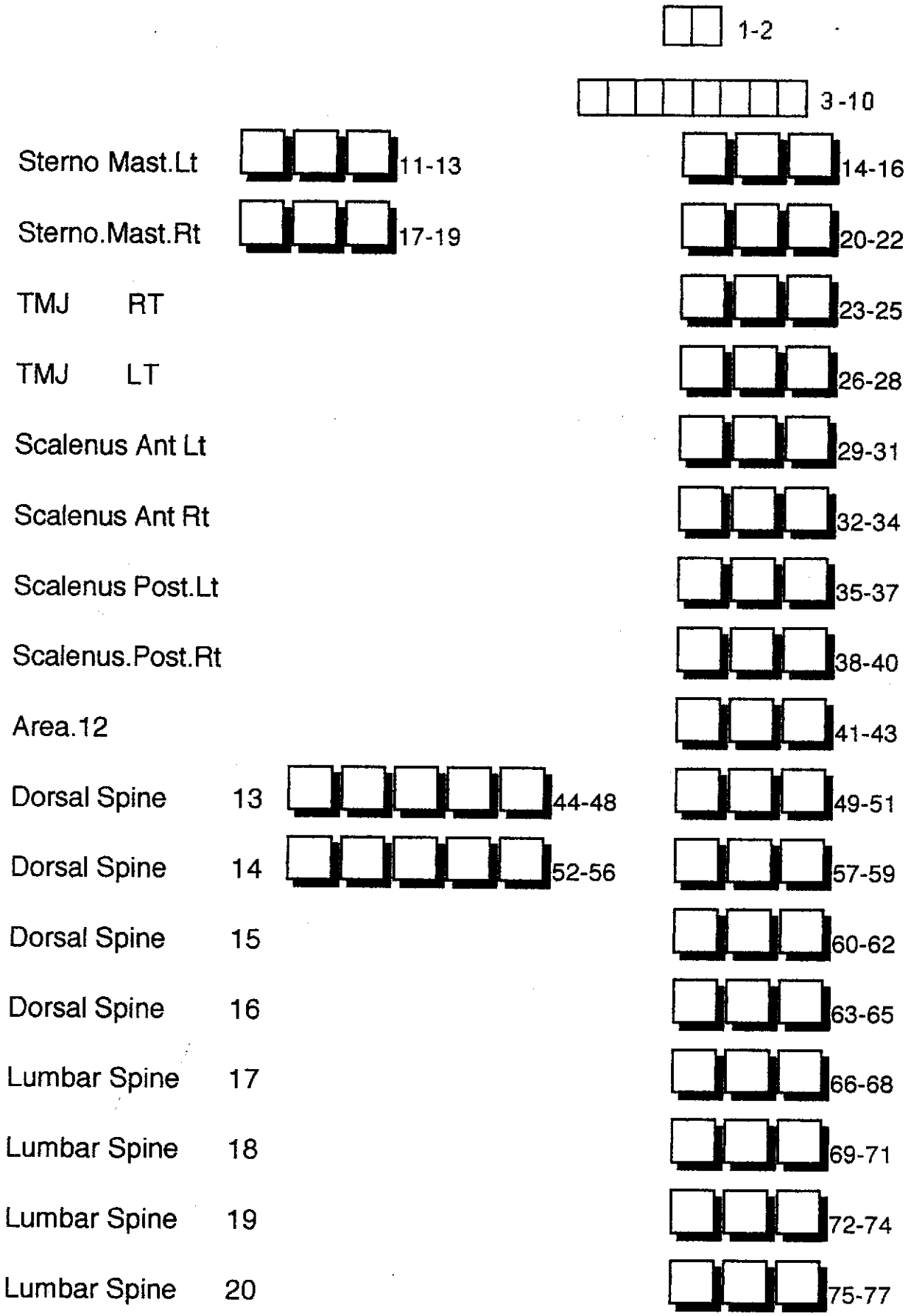
☐ ☐ 67 - 68


1-2


3-10

36.PALPATION OF SOFT TISSUES:

	LEVELS	*NRS*
Lig. Nuchae	11-15	16-18
Area 8	19-23	24-26
Area.9	27-31	32-34
Area.10	35-39	40-42
Area.11	43-47	48-50
Nuchal Line	51-55	56-58
Area.21		59-61
Area.22		62-64
Area.29		65-67
Area.30		68-70
Area.31		71-73
Area.32		74-76
Levator Scap LT		77-79
Levator Scap.RT		80-82


1-2
3-10
Sterno Mast.Lt
11-13
14-16
Sterno.Mast.Rt
17-19
20-22
TMJ RT
23-25
TMJ LT
26-28
Scalenus Ant Lt
29-31
Scalenus Ant Rt
32-34
Scalenus Post.Lt
35-37
Scalenus.Post.Rt
38-40
Area.12
41-43
Dorsal Spine 13
44-48
49-51
Dorsal Spine 14
52-56
57-59
Dorsal Spine 15
60-62
Dorsal Spine 16
63-65
Lumbar Spine 17
66-68
Lumbar Spine 18
69-71
Lumbar Spine 19
72-74
Lumbar Spine 20
75-77

1-2

3-10

37. Radiology

Diagnostic Yes / No 11

LEVEL(S)

1. Normal Lordosis y / n 12

2. Normal Alignment y / n 13

3. Widening of interspinous space y / n 14 15-18

4. Instability y / n 19 21-23

5. Flexion Limitation y / n 24 25-28

6.. Prevertebral Swelling y / n 29 30-33

7. Degenerative Changes y /n 34 35-38

Remarks.....

.....

.....

8. Tomograms.....

.....

.....

.....

38.MRI.....

.....

.....

.....

.....

1-2

3-10

39. Previous Medical History Relevant to Cervical Region

11-12

13-14

15-16

17-18

19-20

21-22

1-2

3-10

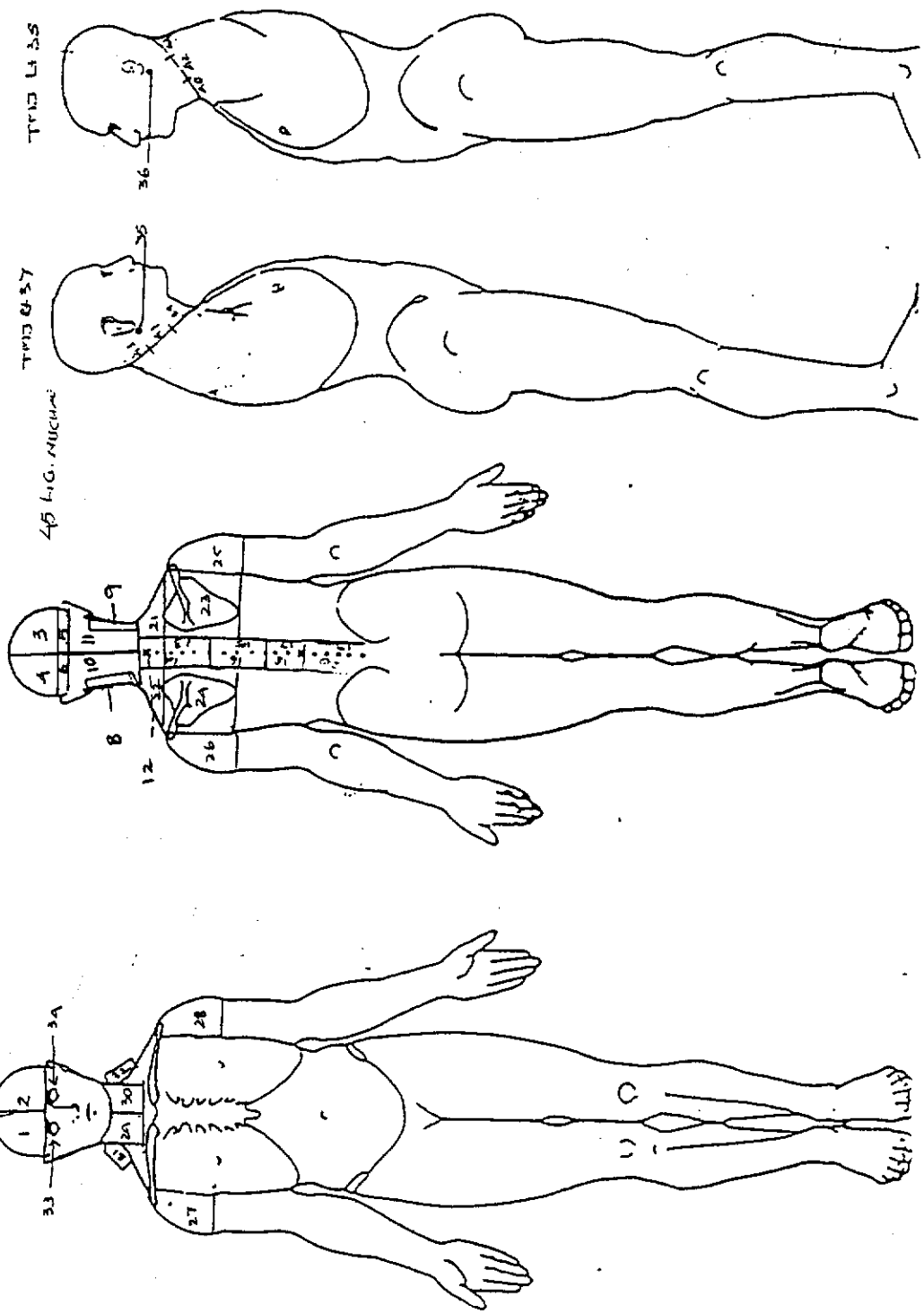
FOLLOW-UP

1. Are you suffering from any of the following:

Nausea	Yes / No	11
Blured vision	Yes / No	12
Double vision Yes / No		13
Black spots in the visual field yes / No		14
Difficulty in focusing your eyes Yes / No		15
Dizziness Yes / No		16
Loss of balance Yes / No		17
Ringing in the ear Yes/No		18
Difficulty in concentrating Yes/No		19
Dropping Objects (Clumsiness) Yes/No		20
Pain Behind the eyes Yes/No		21

Yes=1, No=2, Yes(Right)=3, Yes(Left)=4

2.Please use the body diagram (Chart 3.) to show pain or pins and needles sensation or numbness that you have noticed in the past few days.



 1-2

 3-10

3.Please use the following ***expressions*** to describe the pain that you have shown ***in the chart*** and its severity.

(Please read explanation of 0 - 100 pain scale from the chart).

1 Dull ache ***2.Deep ache*** ***3.Sharp pain***

4.Throbbing pain ***5.Stabbing pain*** ***6.Tooth ache***

7.Burning pain ***8 .Stretching pain***

AREA	DESCRIPTION OF PAIN		INTENSITY	
	11 - 12	 13		14 - 16
	17 - 18	 19		20 - 22
	23 - 24	 25		26 - 28
	29 - 30	 31		32 - 34
	35 - 36	 37		38 - 40
	41 - 42	 43		44 - 46
	47 - 48	 49		50 - 52
	53 - 54	 55		56 - 58
	59 - 60	 61		62 - 64
	65 - 66	 67		68 - 70
	71 - 72	 73		74 - 76
	77 - 78	 79		80 - 82


1-2


3-10

4 Please indicate how often you have been noticing the pain and pins and needles sensation that you have shownin the body diagram. *(Please choose one of the following expressions to answer this question)*

1. All the time 2.Most of the time
3.Now and then 4.After moving the part
5.Occasionally 6.All the time made worse by movement
7.Bending the head forward for a long time(15 minutes +)

AREA of pain	***Frequency***	***AREA ofP&N***	***Frequency***
1 1- 12	13	14- 15	16
17- 18	19	20 - 21	22
23 - 24	25	26 - 27	28
29 - 30	31	3 2 - 33	34
35 - 36	37	38 - 39	40
41 - 42	43	44 - 45	46
47 - 48	49	50 - 51	52
53 - 54	55	56 - 57	58
59 - 60	61	62 - 63	64
55 - 56	57	58 - 59	60
71 - 72	73	74 - 75	76
77 - 78	79	80 - 81	82

1-2

3-10

5. *RANGE OF MOTION*

MOVEMENT	ROM	AREA OF PAIN	NRS	DESCRIPTION
FLEXION	ACT.	11	12-13 14-16 17	
	PAS.	18	19-20 21-23	24
EXTENSION	ACT	25	26-27 28-30	31
	PAS	32	33-34 35-37	38
SIDE FLEXION RT	ACT	39	40-41 42-44	45
	PAS.	46	47-48 49-51	52
SIDE FLEXION LT	ACT	53	54-55 56-58	59
	PAS	60	61-62 63-65	66
ROTATION RT	ACT	67	68-69 70-72	73
	PAS	74	75-76 77-79	80

MOVEMENT	ROM	AREA OF PAIN	NRS	DESCRIPTION
ACT				
ROTATION LT	81	82-83	84-86	87
PAS				
	88	... 89-90	91-93	94

1-2

3-10

6 ***NEUROLOGY***

LEVEL	***MOTOR POWER***		***SENSORY CHANGES***	
	RT	***LT***	***RT***	***LT***
C2	11	12	13	14
C3	15	16	17	18
C4	19	20	21	22
C5	23	24	25	26
C6	27	28	29	30
C7	31	32	33	34
C8	35	36	37	38
T1	39	40	41	42

KEY FOR SENSORY TESTS:

0=NORMAL 1=HYPOSTHESIA 2=PARAESTHESIA 3=ANAESTHESIA TO LIGHT TOUCH

4=ANAESTHESIA TO TEMPERATURE 5=SHARP / BLUNT ALTERED 6=HYPERAESTHESIA

7 . VIBRATORY SENSATION

	ALTERED RT	ALTERED LT	Y/N
Jaw	☐	☐	43 - 44
Acromion	☐	☐	45 - 46
Med epicondyle	☐	☐	47 - 48
Olecronon	☐	☐	49 - 50
RadialStyloid	☐	☐	51 - 52
Ulnar Head	☐	☐	53 - 54
1st MC Head	☐	☐	55 - 56
5th MC Head	☐	☐	57 - 58

8.Reflexes: (FULL=3 DEPRESSED=2 ABSENT=0)

	RIGHT	LEFT	
Biceps	☐	☐	59 - 60
Triceps	☐	☐	61 .- 62
Brach Rad	☐	☐	63 - 64

9.Horner's (+ve= 1) (- ve = 2)

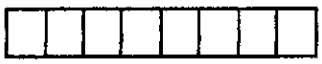
☐ ☐ 65 - 66

10.Light reflex (+ve= 1) (- ve = 2)

☐ ☐ 67 - 68

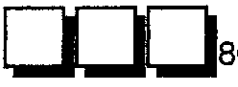

1-2

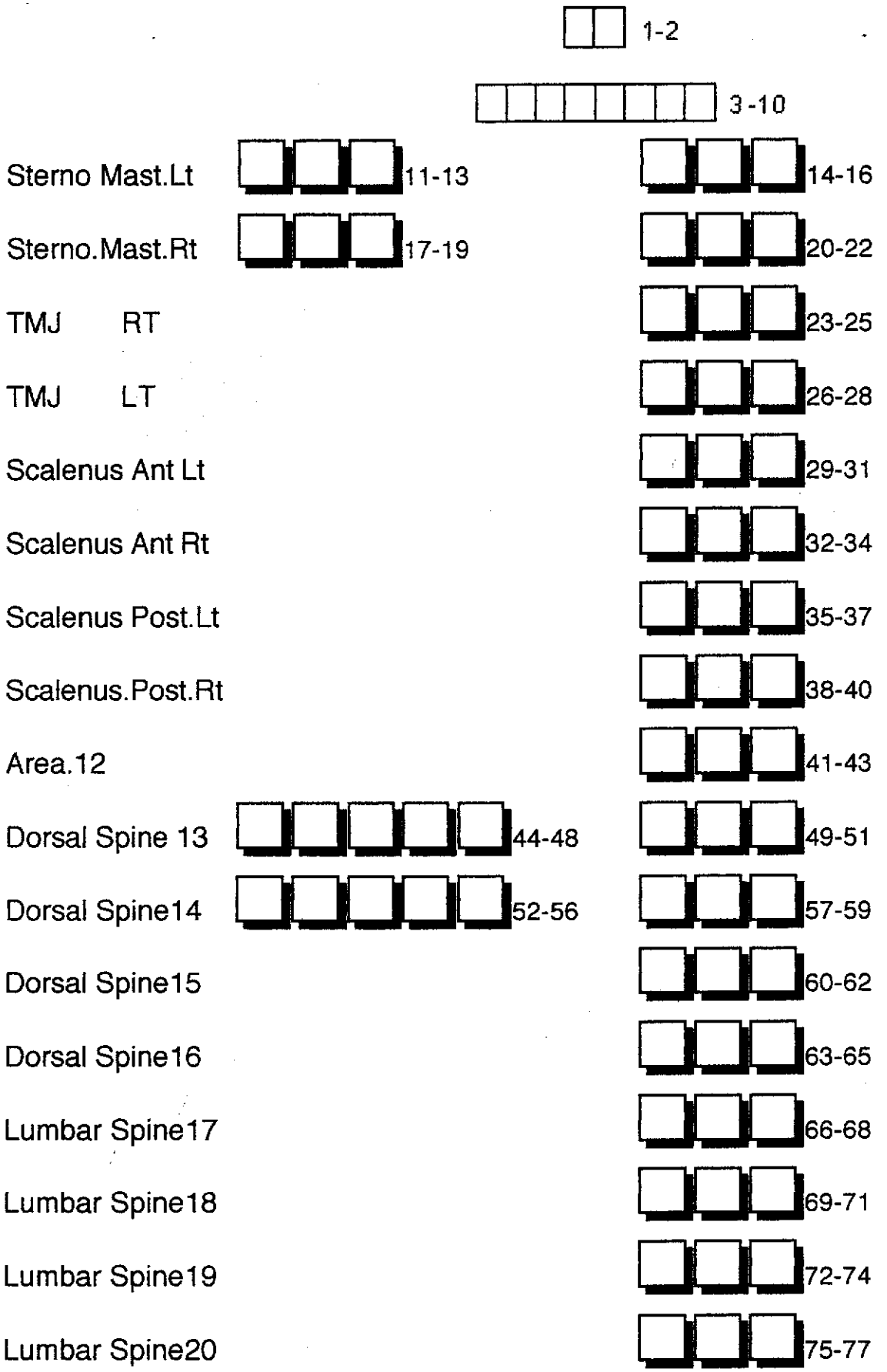
11


3-10

PALPATION OF SOFT TISSUES:

	LEVELS		*NRS*	
Lig. Nuchae		11-15		16-18
Area 8		19-23		24-26
Area.9		27-31		32-34
Area.10		35-39		40-42
Area.11		43-47		48-50
Nuchal Line		51-55		56-58

Area.21		59-61
Area.22		62-64
Area.29		65-67
Area.30		68-70
Area.31		71-73
Area.32		74-76
Levator Scap LT		77-79
Levator Scap.RT		80-82


1-2
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Scalenus Post.Lt
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Dorsal Spine15
60-62
Dorsal Spine16
63-65
Lumbar Spine17
66-68
Lumbar Spine18
69-71
Lumbar Spine19
72-74
Lumbar Spine20
75-77

1-2

3-10

ISOMETRIC MUSCLE TESTING

OF THE PRE AND POST VERTEBRAL NECK MUSCLES

Tested Post Injuyry In weeks 11-12

.Pre Vertebral Muscles

PEAK FORCE IN kgs 13-14

Achieved at seconds 15-16

Maintained for a period of(secs) 17-18

Post Vertebral Muscles

PEAK FORCE IN kgs 19-20

Achieved at seconds 21-22

Maintained for a period of(secs) 23-24

☐☐ 1-2

☐☐☐☐☐☐☐☐ 3-10

13 .Indicate those activities you are totally unable to do now or having difficulty due to pain.

1..... ☐ ☐ 11 - 12

2..... ☐ ☐ 13 -14

3..... ☐ ☐ 15 - 16

4..... ☐ ☐ 17 - 18

5..... ☐ ☐ 19 - 20

6..... ☐ ☐ 21 - 22

7..... ☐ ☐ 23 - 24

8..... ☐ ☐ 25 - 26

9..... ☐ ☐ 27.- 28

10..... ☐ ☐ 29 - 30

14. Medication:

1..... ☐ 31

2..... ☐ 32

3..... ☐ 33

4..... ☐ 34

15 Duration of Back Pain in weeks ☐☐ 46-47

16 Length of Treatment required ☐☐ 48-49

17. RETURN TO WORK:

Normal Working Hours Per week ☐☐ 50-51

18. Resumed work weeks after injury ☐☐☐ 52-54

19. Resumed work:

Hours worked in % ☐☐☐ 55-57

Restricted duties y / n ☐ 58

Modified duties y / n ☐ 59

New occupation y / n ☐ 60

House Chores in % ☐☐☐ 61-63

Hobby in % ☐☐☐ 64-66

20 POINTS OF INTEREST:

SOFT TISSUE NECK INJURY CLINICAL TRIAL

PATIENT INFORMATION AND CONSENT FORM

PATIENT'S STICKER TRIALNO:.....

The effect of injury to the neck arising from motor vehicle accidents seem to be varied. In some patients pain and other discomfort arising from the injury are often felt for a prolonged period, affecting their life style.

These type of injuries are currently treated by different methods. In this clinical trial three different methods are chosen with a view to make comparisons between them. Patients in group 1 will wear a collar for a initial period of 4 weeks and then will commence a specific exercise programme. Patients in the group 2 will receive physiotherapy treatment in the form of specific exercises from the very beginning. Patients in the group 3 will receive treatment as decided by the family doctor. All these three methods are currently used in Australia. This clinical study has been approved by the Ethics Committee of the Royal Perth Hospital and Curtin University of Technology. Please note that the Ethics Committee is a body which is responsible to look after the interest of patients like yourself, who are admitted into research projects at this hospital.

If at anytime you wish to withdraw from the study, you are free to do so and this will not affect further treatment that you might require in any way. The tests used in this study are usually done as a routine and do not pose any threat to your well being.

The trial period covers 52 weeks from the time you are seen at the Royal Perth Rehabilitation Hospital. You will be required to attend a specially set up clinic at 4 weeks , 6 weeks, 12 weeks, 24 weeks and 52 weeks intervals. Each visit would approximately take 30 minutes. These visits will enable the research team to

arrange treatment after an initial examination and record your progress throughout the study period.

Your participation in this trial will be beneficial to numerous patients in our country and other parts of the world. We take this opportunity to thank you for your willingness to participate in this study.

ANY QUESTIONS CONCERNING THE PROJECT ENTITLED.
"A STUDY OF NECK INJURY ARISING FROM MOTOR VEHICLE ACCIDENTS AND ITS CLINICAL MANAGEMENT"
CAN BE DIRECTED TO MR NICK BATALIN, SPINAL SURGEON OF SPINAL UNIT, AT ROYAL PERTH (REHABILITATION) HOSPITAL ON

I.....(THE PARTICIPANT) have read the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this activity, realising that I may withdraw at any time.
I agree that research data gathered for the study may be published provided my name is not used.
Participant or Authorised Representative.....
Date.....
Investigator .....
Date.....

Note: For copyright reasons pages 448 and 449 of Appendix-IV, which contain letters, have not been reproduced.

(Co-ordinator, ADT Project (Retrospective), Curtin University of Technology, 25.11.02)