CARDIOVASCULAR RESPONSES IN PATIENTS WITH CERVICAL SPONDYLOSIS DURING TRACTION AND MANAGEMENT OF CERVICAL SPONDYLOTIC PAIN.

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B.Sc. (Physiotherapy), M.Sc. (Anatomy)

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THESIS SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES, UNIVERSITY OF LAGOS, IN FULFILLMENT OF THE REQUIREMENT FOR THE AWARD OF THE DEGREE OF DOCTOR OF PHILOSOPHY (PH. D.) IN THE DEPARTMENT OF ANATOMY, COLLEGE OF MEDICINE, OF THE UNIVERSITY OF LAGOS, NIGERIA.

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SCHOOL OF POSTGRADUATE STUDIES UNIVERSITY OF LAGOS

CERTIFICATION

This is to certify that the Thesis
SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES
UNIVERSITY OF LAGOS
For the award of the degree of

DOCTOR OF PHILOSOPHY (Ph.D.)

Is a record of original research carried out by

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In the
DEPARTMENT OF ANATOMY
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DEDICATION

TO

The Almighty GOD Through JESUS CHRIST My Redeemer,
For Wisdom, Knowledge, Understanding, Unlimited Love And
Favour HE Bestowed on Me;

My Loving, Supporting And Caring Late Father & Dearest Mum, Who Laboured SO Much To Take Me Through The Rudiments of Live;

4.

And My Loving Children Temitope & Temidayo.

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TO GOD BE THE GLORY

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ABSTRACT

BACKGROUND & OBJECTIVES: Results from past studies have provided evidence that cervical traction is of benefit in the treatment of neck pain, but there is no consensus among clinicians regarding the amount of traction weight to be employed during treatment that will reduce the side effects (vertigo, nausea, mild headache, blurred vision and migraine) associated with this modality, if not properly administered.

The study was designed to investigate the cardiovascular responses and side effects associated with three different cervical traction weights [7.5% total body weight (TBW), 10% TBW and 15% TBW] and to establish the ideal cervical traction weight with minimal side effects and with optimal/highest therapeutic efficacy. This study was carried out in order to establish a safe and efficacious policy in cervical traction therapy.

METHODS: Nine hundred and forty seven (947) adult subjects (432 Men and 515 Women) with five hundred and forty one (541) of them patients with cervical spondylosis and four hundred and six (406) normal subjects (control) participated in the study. The cervical spondylosis subjects were patients with radiological and clinical features of spondylosis while the normal subjects are those that were screened and confirmed to be medically fit for the study.

At the end of the screening exercise, **105** patients and **120** normal subjects (a total of **225** subjects) met the criteria for the study. Sixty of the normal subjects were assigned for the pilot study while the other sixty were for the main study. One hundred and sixty five (**165**) subjects (**105** patients and **60** normal volunteers) participated in the main study. All prospective subjects were fully briefed of the experimental procedures before they volunteered to participate by signing an informed consent.

The main study was implemented in two stages; the **investigative** (**Experiment**One) and therapeutic (**Experiment Two**) stages successively.

Experiment ONE. The experiment investigated the cardiovascular responses, and side effects associated with the different cervical traction weights. One hundred and twenty (120) subjects participated, with 60 patients and 60 normal volunteers (control). The 120 subjects were assigned into 3 Groups; A = 7.5% TBW, B = 10% TBW and C = 15% TBW with 40 subjects (20 patients and 20 normal subjects) in each group. Cardiovascular variables [systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), rate pressure product (RPP), PR interval and QRS complex] in baseline (at rest) and at the end of 5, 10 and 15 minutes following traction application were evaluated and recorded. Data were analysed using descriptive and inferential statistic of t-test and ANOVA.

Experiment TWO. The study assessed the therapeutic efficacy of the three different cervical traction weights. Forty five (45) patients were recruited into this study and were divided into 3 groups of 15 patients in each group. Group D1= Treated with 7.5% TBW traction, thermal therapy (TT) and therapeutic exercise (TE); Group D2= Treated with 10% TBW traction, TT, and TE; Group D3= Treated with 15% TBW traction, TT, and TE. Pain intensity and cervical ROM were assessed Pre and Post treatment and subjected to statistical analysis using Kruskal Wallis test and Wilcoxon test for pain and ANOVA and t-test for neck ROM.

RESULTS:

Experiment One. ANOVA revealed no significant difference at P< 0.05 for the 7.5% in the SBP, DBP and RPP in both patients (A1) and normal subjects (A2) groups. Significant difference existed for the SBP, DBP and RPP in the 10% groups except for the DBP in the normal subject group (B2). A significant difference existed for the 15% groups (C1 & C2) for the SBP, DBP and the RPP.

The heart rate was relatively stable (not significant) throughout the traction periods in the three traction groups, also the traction effects were not statistically significant in terms of the selected ECG variables, that is, the QRS complex and PR interval.

This finding indicates that the cardiac muscles contractility was not adversely affected by any of the traction weights during treatment. Thirty subjects (20 patients and 10 normal subjects) experienced different side effects due to the application of the traction. This study also showed that age is a significant factor in cervical traction therapy as younger subjects recorded less side effects compared with the older ones.

Results from **Experiment Two** revealed that the three traction weights therapy resulted in pain relief and enhances better neck flexibility. But from the mean ranks, the group that was treated with the 10% TBW traction had the least mean rank, lowest pain rating and better neck mobility post treatment; hence 10% traction offered better therapeutic result compared with the 7.5% and 15% TBW tractions.

CONCLUSION: This study revealed that cardiovascular alterations do occur in patients and normal subjects during the application of cervical traction using 7.5%, 10% and 15% TBW traction weights. It also established scientifically the **10% TBW** cervical traction as the ideal weight with minimal side effects and with optimal/highest therapeutic efficacy.

Key Words: Cervical traction, Cervical spondylosis, cardiovascular variables, ECG, Neck Pain, Neck Mobility, Side Effects of Cervical Traction.

CHAPTER ONE

1.0 INTRODUCTION

1.1 INTRODUCTION AND BACKGROUND

Spondylosis (Spinal Osteoarthritis) is a degenerative disorder that may cause loss of normal spinal structure and function. Although aging is the primary cause, the location and rate of degeneration differs in different people. The degenerative process may affect the cervical, thoracic, and/or lumbar regions of the spine affecting the intervertebral disc (IVD) and facet joints. It is however known to be commonest in the mobile segment of the spine, that is, the cervical and lumbar regions (Tamura *et al.*, 1999). The main clinical features of spondylosis are spinal pain and neck stiffness/hypomobility (Yates, 1972; Akinbo, 1996; Shakoor *et al.*, 2002).

Pain is the sensory and emotional sensations that arise as a result of tissue injury/damage or disease and it has different qualities. It is variously described as cutting, stabbing, burning, boring, throbbing, gripping, shooting; it could also be continuous or momentary and may be aggravated by certain activities/actions, e.g. knee pain aggravated by weight bearing or ambulation (Merskey, 1991).

According to the International Association for the Study of Pain (IASP), 'pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage (Merskey, 1986). Pain is a complex phenomenon composed of sensory experiences that include time, space, intensity, emotion cognition and motivation. It is an unpleasant phenomenon that is uniquely experienced by each individual and which cannot be adequately defined, identified or measured by an observer (Merskey, 1986; Merskey, 1991).

Apart from spondylosis other causes of cervical pain (upper back pain), include inflamed lymph nodes, muscle strain and protrusion of the intervertebral disc (Swezey *et al.*, 1999; May, 2001). Most chronic cervical pain is caused by bony abnormalities as a result of cervical spondylosis or other forms of spinal pathology (spondylolysis or spondylolithesis), or by major trauma e.g. road traffic accident (RTA), plane crash or minor trauma e.g. sleeping in an awkward position (Thompson and Rowland, 1995). Cervical pain may be affected by movements of the head, upper limb, especially the shoulder and may be exaggerated during coughing, sneezing or straining during defecation (John and Tony, 2003).

Empirical studies and a review of the literature by Nwuga, (1995), Ross, (1997) and Di Fabio, (1999) provide evidence that spinal traction is of benefit in the treatment of spinal pain. Spinal traction could be in form of cervical or lumbar traction in clinical application (Saunders, 1983). Spinal traction is the

technique in which a pulling force is used to stretch soft tissues and to separate joint surfaces in the spinal column (Saunders, 1983; Judovick and Nobel, 1997). In other words, parts of the spinal column are "pulled" in opposite directions in order to stabilize or change the position of the damaged aspects of the spine or to decompress a nerve. Spinal traction is a form of manual therapy that is used in an effort to reduce pain and improve range of movement (Di-Fabio, 1999). It has been employed in the management of patients with head and neck disorders, including neck (cervical) pain and stiffness, muscle tension, headache and migraine (Kurutzne *et al.*, 2001; Costantoyannis *et al.*, 2002).

If properly and correctly applied, spinal traction strengthens the spinal curves, it is effective in loosening adhesions within the dural sleeves, improves circulation within the epidural space, reduces compression and irritation of the disc by enlarging the inter-vertebral foramina to relieve pressure on compromised nerve roots and can also promote back muscle relaxation (Judovick and Nobel, 1997).

Cervical traction (CT) applies a stretch to muscles, ligaments, and tissue components of the cervical spine. It provides relief by promoting separation of the intervertebral (IV) joint space, which contains the disc and may reduce a "bulge" or impingement of structures within the foramen (Shakoor *et al.*, 2002).

Cervical traction force is usually applied to the skull through a series of weights or a fixation device and requires that the patient is either kept in bed (supine/lying position) or placed in a halo vest i.e. sitting position (Zybergold and Piper, 1985; Valtonen and Kiuru, 1990; Nwuga, 1995).

Cervical traction may be applied continuously or intermittently. Continuous (static) traction is indicated for degenerated (spondylosis) related disorders and disc prolapsed, while intermittent traction is recommended for patients with neck muscle spasm or soft tissue injuries (trauma) to the spinal region (Jette, 1985).

Because of the proximity of the vertebral artery to the cervical articulations, caution must be taken during spinal traction, specifically cervical traction as it is thought that a cerebrovascular accident (CVA) can be induced as a result of mechanical compression or excessive stretching of the arterial wall (Di-Fabio, 1999). However, the pathogenesis of ischemia is unclear.

Previous studies have investigated the tractive force (kilogram weight) needed to cause measurable change in the cervical spinal structures and the ideal position of the patients during cervical traction treatment (Dects *et al.*, 1977; Nwuga, 1995; Judovick and Nobel, 1997). These studies revealed that a force of between 3.5kg – 18.2 kg (minimum – maximum range) is needed to effectively produce a separation of the cervical spine. For optimal results,

most clinicians recommend the supine position, with the head and neck maintained in $25^{\circ} - 35^{\circ}$ of flexion during treatment (Nwuga, 1976; Betge, 1981; Balogun *et al.*, 1990).

There is currently no consensus among clinicians regarding the amount of tractive force to be employed during cervical traction that will correlate precisely with the percentage body weight of the patient.

Nwuga, (1995), recommends an average weight of 11.4 kg (251b) administered for between 10-20 minutes. On the contrary, Gould, (1985), suggests that the initial tractive force should be limited to 3.5-6.8 kg (8 – 151b) and sustained for 5 minutes followed by 15 minutes of rest. Also Kisner and Colby, (1985), suggest that the initial treatment with cervical traction should not exceed 4.5-6.8 kg (10-151 b) and that the duration of treatment may last from 10-30 minutes. Maitland, (1977), believed that patients with cervical problems should be able to accept traction of between 8 kg-10 kg (221b) without discomfort or after effects, Colachis and Strohms, (1976), in their study asserted that 13.6 kg is the ideal traction load for cervical problems requiring cervical traction therapy.

1.2 Side Effects of Cervical Traction

Cervical traction is suspected to induce side-effects (if not properly applied or if excessive load is fitted) such as pain in the neck and arm (brachialgia), weakness not due to overexertion (lassitude), a sensation of lack of balance or equilibrium (vertigo), a feeling of impending vomiting (nausea), mild headache, blurred vision, and migraine. Most of these side effects suggest a perturbation of the patient's cardiovascular system (DI-Fabio, 1999; Ogino *et al.*, 2001; Likhaceav and Ermilor, 2002; Egwu *et al.*, 2003). These side effects have led some clinicians to consider cardiovascular or cardiorespiratory problems as a contraindication to cervical traction therapy, and have also reduced the frequency of its use despite its established benefits (Zhuang, 2000).

1.4 Statement Of Problem.

There is currently no consensus among clinicians regarding the amount of tractive force (traction weight) to be employed during cervical traction that will correlate precisely with the percentage body weight of the patient. In lumbar spondylosis, the lumbar traction force of 26% total body weight (TBW) has been established to be the ideal weight for effective pain reduction and normal range of movement at the lower back region (Borman *et al.*, 2003).

The most feared complication of cervical manipulation, vertebrobasilar artery dissection, can arise if the traction is not properly applied or if excessive load is applied (Di-Fabio, 1999). Because of the proximity of the vertebral artery to the cervical articulations, caution must be taken during manipulation of the cervical spine (spinal traction). It is thought that stroke can be induced as a result of cervical spine manipulation by mechanical compression or excessive stretching of the arterial wall (Di-Fabio, 1999).

Moreover, anecdotal reports indicate that patients, following cervical traction, may experience vertigo, nausea, mild headache, and stroke induced by vertebral artery damage (Nwuga, 1995; Young *et al.*, 2002; Egwu *et al.*, 2003) leading to the possibility of a perturbation of the patient's cardiovascular system (CVS). This has led some clinicians to consider cardiovascular or cardiorespiratory problems as a contraindication to cervical traction therapy (Di-Fabio, 1999). However, these acute effects of cervical traction on the cardiovascular system remain speculative and unquantified. It is therefore important to investigate the cardiovascular responses during cervical traction therapy in order to establish a safe and efficacious policy in cervical traction therapy.

1.5 Aims and Objectives Of Study.

- To investigate the cardiovascular responses and side effects associated with three different cervical traction weights on patients with cervical spondylosis and normal volunteers.
- To establish the ideal cervical traction weight with minimal side effects and with optimal/highest therapeutic efficacy.

1.6 Significance Of The Study

The knowledge provided by this study would help clinicians to;

- Decide the groups of cervical spondylosis patients that would benefit from cervical traction application and preventive measures against adverse effects associated with cervical traction therapy.
- Decide the ideal weight that would be beneficial to patients but with minimal side-effects and with emphasis on individual patients and their body weight.
- 3. This study would also help to establish the effects of cervical traction on normotensive (normal) subjects.

1.7 Operational Definition Of Terms.

Ankylosis Spondylitis: Immobility of the spinal joints as a result of disease condition

Annulus Fibrosus: The outer part of the intervertbral disc.

Arthritis: Diseases of the joints, characterized by pain, swelling and loss of functions of the affected joints.

Arteriosclerosis: (Hardening of the arteries). A variety of conditions caused by fatty deposits in the artery wall resulting in narrowing of the lumen.

Atherosclerosis: Thickening of the inner wall of the arteries, characterized by a narrowing caused by cholesterols rich plagues. It involves mainly the large arteries.

Atrophy: Wasting or decrease in the size (diameter) of the muscles as a result of disease or disuse (non-use)

Cancer: A general disease term in which there is an uncontrolled, abnormal proliferation or growth of cells. Cancer cells can spread through the blood stream and lymphatic system to other part of the body.

Cardiac Arrest: Cessation of the action of the heart. It leads to loss of consciousness in a few seconds and death ensues if cardiac activity is not resumed, or some circulation restored by cardiac massage, within 2-3 min.

Cervical: Relating to the neck, there are seven cervical vertebrae.

spontaneous (non-traumatic) cerebral and subarachnoid haemorrhage, thrombosis and embolism.

Disc: meaning intervertebral disc (IVD). The IVD is the Fibrocartilagenous tissue between the bodies of successive vertebrae Electrocardiogram (ECG): A recording of the electrical activity of the heart. It aids in the diagnosis of heart disease.

Ergometer: A stationary bicycle for exercising and providing strength to the limb muscles. It can also be used to burn off excess fats. They are specially fitted with electronic devices that can be adjusted to assess power output and can assess muscular power.

Hip: (Innominate bone) bone frame formed by the fusion of ilium, ischium and pubis. It forms the pelvis with its fellow of the opposite side and the sacrum.

Hypertension: A condition present when blood flows through the blood vessel with a force greater than normal. It is elevated (high) blood pressure; often asymptomatic and increase the risk of heart attack, stroke and death **Ischemia:** Inadequate flow of blood to a part of the body, caused by constriction or blockage of the blood vessels supplying it.

Laminectomy: The surgical operation by which the lamina of a Vertebra is removed

NSAID (Non Steroid Anti inflammatory Drug): Pain relieving drug used in the treatment of arthritis e.g. Feldene, Oruvail, cataflan etc.

NSAID (Non Steroid Anti Inflammatory Drug): Pain relieving drug used in the treatment of arthritis e.g. Feldene, Oruvail, cataflan etc.

Normotensive: Describing the state in which the arterial blood pressure (BP) is within the normal range.

Nucleus Pulposus: The soft pulpy center of IVD. It disappears in old age.

Oophorectomy: Surgical removal of an ovary.

Osteoarthritis: Degenerative disease of the joints common in the weight bearing joints e.g. knee, hip, and the back.

Prolapsed intervertebral disc (PID): A "slipped disc " protrusion of the inner material of an IVD, causing pressure and compression on the nerve roots and ligaments resulting in severe pain.

Spondylolisthesis: Forward displacement of one vertebra upon the vertebra below, usually occurring between the 5th lumbar vertebra and the sacrum, and due either to injury or to a congenital defect.

Spondylolysis: The breaking down of a vertebra or loosening of the normally stable attachment existing between one vertebra and the next.

Spondylosis: Degenerative disease of the spine or Osteoarthritis of the back joints, common in the lumbar and cervical region, due to the mobility of the two regions.

Stroke: A sudden attack of weakness affecting one side of the body, due to an interruption to the flow of blood to the brain. It can also lead to paralysis, coma and death.

TENS (Transcutaneous Electric Never Stimulation):

TENS is the use of pulsed electrical stimulation through the skin to peripheral

nerve fibres for the management of pain. Melzack and Walls (1965) gate

control theory of pain provides one theoretical foundation for TENS therapy.

The theory proposes that electrical stimulation of the large, afferent high

velocity fibres prevents the smaller slow velocity pain-carrying "A" delta and

"C" fibres from transmitting pain signals, through the substantial gelatinosa of

the dorsal horn to the higher brain centres.

Thermal Therapy: Treatment of painful area using heat apparatus like infra

Red, microwave, short wave diathermy (SWD) etc.

Traction: The application of a pulling force as a means of relieving the

pressure and compression in the nerve root.

Vertigo: Dizziness

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CHAPTER TWO

2.0 LITERATURE REVIEW

2.1 Cervical Spondylosis

One common illness that is frequently misdiagnosed or undiagnosed is cervical spondylosis. Cervical spondylosis due to aging, degenerative changes, and dehydration of the disc spaces in the cervical spine results in a complex clinical picture (Dects *et al.*, 1977; Zaveri and Ford, 2001).

Usually cervical spondylosis is asymptomatic. It is not at all unusual to see all forms of osteophytes, desiccated disc spaces, narrowed disc spaces, and other degenerative changes in cervical spine X-rays of people who are 40 to 45 years and above. Spondylosis should not be called arthritis as there is no inflammation, it is simply nature's defence against drying up (desiccation) of joints and disc spaces due to aging. It usually does not cause any pain unless some form of trauma disrupts nature's fine balance (Dects *et al.*, 1977).

Certain diseases that accelerate aging make the patient more susceptible to the development of cervical spondylosis. These consist of alcohol abuse, which causes dehydration of the disc spaces, excessive cigarette smoking, oophorectomy with resultant early osteoporosis and bone degeneration, and repeated traumas e.g., repeated/recurrent soft tissue injuries, and hard labour. The vast majority of these osteophytes (bony overgrowths) do not cause any nerve problems (Shakoor *et al.*, *2002*), they are signs, however, that the disc between the vertebrae has become degenerative. Osteophytes, may however compress the spinal cord, a condition referred to as myelopathy.

Degeneration of discs can cause pain. The mechanism of pain perception is, unfortunately, not well understood. It is thought to be transmitted by tiny nerve endings that innervate the posterior aspect of the disc and facet joints (Tamura *et al.*, 1999). Degeneration can cause pain from the disc, the facet joint, or both concomitantly. Diagnostic efforts are aimed at determining which of these structures cause the pain and treatment is directed at relieving compression or stress placed on these areas. The sites commonly affected in cervical spondylosis according to Rehbai *et al.*, (2002) are C_4 – T_1 but the commonest is at the lowest 3 cervical vertebrae (C5 – C7).

2.1.1 Epidemiology Of Cervical Spondylosis

- Age of common occurrence is between 30 45 years
 (Akinrolabu, 1992; Saitoh et al., 2002).
- Cervical spondylosis may affect males earlier than females, but this is not true in all studied populations (Akinrolabu, 1992; Adeloye, 1999).

- Cervical spondylosis is also known to be associated with poor posture relating to anxiety, stress, and fatigue in individuals.
- Occupational stress, e.g. computer operators, typists at poorly
 positioned desks, long distance drivers are at risk of developing
 cervical spondylosis early (Bose, 1999).
- It can also arise as a result of post trauma e.g. Road Traffic Accidents
 (RTA), or soft tissue injury at joints.

Furthermore, Irvine, (1992), using radiographic evidence described the prevalence of cervical spondylosis. In males, prevalence was 13% in the third decade, rising to nearly 100% by age 70 years, while in females, the prevalence ranged from 5% in the 4th decade to 96% in those older than 70 years.

Another study, by Holt and Yates, (1996), examined patients at autopsy. At age 60 years, 60% of the men and 40% of the women had a significant amount of the disease.

Rahim and Stambough, (1992), noted that spondylotic changes are most common in those older than 40 years, eventually, more than 70% of men and women are affected, but radiographic changes are more severe in men than women.

2.1.2 Pathophysiology Of Cervical Spondylosis and Symptoms.

Normally the intervertebral disc gradually degenerates throughout life, with a resulting loss of intervertebral height and a bulging of the annulus fibrosus. The biomechanical properties of the disc are compromised, which cause local or "segmental" instability. The processes causes increased load on the facet joints, resulting in hypertrophy of the bone and capsular ligaments (Rehbai *et al.*, 2002).

Continuous stress on the attachment of the annulus to the bone results in the deposition of calcium, which appears on a radiograph as a "bone spur" called an osteophyte. These degenerative changes in most cases result in a cracking sound (crepitation) of the affected joint during movement. The processes of annular bulging, osteophyte formation, joint enlargement and ligamentous hypertrophy result in narrowing of the central spinal canal and the intervertebral foramen (Tsiptsios *et al.*, 2001).

Clinically, several symptoms, both overlapping and distinct, are seen in cervical spondylotic patients. These includes neck and shoulder pain, neck stiffness/hypomobility, headache, radicular symptoms, and cervical spondylotic myelopathy in some cases (Akinrolabu, 1992). Frequently, associated degenerative changes in the facet joints, hypertrophy of the ligamentum flavum, and ossification of the posterior longitudinal ligament do

occur, all can contribute to impingement on pain sensitive structures (nerves and spinal cord), thus creating various dinical symptoms.

2.1.3 Diagnosis Of Cervical Spondylosis

Diagnosis and investigation are important components of cervical spondylosis rehabilitation. They include the following;

2.1.3.1 Physical Examination

A thorough physical examination reveals a lot about the health and general fitness of the patient. Examination is also essential to identify precipitating factors in the patient's lifestyle, for example;

- Working conditions that demand concentration resulting in 'poking chin and round shoulder'
- 2. Habit of holding the telephone on one shoulder
- 3. Sitting or standing still for long periods
- 4. Driving for a long time, especially in traffic jams
- 5. Sleeping in awkward positions and,
- 6. Long periods working on the computer with bad sitting posture.

The examination will also include a review of the patient's medical and family history. Often laboratory tests such as complete blood count and urinalysis may be ordered and the physical examination may include:

Palpation to determine spinal abnormalities, areas of tenderness, and muscle spasm.

Range of Motion Assessment which measures the degree to which a patient can perform movement of flexion, extension, lateral flexion, and cervical rotation. This is done using the universal goniometer.

2.1.3.2 Neurological Evaluation

The neurologist assesses the patient's symptoms including pain, numbness, parasthesia (e.g. tingling), extremity sensation and motor function, muscle spasm, weakness, and bowel/bladder changes. Particular attention may be given to the extremities.

2.1.3.3 X-Rays And Other Tests

Lipping of the vertebral bodies, osteophytes at the margins of the apophyseal joints and diminished space between the vertebral bodies can be seen (Figure 1). It is important to remember that severity of signs and symptoms and X-ray findings do not very often correlate. Radiographs will almost always be "positive" for bone spurs, decrease disc height and facet hypertrophy in older patients. Magnetic resonance imaging (MRI) and computerised tomography (CT) scanning do give a more accurate and detailed picture of the bony anatomy (Tsiptsios *et al.*, 2001). These are particularly required if there is evidence of neurological dysfunction.

MRI, CT scans, Discography and other comprehensive diagnostic tests are also indicated in most cases when patient has poor response to initial conservative therapy.

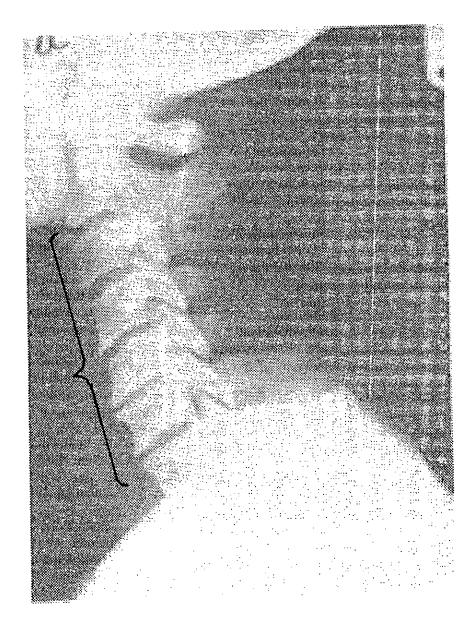


Figure 1: Radiograph Of Cervical Spondylotic Patient (One of the Subjects).

Shows Degenerative Changes in the Spine

2.1.4 Management of Cervical Spondylosis

Management of cervical spondylosis is directed at:

- Relief of pain.
- Restoration of movement
- Strengthening of spinal muscles
- Education on posture
- Sustenance or restoration (if impaired) of activities of daily living (ADL)
- Analysis of precipitating factors to reduce recurrence of patients' problems.

2.1.4.1 Conservative Management

Conservative treatment is successful in 75% of the cases (Onel *et al.*, 1989; Neuwirth and Marssicano, 1996). Many patients find their pain and other symptoms being effectively treated without surgery (Bhatoe, 2000; Singh *et al.*, 2001). During the acute phase, anti-inflammatory agents, analgesics, oral corticosteroids, injection (epidural steroid or trigger point) and muscle relaxants may be prescribed for a short period of time.

The affected area may be immobilized and/or braced. Cervical collars may be used to restrict movement and alleviate pain (Figure 2). In physical therapy (physiotherapy), heat/thermal therapy, traction therapy, electrical stimulation, therapeutic exercises e.g. isometric neck strengthening exercise, neck and shoulder stretching and flexibility exercises, back strengthening exercise,

pulley exercise, TENS (Transcutaneous Electric Nerve Stimulation) and other modalities may be incorporated into the treatment plan to control muscle spasm, alleviate pain and combat neck stiffness (Nelson, 1979; Junkin and Adria, 2002; Lee et al., 2002).

No single physical modality can effectively manage cervical spondylosis. Therefore the best physiotherapy approach is the combination of modalities (Sypert and Cole, 1999; Borman *et al.*, 2003; Akinbo *et al.*, 2004*). A combination that has been proved to offer 75% - 80% symptomatic relief of spondylotic pain is thermal therapy, therapeutic exercise and cervical traction (Uttely and Monro, 1989; Borman *et al.*, 2003).

Physical therapy teaches the patients how to strengthen their para-vertebral and abdominal muscles to lend support to the spine. Isometric exercises can be helpful when movement is painful or difficult. Exercise in general helps to build strength, flexibility, and increase the range of movement (Fouyas and Statham, 1998; Fouyas *et al.*, 2002).

Lifestyle modification may be necessary. This may include an occupational change, back care education, losing weight if obese/overweight and quitting smoking. Occupational change include, change of job from manual labour, load carrying on the head, prolonged sitting and long hours of Visual Display Terminal (VDT) keyboard work (increased computerization in office, at home

or even inside the car, Laptop), job involving standing for a long period of time e.t.c., if established as symptoms' aggravating factors. Back care education involves, back care and posture education, ergonomic instructions, home program, and instruction on activities of daily living (ADL) (John and Tony, 2003).

Failure to respond at any time to conservative treatment may require additional diagnostic tests and/or treatment consistent with greater level of severity.

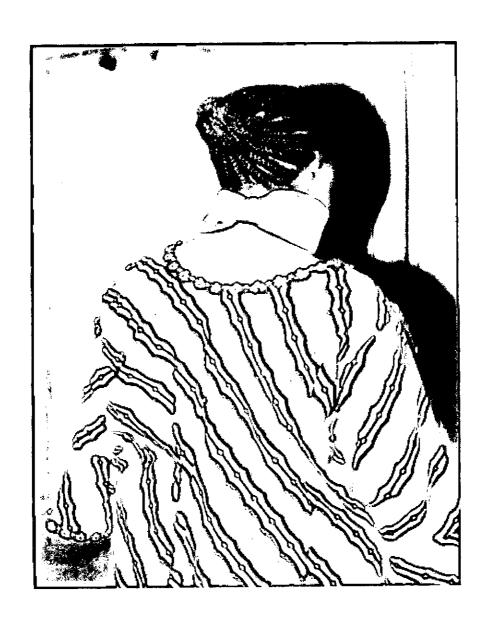


Figure 2: A Cervical Spondylotic Patient On Cervical Collar. (One of the Subject)

2.1.4.2 Surgery

Surgery is seldom used to treat spondylosis or spinal osteoarthritis.

Conservative forms of treatment are tried first, but if there is neurological deficit, certain surgical procedures may be considered (Nelson, 1979).

Indication for surgery includes; intractable pain, progressive neurological deficits, and documented compression of nerve root or of the spinal cord that leads to progressive symptoms.

Surgery options are anterior cervical discectomy (ACD), laminectomy, and laminoplasty, however, before surgery is recommended, the patient's age, lifestyle, occupation, and number of vertebral levels involved are carefully evaluated.

2.1.4.3 Summary Of Management

The management of patients with cervical spondylosis can be summarized as follows:

2.1.4.3.1 Pharmacological Pain Control Methods

- Muscle relaxants
- Non-steroidal anti-inflammatory drugs (NSAID).
- Oral corticosteroids.
- Injection (epidural steroid or trigger point).

2.1.4.3.2 Physical Therapy Pain Control Methods

- Manual therapy- soft tissue massage
- Thermal therapy.
- Traction therapy
- Transcutaneous electric nerve stimulation (TENS).
- Brace/ collar
- Acupuncture therapy.
- Cryotherapy/Hydrotherapy.

2.1.4.3.3 Therapeutic Exercise.

- Isometric neck strengthening exercise
- Neck and shoulder stretching and flexibility exercise.
- Back strengthening exercise.
- Aerobic exercise.
- Pulley exercise

2.1.4.3.4 Back Care Education

- Back care and posture education
- Ergonomic instructions
- Home programme.

2.2 What is Traction?

Traction is the technique in which a pulling force is used to stretch soft tissues and to separate joint surfaces or bone fragments. Traction in patient management is categorized into three namely; skeletal traction, skin traction and spinal traction. Skeletal and skin traction are used in the management of bone fracture.

Spinal traction is the application of a longitudinal force to the axis of the spinal column (Saunders, 1983; Judovick and Nobel, 1997). In other words, parts of the spinal column are "pulled" in opposite directions in order to stabilize or change the position of damaged aspects of the spine. Two main form of spinal traction, depending on the area of application in the spinal column, are the cervical and lumbar traction.

2.2.1 History of Spinal Traction

Physicians have been aware of the concept of traction for many centuries, however, it was not used as a therapeutic option until the late 18th century (Betge, 1981). At that time, the primary indications for spinal traction were the correction of scoliosis and spinal deformity, the management of rickets and for relieving backache of any origin or location.

Later in the 19th century (Caldwell and Krusen, 1962), attempts were made to treat a multitude of neurological disorders with spinal traction including conditions such as Parkinson's disease and impotence. The results were generally not consistent and the technique did not gain much support among those in the medical community (Caldwell and Krusen, 1962).

By the first half of the 20th century, the accepted use of spinal traction became primarily focused in the areas of cervical spine surgery and, more frequently, in the management of spinal trauma and pain (Betge, 1981).

2.2.2 Cervical Traction

Cervical traction (CT) applies a stretch to muscles, ligaments, and tissue components of the cervical spine. It provides relief by promoting separation in the intervertebral (IV) joint space, which contains the disc and may reduce a "bulge" or impingement of structures within the foramen (Shakoor *et al.*, 2002).

Cervical traction force is usually applied to the skull through a series of weights or a fixation device and requires that the patient is either kept in bed (supine), placed in a halo vest i.e. sitting position, or in standing position (Figures 3-5) (Nwuga, 1976; Zybergold and Piper, 1985; Valtonen and Kiuru, 1990).

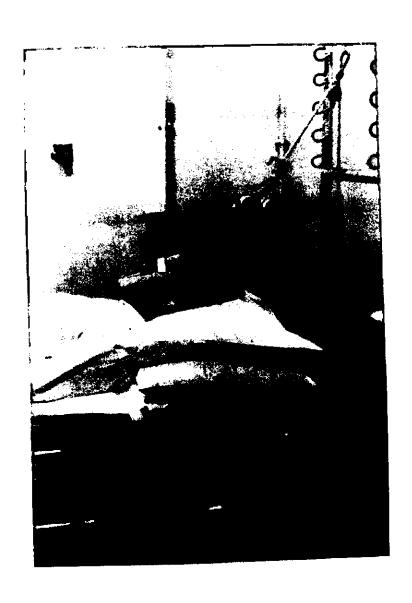


Figure 3: Cervical Traction In Supine Position Using Pulley And Weight.

(Nwuga, 1976)

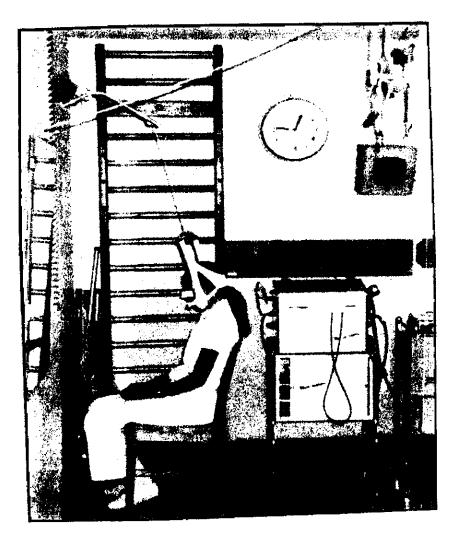


Figure 4: Cervical Traction In Sitting Position Using An Overhead Traction Unit. (Nwuga, 1976)

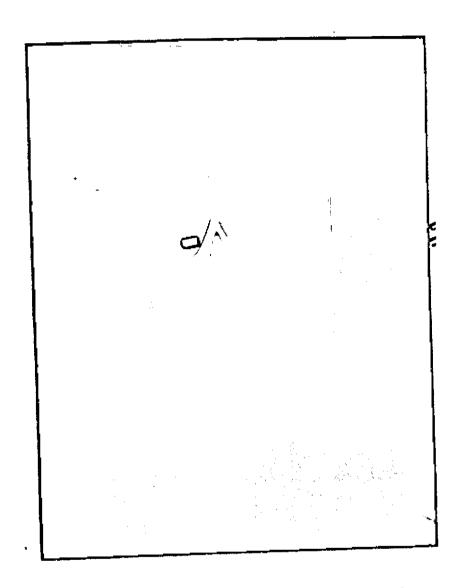


Figure 5: Cervical Traction In Standing Position (Nwuga, 1976)

2.2.2.1 Uses of Cervical Traction

A recent retrospective study by Swezey *et al.*, (1999) found that cervical traction provided symptomatic relief in 81% patients with mild-to-moderate-severe cervical spondylosis. Therapeutic spinal traction uses manually or mechanically created forces/weights and pulley to put tension on a displaced bone or joint, to realign the bone and immobilize it. Traction is also used to keep a group of muscles stretched to reduce muscle spasms. Spinal traction is also used to mobilize soft tissues or joints, decompress pinched nerve roots, and reduction of herniated intervertebral discs (Harris, 1977).

Currently, the most important use of traction is for the management of cervical spine instability, specifically cervical spondylosis (Harris, 1977; John and Tony, 2003). Traction is an extremely effective means of realigning a cervical spine dislocation and providing stabilization in cervical spine injury (Jette, 1985; Olson, 2001).

2.2.2.2 Effects of Cervical Traction

Clinical effects are relief of pain or pins and needles sensations and increased mobility. How these effects are achieved is speculative (May, 2001). The following explanations have been given by some researchers (Nwuga, 1995; Ross, 1997):

- 1. Separation of vertebral bodies enables fluid flow and therefore improves nutrition of the IVD.
- 2. Stretching of tight fibrous tissue for 5-10 minutes also allows fluid flow and increases movement which is retained by exercise
- 3. Stretching of tissues around the nerve root in the intervertebral canal enables free circulatory flow. This improves nutrition to the nerve and removes metabolites and exudates produced by low-grade inflammation. This may explain why root pain remains diminished after traction is removed.
- 4. Sliding (accessory movement) of the facet joints facilitates synovial sweep-aiding lubrication and nutrition.
- 5. Traction counteracts the effects of gravity and poor posture and is useful as an adjunct to posture training.

2.2.2.3 Application of Cervical Traction

The variables to consider in the application of traction are:

- 1. Position of spine
- 2. Traction force/load
- 3. Traction duration
- 4. Traction technique

2.2.2.3.1 Position of spine

The spine is positioned to enable the force to be effective at the target segment of the spinal column. Generally, the lower the level to be treated in the cervical spine the more the neck is positioned in flexion. Also, the lower in the lumbar spine the level is, the more the spine is flattened by flexing the knees and hips and tilting the pelvis backwards. The clinician should always palpate the vertebral spines of the target segment to check for effect.

2.2.2.3.2 Traction Force/Weight

There is currently no consensus among the clinicians regarding the amount of traction force to be employed during cervical traction that will correlate precisely with the percentage body weight of the patient.

In lumbar spondylosis, the lumbar traction force of 25% - 26% TBW has been established to be the ideal weight for effective pain reduction and normal range of movement at the lower back region (Borman *et al.* 2003).

2.2.2.3.3 Traction Duration

Time varies according to the condition treated. For nerve root pain, sustained traction may be left on for 20-30 minutes, released for 5-10 minutes rest period and re-applied for a further 20-30 minutes. Treatment is generally best on daily basis until the overall pain reduction is achieved. For degenerative changes or regional spinal stiffness, 10-20 minutes is the order of time used, repeated three times a week until symptomatic pain relief is achieved.

2.2.2.3.4 Traction Techniques/Types

Techniques applied in spinal traction are dependent on the patient's physical condition, disorder, individual tolerance, and the spinal level(s) to be treated. The techniques presented below are not all inclusive (Harris, 1977; Swezey *et al.*, 1999; Shakoor *et al.*, 2002).

2.2.2.3.4.1 Mechanical Traction

Mechanical traction can be a mechanical device or can be electrically controlled (Figure 6). It could be continuous (static) or intermittent. For a static model, static tension is constant throughout the duration of treatment. For an intermittent model, the ratio of on: off time of treatment could be; 3: 1 or 4: 1. Continuous traction is indicated for degenerative related disorders and disc prolapse, while intermittent traction is recommended for patients with neck muscle spasm (Jette, 1985; Judovick and Nobel, 1997).

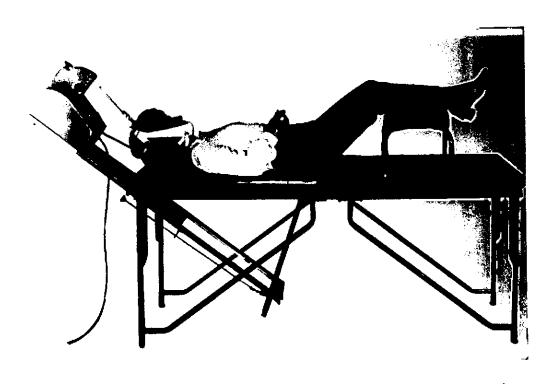


Figure 6: Cervical Traction In Supine Position Using An Electronic Device (Nwuga, 1976)

2.2.2.3.4.2 Manual Traction

This traction is applied manually by the therapist. It can be used as a clinical test for mechanical traction (Figure 7).

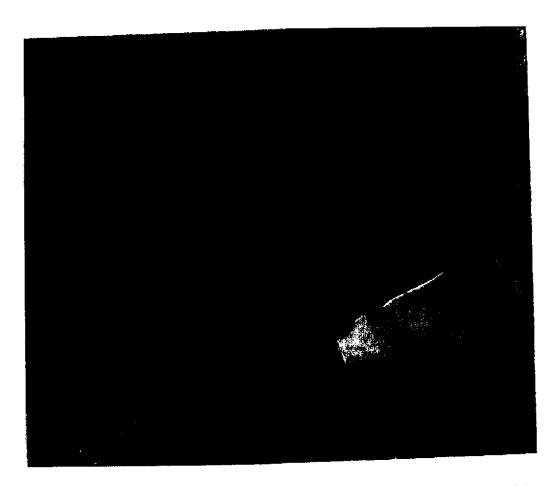


Figure 7: Manual Cervical Traction With The Subject In Supine Position.

(Nwuga, 1976)

2.2.2.3.4.3 Traction by inversion

The patient is held in an inverted position by the ankle or the area of anterior thigh. It has been proved to be effective in managing chronic low back pain but has the risk of cardiovascular stress and an increased blood pressure during application (Ogino *et al. 2001*)

2.2.2.3.4.4 Traction in water/Hydrotherapy

Traction in which the flotation device in water provides the traction pulls. It has an added advantage of enhancing muscular relaxation and faster pain relief.

2.2.2.3.4.5 **Self Traction**

Traction applied by the patient on his/herself. Not effective when high force is needed.

Cervical traction could be applied either in the sitting or lying (supine) position, but the supine position is widely accepted with the knee bent at 45 degree with the neck placed at 20 – 30 degree of flexion (forward tilt). Using traction in this position helps stretch the posterior neck muscles and facilitate intervertebral separations which relieve pressures that may be pinching the nerves, and thereby promoting muscle relaxation (Zybergold and Piper, 1985; Nwuga, 1995).

2.2.2.4 Cervical Traction Indications & Contraindications.

It's not all patients with cervical spondylosis that could be managed with cervical traction; the following are the indications and contraindications of cervical traction (Ogino *et al.*, 2001).

2.2.2.4.1 Indications

Cervical traction can be used in the management of the following conditions:

- Muscle spasm.
- Degenerative Osteoarthritis/spondylosis of the spine.
- Herniated/protruding IVD.
- Nerve root compression.
- Capsulitis of the vertebral joints.
- Pathology of the anterior or posterior longitudinal ligaments
- Management of facet joint impingement or pain.
- Neck stiffness/hypomobility.

2.2.2.4.2 Contraindications.

Cervical traction is contraindicated in the following conditions:

- Unstable or fractured spine.
- Extruded disc fragmentation or rupture
- Spinal cord compression.
- Acute strains or sprains (inflammatory diseases of the spine).
- Pregnancy

- Conditions in which vertebral flexion is contraindicated,
- Osteoporosis.
- Mental disorders.

2.2.2.5 Precautions on Cervical Traction Application

- Treatment should be discontinued if (a) symptoms increase, or
 (b) if pain, or parasthesia is experienced (Nanno, 1994).
- Instruct patient on what to expect and to relax during treatment.
- Patient should be given emergency signal or off button (panic button),
 which should be used if there is discomfort or increase of pain during treatment.
- If traction device does not automatically shut off at the end of traction period, the tension/force should be gradually removed after treatment.
- Patients should be instructed to lie still for 5 10 minutes post treatment. Standing up too quickly, can be detrimental to any benefits received from the traction (Klougart et al., 1996).

2.3 Anatomy Of The Vertebral Column

For the spinal pathology to be appreciated we must understand the anatomy of the spine (vertebral column). The vertebral column is composed of 33 short bones called vertebrae (7 cervical, 12 thoracic, 5 lumbar, 5 sacral and 3-5 coccygeal) and 23 intervertebral discs, there are thus 25 mobile segments (Paris, 1990).

The column is divided into the following five regions: cervical thoracic, lumbar, sacral and coccygeal (Figure 8). The vertebrae adhere to a common basic structural design but show regional variations in size and configuration. The vertebrae increase in size from the cervical to the lumbar region and decrease in size from the sacral to the coccygeal region. Structural abnormalities in the lower lumbar, sacral and coccygeal regions are a common finding (Carmicheal and Burkart 1979). The principal functions of the vertebral column include the transmission of the weight of the head, shoulder girdle and upper extremities.

Additionally, the column/spine is a protective organ to the central nervous system (CNS) and the origin of the peripheral nerves. It also permits movement and serves as an excellent shock absorber. It is assisted in this capacity by the curves of the spine (Gracovetsky, 1987).

2.3.1 The Spinal Curves

When the vertebral column is viewed from the posterior aspect, all regions together present a single vertical line that bisects the trunk (Figure 8). When the column is viewed from the side (lateral), a number of curves that vary with age are evident. The shape of the vertebral bodies and their interposed intervertebral discs form the curves of the spine. The vertebral column of a baby at birth exhibits one long curve that is convex posteriorly (Figure 9). However, when the column of an adult is viewed from the side, four distinct anterior-posterior curves are evident (Figure 8). The two curves (thoracic and sacral) that retain the original posterior convexity throughout life are called *primary curves*, while the two curves (cervical and lumbar) that show a reversal of original posterior convexity are called *secondary curves*.

Curves that have a posterior convexity (anterior concavity) are referred to as kyphotic curves, while curves that have a posterior concavity (anterior convexity) are called lordotic curves (Gracovetsky, 1987). The function of the curves is principally to increase the load bearing capacity of the spine (Slaby et al., 1994).

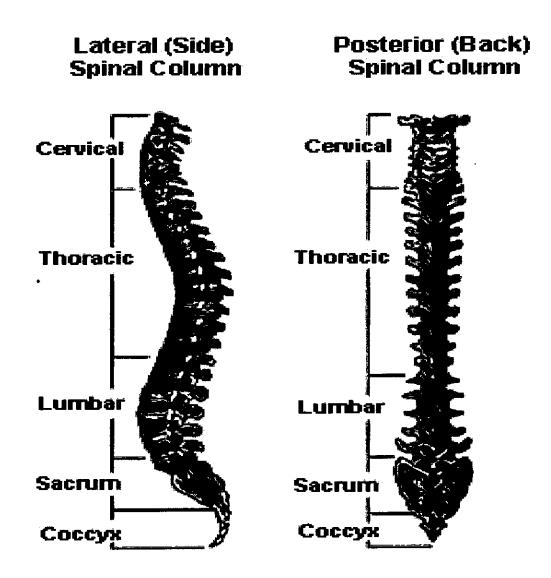


Figure §: The Spinal Column Showing The Spinal Curves. (Slaby *et al.,* 1994).

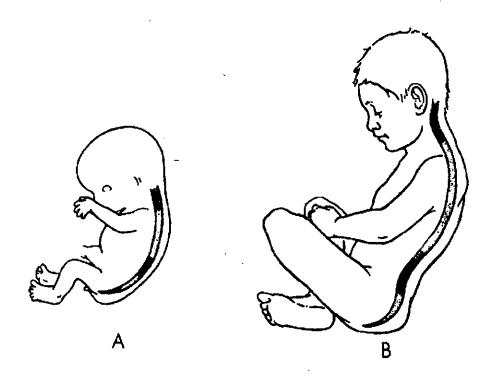


Figure 9: The Normal Curvatures Of The Vertebral Column In The (A)
New Born And (B) 4-year Old Child (Norkin and Levangie, 1992).

2.3.2 The Intervertebral Discs

The intervertebral disc (IVD) is one of the principal structures implicated in cervical spondylosis pathology (Roberts, *et al.*, 1989). The joints between the bodies of all pre-sacral vertebrae, except those between the occiput, atlas and axis are united by secondary cartilaginous joints comprising what is known as the intervertebral disc (Roberts, *et al.*, 1989), (Figure 10).

The IVD is said to be a unique structure of considerable mechanical and pathological entity (Farfan and Sullivan, 1967). This assertion was supported by the epoch-making paper delivered in 1933 by Mixer and Barr (Farfan and Sullivan, 1967). The disc till date is considered by researchers to be the principal site of spondylosis (cervical and lumbar) and spinal disability (Farfan and Sullivan, 1967; Bogduk *et al.* 1981).

Degenerative diseases of the disc, primary or secondary, do have serious consequences often requiring surgical intervention; and if unmanaged may result in permanent physical disability. Of all the structures of the spine, the disc carries the greatest responsibility for the preservation of function of the vertebral column (Farfan and Sullivan, 1967). There are 23 vertebrae in the spine that provide a flexible and resident motion as well as shock absorption and transmission during weight bearing (Bogduk *et al.* 1981).

The disc makes up approximately, 25% of the total length of the normal adult spine: 33% of the lumbar spine, 25% of the cervical spine and 20% of thoracic spine. At birth they make up 50% of the total length of the spine (Gracovetsky, 1987).

The shape of the disc varies from region to region. The thicker the shape the more the movement it permits. The thoracic spine discs posses the same anterior and posterior thickness and are quite narrow hence the relative immobility of this region. The cervical and the lumbar discs are wedge-shaped, and all are thicker anteriorly.

The intervetebral disc has 3 principal functions which are:

- They bind together the vertebral bodies
- They permit movement within the segment
- They permit load to be transmitted across the spine

In order for these and other functions to be carried out efficiently, the disc is composed of 2 principle parts. The inner nucleus pulposus and outer annulus fibrosis.

Moreover, as people age, certain biochemical changes occur affecting tissues found throughout the body. In the spine, the structure of the intervertebral discs-annulus fibrosus, lamellae, nucleus pulposus may be compromised. The

annulus fibrosus is composed of 60 or more concentric bands of collagen fibers termed lamellae. The nucleus pulposus is a gel-like substance inside the intervertebral disc encased by the annulus fibrosus. Collagen fibers form the nucleus along with water, and proteoglycans (Bogduk *et al.*, 1981).

The degenerative effects from aging may weaken the structure of the annulus fibrosus causing the 'tire tread' to wear or tear (Paris and Nyberg, 1989). The water content of the nucleus decreases with age, affecting its ability to rebound following compression (e.g. shock absorbing quality). The structural alterations from degeneration may decrease disc height and increase the risk for disc herniation (Burkart and Beresford, 1979).

Examples of Disc Problems

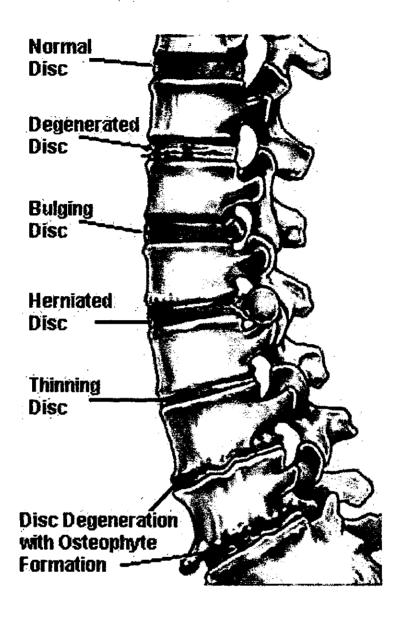


Figure 10: Intervertebral Disc With Pathological Changes

2.3.3 The Facet/Zygapophyseal Joints

The facet joints are also termed zygapophyseal joints. Each vertebral body has four facet joints that work like hinges. These are the articulating (moving) joints of the spine enabling extension, flexion, and rotation. Like other joints, the bony articulating surfaces are coated with cartilage which is a special type of connective tissue that provides a self-lubricating low-friction gliding surface. Facet joint degeneration causes loss of cartilage and formation of osteophytes (bone spurs) and these changes may cause hypertrophy or osteoarthritis, also known as degenerative joint disease (Slaby *et al.*, 1994; Weigel, 1995).

2.3.4 Spinal Ligaments

The spinal ligaments are also of relative importance to the pathogenesis of cervical spondylosis. The principal function of the spinal ligaments is to limit or modify movement occurring at the spinal segments (Smith-Petersen, 1990). Additionally, they serve to resist postural and traumatic strains and they also slow down movement and assist the spine in regaining the neutral or rest position (Smith-Petersen, 1990).

Some of the spinal ligaments span many segments (multisegment) e.g. anterior longitudinal ligament, posterior longitudinal ligament, supraspinous ligament and ligamentum nuchae, others only pass between 2 vertebrae

(Unisegment) e.g. interspinous, intertransverse ligaments, ligaemtum flava and lumbosacral ligaments). The summary of the ligaments of the vertebral column and functions are given in Table 1.

2.3.4.1 Anterior longitudinal ligaments

The anterior longitudinal ligament is perhaps the strongest ligament in the body (Gracovertsky, 1987), it runs along the anterior and lateral surfaces of the vertebral column (Figure 11). Functionally, this ligament has a high resistance to distraction of the vertebrae, and becomes taut on backward bending and slack on forward bending.

2.3.4.2 Posterior longitudinal Ligament

The posterior longitudinal ligament is situated within the spinal canal (Figure 11). It narrows as it descends the vertebral column from the axis to sacrum and onto the coccyx, but widens across the intervertebral discs. The ligament is thicker in the thoracic region than in the cervical and lumbar regions.

Table 1: Ligaments Of The Vertebral Column (Norkin and Levangie, 1992).

Ligaments	Function	Region
Anterior longitudinal (ALL)	Limits extension and reinforces	Axis to sacrum well-
	anterior portion of annulus	developed in lumbar thoracio
	fibrosus	
Anterior atlantoaxial	Limits flexion	Axis to atlas
(continuation of ALL)		
Posterior longitudinal (PLL)	Limits flexion and reinforces	Axis to sacrum
	posterior portion of annulus	
	fibrosus.	
Tectorial membrane	Limits flexion.	Axis to occipital bone.
(continuation of PLL)		
Ligamentum flavum (LV)	Limits flexion especially	Axis to sacrum
	in the lumbar area.	
Posterior atlantoaxial	Limits flexion.	Atlas and axis
(continuation of LV)		
Supraspinous (SL)	Limits flexion.	Thoracic and lumbar
Ligamentum nuchae	Limits flexion	Cervical region
(continuation of SL)		
Interspinous	Limits flexion	Primarily lumbar
Intertransverse	Limits lateral flexion	Primarily lumbar
Alar	Limits rotation of the head	Axis to skull
	to the same side and lateral	
	flexion to opposite side.	

ANTERIOR VIEW

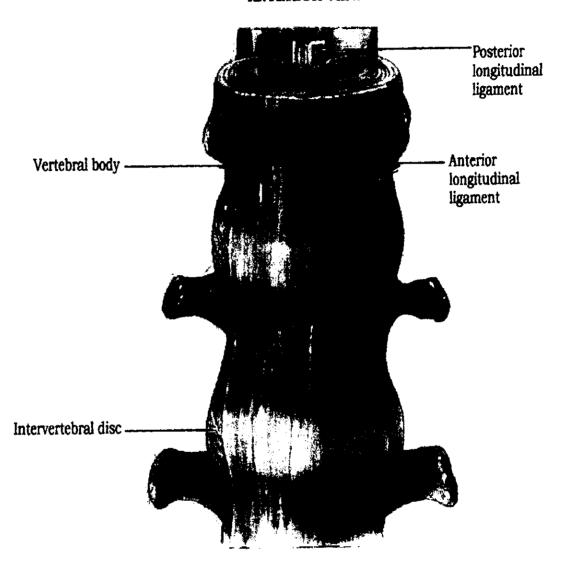


Figure 11: Ligaments of the Vertebral Column (Smith-Petersen, 1990).

2.3.5 The Back Bones

The segments of the spinal column that are mostly affected by spondylosis are the cervical and lumbar spine because of the relative mobility of these segments (Slaby *et al.*, 1994). The thoracic spine is rarely affected though it cannot be completely ruled out of degenerative changes as a result of aging.

2.3.5.1 Cervical Spine

The complexity of the anatomy of the cervical spine and its wide range of movement make this spinal segment susceptible to disorders associated with degenerative changes. Neck pain and stiffness from spondylosis are common and may spread (radiate) to the shoulder or down the arm. When a bony spur (osteophyte) causes nerve root compression, extremity (arm) weakness may result (Slaby *et al.*, 1994) and in rare cases, bone spurs that form at the front of the cervical spine, may cause difficulty in swallowing (dysphagia).

2.3.5.1.1 Functions of The Cervical Region

The two main functions of the cervical region are stability and mobility (Table 2). The cervical region differs from the thoracic and lumbar regions in that the cervical region bears less weight and is generally more mobile. Although the cervical region demonstrates the most flexibility of any of the regions of the vertebral column, stability of the cervical region, especially of the atlanto-occipital and atlanto-axial joints, is essentially for support of the head and protection of the spinal cord and vertebral arteries.

The design of the atlas is such that it provides more free space for the spinal cord than any other vertebra. The extra space helps to ensure that the spinal cord is not impinged on during motion. The bony configuration of the atlanto-occipital articulation confers some stability, but the application of small loads produce large rotations across the occipito-artiantoaxial complex and also across the lower cervical spine (Goel, 1988).

The neural zone across the occipital-atlantoaxial complex has been estimated to be 50 percent larger than in the lower cervical spine (Goel, 1988). The existence of a large neural zone implies that the ligaments and joint capsules are lax and that the muscles play an important role in providing stability for the occipito-atlantoaxial complex (Goel, 1988).

The motion of flexion and extension, lateral flexion and rotation are permitted in the cervical region. The range of motion (ROM) in lateral flexion and rotation is greater in the cervical region than in any other region. The largest range of rotation occurs between C-1 and C-2. Lateral flexion below the level of C-2 is coupled and is accompanied by rotation. Similarly, rotation initiates lateral flexion owing to the configuration of the articulating facets.

It is generally agreed that the atlanto-occipital joint permits primarily a nodding motion of the head (flexion and extension in the sagittal plane around a coronal axis), however, some axial rotation and lateral flexion is

possible (Panjabi, 1988). There is less agreement about the range of motion. The combined mobility for flexion-extension reportedly ranges from 10 to 30 degrees (Kent, 1974; Cailliet, 1981).

Table 2: Mobility And Stability Of The Vertebral Column (Norkin and Levangie, 1992).

Region	Bony Factors	Soft Tissue Factors
CERVICAL		
Atlanto-occipital	Bony contact of the anterior	Tectorial membrane limits flexion.
	ring of the foramen magnum	
	limits flexion.	
Atlantoaxial		Tectorial membrane limits flexion and extension.
Adamodaldi		Posterior atlantoaxial ligament limits flexion.
		Anterior atlantoaxial ligament limits extension.
		Transverse ligaments prevents anterior
		dislocation of C1 on C2.
		Alar ligaments limits rotation and lateral flexion
		•
		of the opposite side.
C2 - C7	Uncinate processes	The annulus fibrous provides stability by limiting
	Prevent posterior	the amount of vertebral tilting. Great height and
	Translation of the	small diameter of disc permit large range of sagittal
,	Vertebral body and	and frontal plane motion. The posterior longitudinal
L	imits flexion.	ligament, ligamentum nuchae and ligamentum
		flavum limits flexion, while the anterior
		longitudinal ligament limits extension.
H	yperextension is	Zygapophyseal capsular ligaments are lax and
Li	mited by contact	permit a large range of motion.
Of	spinous processes	·

2.3.5.1.2 Cervical Joint Range of Motion/Goniometry.

Joint mobility/flexibility is defined as the range of motion (ROM) allowed at a joint. A joint's ROM is usually measured by the number of degrees from the starting position of a segment to its position at the end of its full range of the movement. The most common way this is done is by using a double-armed/universal goniometer. A stationary arm holding a protractor is placed parallel with a stationary body segment and a movable arm moves along a moveable body segment. The pin (axis of goniometer) is placed over the joint.

When anatomical landmarks are well defined, the accuracy of measurement is greater. If there is more soft tissue surrounding the joint area, measurement error can be more frequent (Luttgens and Hamilton, 1997). Common anatomical landmarks used in the assessment of cervical ROM are; the vertex of the skull (top of the head) for neck rotation, the spinous process of C₇ for lateral flexion and external auditory meatus for flexion and extension. The best starting position for these assessments is that the subject must be seated on low chair (Norkin and White, 1995; Luttgens and Hamilton, 1997).

Furthermore, the term goniometry is derived from two Greek words, *gonia*, meaning angle, and *metron*, meaning measure. Therefore, goniometry refers to the measurement of angles, in particular the measurement of angle created at human joints by the bones of the body (Norkin and White, 1995).

When using a universal goniometer, the examiner obtains these measurements by placing the parts of the measuring instrument along the bones immediately proximal and distal to the joint being evaluated (Capuano-Pucci, 1991).

Table 3 provides cervical spine ROM values from selected sources. These measurements were obtained using the universal goniometer on normal volunteer subjects (male and female) with no history of cervical problem. The mean age of the subjects was 33.5 years (range 25 – 67 years). One hundred and thirty subjects were involved in the three studies.

Table 3: Cervical Spine Motion: Mean Values In Degrees From Selected Sources.

Motion	*AAOS	†AMA	‡Capuano-Puci,	Mean *, † & ‡
	N = 50	N = 50	N = 30	
Flexion	45	60	51	52
Extension	.45	75	70	63.3
Rt.Lat.Flexion	. 45	45	45	45
Lt. Lat. Flexion	45	45	45	45
Rt. Rotation	60	80	71	70.3
Lt. Rotation	60	80	71	70.3
	<u> </u>			<u> </u>

<u>Keys</u>

*AAOS = Values obtained from the American Academy of Orthopaedics

Surgeons, (1965).

†AMA = Values obtained from the American Medical Association, (1988).

‡Capuano-Puci = Values obtained from Capuano-Puci, (1991).

2.3.5.2 Thoracic Spine

Pain associated with degenerative disease is often triggered by forward flexion and hyperextension. In the thoracic spine disc pain may be caused by flexion - facet pain by hyperextension.

2.3.5.3 Lumbar Spine

Spondylosis often affects the lumbar spine in people over the age of 40. Pain and morning stiffness are common complaints and usually multiple levels are involved. The lumbar spine carries most of the body's weight, therefore, when degenerative forces compromise its structural integrity, symptoms including pain may accompany activity. Movement stimulates pain fibers in the annulus fibrosus and facet joints. Sitting for prolonged periods of time may cause pain and other symptoms due to pressure on the lumbar vertebrae and repetitive movements such as lifting and bending during manual labour may increase pain (Slaby *et al.*, 1994).

2.3.6 Bony and Ligamentous changes in Spondylosis

Osteophytes (bone spurs) may form adjacent to the end plates, these may compromise blood supply to the vertebra causing the end plates to stiffen due to sclerosis; a thickening/hardening of the bone under the end plates. Ligaments are bands of fibrous tissue connecting spinal structures (e.g. vertebrae) and they protect against the extremes of motion (e.g. hyperextension), however, degenerative changes may cause ligaments to lose

some of their strength. The ligamentum flavum (a primary spinal ligament) may thicken and/or buckle posteriorly toward the dura mater (Frymoyer and Gordon, 1989).

2.4 Pain As An "Alarm Bell"

Four centuries ago Descarte described pain in terms of an alarm bell ringing in a bell tower (Hoheisel *et al.* 1994). From this came the concept that there can be false alarms and we have therefore come to distinguish "psychogenic pain" from "real pain". This is now known to be a false distinction, but still we hear today the concept of hurt being not the same thing as harm, with the implication that much that hurts may be ignored, harm cannot be ignored (Wall, 1996).

2.4.1 What is Pain?

The International Association for the Study of Pain has published the following definition of pain which reflects what has been learned about pain in the last four centuries, and primarily in the past half century (Jensen, 1996; Merskey and Bogduk, 1994).

Pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey, 1986; Merskey, 1991).

From this definition we see that pain is a perception, not really a sensation, in the same way that vision and hearing are. It involves sensitivity to chemical changes in the tissues and then interpretation that such changes are harmful. This perception is real, whether or not harm has occurred or is occurring. Cognition is involved in the formulation of this perception and there are emotional consequences and behavioural responses to the cognitive and emotional aspects of pain.

Pain in most clinical settings refers to the 'sensory dimension' having intensity, location and quality and can also be a mental and emotional assault e.g. death of a loved one, armed robbery, rape e.t.c.

2.4.2 The Experience of Pain

Three systems interact to produce pain. They are;

- (1) The sensory discriminative system, which processes information about the strength, intensity, temporal and spatial aspects of pain. These are mediated through afferent nerves to spinal cord and brain.
- (2) The motivational/affective system, which determines the individual conditioned or learned approach/avoidance. These behaviours are mediated by interaction of the reticular formation, the limbic system and brain stem.

(3) The cognitive/evaluative system, which is the individual learned behaviour concerning the experience of pain. The influence of this system may block, modulate or enhance pain, e.g. the fear of dentists may intensify pain, and the joy of childbirth may suppress pain.

2.4.3 Classifications of Pain.

Pain could be classified as follows;

2.4.3.1 Peripheral pain

Peripheral pain originates in muscles, tendons, ligaments, and joints, or in the peripheral nerves themselves. Pain originating in the peripheral nerves, i.e. via trauma to the nerves, is neurogenic pain.

2.4.3.2 Central pain

Central pain arises from central nervous system pathology ... a "primary" CNS dysfuntion. Some of this may arise due to maladaptive thought processes, true "psychogenic" pain. But most of it are due to structural changes in the CNS, e.g., spinal cord injury, multiple sclerosis, stroke and epilepsy (Boivie, 1996).

Another classification that distinguishes between normally functioning nerves and nerves whose function has been altered by pathology is as follows:

2.4.3.3 Nociceptive Pain

This is a pain in which normal nerves transmit information to the central nervous system about trauma to tissues (nocere = to injure, Latin).

2.4.3.4 Neuropathic Pain

Neuropathic pain is pain in which there are structural and/or functional nervous system adaptations secondary to injury, that take place either centrally or peripherally (Jensen, 1996). Much of what has previously been considered psychogenic pain is now better understood as neuropathic pain of central origin. The IASP defines *central pain* as "pain initiated or caused by a *primary* lesion or dysfunction in the central nervous system" (Merskey and Bogduk, 1994). "Neuropathic" should not be confused with "neurogenic", a term used to describe pain resulting from injury to a *perlpheral nerve* but without necessarily implying any "neuropathy".

2.4.3.5 Acute Pain

Acute pain is a warning of actual or impending tissue injury. Physiologic responses therefore include increased heart rate, increased respiratory rate, elevated blood pressure, pallor or flushing dilated pupils and diaphoresis (Johnson, 1997). Blood sugar is elevated, so also is gastric acid secretion. Acute pain is a protective mechanism that alerts the individual to a condition or experience that is immediately harmful to the body. Onset is usually sudden and pain is relieved after clinical intervention.

2.4.3.6 Chronic/Persistent Pain

Chronic pain is persistent – usually defined as lasting at least 6 months. The cause is often unknown and pain does not respond to usual therapy. Chronic pain often develops gradually and individuals generally experience more suffering over time. Chronic pain is often associated with a sense of hopelessness and helplessness.

Physiologic response depends on whether it is persistent or intermittent. Intermittent pain produces a physiologic response similar to acute pain, whereas persistent pain allows for physiologic adaptation, without relief of pain. Moreover, the persistent pain often experienced in chronic work-related musculoskeletal injuries (e.g. cervical spondylosis), as well as in those with long continued pain for other reasons, may persist because of a Central Nervous System dysfunction. But this is a CNS dysfunction *secondary* to long continued peripheral pain. This is a concept yet to be addressed by any official declaration of the International Association for the Study of Pain (IASP).

2.4.3.7 Referred Pain

This could be described as a pain sensation in an area distant from its point of origin. That is, it originates in one place and the pain sensation is felt in another place(s) which has the same segmental innervation. Referred pain in cervical spondylosis could be any of the following;

- There may be no pain perceived in the neck but there is pain in the arm.
- Pain down to the elbow could be as a result of the compression of the 5th cervical rami.
- 3. Pain to the thumb and index finger could be as a result of the compression of the 6th cervical rami.
- 4. Pain on the middle three fingers and forearm could be as a result of the compression of the 7th cervical rami.
- 5. Pain on the inside of forearm, little fingers and possibly chest could be as a result of the compression of the 8th cervical and 1st thoracic ramus.
- 6. In addition pain may be referred down to the thoracic area, e.g. the medial border of the scapula.

2.4.4 Peripheral Sensitization of Pain

Tissue damage results in a drop in pH and a release of chemicals, e.g. histamines and bradykines, to which small non-myelinated C fibres are sensitive. Fitzgerald and Woolf, (1984), have hypothesized that C fibres are primarily chemical sensors, although they do respond also to high level mechanical and thermal stimulation.

The C fibres respond by generating an electrical impulse which travels along the nerve to the dorsal horn of the spinal cord. Activity of the C fibres may be up-regulated peripherally by serotonin (i.e., 5-hydroxy tryptamine), prostaglandins, thromboxane, and leucotrienes in the damaged tissues. This is referred to as *peripheral sensitization* in contrast to central sensitization which occurs at the dorsal horn. Both occur in chronic pain. 'Substance P' may also be released peripherally with resultant increase in peripheral vasodilatation and further sensitization of the C fibre's peripheral ending. Even chemical products of tissue breakdown may sometimes enter the neuron and be transported centrally to exert an effect at the dorsal horn synapse (Hirshberg *et al.* 1996).

2.4.5 Theories of Pain

There are four theories of pain:

- 1. The intensity theory,
- 2. The specificity theory,
- 3. The pattern theory and
- 4. The gate control theory.

Of these theories, the gate control theory is most relevant to the modern mechanism of pain and pain controls.

2.4.5.1 The Intensity Theory of Pain.

This theory is the first to be formulated of all the pain theories. The intensity theory implied that pain resulted from excessive stimulation by certain stimulus e.g. excessive heat or cold, excessive damage to the tissues e.t.c. This theory was first suggested by Darwin and subsequently embraced by Romberg, in the 1840s. The theory was explicitly supported by Erb in 1874, it maintained that every sensory stimulus was capable of producing pain if it reached sufficient intensity (Bishop, 1980).

In 1894 Goldscheider fully developed the theory and stated that stimulus intensity and central summation were the critical determinants of pain. This theory suggested that the particular pattern of nerve impulses that evoked pain is produced by the summation of the skin sensory input at the DORSAL HORN CELLS.

According to this concept, pain results when the total output of cells exceeds a critical level as a result of excessive stimulation of receptors that are normally fired by noxious thermal or tactile stimuli or at pathological conditions that enhance the summation of impulses produced by non-noxious stimuli (Bishop, 1980; Michlovitz and Wolf, 1996).

Goldscheilder reported that the long delay and persistent pain observed in pathological conditions were due to abnormally prolonged periods of summation. He further proposed that the spinal "summation pain" that

transmitted the pain signal to the brain consisted of slowly conducting multi synaptic fibre chain and that the large fibres that project up to the dorsal column pathway carried the tactile discrimination properties of cutaneous sensation.

It must be pointed out, however, that not all researchers accept the concept of intensity theory of pain. Knowledge postulated in the 20th century has shown that the intensity theory of pain is wrong (Melzak and Wall, 1965).

2.4.5.2 The Specificity Theory of Pain

The specificity theory (Figure 12) proposed that each sensation perceived is elicited by the activation of a specific receptor anatomically located in the skin. When the receptor is stimulated, a signal is sent to the brain centre via direct pathway, such as the lateral spinothalamic track to the ventrolateral portion of the thalamus. Originally, it was thought that pain, touch, warmth and cold sensations were the result of the stimulation of those receptors specifically and anatomically related to the sensation. The specificity theory was questioned, however based on the fact that severing the spino-thalamic track did not necessarily reduce pain (Michlovitz and Wolf, 1996).

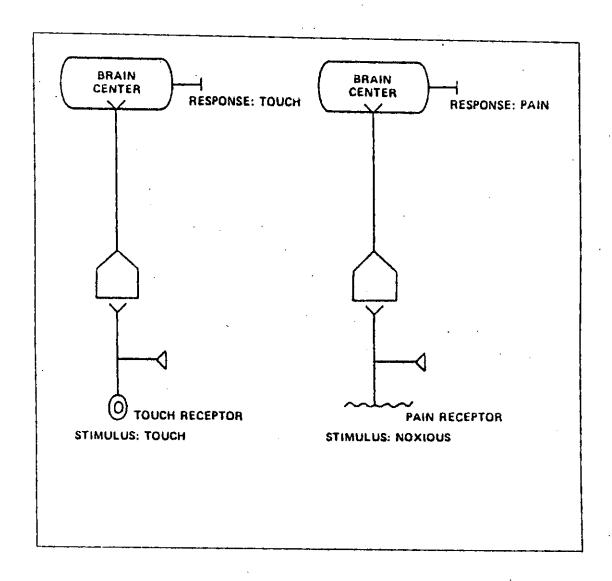


Figure 12: Specificity Theory of Pain (Michlovitz and Wolf, 1986).

2.4.5.3 The Pattern Theory of Pain

The pattern theory proposed that a non-specific receptor stimulated by varying intensities produces different sensations that result from a code or a pattern of sensory input (Figure 13). It was suggested that a temporal (frequency) or spatial (intensity) pattern of impulses from a non-specific receptor determine the type of sensation perceived, with a specific stimulus, such as pain, resulting in the generation of a specific pattern of action potentials that is perceived in the brain as pain.

This theory was questioned because of the existence of specific receptors that are anatomically located in the skin (Michlovitz and Wolf, 1996).

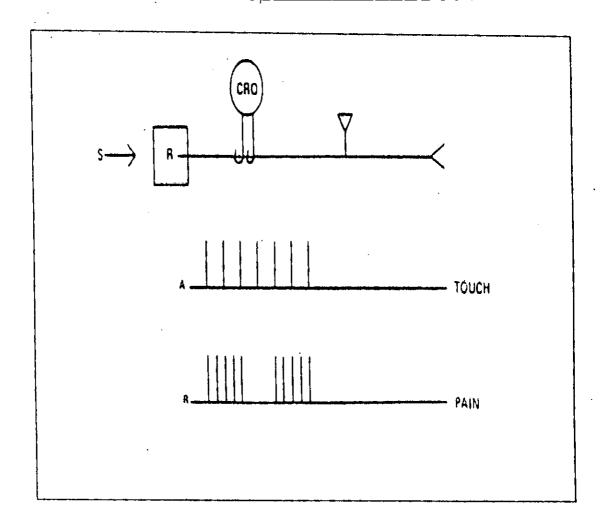


Figure 13: The pattern Theory of Pain.

S = stimulus; R = response; CRO = cathode ray oscilloscope; A = action potentials resulting from touch stimulus recorded on oscilloscope, touch perceived; B = action potentials resulting from pain stimulus, pain perceived (Michlovitz and Wolf, 1986).

2.4.5.4 The Gate Control Theory of Pain

The gate control theory, proposed by Melzak and Wall, (1965), stated that a gating mechanism located in the spinal cord modulates pain transmission (Figure 14). The gate control system consists of large diameter A-beta sensory fibres, small diameter A-delta fibres and C-fibres, all of which synapse on the substantial gelatinosum (**SG**) (located in the gray matter of the dorsal horn, laminae II and III) and on the transmission or tract cells (T cells) in the dorsal horn.

The C-fibres are pain-and temperature-afferent nerve fibres. The SG terminates pre-synaptically on the large and small sensory fibbers, which in turn terminate on the T cells. Thus, the SG acts as a gate control mechanism by modulating impulses to the T cells. The activation of the SG by the A-beta fibres increases the presynaptic control on the A-delta fibers and C-fibers. The "gate "is said to be closed, reducing the impulses to the T cells and consequently, attenuating pain (pain reduction).

Conversely, a decrease in the SG activity results in a reduction of the presynaptic control of the A-delta fibers and C-fibers; the gate is open, maintains the input to the T cells, and pain is perceived. Impulses from the peripheral receptor through the T cells to the thalamus and cortex can be considered an ascending pathway of pain control. Regions in the brain stem exert an inhibitory influence on the ascending pathways originating in the spinal cord

(Figure 15). The pituitary gland, the paraqueductal gray matter, the raphe nucleus magnus, and the lateral dorsal funiculus, collectively, can be considered the descending inhibitory pathway.

High levels of stimuli or stress seem to attenuate pain via the descending control mechanism. Impulses from the A-delta fibers and C-fibers pass through the T cells to the nucleus reticularis magocellularis, gigantocellularis, and medulla. Output from the pituitary gland and gigantocellularis then travel to the paraqueductal gray matter, which sends axons to both the raphe nucleus magnus and the nucleus reticularis magocellularis.

Efferents from these nuclei travel through the dorsal lateral furniculus to terminate on the enkephalinergic interneurons in the SG. The raphe nucleus magnus release the neurotransmitter serotonin and inhibits A delta fibres to a greater extent than the A-beta fibers (Michlovitz and Wolf, 1996).

It is generally recognised that the 'Pain gate' can be shut by stimulating nerves responsible for carrying the touch signal (mechanoreceptors) which enables the relief of pain through some physical modalities and methods e.g., massage techniques, manipulation, thermal therapy and also the application of "wheat bags" and ice packs (Jensen et al., 1986).

The Gate can also be shut by stimulating the release of "endogenous opioids" which are oploid (pain-relieving) type chemicals released by the body in response to pain stimuli. Acupuncture and electrical analgesia (TENS) are thought to stimulate their release as a response to stimulation, the opioids then inhibiting the transmission of pain signals in the SG part of the spinal cord - what is often referred to as the spinal root part of the nerve (Melzak and Wall, 1965; Taylor et al., 1983). Encephalins have a half-life of 2 minutes, and the pain reduction effect generally ceases when stimulation stops. Endorphins, however, are more potent, having a half-life of 4 hours, and provide longer pain relief after the cessation of stimulation (Melzak and Wall, 1965; Bishop, 1980).

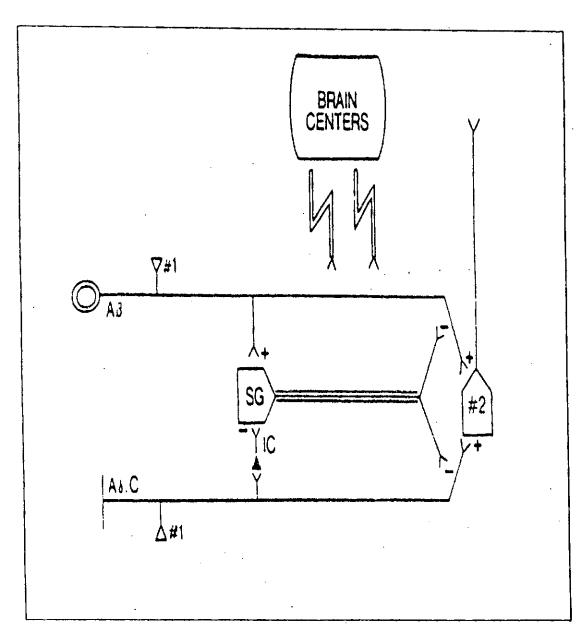


Figure 14: Gate Theory of Pain.

 $A\beta$ = A-beta fibers, SG = substantia gelatinosa, IC = inhibibitory interneuron, $A\partial$ = A-delta fibers, C = C-fibers (Michlovitz and Wolf, 1986).

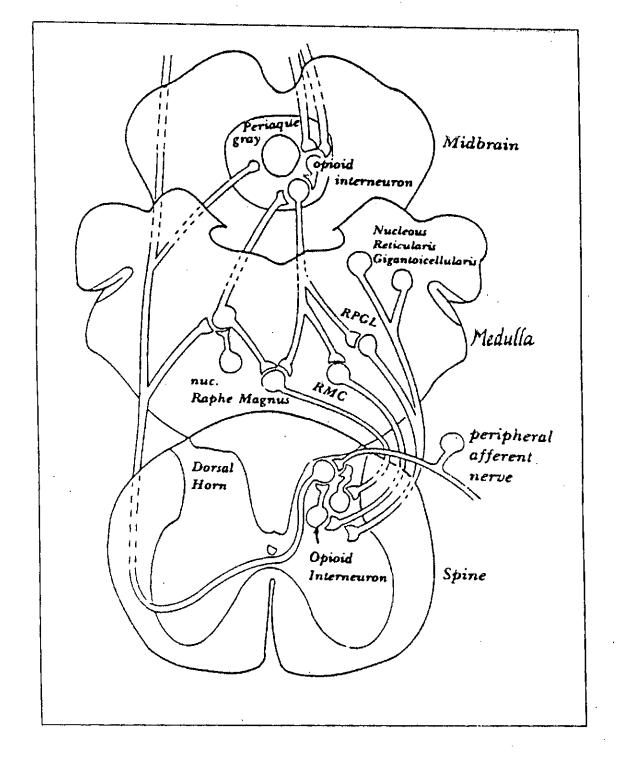


Figure 15: Descending Feedback Pathway of Pain (Tapio and Hymes, 1987)

2.4.7 Pain Producing Substances

It has been established that pain-producing chemical substances accumulate near nociceptors following tissue injury (Coker, 2003). These compounds arise from cell leakage, biosynthesis by local substrates released via enzymes induced by damage, or release by the nociceptor itself. Common pain producing substances are;

2.4.7.1 Histamine and Potassium

Histamine and potassium, amongst many substances released after injury, excites nociceptor and produce pain.

2.4.7.2 Bradykinin

Bradykinin, a polypeptide, produced by plasma protein following injury has been recognized as potent activator of nociceptor.

2.4.7.3 Prostaglandins and Leukotrines

They are also known to be synthesized at the sites of injury or tissue damage; they are the by-products of Arachidonic Acid Metabolism. These chemicals are found in high concentration in inflammatory fluids and are potent mediators of inflammation.

Aside from substances released in the regions of injuries, nociceptors themselves are known to produce pain-enhancing substances.

2.4.7.4 Substance P

Substance P, a polypeptide, is discharged from the nociceptors and excites pain transmission pathways in the dorsal horn of the spinal cord. Substance P has also been shown to be a potent vasodilator and augments the release of histamine from mast cell.

Table 4: Chemicals Actively Involved In Pain Transmission.

Substance	Source
Nociceptor Activators	
Histamine	Released from mast cell
Potassium	Released from damaged cells
Bradykinin	Plasma protein
Nociceptor Sensitizers	
Prostaglandins	Arachidonic acid released by damaged cells
Leukotrienes	Arachidonic acid released by damaged cells
Substance P	Released from 1º afferent of nociceptor
, ,	

2.4.8 Pain Management

Pain that commonly follows injury or illness can impair physical, psychological, social, and vocational functions (Brena and Chapman, 1983; Bonica, 1990). The International Association for the Study of Pain (IASP) has emphasized the need for multidisciplinary management of patients with pain.

Approaches to managing pain include:

- (1) Surgical/anesthesiological;
- (2) Pharmacological;
- (3) Physical rehabilitative/Physical Therapy;
- (4) Psychological; and
- (5) Alternative/complementary (Herbal) Therapy.

2.4.8.1 Physical Therapy Management of Pain.

Historically, the use of physical agents to treat pain and other impairments has been empiric. Developed countries have emphasized pharmacological or surgical interventional, and advanced anesthetic approaches for pain management. Psychological/behavioral and cognitive methods are being increasingly utilized, especially in comprehensive pain centers.

In most developed countries including the United States, physical agents (physiotherapy) merit consideration as *first-line treatment*. Physical rehabilitation emphasizes the use of modalities such as heat, cold, and electricity, and hands-on techniques such as manipulation, mobilization, massage, and traction. It also involves planning so as to balance rest for the injured part and prevention of re-injury through use of orthotic devices (Bonica, 1990.

Heat or thermal therapy, either superficial or deep was one of the oldest modalities used to relieve pain. It can also decrease muscle spasm and improve function.

Physiological effects of heat include analgesia, increased flexibility of collagenous tissues, and reduction of muscle spasm through selective decrease in excitation of nociceptive nerve endings. Increased muscle temperature also decreases spindle sensitivity and reduces "muscle spasm." Heat increases blood flow to the warmed area, which also may accelerate healing.

2.4.8.1.1 Cryotherapy

Application of cold therapy is a common and practical treatment for pain, they are mostly used in acute pain e.g. sport injury. Ice packs, commercially prepared chemical gel packs, and cold packs are easily available and can be used at home. Cold packs are usually applied for 10 - 15 minutes and are helpful because they conform to body contours and produce comfortable and safe pain relief (Michlovitz and Wolf, 1996). Ice massage, a specific technique in which the skin is rubbed with a block of ice, produces three stages of sensation. The patient first experiences coolness lasting a few minutes, followed by a burning sensation for a few minutes, and then numbness and pain relief, alternate vasoconstriction and vasodilatation of the blood vessel in the injured area on cold application, this mechanism is called the "Lewis hunting reaction".

Ice massage is particularly useful in treating small areas such a trigger points, tendons, and bursae, and should precede massage and stretching programs.

2.4.8.1.2 Electrotherapy

Electricity has been a pain treatment modality since ancient times when "torpedo fish" that produced electric currents were used to treat gout and headaches (Brena and Chapman, 1983). Today the most common mechanism

for applying therapeutic electricity is transcutaneous electrical nerve stimulation (TENS). Electrogalvanic stimulation (EGS), electrical muscle stimulation (EMS), focus stimulation, and neuroaugmentative stimulation are other rehabilitative methods that employ electrical current.

2.4.8.1.3 Manual Therapy

Manual therapy includes techniques that use a "hands-on" approach such as massage (stroking, friction, kneading), manipulation, and mobilization (Kottke and Lehmann, 1990; Leek, 1982).

2.4.8.1.4 Therapeutic Exercise

The most important element of physical rehabilitation addresses improvement in function through therapeutic exercises designed to increase functional activity.

2.4.9 Pain Assessment/Rating

Pain assessment has been a subject of controversy and heated debate among healthcare professionals and opinion leaders over the years. It is said that pain cannot be adequately defined, identified or measured by an observer (Merskey, 1986), but lately different methods exist.

Depending on pain responses, it is now possible to distinguish between categories of humans: Pain-sensitive (PS) human experiencing pain with qualitative differences, which depend more on psychological variables than is the case with Pain-tolerant (PT) subjects (Coker, 2003).

In advanced countries it is known that pain can be measured with Electroencephalographs devices.

2.4.9.1 Physiotherapy Pain Assessment Methods

Common pain rating scales used by physiotherapists in assessing adult patients has both numerical index (Numerical Rating Scale; NRS) and verbal component (Verbal Rating Scale; VRS). Some of the common methods are as follows;

- 1. **Pain Scale** (Borg, 1982)
- 2. Present Pain Index (PPI), (Finch and Melzack, 1987)
- 3. Visual Analogue Scale (Waterfield and Sim, 1996)
- 4. Modified Verbal Rating Scale, (Olaogun et al., 2003)

These are pain-measuring scales that allow the patient to describe his or her pain intensity in term of numbers/numerically and with corresponding verbal ratings. It is a progressive scale with the numbers being positively related to the pain intensity. The scales except the PPI (0-to-5) are rated using the 0-to-10 Numerical Rating Scale on which O equals no pain while 10 represents the worst possible pain.

Table 5: Pain Scale (Borg, 1982)

Numerical Index	Pain Description.
0	No pain/discomfort
1	Very very mild
2	Very mild
3	Mild
4	Mild
5	Very Moderate
6	Moderate
7	Uncomfortable
8	Very uncomfortable
9	Unbearable
10	Excruciating/Worst

Table 6: Present Pain Index (PPI), (Finch and Melzack, 1987)

Numerical Index	Pain Description
0	No pain
1	Slight Pain
2	Mild Pain
3	Moderate Pain
4	Severe Pain
5	Extreme/Very Severe

Table 7: Visual Analogue Scale (VAS) (Waterfield and Sim, 1996)

Numerical Index	Pain Description.		
0	No pain		
1	Slight		
2	Very mild		
3	Mild		
4	Very Moderate		
5	Moderate		
6	Moderate		
7	Severe pain		
8	Very Severe		
9	Horrible pain		
10	Worst/Extreme pain		

Table 8: Modified Verbal Rating Scale (MVRS) (Olaogun et al., 2003)

Pain Description	Pain Description	Numerical Index
English	Yoruba	
No pain	Kosi irora rara	
Barely perceptible pain	Irora ti ko lonkan lati furasi	2
Mild pain	Irora to se faramo	
Moderate pain	Irora ni won ba	4
Barely strong pain	Irora to po die	
Strong/severe pain	Irora to le	6
Intense pain	Irora to po	
Very intense pain	Irora to po lopolopo	8
Horrible pain	Irora to lini lara	
Worst pain	Irora to pojulo	10

Mark the one that corresponds to the pain you feel at this moment.

2.4.9.2 Key To Successful Pain Screening

Pain is described as the 5th vital sign. Other vital signs are BP, HR, respiratory rate and body temperature. Successful pain screening relies on the clinicians' consistent commitment to several core concepts:

- The patient's self-report of pain is the single most reliable indicator of pain.
- Observations of behaviour and vital signs should not be used instead of self-report unless the patient is unable to communicate.
- Pain can occur when there is no physiological cause and it is just as real to the patient.

2.4.9.3 The Numerical Rating Scale (NRS) For Pain

There is no pain thermometer. Measurement of pain must rely on patient's self-reports or the interferences we can make based on their behaviours. Screening for pain intensity is an important aspect of patient care (Chapman, 1985; Downie, 1978).

2.4.9.4 Tips For Successful Use of NRS.

The under-listed points must be critically followed;

- 1. Allow sufficient time to elicit the patient's self-reported pain rating.
- Provide an environment that is quiet and free of distractions.
- Have appropriate aids for hearing and vision available, e.g., charts with enlarged words, numerical scales, anatomical drawings.
- 4. Speak slowly, clearly, and as loudly as needed.
- Involve family members and/or caregivers.
- 6. Use enlarged copies of the NRS.
- 7. Teach the patient how to use the pain rating scale.
- 8. Explain the use of the scale each time it is administered.
- Provide ample time for the patient to respond to questions.
- If the patient cannot respond verbally, try having him or her point to enlarged words, numerical scales, or anatomical drawings.
- 11. Have the patient provide a single, global estimate of pain intensity.

2.5 Cardiovascular And Conducting Systems Of The Heart.

The cardiovascular system is made up of the heart together with the two main networks of blood vessels; the systemic circulation and the pulmonary circulation. The cardiovascular system effects the circulation of blood around the body, which brings about transport of nutrients and oxygen to the tissues and the removal of waste products.

Furthermore, the heart normally beats in an orderly sequence: Contraction of the atria (atria systole) is followed by contraction of the ventricle (ventricular systole), and during diastole all four chambers are relaxed.

The heart beat originates in a specialised cardiac conducting system and spreads via this system to all part of the myocardium. The structures that make up the conducting system are the sinoatrial node (SA node), the internodal atrial pathways, the atrioventricular node (AV node), the bundle of His and its branches, and the Purkinje system. The SA node is the normal Cardiac Pacemaker, its rate of discharge determines the rate at which the heart beats. In the normal human heart, the heart beats about 70 times in a minute at rest. The rate is slowed (bradycardia) during sleep and is accelerated (tachycardia) by emotion, Physical exertion, fever, and many other stimuli (Ganong, 1991).

The blood pressure in the brachial artery in young adults in the sitting or lying position at rest is approximately lower at night and is lower in women than in

men. Since the arterial pressure is the product of the cardiac output and the peripheral resistance, it is affected by conditions that affect either or both of these factors. Emotion, for example increases the cardiac output; and it may be difficult to obtain a truly resting blood pressure in an excited or tense individual. It is established that heart diseases and ischemia of the vital organs do cause alteration in blood pressure, with clinical consequences which may be mild or severe depending on the severity of the pathology (O'Brien, 1991). Also, in apparently healthy humans both the systolic and diastolic pressures rise with age.

2.5.1 Regulation Of Blood Pressure (Baroreceptor Activities)

The baroreceptor is a stretch receptor in the wall of the heart and blood vessels. The carotid sinus (located at the bifurcation of the common carotid artery in the neck) and aortic sinus (at the arch of the aorta) are receptors that monitor the arterial circulation (Figure 16).

These receptors are called cardiopulmonary receptors (Brannon *et al.*, 1993). The baroreceptors are stimulated by distension of the structures in which they are located, and so they discharge at an increased rate when the pressure in these structures rises. Their afferent fibers pass via the glossopharyngeanl (IX cranial nerve) and vagus (X cranial nerve) nerves to the medulla. Thus,

increased baroreceptor discharge inhibits the tonic discharge of the vasoconstrictor nerves and excites the vagal innervation of the heart, producing vasodilatation, venodilatation, a drop in blood pressure, bradycardia (decrease HR), and a decrease in cardiac output (Dehn, 1980).

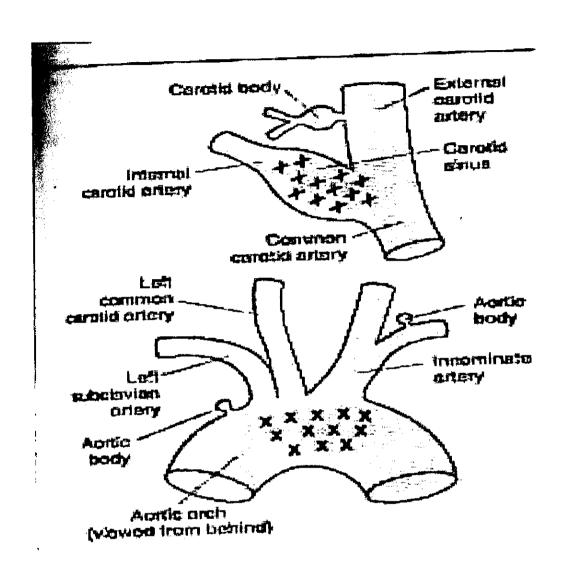


Figure 16: Baroreceptor Areas In The Carotid Sinus And Aortic Arch. X,
Sites Where Receptors Are Located (Dehn, 1980).

2.5.2 The Electrocardiogram (ECG)

An ECG is a graphic representation of the electrical activity generated by the atria and ventricles of the heart. The ECG allows for indirect observation of the sequence of cardiac muscle excitation over any given period of time (Conover, 1988). As electrical activity passes through the myocardium, it is detected by external skin electrodes placed at specific points on the body surface and recorded as a series of deflections on the ECG. The deflections, or waves, are known as P, Q, R, S and T (Figure 17).

The upward deflections are positive; toward the skin electrode. The downward deflections are negative; they represent an electrical current moving away from the skin electrode. In both instances, the magnitude of the deflection represents the thickness of the muscle mass through which the current is being conducted (Conover, 1988).

Each deflection, or wave, represents an aspect of the depolarization or repolarization of the cardiac muscle cells. Depolarization, the change of the internal potential of the cell from negative to positive, causes almost immediate myocardial contraction. Depolarization is followed by repolarization. During repolarization, the cells regain their electro-negative state, and the heart is in a physically "quiet" state (Brannon *et al.*, 1993). Although the change in electrical potential during repolarization is seen in the ECG, no physical activity accompanies this electrical activity.

It is important to note that the ECG is a recording of the electrical activity of the heart, depolarization and repolarization of the muscle cells and not a recording of the actual contraction and relaxation of the myocardium (Brannon *et al.*, 1993).

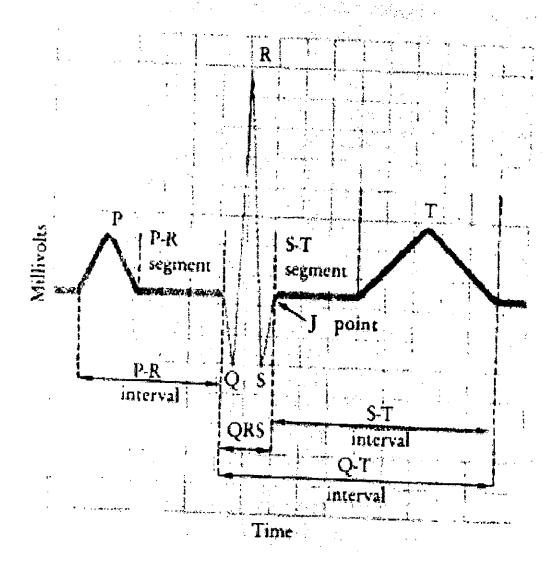


Figure 17: Graphic Representation Of Normal Electrocardiogram (Brannon *et al.*, 1993).

2.5.2.1 PR Interval

It represents the time required for the impulse to travel from the atrium through the conduction system to the Purkinje fibers. The P-wave is produced by atria depolarization (AD. It has an average duration of 0.18 second, range = 0.12-0.20 second (Dehn, 1980; American Heart Association, 1966).

2.5.2.2 QRS Complex

The QRS represents the period of ventricular depolarization (VD) and atria repolarization (AR) in the heart. It has amplitude of 20-30mm and duration of 0.06-0.10 second (Dehn, 1980; American Heart Association, 1966).

An increase in duration is a sign of delayed conduction through the ventricle. Amplitude greater than 35mm indicates ventricular hypertrophy. Amplitude less than 5mm may indicate coronary artery disease (CAD), emphysema, marked obesity, generalized oedema, or pericardial effusion (Conover, 1988).

2.5.2.3 Normal ECG Changes Seen With Physical Exertion.

Individuals with healthy hearts demonstrate a numbers of expected and insignificant ECG changes during exercise or physical exertion e.g. during cervical traction application. Most notable is the significant tachycardia that occurs with moderate to heavy physical exertion accompanied by a rapid return to the pre-exercise heart rate following cessation of physical exertion (during recovery).

The healthy individual may experience single or rare premature atrial, junctional, or ventricular contractions during physical exertion. These arrhythmias are without hemodynamic/clinical consequence (Dehn, 1980).

2.5.2.4 Abnormal ECG Seen With Physical Exertion.

Abnormal ECG responses observed during exercise or physical exertion reflect an imbalance between myocardial oxygen supply and demand. They are either exertional arrhythmias or alterations in the PR interval, QRS complex, or ST segment of the ECG (Sheppard, 1983). Exertional arrhythmias occur during both physical exertion and recovery period. They are significant in their relations to an individual's cardiac output.

If the arrhythmias cause inadequate cardiac output, it may induce syncope, angina, or congestive heart failure (CHF) (Dehn, 1980; Sheppard, 1983).

Many physiological conditions, including anaemia, hypoxemia, and ventricular aneurysm, as well as cardioactive drugs, cause the same ECG changes commonly induced by physical exertion. Other abnormal ECG changes that may be observed during physical exertion includes exertional fatigue, chest pain, dizziness, and palpitations.

When any of these symptoms is observed during exercise or physical exertion, treatment should be terminated. Observation of arrhythmias and

assessment of patients, including vital signs and objective and subjective symptoms, should be done immediately and appropriate treatment should be instituted (Dehn, 1980).

CHAPTER THREE

3.0 MATERIALS AND METHODS

3.1 Subjects Selection.

Nine hundred and forty seven (947) adult subjects (432 Men and 515 Women) with five hundred and forty one (541) of them patients with cervical spondylosis and four hundred and six (406) normal subjects (control) participated in the study.

The cervical spondylosis subjects were patients with radiological and clinical features of spondylosis analysed and confirmed by a radiologic report and referred for physiotherapy rehabilitation. The normal subjects are those that were screened by the physician and the physiotherapist and confirmed to be medically fit for the study.

3.2 Inclusion Criteria

- Cervical spondylosis patients (Men and women) with radiological evidence and clinical features of cervical spondylosis.
- 2. Subjects with no previous experience of cervical traction
- 3. Normal volunteers, medically fit without the symptoms of cervical spondylosis and age matched with cervical spondylosis subjects.

3.3 Exclusion Criteria

- Patients with structural disease or condition affecting the bones of the spine e.g. malignant lesion, osteoporosis and tuberculosis.
- Subjects with history of injury to the spine e.g. fracture, unstable spinal joint, dislocation or subluxation.
- 3. Subjects with previous history of chronic obstructive respiratory disease.
- Subjects to whom the pressure of cervical belts could be hazardous
 e.g. pregnancy, or cerebrovascular compromise.
- 5. Subjects experiencing claustrophobia and/or other psychological aversion to traction.
- 6. Hypertensive patients with cervical spondylosis.

At the end of the screening exercise, one hundred and five (105) patients and one hundred and twenty (120) normal subjects (a total of 225 subjects) met the criteria for the study (Table 9). Sixty of the normal subjects were assigned for the pilot study while the other sixty were for the main study. One hundred and sixty five (165) subjects (105 patients and 60 normal volunteers) were involved in the main study. All prospective subjects were fully briefed of the experimental procedures before they volunteered to participate by signing an informed consent.

Table 9: Classification Of Subjects According To Age And Sex.

Age range (years)	Male	Female	Total	%Total	
31 – 40	12	18	30	13.3	
41 – 50	33	40	73	32.4	
51 – 60	35	37	72	32.0	
61 & above	22	28	50	22.3	
Total	102	123	225	100.0	

3.4 The Pilot Study

The pilot study was done to make a tryout of the instruments and to identify some methodological and logistic problems that could affect the study.

Sixty (60) normal volunteers were recruited for the pilot study. They were divided into three groups of twenty subjects (9 men and 11 women in each group).

Group X: Investigated with 7.5% total body weight (TBW) cervical traction.

Group Y: Investigated with 10% TBW cervical traction.

Group Z: Investigated with 15% TBW cervical traction.

Cardiovascular variables [Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Heart Rate (HR), Rate Pressure Product (RPP), PR interval, and QRS complex] were evaluated at supine resting position (baseline) and at the end of 5, 10 and 15 minutes following cervical traction application.

Analysis of variance was done to evaluate for significance difference between the baseline cardiovascular variables and variables at the end of the three experimental phases (5, 10, and 15 minutes successively) in the various traction groups. Side effects associated with various traction weights were also evaluated.

traction groups. Side effects associated with various traction weights were also evaluated.

The pilot study had very similar results with the main study in all the variables analysed. Since there was a close similarities between the pilot study and main study results (for the normal subjects, since normal subjects only participated), detailed analysis of these results were given in the main study results and discussion chapters.

3.5 Experimental Design

The main study was implemented in two stages; the **investigative**(Experiment One) and therapeutic (Experiment Two) stages successively. The investigative stage was concluded before the commencement of the therapeutic stage.

A sample of convenience method was used to assign subjects into groups, that is, subjects were assigned to the various groups as they became available. Equal numbers of male and female subjects were also maintained in each group for uniformity (Table 10).

Table 10: Classification of Subjects into Experimental Groups with X± S.D Weight & Height (Main Study)

Experimental Groups	Age	Weight (kg)	Height (m)	Men	Women
A1	44.3±9.9	75.8±10.1	1.67±0.07	9	11
A2	42.1±10.5	71.2±8.5	1.69±0.07	9	11
B1	44.6±10.7	73.9±8.0	1.68±0.06	9	11
B2	44.0±7.8	72.5±7.8	1.67±0.06	9	11
C1	43.8±11.1	72.1±7.0	1.66±0.07	9	11
C2	41.8±10.3	73.1±8.0	1.67±0.08	9	11
D1	45.0±8.1	70.5±7.5	1.67±0.07	7	8
D2	43.9±9.5	73.1±8.2	1.65±0.06	7	8
D3	44.5±10.0	73.0±9.3	1.67±0.08	7	8
	43.9±9.8	72.6±8.4	1.67±0.07	75	90

3.5.1 Investigative Stage (Experiment ONE)

This study investigated the cardiovascular responses, and side effects associated with three different cervical traction weights.

Group A = 7.5% total body weight (TBW), Group B = 10% TBW, and Group C = 15% TBW

One hundred and twenty (120) subjects participated in this study, with sixty cervical spondylosis patients and sixty normal volunteers (control).

Group A

Group A1. = 20 Cervical spondylosis patients investigated with 7.5% TBW cervical traction (CT).

Group A2 = 20 Normal/Control volunteers investigated with 7.5% TBW CT.

Group B

Group B1. = 20 Cervical spondylosis patients investigated with **10% TBW CT**..

Group B2 = 20 Normal/Control volunteers investigated with 10% TBW CT.

Group C

Group C1 = 20 Cervical spondylosis patients investigated with 15% TBW CT..

Group C2 = 20 Normal/Control volunteers investigated with **15% TBW CT**.

3.5.2 Therapeutic Stage (Experiment TWO)

This experiment was carried out to evaluate the therapeutic effectiveness of three different cervical traction weights (7.5%, 10% & 15% TBW) in the treatment of cervical spondylotic pain.

Forty five (45) cervical spondylosis patients were recruited into this study and were divided into 3 groups of fifteen (15) patients in each group.

Group D

Group D1= Treated with **7.5% TBW** traction, thermal therapy (TT) and therapeutic exercise (TE)

Group D2= Treated with 10% TBW traction, TT, and TE

Group D3= Treated with 15% TBW traction, TT, and TE.

In the investigative stage (**Experiment ONE**) the baseline physiological response was compared with responses during cervical traction treatment using different weights 7.5%, 10% and 15% of total body weight (TBW) at various time intervals.

Seven and half percent (7.5%) TBW was selected to reflect the lower weight (3.5 - 6.8kg) recommended by Kisner and Colby (1985) and Gould (1985), 10% TBW to reflect the weight range of 8.0-11.4kg limit recommended by Nwuga (1995) and Maitland (1977), and 15% TBW to reflect the upper traction load of 13.6kg recommended by Colachis and Strohm (1976).

Using each subject as his/her own control, the subjects' cardiovascular responses in a supine resting position (baseline) and under 3 experimental conditions (using 7.5%, 10% and 15% TBW) cervical traction was investigated. Subjects were required to wear unrestrictive clothing during experiment.

3.6 Instrumentation

The following materials were used in this study;

3.6.1 ECG Machine

Portable Electrocardiography (ECG) machine (KENZ 201) was used to investigate the PR interval and QRS complex.

3.6.2 Guthrie Smith Suspension Unit (Multi-Gym Model)

A Guthrie Smith suspension unit was employed to provide the cervical traction treatment. The arrangement included a head halter, a plinth, a pulley system, a spreader bar, and standard sets of weights.

3.6.3 Digital (Electronic) B.P meter

Blood pressure was monitored over the brachial artery using Automatic Electronic blood pressure meter (SE-2000). This device simultaneously monitors the HR. This instrument was standardised with the commonly used mercury sphygmamometer and gave same readings.

3.6.4 Height meter, weighing scale

The subjects' height and weight was recorded using the weight meter (Seca Model) in Kg and the height in meters.

3.6.5 Infra Red Radiation Machine (Thera-Lux Model).

Thermal therapy was administered with the aid of the standing luminous IRR (Infra Red Radiation) apparatus.

3.6.6 Mat Exercise

Therapeutic Exercise was done using the following regimen; isometric neck strengthening exercise, neck and shoulder stretching and flexibility exercise, pulley exercise and back strengthening exercise.

3.6.7 Visual Analogue Scale (VAS)

(Waterfield and Sim 1996; Olaogun et al. 2003).

This is a pain-measuring scale that allows the patient to describe his or her pain intensity in term of numbers. This scale is graded from 0-10. It is a progressive scale with the numbers being positively related to the pain intensity. Thus, 0 represents a situation of no pain, while 10 represents the most excruciating pain level.

3.6.8 Universal Goniometer (Andra)

This instrument was used to assess the range of neck movements on the subjects.

3.7 Experimental Procedure (Experiments ONE)

The approval of the Lagos University Teaching Hospital, (LUTH) Research and Ethical Committee was sought and obtained before the commencement of the experimental procedure.

All patients received the normal physiotherapy treatment during the experiment. They also had their BP, HR, and ECG recorded during the traction therapy apart from the usual pre-treatment vital sign assessment. The normal subjects (control) had free medical screening. They were offered lunch and transportation to the research venue during the experiment as an incentive.

3.7.1 Traction Procedure

Subjects were informed of the research protocol and were introduced to the equipment to be used. Subjects' weights and heights were measured. Following the above procedures, and after remaining in a quiet sitting position for 5-10 minutes, subject was placed in a supine position.

ECG was recorded according to the recommendations of the American Heart Association, (1966). The ECG results was analysed by a cardiologist. The blood pressure was measured with the electronic sphygmomanometer secured on the right arm through out the procedure.

A head halter was applied and strapped to the subject's head and neck. The supine lying cervical traction protocol described by Nwuga, (1976), was utilized. Two pillows were placed under the head and neck in order to provide appropriate neck flexion of 25-30 degrees recommended by most clinicians, and also a pillow under the knee, (Nwuga, 1976; Balogun *et al.*, 1990). This treatment protocol is demonstrated in Figures 18 and 19.

As the head halter and the external weight applied hinder the ability of subjects to communicate, a hand signal was arranged to enable the subjects terminate the treatment or respond at a specific period in case of the development of any discomfort or an unbearable reaction during the experiment.

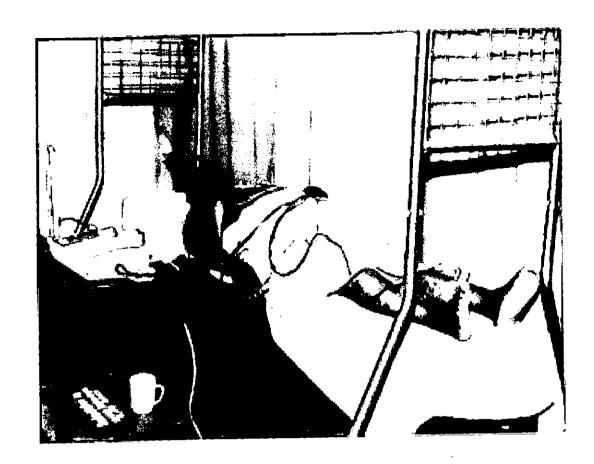


Figure 183: Procedure Showing The ECG And Guthrie Smith Suspension Unit.



Figure 19: Procedure Showing The Traction Weight.

Cardiovascular variables [SBP, DBP, HR, rate pressure product (RPP), PR interval, and QRS complex] were evaluated with subjects in the supine resting position (baseline). Following this, the static cervical traction was administered for 15 minutes. The cardiovascular responses (CVR) were taken at the end of 5 minutes, 10minutes and 15 minutes successively.

For all the subjects in the experiments one of this study, the cardiovascular evaluation followed the same procedure. The rate pressure product (RPP), which is a useful index of the cardiac stress and is known to be a valid predictor of the myocardial oxygen consumption at rest and during exercise was also evaluated using the formula; $RPP = SBP \times HR$, (Kispert, 1987).

Subjects remained seated for between 10-15 minutes post cervical traction application and were questioned concerning any discomfort (side effects) they might have felt during the cervical traction experiment.

3.7.2 Experimental Procedure (Experiment TWO)

- A brief history of the origin and duration of the pain and any associated problems was conducted.
- Assessment and examination of the back for each fresh referral was conducted to identify the painful areas. Palpation and localisation of the painful areas was by mild pressure via the thumb on the spinous process of the cervical spine and upper back region.
- A cervical mobility test (cervical movements in flexion, extension, lateral/side flexion and rotation in degrees) was done adopting Luttengens and Hamilton, (1997) protocol using the universal goniometer. Movements eliciting pain were noted. Intensity of pain was rated using the visual analogue scale and MVRS (Waterfield and Sim, 1996; Olaogun et al., 2003)
- Thermal therapy was administered using the luminous Infra red
 radiation (IRR) in prone position. Exercise regimen was given to all
 subjects, using the pulley, isometric neck strengthening exercise,
 neck and shoulder stretching and flexibility exercise.
- Experiment TWO was conducted four consecutive weeks with
 3- treatment sessions per week (Monday, Wednesday and Friday).
 The duration of each treatment session was between 45 minutes
 and 60 minutes per patient.

All subjects in Experiments TWO were instructed not to take any
non steroid anti-inflammatory drugs (NSAID) and other
analgesics for the duration of the study and were instructed to
report any medical problem immediately.

3.8 Computation

Following the data collection for Experiment One the mean cardiovascular responses in baseline position, end of 5 minutes, 10 minutes and 15 minutes were recorded.

For Experiments two and three the mean pain intensity and neck ROM were also evaluated.

3.9 Data Analyses

Obtained data were analyzed using descriptive statistics and inferential statistics with a Statistical Package (SPSS-PC, Version 7) design for window based IBM Compatible Computer.

3.9.1 Data Analysis; Experiment ONE

Analysis of variance (ANOVA) was employed to determine if there was a significant difference in the cardiovascular variables between the baseline position and the different experimental phases (end of 5, 10 and 15 minutes successively) within the same group for all the three groups (7.5% TBW, 10% TBW and 15% TBW).

Cardiovascular responses were monitored during recovery phase for safety reasons. Statistical analyses were not performed to compare the recovery data with the baseline and experimental phases.

For all statistical tests, the level of significance was set at 0.05 alpha level.

3.9.2 Data Analysis; Experiment TWO

Descriptive statistic of mean (X) and standard deviation (SD) was used to summarize the cervical ROM and pain intensities before (pre) treatment and at the end of the study (post treatment) in all groups.

Analysis of variance (ANOVA) was used to determine whether there was a significant difference between the mean neck ROM across the three groups for pre treatment and post treatment differently. Also, t- test was used to test for significant difference between pre and post treatment neck ROM within each group.

Kruskal Wallis independent test was used to test for pain intensity across the three groups, while Wilcoxon signed ranks test was used to determine significant difference between pre and post treatment pain intensity within each group

Significant difference was also set at 0.05 alpha level.

CHAPTER FOUR

4.0 RESULTS

4.1 Experiment ONE.

This study investigated the cardiovascular responses, and side effects associated with three different cervical traction weights.

Group A = 7.5% TBW, Group B = 10% TBW, and Group C = 15% TBW cervical traction weights respectively.

There was a drop in the blood pressure (SBP and DBP), and myocardial oxygen consumption (RPP) from the baseline in the three groups on the application of the cervical traction.

Analysis of variance comparing the baseline cardiovascular variables with the three phases during the traction therapy, that is, end of 5, 10, and 15 minutes successively for the 7.5% TBW traction groups did not show any significant difference at P<0.05 in both the patients (A1) and normal subjects (A2) for the SBP, DBP and RPP variables (Table 11).

The 10% TBW traction group B1 (patients) and group B2 (normal subjects) also experienced decreases in the SBP, DBP, and RPP, on the application of the traction. The decreases demonstrated significant difference (P<0.05) for the both the patients and normal subjects in the SBP and RPP variables and

patients group (B1) only for the DBP. The DBP for the normal subject (group B2) was not significant (Table 12).

The pattern of cardiovascular alterations experienced by subjects in 15% traction weight groups demonstrated a significant difference in both the patients and normal subjects groups for the SBP, DBP and RPP (Table 13).

An increase in **HR** above the baseline value was recorded at the end of 5 minutes with further increase at the end of 10 minutes and 15 minutes for the 15% TBW traction (Group C) in both patients (Group C1) and normal subjects (Group C2), Table 13. But this increase was very minimal in the 10% TBW traction groups (B1 and B2), Table 12. There was a slight drop in the HR at the end of 5 minutes, with a little increase at the end of 10 minutes and 15 minutes but the increase did not attain the baseline HR values at the end of the traction for the 7.5% TBW traction (Group A) in both the patient (Group A1) and normal subjects (Group A2), (Table 11).

The **HR** variables were not statistically significant (P>0.05) in any of the groups, (A, B, and C) comparing the baseline values with the three experimental phases during the traction application.

Table 11: Statistical Analysis Of Cardiovascular Responses Of Patients and Normal Subjects

During The **7.5% Traction** Using The ANOVA.

	_	7.5% - GROUPS (A1 & A2)					
		BL	5mins	10mins	15mins	P <i>value</i>	
SBPmmHg A1	(Patients=Pt)	130.3 ± 7.9	123.4± 6.2	125.9±6.8	127.3 ±7.1	0.079	
A2 (NS/	Control=Ctr)	127.4±73	121.9± 8.0	123.7±8.2	124.7 ±7.4	0.177	
DBP mmHg	A1 (Pt)	79.8:4.4	75.6 ± 4.8	76.4 ± 4.2	77.3 ± 4.4	0.065	
	A2 (Ctr)	80.6±5.5	77.7 ± 5.2	78.6 ± 4.9 ·	78.9 ± 5.1	0.326	
HR beats/min	A1 (Pt)	75.0 ± 8.6	74.0 ± 7.9	74.4 ± 8.0	74.6 ± 8.0	0.965	
	A2 (Ctr)	75.9 ± 8.1	74.9 ± 7.5	75.0 ± 8.0	.75.1 ± 7.6	0.939	
RPP X10 ²	A1 (Pt)	97.7 ± 16.0	91.3 ± 13.2	93.7 ± 14.0	95.0± 14.4	0.515	
	A2 (Ctr)	96.7 ± 13.7	91.3 ± 13.1	92.8 ± 13.0	93.7±13.1	0.612	
QRS X10 ⁻² sec	A1 (Pt)	8.0 ± .15	9.0 ± . 20	8.5 ±.17	8.0 ± .2	0.900	
	A'Z (Ctr)	8.0 ± .15	8.9 ± . 20	8.5 ±10	8.3 ±.14	0.817	
PR X10 ⁻² sec	A1 (Pt)	19.0 ± 0.06	19.8 ±. 0.06	19.0 ± 03	18.4±.06	0.890	
	A2 (Ctr)	18.0 ± .06	20.0 ±03	19.0 ±05	18.5± . 25	0.710	

^{*} The mean difference is significant at P < 0.05 level

KEY:

BL = Baseline Value

Mins = Minutes

RPP = Rate Pressure Product = SBP x HR

Table 12: Statistical Analysis Of Cardiovascular Responses Of Patients and Normal Subjects During The 10% Traction Using The ANOVA

		10% - Groups (B1 & B2)						
		BL	Smins	10mins	15mins	P <i>value</i>		
SBPmmHg B1 (Patients=Pt)		128.2 ±9.3	116.8±7.9	116.9± 6.9	119.4 ± 6.9	< 0.001*		
B2 (NS	i/Control≃Ctr)	125.4 ±12.3	116.0±8.2	117.2 ± 8.9	119.1±9.4	0.017*		
DBP mmHg	B1 (Pt)	81.5 ±6.2	75.5±5.4	75.3±5.0	75.7±5.0	0.001*		
•	B2 (Ctr)	83.4 ±7.8	77.5±6.2	77.9±6.1	79.3±6.9	0.055		
HR beats/min	B1 (Pt)	76.0±8.7	76.3±8.2	76.6 ±8.7	76.9±8.3	0.886		
	B2 (Ctr)	76.3±8.7	76.6±8.2	76.8 ±8.7	77.3±8.3	0.683		
RPP X10 ²	B1 (Pt)	97.4±18.2	89.1±12.6*	89.6±13.9*	91.8±13.3	0.024*		
	B2 (Ctr)	95.7±16.4	88.7±12.0*	89.8±112.6*	91.5±12.4	0.031*		
QRS X10 ⁻² sec	B1 (Pt)	8.5±.14	9.5±.10	9.0±.15	8.5± 15	0.835		
	B2 (Ctr)	8.2 ±.15	9.0±.20	8.7±.17	8.5±20	0.819		
PR X10 ⁻² sec	B1 (Pt)	18.7±.05	18.9±.06	17.9 ±.03	18.0 ±.06	0.899		
	B2 (Ctr)	18.0±.05	19.0±.06	18.0 ±.03	18.0 ±.06	0.900		
						•		

^{*} The mean difference is significant at P < 0.05 level

KEY:

BL = Baseline Value

Mins = Minutes

RPP = Rate Pressure Product = SBP x HR

Table 13: Statistical Analysis Of Cardiovascular Responses Of Patients and Normal
Subjects During The 15% Traction Using The ANOVA

		15% - GROUPS (C1 & C2)					
		BL	5 mins	10mins	15mins	P <i>value</i>	
SBPmmHgC1(Patients=Pt)		126.6±11.0	114.0±7.9	111.7 ±7.4	113.0 ±7.7	< 0.001*	
C2 (NS/Control=Ctr)		125.9±9.1	116.3±7.5	116.0 ±7.2	117.5 ±7.2	< 0.001*	
DBP mmHg	C1 (Pt)	82.7±6.8	73.0±6.1	71.6±4.0	72.3±3.9	< 0.001*	
	C2 (Ctr)	82.6±7.5	77.1±6.2	76.3±5.9	76.6±5.9	0.010*	
HR beats/min	C1 (Pt)	75.0±5.7	77.9±4.4	79.0±3.8	80.6±4.4	0.320	
	C2 (Ctr)	75.8±4.2	76.9±4.4	77.5±3.8	78.6±4.4	0.717	
RPP X10 ²	C1 (Pt)	96.0±13.9	88.8±9.6	88.2 ±8.6	91.1±9.4	< 0.001*	
	C2 (Ctr)	95.4±9.9	89.4±19.0	90.3 ±19.5	92.0±19.0	0.040*	
QRS X10 ⁻² sec	C1 (Pt)	8.0±.14	9.0±.20	8.5 ±8.6	8.5 ±.14	0.690	
٠	C2 (Ctr)	8.2±.14	10.0±.10	9.10±8.6	8. 0±.15	0.656	
PR X10 ⁻² sec	C1 (Pt)	18.3±.06	19.1±.03	19.0 ±.04	18.4±.28	0.758	
	C2 (Ctr)	18.5±.05	20.0±.06	19.8 ±.03	18.5±06	0.679	
•							

^{*} The mean difference is significant at P < 0.05 level

KEY:

BL = Baseline Value

Mins = Minutes

RPP = Rate Pressure Product = SBP x HR.

4.1.1 Effects of Traction Weights on Cardiac Muscles

The ECG variables (PR interval and QRS complex durations) were relatively stable (though with very mild alterations) in all the Experimental groups (A, B, & C) throughout the 15 minutes traction periods. The values were within the normal adult recordings of **0.12-0.20 sec** for **PR interval** and **0.06-0.10 sec** for **QRS complex** (American Heart Association, 1966). Statistical analysis did not show any significant difference (p> 0.05).

These results signified that the cardiac muscles were not adversely affected by any of the traction weights during the traction application.

4.1.2 Side Effects Associated With The Different Cervical Traction Weights (Experiment One)

Thirty (30/120) subjects (20 patients and 10 normal subjects, 25% of total subjects population) had various complaints (side effects): 4, 9 & 17 in subjects investigated with 7.5% (4/30 = 13.3%), 10% (9/30 = 30%), and 15% (17/30 = 56.7%) TBW cervical traction weight respectively (Table 14).

Ninety (90/120) subjects (75%) did not give any complaint. Seven subjects (between the age range of **50** and **65** years), one in group investigated with 10% traction (B1), and six in group (C1=4, C2=2) investigated with 15% TBW traction weights did not complete the experiment because of severe pain during the traction hence treatment was terminated.

Twelve subjects experienced neck muscle tenderness during the experiment, seven had mild headache, three subjects experienced dizziness when moving from supine position to sitting at the end of the traction and one complained of nausea at the end of the traction.

Moreover, twenty (67%) of the thirty subjects with side effects were above the age of 50 years. Also there was a progressive rise in the side effect with increases in age of the subjects. Fifteen percent (15%), 17%, 33% and 34% subjects were affected in the age groups (in year), 31-40, 41-50, 51-60 and 61 years and above respectively (Figure 20).

Table 14: Classification Of Side Effects Associated With The Various Traction Weights

	Groups A 7.5% TBW		Groups B 10% TBW		Groups C 15% TBW		
Side Effects	n=20 A1=Pt	n=20 A2=Ns	n=20 B1=Pt	n=20 B2=Ns	n=20 C1=Pt	n=20 C2=Ns	% Total
Mild Headache	1	-	2	1	2	1	5.8
Neck muscle Tenderness	2	1	2	2	3	2 .	10.0
Dizziness	-		1	•	1	i	2.6
Mild nauseas		-	- .	-	1	•	0.8
Treatment Terminated	-	· <u>•</u>	1	· -	4	2	5.8
Total	3	1	6	3	11	6	25.0

Keys

Pt = Patients

Ns = Normal subjects

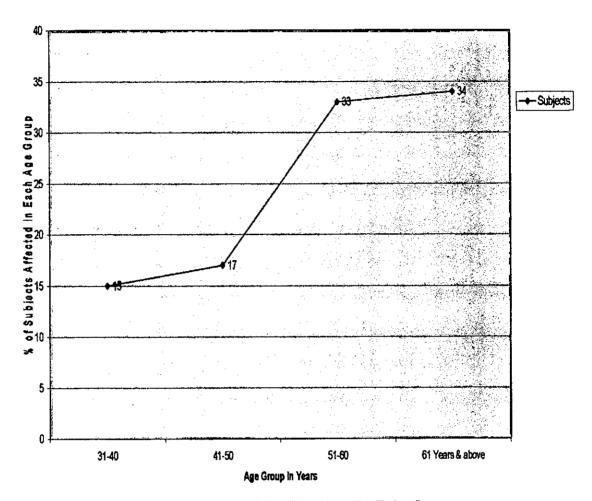


Figure 20: Classification of Side Effects According To Age Group

4.2 Experiment TWO Results

This Experiment Assessed The Therapeutic Effectiveness of The Different Traction Weights

Cervical Mobility (Range of Movement, ROM)

All the subjects had neck stiffness with associated pain of different severity during cervical movements (extension, lateral/side flexion, rotation and flexion) at the beginning of the study (pre-treatment). Analysis of variance do not established a significant difference pre-treatment for the cervical movements except lateral/side flexion only.

Post-treatment analysis established a significant difference for flexion and rotation, while extension and lateral flexion do not show a significant difference (Table 15). Significant difference was also established comparing the pre and post treatment ROM using the t-test for the four neck motions in group **D2**. Extension and rotation only exhibited significant difference for subjects in group **D3** and no significant difference for all the neck ROM for group **D1** subjects (Table 16).

Neck Pain

All the subjects reported different pain intensities pre-treatment. There was a progressive drop in pain intensity on a weekly basis till the end of the study in

all the groups (Figure 21). Pain intensity post treatment ranges from no pain (0) to moderate pain (5). Two (2) patients in the group treated with the 15% TBW traction weight did not complete the experiment because of severe pain during the traction therapy; hence treatment was terminated immediately.

There was no significant difference comparing pre-treatment, end of week 1, and 2, pain scores between the three groups (p>0.05), but a significant difference existed comparing end of week 3 and the post treatment pain scores between the groups (p<0.05).

The three traction weights (7.5%, 10%, and 15% TBW) resulted in pain relief because analysis of pre and post treatment pain intensity within each group using the Wilcoxon test established a significant difference (P<0.05). But from the mean ranks (D1 = 15.35, D2 = 13.25, D3 = 15.10), the group that was treated with the 10% TBW traction (D2) has the least mean rank and also the lowest pain rating post treatment. Hence 10% traction offered better and faster pain relief compared with the other two groups. There was just little difference in the mean rank and pain rating post treatment between the patients treated with 7.5% TBW (D1) and 15% TBW (D3) cervical traction, but because two patients had adverse reaction during traction that led to the termination of the treatment, we suggest extreme caution using 15% TBW cervical traction for safety reason.

Table 15: Analysis of Pre and Post Treatment ROM Across The Groups Using The ANOVA

	Pre-Treatment							
Cervical ROM (in Degree)	Grp D1	Grp D2	Grp D3	P <i>value</i>				
Flexion	37.4± 4.7	35.3 ± 5.0	40.1 ± 5.4	0.175				
Extension	41.8 ± 5.1	41.0 ± 4.9	39.7 ± 4.2	0.341				
Lateral Flexion	33.9 ± 3.6	37.9 ± 4.1	38.7 ± 4.5	0.020*				
Rotation	50.3 ± 7.5	49.7 ± 5.5	52.7 ± 6.2	0.407				
	Post-Treatment							
Flexion	41.5± 4.7	47.5 ± 5.8	46.7 ± 5.4	0.030*				
Extension	47.8 ± 6.0	52.6 ± 6.5	48.7 ± 4.2	0.348				
Lateral Flexion	37.7 ± 4.6	45.0 ± 3.9	42.5 ± 5.0	0.40*				
Rotation	55.7 ± 7.7	59.9 ± 7.1	61.0 ± 6.9	0.655				

^{*} The mean difference is significant at P < 0.05 level

Table 16: Analysis Of Cervical ROM, Pre and Post Treatment Within The Group Using The t-test.

Cervical Rom	Group D1			Group D2			Group D3		
(in Degree)	PreRx	PostRx	Pv.		PostRx			PostRx	
Flexion	37.4 ± 4.7	41.5 ±4.7	0.315	35,3 ± 5.0	47.5 ± 5.8	0.02*		46.7 ± 5.4	0.150
Extension	41.8 ± 5.1	47.8 ± 6.0	0.151	41.0 ± 4.9	52.6 ± 6.5	0.02*	39.7 ± 4.2	48.7 ± 4.2	0.035*
Lat. Flexion	33.9 ± 3.6	37.7 ± 4.6	0.457	37.9 ± 4.1	45.0 ± 3.9	0.04*		42.5 ± 5.0	0.601
Rotation	50.3 ± 7.5	55.7 ± 7.7	0.267	49.7 ± 5.5	59.9 ± 7.1	0.03*	52.7 ± 6.2	61.0 ± 6.9	0.04*

^{*} The mean difference is significant at P < 0.05 level

<u>Key</u>

PreRx = Pre-Treatment

PostRx = Post- Treatment

Pv. = P value

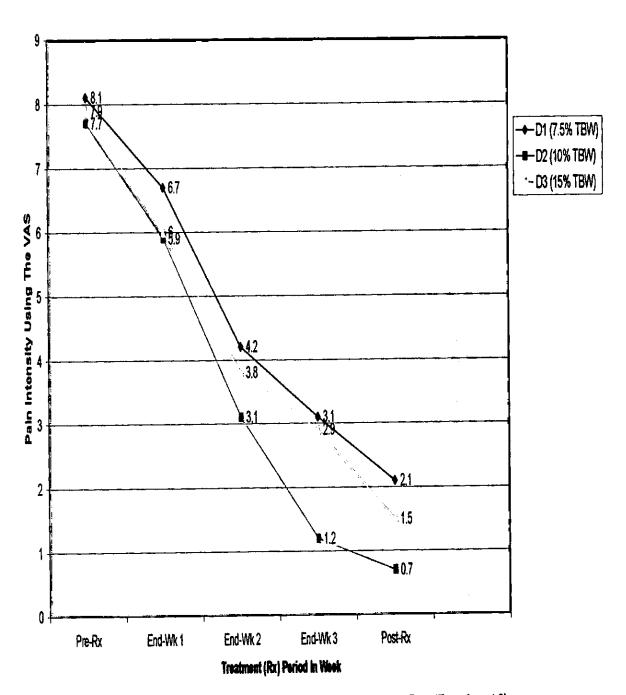


Figure 21: Analysis of Pain Intensity Using The Kruskal Wallis Test (Experiment 2)

CHAPTER FIVE

5.0

DISCUSSION

5.1 General Discussion

Many clinicians reported that patients following cervical traction therapy may experience various types of side effects, which may be mild/moderate and serious/severe (Maitland, 1977; Kisner and Colby, 1985; Akinbo *et al.*, 2004). Thirty (30) subjects comprising of twenty (20) patients and ten (10) normal subjects (Twenty five percent of total subjects population) in this study experienced various types of side effects that fall under the mild and severe classifications during and/or following cervical traction treatment.

Side effects are classified as mild/moderate and serious/severe in terms of clinical implication in reference to cervical traction application (Di-Fabio, 1999; Ogino et al., 2001). Those side effects that are classified as mild/moderate without any serious clinical consequence are; headache, neck muscle spasm and tenderness, and lassitude (tiredness/weakness) most of which disappear 10-30 minutes post traction application. Those classified as severe with serious clinical implication are nausea, dizziness, blurred vision post treatment, severe pain during treatment, and migraine. The serious or severe side effects are referred to as adverse effects which may lead to

cerebrovascular accident or other serious clinical complications (Di Fabio, 1999).

The present result also showed that age is a significant factor in cervical traction therapy as younger patients recorded less side effects compared with the older patients. That is, the older the patient the higher the risk of side effects due to cervical traction (Figure 20). Also patients above 50 years were able to tolerate the 7.5% TBW cervical traction without any adverse effects compared with the 10% and 15% traction weight. These revelations suggest that individuals react differently to cervical traction treatment. The results of this study also support the findings by Jette, (1985), that Intermittent supine traction does not produce relaxation of the upper trapezius muscle during and after treatment.

Continuous cervical traction causes sustained stretching and contraction of the skeletal muscle (Ellemberg *et al.*, 1994; Valtonen and Kiuru, 1999). The decrease in blood pressure observed during cervical traction in all the experiments may partly be due to the stretching of the baroreceptors located in the carotid sinuses (Maitland, 1977). In addition, direct pressure of the head halter over the region of the carotid sinus may be responsible for the decreased blood pressure responses observed in this study (Berne and Levy, 1983).

Dehn, (1980) and Berne and Levy, (1983) suggested that stimulation of the baroreceptors send impulses via the afferent nerves to the vasomotor and cardiac regions in the medulla to induce (1) slowing of the heart, (2) reduction of cardiac contractility, and (3) dilatation of peripheral arteries and veins. These physiological changes lower the blood pressure by decreasing cardiac output. These observations support our findings with the drop in the blood pressure following the application of the traction weight.

Regulation of blood pressure occurs by interacting relationships between cardiac function and arteriolar resistance to the blood flow. The most important receptors that bring information to the vasomotor and cardiac region for the control of blood pressure are the baroreceptor, chemoreceptor, and joint proprioceptor (Berger, 1982; Berne and Levy, 1983; Brannon *et al.*, 1993). However, the baroreceptors have a domineering effect over the latter two (Berne and Levy, 1983; Poulter *et al.*, 2005).

The mild headache, dizziness and mild nausea experienced by some of the subjects in the study may be due to decreased cardiac output as observed by Yates, (1972). Alternatively, the decreased cerebral blood flow may be attributed to stretching and compression of the vertebral arteries during the traction therapy (Bess and O'Sullivian, 1984). With their subjects in the supine position, Colachis and Strohm, (1976) demonstrated that a 13.6 kg tractive force is needed to cause separation of the cervical spine.

Joint proprioceptors are stimulated in response to separation of the vertebrae by manipulation or traction (Wyke, 1979; Junkin and Adria, 2002). It is possible that the 7.5% TBW traction was not adequate to excite the cervical joint proprioceptors hence the minimal side effect and less blood pressure alterations in this group. On the other hand, the greater stimulation of the joint proprioceptor by the 15% TBW traction may partly explain why more side-effects/adverse reactions were recorded in this group.

Joint proprioceptors, when stimulated, increase HR and cause vasoconstriction, resulting in blood pressure alterations (Conover, 1988). The stimulation of the joint proprioceptor may partly explain why blood pressure responses for the 15%TBW groups are slightly higher than the 10% TBW and 7.5%TBW respectively.

The HR responses for the 15% groups were slightly higher than the 10% and 7.5% groups. The increase in HR during the 15%TBW traction was an attempt by the circulatory system to regulate the decreased blood pressure. However, the increased HR did not appear to be sufficient to maintain the blood pressure at the baseline levels at the end of the 15 minutes treatment period.

The traction effects were not statistically significant (p>0.05) in terms of the selected ECG variables, that is, the QRS complex and PR interval durations. This finding indicates that the cardiac muscles contractility were not adversely affected by any of the traction weights during treatment, hence the ECG variables did not change significantly from the baseline during the experiment.

Pain in this study refers to the 'sensory dimension' having intensity, location and quality. It is understood to constitute one component of discomfort associated with degenerative lesions of the spine. Also the pain rating scale (VAS) used has been shown to posses differential properties and is clinically applicable in quantifying the pain experience of patients (Waterfield and Sim, 1996; Olaogun *et al.*, 2003).

The pathogenesis of adult cervical pain has been an issue of controversy for many decades. It is however, widely believed that degenerative changes associated with ageing are a major factor (Adams and Hutton, 1982).

Burkar and Beresford, (1979), best summarized the effects of ageing on the spine. They postulated that at about 32 years of age, spinal degenerative changes start manifesting in the majority of Individuals. And as degeneration progresses with age, the intervertebral disc water content decreases; consequently, it shrinks, tears and sometimes prolapses and no longer

correctly attaches, restrain, space, and position its vertebrae. Also its ability to absorb shock and distribute pressure becomes impaired while the cartilaginous end-plate become thin and crack, vertebrae experience osteoporosis and develop osteophytes, ligaments become lax and the facet joints become arthritic with some cracking sound (crepitation) during movement in some patients.

Also this degenerative change is known to place neurons (motor, sensory and autonomic) in a hyper-excitable state, increase blood vessel tone, and render connective tissues more susceptible to injury without necessarily being painful (Gunn and Milbrant, 1978). Since none of the subjects had a history of trauma, it is believed that subjects in this study shared the above characteristic, and that pain a major factor in spondylosis arose when these degenerative conditions affected pain-sensitive structures in their mechanical interface and constituted the noxious mechanical stimulus encoding the action potential on A-delta and C fibers which transmit noxious impulses to the central nervous system as observed by Johnson, (1997).

For all cases of cervical spondylosis, cervical traction was preceded by thermotherapy. This has likely resulted in effective pain reduction, muscle relaxation, and cervical joint flexibility as observed in this study. This finding support the study of Hattori *et al.*, (2002) who also observed that, that was an arrest of further degeneration of the cervical vertebrae and lesion of the intervertebral discs

Cervical hypomobility/stiffness has been attributed to neck pain in most cases of cervical spondylosis. All the patients with cervical spondylosis in this study presented with neck stiffness pre-treatment, induced by pain. All patients with pain reduction also had a corresponding improvement in neck mobility post-treatment. This result was in agreement with the assertion that pain reduction will automatically lead to improved neck mobility/flexibility (Caldwell and Krusen, 1962; Akinbo, 1996).

Most of the physical modalities commonly used for degenerative spinal diseases are most efficacious in combination (Nanno, 1994; Borman *et al.*, 2003; Akinbo *et al.*, 2004*), therefore the combination of thermal therapy, soft tissue massage, therapeutic exercise and cervical traction brought about pain relief in the three groups. That two patients did not complete the experiment due to discomfort (pain) as a result of the 15%TBW traction confirmed the findings of the earlier experiment where six subjects in the 15% groups terminated the experiment because of severe pain during the application of the traction weight.

All the traction weights brought about pain relief, considering the post treatment pain rating. There was no significant difference (p>0.05) comparing the pre-treatment pain scores in the three groups, but significant differences existed (p<0.05) comparing the post treatment pain scores in the three groups. Also, from the Kruskal Wallis post treatment mean ranks, the

patients treated with the 10%TBW cervical traction had the least mean rank and lowest pain rating post treatment, hence we can infer that the modalities used in that group offered better and faster pain relief compared with the modalities in the other groups.

The rationale for the non significant differences recorded pre-treatment for one of the neck movement (lateral flexion) may be attributed to the fact that normal neck mobility varies in people according to the different body types (ectomorph, mesomorph and endomorph), (Norkin and Levangie, 1992; Norkin and White, 1995; Luttgens and Hamilton, 1997). The ranges of normal flexion, extension, lateral flexion and rotation in the cervical region are given in table 3 (page 60) obtained from three different independent studies.

The results of pre and post-treatment analysis within each group, tables 16 (page 135) gives an insight into which treatment combination or modality offers better therapeutic result in terms of neck flexibility/ROM. Group **D2** exhibited a significant difference comparing the pre and post-treatment values for all the neck movements and also recorded the least post-treatment pain intensity. This revelations support the findings of Caldwell and Krusen, (1962); Panjabi, (1988) Norkin and White, (1995) and Akinbo, (1996) that reduction in neck pain will automatically lead to better mobility/flexibility in

neck stiffness because cervical hypomobility/stiffness has been attributed to neck pain in most cases of cervical pathology, specifically spondylosis.

5.2 Clinical Implication

The findings from this study revealed that the higher cervical traction weights, that is, 10% and 15% TBWs elicited more side effects compared with the 7.5% TBW traction. This study also showed that age is a significant factor in cervical traction therapy as younger patients and normal subjects recorded less side effects compared with the older subjects (Figure 20).

Moreover, subjects employed in this study reacted differently to cervical traction; therefore we recommend that clinicians must monitor the blood pressure responses of all patients especially the 'high risk' ones before, during, and after continuous cervical traction treatment.

"High risk" patients are individuals who are particularly sensitive to pressure over the baroreceptors area (neck region) and have difficulty tolerating even light collars (carotid sinus syndrome) or older patients who may have atherosclerotic plaques in their carotid arteries and are prone to fainting (carotid sinus syncope) must be treated with extreme caution using cervical traction.

In the five minutes post cervical traction application (recovery phase period), the subjects' blood pressure did not re-attain pre-treatment/baseline levels. This finding suggests that all patients especially the 'high risk' ones should be monitored in the sitting position following cervical traction treatment. As a precaution against adverse effects, all patients treated with cervical traction should not be allowed to leave the treatment area post cervical traction application until the vital signs (SBP, DBP and HR) have fully returned to stable values approximating the baseline as closely as possible.

Some healthy persons (normal individuals) can experience a considerable drop in blood pressure with external compression (using collar or traction application) of the neck region, (Zaverl and Ford, 2001). This phenomenon was demonstrated in this study as the cardiovascular alteration also occurs in normal subjects. In older subjects with atherosclerotic plaque formation in the carotid arteries, the response can lead to a precipitating drop in blood pressure, with fatal consequences.

Therefore to determine the suitability of patients for cervical traction, we urge clinicians to question patients regarding any unusual sensitivity they may have with respect to clothing around their neck, history of fainting, and precipitating causes. It would also be advisable to identify patients with history of hypotension or hypertension and those on medication. It is possible

that hypotensive patients will show a greater cardiovascular response to cervical traction.

It is known that the baroreceptor accommodates to high pressures over the course of a few day (Conover, 1988; Adeloye, 1999). Thus, the apparent cardiovascular perturbation observed with cervical traction may be less pronounced in hypertensive patients (Dehn, 1980).

Furthermore, manual weight applied during cervical traction treatment should be released gradually to prevent injury to the vertebral artery, which may lead to cerebrovascular accidents (Bose, 1999).

CHAPTER SIX

6.0 CONCLUSION AND RECOMMENDATIONS

6.1 GENERAL CONCLUSION

This study revealed that cardiovascular (SBP, DBP and RPP) alterations do occur in patients with cervical spondylosis and normal subjects using 7.5%, 10% and 15% TBW traction weights. The heart rates were relatively stable throughout the traction periods. The traction effects were not statistically significant in terms of the selected ECG variables, that is, the QRS complex and PR interval durations. This finding indicates that the cardiac muscles contractility was not adversely affected by any of the traction weights during treatment. Thirty (30/120) subjects experienced different side/adverse effects due to the application of the traction.

Also the study established the effectiveness of the three cervical traction weights (7.5%, 10% and 15% TBW) combined with thermal therapy and therapeutic exercise in the management of patients with cervical spondylosis. Ten percent (10%) TBW cervical traction recorded the best therapeutic

results in term of pain relief and neck flexibility/mobility compared with the 7.5% TBW and 15% TBW traction therapy. Therefore this study established scientifically the **10% TBW** cervical traction as the ideal weight with minimal side effect and with optimal/highest therapeutic efficacy.

In conclusion, degenerative disorders of the spine continue to be a significant cause of neck pain in today's population. Understanding of these problems continues to grow and with that, patients' understanding should follow suit. Affected patients should be aware of the possible treatment modalities, including medication/drugs, physiotherapy, braces, selective injections and surgery. The best patient is an informed one who understands the natural progression of these disorders, as well as the benefits, risks, and complications of available treatment methods.

6.2 RECOMMENDATIONS

- 1. Based on the results from this study, we recommend the use of 7.5% TBW cervical traction for patients above 50 years (elderly patient) because subjects in this age group were able to tolerate the 7.5% TBW cervical traction without any adverse effects. This traction weight (7.5%TBW) also recorded the lowest side effects compared with the other traction weights.
- The two adverse effects (severe pain during treatment resulting in termination of treatment and dizziness) associated with the 10% TBW cervical traction occurs in subjects above 50 years (a 57 year woman and 63 year man).
- 3. Severe side effects were not recorded for any subjects below 50 years treated with the 10% traction. Therefore 10% TBW cervical traction is recommended for patients of 50 years and below because it recorded the highest therapeutic effectiveness with minimal side effects.

- 4. Based on the high adverse effects associated with the 15% TBW cervical traction, clinician should avoid this traction weight.
- However, cervical traction treatment is a relatively safe procedure when the ideal weight and safety measures are taken.

REFERENCES

Adam M.A. and Hutton W.C. (1982). Mechanics of prolapsed intervertebral disc. *International Orthopedics*, **5**; 6:249-253.

Adeloye A. (1999). Syndrome of Cervical spondylosis in Blantyre, *Malawi* East Afr. Med. J. **76**; 10: 575-9.

Akinbo S.R.A. (1996). Clinical presentation and management of Osteoarthritis: The Lagos University Teaching Hospital Approach. *Journal Nig. Med. Rehab.Ther.* **5**; 2: 51 – 55.

Akinbo S.R.A., Oke D. A., Okanlawon A.O. (2004). The effect of cervical traction on the cardiovascular system in normotensive Volunteers.

Nig. J. Health & Biomedical Sciences, 3; 2: 56

Akinbo S.R.A., Odebiyi D.O., Giwa S.O. (2004).* Comparative Study of the Effect of Thermal Therapy and TENS in the management of cervical spondylotic pain. *Nig. J. of Medical Rehabilitation*, **9**: 1-4.

Akinrolabu B.O. (1992). The pattern of cervical spondylosis in University College Hospital Ibadan between 1965 & 1990. *J. Nig. Soc. of Physiotherapy*. **2**:17 – 26.

American Academy of Orthopeadic Surgeons (1965). Joint Motion: Methods of measuring and recording. AAOS, Chicago.

American Heart Association Committee Report, (1966).

Recommendations for Standardization of leads and the specifications for instruments in electrocardiography and ventriculography. *Circulation supplement*. 57-62.

American Medical Association (1988). Guides to the evaluation of permanent impairment, ed 3. AMA, Chicago.

Balogun J.A., Abereoje O.K., Olaogun M.O., Okonofua F.E. (1990). Cardiovascular responses of healthy subjects during cervical traction. *Physiotherapy Canada*, **42**: 16-22.

Berger R.A. (1982). Applied exercise physiology. Philadelphia: Lea and Febiger, 181-189.

Berne R.M. and Levy M.N. (1983). Physiology. St. Louis: CV Mosby, 542-549.

Bess P.K. and O'Sullivan S.D. (1984). Cardiovascular responses during nonweight-bearing and touchdown ambulation. *Physical Therapy,* **64**;1: 14-18.

Betge C. (1981). The effects of intermittent traction on the cervical spine. *Swiss Ann;* **7**; 10: *100-5*.

Bhatoe H.S. (2000). Spinal cord injury without radiological abnormality in adult. *Neurol. India*, **48**; 3: 243-8

Bishop B. (1980). Pain: Its physiology and rationale for management. *Physical Therapy.* **60**; 21-23.

Bogduk N., Tynan W., Wilson A.S. (1981). The nerve supply of the human lumbar intervertebral disc. *J. Anat.* **27**; 132: 39-56.

Boivie J. (1990). Central pain syndromes. Campell JN (2ed); An Updated Review, IASP Press, *Seattle*, p.23-29.

Bonica J.J. (1990). The Management of Pain, 2nd ed. Philadelphia: Lea and Febiger, 90 - 97.

Borg S. (1982). Objective pain rating scale for clinical evaluations. *Physical Therapy*, **17**: 317-321.

Borman P., Keskin D., Bodur H. (2003). The efficacy of lumbar traction in the management of patients with low back pain. *Rheumatology Int. J,17;* 28:82-6.

Bose K. (1999). The efficacy and safety of eperisone in patients with cervical spondylosis; results of a randomised, double-blind, placebo-controlled trial. *Clin. Pharmacology,* **21**; *3: 209-13.*

Brannon F.J., Foley M.W., Starr J.A., Black M.G. (1993).

Cardiopulmonary Rehabilitation: Basic Theory & Application. FA Davis

Publication. 1st ed. 340-351.

Brena S.F. and Chapman S.L. (1983). Management of Patients with Chronic Pain, Great Neck, NY: PMA Publications 105 - 110

Burkart B.S. and Beresford A.W. (1979). The ageing intervertebral disc. *Physical Therapy*, **58**; 8: 969-971.

Cailliet R. (1981). Neck and arm pain, ed 2. FA Davis, Philadelphia; pp. 102-104.

Caldwell J.W. and Krusen E.M. (1962). Effectiveness of cervical traction in treatment of neck problems: Evaluation of various methods. *Arch Phys Med. Rehabil.* 62; 43: 214-19.

Capuano-Pucci D. (1991) Intratester and Intertester reliability of cervical range of motion. Arch Phys Med Rehabil 72: 338-343.

Carmicheal S.N. and Burkart S.L. (1979). Clinical anatomy of the lumbosacral complex. *Phys Ther* **59**; 56-60.

Chapman C.R. (1985). Pain measurement: An overview. Pain. 22; 67-70.

Coker H.A.B. (2003). Pain modulator and inhibitors. *Journal Nig. Med. Rehab. Therapists*, **8**; 2: 7-19.

Colachis S.C. and Strohm B.R. (1976). Effect and duration of Intermittent cervical traction on vertebral Separation. *Arch Phys Med Rehab.* **6**; 47: 353 – 359.

Conover M. (1988). *Understanding Electrocardiography: Physiology & Interpretation concepts.* Mosby, 5th ed. 559-564

Constantoyannis C., Konstantinou D., Papadakis N. (2002). Intermittent cervical traction for cervical radiculopathy caused by volume herniated disks. *J. Manipulative Physical Therapy*, **25**;3: 188-189.

Dabbs V. and Lauretti W.J. (1995). 'A risk assessment of cervical manipulation versus NSAIDs for the treatment of neck pain', *Pain*, **18**; 8: 530-536.

Dects D., Hands K.L., Hoop S.S. (1977). Cervical traction: A comparison of sitting and supine position. *Phys Ther.* **9**; 57: 255 – 261.

Dehn M.M. (1980). The effects of rehabilitation exercise on cardiac patients:. *Am. J. of Nursing;* **80**: 9: 50-55.

De-Lacerda F. G. (1980). 'Effect of angle of traction pull on upper trapezius muscle activity', *Journal of Orthopedic Sports Physical Therapy*, **1**, 6, 205-209.

Di Fabio R.P. (1999). Manipulation of the cervical spine: Risks and Benefits. *Phys. Ther.* **19**; 79: 50 – 69.

Downie W.W. (1978). Studies with pain rating scales. *Annals of Rheumatic Diseases*. **37**; 378-381.

Egwu M.O., Moromoke A.M., Nwuga V.C.B. (2003). Effect of a cervical oscillatory pressure on neck pain and some cardiovascular variables. *Physiotherapy.* **89**; 6:666-74.

Ellemberg M.R., Honet J.C., Treancer W.J. (1994). Cervical radiculopathy. *Arch Phys Med. Rehabil*, **75**; 3: 342-52.

Farfan H.F. and Sullivan J.D. (1967). The relation of facet orientation to intervertebral failure. *Can. J. Surg.* 10: 179-185.

Finch L. and Melzack R. (1987). Objective pain measurement, a case for increase clinical usage. *Physiotherapy Canada*; **34**: 343-345.

Fitzgerald M. and Woolf C.J. (1984). Axon transport and sensory C fibre function. In Chahl LA, Szolcsanyi J, Lembeck F (eds): Antidromic Vasodilatation and Neurogenic Inflammation, Akademiai Kiado, Budapest, p.119-140.

Fouyas I.P. and Statham P.F. (1998). Operatives treatment of cervical spondylosis. *Br.J.Neurosurg*, **12** 6: 594-5

Fouyas I.P., Statham P.F., Sandercock P.A. (2002). Cochrane review on the role of surgery in cervical spondylotic radiculomyelopathy. *J. spine*, **27**; 7: 736-47.

Frymoyer J.W. and Gordon S.L. (1989). New perspective on low back pain. America Academy of Orthopaedics Surgeons. 2nd eds. Park Ridge, Illinois **Ganong W.F.** (1999). Blood Circulation, *Medical Physiology*, 19th ed. Lange Medical Book, 493-600.

Goel V.K. (1988). Moment-rotation relationship of the ligamentous occipitoatlanto-axial complex. *Biomeech. 8*: 673-680.

Gould J.A. (1985). The spine and dysfunctions. *Orthopaedic and sports* physical therapy, **12**; 10: 518 – 549.

Gracovetsky S. (1987). The spinal Engine. 1st eds Springer-Verlag, New York.

Gunn C.C. and Milberant T. (1978). Early subtle signs in low back pain. Spine, 3;3: 251-272.

Harris P.R. (1977). Cervical traction: Review of Literature and treatment guideline. *Phys Ther J. 7*; *57*: *910* – *914*.

Hattori M., Shirai Y., Aoki T. (2002). 'Research on the effectiveness of intermittent cervical traction therapy using short- latency somatosensory evoked potential', *Journal Orthopaedic*, **7**, 2, 208-216.

Hirshberg R.M., Al-Chaer E.D., Lawand N.B., Westlund K.N., Willis W.D. (1996). Is there a pathway in the posterior funiculus that signals visceral pain? *Pain* 67: 291-305.

Hoheisel V., Koch K., Mense S. (1994). Functional reorganization in rat dorsal horn during experimental myositis. *Pain* **59**: 111-118.

Holt S.T. and Yates J. (1996). Management of cervical discogenic spondylosis. *Phys Ther; 17: 106-10*.

Hurwitz E. L., Aker P. D., Adams A. H. (1996). 'Manipulation and mobilization of the cervical spine: A systematic review of literature'. *Spine*, 12, 21, 1746-1759.

Irvine A. (1992). *Cervical* spondylotic myelopathy; Diagnosis and management. *Spine,* **7:** 128-31.

Jensen M.P. (1996). Mechanisms of neuropathic pain. In Campbell JN (ed) - An Updated Review, IASP Press, *Seattle*, p.77-86.

Jensen M.P., Karoly P., Braver S. (1986). The management of clinical pain intensity: A comparison of six methods. *Pain.* 27; 117-126.

Jette D.U. (1985). Effect of intermittent, supine cervical traction on the myoelectric activity of the upper trapezius muscle in subjects with neck pain. *Phys Ther.* **5**; 65: 173-76.

John A.D. and Tony G. (2003). The chiropractic management of two cases of cervical spondylotic radiculopathy. *J. Can. Chiropr Assoc.* **20**; 47: 121-6.

Johnson M.I. (1997). The Physiology of the sensory dimension of clinical pain. *Physiotherapy*, **83**; 10: 526-535.

Judovick B.D. and Nobel G.R. (1997). Traction Therapy, a study of resistance force: a preliminary report on a new method of lumbar traction. *Am. J. Surg.* **93**: 12: 108 – 112.

Junkin J.B. and Adria L.M. (2002). Comparative term post-treatment changes in hyper divergent patients early cervical traction treatment. *Orthopaedic*, **72**; 1: 5-14.

Kent B.A. (1974). Anatomy of the trunk: A review. Part 1. Phys Ther. 7: 54-59

Kisner C. and Colby L.A. (1985). Therapeutic Exercise: *Foundation and Techniques. Philadelphia: FA. Daris, 483 – 485*.

Kispert C. P. (1987). Clinical measurement to assess cardiopulmonary function. *Phys Ther J.* **19**; 67: 1886 – 1890.

Klougart N., Leboeuf C., Rasmussen L.R. (1996). Safety in chiropractic practice, part I: the occurrence of cerebrovascular accident after manipulation of the neck in Denmark from 1978- 1988. *J Manipulative Phys Ther.* 19: 371-377.

Kottke F.J. and Lehmann J.F. (1990). Krusens Handbook of Physical Medicine and Rehabilitation, 4th ed. Philadelphia: W.B. Saunders, 57 - 67.

Kurutzne K. M., Bene E., Lovas A. (2001). Biomechanical experiments for measuring traction lengthening of lumbar spine during weight bath therapy. *Orthop J,* **143**;13: 673-684.

Lee K.Y., Siff T.E., Heggeness M.H., Esses S.I. (2002). Anterior cocking screw plate fixation in cervical spondylosis. *Am. J. Orthop, 31; 1: 27-30.*

Leek J.C. (1982). Principles of Physical Medicine and Rehabilitation. Orlando: Grune and Stratton, 275–350.

Likhaceav M. and Ermilor P.V. (2002). Traction of the cervical spine region in osteochondrosis of cerebral circulatory insufficiency. *Vopr Leeh Fiz Kult,* **21**; 2:41-6.

Luttgens K. and Hamilton N. (1997). Kinesiology: Scientific Basis of Human Motion, 9th edn. Brown & Benchmark: London 1997. , pp78-85.

Maitland G.D. (1977). Vertebral manipulation. Boston: *Butterworths* publication: 109 – 118.

May S.J. (2001). Patient satisfaction with management of back pain (part 1). Physiotherapy, 87; 1: 4-9.

Melzack R. and Wali P.D. (1965). The challenges of pain (2nd ed) London: Penguin books. Pp 105 -147

Merskey H.M. (1986). Pain terms. Pain, suppl. 3: pp 215 - 221.

Merskey H.M. (1991). The definition of pain. *European Journal of Psychiatry*, **9**; 6: 153-159.

Merskey H.M. and Bogduk N. (1994). Classification of Chronic Pain, 2nd ed., IASP Press, Seattle, p.211 - 217.

Michlovitz S.L. and Wolf R. (1996). Thermal Agents in Rehabilitation. Philadelphia: F..A. Davis, 98 - 105.

Nanno M. (1994). The effect of intermittent cervical traction on muscle pain. Flow metric and electromyography studies on the cervical Para spinal muscles. *Nippon. Gakkai – Zasshi. J.* **7**: 371 – 85.

Nelson M.A. (1979). Surgical management of cervical spondylosis. *Physiotherapy*, **65** *4*: *106-9*.

Neuwirth M. and Marsicano J. (1996). Cervical spondylosis: diagnosis, symptomatology, and treatment. *Orthop Nurs*, **15**; 1: 31-6.

Norkin C.C. and Levangie P.K. (1992). The vertebral column; Joint Structure And Function. F.A. Davis publication, Philadelphia: pp 125-170.

Norkin C.C. and White D.J. (1995). Measurement of joint motion: A guide to goniometry. F.A. Davis publication, Philadelphia: pp 181-198.

Nwuga V.C.B. (1976). Manipulation of the spine. Baltimore: *Williams and Wilkins;* 82 – 95.

Nwuga V.C.B. (1995). An in sight into Back pain and manual therapy (Part I). J. Nig. Soc. of Phys, **5**;13: 8 – 12.

O'Brien E. (1991). Twenty four hours ambulatory blood pressure in men and women aged 17-80 years: The Allied Irish Bank Study. *Journal Hypertension*, **9:** 355-360.

Ogino M., Kawamoto T., Kim P. (2001). Proper management of the rotational vertebral artery occlusion secondary to spondylosis. *Clin Neurol Neurosurg.* **35**;40: 250-3.

Olaogun M.O.B., Adedoyin R.A., Anifaloba O. (2003). Reliability and concurrent validity of VAS and modified verbal rating scale (MVRS) of pain assessment in adult patients with knee Osteoarthritis in Nigeria. *South African Journal Of Physiotherapy*. **59**; 2: 12-15.

Oison V.K. (2001). Chronic whipiash associated disorder treated with home cervical traction. *J. Back & Musculoskeletal Rehab.* **19**; 9: 181-90.

Onei D., Tuziaci M. Sari H. (1989). Computed Tomographic investigation of the effect of traction on lumbar disc herniation. Spine, 69, 14. 62-90.

Panjabi M. (1986). Three dimensional movements of the upper cervical spine. Spine. 13: 726-730.

Paris S.V. (1990). Introduction to spinal evaluation and manipulation.
Institute Press St, Augustine Florida; 305-310

Paris S.V. and Nyberg R. (1989). Innervations of the posterior lateral aspect of the lumber intervertebral disc. Abstract, Presented at the International Society of the Lumbar Spine, Kyoto, Japan.

Poulter N. R., Wedel H., Sever P. S. (2005) Role of BP and other variables in the differential cardiovascular event rates noted in the Anglo-Scandinavian Cardiac outcome trials-Blood Pressure Lowering Arm (ASCOT-BPLA). *The Lancet*, **366**; 9489: 907-913.

Rahim K. and Stambough R.V. (1992). Cervical radiculopathy. *Arch Phys Med. Rehabil.* **75**; *3: 342-52*.

Rehbai R., Boudawara M.Z., Ben Y., Ben M.H. (2002). Syringomyelobulbia associated with cervical spondylosis. Pathophysiology and therapeutic implications. *Neurology J, 5*, 48: 120-3.

Roberts S., Menage J., Urban J. P.G. (1989). Biomechanical and structural properties of the cartilage end-plate and its relation to the intervertebral disc. *Spine* **14**; 166-173.

Ross M. (1997). Manipulation and back school in the treatment of low back pain. *Physiotherapy*, **83**; 4: 181-183.

Saitoh K., Kai M., Mitamura J., Seo N. (2002). Cervical spondylosis revealed after general anaesthesia. *Masui*; **51**; 6: 667-9.

Saunders H.D. (1983). Use of spinal traction in the treatment of neck and back conditions. *Clin Orthop.* **83**; 79: 31-37.

Shakoor M.A., Ahmed M.S., Kibria G. (2002). Effects of cervical traction and exercise therapy in cervical spondylosis. Bangladesh Med Res 2; 28:61-9.

Sheppard R.J. (1983). The value of exercise in ischeamic heart disease: A cumulative analysis. *Journal Cardiac Rehabilitation* **3**: 294-298.

Singh A., Crockard H.A., Plats A., Stevens J. (2001). Clinical and radiological correlates of severity and surgery-related outcome in cervical spondylosis. *J. Neurosurgery*, **22** 94:189-98.

Slaby F.J., McCune S.K., Summers R.W. (1994). Gross Anatomy; In the practice of Medicine. Lea & Ferbiger, Field Parkway USA.

Smith-Petersen M.N. (1990). Routine examination of low back cases with particular references to differential points between lumbosacral and sacroiliac regions. *J. Bone and Joint Surg.* 6: 819-826.

Swezey R.L., Swezey A.M., Warner K. (1999). Efficacy of home cervical traction therapy. *Am. J. Phys Med Rehab*, *9*; *78*: *30-32*.

Sypert G.W. and Cole H.O. (1999). Management of multilevel cervical spondylosis with myelopathy. *Surgical Neurology*, **51**;1:4-5.

Tamura M., Shinso S., Ogawa K., Tamiya T., Takasu T. (1999). The Clinical features of cervical spondylosis with boule musculature. *Rinsho Shinkeigaku* 39; 1: 1097-103.

Tapio D. and Hymes A.C. (1987). New frontiers in Transcutaneous Electrical Stimulation. Minnetonka, Minn: LecTec Corp; 107.

Taylor A.G., West B.A., Simon B., Skeleton J., Rowlingson S. (1983). How effective is TENS for acute pain? *American Journal of Nursing*, **83**, 1171-1174.

Thompson A. and Rowland J. (1995). Diagnosis of pain and parasthesia:

Merit Textbook of Neurology, 9th Ed. Williams & Wilkins, USA.

Tsiptsios I., Fotiou F., Sitzogblou K. (2001). Neurophysiological investigation of cervical spondylosis. *Electromyography Clin. Neurophysical*, **41**; 5: 305-13.

Uttely D. and Monro P. (1989). Neurosurgery for cervical spondylosis. *Br. J. Hosp.* **42**; 1: 62-70.

Valtonen E.J. and Kiuru . (1990). Cervical traction as a therapeutic tool. Scand. J Rehab. Med. 19; 2: 29-36.

Wall P.D. (1996). The mechanisms by which tissue damage and pain are related. In Campbell JN (ed) Pain - An Updated Review, IASP Press, Seattle, p.123-126.

Waterfield J. and Sim J. (1996). Clinical assessment of pain by the visual analogue scale. *British Journal of Physic. Therapy Rehabilitation*, *66*; 3: 94-97.

Weigel H. (1995). The articular surfaces of the sacroiliac joint and their relation to the sacrum. *Acta. Anat.* **17**; 22: 65-70.

Wyke B. (1979). Neurology of the cervical spine joints. *Physlotherapy*, **65**; 9: 72-76

Yates D. (1972). Indications and contraindications for spinal traction. *Physiotherapy*, **58**; 12: 55-57.

Young I.A., Burns S.P., Little J.W. (2002). Sudden unset of cervical spondylotic myelopathy during sleep: a case report. *Arch phys. Med. Rehab*, 83; 3: 427-9

Zaveri G.R. and Ford M. (2001). Cervical spondylosis: The role of anterior instrumentation after decompression and fusion. *J. Spinal discord*, **14**; 1: 10-6.

Zhuang L. (2000). Twenty one cases of vertebral-artery-type cervical spondylosis treated with acupuncture and *moxibuston*. *J. Tradit Clin Med.* **20**; 4: 280-4.

Zybergold R.S. and Piper M.C. (1985). Cervical spine disorder- A comparison of two techniques of traction. *Spine.* 85; 102-107.