UNIVERSITEIT VAN PRETORIA
UNIVERSITY OF PRETORIA
YUNIBESITHI YA PRETORIA

A biomechanical investigation into the effects of decompressive surgery on the stability of the lumbosacral joint in the dog

by

Gregory Stuart Irvine-Smith

Submitted to the Faculty of Veterinary Science,

University of Pretoria, in partial fulfilment

of the requirements for the

MMedVet (Chir.) (Small Animals) degree

January 2009

PROMOTOR: NE Lambrechts

Associate Professor, Department of Companion Animal Clinical Studies

Onderstepoort, Faculty of Veterinary Science

University of Pretoria

Current address:

Purdue University

Department of Veterinary Clinical Sciences

West Lafayette

Indiana, USA

Contents

Summary

The primary objective of this biomechanical study was to investigate the effect of decompressive surgery, specifically dorsal laminectomy and discectomy, on the stability of the lumbosacral joint in the dog. Different size laminectomies were compared with respect to their effect on lumbosacral stability.

A total of eighteen lumbosacral motion units were collected from cadavers and divided into three groups. Group 1 was a control group and received no modification, Group 2 specimens received mini-dorsal laminectomies and discectomies (lamina of L7 caudal to the dorsal spinous process excised, lamina of S1 not affected) while Group 3 specimens received standard dorsal laminectomies and discectomies (75% of L7 lamina and 50% of S1 lamina excised). All specimens were potted in aluminium tubing and mounted in a four-point bending jig and tested in a load cell. Specimens were stressed to 21° in dorsiflexion and ventroflexion. The relevant surgical modification was then performed and the specimens re-tested to 21° in dorsiflexion and ventroflexion. All specimens were then tested to failure in ventroflexion. Force and angular displacement was recorded and used to obtain load-deformation curves for each specimen (5 curves for each specimen). From the load-deformation curves the stiffness (gradient of the graph) was determined at three set angles of deflection. These points were 6°-8°, 12°-16° and 18°-20°. The percentage change in stiffness for each specimen in both dorsiflexion and ventroflexion was obtained. Peak force at failure and angular deformation at failure were obtained when tested to failure in ventroflexion.

When examining the overall stiffness of the specimen (dorsiflexion and ventroflexion and all angles of deflection) mini-dorsal laminectomy was shown to result in a 48.3% reduction in stiffness ($P < 0.001$) while standard dorsal laminectomy and discectomy resulted in a 59.8% reduction in stiffness ($P < 0.001$). These results were statistically significant. The difference

between the two different types of laminectomies could be described as approaching significance (P=0.066). Larger group size would be required to determine whether this is in fact statistically significant

Dorsal laminectomy combined with discectomy does have an effect on the stability of the lumbosacral joint. This may contribute to the relatively high recurrence rate following surgical treatment of degenerative lumbosacral stenosis especially in large breed highly active dogs. The study provides further support for decompressive surgery combined with a stabilisation technique when treating degenerative lumbosacral stenosis. It also provides potential support for the use of mini-dorsal laminectomies.

Acknowledgements

Without the contributions of many colleagues, this project would not have been possible. I extend my sincerest thanks and gratitude to the following people:

Prof. Nic Lambrechts (Promotor) who provided motivation, guidance and support throughout this project and my post-graduate degree.

Dr Peter Turner for support and guidance throughout my post-graduate studies and for proof reading this dissertation

To all the staff at **Bryanston Veterinary Hospital** , especially my colleagues **Dr Bruce Irvine-Smith**, **Dr Chris Collie** and **Dr Remo Lobetti**, for providing me with the necessary motivation, encouragement and time to complete this project.

To the **Johannesburg Animal Anti-Cruelty League** for allowing me to collect the cadavers from them.

Sr Cindy-Lee Price of the Johannesburg Animal Anti-Cruelty League for her assistance in collecting cadavers.

Sr Tracy Phillips and **Sr Theresa Malan** of BVH for their assistance with specimen preparation.

Dr Peter Thompson for his guidance, assistance and statistical analysis.

Mr Danie Drent and staff at the Sasol Laboratory of the Department of Mechanical and Aeronautical Engineering, University of Pretoria, for their assistance with specimen testing and data analysis.

Dr Kenneth Joubert for his encouragement and support while writing this dissertation

The **University of Pretoria** for the financial support that made this research project possible.

Introduction

"Canine lumbosacral disease" is a collective term used for a variety of conditions that result in compression and/or inflammation of the *Cauda equina* and its vasculature [7, 26]. The *Cauda equina* is formed by the roots of the sacral and caudal spinal nerves, and occupies the vertebral canal from the level of the 5th to the 7th lumbar vertebrae (L5 – L7) caudally [26]. Conditions that predispose to lumbosacral disease include malformation/malarticulation of the lumbosacral joint, infection (discospondylitis of L7/S1), osteochondrosis of L7, trauma to the lumbosacral joint, decreased circulation of the vasculature (ischaemia) of the *Cauda equina*, and degenerative changes within the lumbosacral intervertebral disc [26]. The term "*Cauda equina* syndrome" refers to a clinical syndrome characterized by inflammation and/or compression of the *Cauda equina.* The clinical symptoms of this syndrome progress from lumbosacral pain, to pelvic limb ataxia, to faecal and urinary incontinence, with increasing severity of the condition [26,29]. The most common cause of *Cauda equina* syndrome is degenerative lumbosacral stenosis (DLS), which usually involves Hansen type II (fibroid) degeneration of the lumbosacral (L7 – S1) intervertebral disc [8]. It is the author's opinion that instability of the lumbosacral joint may play a significant role in the pathophysiology of DLS. The instability of the lumbosacral joint may contribute to the so called secondary changes of DLS (including hypertrophy of the *Lig. flavum* and articular processes' joint capsules) which result in stenosis of the vertebral canal and/or intervertebral foramen, which in turn causes compression, and potentially, ischaemia of the *Cauda equina.*

Surgical and conservative treatment options exist for clinical cases of DLS. Surgical treatment usually consists of decompression by means of a dorsal laminectomy (with or without discectomy), facetectomy or foraminotomy. Surgical treatment of DLS results in a clinical improvement in 67-93% of cases [7, 8,18]. However, as many as 18-33% of cases have recurrence of clinical symptoms within 18 - 24 months post-operatively [6, 7, 9, 29]. Force plate analysis of dogs with DLS that were treated surgically revealed improved function within 6 months, but not a return to normal [18, 35]. Recurrence after surgery and incomplete recovery may be due to numerous factors [7, 13, 29]. One possibility is that surgical decompression may exacerbate lumbosacral

instability, and therefore result in further/ongoing degenerative disease and/or compression and/or inflammation.

DLS is more commonly seen in certain breeds, the German shepherd dog being over represented [7, 13, 33]. Because this breed is commonly used as a working dog (e.g. police, military /security and guide dogs), there are significant financial implications to this condition in these animals. Surgical treatment requires an extended rest period, and some individuals will never return to normal strenuous activity. The relatively high recurrence rate has further financial implications.

The purpose of this study is, therefore, to investigate what effect surgical decompression of the lumbosacral region (dorsal laminectomy and discectomy) has on the stability of the lumbosacral joint and whether the size of the laminectomy has an effect of lumbosacral stability.

Index of Tables

Literature Review

Results

Index of Figures

Materials and Methods

Results

Chapter 1: Literature Review

1.1 Anatomy and vertebral kinematics

The lumbosacral articulation involves three distinct joints: the L7 intervertebral symphysis (with intervertebral disc) and the paired synovial articular process joints between L7 and S1 [26].

The stability of the lumbosacral joint is provided by three complementary systems. These systems are the passive system, the active system and the neural control system. The passive system is further divided into dorsal and ventral compartments [21, 22].

- The dorsal compartment of the passive system consists of :
    - the lamina, pedicles, paired articular process (facet) joints and the dorsal spinous process of L7;
    - the lamina and median sacral crest of S1;
    - the *Lig. flavum* (between the caudal lamina of L7 and the cranial lamina of S1), the supraspinous ligament (between the tip of the spinous process of L7 and the median crest of S1), and the interspinous ligament (between the caudal surface of the dorsal spinous process of L7 and the cranial edge of the median sacral crest).

- The ventral compartment of the passive system consists of :
    - the vertebral bodies of L7 and S1;
    - the L7/S1 intervertebral disc;

- the dorsal longitudinal ligament (situated on the dorsal aspect of the vertebral bodies, within the vertebral canal), and the ventral longitudinal ligament (on the ventral aspect of the vertebral bodies).

The components of the dorsal system, specifically the supraspinous ligament, interspinous ligament, *Lig. flavum*, laminae of L7 and S1, and articular process joints are the structures that are most likely to be disrupted during laminectomy, facetectomy and foraminotomy. The intervertebral disc, specifically the dorsal *Anulus fibrosus* and the *Nucleus pulposus*, as well as the dorsal longitudinal ligament are disrupted during discectomy.

The active system consists of the epaxial and sublumbar musculature and their tendons, while the neural control system is responsible for activation of and feedback to the active system [21, 22].

The relative importance of the passive vs. the active systems is impossible to deduce from *in vitro* biomechanical studies as the effect of the active system is impossible to evaluate in these studies. It is possible, and in fact probable, that the active and neural systems are more important than previously thought.

The tension surface of the vertebral column (dorsal surface in ventroflexion and ventral in dorsiflexion) will be subjected to tremendous force. Therefore the dorsal and ventral components will have altered importance and function in ventroflexion compared to dorsiflexion [28].

The lumbosacral joint serves as the hinged connection between the pelvic limbs (including the pelvis) on one side and the vertebral column on the other. Most of the force used for forward propulsion is generated by the pelvic limbs and is transmitted to the vertebral column and rest of the body through the lumbosacral joint. The actual force transmitted through the lumbosacral joint is unknown. At a stationary stance position it is generally accepted that approximately 40% of body weight is supported by the hindlimbs, this is however not the case during ambulation. It has been calculated that hip joint reaction force (acting on the acetabulum) during a three legged stance ranges between 0.73 and 1.04 times body weight [25]. It is therefore evident that during forward propulsion there are increased forces (>40% body weight) acting across the joints of the hindlimb. We can therefore assume that the force transmitted across the lumbosacral joint would be equivalent to at least 50% of body weight.

The range of motion between consecutive lumbar vertebrae increases as one proceeds caudally, with the lumbosacral joint having the largest range of motion (2.5 times the range of motion of the L6-L7 joint) [29]. A previous study into the three dimensional motion pattern of the lumbosacral joint in 25 clinically normal dogs determined the range of motion for the lumbosacral joint [4]. The results indicated a range of motion in flexion-extension of 37° (±5.7°). The range of motion in lateral bending was 9.5° (±2.6°), and in axial rotation was 2.0° (±1.2°) [4]. Movement in this joint is thus largely restricted to extension and flexion in a sagittal plane [18, 26]. There is also limited movement in lateral bending and axial rotation. The vertical orientation of the articular process joints limits both lateral bending and axial rotation, while allowing flexion and extension in the sagittal plane [18]. The lumbosacral joint is therefore a high motion, high stress joint, when compared to the rest of the intervertebral joints of the thoracic, thoracolumbar and lumbar spine.

The lumbosacral disc is the largest disc of the vertebral column. The disc consists of an outer region, the *Anulus fibrosus* and an inner region, the *Nucleus pulposus.* The inner *Nucleus pulposus* has a gel-like consistency and helps to dissipate stress associated with normal movement while the outer *Anulus fibrosus* contains the *Nucleus pulposus.*

The *Conus medullaris* is the caudal end of the spinal cord and is generally situated at the level of L6 (sometimes over the body of L5). This does vary according to the dog's size, in smaller dogs (<7kg), the *Conus medullaris* may be situated within L7/S1. It is therefore evident that stenosis of the lumbosacral vertebral canal in large breeds results in nerve root compression and not spinal cord compression. The nerve roots that traverse the lumbosacral joint include the 7th lumbar nerve, the sacral nerves and the caudal nerves. These nerve roots contribute to the following peripheral nerves: *N. gluteus cranialis, N. gluteus caudalis, N. ischiadicus, N. tibialis (*branch of *N. ischiadicus), N. peroneus communis (*branch of *N. ischiadicus), N. cutaneus femoris caudalis, N. pudendus* as well as the *Nn. pelvini.* (See Table 1-1)

The clinical symptoms seen in degenerative lumbosacral stenosis (DLS) are related to compression and compromised function of the nerves listed above. From the anatomical location of the nerve roots crossing the lumbosacral joint, it is evident that compression of these nerve roots will result in lower motor neuron (LMN) signs in the pelvic limbs. The most basic unit of skeletal muscle is the motor unit. This consists of the lower motor neuron and its effector organ i.e. skeletal muscle fibres. The cell body of the lower motor neurons of the pelvic limbs are situated in the 4th lumbar spinal segment to the 3rd sacral spinal segment (spinal segments L4 – S3) Lower motor neuron signs are characterized by one or more of the following: absent or depressed spinal reflexes, flaccid muscle tone, poor muscle strength, muscle fasciculation, muscle atrophy, root signature and an easily expressed bladder.

Nerve	Spinal cord segment	Effector organs
N. gluteus cranialis	L6 – S1	*M. gluteus medius*, *M. gluteus profundus* and *M. tensor fascia latae*
N. gluteus caudalis	S1 – S2	*M. piriformis*, *M. gluteus medius* and *M. gluteus superficialis*
N. ischiadicus	L6 – S1	*Mm. gemelli*, *M. quadratus femoris*, *M. obturator internus*, joint capsule of the hip, *M. biceps femoris*, *M. semitendinosus*, *M. semimembranosus* and sensation to the skin of the caudal crus
N. peroneus communis	L6 – L7	Sensation to the skin of the lateral aspect of the distal thigh, stifle and proximal crus, *M. extensor digitorum lateralis*, *M. peroneus longus*, *M. tibialis cranialis*, *M. extensor digitorum longus* and *M. extensor digitorum brevis*
N. tibialis	S1 – S2	*M. gastrocnemius*, *M. flexor digitorum superficialis*, *M. popliteus*, *M. flexor digitorum profundus*, joint capsule of the stifle, sensation to the skin of the hock and the tarsal join capsule
N. cutaneous femoris caudalis	S1 – S2	Sensation to the skin of the lateral caudal and medial surfaces of the proximal thigh and the skin dorsal to the *Trochanter major*
N. pudendus	S1 – S3	*M. sphincter ani externus*, *M. urethralis*, *M. ischiourethralis*, *M. bulbospongiosus*, *M. ischiocavernosus*, *M. retractor penis / clitoridis*, sensation to the skin of the perineum and caudomedial thigh
Nn. pelvini	S1 – S2	Rectum, descending colon and the erectile tissue of the penis / clitoris via the *Plexus pelvinus* on the lateral wall of the rectum

Table 1-1: List of the nerves that arise from spinal roots that traverse the lumbosacral joint, their spinal cord segment of origin and their effector organs

1.2 Pathophysiology

As mentioned in the Introduction, DLS is thought to be the most common cause of *Cauda equina* syndrome in large breed dogs. Stenosis of the vertebral canal at L7/S1 and/or the intervertebral foraminae at L7/S1 is due to a combination of changes including intervertebral disc prolapse, hypertrophy of ligamentous structures, osteophyte formation and possible subluxation of the lumbosacral joint [21]. The clinical signs may be caused by nerve root compression and/or ischaemia of the nerve roots. Compression may also be dynamic in nature [3, 17]. Neuritis in the absence of *Cauda equina* compression has also been proposed as a possible aetiology [17]. There is controversy and uncertainty within the literature as to what changes are initiating changes versus those that are subsequent changes.

The pathophysiology of DLS may revolve around the degeneration of the lumbosacral intervertebral disc [7]. This is believed to be initiated by mechanical stress (e.g. working dogs), malformation of the osseous structures, osteochondrosis, and age related changes. Constant repetitive mechanical stress, especially in very active working dogs (large breeds), is thought to be the most important factor [3, 5, 7, 15, 26, 29]. Lumbosacral joint instability or hypermotility is suspected to play a major role in initiating DLS. Very active large breed and working dogs are at much higher risk for developing the condition [7]. There is an increased incidence in male compared to female dogs as well and an increased incidence in German Shepherd Dogs (GSD) [7, 13, 17, 33]. There appears to be an increased incidence of more sagittally orientated articular process (facet) joints at the lumbosacral joint in GSDs [33]. This may alter the motion characteristics of the lumbosacral joint. Articular process (facet) joint tropism as seen in the GSD breed may potentially also contribute to an increased incidence of DLS in the breed [33].

Degenerative lumbosacral stenosis is characterized by fibroid degeneration of the lumbosacral disc with Hansen type II disc protrusion. The intervertebral disc consists of the outer *Anulus*

fibrosus and the inner *Nucleus pulposus*. Hansen type II disc protrusion is characterized by partial rupture of the annulus and subsequent bulging of the dorsal annulus. As the disc degenerates it loses its ability to perform its function of pressure distribution, shock absorption and joint stabilization. Other changes characterising DLS include lumbosacral subluxation, hypertrophy of the articular process joint capsule and *Lig. flavum*, as well as peri-articular osteophyte formation [3, 7, 8, 26, 29]. These changes lead to collapse of the lumbosacral disc space: the articular joint spaces, in turn, collapse resulting in subluxation of the lumbosacral joint. This collapse/subluxation results in increased laxity of the dorsal *Anulus fibrosus, Lig. flavum* and the joint capsules of the articular process joints. Due to the laxity, these structures will have a reduction in their contribution to the stability of the lumbosacral joint. The response of the body to this instability is to initiate osteophyte formation around the articular process (spondyloarthrosis) and vertebral end plates (spondylosis), as well as hypertrophy of the ligaments and soft tissues surrounding the lumbosacral joint. The result is osseous and soft tissue impingement of the neural and vascular structures of the *Cauda equina* and stenosis of the vertebral canal and the foramina.

The secondary or compensatory response to disc degeneration and resulting lumbosacral instability are responsible for the narrowing of the vertebral canal with potential compression of the *Cauda equina*. There is also a dynamic component to DLS. That is to say, the diameters of the vertebral canal at L7-S1 and of the L7-S1 intervertebral foraminae vary with flexion and extension of the lumbosacral joint [17]. The compression will be exaggerated in extension and reduced during flexion. Instability or hypermotility of the lumbosacral joint could potentially exacerbate this dynamic component of DLS [26, 36].

The bulging *Anulus fibrosus* can be substantial and may be the main cause of the nerve root compression. This compression may be central (vertebral canal stenosis) or may extend laterally into the L7-S1 intervertebral foramen (foraminal stenosis) with resulting compression of the L7 nerve root. Narrowing of the intervertebral foramen may be caused by bone and/or soft tissue

proliferation. The soft tissue component includes a bulging dorsal *Anulus fibrosus* or hypertrophied articular process (facet) joint capsule. The intervertebral foramen has been divided into three zones: entrance, middle and exit zones [10, 36]. The foraminal stenosis is therefore classified according to the zone were the stenosis occurs [10, 36].

Another factor causing narrowing of the vertebral canal can be ventral subluxation of the sacrum relative to the L7 vertebra, so called “stepping” [33, 34]. Elongation of the lamina of the sacrum cranioventrally into the vertebral canal was identified in 31% of dogs with DLS in one study [34] and may significantly contribute to vertebral canal stenosis. Instability of the lumbosacral joint may result in dynamic interference with blood flow to the nerve roots. This results in nerve root ischaemia which significantly contributes to the neurological / clinical symptoms. The term used to describe this phenomenon i.e. dynamic nerve root ischaemia is claudication [26].

DLS results in compression of the nerve roots, i.e. it affects the peripheral nerves of the *Cauda equina* rather than the spinal cord. The condition is also generally chronic in nature (symptoms present for 14 months (mean) in one study) [13]. Chronic compression results in neurological damage by causing demyelination, damaging axonal membranes and ultimately destroying axons. The ability to recover and heal is dependent on the extent of the injury and the duration of compression. Three grades of injury may be seen, neuropraxia, axonotmesis and neurotmesis. Neuropraxia is a transient dysfunction of the neuron due to myelin damage without disruption of the axon. Potential causes include compression, ischaemia, or blunt trauma. Fortunately this is the most common type of injury seen with DLS. Axonotmesis occurs when axon integrity is lost but the endoneurium and Schwann cell sheath remain intact. Neurotmesis is the most severe type of injury and occurs when the entire nerve is severed. Regenerating nerves (following neuropraxia and axonotmesis) may result in paresthesia and hyperaesthesia. This may in turn cause self-mutilation.

Four different causes for the pain in degenerative lumbosacral stenosis have been proposed [26]. Discogenic pain from receptors in the *Anulus fibrosus*; osteoarthritic pain from receptors in the periosteum, joint capsule and ligaments; meningeal pain from stimulation/irritation of the dura mata, and radicular pain from nerve root compression, inflammation and/or compromised blood flow to the nerve roots. Nerve root ischemia or claudication is dynamic and is experienced when the blood vessels are prevented from dilating during physical activity. Discogenic pain is suspected to be one of the main cause of pain in dogs with DLS.

1.3 Clinical Symptoms

Symptoms of DLS (*Cauda equina* syndrome) are neurological dysfunction and pain as a result of nerve root compression, entrapment and/or inflammation as well as discogenic pain. Symptoms of DLS may also be caused by neural ischaemia secondary to vascular compression (claudication). Pain (lumbosacral) and difficulty rising and jumping are the most common presentation [13]. An abnormal tail carriage may be associated with DLS due to lumbosacral pain; this may include lower tail position, decreased or absent tail wag, pain on manipulation (hyperextension) and decreased sensation.

Faecal and urinary incontinence may be seen in advanced cases [3, 5, 6, 7, 8, 26]. Urinary incontinence will usually precede faecal incontinence. In advanced cases decreased anal tone will be noted. Loss of sensation in the perineum and depressed to absent spinal reflexes of the pelvic limb may be noted. Conscious proprioception deficits are not a consistent clinical finding in dogs with DLS [10]. When one considers the nerve roots that traverse the lumbosacral joint, it is apparent that compression of these structures will result in LMN signs to the pelvic limbs. The specific nerves affected and the LMN signs are described in section 1.1. Another important clinical observation may be an exaggerated patella reflex. This should not be confused with an upper motor neuron (UMN) lesion. The reason for the exaggerated patella reflex is the loss of the

antagonistic action of the muscles innervated by ischiadic nerve. Moderate to severe atrophy of the hamstring and gastrocnemius muscle groups may be detected [13]. Paraesthesia and hyperaesthesia of the tail, lateral digits and perineum may be present [13]. This may generally result in some degree of self-mutilation of the tail, perineum or pelvic limb.

1.4 Diagnosis

Diagnostic confirmation of DLS is based on an accurate history, clinical examination, neurological examination, survey radiographs (including stressed views), myelography, epidurography, computed tomography (CT) and magnetic resonance imaging (MRI) and electrophysiological examination.

Differential diagnoses include but are not limited to L/S discospondylitis, orthopaedic disease (e.g. coxarthrosis) meningitis, iliospoas myopathy etc.

Physical examination should include direct palpation over the lumbosacral joint, hyperextension of the lumbosacral joint and lifting by the tail base. These techniques are used to confirm the presence of lumbosacral pain. Unilateral pelvic limb lameness may be present due to unilateral compression of the L7 and/or S1 nerve root. Neurological examination will confirm a LMN lesion to the pelvic limb/s in more severe cases. Lower motor neuron signs in the pelvic limbs include absent or depressed spinal reflexes, flaccid muscle tone, poor muscle strength, muscle fasciculation, muscle atrophy, root signature and an easily expressed bladder. Sensation should be assessed over the perineum and lateral digits. Anal tone should be examined as signs of incontinence are generally related to a much poorer prognosis [8].

Survey and stress (ventro- and dorsiflexion) radiographs of the lumbosacral joint should be obtained. They are used to evaluate for any abnormalities of the area: spondylosis, subluxation, transitional vertebrae, L/S osteochondrosis, discospondylitis, spondyloarthrosis, vertebral

neoplasia etc as well as lumbosacral stepping. Myelography is generally not diagnostic due to the fact the spinal cord (subarachnoid space) usually ends within the body of L6. Epidurography and discography were the imaging modalities of choice prior to MRI and CT being readily available [3426]. Epidurography by definition is injection of radiographic contrast into the epidural space while discography is injection of radiographic contrast into the intervertebral disc itself. The newer imaging modalities, MRI and CT, give far superior diagnostic information [34]. Both MRI and CT allow cross-section imaging of the lumbosacral region. It is also important that all images are obtained with the pelvic limbs in extension as this will exacerbate any lumbosacral instability and results in further narrowing of the vertebral canal. MRI gives excellent soft tissue imaging, while CT gives excellent bone detail. MRI is generally considered the gold standard for imaging of the nervous system due to its soft tissue detail.

CT and especially MRI have become the imaging modalities of choice for the diagnosis of DLS, although due to their sensitivity, a number of clinically insignificant lesions may be detected. MRI provides a sensitive, accurate and non-invasive imaging modality with excellent soft tissue visualization. In a MRI study investigating the association of *Cauda equina* compression and clinical signs in 27 dogs, no correlation was found between the severity of MRI compression and severity of clinical signs [17]. Another study concluded that although there was a high degree of agreement between CT and MRI findings in dogs with DLS, the degree of agreement between diagnostic imaging findings (CT and MRI) and surgical findings was lower [34]. The risk of over-diagnosing DLS with these modalities exists, and the clinical findings must therefore always be carefully correlated with the CT or MRI findings [8, 26]. Up to 68% of dogs with DLS have MRI evidence of foraminal stenosis [2] although only 50% of these cases showed clinical signs associated with foraminal stenosis [2, 17]. A clinical study using MRI to investigate dogs showing signs of lumbosacral pain and/or neurological deficits revealed MRI findings consistent with surgical findings in 7 dogs [2]. It is clear that MRI offers significant benefits over radiography (survey and contrast) in the diagnosis of DLS due to its ability to accurately define soft tissue compression of the *Cauda equina*.

1.5 Treatment

Various options exist for the treatment of DLS in dogs. Non-surgical treatment consists of rest and anti-inflammatory medication. Conservative treatment is however unlikely to give long term relief, especially in very active dogs or dogs with severe symptoms [6]. Conservative treatment is therefore usually reserved for dogs showing signs of pain only or for cases were anaesthesia and surgery may be contraindicated for other reasons (age, renal disease etc.). Reoccurrence of symptoms is common once treatment has ceased and normal activity resumes. Conservative or medical management generally results in a poor outcome in large breed active dogs, especially working dogs [7, 8, 15, 26, 29]. Surgical treatment is normally the treatment of choice, especially in active or working dogs, those that have shown recurrence of symptoms following conservative treatment and those with neurological deficits [5, 6, 7, 8, 26,]. Surgical decompression is achieved by means of a laminectomy (with or without partial discectomy), foraminotomy and facetectomy. The extent of the laminectomy is variable and may be extended cranially and caudally dependant on the extent of the compression. The laminectomy will however usually include excision of the dorsal spinous processes of L7 and S1 [18]. The generally accepted dimensions of a lumbosacral laminectomy are at least 50% of the lamina of L7 and the entire lamina of S1 [26]. Facetectomy or foraminotomy may be required to decompress nerve roots as they exit via the intervertebral foramina. Facetectomy involves excision of the caudal articular facet of L7. This may be done unilaterally or bilaterally [26, 29]. Facetectomy is however no longer generally recommended due to the risk of increased hypermotility and instability of the lumbosacral joint [10]. Foraminotomy is the enlargement of the intervertebral foramen in order to decompress the nerve root as it exits the intervertebral canal. Treatment of foraminal stenosis is difficult when using a dorsal laminectomy approach. The difficulties encountered include poor access and visualisation of the nerve root and foramen, especially the middle and exit zones [10]. This increases the risk of inadequate decompression and iatrogenic nerve injury. Endoscopically-assisted lumbosacral foraminotomy has recently been described [36]. The study, in normal animals, was able to demonstrate good visualization of the nerve root within the canal, which permitted a minimally invasive foraminotomy, through a mini-dorsal laminectomy [36]. Lateral foraminotomy via a lateral approach has also recently been described as a treatment option in dogs with DLS and foraminal

stenosis of L7/S [10]. The lateral foraminotomy may also be performed bilaterally and can be combined with a dorsal laminectomy if required [10].

Dorsal laminectomy may be combined with a stabilization technique that results in distraction and/or fusion [3, 27]. Stabilization techniques have been developed due to the implication that instability has a role to play in the underlying pathogenesis of *Cauda equina* syndrome. Various techniques exist, the most commonly used being the placement of either positional or lag screws across the articular facets of the L7-S1 joint. The articular cartilage may be removed and the area packed with autogenous cancellous bone to promote fusion [3]. Threaded or non-threaded pins may be used instead of screws. These pins may be driven through the dorsal spinous process of L7 before traversing the L7-S1 articular process (facet) joint [27]. Other techniques that may be used include pedicle screws and rod fixation [5, 18], pins and polymethylmethacrylate (PMMA) [19] and external fixation [1]. A distraction-fusion technique without laminectomy has also been described [27]. It does however remain debateable as to whether a true arthrodesis is achieved with these techniques or whether a fibrosis of the articular process (facet) joints results in increased stability.

There are no recognized criteria to differentiate between those cases that require stabilization and those that do not. It appears largely personal choice and depends on an intra-operative visual assessment of the “stability” of the lumbosacral joint. Radiographic assessment of lumbosacral stepping, especially during stressed views my provide addition information regarding instability. Specific guidelines are not available with regard to assessment of lumbosacral stability and it is conceivable that a large range of normal variation exists. There is some documented evidence to suggest that stabilization should be promoted. This includes the fact that the pressure on the *Cauda equina* is dynamic i.e. the compression is worsened with the lumbosacral joint in a neutral or extended position as opposed to a flexed position [3, 17]. This fact is also demonstrated by fluctuations in epidural pressures during flexion/extension of the lumbosacral joint [3, 17]. It is also often very noticeable (intra-operatively) that there is overriding of the L7-S1 articular processes

(facets) i.e. the L7 articular facets are caudally displaced relative to the S1 articular facets [3]. Distraction and fusion procedures have the advantage that they distract L7 from S1 slightly thereby "opening" the intervertebral foramen at L7/S1[10, 27]. They also alleviate or reduce the dynamic component of DLS [10].

1.6 Prognosis

The prognosis for dogs with DLS treated surgically is largely dependent on the severity of the clinical symptoms prior to treatment. Dogs with faecal and or urinary incontinence have a poorer prognosis than those showing signs of pain only [8, 15]. Force plate analysis has shown significant improvement in function of the pelvic limbs following surgical treatment of dogs with DLS over a 6 month period but with very few individuals returning to normal [35]. Initial improvement post-operatively may be followed by deterioration and recurrence of the clinical symptoms.

Reoccurrence following surgical decompression (laminectomy) is reported to be as high as 18 – 33% [6, 7, 9, 29]. There are a number of factors that may be responsible for this: Compression of the neural tissues may reoccur as a result of scar tissue formation (laminectomy membrane). The incidence of this may be reduced by placing a free autogenous fat graft over the laminectomy defect [13]. Persistent discogenic pain may be present in some cases. [5, 15, 29]. Another potential cause of persistent pain and dysfunction includes undiagnosed foraminal stenosis, as discussed previously foraminal stenosis is seen in up to 68% of dogs with DLS [10].

A major cause of reoccurrence is believed to be persistent instability and hypermotility [29]. As described earlier, instability is thought to play a pivotal role in the initial pathogenesis of DLS. This instability may however still be present after surgery and therefore result in reoccurrence or persistence of clinical symptoms. The other possibility is that the decompressive surgery (dorsal

laminectomy, discectomy, facetectomy or foraminotomy) may in fact exacerbate any underlying instability of the lumbosacral joint and therefore result in reoccurrence of the symptoms, the so called “failed back surgery syndrome” [5]. Any ongoing hypermotility or instability would result in further soft tissue proliferation and impingement of the neural structures [5, 29]. This reoccurrence is much more likely in very active working dogs. This has economic implications as significant time and financial resources are spent training these dogs.

Should it be found that specific components of the decompressive surgery contribute to lumbosacral instability and ongoing clinical disease; steps could be taken to reduce the effects or magnitude of these effects by either modification of the decompressive technique (limited laminectomy) or by adding a stabilization technique. This may significantly reduce the rate of reoccurrence.

1.7 Spinal Biomechanical Studies

Numerous studies have been performed evaluating the effect of various procedures on the stability and stiffness of the canine spine. The majority of these studies have focused on the thoracolumbar and lumbar regions and utilized a four-point bending jig to test spinal stiffness. Their relevance to the lumbosacral joint is difficult to accurately evaluate due to the unique anatomical and biomechanical characteristics of the lumbosacral joint.

A study by Schulz *et al* [24] investigating the biomechanics of the canine thoracolumbar vertebral motion unit in lateral bending showed no significant decrease in stiffness in specimens undergoing unilateral and bilateral facetectomy and fenestration compared to control specimens [24]. A statistical difference in stiffness was found between discectomy specimens and specimens not subjected to discectomy. Discectomy has the most significant effect on spinal stiffness in the thoracolumbar and lumbar regions [24]. Circumferential discectomy resulted in decreased stiffness

and load at failure in lateral bending in the thoracolumbar region [24, 29]. Hemilaminectomy combined with fenestration had the greatest effect on stiffness of the thoracolumbar spine with fenestration the most significant cause [12, 19, 29]. In a study on decompressive surgeries and dorsal compartment injuries (of the L3-L4 motion unit) Smith *et al* found that bilateral facetectomy decreased bending strength by 56%, and that a dorsal laminectomy in addition to the bilateral facetectomy reduced the bending strength by 75% [28]. The same study showed a 36.2% decrease in vertebral column rigidity in extreme flexion after excision of the supraspinous and interspinous ligaments and a 62.4% decrease in ultimate bending strength [28]. Fenestration has also been shown to significantly increase instability of the C5-C6 disc space [16]. (See Table 1-2)

Study (ref)	Site	Testing methodology	Results
Smith *et al* [28] 1988)	**L3 – L4**	Swing arm 4 point bending jig	Hemilaminectomy did not significantly affect spinal stability. Excision of supra- and interspinous ligaments decreased flexion bending strength by 62%. Dorsal laminectomy decreased flexion bending strength by 75%.
Schulz *et al* [24] (1996)	**T13 – L1**	Swing arm 4 point bending jig	Discectomy resulted in significant decrease in stiffness and load at failure.
Macy *et al* [16] (1999)	**C5 – C6**	Specimen loaded as a cantilever beam	Range of motion and individual flexion and extension angles significantly increased after fenestration.
Hill *et al* [12] (2000)	**L1- L4**	4 point lateral bending	Anulus fibrosus is an important stabilising structure between vertebrae. Fenestration had the greatest negative effect on stability.
Smith *et al* [29] (2004)	**Lumbosacral**	4 point bending jig	Dorsal laminectomy had no significant effect of Lumbosacral stiffness. Dorsal laminectomy and discectomy decreased mean stiffness by 31% in ventro-flexion.
Meij *et al* [18] (2007)	**Lumbosacral**	4 point bending jig, cyclical testing	Dorsal laminectomy and partial discectomy did not affect neutral zone or the range of motion. Neutral zone and range of motion were significantly decreased following pedicle screw-rod fixation.

Table 1-2: Comparison of biomechanical studies of the thoracolumbar, lumbar and lumbosacral spine (arranged in chronological order).

The role of the active system (i.e. the epaxial musculature) on stabilization of the vertebral column is not addressed by these biomechanical studies. Studies investigating the effect of epaxial musculature on the stability of the vertebral column show that stretching of the interspinous ligaments in cats caused electromyographic activity in the epaxial muscles adjacent to the vertebral segment. This muscle contracture potentially plays a very important role in stabilisation of the lumbar and lumbosacral spine. The location of the receptors responsible for this contraction of epaxial muscles is unknown [30, 32]. Excision of the interspinous ligaments and dorsal spinous processes may reduce or eliminate this protective contraction of the epaxial musculature [30, 32].

There appear to be only two biomechanical studies which have specifically investigated the effect of decompressive surgical techniques on the lumbosacral spine. In the study by Smith *et al*, a four-point bending jig was used to evaluate the stiffness of the lumbosacral joint before and after various combinations of decompressive surgeries [29]. Dorsal laminectomy was not found to have any significant effect on the stability of the lumbosacral joint in either dorsiflexion or ventroflexion. A dorsal laminectomy combined with a discectomy had no significant effect on stiffness in dorsiflexion, but did decrease mean stiffness in ventroflexion by 33%. It was found that the combination of dorsal laminectomy, discectomy and bilateral facetectomy resulted in the greatest decrease in stiffness (48% in dorsiflexion and 56.4% in ventroflexion) [29].

The study by Meiji *et al* was a biomechanical investigation of the effect of dorsal laminectomy and discectomy and pedicle screw-rod fixation on the flexion-extension forces in the lumbosacral joint of normal dogs [18]. This study used cyclical testing in a four-point bending device. The findings were that dorsal laminectomy and discectomy did not significantly decrease spinal stability while pedicle screw-rod fixation did effectively stabilize the lumbosacral spine [18]. The study did however conclude that stiffness in the neutral zone (Neutral zone is defined as that

range were the specimen moves without external force being applied i.e. the laxity of the specimen) did decrease after dorsal laminectomy and discectomy.

The major difference in the results of the two studies is as follows : Smith *et al* [29] showed a significant decrease in stiffness following dorsal laminectomy and discectomy in ventroflexion (33%), while Meij *et al* [18] showed no significant change/decrease in stiffness after dorsal laminectomy and discectomy. It is difficult to explain these differences. The different testing methodologies may play a role in the differing results. The study by Meij *et al* used cyclical testing of the specimens as opposed to the study by Smith *et al* which stressed the specimens to a point and then returned to zero.

The study reported here will investigate the effect of decompressive surgery on the stability of the lumbosacral joint. Although a previous study [29] has investigated the biomechanics of the lumbosacral joint after surgical modification, there are some fundamental differences between that study and the present investigation. In that study the effect of different sized laminectomies on the stiffness of the lumbosacral joint was not investigated. This aspect was included in the current investigation, by comparing a mini-dorsal laminectomy (MDL) and standard dorsal laminectomy (SDL) (sizes defined in the Material and Methods section). With the advent of minimally invasive surgery and the endoscopically-assisted foraminotomy technique [36], mini-laminectomy may become more commonly performed. The discectomy performed in the Smith *et al* study [29] was performed with power drill, but this is not common practice in a clinical situation. The discectomy in this study was performed by means of a scalpel incision in the dorsal annulus and removal of *Nucleus pulposus* with a 2mm curette. This was considered more controlled and reproducible than the power burring. Bilateral facetectomy, as performed in the Smith *et al* study [29] is rarely, if ever performed in a clinical setting, therefore its clinical relevance is in question.

The study by Meiji *et al* [18] applied cyclical loading to the specimens, while the current investigation used a single stress applied once before and after surgical modification. The Meiji *et al* study did not investigate the effect of dorsal laminectomy size on the outcome.

There are therefore some unanswered questions that remain with regard to DLS and lumbosacral instability. The purpose of this study is to address some of these issues as outlined in the next chapter.

Chapter 2: Objectives and Hypothesis

It is unclear from previous biomechanical investigations of the lumbosacral joint (Smith *et al* and Meij *et al*) whether the extent of a dorsal laminectomy has an effect of lumbosacral stability post-operatively. There is a relatively high incidence of recurrence (18-33%) of clinical symptoms following lumbosacral decompression. The presence of instability, both pre-operatively and post-operatively is thought to play a major role in the pathophysiology of this condition. It is therefore important to determine whether or not surgical decompression potentially exacerbates the underlying instability, and increases the risk of recurrence of clinical symptoms. The primary objectives of this study were to determine whether or not lumbosacral decompressive surgery (dorsal laminectomy and discectomy) for degenerative lumbosacral stenosis results in significant instability of the lumbosacral joint, and secondly whether the type and/or extent of the dorsal laminectomy had an influence on the degree of instability in the lumbosacral joint.

The hypothesis was that dorsal laminectomy and discectomy would result in decreased lumbosacral stability compared to the intact spine, and that a mini-dorsal laminectomy combined with a partial discectomy would result in less lumbosacral instability than a standard dorsal laminectomy and discectomy.

The null hypothesis was that dorsal laminectomy and partial discectomy has no effect on spinal stability and secondly that the size of the laminectomy has no effect on lumbosacral stability.

The paraspinal muscles and tendons are thought to play an important role in lumbosacral joint stability in the live animal. Their role and that of the neural control system are obviously negated in the cadaver and are thus impossible to quantify in this study. Spinal attachments of the epaxial muscles are reflected during surgical exposure of the lumbosacral joint. The significance of this and whether it will have an influence on the functioning of the active system is unknown and cannot be investigated in a cadaver study.

The results of this investigation would then allow more objective decision making with regard to type of decompressive surgery to be performed, and whether or not adjunctive lumbosacral stabilisation should be performed. This information may then allow improved treatment and outcome of this condition by helping to reduce the relatively high recurrence rate experienced with current surgical treatments.

Chapter 3: Materials and Methods

3.1 Outline of study design

This was an *in vitro* biomechanical cadaver study investigating the stiffness of the lumbosacral motion unit before and after surgical modification. The lumbosacral motion unit is defined as the articulations between L7 and S1. This includes the vertebral body of L7 and the body of S1 as well as all the soft tissues (intervertebral disc and ligaments). Each lumbosacral motion unit was rigidly mounted in a four-point bending jig and subjected to dorsiflexion and ventroflexion in the sagittal plane.

Each specimen was tested twice, non-destructively, first in dorsiflexion followed immediately by ventroflexion. Following an interval of 20 minutes during which time the specimen was surgically modified, the testing was repeated in both dorsiflexion and ventroflexion. Each specimen was then tested to failure in ventroflexion.

The collected data was then used to obtain load-deformation curves for each specimen (pre- and post-modification) in dorsiflexion and ventroflexion. The stiffness of each specimen was then determined from the load-deformation curves at specific points of angular displacement. Change in stiffness was then calculated from pre- and post modification curves for each specimen.

3.2 Study details

3.2.1 Specimen collection

Dog cadavers were obtained from the Johannesburg Animal Anti-Cruelty League. These animals were all euthanised with an intravenous barbiturate overdose for reasons unrelated to the study. Only animals weighing between 25 and 40kg were considered. The following information was noted at the time of cadaver collection: weight, sex, and whether the animal was sterilised or not. None of the animals had known ages. From radiographs it was determined that all were skeletally mature, and from appearances and history, none of the animals were regarded as being geriatric.

All cadavers were radiographed at Bryanston Veterinary Hospital. Both lateral and ventrodorsal views of the lumbosacral joint were obtained. Radiographs included the lumbar spine from L4 distally to the coccygeal vertebrae in both the lateral and ventrodorsal views. The radiographs were carefully screened for any pathology or anatomical abnormalities (transitional vertebrae, lumbosacral spondylosis etc) as well as open physes. If any of these conditions were identified or even suspected the cadaver was immediately rejected from the study.

All cadavers were identified with a number once they had been radiographed. This reference number remained the same for each specimen throughout the study.

3.2.2 Specimen preparation

Stage 1:

The lumbar spine from L5/6 to the sacro-coccygeal joint was the harvested from the cadavers immediately following euthanasia and radiography. The skin was incised on the dorsal midline from thoracolumbar junction to the proximal coccygeal vertebrae and reflected laterally. Care was taken not to damage the supraspinous ligaments. The abdominal musculature was transected from the transverse processes. A saw was then used to transect the vertebral column at L5/6 and at the saccro-coccygeal junction. The ilial wings were disarticulated from the wings of the sacrum using a small osteotome and mallet. All musculature covering the specimen was left intact at this stage. The specimens were then wrapped in paper towel and soaked in sterile lactated Ringer's solution (Sabax, Adcock Ingram, Johannesburg, South Africa). Care was taken to ensure that the entire specimen was wrapped in paper towel and that the paper towel was completely soaked in sterile Ringer's solution. They were then placed in individual numbered sealed plastic bags (Ziploc® bags), which were then sealed within a second bag. The specimens were then frozen at -20˚C in a chest freezer. The process from euthanasia to refrigeration took approximately 45 minutes on average. All cadaver remains were incinerated by an animal cremation company (Envirocin, 191 Homestead Ave, Randburg).

Prior to potting the lumbosacral motion units in aluminium tubing (for mounting in the four-point bending jig), the specimens were partially thawed at 4˚C in a sterile lactated Ringer's bath (still in sealed plastic bags). All the musculature except for a very thin layer of *Mm. multifidi* and *Mm. interspinales* over the articular process (facet) joint capsule, *ligamentum flavum* and interspinous ligaments was dissected away. Care was taken to ensure that all ligamentous structures associated with the stability of the lumbosacral joint remained intact. These structures included *Lig. Flavum,* interspinous ligaments, supraspinous ligaments, articular process (facet) joint capsule, *Anulus fibrosus,* and the dorsal and ventral longitudinal ligaments. (Figure 3-1)**.** Once the dissection was completed, accurate measurements of the length of the lamina of L7 and

the sacrum were taken with a vernier calliper (Omni-tech®, 150mm, accuracy to 0.02mm). All measurements were made to the nearest millimetre. The transverse processes of L6 and L7 and the dorsal spinous process of L6 were shortened with bone rongeurs so that the specimen would fit into the square (60mm x 60mm) aluminium tubing. (Figure 3-2)

Each specimen was then prepared to be potted with a polyester resin in the aluminium tubing. Each section of aluminium tubing was 10cm long. A custom made aiming device was used to drill a 5mm hole transversely in the vertebral body of L6 and the sacrum. The aiming device was fixed in a table-mounted vice and the specimen was held in the aiming device while the holes were drilled at low speed. The hole in the sacrum was positioned just caudal to the wings of the sacrum. A drill press was used to drill holes accurately in the walls of the aluminium tubing.

L6 was placed in one section of tubing while the sacrum was placed within another section of aluminium tubing. Transfixtion bolts (5mm diameter) were then placed through the holes in the aluminium tubing and through the holes drilled in the specimen. The positioning of the holes was such that the specimens were accurately fixed with the median plane of the vertebrae parallel to the walls of the tubing. This ensured that the specimens were loaded symmetrically during testing.

A polyester resin (NCS 964 PA, NCS Resins (Pty) Ltd, Edenvale, South Africa) was used to pot the specimen within the aluminium tubing. The resin (NCS 964PA) was mixed with the catalyst (Curox M-200, NCS Resins (Pty) Ltd, Edenvale) in a 1.5% ratio (1.5 parts catalyst to 100 parts resin). Carbonate powder (Kulubrite 5, NCS Resins (Pty) Ltd, Edenvale) was added in equal volume to the resin. This was to provide a filler which reduced the amount of resin used, increased the strength and decreased the temperature of the exothermic reaction. The resin was inserted into the aluminium tubing using a 60ml catheter-tipped syringe (Terumo, Terumo

Corporation, Tokyo, Japan). Masking tape was then used to seal the ends of the aluminium tubing.

When setting, the aluminium tubing was warm to the touch and setting took 20-30 minutes to complete. While the resin was setting the specimens were kept moist and cool by covering them with lactated Ringer's soaked paper towels. When the specimens were mounted within the aluminium tubing care was taken to ensure that there was sufficient access to the specimen to allow the surgical modifications to be performed accurately. This gap also allowed unhindered flexion and extension of the lumbosacral joint during testing.

The specimens were then re-wrapped in Ringer's soaked paper towels and double bagged in sealed plastic bags. All specimens and bags were clearly marked with a permanent marker. The number corresponded to the original identity number of the cadaver and therefore all relevant data pertaining to the specimen. Specimens were then refrozen in the chest freezer. Freezing at -20˚C has been demonstrated to have negligible effect on the biomechanical properties of the tissues of the vertebral motion unit (bone, ligament and disc) [17]. The above protocol (freezing at -20˚C and thawing prior to testing) is a well accepted method when testing the biomechanics of vertebral motion units [17]. All specimens were exposed to two cycles of freezing and thawing during the preparation process.

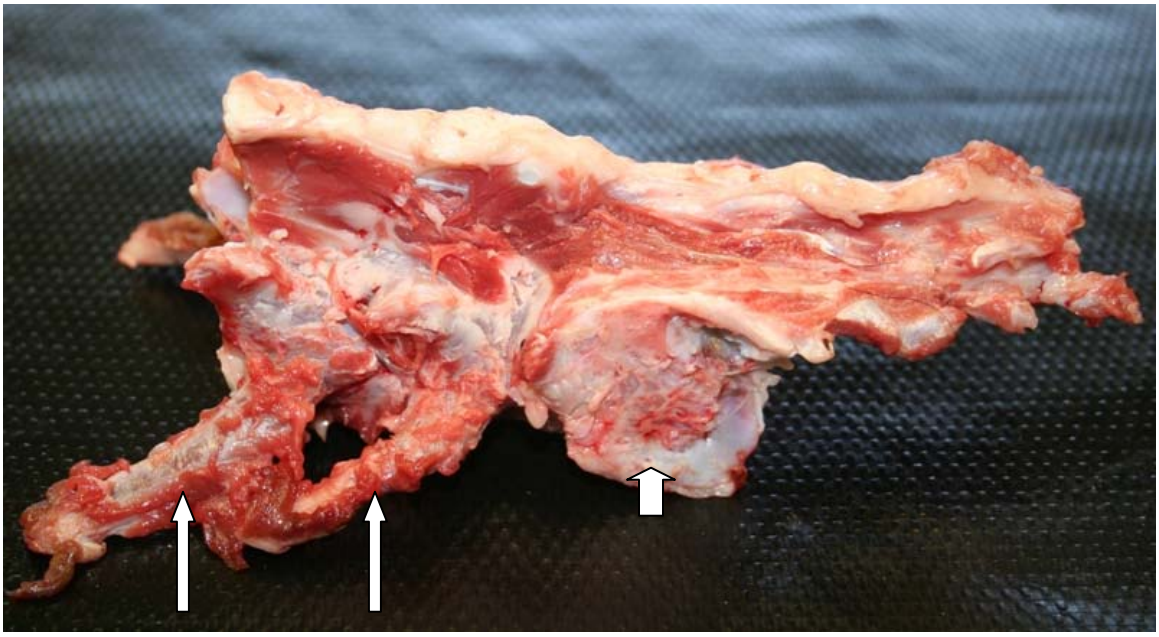

Figure 3-1 : Specimen during preparation (1). Most of the musculature dissected away with all ligamentous structures left intact. (long arrows – transverse processes of L6 & L7, short arrow – wing of the sacrum, cranial to the left)

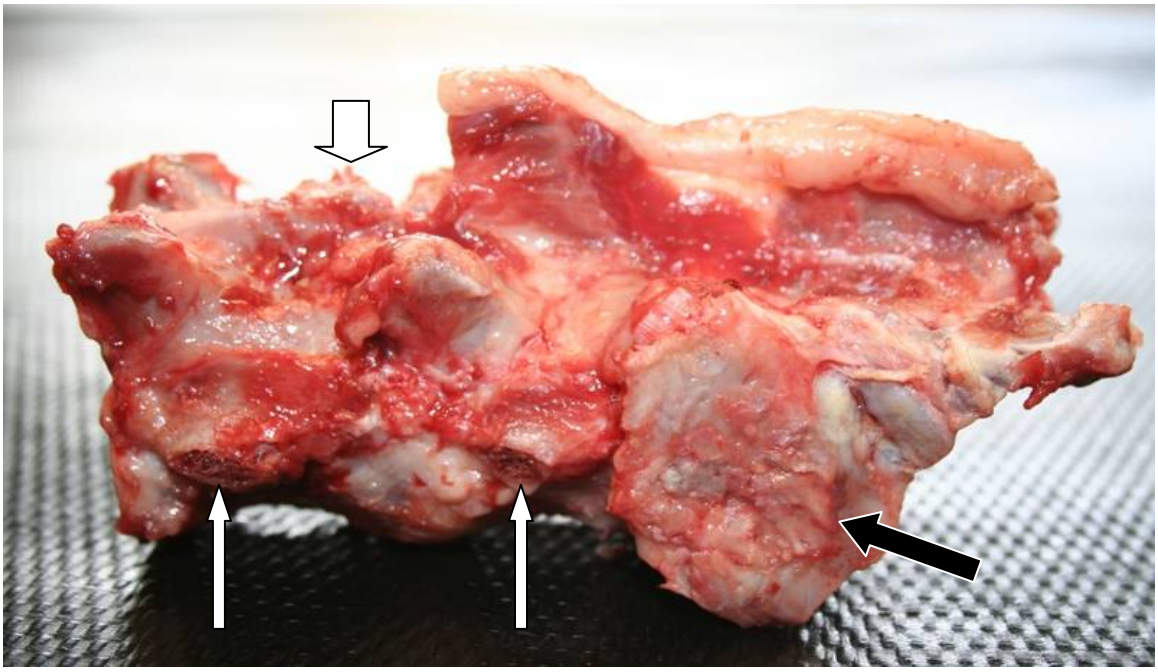

Figure 3-2: Specimen during preparation (2). Specimen ready for potting. Note the transverse processes of L6 and L7 (long arrows) have been removed and the spinous process of L6 has been removed (short arrow) to allow fixation within the aluminium tubing. (black arrow = sacrum, cranial to the left)

Stage 2:

Twenty one specimens were randomly allocated to three groups.

The three groups were as follows:

Group 1 – No surgical modification

Group 2 – Mini-dorsal laminectomy (MDL) and discectomy

Group 3 – Standard dorsal laminectomy (SDL) and discectomy

The allocation of the specimens to the groups resulted in the following group sizes: Group 1 = 7, Group 2 = 7 and Group 3 =7.

Due to the nature of the study, each specimen served as its own control. Because each specimen was tested once prior to modification and once after modification it allowed assessment of the change within that specimen. Group 1 specimens had no surgical modification between tests, and therefore allowed assessment for any change due to the testing process and/or time interval between tests 1 and 2.

Stage 3

All specimens were tested once in dorsiflexion and once in ventroflexion. Specimens were removed from the jig and inverted between the tests in dorsiflexion and ventroflexion. The angle achieved during testing was approximately 21˚ in dorsiflexion and 21° in ventroflexion). The tests in dorsiflexion and ventroflexion were then repeated after an interval of 20 minutes. This was to ensure that sufficient time was allowed for the surgical modification of the specimens in groups 2 and 3. All specimens, regardless of group therefore had a 20 minute interval between the first tests in dorsiflexion and ventroflexion and the second tests in dorsiflexion and ventroflexion. This minimized any changes in the results due to changes in the viscoelastic properties of the specimens over time i.e. all specimens would be tested over the same time

period. All specimens were tested in the same order, i.e. first in dorsiflexion and then in ventroflexion. The test to failure in ventroflexion was recorded on a digital video camera.

To track the data of each specimen a letter and number were added to the original specimen number. The software required a letter prior to the specimen number, this was the letter “t”. The letter “a” after the specimen number indicated a test in dorsiflexion, “b” indicted a test in ventroflexion and “c” indicated a test to failure in ventroflexion. The number “1” after the “a” or “b” indicated the second or post modification test.

e.g. : t7a = Specimen 7, initial test / pre-modification test in dorsiflexion

t7a1 = Specimen 7, second test / post modification test in dorsiflexion

t7b = Specimen 7, initial test / pre-modification test in ventroflexion

t7b1 = Specimen 7, second test / post modification test in ventroflexion

t7c = Specimen 7 tested to failure in ventroflexion

Mini-dorsal laminectomies were performed on all Group 2 specimens. In the mini-laminectomy the caudal lamina of L7 was removed from the caudal margin of the dorsal spinous process extending caudally to the caudal margin of the lamina (L7) and laterally to the medial aspect of the caudal articular processes (facets) of L7. The dorsal spinous process was not disturbed. The lamina of S1 was not disturbed during the MDL (Figure 3-3). These dimensions were chosen as they were found to provide sufficient space to perform a discectomy, fenestration and permit probing of the foramina. Standard dorsal laminectomies (SDL) were performed on Group 3 specimens. The SDL removed the caudal 75% of the lamina of L7 (including excision of the dorsal spinous process of L7) and the cranial 50% of the lamina of S1. The lateral limits of the

standard laminectomy were the same as for the mini-laminectomies (Figure 3-4). The dimensions for the SDL were taken from the literature where most dorsal laminectomies are described as being the majority of the lamina of L7 and the majority of the lamina of S1. The above dimensions were decided upon in order to standardize the extent of the laminectomies in all specimens. The lamina lengths of L7 and S1 were recorded in mm and then 75% of the lamina of L7 and 50% of the lamina of S1 were calculated for each specimen. This minimized any variability between specimens. Laminectomies were performed with a rotary power drill (Dremel Multipro 395, Racine, Wisconsin, USA) using a 1mm diameter burr. Both mini-laminectomies and standard laminectomies included excision of the *Lig. flavum* as well as the interspinous and supraspinous ligaments of the L7-S1 joint.

Partial discectomies were performed on all specimens in both Groups 2 and 3. The partial discectomy was performed by making a rectangular incision in the dorsal *Anulus fibrosus* with a number 11 scalpel blade. The cranial and caudal limits of the incisions for the discectomy were the caudal endplate of L7 and the cranial endplate of S1. The lateral borders of the discectomy were the vertebral sinus within the vertebral canal, which were clearly visible during dissection. After removing the rectangular piece of *Anulus fibrosus*, a 2mm curette was the used to remove the *Nucleus pulposus.* The disc was curetted until no further disc material could be retrieved. This was generally achieved with approximately five sweeps of the curette.

Once the testing of a specimen was completed the specimen was wrapped in paper towel soaked in lactated Ringer's and sealed in numbered plastic bags (Ziploc bags). The specimens were then replaced in a cool lactated Ringer's bath and then replaced in the chest freezer to be re frozen. All specimens were disposed of by incineration

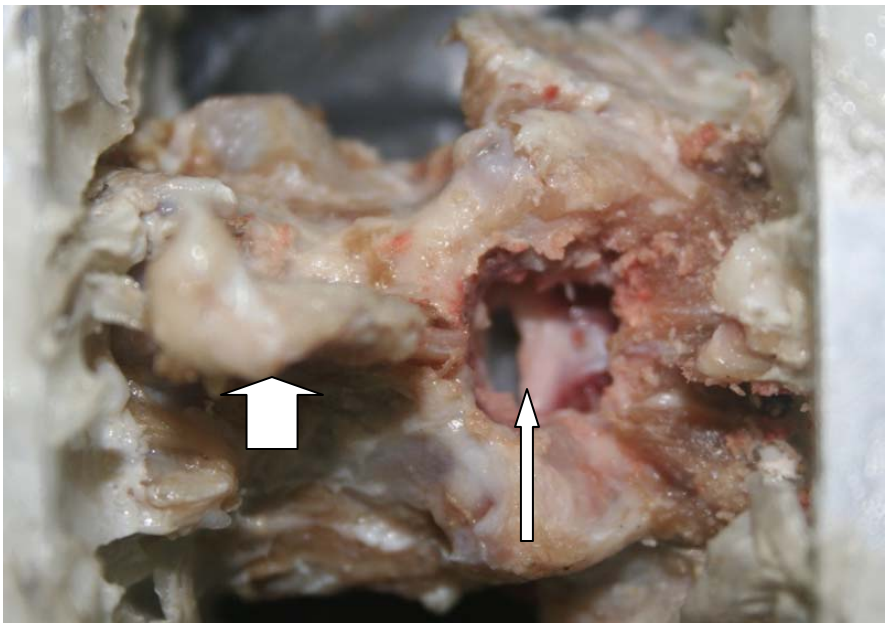

Figure 3-3: Mini-dorsal laminectomy. The dorsal annulectomy (long arrow) is clearly visible. Note the dorsal spinous process of L7 (short arrow) is undisturbed.(cranial is to the left)

.

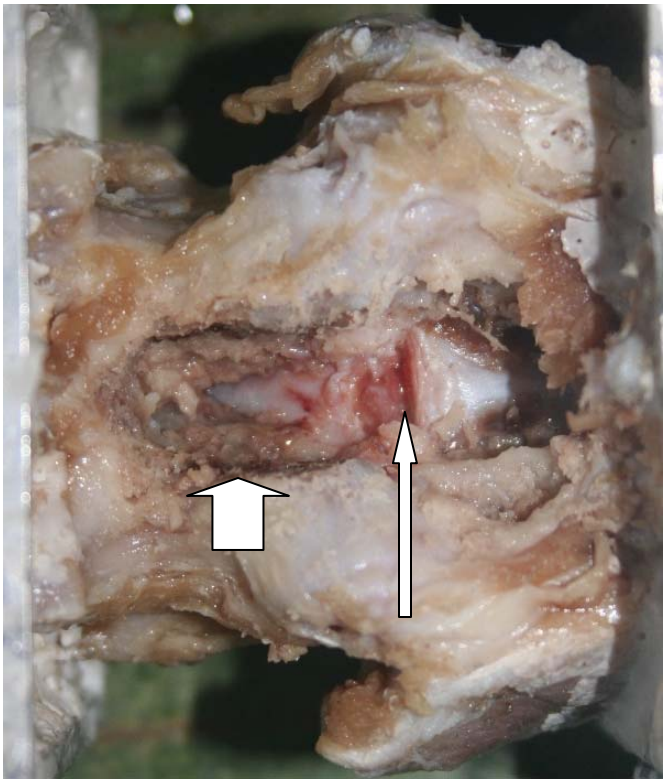

Figure 3-4: Partially completed standard dorsal laminectomy. Note the dorsal spinous process of L7 (short arrow) has been excised. The partially completed annulectomy (long arrow) is clearly visible. (cranial is to the left)

3.2.3 Testing methodology

The specimens were placed in a lactated Ringer's bath at 4˚C 12 hours prior to testing. The specimens were all kept in their sealed plastic bags until testing. As soon as each specimen was tested, it was replaced in a sealed plastic bag in the lactated Ringer's bath. All specimens were transported to the laboratory for testing in a cool box (Coleman®). All testing was done at the Sasol Laboratory of the Faculty of Mechanical and Aeronautical Engineering at the University of Pretoria.

A custom-made four-point bending jig (Figure 3-5) was designed and manufactured according to the dimensions of the aluminium tubing that the specimens were potted in.

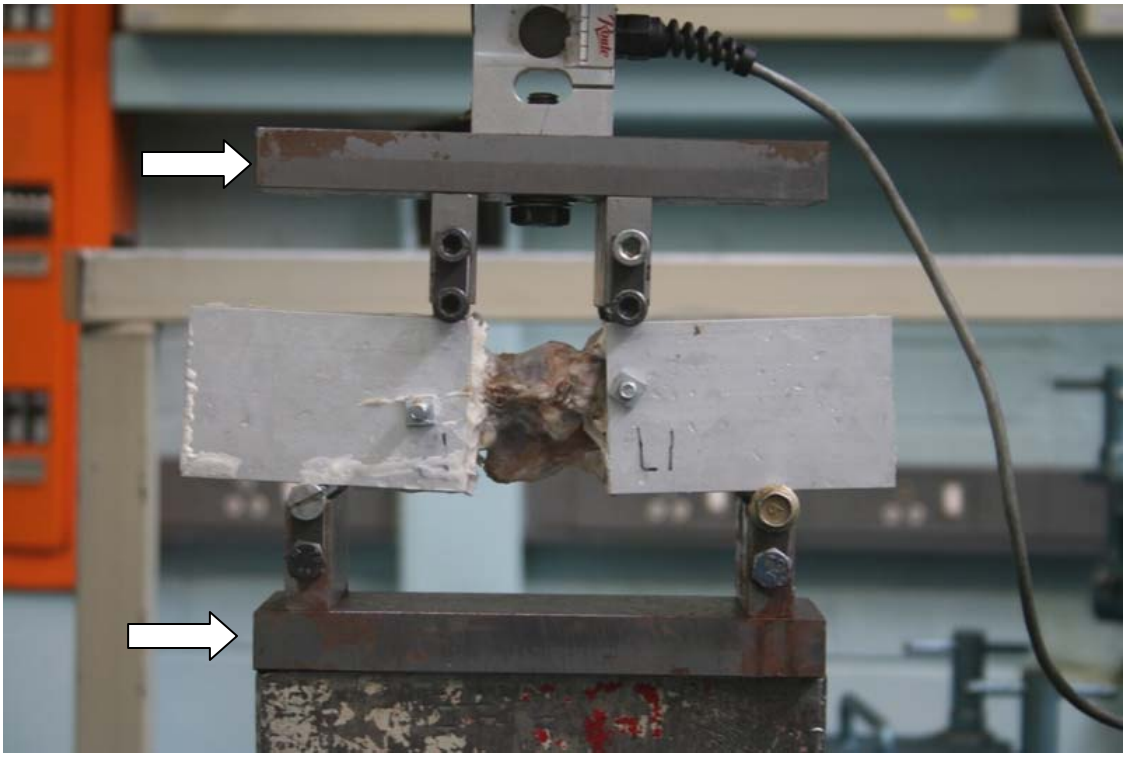

Figure 3-5: The 4 point bending jig (arrows). The potted specimen is visible in the bending jig

The four-point bending jig was mounted in a servo hydraulic actuator (100kN Schenck Hydropuls ®, Schenck, Darmstadt, Germany) which applied the force. The actuator was

equipped with a linear variable displacement transducer (LVDT) (HBM GmbH, Darmstadt, Germany) to measure the displacement and was calibrated at 1 volt = 5 mm. A 300 Kg load cell (Load Cell Services, Pretoria, South Africa) was attached to the jig to record the force produced by the actuator. The load cell was calibrated at 1 volt = 200 Kg. An amplifier (Measurements Group, Raleigh, North Carolina, USA) was used to amplify the voltage signal received from the LVDT, and to calibrate the apparatus.

The data acquisition system was a Pentium computer (ICP Intel Pentium I) with a C-DAS system. The C-DAS system consists of Analogue to Digital and Digital to Analogue cards, as well as filters to filter the measured data. Matlab version 4.1® (Mathworks, Natick, Massachusetts, USA) was used in the data acquisition. This is a high level technical computing programme that allows for algorithm development, data visualization, data analysis and mathematical calculations based in arrays. Qantim (written by Prof. A.D. Raath, Sasol Laboratory, University of Pretoria) was used to log the data. The equipment was calibrated daily to ensure accuracy.

Two drive signals were written in Matlab®. The loading protocol was displacement controlled. In other words, the actuator displaced the specimen to a set displacement over a set time and the force applied to achieve this was recorded. The cross head speed was 4.8 mm/minute. The load and vertical displacement of the cross head on the actuator were recorded at a frequency of 100Hz.

The first drive signal was used for the non-destructive testing and resulted in the actuator exerting an increasing force to achieve a constant displacement (5.5 mm). This was found to result in non destructive testing of the specimen as determined in a small trial. The second drive signal was used for testing to failure in ventroflexion. This resulted in displacement to 15 mm. Due to the dimensions of the specimen in the testing apparatus it was not possible to test beyond 15mm of displacement as the aluminium tubing impinged on the four-point bending jig (15mm of displacement did however result in testing to failure in ventroflexion).

Figure 3-6: Testing apparatus. The 4 point bending jig (green arrows) mounted in the actuator (white arrows) with the load cell (blue arrows). The potted specimen (red arrow) is visible in the bending jig. The image on the right is a close up of the testing apparatus.

3.2.4 Data collection and processing

Sampling frequency during testing was 100Hz. This resulted in approximately 7000 data points per test run (non-destructive testing) and more than 20 000 data points when testing to failure. Data was saved in the Matlab® format. It was then converted from the Matlab® format and saved as a text file which was then opened using Microsoft Excel (Microsoft Corporation, Get City, USA). It was apparent from the raw data that there was a significant amount of "noise" in the results. "Noise" is caused by the inherent vibrations of the actuator and may have included electronic spikes on the controller; the C-DAS data acquisition system may also have created some "noise". The "noise" was filtered with a Butterworth filter which filters all data from 100Hz down to 10Hz. This filter is a standard function of Matlab®. Each test's data was saved as a separate spreadsheet in Microsoft Excel. The raw data (volts) was then converted to kilograms (force) and millimetres (displacement).

The load cell and servo hydraulic actuator were calibrated as follows:

1 volt = 200 Kg (Force)

1 volt = 5 mm (Displacement)

The following formulae were used to convert force and displacement into kilograms and millimetres:

Force (Kg) = Volts x 200

Displacement (mm) = Volts x 5

Force was then converted to Newtons (N) by the following formula:

Force (N) = Kg x 9.81

Displacement in millimetres was converted into angular displacement by the following trigonometry formula :

Angular displacement (°) = 2 x [ATAN(Displacement in mm/30) x 180/Pi()].

ATAN = returns the arctangent (inverse tangent) of a number in radians, in the range -Pi/2 to Pi/2.

30mm = the distance between the centre of the jig to the first hinge point.

Pi() = converts the angle from radians to degrees

All formulae were applied to the data using Microsoft Excel .

The load deformation curves were then obtained using Microsoft Excel plotting force (Newtons) against displacement (angle in degrees). The slope or gradient of the curve was equivalent to the stiffness of the specimen. Stiffness is defined as the mechanical resistance to deformation of the specimen.

The stiffness was calculated for specific regions of the curve by the following formula:

Stiffness = (X1 – X2)/(Y1-Y2)

where X = force (N) and Y = angle of displacement (˚).

Example:

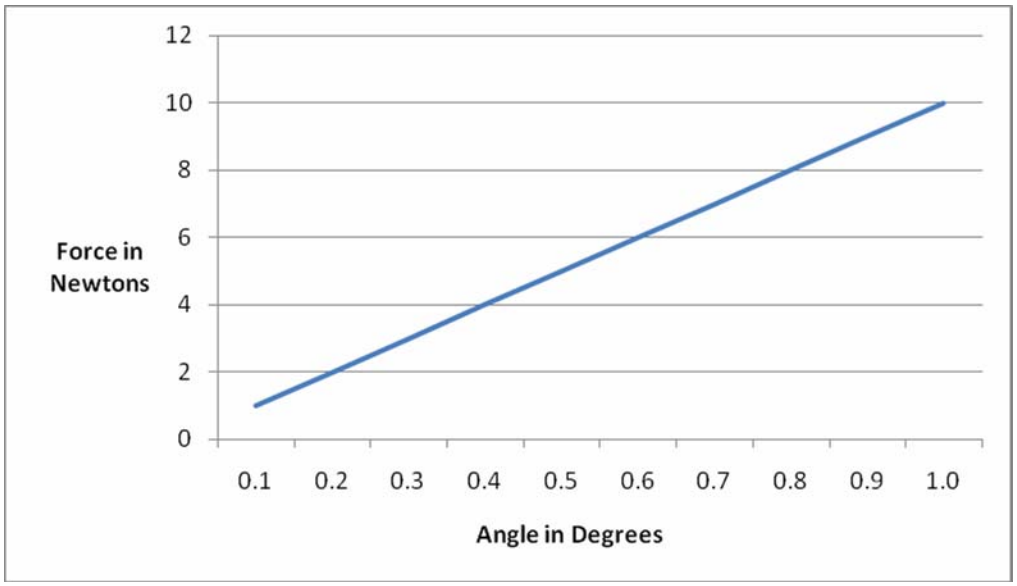

Figure 3-7: Stiffness calculation

To calculate the stiffness between 0.2° and 0.4° of angular displacement :

Stiffness = (4-2) / (0.4-0.2)

= 10 N/˚ (Newtons / degree)

Stiffness is therefore expressed as Newtons / Degree.

Stiffness was calculated at three specific ranges of angular deformation on the load deformation curves. These ranges were:

i) 6° - 8°
ii) 12° - 16°
iii) 18° - 20°

There were two reasons why the stiffness was compared for specific regions of the graph: Firstly, the force required to achieve equal angular displacement varied between specimens, and it was therefore decided to compare areas of equal angular displacement. Secondly, and more importantly, the formula (stiffness = (X1-X2)/(Y1-Y2)) assumes the curve between points X1 and X2 is linear. Due to the fact this was not a linear curve, short sections of the curve had to be compared i.e. to ensure that X1 and X2 were close enough together that the curve between them could be considered linear. The three ranges of angular deformation chosen represent areas of the load deformation curve at low, medium and high angular displacement,

Once stiffness had been calculated for specific regions of each curve, it was possible to calculate the percentage change in stiffness for dorsiflexion and ventroflexion for each specimen. This change in stiffness was between pre- and post-modification load deformation curves, or in the case of Group 1, it was the change in stiffness between the first and second tests without modification.

% change in stiffness = [(Pre-mod stiffness – Post-mod stiffness) / Pre-mod stiffness] x100

The mean percentage change in stiffness for each group was then calculated.

Failure was defined as a sudden, unexpected change in the slope of the load deformation curve.

3.3 Statistical analysis

All data was entered onto a spreadsheet (Microsoft Excel, Microsoft Corporation, Get City, USA). Statistical analysis was performed using Stata 10.0 (StatCorp, College Station, Texas, USA). Statistical significance was set at $p< 0.05$. A mixed effects multiple regression model was used to estimate the effects of group (1, 2 or 3), direction (dorsiflexion and ventroflexion), body weight and angular displacement (at which stiffness was calculated) on the percentage reduction in stiffness. Fixed effects were group, direction, body weight and angle, and subject was included as a random effect to account for the fact that multiple outcomes were clustered within subject.

The Kruskal-Wallis One Way Analysis of Variance (ANOVA) and the Tukey-Kramer Multiple-Comparison Test were then used to compare the three groups in both dorsiflexion and ventroflexion.

Force and angle at failure were compared using the One Way Analysis of Variance (ANOVA) test and the Tukey-Kramer Multiple-Comparison Test.

Means and standard deviations were calculated for the percentage change in stiffness for each specimen in both dorsiflexon and ventroflexion, and at all three angles of stiffness calculation. This was done in order to evaluate for repeatability.

3.4 Ethical considerations

Ethical aspects of this investigation was scrutinized and approved by the Animal Use and Care Committee of the Faculty of Veterinary Science, University of Pretoria.

Chapter 4: Results

4.1 Specimens

Twenty one specimens were randomly allocated to groups 1, 2 or 3.

The three groups were as follows:

Group 1 – No surgical modification

Group 2 – Mini-dorsal laminectomy (MDL) and discectomy

Group 3 – Standard dorsal laminectomy (SDL) and discectomy

The allocation of the specimens to the groups resulted in the following group sizes: Group 1 = 5, Group 2 = 7 and Group 3 = 6. Three specimens were lost to the investigation due to power failures resulting in excessive time intervals between tests; 2 from Group 1 and 1 from Group 3.

Data from a total of 18 lumbosacral motion units was collected. Specimen 5 (Group 1) was tested to failure in ventroflexion but the data was lost due to a computer malfunction during data collection. The test could not be re-run as it was a test to failure.

The breeds represented included: 3 German Shepherd Dogs (GSD), 3 German Shepherd Dog crosses, 2 crossbreeds (X Breed), 2 Boerboels (mastiff-type breed), 2 Rottweilers, 1 Boxer cross, 1 Ridgeback, 1 Doberman, 1 Chow Chow cross, 1 Labrador cross and 1 Husky cross. All dogs were adjudged to be young adults. None of the specimens displayed any radiographic signs of lumbosacral disease or pathology. Specimens were from 9 entire males, 7 entire females and 2

neutered male dogs. Mean body weight was 30.60kg (range 25.4 – 40). Mean body weight in Group 1 was 31.68 Kg, in Group 2, 30.88 Kg, and in Group 3, 29.38 Kg. Group allocation is shown in Table 4-1

Group	Specimen No.	Sex	Weight (kg)	Breed
Group 1 (n=5)	5	F	26.4	GSD X
	10	F	33.9	Boxer X
	12	F	32.1	Ridgeback
	13	M	37	Boerboel
	16	M	29	Doberman
Group 2 (n=7)	6	M	28.4	GSD
	7	M(N)	40	GSD
	15	F	31.9	X Breed
	17	M	25.4	Husky X
	19	M	31	GSD
	20	M	33.6	Rottweiler
	26	M	26	X Breed
Group 3 (n=6)	9	M	29.5	GSD X Lab
	11	M(N)	32	Rottweiler
	18	F	34	Boerboel
	21	M	26.3	GSD X
	22	F	28	Chow X
	27	F	26.6	Lab X

Table 4-1: Specimen allocation to Groups 1, 2 and 3.

4.2 Biomechanical study

All the load deformation curves for each specimen are shown in the Appendix (Figures A-1 to A-89).

A neutral zone was observed on some load displacement curves. This is defined as a zone where change in angular displacement occurs at zero loading. The neutral zone (NZ) was measured in degrees i.e. it was that angular displacement that occurred without a force being applied to the specimen. This was noticeable during testing as sagging of the specimen before the actuator applied a load. The neutral zone indicates a range of angular deformation were the stiffness is zero. A large neutral zone represents a large degree of angular deflection achieved at zero force i.e. the specimens stiffness is zero. The neutral zone was an observation in the study and was not a parameter that the study had intended to investigate or measure. It is included as an observation.

4.2.1 Load-deformation curves (non destructive testing)

Group 1:

All load deformation curves displayed similar tendencies in that the gradient increased as the force applied and resultant angular displacement increased. The gradient of the curve is indicative of the stiffness of the specimen i.e. with increased load and angular displacement the stiffness increased. The load deformation curves for Group 1 are presented in Figures A-1 to A-20. The load deformation curve for specimen 5 when tested in dorsiflexion the second time (Figure A-2) showed a deviation in the curve at approximately 20°. This was due the specimen slipping within the 4 point bending jig and was not due to failure of the specimen. Data from this specimen was included as no evidence of failure was detected.

There was large variation between individual specimens regarding the force required to achieve approximately 21° of angular displacement. See Table 4-2 for the forces required to achieve 21°

of angular deflection as well as the mean and standard deviation for dorsiflexion (pre- and post-modification) and ventroflexion (pre- and post modification). All specimens in Group 1 required greater force for ventroflexion compared to dorsiflexion.

Specimen No.	Force (N) at 21° (Dorsiflexion Pre-Mod)	Force (N) at 21° (Dorsiflexion Post-Mod)	Force (N) at 21° (Ventroflexion Pre-Mod)	Force (N) at 21° (Ventroflexion Post-Mod)
5	108.12	98.97	243.15	196.80
10	53.91	45.15	62.24	48.25
12	27.89	20.81	149.18	146.24
13	51.26	56.46	185.99	139.94
16	79.62	57.19	205.25	162.33
Max	108.12	98.97	243.15	196.80
Min	27.89	20.81	62.24	48.25
Mean	64.16	55.72	169.16	138.71
S.D.	30.65	28.30	68.72	55.17

Table 4-2: Force required to achieve 21° angular deflection of Group 1 specimens. All values given in Newtons (N). S.D. = Standard deviation, Pre-Mod = Pre-modification, Post-mod = Post-modification.

The observations of the neutral zones are summarized in Tables 4-3 and 4-4. From the tables it is noted that there was no neutral zone in ventroflexion in group 1.

Specimen No.	Pre-mod NZ	Post-mod NZ	Change in NZ
5	0°	0°	0°
10	2°	2°	0°
12	5°	8°	3°
13	5°	5°	0°
16	3°	3°	0°
Mean	3°	3.6°	0.6°
SD*	2.12	3.05	1.34

Table 4-3: Neutral zone in dorsiflexion (Group 1). SD =Standard deviation, NZ = neutral zone, Pre-mod = Pre-modification, Post-mod = Post-modification**.**

Specimen No.	Pre-mod NZ	Post-mod NZ	Change in NZ
5	0°	0°	0°
10	0°	0°	0°
12	0°	0°	0°
13	0°	0°	0°
16	0°	0°	0°
Mean	0°	0°	0°
SD*	0	0	0

Table 4-4: Neutral zone in ventroflexion (Group 1). SD = Standard deviation, NZ = neutral zone, Pre-mod = Pre-modification, Post-mod = Post-modification.

Group 2:

Similarly to Group 1, the load-deformation curves in Group 2 displayed an increasing gradient as force and angular displacement increased. The load deformation curves for Group 2 are shown in Figures A-21 to A-48. There was a large variation in forces required to achieve the set angular displacement between specimens. Within Group 2, two specimens (6 and 15) required greater loading in dorsiflexion compared to ventroflexion, two specimens (19 and 26) required greater loading in ventroflexion and three specimens (7, 17 and 20) required approximately equal loading in dorsiflexion and ventroflexion to achieve the set displacement. See Table 4-5 for the forces required to achieve 21° of angular deflection as well as the mean and standard deviation for dorsiflexion (pre- and post-modification) and ventroflexion (pre- and post modification).

Specimen No.	Force (N)at 21° (Dorsiflexion Pre-Mod)	Force (N) at 21° (Dorsiflexion Post-Mod)	Force(N) at 21° (Ventroflexion Pre-Mod)	Force (N) at 21° (Ventroflexion Post-Mod)
6	208.97	133.16	55.38	13.68
7	216.15	136.63	231.73	66.03
15	281.21	12.15	45.70	24.18
17	134.16	39.67	142.62	26.92
19	125.10	50.93	191.73	60.82
20	197.49	67.31	159.90	91.25
26	85.81	43.93	73.83	14.31
Max	281.21	136.63	231.73	91.25
Min	85.81	12.15	45.70	13.68
Mean	178.41	69.11	128.70	42.46
S.D.	66.66	47.85	71.92	30.19

Table 4-5: Force required to achieve 21° angular deflection of Group 2 specimens. All values given in Newtons (N). S.D. = Standard deviation, Pre-Mod = Pre-modification, Post-mod = Post-modification.

The observations of the neutral zones in Group 2 are summarised in Tables 4-6 and 4-7.

Specimen No.	Pre-mod NZ	Post-mod NZ	Change in NZ
6	0°	0°	0°
7	0°	3°	3°
15	0°	8°	8°
17	3°	8°	5°
19	0°	4°	4°
20	0°	0°	0°
26	2°	10°	8°
Mean	0.71°	4.71°	4.00°
SD*	1.25	4.03	3.32

Table 4-6: Neutral zone in dorsiflexion (Group 2). SD = Standard deviation, NZ = neutral zone, Pre-mod = Pre-modification, Post-mod = Post-modification.

Specimen No.	Pre-mod NZ	Post-mod NZ	Change in NZ
6	8°	12°	4°
7	1°	6.5°	5.5°
15	3°	9°	6°
17	0°	6°	6°
19	0°	1.5°	1.5°
20	0°	2°	2°
26	5°	15°	10°
Mean	2.43°	7.43°	5.00°
SD*	3.10	4.97	2.87

Table 4-7: Neutral zone in ventroflexion (Group 2). SD = Standard deviation, NZ = neutral zone, Pre-mod = Pre-modification, Post-mod = Post-modification.

A number of the load deformation curves displayed oscillations of the curve. This was particularly evident when the magnitude of the force required to achieve angular displacement was very low. It was also more evident in the neutral zones. The load deformation curves displaying this tendency are depicted in Figures A-24, A-32, A-36, A-40 and A-48.

Group 3:

As for Groups 1 and 2, the load-deformation curves followed a similar pattern with increasing gradient at increased loads and angles of displacement. The load deformation curves from group 3 are found in Figures A-49 to A-72. Similarly to Groups 1 and 2, there was a large variation in size of force / load required to achieve the set displacement. See Table 4-8 for the forces required to achieve 21° of angular deflection as well as the mean and standard deviation for dorsiflexion (pre- and post-modification) and ventroflexion (pre- and post modification).

Specimen No.	Force (N) at 21° (Dorsiflexion Pre-Mod)	Force (N) at 21° (Dorsiflexion Post-Mod)	Force (N) at 21° (Ventroflexion Pre-Mod)	Force (N) at 21° (Ventroflexion Post-Mod)
9	18.20	0.11	155.23	16.93
11	68.68	35.56	95.04	33.89
18	33.33	7.56	347.27	115.39
21	57.82	5.90	160.81	25.89
22	46.89	18.30	56.66	7.29
27	80.09	25.94	57.49	9.50
Max	80.09	35.56	160.81	115.39
Min	18.20	0.11	56.66	7.29
Mean	50.84	15.56	145.42	34.82
S.D.	22.85	13.50	108.83	40.72

Table 4-8: Force required to achieve 21° angular deflection of Group 3 specimens. All values given in Newtons (N). S.D. = Standard deviation, Pre-Mod = Pre-modification, Post-mod = Post-modification.

The observations of the neutral zones in group 3 are summarised in Tables 4-9 and 4-10.

Specimen No.	Pre-mod NZ	Post-mod NZ	Change in NZ
9	10°	21°	11°
11	0°	2°	2°
18	9°	17°	8°
21	6°	18°	12°
22	2°	10°	8°
27	1°	8°	7°
Mean	4.67°	12.67°	8.00°
SD	4.27	7.20	3.52

Table 4-9: Neutral zone in dorsiflexion (Group 3). SD = Standard deviation, NZ = neutral zone, Pre-mod = Pre-modification, Post-mod = Post-modification.

Specimen No.	Pre-mod NZ	Post-mod NZ	Change in NZ
9	0°	10°	10°
11	1°	6°	5°
18	0°	0°	0°
21	0°	8°	8°
22	4°	17°	13°
27	2°	11°	9°
Mean	1.17°	8.67°	7.50°
SD	1.60	5.65	4.51

Table 4-10: Neutral zone in ventroflexion (Group 3). SD = Standard deviation, NZ = neutral zone, Pre-mod = Pre-modification, Post-mod = Post-modification.

There were a number of the load deformation curves in this group that displayed oscillation of the curve. The most severe are depicted in Figures A-50, A-58, A-68 and A-72.

The changes in the neutral zones between pre- and post-modification testing of specimens are summarized in Figure 4-1. In Group 1 there is a mean increase of 0.6° in the neutral zone in dorsiflexion while the neutral zone in ventroflexion did not change. In Group 2 the neutral zone increased by a mean of 4° and 5° in dorsiflexion and ventroflexion respectively Group 3 showed a mean increase in the neutral zone of 8° and 7.5° in dorsiflexion and ventroflexion.

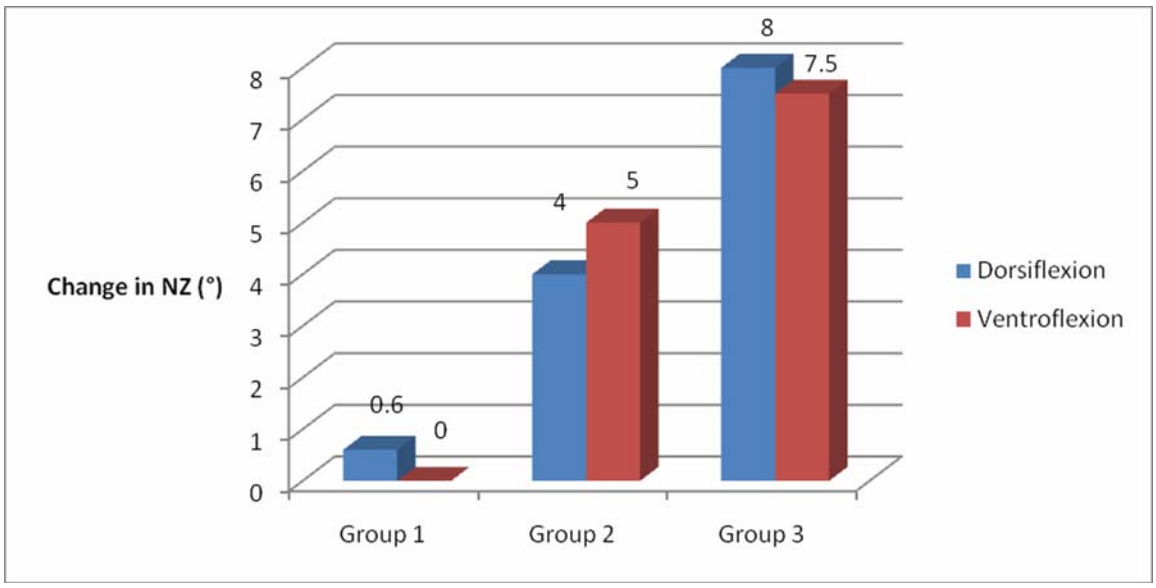

Figure 4-1: Changes in mean neutral zones between pre- and post-modification tests in Groups 1, 2 and 3 in both dorsiflexion and ventroflexion (changes are in degrees and represent increased mean NZ post-modification).

4.2.2 Stiffness calculations

Stiffness was calculated from each load deformation curve at three specific ranges of angular displacement. These ranges were:

1. 6° - 8°
2. 12° - 16°
3. 18° - 20°

The pre-modification and post-modification stiffness at the 3 ranges of angular displacement, as well as the percentage change in stiffness are shown in the Appendix (Tables A-1 to A-6). Mean percentage change in stiffness was calculated for each group in dorsiflexion and ventroflexion. These results are summarised in Tables 4-11 and 4-12.

	Angle	Min*	Max*	Mean*	Median*	SD
Group 1	6° - 8°	-43.71	29.08	-7.11	-7.35	33.59
	12° - 16°	-20.01	30.56	8.69	20.93	22.5
	18° - 20°	-48.54	49	9.62	22.25	37.12
Group 2	6° - 8°	45.54	94.52	69.29	74.68	17.96
	12° - 16°	33.27	71.22	52.46	51.31	14.19
	18° - 20°	16.54	81.53	38.87	30.73	24.92
Group 3	6° - 8°	0	100	50.93	52.8	51.48
	12° - 16°	32.84	100	73.12	77.63	29.7
	18° - 20°	34.27	96.19	58.02	54.1	21.65

Table 4-11: Mean percentage change in stiffness in dorsiflexion at the three ranges of angular displacement. * values given in percentage points. A negative value indicates increased stiffness; a positive value indicates decreased stiffness. SD: standard deviation, angle = range of angular displacement on the load-deformation curve where stiffness calculated

	Angle	Min*	Max*	Mean*	Median*	SD
Group 1	6° - 8°	-12.11	80.12	27.38	20.75	40.11
	12° - 16°	-15.36	32.65	7.69	17.12	21.63
	18° - 20°	-21.31	45.79	13.91	18.42	26.51
Group 2	6° - 8°	13.87	104.96	69.95	66.25	32.15
	12° - 16°	32.94	87.38	65.05	70.38	19.82
	18° - 20°	31.21	94.42	63.20	66.12	20.53
Group 3	6° - 8°	71.96	107.41	92.91	98.63	15.51
	12° - 16°	53.40	95.91	79.43	84.04	14.87
	18° - 20°	56.14	78.11	69.31	73.50	9.69

Table 4-12: Mean percentage change in stiffness in ventroflexion at the three ranges of angular displacement. * values given in percentage points. A negative value indicates increased stiffness; a positive value indicates decreased stiffness. SD: standard deviation, angle = range of angular displacement on the load-deformation curve where stiffness calculated. The percentage change in stiffness for specimen 10 (Group 1) in ventroflexion at 6 - 8° (-594.29%) was considered an outlier and was excluded from the statistical model.

Figures 4-2 and 4-3 summarise the change in percentage stiffness at the various angles of displacement and show the trend that was observed. In dorsiflexion Group 1 showed an initial marginal increase in stiffness (Mean = -7.11% and Median = -7.34%) at 6 - 8° followed by a decrease in stiffness at 12 - 16° (Mean = 8.69% and median = 20.93%) and 18 - 20° (Mean = 9.62% and Median = 22.25%). In dorsiflexion Group 2 showed a decrease in stiffness (Mean = 69.29 % and Median = 74.68 %) at 6 - 8° followed by a decrease in stiffness at 12 - 16° (Mean = 52.46% and median = 51.31%) and 18-20° (Mean = 38.87% and Median = 30.73%). In dorsiflexion Group 3 showed decrease in stiffness (Mean = 50.93% and Median = 52.80%) at 6 - 8° followed by a decrease in stiffness at 12 - 16° (Mean = 73.12% and median = 77.63%) and 18-20° (Mean = 58.02% and Median = 54.10%). These results illustrated by the box plots in Figures 4-4, 4-5 and 4-6.

In ventroflexion Group 1 showed a decrease in stiffness (Mean = 27.38% and Median = 20.75%) at 6 - 8° followed by a decrease in stiffness at 12 - 16° (Mean = 7.69% and Median = 17.12%) and 18-20° (Mean = 13.91% and Median = 18.42%). In ventroflexion Group 2 showed a decrease in stiffness (Mean = 69.95% and Median = 66.25%) at 6 - 8° followed by a decrease in stiffness at 12 - 16° (Mean = 65.05% and median = 70.38%) and 18-20° (Mean = 63.20% and Median = 66.12%). In ventroflexion Group 3 showed a decrease in stiffness (Mean = 92.91% and Median = 98.63%) at 6 - 8° followed by a decrease in stiffness at 12 - 16° (Mean = 79.43% and median = 84.04%) and 18-20° (Mean = 69.31% and Median = 73.50%). These results illustrated by the box plots in Figures 4-7, 4-8 and 4-9.

In dorsiflexion in Group 1 the greatest decrease in stiffness was found at 18 - 20°. This in contrast to Groups 2 and 3 where the greatest decrease in stiffness was found at 6 - 8° and 12 - 16° respectively. In ventroflexion however Groups 1, 2 and 3 all show the greatest decrease in stiffness at 6 - 8° of angular deflection.

It was noted that the difference between the 3 groups became less when the stiffness was measured at greater degrees of angular displacement. Group 1 (control group) showed an initial

increase in stiffness at 6°- 8° in dorsiflexion. The percentage change in stiffness for specimen 10 (Group 1) in ventroflexion at 6 - 8° was considered an outlier and was excluded from the statistical model.

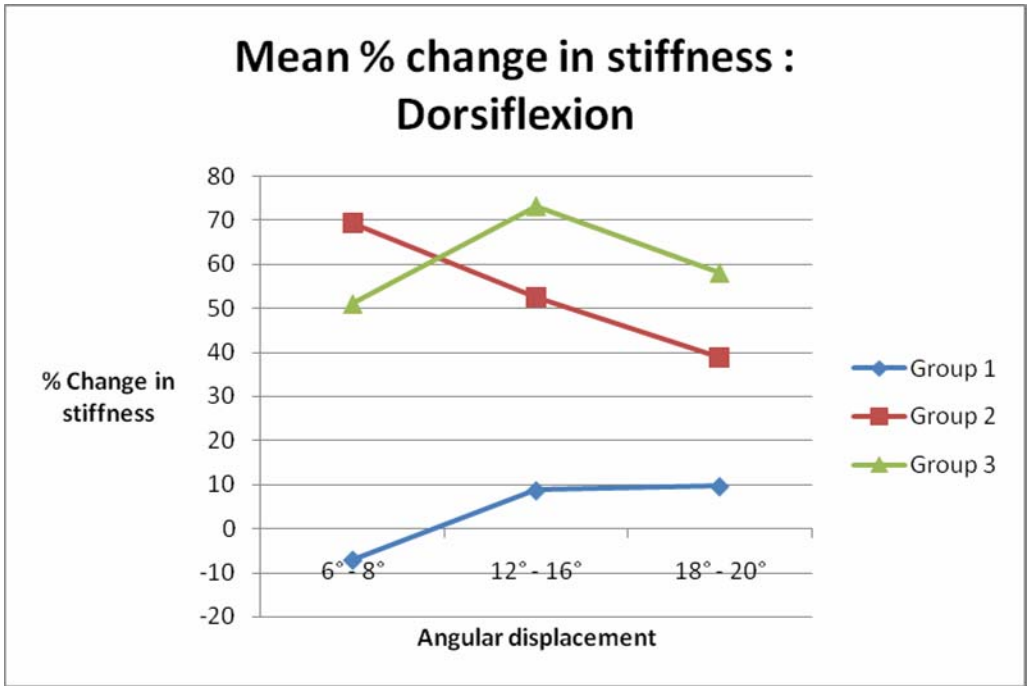

Figure 4-2: Mean percentage change in stiffness in dorsiflexion. Note the difference between Groups 1, 2 and 3 decreases at increasing angular displacement (negative % = increase in stiffness).

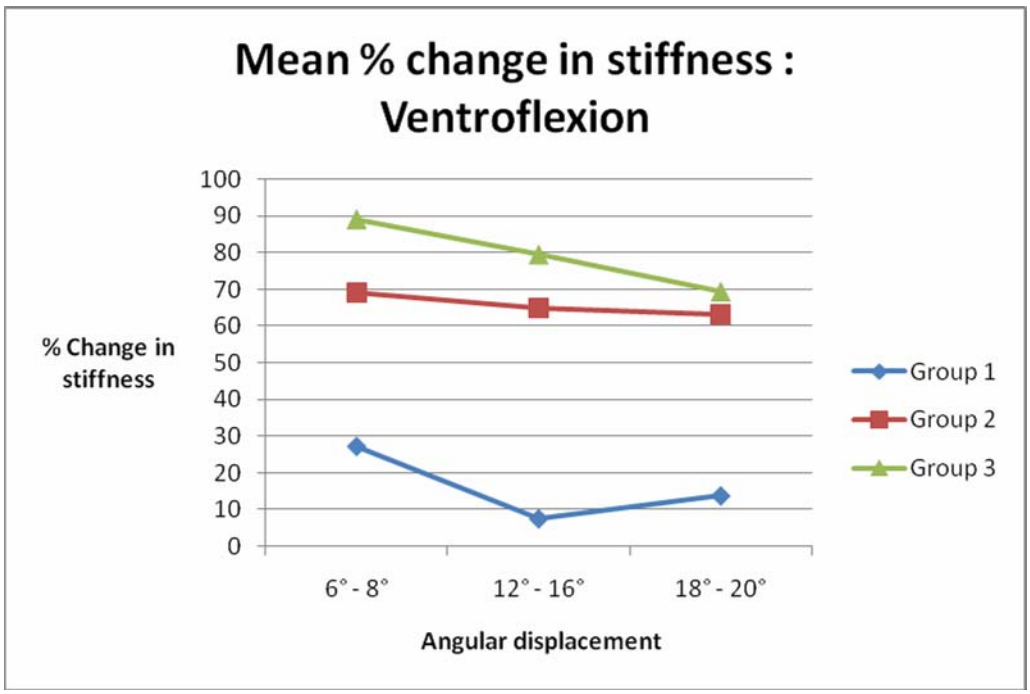

Figure 4-3: Mean percentage change in stiffness in ventroflexion. Note the difference between groups 1, 2 and 3 decreases at increasing angular displacement. (negative % = increase in stiffness). Note in this figure the data from specimen 10 has been omitted at the 6° - 8° angle as it was considered an outlier.

When comparing the groups in dorsiflexion at the three angles of deflection (6°-8°, 12°-16° and 18°-20°) using the Kruskal-Wallis One Way Analysis of Variance (ANOVA) and the Tukey-Kramer Multiple-Comparison Test following results were found. There was a statistically significant difference between Group 1 and Group 2 ($p<0.05$), and between Group 1 and Group 3 ($p<0.05$), but not between Group 2 and Group 3 at 6°-8° and at 12°-16°.

At 18°-20° of deflection, there was a statistically significant difference between Group 1 and Group 3 ($p<0.05$), but no statistically significant difference between Group 1 and Group 2, or between Group 2 and Group 3.

Comparisons between the groups in ventroflexion revealed similar results i.e. statistically significant differences between Group 1 and Group 2, and between Group 1 and Group 3 ($p<0.05$) at 6°-8° and 12°-16° of deflection.

At 18°-20° of deflection there was a statistically significant difference between Group 1 and Group 3 ($p<0.05$), but no statistically significant difference between Group 1 and Group 2, or between Group 2 and Group 3.

It is evident that Group 1 showed the least reduction in stiffness whilst Group 3 demonstrated the greatest reduction in stiffness.

When combining dorsiflexion and ventroflexion in a single statistical model (mixed effects multiple regression model) the results were as follows: There was a 48.3 percentage point reduction in stiffness for Group 2 compared to Group 1 and this was highly statistically significant ($P < 0.001$). Group 3 showed a 59.8 percentage point reduction in stiffness compared to Group 1 ($P < 0.001$) which was also highly statistically significant. The difference in reduction in stiffness between Groups 2 and 3 was 11.5 percentage points ($P = 0.066$) and was marginally significant.

Reduction in stiffness was more pronounced at smaller deflection angles. Comparing reduction in stiffness at 6°-8° to reduction in stiffness at 12°-16° showed no statistically significant difference ($P = 0.221$). There was a statistically significant difference in reduction in stiffness at 6°-8° compared to 18°-20°, ($P = 0.002$).

Figures 4-4 to 4-9 are box plots depicting the range of the change in stiffness for Groups 1, 2 and 3 in dorsiflexion and ventroflexion at all three angles of deflection. The medians, 10^{th}, 25^{th}, 75^{th} and 90^{th} percentiles are indicated in the figures.

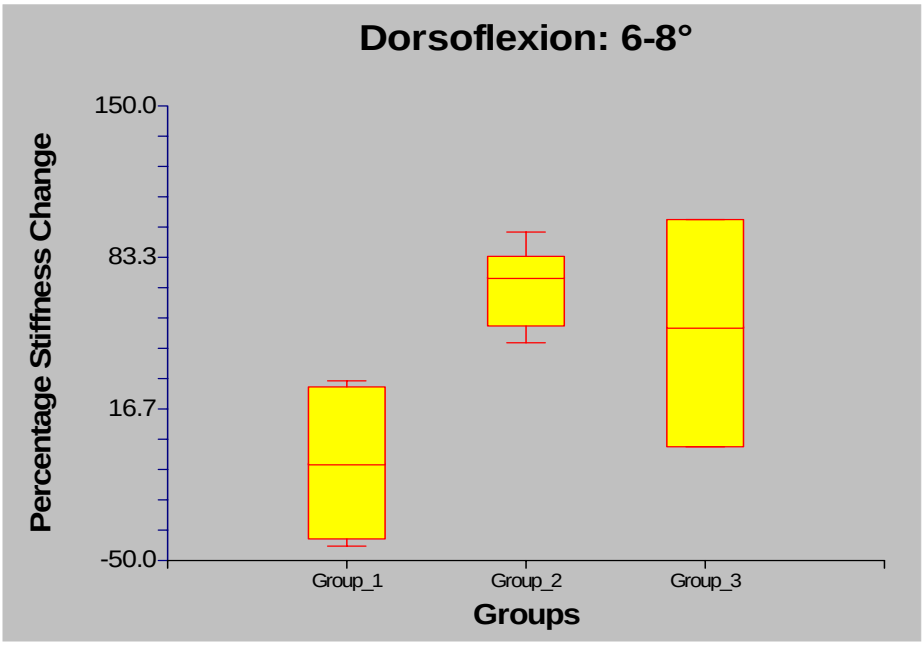

Figure 4-4: Percentage change in stiffness in dorsiflexion at 6°-8° of deflection. Data are shown as median (horizontal line within box), 25th and 75th percentiles (horizontal ends of boxes), and 10th and 90th percentiles (T-bars).

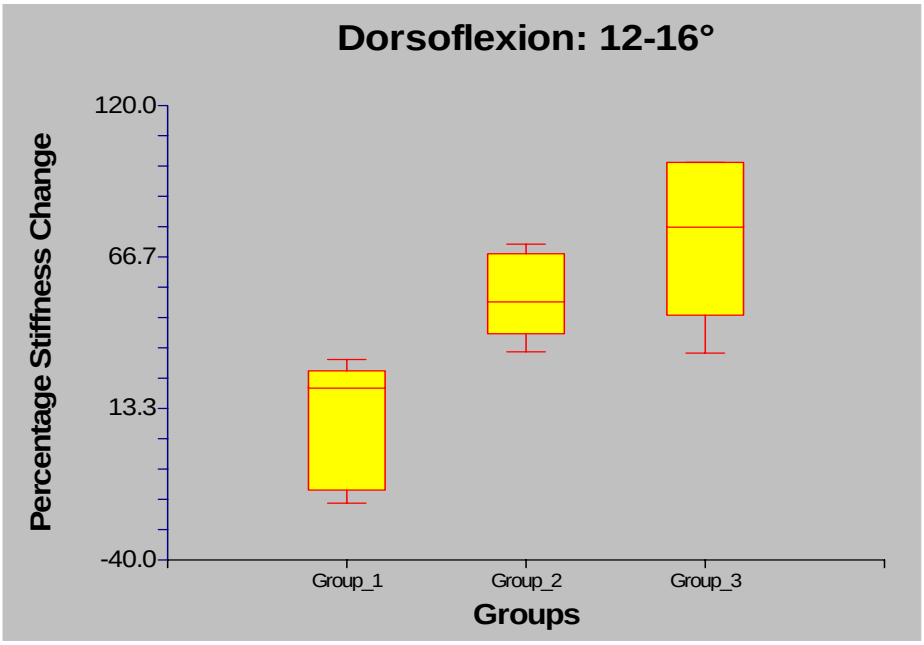

Figure 4-5: Percentage change in stiffness in dorsiflexion at 12°-16° of deflection. Data are shown as median (horizontal line within box), 25th and 75th percentiles (horizontal ends of boxes), and 10th and 90th percentiles (T-bars).

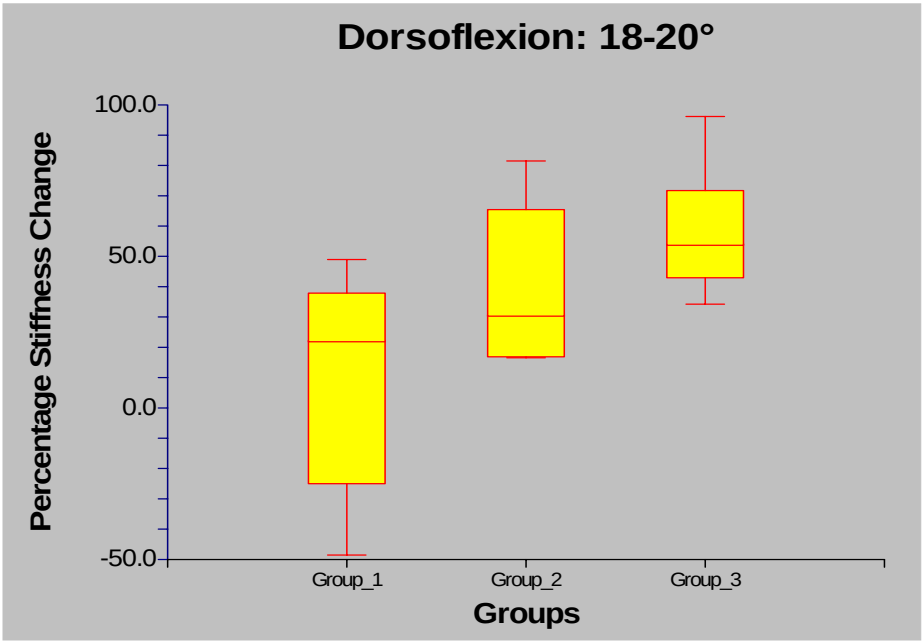

Figure 4-6: Percentage change in stiffness in dorsiflexion at 18°-20° of deflection. Data are shown as median (horizontal line within box), 25th and 75th percentiles (horizontal ends of boxes), and 10th and 90th percentiles (T-bars).

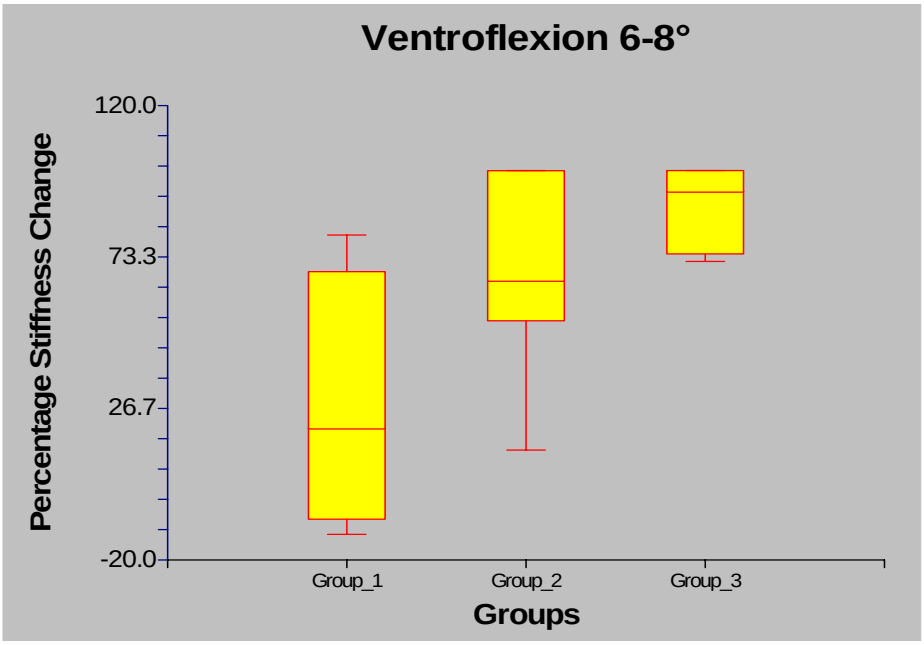

Figure 4-7: Percentage change in stiffness in ventroflexion at 6°-8° of deflection. Data are shown as median (horizontal line within box), 25th and 75th percentiles (horizontal ends of boxes), and 10th and 90th percentiles (T-bars). Note in this figure the data from specimen 10 has been omitted at the 6° - 8° angle as it was considered an outlier.

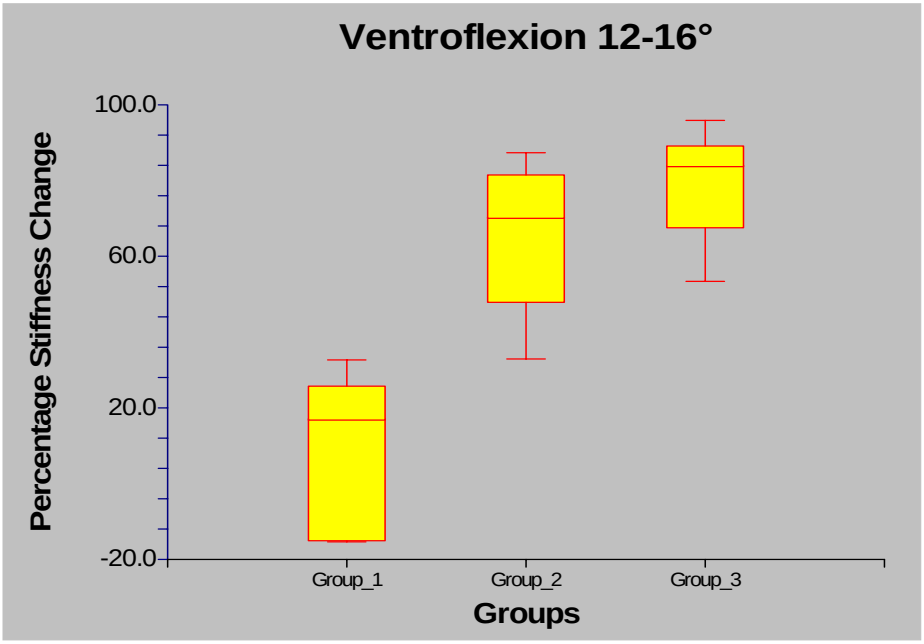

Figure 4-8: Percentage change in stiffness in ventroflexion at 12°-16° of deflection. Data are shown as median (horizontal line within box), 25th and 75th percentiles (horizontal ends of boxes), and 10th and 90th percentiles (T-bars).

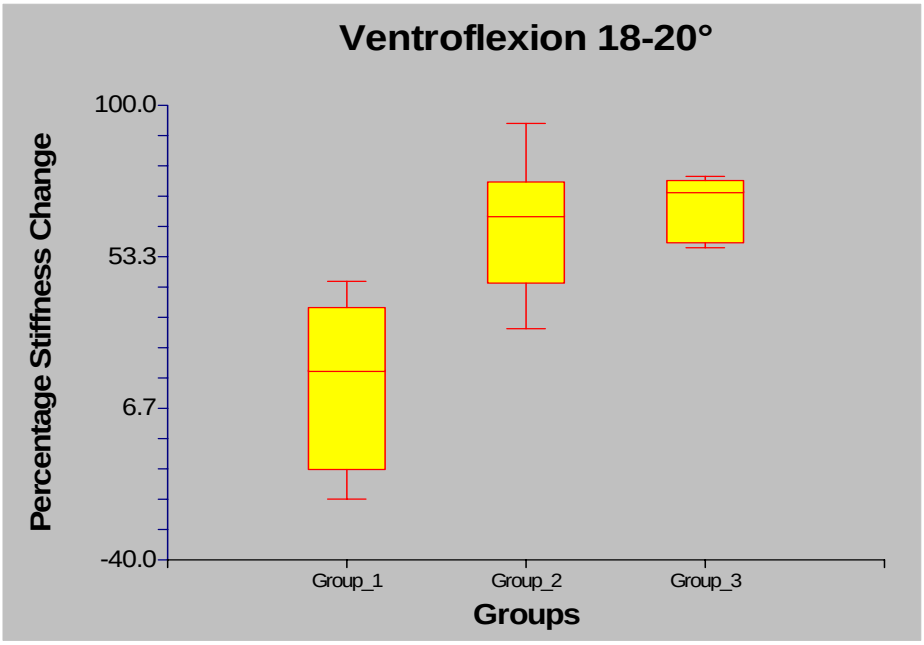

Figure 4-9: Percentage change in stiffness in ventroflexion at 18°-20° of deflection. Data are shown as median (horizontal line within box), 25th and 75th percentiles (horizontal ends of boxes), and 10th and 90th percentiles (T-bars).

4.2.3 Test to failure

All specimens were tested to failure in ventroflexion once the non-destructive testing was completed. The results of specimen 5 (Group 1) when tested to failure in ventroflexion was lost due to a computer malfunction therefore data is only available for 17 specimens in total. The load-deformation curves for the tests to failure in ventroflexion are found in Figures A-73 to A-89. The results are summarized in Table A-7 in the Appendix. Figures 4-10 and 4-11 illustrate the angle of displacement and the force at failure.

The nature of the failure was determined from observation of the specimen during testing, observation of the recorded video footage and examination of the specimen after testing. The nature of the failure was similar in all specimens i.e. rupture of the supra- and interspinous ligaments first, followed by rupture of the articular joint capsule in Group 1. In Groups 2 and 3 failure was characterised by rupture of the articular process (facet) joint capsules. No fractures were noted in any specimens during failure. The load deformation curves of Specimens 11, 15, 16 and 18 did not show typical back sliding of the graph typical of failure. Macroscopic failure was however evident in these specimens. All these specimens showed a sudden change in the gradient of the graph which corresponded with the point at which macroscopic failure was noted. It was therefore accepted that failure had occurred.

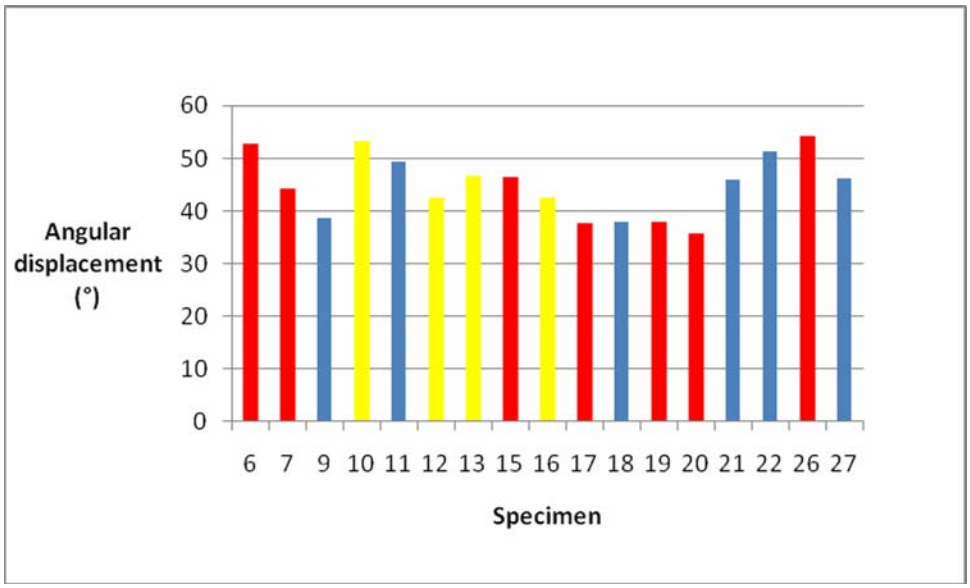

Figure 4-10: Angular displacement at failure. (yellow = Group 1, red = Group 2 and blue = Group 3)

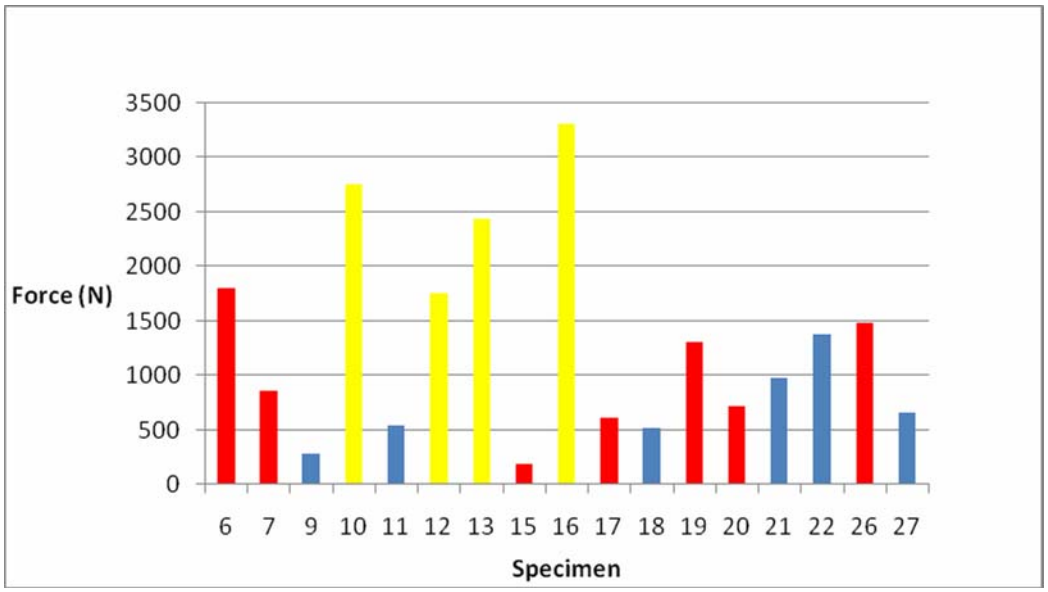

Figure 4-11: Peak force at failure. (yellow = Group 1, red = Group 2 and blue = Group 3)

From Figures 4-10 and 4-11 it is clear that the interspecimen variation in terms of force at failure was large whilst the interspecimen variation in terms of angular displacement at failure was small. The mean force at failure (for all groups combined) was 1270.476N (SD = 898.599) and the mean angle of deflection at failure (for all groups combined) was 45.18° (SD = 5.69). For

Group 1 the mean force at failure was 2566.688N (SD = 647.277) and the mean angle at failure was 46.29° (SD=5.06). For Group 2 the mean force at failure was 995.225N (SD = 559.394) and the mean angle at failure was 44.81° (SD=6.92). For Group 3 the mean force at failure was 727.461N (SD = 391.163) and the mean angle at failure was 44.86° (SD=5.43).

The strength of the lumbosacral motion unit in ventroflexion decreased by 61.3% after MDL + D (Group 2) and by 71.7% after SDL + D (Group 3) when compared to the control specimens (Group 1)

There was a statistically significant difference between the force at failure in Groups 2 and 3 compared to Group 1 ($p<0.001$). There was no statistically significant difference between Groups 2 and 3 with regards to force at failure. No statistically significant difference was detected with regard to the angular displacement at failure between the three groups ($p=0.86$).

Chapter 5: Discussion

5.1 Context and Objective

This study was a biomechanical *in vitro* investigation to determine the effect of two different sized laminectomies combined with a standardised discectomy, on the stiffness of the lumbosacral motion unit. Clinically, lumbosacral instability is thought to play a role in the pathogenesis of degenerative lumbosacral stenosis (DLS) [17, 33]. The recurrence rate associated with surgical treatment of DLS is between 18 and 33% [6, 7, 9, 29]. If surgical treatment exacerbates pre-existing lumbosacral instability it may be responsible for the relatively high recurrence rate. To my knowledge, no studies have investigated whether the size of the dorsal laminectomy had an effect on the stability of the lumbosacral motion unit. The primary objective of this study was therefore, to determine whether dorsal laminectomy and discectomy resulted in significant instability of the lumbosacral motion unit and secondly whether the size of the dorsal laminectomy had an effect on lumbosacral stability.

5.2 Methodology

This study was performed using a four-point bending jig which is the recognized testing methodology for applying pure bending force to a specimen. The non-destructive testing was displacement controlled, i.e. each specimen was displaced to a set point and the force required to achieve this displacement was recorded. Load deformation curves were obtained for each specimen tested. The specimens were tested non-destructively in both dorsiflexion and ventroflexion prior to and after surgical modification, except for Group 1 which had no modification between tests. Each specimen was then tested to failure in ventroflexion. The investigation was performed such that each specimen acted as its own control during the non

destructive testing, whilst Group 1 acted as the controls for Group 2 and 3 during testing to failure in ventroflexion.

The two different size laminectomies were standardised by making accurate measurements of the laminae with a vernier calliper and then removing a predetermined percentage of the lamina. For the MDL the caudal aspect of the lamina of L7 was removed without disturbing the dorsal spinous process. For the SDL 75% of the lamina of L7 and 50% of the lamina of S1 was removed. The width of the laminectomies for both MDL and SDL was the same i.e. laterally to the medial aspects of the articular processes (facets) of L7. The discectomy was performed by making a rectangular incision in the dorsal *Anulus fibrosus* which extended from vertebral sinus to vertebral sinus laterally and to the vertebral endplate of L7 cranially and the vertebral endplate of S1 caudally. The *Nucleus pulposus* was curetted until no further material could be retrieved. This ensured that laminectomies and discectomies were standardised within the groups.

The angle of deflection was chosen after a small pilot study which demonstrated no evidence of failure at 21° of deflection. This angle is certainly larger that those used in previous studies as discussed below but does fall within the calculated range of motion for flexion and extension of the lumbosacral joint [4]. The specimens were loaded at a rate of 4.8mm per minute. This is a higher rate of loading that that used in the study by Smith *et al* [29] (2mm per minute). It could be assumed that this may more closely resemble the rate of loading in the live animal.

It is very difficult to compare the forces used in a biomechanical *in vitro* study to those generated in the lumbosacral joint of a live animal. It is however known that between 0.73 and 1.04 times the body weight is transmitted through the hip during a three legged stance [25]. The testing was displacement controlled. This meant the specimens were stressed to a set angular deflection and the force required to achieve this was recorded. This force varied greatly between specimens. It is unlikely that these forces correlate accurately with those in the live animal due to the fact that this study has by definition negated the effects of the active control system i.e. musculature.

In this investigation a deflection of approximately 21° was achieved in both dorsiflexion and ventroflexion. There was no evidence at the time of testing of macroscopic failure in any of the specimens during non-destructive testing. The resultant load deformation curves did not reveal

any evidence of failure in any of the specimens either. Failure of the specimen would have been identified as a sudden drop (back sliding) in the graph of the load-deformation curve. The study by Smith *et al* [29] stressed the lumbosacral motion units to approximately 10 - 12° in both dorsiflexion and ventroflexion during non destructive testing. In the study by Meij *et al* [18] specimens were tested to an angular deflection of approximately 15° in dorsiflexion and 15 - 20° in ventroflexion. It is therefore evident that this study resulted in greater angular deflection of the specimens in both dorsiflexion and ventroflexion when compared to the studies by Smith *et al* and Meij *et al.*

5.3 Results and Comparisons

The results of this investigation showed that a standard dorsal laminectomy combined with a discectomy (SDL + D) resulted in a 59.8% reduction in stiffness of the lumbosacral motion unit compared to unmodified specimens (Group 1), while a mini-dorsal laminectomy and discectomy (MDL + D) resulted in a 48.3% reduction in stiffness of the lumbosacral motion unit compared to unmodified specimens (Group 1). These figures are the overall reduction in stiffness i.e. combining all angles of deflection and both dorsiflexion and ventroflexion. Dorsal laminectomy (regardless of extent) combined with a discectomy results in significant reduction in stiffness of the lumbosacral joint. These results indicate a greater reduction in stiffness than the study by Smith *et al* [29] who found that dorsal laminectomy + discectomy had no statistically significant effect of stiffness in dorsiflexion and resulted in a 33.1% decrease in stiffness in ventroflexion. This may be explained by the fact that this study was performed at greater degrees of angular displacement, 21° compared to 10 - 12°. Greater angular deflection would be expected to place more strain on the tissues and structures of the lumbosacral motion unit. It may therefore explain the fact that this study found a greater decrease in stiffness following laminectomy and discectomy compared to previous studies.

The second possible reason for our results showing a greater decrease in stiffness may be the type of discectomy performed. Smith *et al* [29] performed their discectomy with a power burr. In this investigation the discectomy was performed by making a rectangular incision in the dorsal

annulus (from vertebral sinus to vertebral sinus and end plate to end plate). Effectively the majority of the dorsal *Anulus fibrosus* was excised, i.e. an annulectomy was performed. *Nucleus pulposus* was curetted until no further disc material could be retrieved. Our annulectomy was possibly more aggressive and extensive than that described by Smith *et al* [29]. It is also possible that the pulpectomy performed in this study was more extensive than that in the Smith *et al* study [29] as the disc was curetted until no further *Nucleus pulposus* could be retrieved as opposed to the discectomy being performed with a power bur. It is unclear what volume of *Nucleus pulposus* was removed by this technique. Previous studies have concluded that discectomy is the single procedure responsible for the most of the reduction in stiffness in the lumbar spine when subjected to lateral bending [12, 19, 24, 29].

The study by Meij *et al* [18] showed no statistically significant change in the stability of the lumbosacral motion unit after dorsal laminectomy and partial discectomy. The discectomy described in the study by Meij *et al* was similar in size and extent to that used in this study. These results may differ from those of the present study due to larger angular deflection of the specimens during testing i.e. 21° as opposed to approximately 15° in dorsiflexion and 15 - 20° in ventroflexion in the Meij *et al* study.

This study did not compare groups with and without discectomies. It is therefore impossible in this study to determine the relative contribution to the change in stiffness of the laminectomy and discectomy. From previous studies [12, 19, 24, 29] it is likely that discectomy plays a major role in reducing the stiffness of the lumbosacral motion unit. My feeling is that the discectomy and specifically the dorsal annulectomy is a significant contributor to the reduction in overall stiffness but particularly stiffness in ventroflexion of the lumbosacral motion unit. Observation of the specimens when performing the laminectomies and discectomies tended to indicate that the subjective stiffness of the specimen reduced notably after the discectomy had been performed. As mentioned previously this study did not compare stiffness between groups with and without discectomy – this was merely a subjective assessment made during specimen preparation. The conclusion drawn from this is that the discectomy, specifically the dorsal annulectomy played the dominant role in reducing stiffness while the laminectomy probably has a minor role in reducing stiffness of the lumbosacral motion unit.

Previous studies have also concluded that the volume of *Nucleus pulposus* removed during discectomy has an influence on the ultimate stability of the joint [14]. In this study the disc was curetted until no further nucleus pulposus could be retrieved. The fact that all retrievable *Nucleus pulposus* was removed implies that this was an extensive discectomy and is therefore likely to have a significant effect on the stability of the lumbosacral motion unit.

In a previous retrospective study [33], it was found that clinical cases of DLS that had surgical treatment by means of a dorsal laminectomy alone had a better outcome and prognosis than those that received dorsal laminectomy combined with a partial discectomy. This may indicate that the partial discectomy leads to increased lumbosacral instability and therefore recurrence of symptoms and/or poor response to surgery [33]. This fact does tend to provide added support for the concept that instability, either persistent after surgery or exacerbated by surgery, especially discectomy, may play a significant role in the pathogenesis of DLS. It therefore raises the question as to whether discectomy should be performed at all and whether stabilisation should be routinely performed. Another option would be to consider stabilisation if a discectomy has been performed.

Human biomechanical investigations have shown that the stability of the lumbar spine segments (cadavers) is negatively influenced by the extent of the discectomy. It was reported by Panjabi *et al* [20] that incision of the annulus and removal of *Nucleus pulposus* changed the biomechanical properties of the lumbar spine [11, 20]. The more extensive the discectomy, the greater the reduction in stiffness. This refers to both the size of the annulectomy and the volume of *Nucleus pulposus* removed [11, 20]. Due to anatomical and biomechanical differences between human and animal spines it is difficult to know the significance and accuracy of comparisons between human and animal spinal biomechanical studies.

Results of this study indicate an 11.5% reduction in stiffness of the lumbosacral motion unit in the SDL + D group compared to the MDL + D group (P = 0.066). This difference could be considered to be approaching significance or indicating a trend towards significance. Larger group sizes may have resulted in a statistically significant difference. The opposite is also possible i.e. larger group sizes may in fact indicate no statistically significant differences between Groups 2 and 3. With the recent description of minimally invasive procedures and

endoscopically assisted lumbosacral foraminotomies, there may well be indication to perform mini-dorsal laminectomies.

It is difficult to explain the difference between the 2 laminectomy groups. Anatomically the difference between the SDL + D group and the MDL + D group is the amount of the lamina excised. In the MDL the dorsal spinous process of L7 is preserved and the lamina of S1 is left intact. It is unlikely that the excision of the spinous process of L7 in the SDL + D group resulted in a difference as in both SDL + D and MDL + D groups, the interspinous and supraspinous ligaments were excised. One possible explanation is that in some individuals there is a telescoping of the lamina of S1 ventral to the caudal lamina of L7 [33]. This "overlapping" of the laminae may result in some impingement and therefore increased stability. With a SDL both the caudal portion of the lamina of L7 as well as the cranial portion of the lamina of S1 is excised thereby eliminating this impingement and possibly reducing stability and increasing range of motion. In contrast, with the MDL the lamina of S1 is untouched and therefore may still impinge with the remains of the L7 lamina especially in ventroflexion.

The neutral zone of the load-deformation curve is defined as that area were angular displacement occurs at a zero force, i.e. the specimen bends under its own weight. This was noted in the study as sagging of the specimen in the four-point bending jig before the actuator had applied a force. The results of this study indicate an increase in the neutral zone post-modification in Groups 2 and 3 in comparison to Group 1. This is consistent with findings of a previous study (Meiji *et al* [18]) who found that the neutral zone stiffness decreased following dorsal laminectomy and partial discectomy. From the results it is clear that reduction in stiffness was greatest at lower angles of displacement. This correlates to the neutral zone. It is therefore concluded that stiffness reduction was greatest in the neutral zone and was the least at larger angles of the displacement. The change in stiffness (reduction is stiffness) was also smallest at the greater angles of displacement.

The clinical relevance of this observation could be that there is a reduction in lumbosacral stability post-operatively, especially at small angles of flexion. These smaller angles will be well within the normal range of motion of the lumbosacral joint. This instability may in turn lead to soft tissue proliferation in an attempt to stabilise the lumbosacral joint. This soft tissue proliferation may then result in impingement on the *Cauda equina*.

There was a greater reduction in stiffness when tested in ventroflexion compared to dorsiflexion. Reduction in stiffness of Groups 2 and 3 at all three angles of deflection was greater in ventroflexion compared to dorsiflexion. This may be explained by the fact that the laminectomy and discectomy were all performed on the dorsal aspect of the specimen i.e. the dorsal compartment. When the specimen is tested in ventroflexion the dorsal aspect is the tension surface of the lumbosacral motion unit. The ligamentous structures are very important in terms of resisting these forces on the tension surface. SDL + D and MDL + D resulted in the excision of interspinous and supraspinous ligaments, dorsal annulus as well as varying amounts of lamina.

The mode of failure in all specimens tested to failure in ventroflexion was similar. Failure in Group 1 specimens was characterised by rupture of the interspinous and supraspinous ligaments as well as the articular process (facet) joint capsule. In Groups 2 and 3 failure was characterised by rupture of the articular process (facet) joint capsule. Maximum angular displacement achieved was approximately 55°. Due to the dimension of the four-point bending jig, further displacement resulted in the aluminium tubing impinging on the jig. It is possible that further angular displacement would have resulted in the failure of additional structures e.g. articular process joint fracture. It is the author's opinion that these interspinous and supraspinous ligaments are important in providing support, particularly when stressed in ventroflexion, as stated in the previous paragraph these structures are situated on the tension surface when the lumbosacral motion unit is stressed in ventroflexion.

A previous study by Smith GK *et al* [28] showed a 36.2% decrease in vertebral column rigidity in extreme ventroflexion after excision of the supraspinous and interspinous ligaments, and a 62.4% decrease in ultimate bending strength [28]. The reduction in strength at failure of Group 2 compared to Group 1 was 61.3% and Group 3 compared to Group 1 was 71.7%. These results are similar to those of Smith GK *et al* [28], and again illustrate the importance of these ligaments in maintaining vertebral stability.

Angular displacement at failure (yield point) during ventroflexion showed no significant difference between the groups. This indicates that anatomical structures, specifically the interspinous and supraspinous ligaments as well as the articular process (facet) joint capsule are only exposed to excessive strain once a certain angular deflection is reached. It thus appears that

failure will occur at similar angular displacements regardless of stiffness. However in specimens with reduced stiffness (Groups 2 and 3) the force required to achieve this angular displacement and hence failure will be significantly less. This is confirmed by the significant reduction in strength of the specimens in Groups 2 and 3. One can however conclude that there is no significant difference in strength between those specimens undergoing MDL and those undergoing SDL.

Further studies would be required to compare mini-dorsal laminectomies with and without excision of the interspinous and supraspinous ligaments. This could be another potential advantage of a mini-dorsal laminectomy, i.e. an ability to perform a decompressive procedure without compromising these ligamentous structures. Clinically it would be possible to perform mini-laminectomies and preserve the supra- and interspinous ligaments as the dorsal spinous process of L7 is not excised with a mini-laminectomy. This is potentially beneficial especially with endoscopically assisted foraminotomies.

Potential limitation of mini-laminectomies however would include limited ability to decompress the neural structures within the vertebral canal if the compression extends cranially and/or caudally a significant distance from the actual lumbosacral disc.

Another potential advantage of preservation of the interspinous ligaments relates to the support provided by the epaxial musculature. Studies in cats have shown that there is a neural feedback after stretching of the interspinous ligaments that result in contracture of the epaxial musculature [30, 32]. Contraction of the epaxial musculature would play significant role in providing dynamic stability and support to the lumbosacral motion unit in the live animal. The exact location of these receptors remains unknown but the interspinous ligaments may play an integral role in this active mechanism which adds to lumbosacral stability.

5.4 Lumbosacral stabilisation

Stabilisation of the lumbosacral joint remains a controversial topic. There are currently no objective criteria that indicate which cases should be stabilized and which cases not, nor is there

a clear understanding whether and how lumbosacral instability is associated with the pathogenesis or poor long-term post-operative results. Lumbosacral "stepping" or incongruency can be assessed on survey and stressed radiographs as well as CT and MRI [33, 34]. "Stepping" is commonly seen in clinical cases of DLS; 78% of cases in one report [33, 34]. Although this "stepping" is considered an indication of instability it is unknown as to what degree of "stepping" should be considered clinically significant. It is possible that a step as small as 2mm may in fact represent clinically significant instability [33, 34]. The decision to stabilise the lumbosacral joint seems to be largely dependent on the surgeon's personal preference. The size of the "step" as determined on diagnostic imaging, whether the step increases significantly on stressed radiographs (dorsiflexion) and the presence of overriding articular process (facet) joints intra-operatively are all factors that can be used to assess for the presence of clinical lumbosacral instability. The results of this study seem to provide added evidence that stabilisation may be indicated more frequently than previously thought although it does not look at the dynamic spinal stabilizers.

Another factor supporting stabilisation is the fact that DLS is a dynamic condition [26, 36]. That is to say that compression of the C*auda equina* is worse in extension and reduced in flexion [3, 17].

Many stabilisation techniques have been described. These include pedicle screw-rod fixation [5, 18], articular process (facet) screws or pins [3, 27], pins and polymethylmethacrylate (PMMA) [19] and external skeletal fixation [1]. A recent study by Meiji *et al* investigated the biomechanics of the lumbosacral joint after stabilisation with pedicle screw- rod fixation [18]. Results indicated that this was a useful and successful method of lumbosacral stabilisation in the dog.

The effect of cyclical loading on the lumbosacral joint was not investigated in this study. Investigations of the human lumbar spine [14] following discectomy have shown increased range of motion and instability after cyclical flexion / extension loading after a period of time, even if this was not apparent immediately after surgery. Factors that appear to affect spinal instability (human) over time include volume of *Nucleus pulposus* removed, location and extent of the annulectomy as well as repetitive (cyclical) loading [14]. This provides added support for some sort of stabilisation technique to be performed with lumbosacral decompression. This fact may be a

major contributor to the high percentage of cases which show recurrence of clinical symptoms after a period of time.

Another argument for stabilisation is the fact that many of these cases have compression of the nerve roots in the foraminae. In some cases distraction and stabilisation will “open up” the intervertebral foramen and relieve nerve root compression. It may also be argued that distraction and stabilisation of the lumbosacral joint may result in thinning and remodelling of the hypertrophied soft tissue that was contributing to the compression. When the joint is distracted these tissues will also be stretched out and therefore not “sag” into the vertebral canal as much thereby potentially reducing compression. This concept is similar to the rationale behind distraction and fusion for traction responsive cervical spondylopathy .

The findings of this study indicate an increase in instability following dorsal laminectomy and partial discectomy. The relatively high recurrence rate associated with the surgical treatment of DLS may be in part related to this increase in instability. This instability may perpetuate or result in delayed (6 – 18 months) [13, 33] recurrence of the clinical symptoms of DLS. Decompression combined with a stabilisation technique may reduce the likelihood of recurrence of clinical symptoms as well as improve the overall success rate for the surgical treatment of DLS.

5.5 Study limitations

There were certain limitations in this study. In some of the load deformation curves obtained in this study there was irregularity/ oscillation of the curve. This was most apparent in specimens which required a very small force to achieve the set displacement. The cause of the irregularity of the curve was “noise” mainly due to the fact that the load cell used was a 300 Kg load cell (3000N). Therefore at lower forces (< 5N) there was significant noise in the signal. Another factor which influenced the load deformation curves was the fact that over 6000 data points were collected for each test. This resulted in an extremely sensitive curve which would detect any abnormality or variation. In other similar studies data sampling was at much lower frequencies and significantly less data points were collected (between 6 and 40 data points) [29].Future studies could collect data at a lower frequency (10Hz) compared to this study (100Hz). The use of a

smaller load cell (100kg or less) would have further improved data acquisition and reduced noise.

Specimens in this study underwent a double freeze / thaw cycle during preparation. All specimens were subjected to the same protocol. This together with the fact that each specimen served as its own control eliminates any effect that the double freeze / thaw cycle would have had on the results of the study.

Further limitations of this study included those of any *in vitro* biomechanical study. The effects and contributions to lumbosacral stability of the active (musculature) and neural feedback systems have been ignored. The cyclical loading as experienced in the live animal has also not been replicated, and it appears that repetitive loading over a prolonged period may have significant effect on range of motion and stability of the lumbosacral joint [14].

It is important to note that all test specimens were from cadavers without any evidence of lumbosacral disease. The selection of specimens was based on radiographic findings, i.e. specimens with radiographic evidence of lumbosacral disease, transitional vertebra or other forms of lumbosacral pathology were excluded. Magnetic resonance imaging (MRI) and computed tomography (CT) are considered the diagnostic imaging modalities of choice for DLS due to their ability to obtain cross sectional images as well as identify soft tissue compression accurately (especially MRI) [33]. It is possible that specimens with subtle changes associated with DLS that were not detected on radiographs were included in the study. These changes may potentially have been detected if MRI or CT had been utilised to screen the specimens for inclusion in the study. If present, these changes may have influenced the results above.

How stability and stiffness in this biomechanical study would be affected in specimens with DLS remains unknown. Two scenarios are proposed:

- One situation that could be expected is that in clinical cases of DLS there is already instability present. These lumbosacral joints are then further destabilized during surgery which may result in clinically significant lumbosacral instability.
- The second option is that in chronic cases of DLS there is often already substantial ventral spondylosis at L7/S1 and associated hypertrophy of all the ligamentous structures. This may

in turn have increased lumbosacral stability and make it more resistant to any instability associated with surgery.

This study was done using lumbosacral motion units from normal cadavers. Clinical cases with DLS, ventral spondylosis and lumbosacral instability may show different biomechanical responses to surgical modification. A further study investigating groups with specimens from animals with clinical DLS would address this aspect. It would however be very difficult to standardise the groups as there would inherently be a lot of variability.

The effect surgical modification on axial rotation and lateral bending were also not evaluated in this study. Although movement in these directions is significantly less than that of flexion and extension in the sagittal plane, there may be a significant interaction between movements in all three of these planes.

As mention previously group sizes were small and therefore there is the possibility of type II statistical errors. Future studies should therefore make use of larger group sizes.

5.6 Future research

Future research should be directed at the influence of repetitive cyclical loading on lumbosacral stability, the contribution that the interspinous and supraspinous ligaments make to stability, and an evaluation of the different stabilisation techniques and their ability to successfully stabilize the lumbosacral joint. The ability to perform *in vivo* studies would significantly increase our understanding of the complex interaction between all the stabilizers of the lumbosacral joint (passive, active and neural) and the effect of surgical modification on the stability of the lumbosacral joint in the live animal. This would also allow determination of the clinical relevance or significance of surgically induce instability. These techniques are being utilized in human spinal research and would greatly improve our understanding of lumbosacral biomechanics in the canine field [23]. An accurate understanding of the magnitude of the forces exerted on the lumbosacral joint and its response to this is essential in determining clinical treatment modalities and stabilisation techniques.

Chapter 6: Conclusion

This investigation confirms that decompressive surgery of the lumbosacral joint by means of dorsal laminectomy combined with a discectomy does result in decreased stiffness of the lumbosacral joint. Mini dorsal laminectomy resulted in less instability compared to a standard dorsal laminectomy. This difference however was not statistically significant although could be seen as approaching significance. Larger group sizes would be required to determine this fact.

The results of this study indicate that there may well be increased indication for lumbosacral stabilisation in combination with lumbosacral decompression, and that this may indeed reduce the reoccurrence rate of DLS post operatively.

Further studies should include larger studies (larger group sizes) and, if possible, include specimens from cadavers with lumbosacral disease. One should however always bear in mind that biomechanical studies have their inherent weaknesses due to the fact that the muscular and neural systems are not evaluated.

References

1. Auger J, Dupuis J, Quesnel A, *et al*: Surgical treatment of lumbosacral instability caused by discospondylitis in four dogs. *Veterinary Surgery* 29: 70-80, 2000
2. Adams WH, Daniel GB, Pardo AD, *et al*: Magnetic resonance imaging of the caudal lumbar and lumbosacral spine in 13 dogs. *Veterinary Radiology and Ultrasound* 36: 3-13, 1995
3. Bagley RS: Surgical stabilization of the lumbosacral joint. In Slatter D(ed) *Textbook of Small Animal Surgery* (3rd edition). Saunders, Philadelphia: 1238-1243, 2003
4. Benninger MI, Seiler GS, Robinson LE, *et al*: Three-dimensional motion pattern of the caudal lumbar and lumbosacral portions of the vertebral column of dogs. *American Journal of Veterinary Research* 65: 544-551, 2004
5. Carpenter LG, Taylor B, Nye R, *et al*: Treatment of degenerative lumbosacral stenosis in military working dogs with dorsal laminectomy and pedicle screw and rod fixation. *Proceedings ACVIM Congress* 2003, CD ROM.
6. Chambers JN, Barbara A, Selcer JE, *et al*: Results of treatment of degenerative lumbosacral stenosis in dogs by exploration and excision. *Veterinary and Comparative Orthopaedics and Traumatology* 3: 130-133, 1988
7. Danielsson F, Sjostrom L: Surgical treatment of degenerative lumbosacral stenosis in dogs. *Veterinary Surgery* 28: 91-98, 1999
8. De Risio L, Sharp N, Olby NJ, *et al*: Predictors of outcome after dorsal decompressive laminectomy for degenerative lumbosacral stenosis in dogs: 69 cases (1987-1997). *Journal of the American Veterinary Medical Association* 219: 624-628, 2001
9. Flynn J, Rubert MJ, Olson E, *et al*: The effects of freezing or freeze-drying on the biomechanical properties of the canine intervertebral disc. *Spine* 15: 567-570, 1990
10. Gödde T, Steffen F: Surgical treatment of lumbosacral foraminal stenosis using a lateral approach in twenty dogs with degenerative lumbosacral stenosis. *Veterinary Surgery* 36: 705-713, 2007
11. Goel VK, Nishiyama K, Weinstein JN *et al*: Mechanical properties of lumbar spinal motion segments as affected by partial disc removal. *Spine* 11:1008-1012, 1986

12. Hill TP, Lubbe AM, Guthrie AJ: Lumbar spine stability following hemilaminectomy, pediculectomy, and fenestration. *Veterinary and Comparative Orthopaedics and Traumatology* 13: 165-171, 2000
13. Janssens LAA, Moens Y, Coppens P *et al*: Lumbosacral degenerative stenosis in the dog. *Veterinary and Comparative Orthopaedics and Traumatology 13: 97-103, 2000*
14. Kuroki H, Goel VK, Holekamp SA *et al*: Contributions of flexion – extension cycle loads to the lumbar spinal segment stability following discectomy procedures. *Spine* 29: 39-46, 2004
15. Linn LL, Bartels KE, Rochat MC, *et al*: Lumbosacral stenosis in 29 military working dogs: Epidemiologic findings and outcome after surgical intervention (1990-1999). *Veterinary Surgery* 32: 21-30, 2003
16. Macy NB, Les CM, Stover SM, *et al*: Effect of disk fenestration on sagittal kinematics of the canine C5-C6 intervertebral space. *Veterinary Surgery* 28: 171-179, 1999
17. Mayhew PD, Kapatkin AS, Wortmann JA, *et al*: Association of cauda equina compression on magnetic resonance images and clinical signs in dogs with degenerative lumbosacral stenosis. *Journal of the American Animal Hospital Association* 38: 555-562, 2002
18. Meij BP, Suwankong E, Van Der Veen AJ, *et al*: Biomechanical flexion-extension forces in normal canine lumbosacral cadaver specimens before and after dorsal laminectomy-discectomy and pedicle screw-rod fixation. *Veterinary Surgery* 36: 742-751, 2007
19. Moens NMM, Runyon CL: Fracture of L7 vertebral articular facets and pedicles following dorsal laminectomy in a dog. *Journal of the American Veterinary Medical Association* 221: 807-810, 2002
20. Panjabi MM, Krag MH, Chung TQ: Effects of disc injury on the mechanical behaviour of the human spine. *Spine* 9: 707-713, 1984
21. Panjabi MM: The stabilizing system of the spine. Part I. Function, dysfunction, adaptation and enhancement. *Journal of Spinal Disorders* 5: 383-389;1992
22. Panjabi MM: The stabilizing system of the spine. Part II. Function, dysfunction, adaptation and enhancement. *Journal of Spinal Disorders* 5: 390-396; 1992

23. Rohlman A, Graichen F, Weber U *et al*: Monitoring in vivo implant loads with a telemeterised internal spinal fixation device. *Spine* 25: 2981-2986, 2000
24. Schulz KS, Waldron DR, Grant JW, *et al*: Biomechanics of the thoracolumbar vertebral column of dogs during lateral bending. *American Journal of Veterinary Research* 57: 1228-1232, 1996
25. Shahar R, Banks-Sills L: Biomechanical analysis of the canine hind limb:calculation of forces during three legged stance. *Veterinary Journal* 3: 240-250, 2002
26. Sjöström L: Degenerative lumbosacral stenosis: Surgical decompression. In Slatter D (ed) *Textbook of Small Animal Surgery* (3rd edition). Saunders, Philadelphia: 1227-1237, 2003
27. Slocum B, Devine T: L7-S1 fixation-fusion for treatment of Cauda equina compression in the dog. *Journal of the American Veterinary Medical Association* 188: 31-35, 1986
28. Smith GK, Walter MC: Spinal decompressive procedures and dorsal compartment injuries: Comparative biomechanical study in canine cadavers. *American Journal of Veterinary Research* 49: 266-273, 1988
29. Smith MEH, Bebchuk TN, Shmon CL,*et al*: An in vitro biomechanical study of the effects of surgical modification upon the canine lumbosacral spine. *Veterinary and Comparative Orthopaedics and Traumatology* 17: 17-24, 2004
30. Solomonow M, Zhou BH, Harris M, *et al*: The ligamento-muscular stabilizing system of the spine. *Spine* 23: 2552-2562, 1988
31. Steffen F, Berger M, Morgan JP: Asymmetrical, transitional, lumbosacral vertebral segments in six dogs: A characteristic spinal syndrome. *Journal of the American Animal Hospital Association* 40: 338-344, 2004
32. Stubbs M, Harris M, Solomonow M *et al*: Ligamento-muscular protective reflex in the lumbar spine of the feline. *Journal of Electromyography and Kinesiology* 8: 197-204, 1998
33. Suwankong N, Meij BP, Voorhout G *et al*: Review and retrospective analysis of degenerative lumbosacral stenosis in 156 dogs treated by dorsal laminectomy. *Veterinary and Comparative Orthopaedics and Traumatology* 21: 285-293, 2008

34. Suwankong N, Voorhout G, Hazewinkel HAW *et al*: Agreement between computed tomography, magnetic resonance imaging, and surgical findings in dogs with degenerative lumbosacral stenosis. *Journal of the American Veterinary Medical Association* 12: 1924-1929, 2006
35. Van Klaren NJ, Suwankong E, De Boer S *et al*: Force plate analysis before and after dorsal decompression for treatment of degenerative lumbosacral stenosis in dogs. *Veterinary Surgery* 34: 450-456, 2005
36. Wood BC, Lanz OI, Jones JC, *et al*: Endoscopic-assisted lumbosacral foraminotomy in the dog. *Veterinary Surgery* 33: 221-231, 2004

Appendices

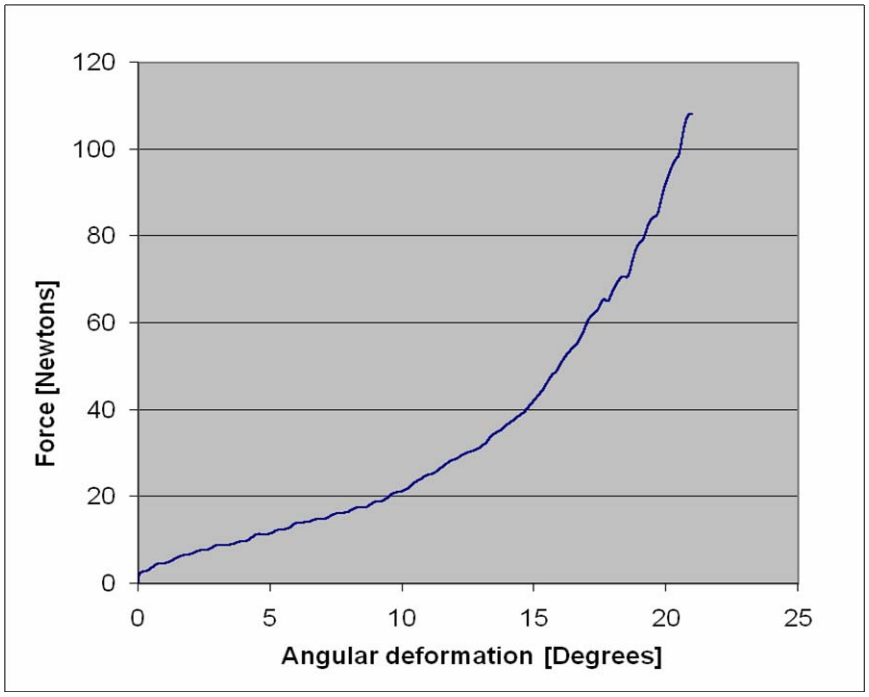

Figure A - 1: Load Deformation Curve – Specimen 5 (Group 1): Dorsiflexion Pre-modification.

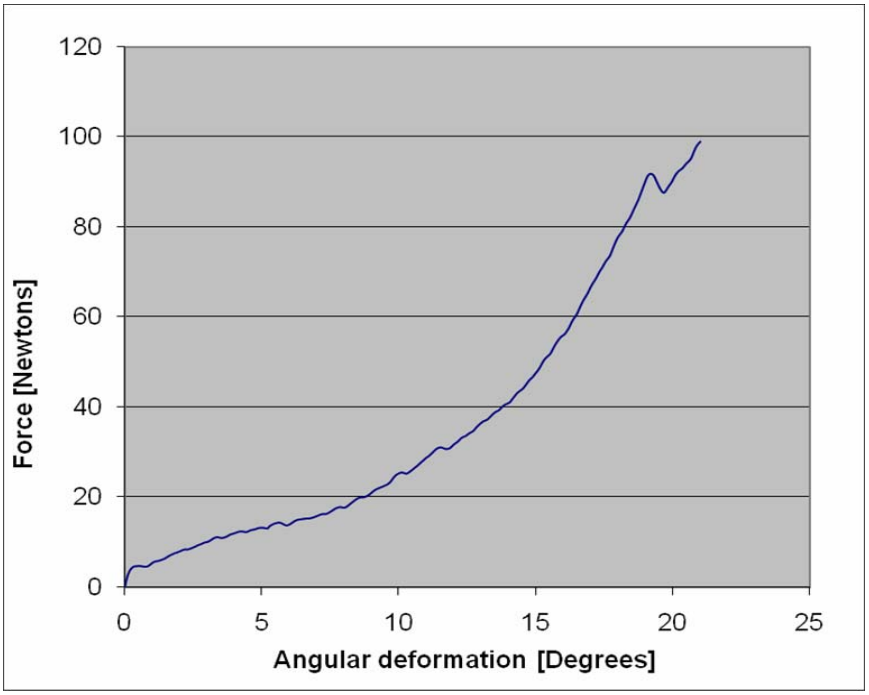

Figure A - 2: Load Deformation Curve – Specimen 5 (Group 1): Dorsiflexion Post-modification.

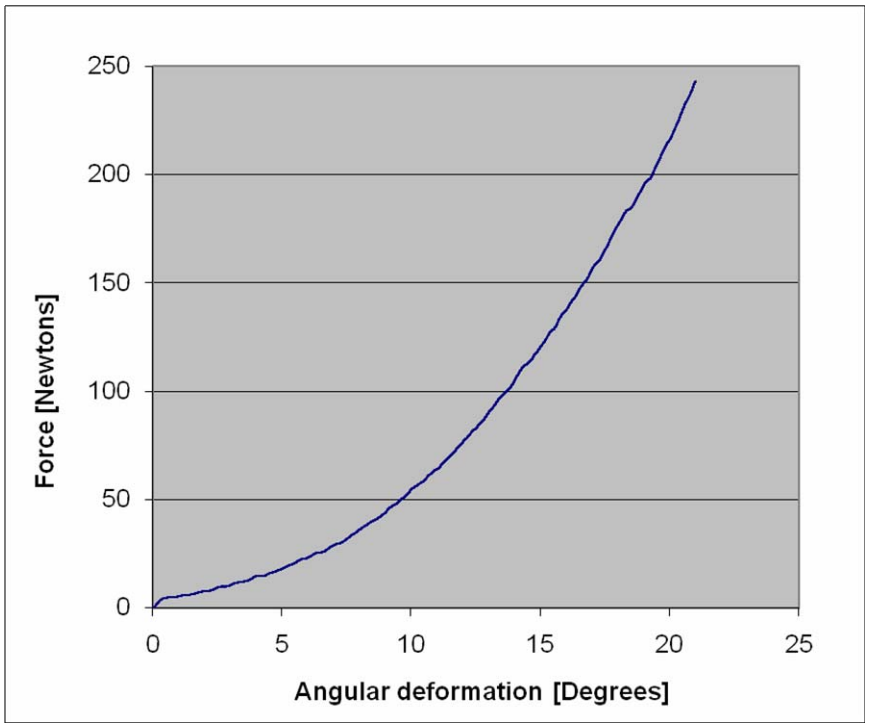

Figure A - 3: Load Deformation Curve – Specimen 5 (Group 1): Ventroflexion Pre-modification.

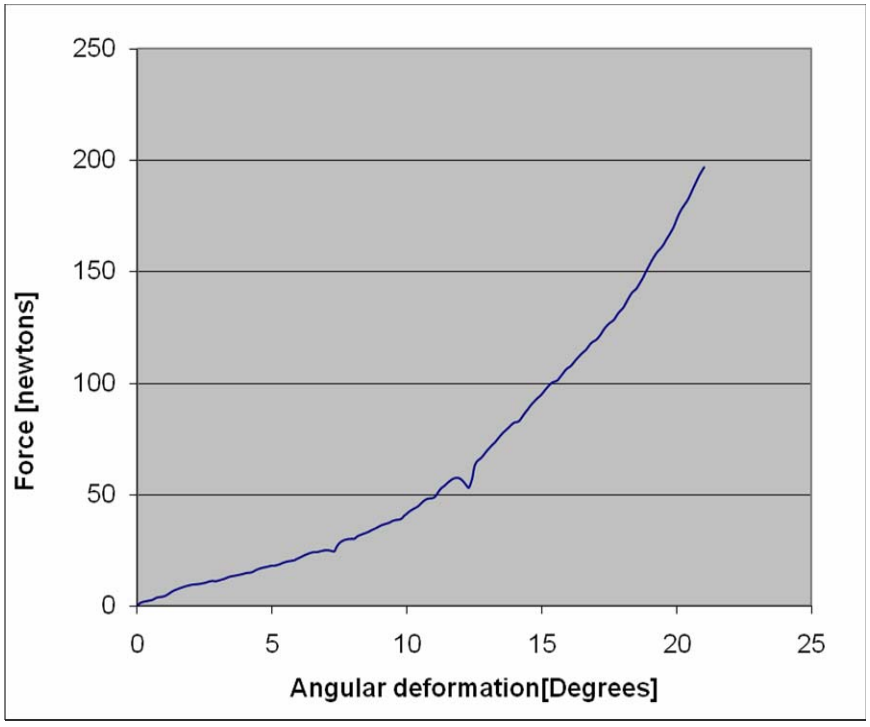

Figure A - 4: Load Deformation Curve – Specimen 5 (Group 1): Ventroflexion Post-modification.

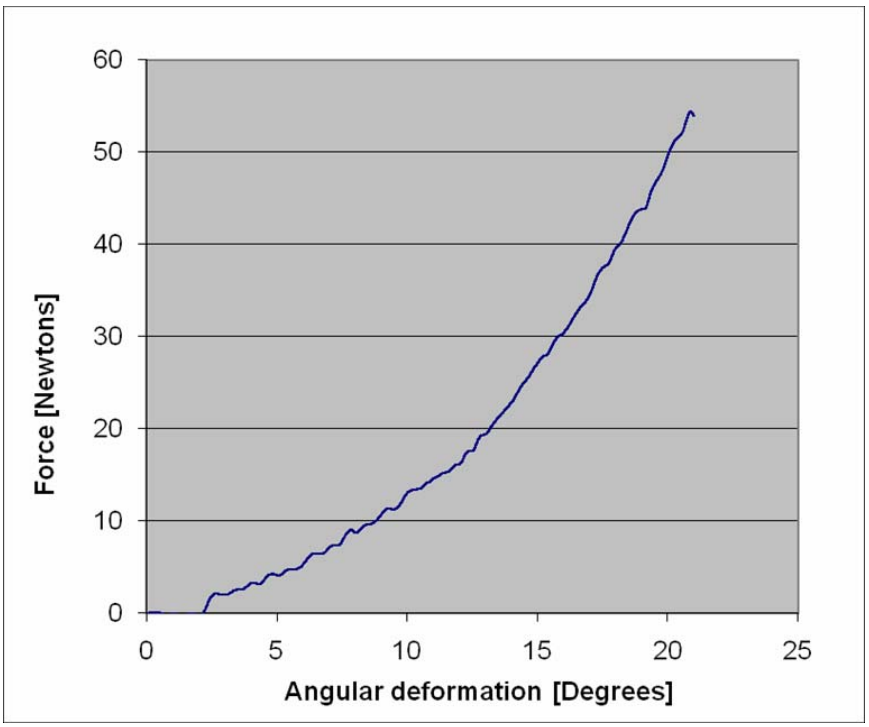

Figure A - 5: Load Deformation Curve – Specimen 10 (Group 1): Dorsiflexion Pre-modification.

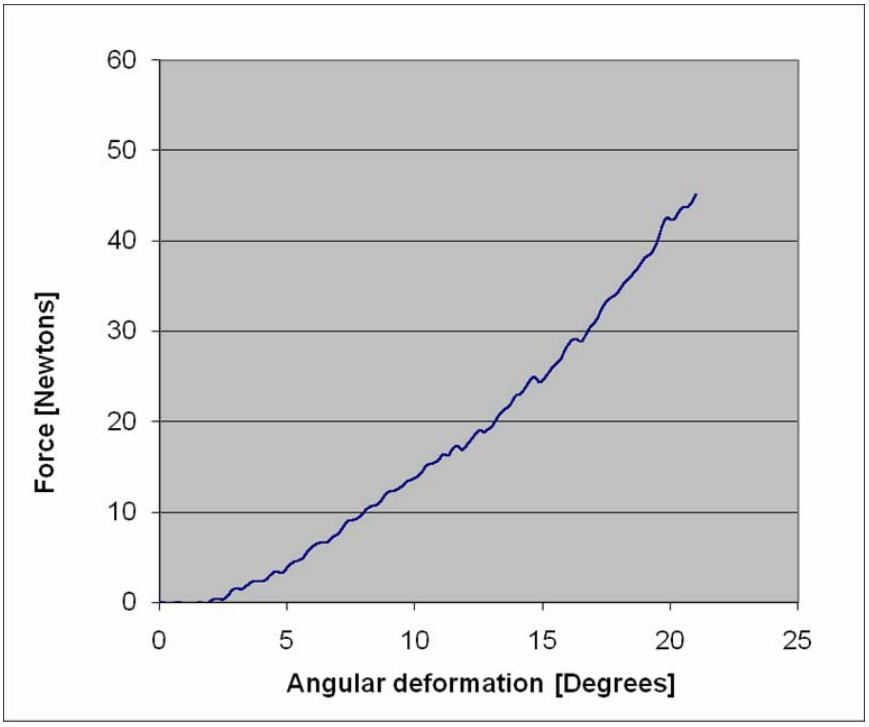

Figure A - 6: Load Deformation Curve – Specimen 10 (Group 1): Dorsiflexion Post-modification.

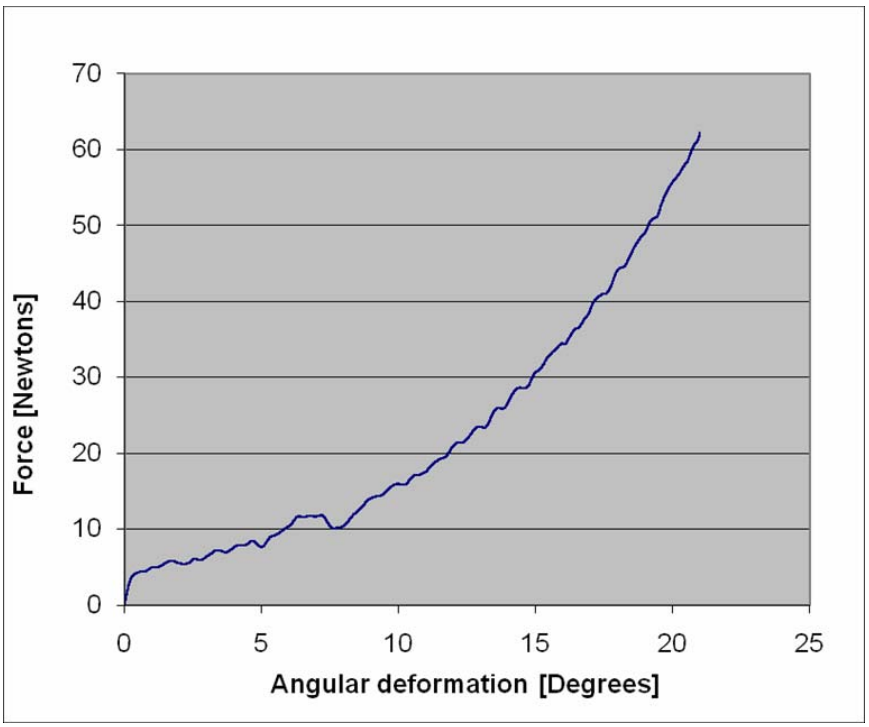

Figure A - 7: Load Deformation Curve – Specimen 10 (Group 1): Ventroflexion Pre-modification.

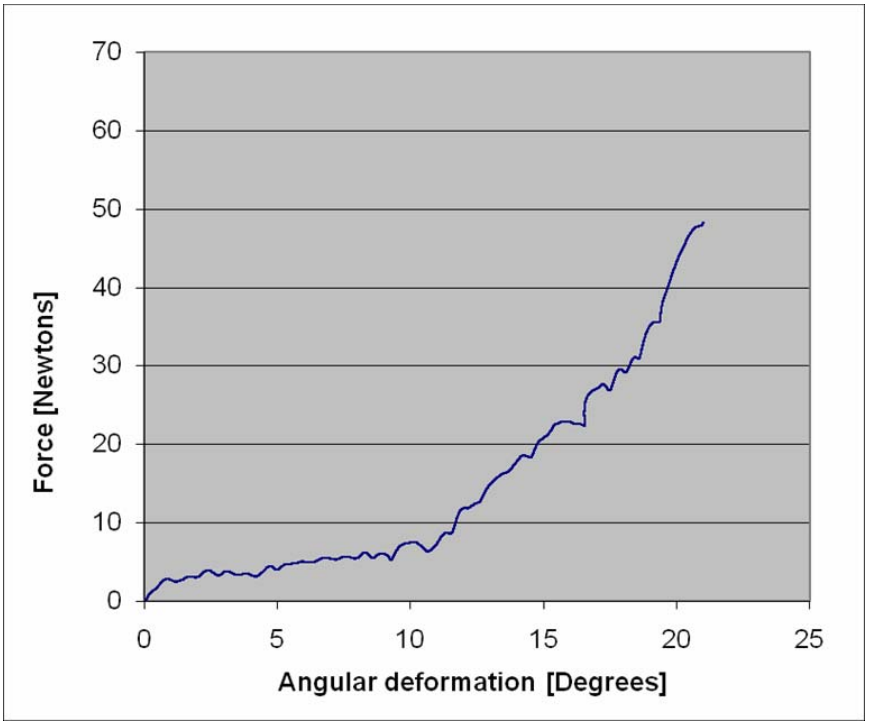

Figure A - 8: Load Deformation Curve – Specimen 10 (Group 1): Ventroflexion Post-modification.

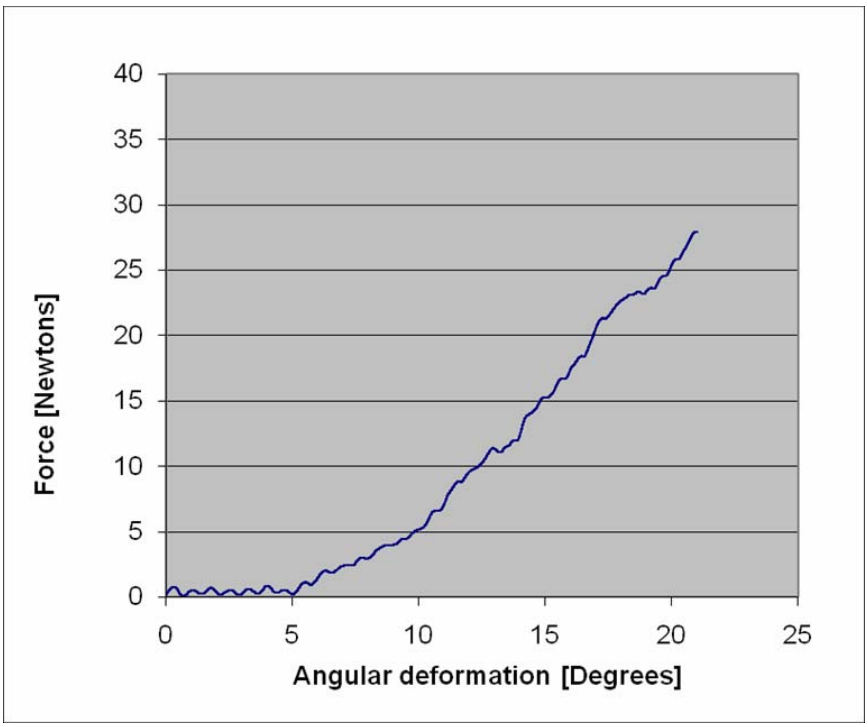

Figure A - 9: Load Deformation Curve – Specimen 12 (Group 1): Dorsiflexion Pre-modification.

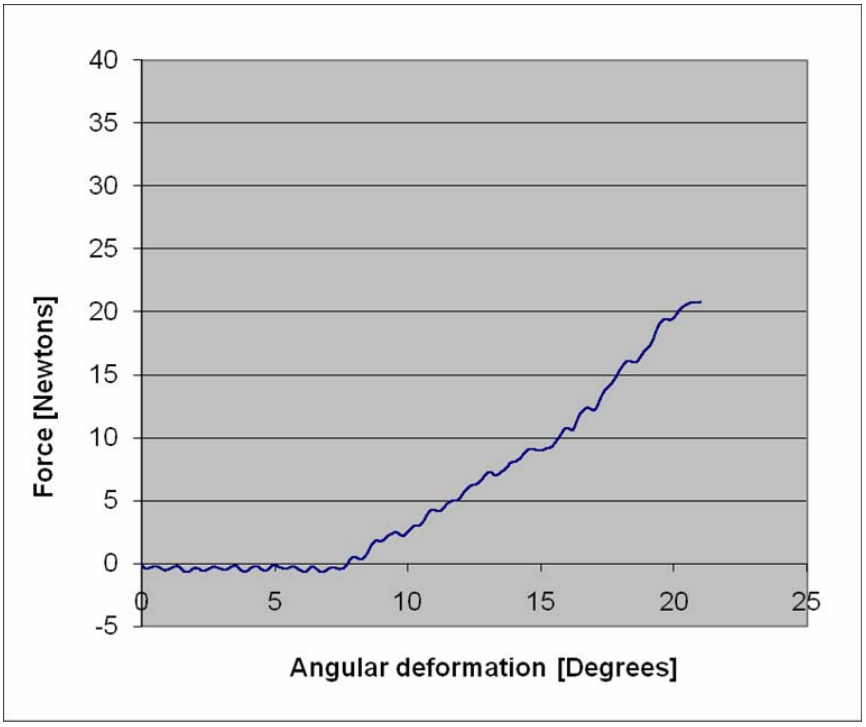

Figure A - 10: Load Deformation Curve – Specimen 12 (Group 1): Dorsiflexion Post-modification.

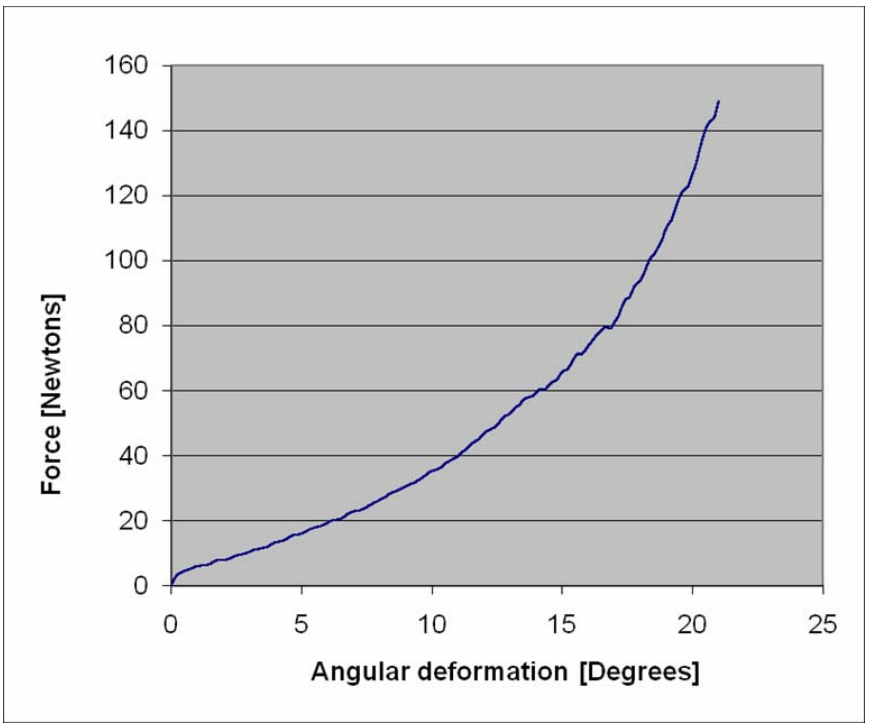

Figure A - 11: Load Deformation Curve – Specimen 12 (Group 1): Ventroflexion Pre-modification.

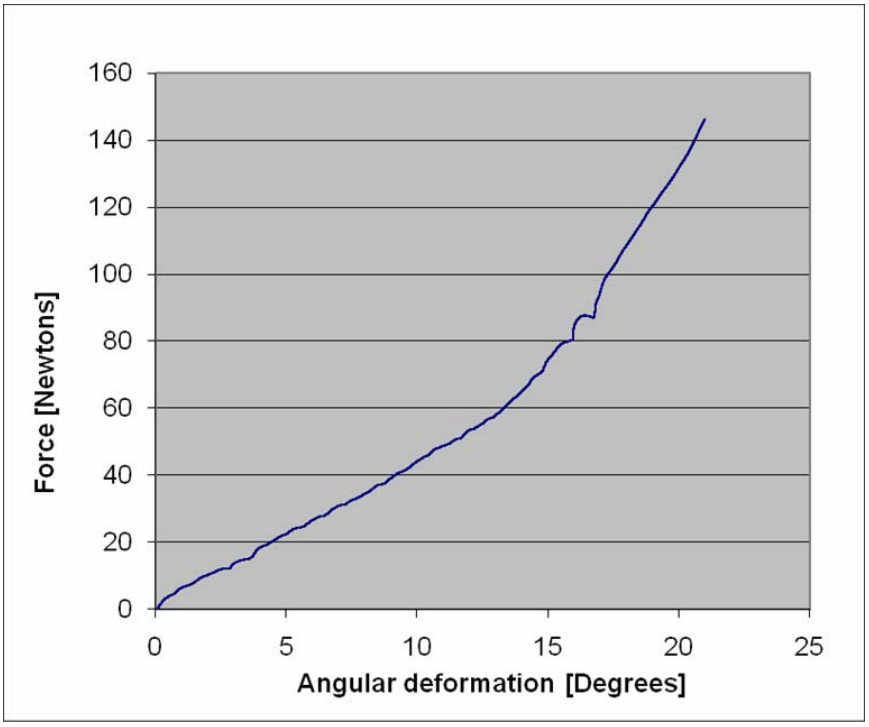

Figure A - 12: Load Deformation Curve – Specimen 12 (Group 1): Ventroflexion Post-modification.

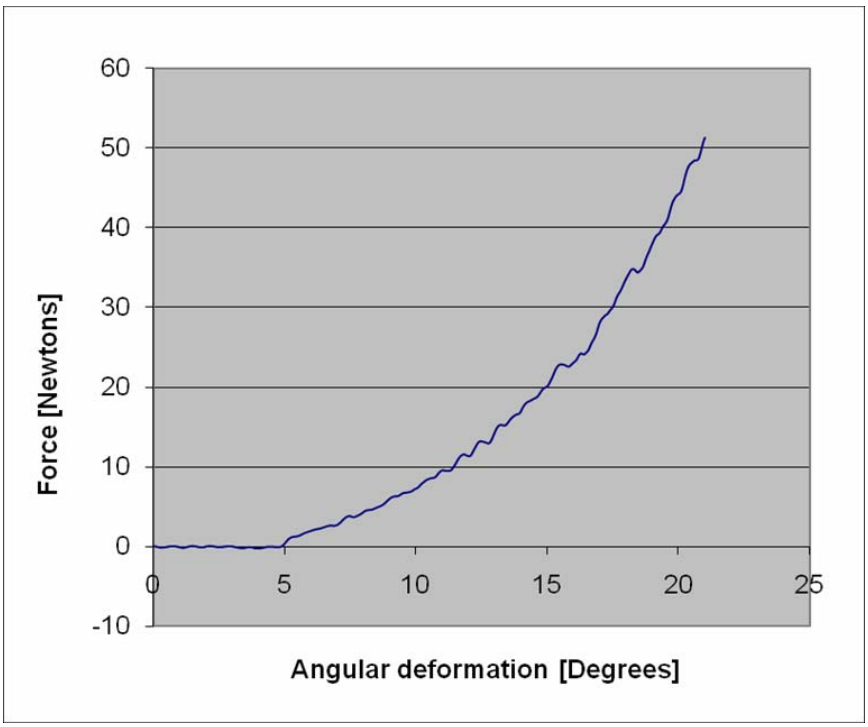

Figure A - 13: Load Deformation Curve – Specimen 13 (Group 1): Dorsiflexion Pre-modification.

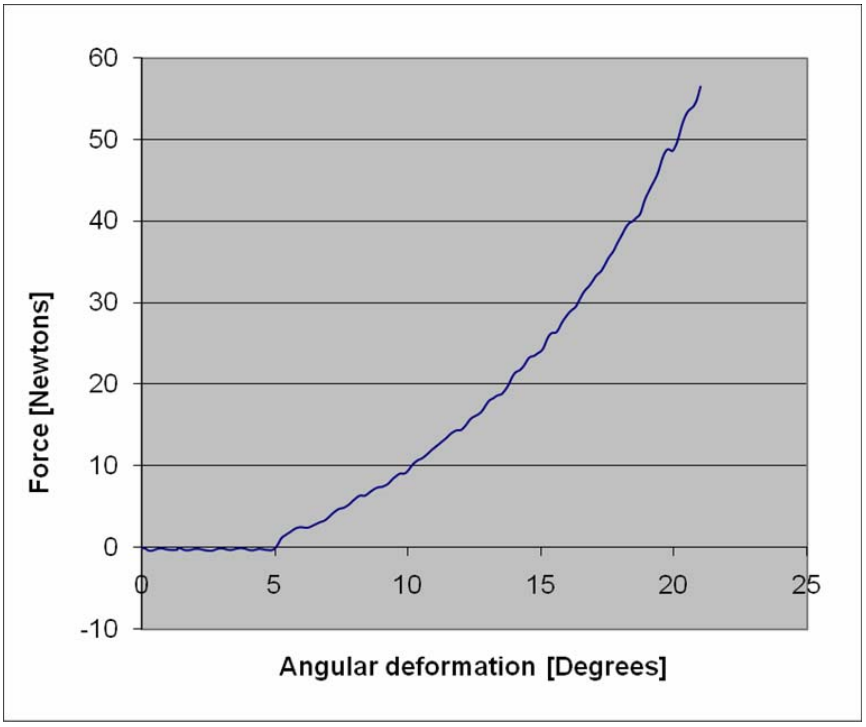

Figure A - 14: Load Deformation Curve – Specimen 13 (Group 1): Dorsiflexion Post-modification.

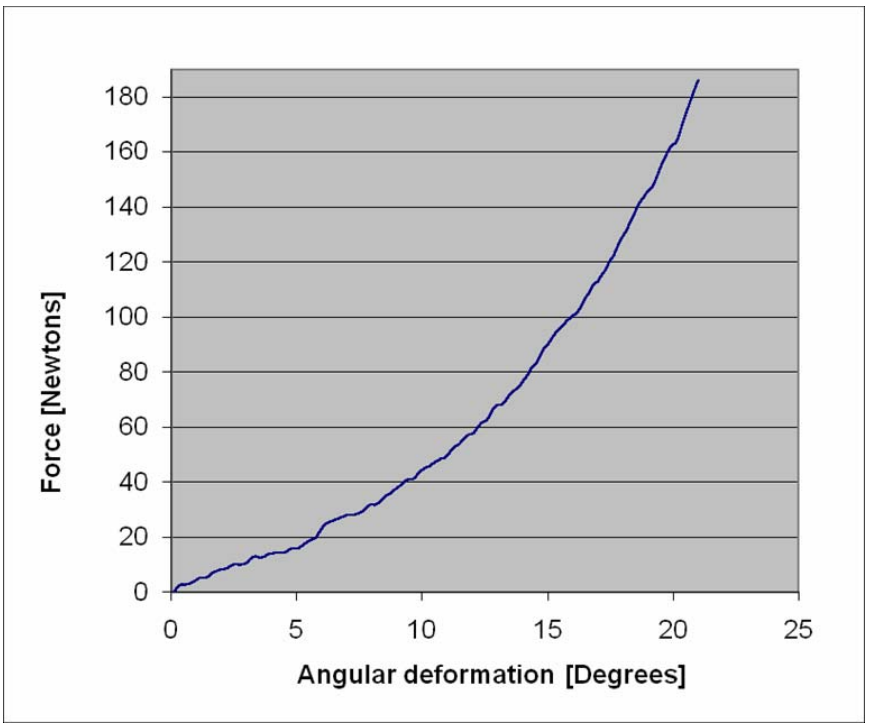

Figure A - 15: Load Deformation Curve – Specimen 13 (Group 1): Ventroflexion Pre-modification.

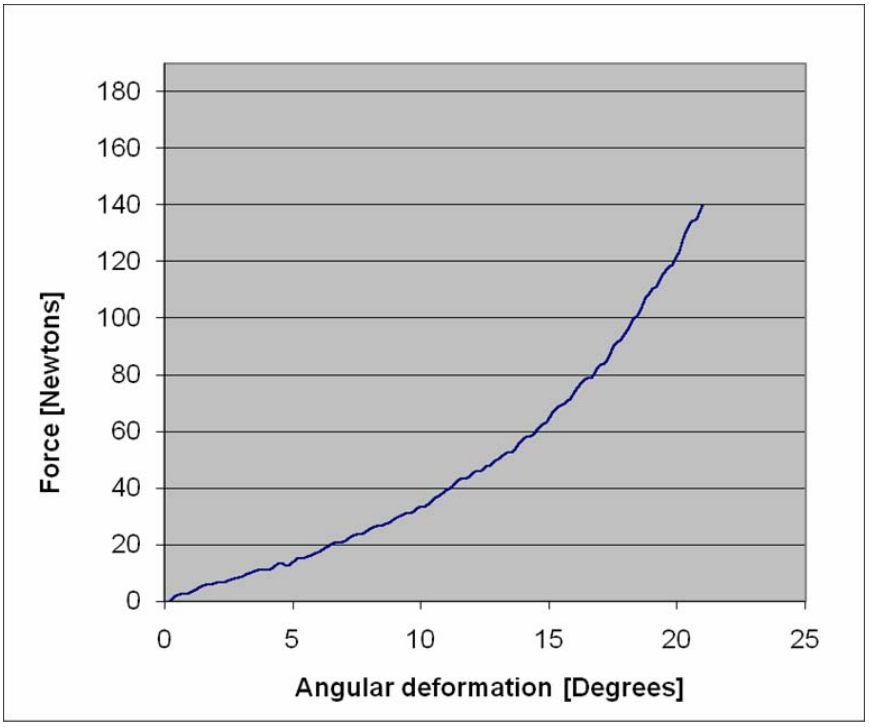

Figure A - 16: Load Deformation Curve – Specimen 13 (Group 1): Ventroflexion Post-modification.

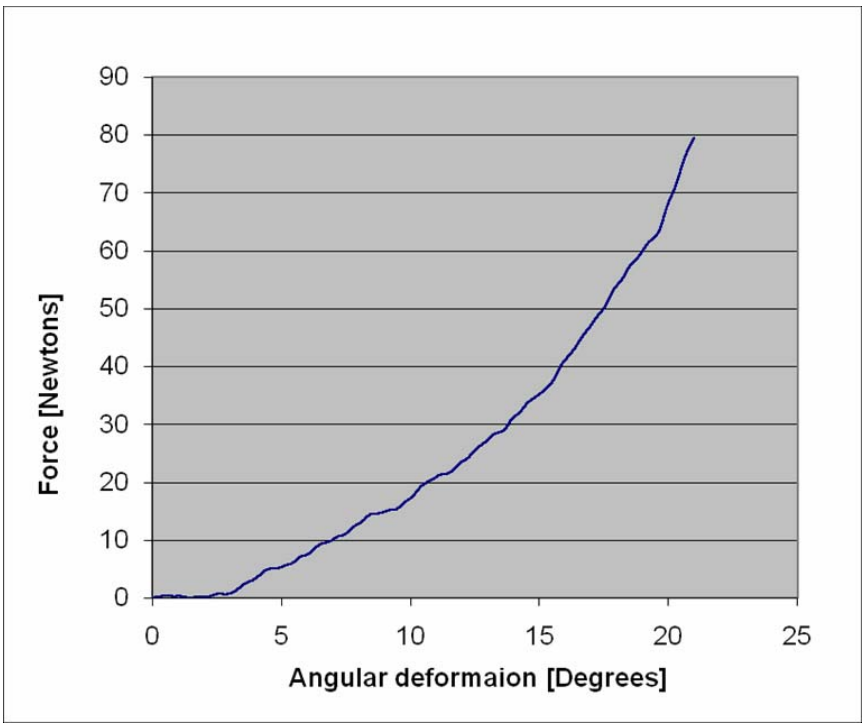

Figure A - 17: Load Deformation Curve – Specimen 16 (Group 1): Dorsiflexion Pre-modification.

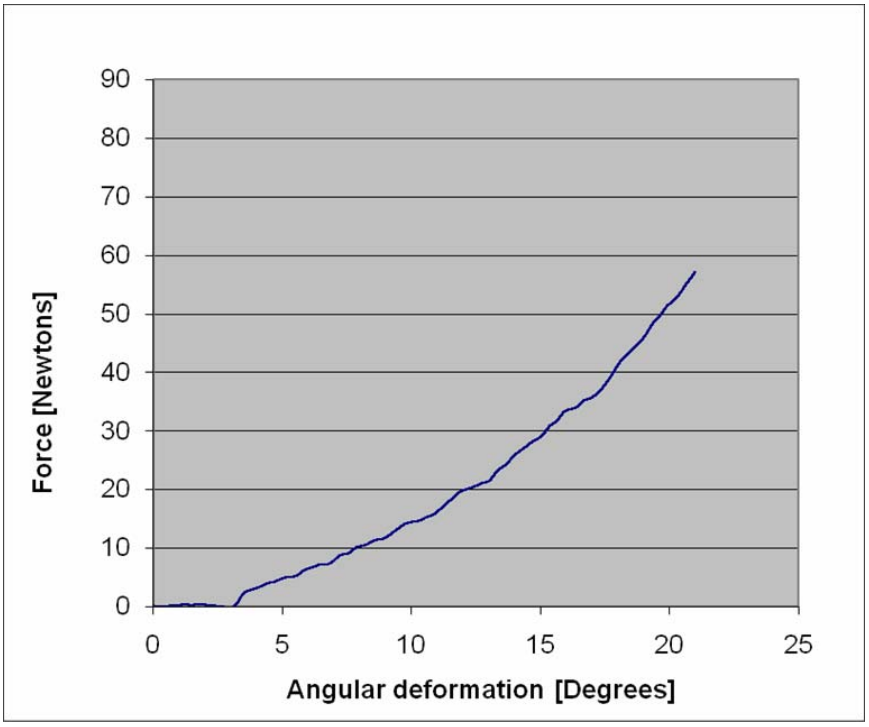

Figure A - 18: Load Deformation Curve – Specimen 16 (Group 1): Dorsiflexion Post-modification.

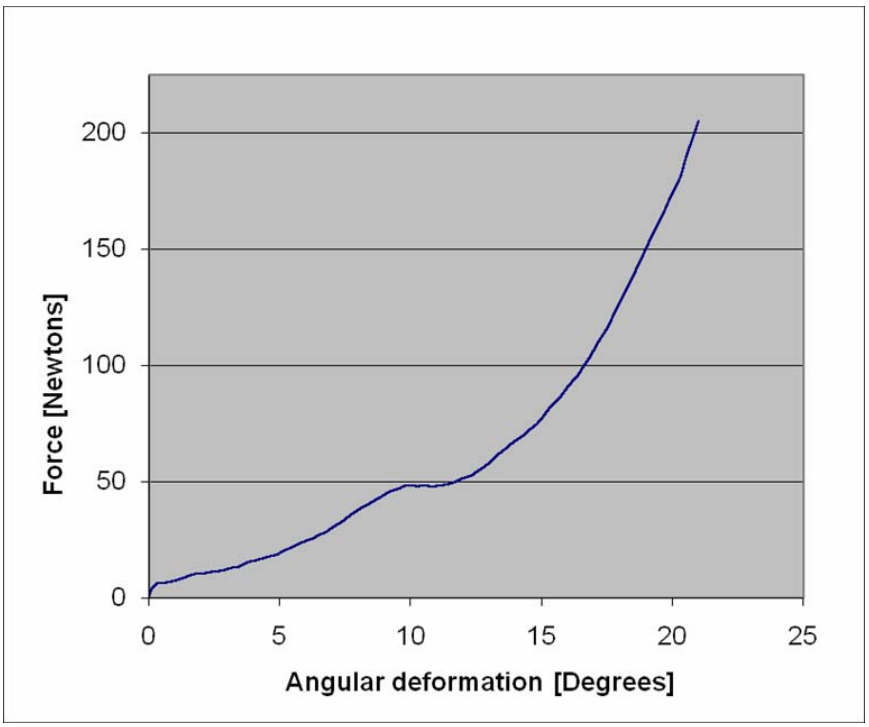

Figure A - 19: Load Deformation Curve – Specimen 16 (Group 1): Ventroflexion Pre-modification.

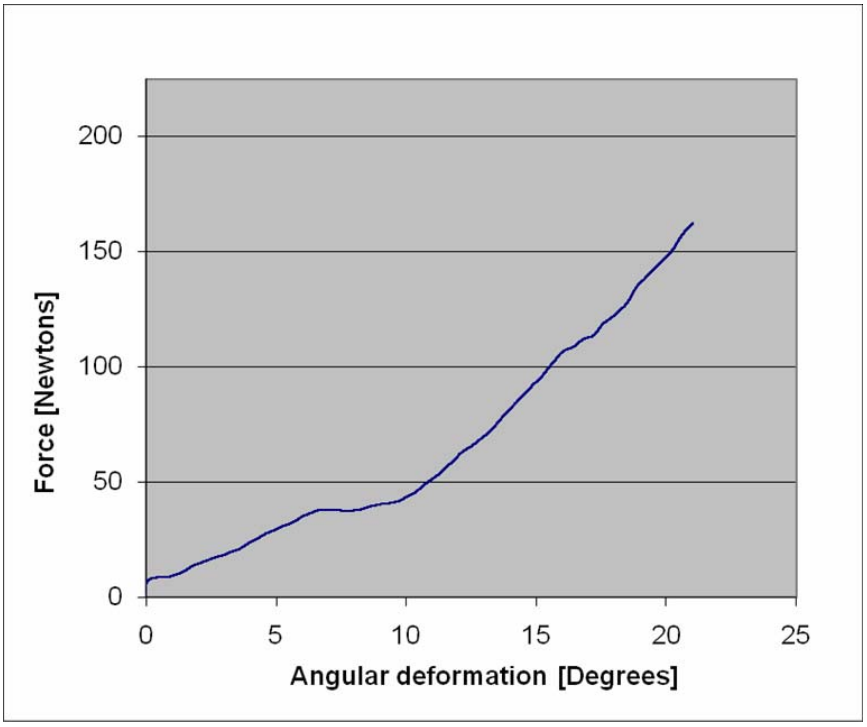

Figure A - 20: Load Deformation Curve – Specimen 16 (Group 1): Ventroflexion Post-modification.

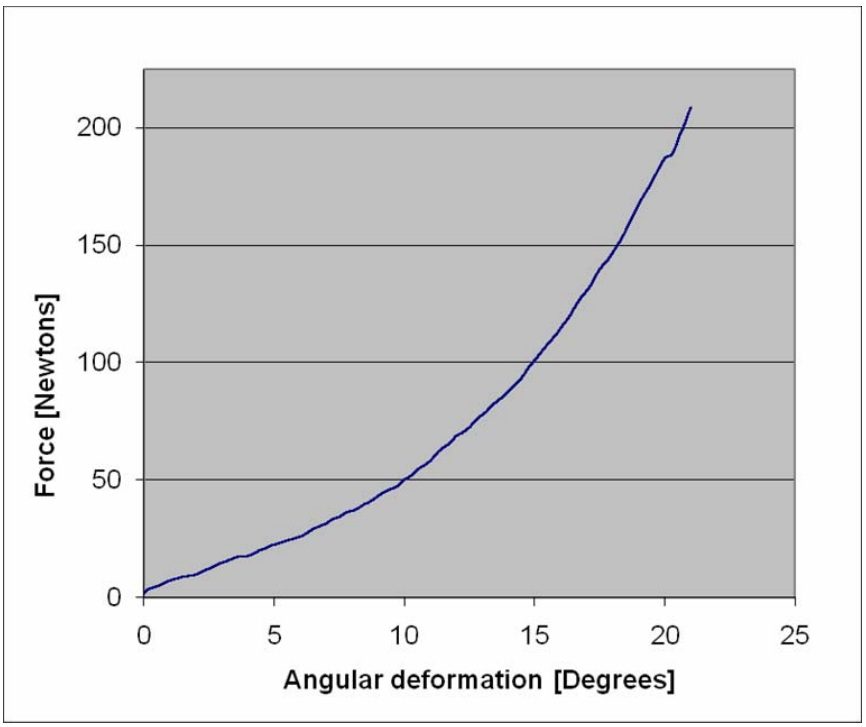

Figure A - 21: Load Deformation Curve – Specimen 6 (Group 2): Dorsiflexion Pre-modification.

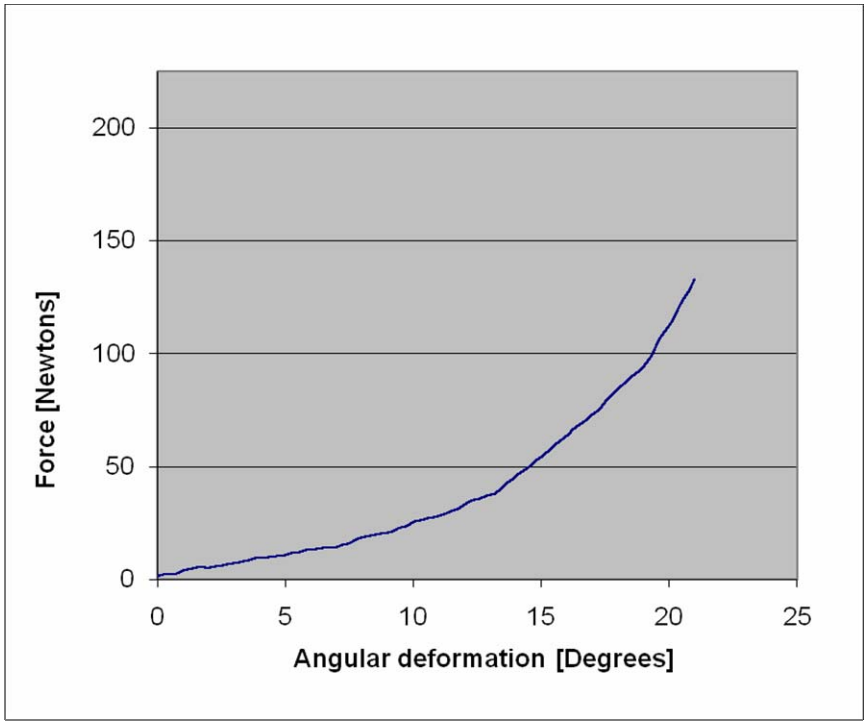

Figure A - 22: Load Deformation Curve – Specimen 6 (Group 2): Dorsiflexion Post-modification.

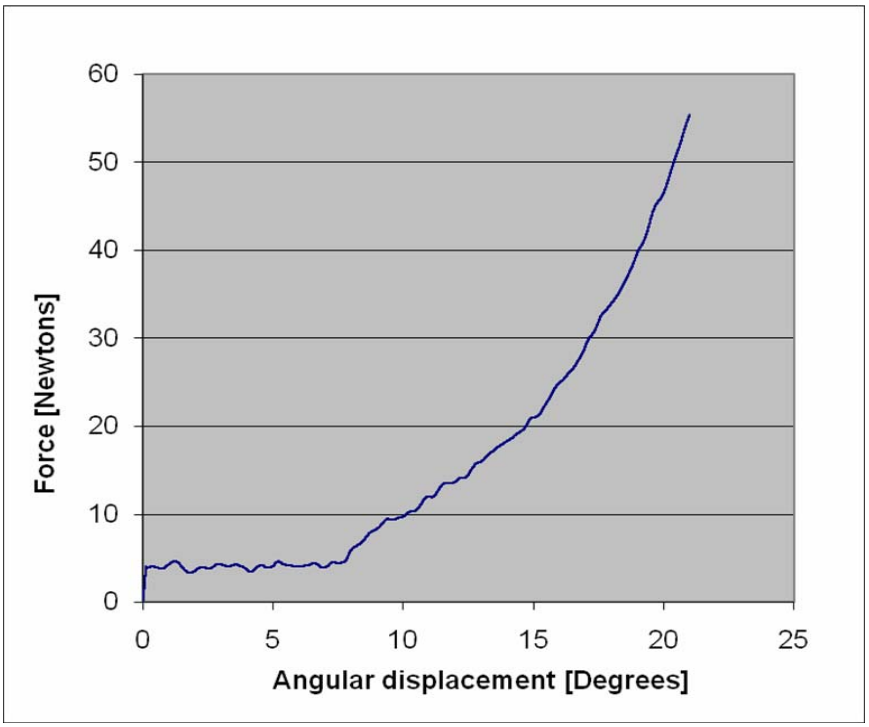

Figure A - 23: Load Deformation Curve – Specimen 6 (Group 2): Ventroflexion Pre-modification.

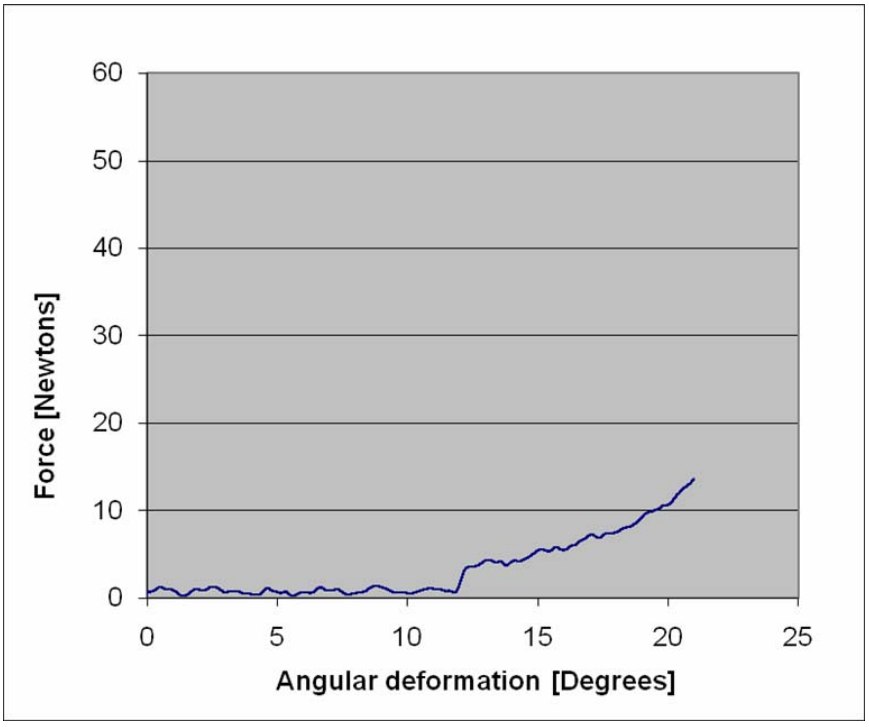

Figure A - 24: Load Deformation Curve – Specimen 6 (Group 2): Ventroflexion Post-modification.

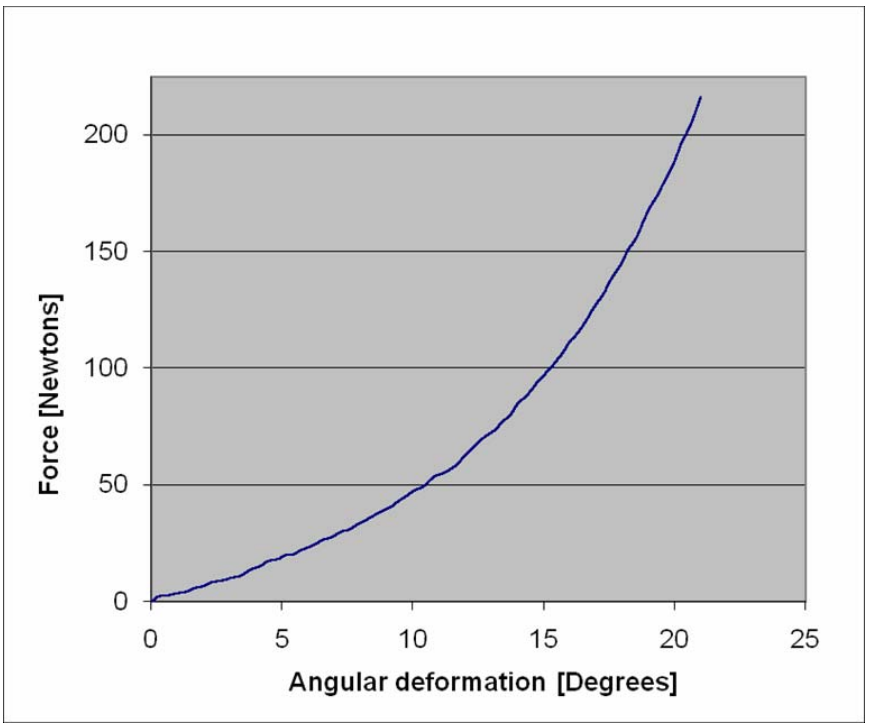

Figure A - 25: Load Deformation Curve – Specimen 7 (Group 2): Dorsiflexion Pre-modification.

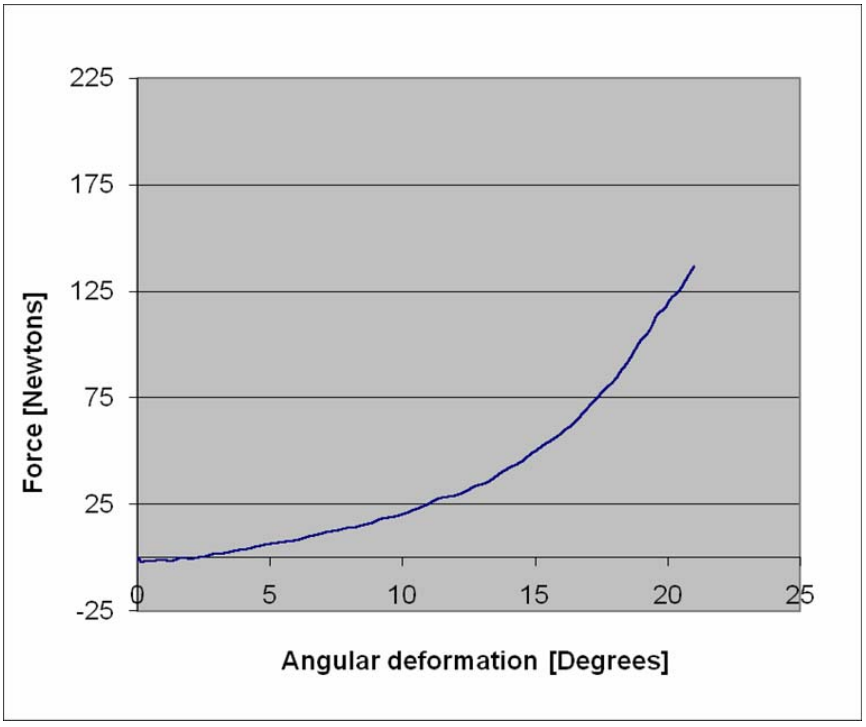

Figure A - 26: Load Deformation Curve – Specimen 7 (Group 2) : Dorsiflexion Post-modification.

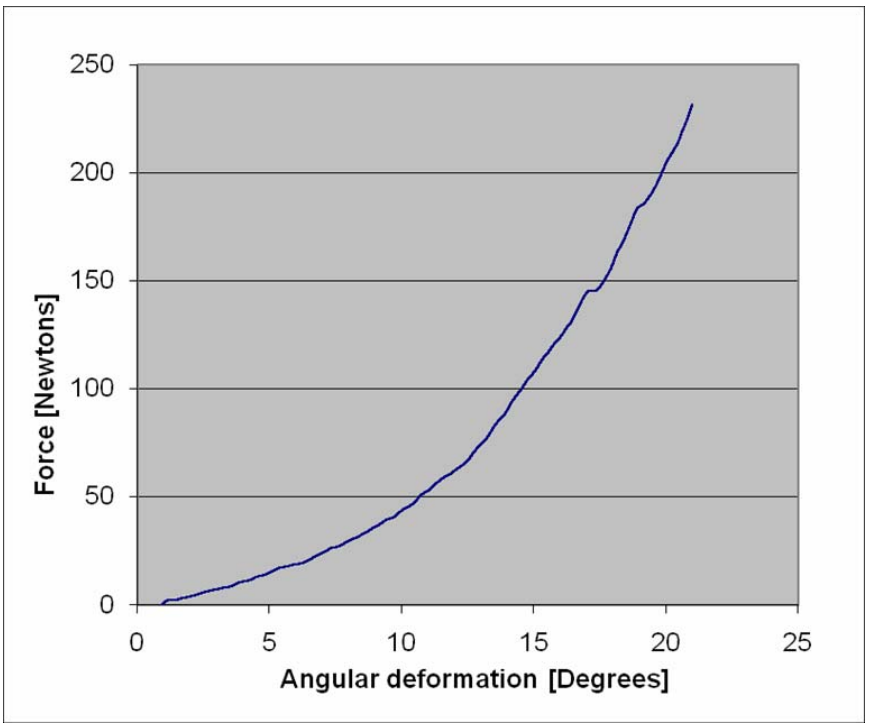

Figure A - 27: Load Deformation Curve – Specimen 7 (Group 2): Ventroflexion Pre-modification.

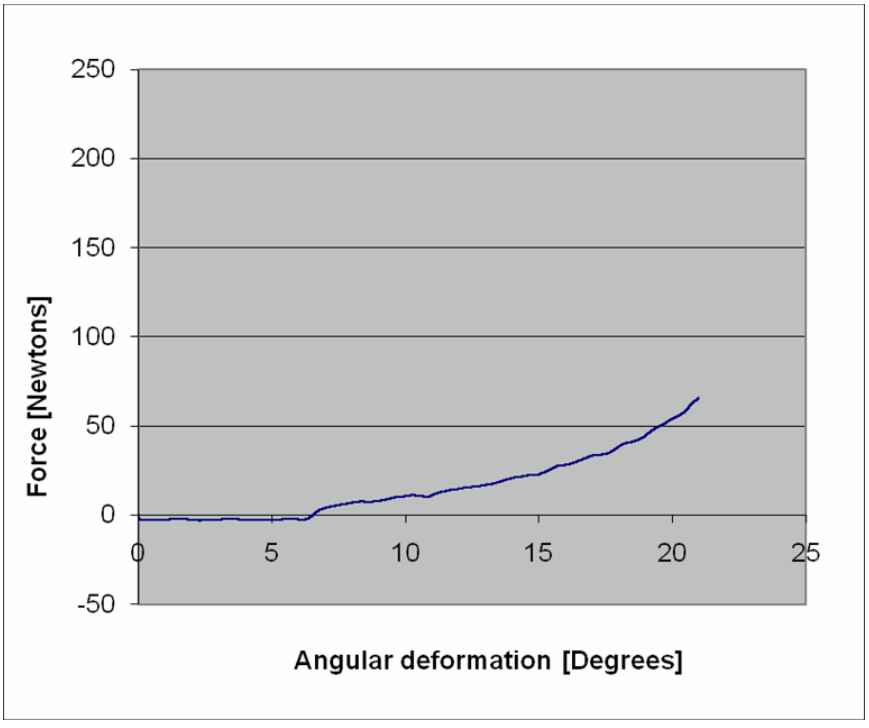

Figure A - 28: Load Deformation Curve – Specimen 7 (Group 2): Ventroflexion Post-modification.

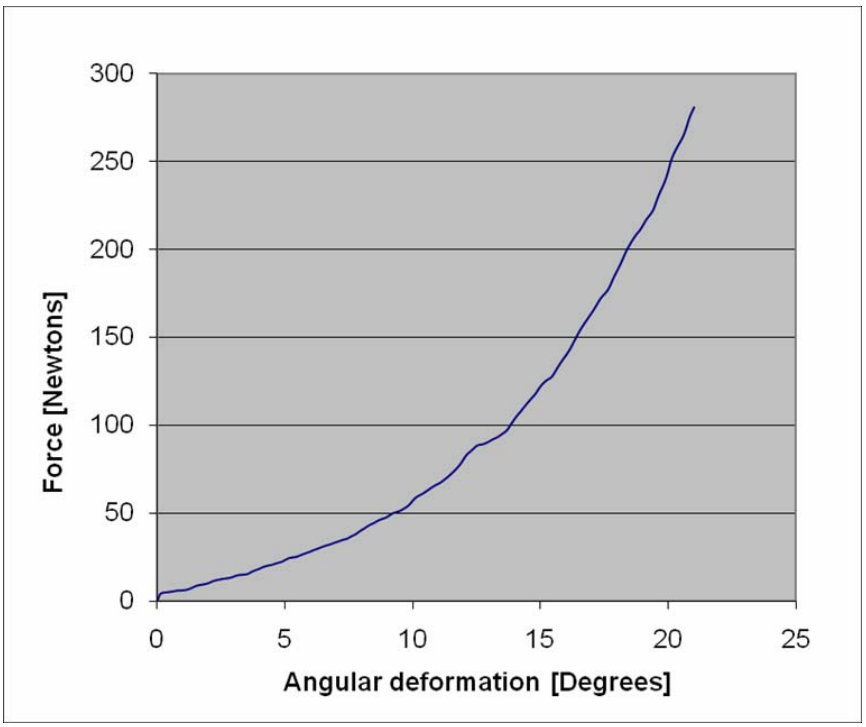

Figure A - 29: Load Deformation Curve – Specimen 15 (Group 2): Dorsiflexion Pre-modification.

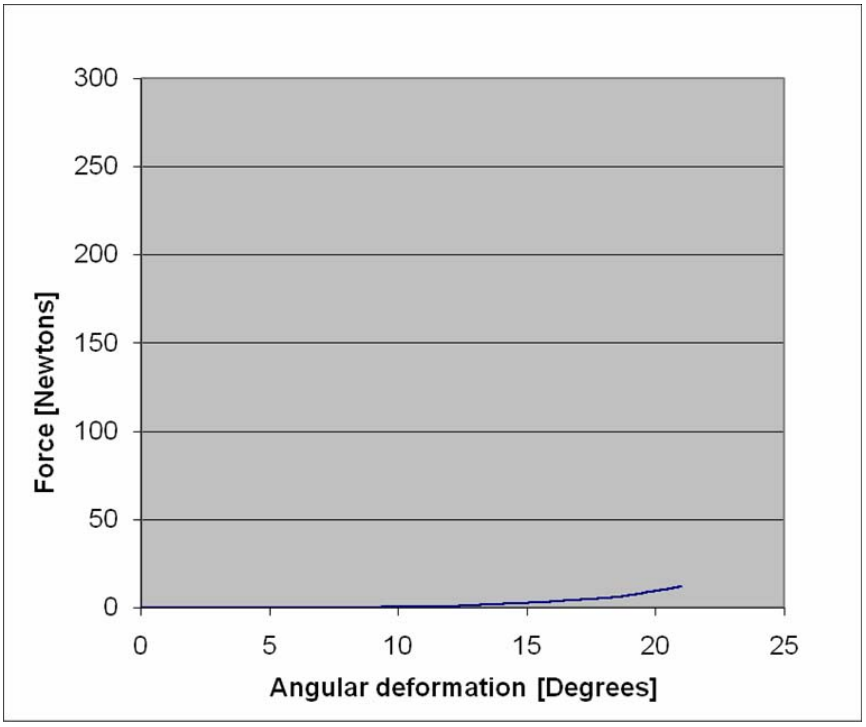

Figure A - 30: Load Deformation Curve – Specimen 15 (Group 2): Dorsiflexion Post-modification.

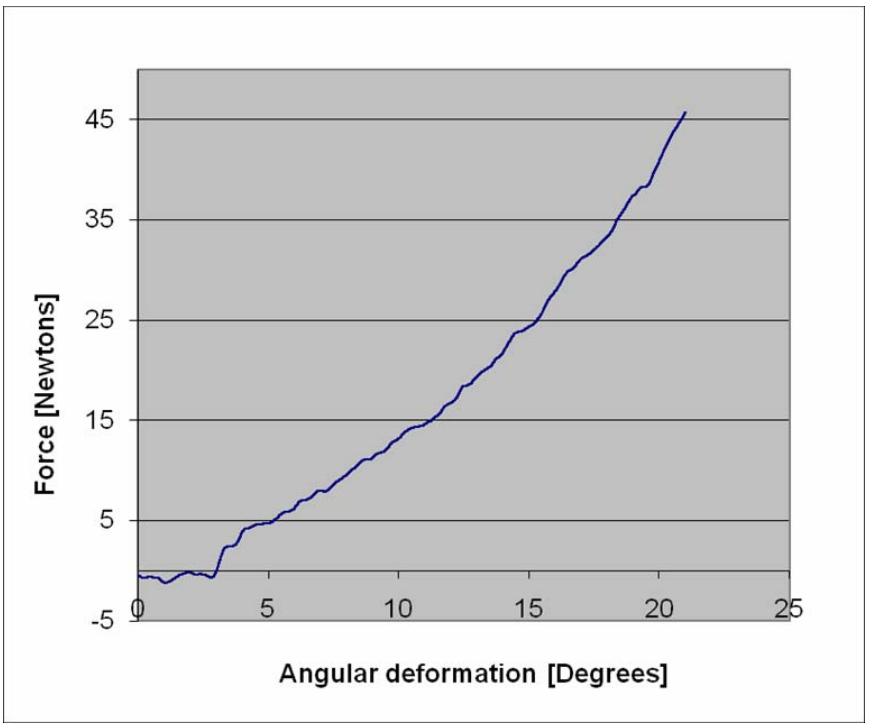

Figure A - 31: Load Deformation Curve – Specimen 15 (Group 2): Ventroflexion Pre-modification.

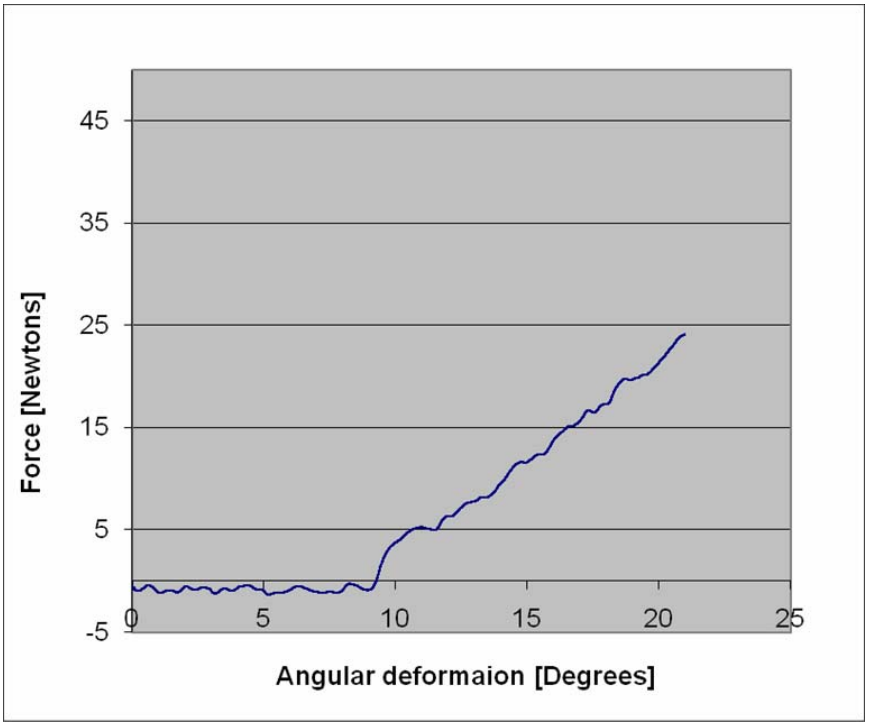

Figure A - 32: Load Deformation Curve – Specimen 15 (Group 2): Ventroflexion Post-modification.

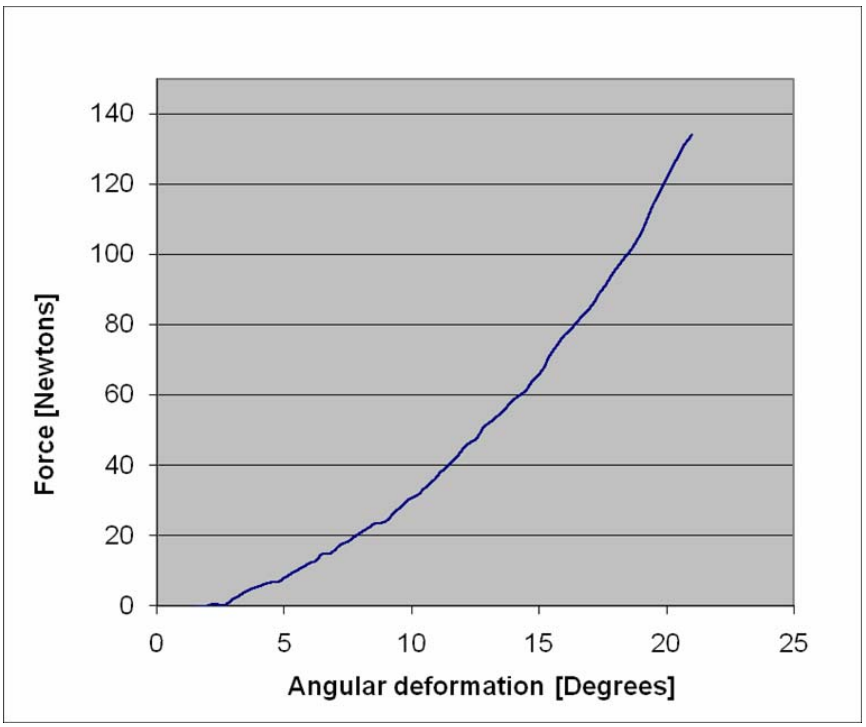

Figure A - 33: Load Deformation Curve – Specimen 17 (Group 2): Dorsiflexion Pre-modification.

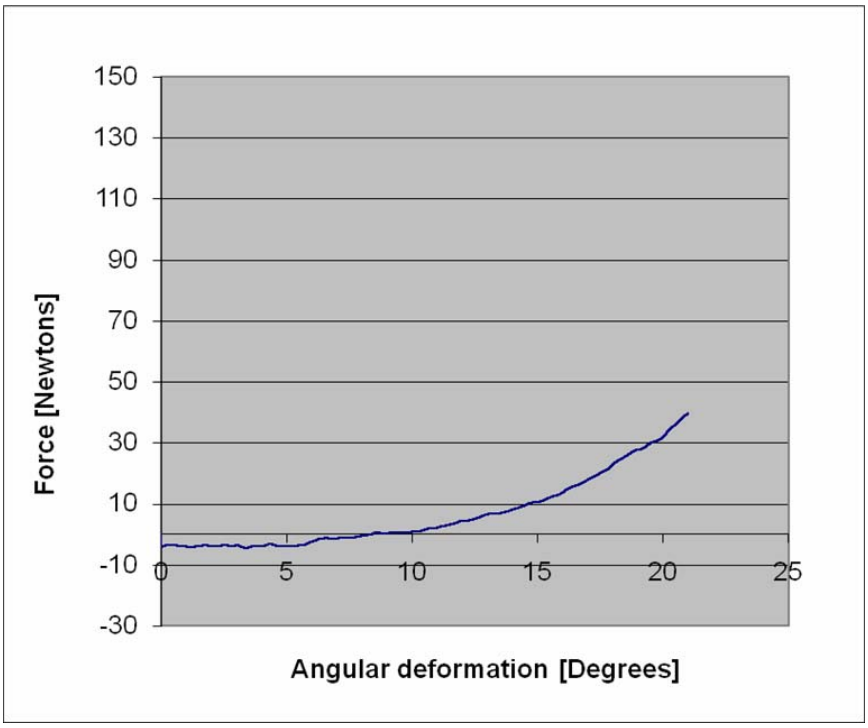

Figure A - 34: Load Deformation Curve – Specimen 17 (Group 2): Dorsiflexion Post-modification.

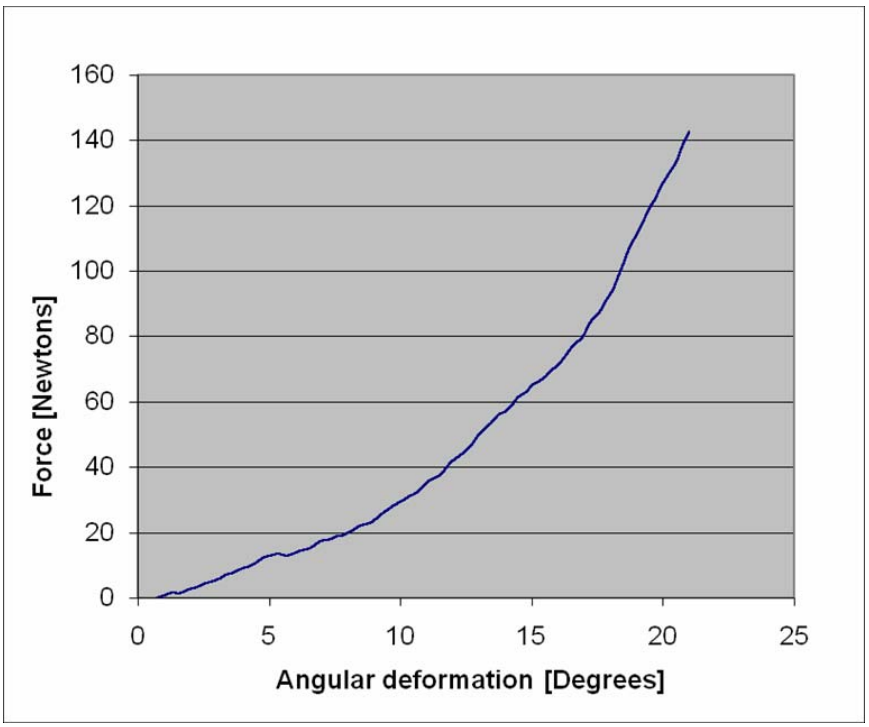

Figure A - 35: Load Deformation Curve – Specimen 17 (Group 2): Ventroflexion Pre-modification.

.

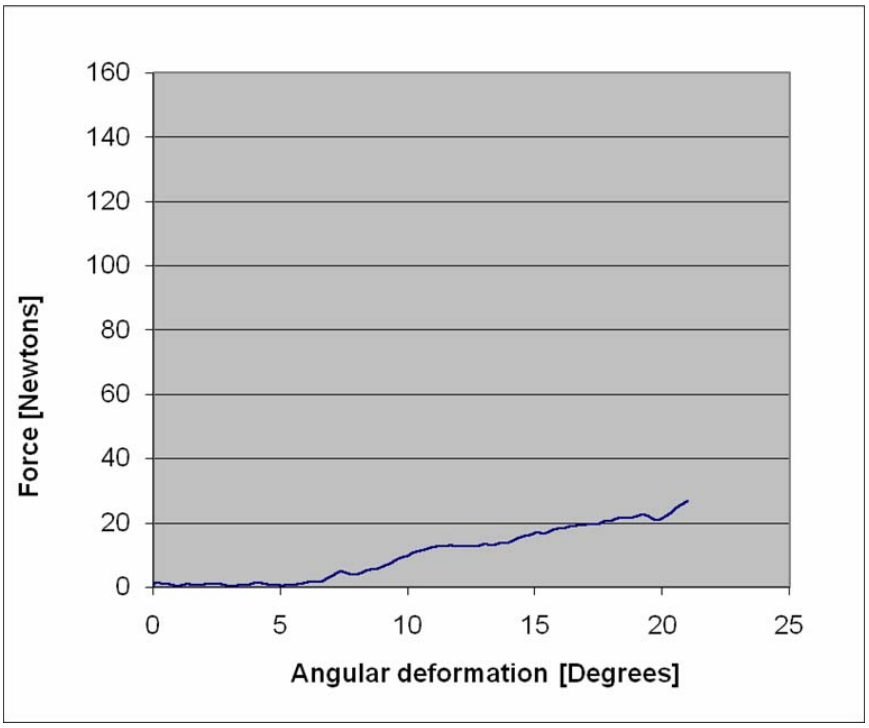

Figure A - 36: Load Deformation Curve – Specimen 17 (Group 2): Ventroflexion Post-modification.

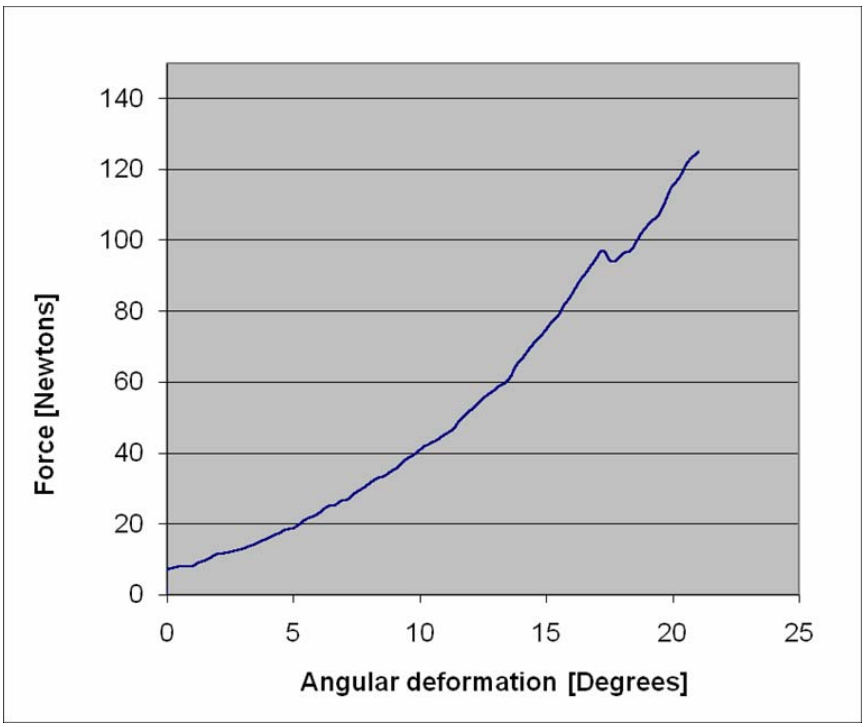

Figure A - 37: Load Deformation Curve – Specimen 19 (Group 2): Dorsiflexion Pre-modification.

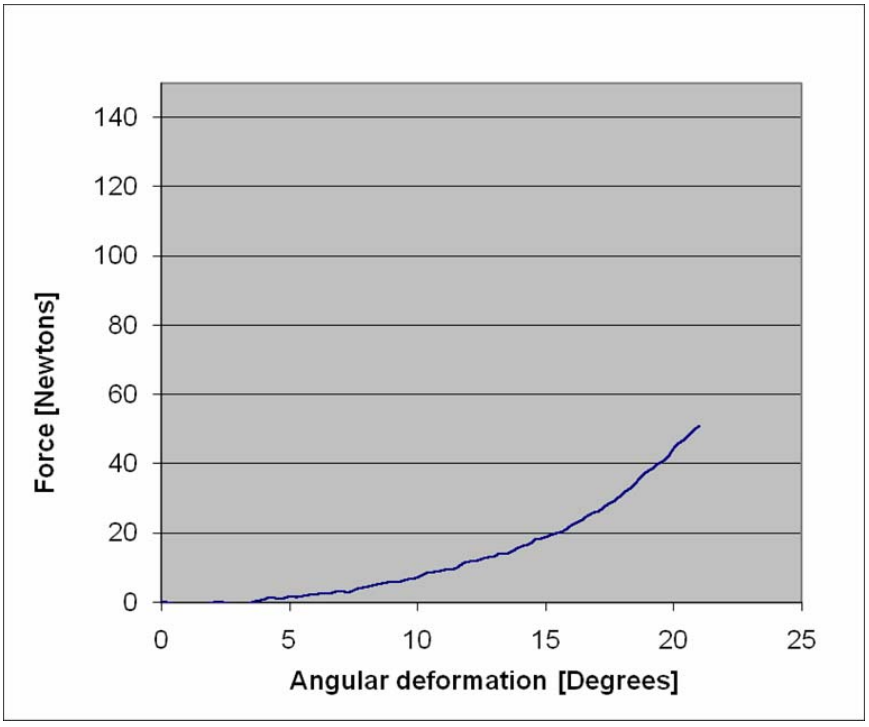

Figure A - 38: Load Deformation Curve – Specimen 19 (Group 2): Dorsiflexion Post-modification.

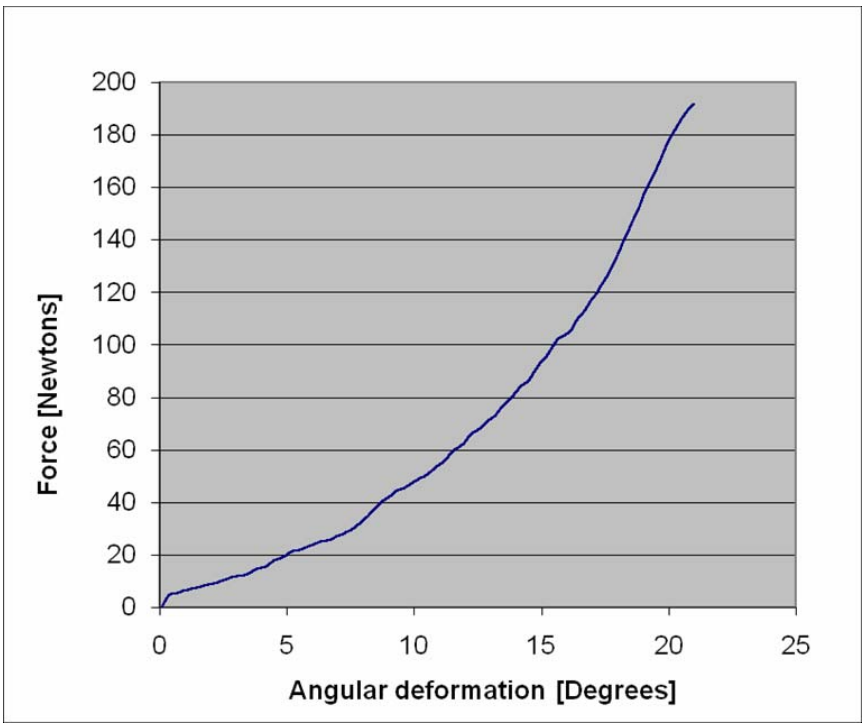

Figure A - 39: Load Deformation Curve – Specimen 19 (Group 2): Ventroflexion Pre-modification.

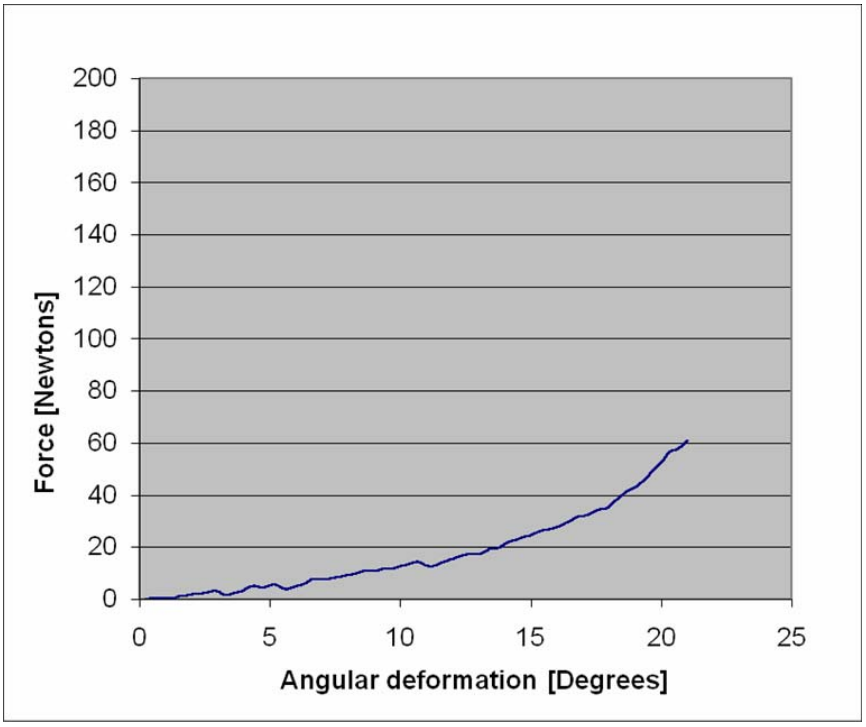

Figure A - 40: Load Deformation Curve – Specimen 19 (Group 2): Ventroflexion Post-modification.

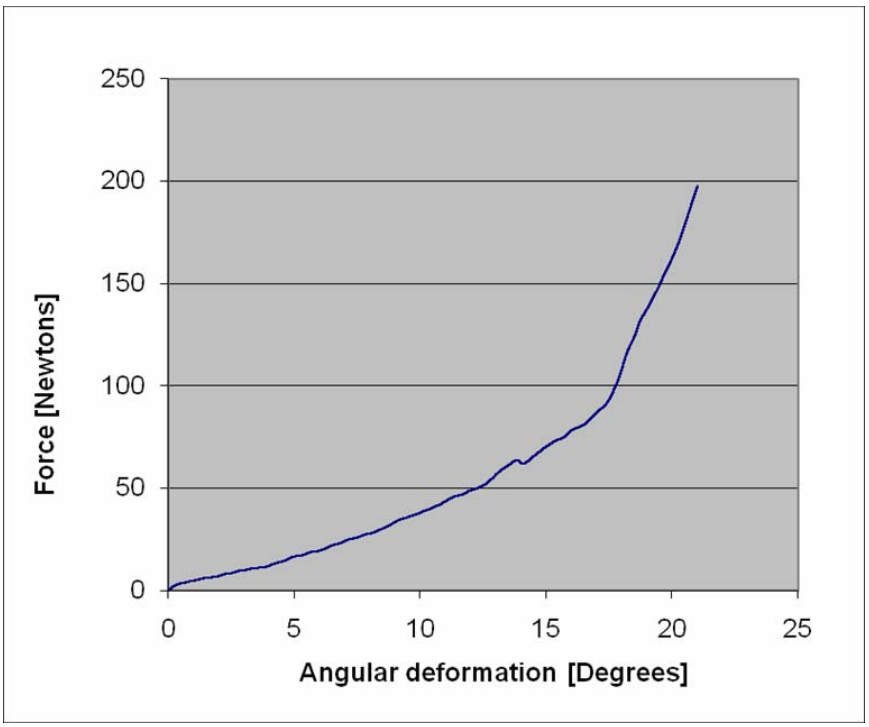

Figure A - 41: Load Deformation Curve – Specimen 20 (Group 2): Dorsiflexion Pre-modification.

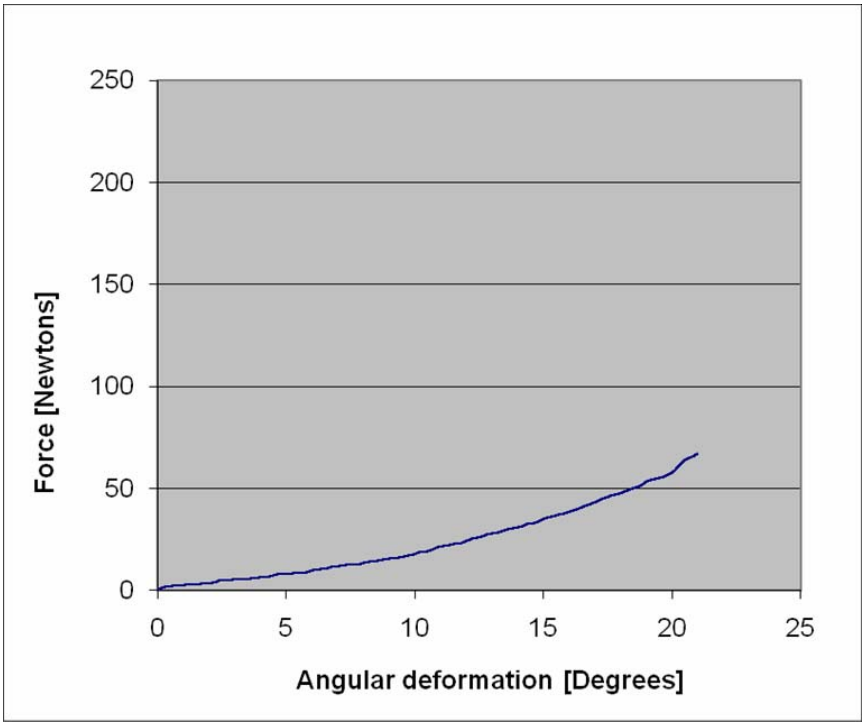

Figure A - 42: Load Deformation Curve – Specimen 20 (Group 2): Dorsiflexion Post-modification.

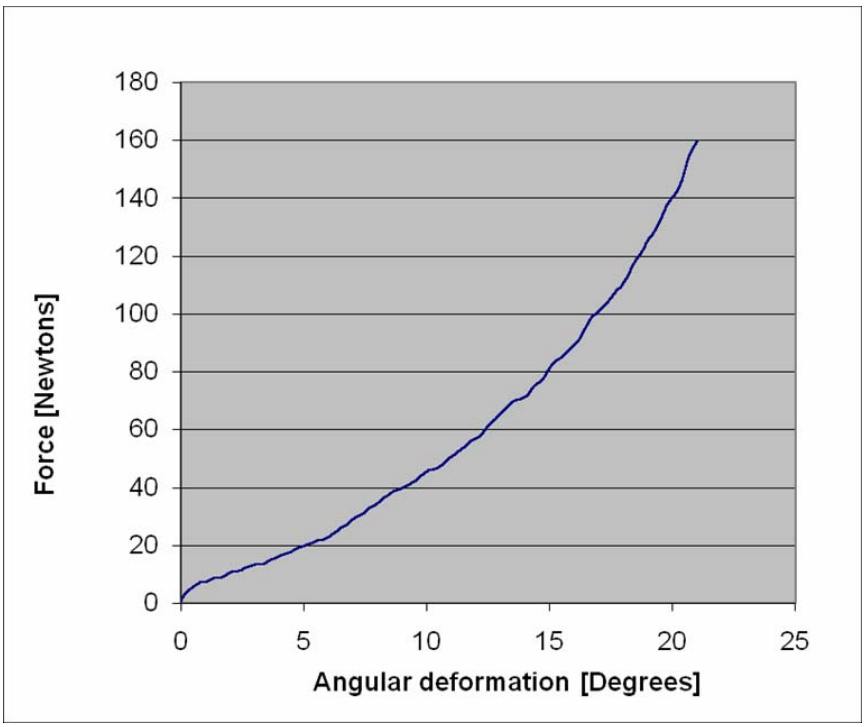

Figure A - 43: Load Deformation Curve – Specimen 20 (Group 2): Ventroflexion Pre-modification.

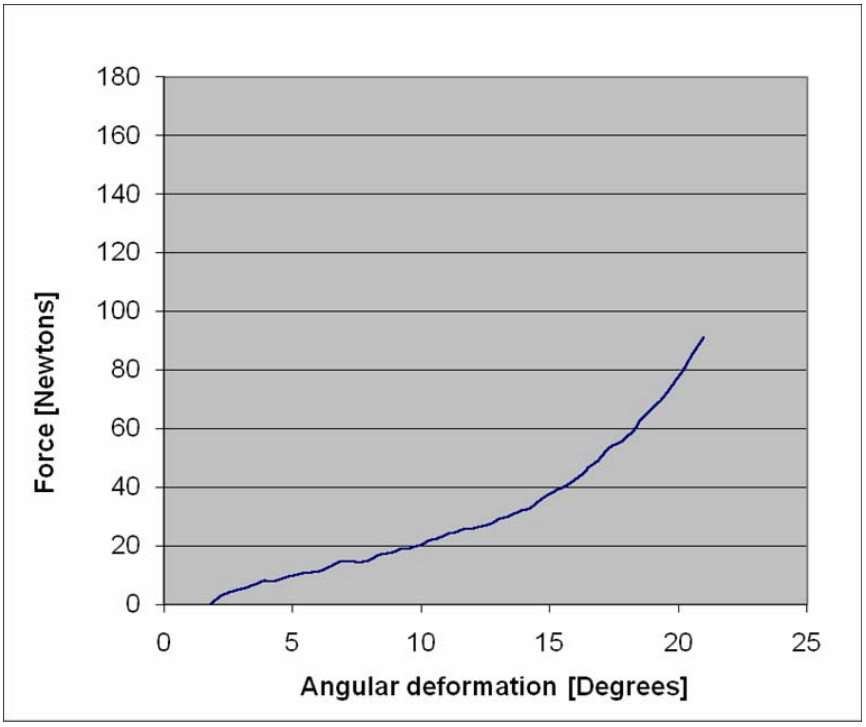

Figure A - 44: Load Deformation Curve – Specimen 20 (Group 2): Ventroflexion Post-modification.

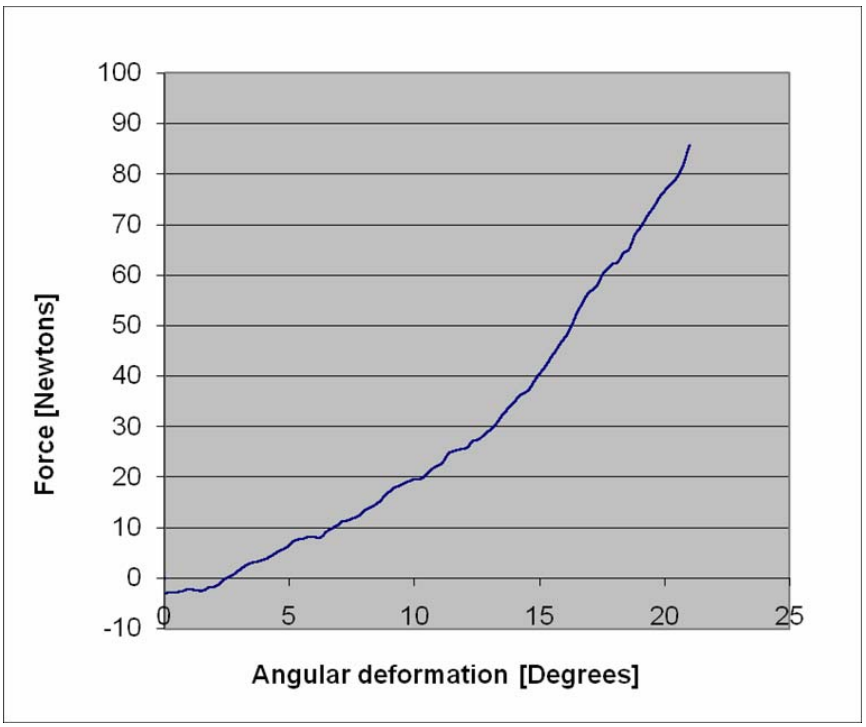

Figure A - 45: Load Deformation Curve – Specimen 26 (Group 2): Dorsiflexion Pre-modification.

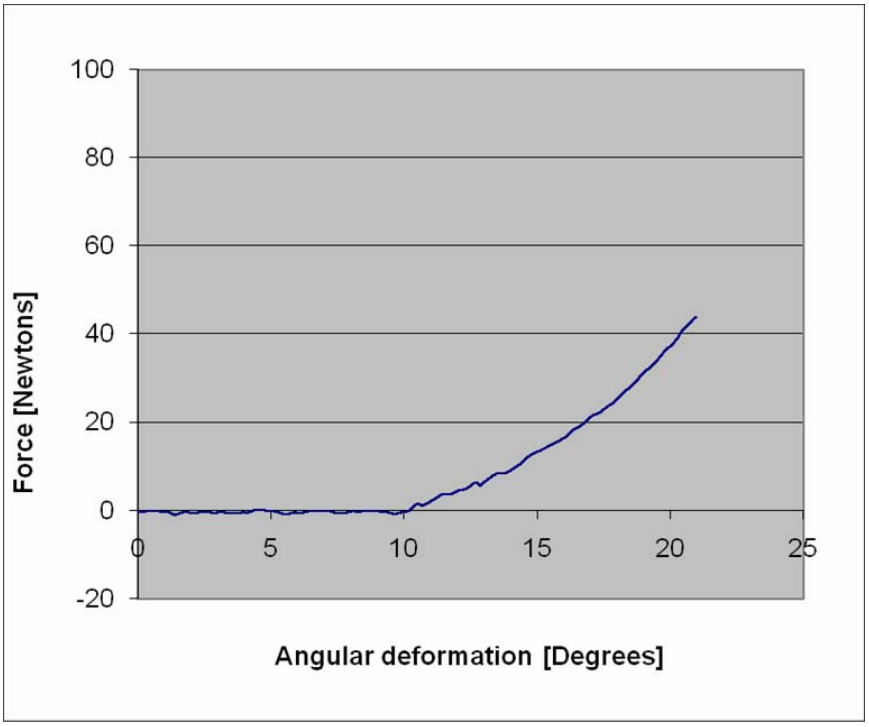

Figure A - 46: Load Deformation Curve – Specimen 26 (Group 2): Dorsiflexion Post-modification.

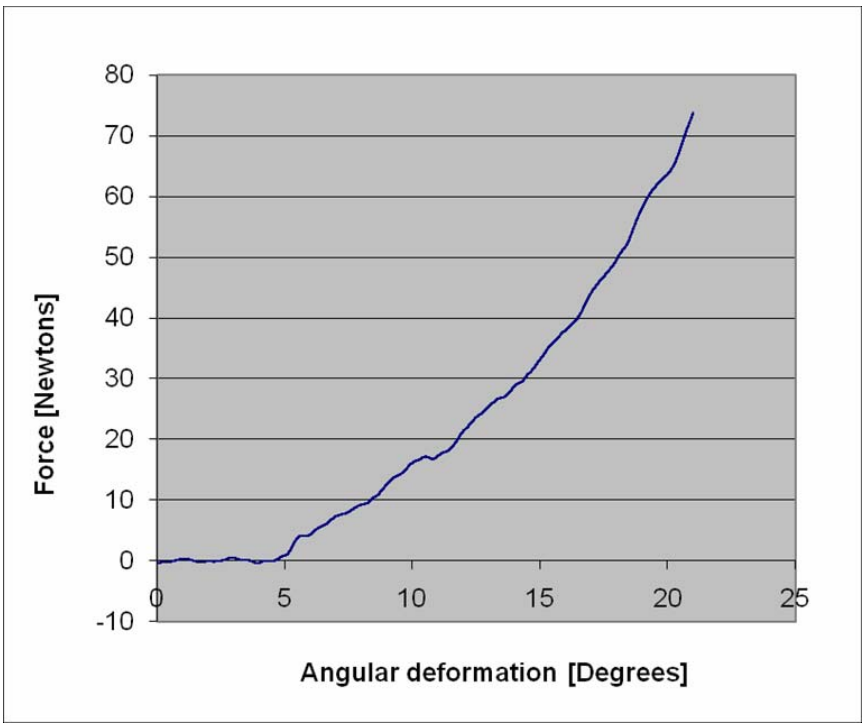

Figure A - 47: Load Deformation Curve – Specimen 26 (Group 2): Ventroflexion Pre-modification.

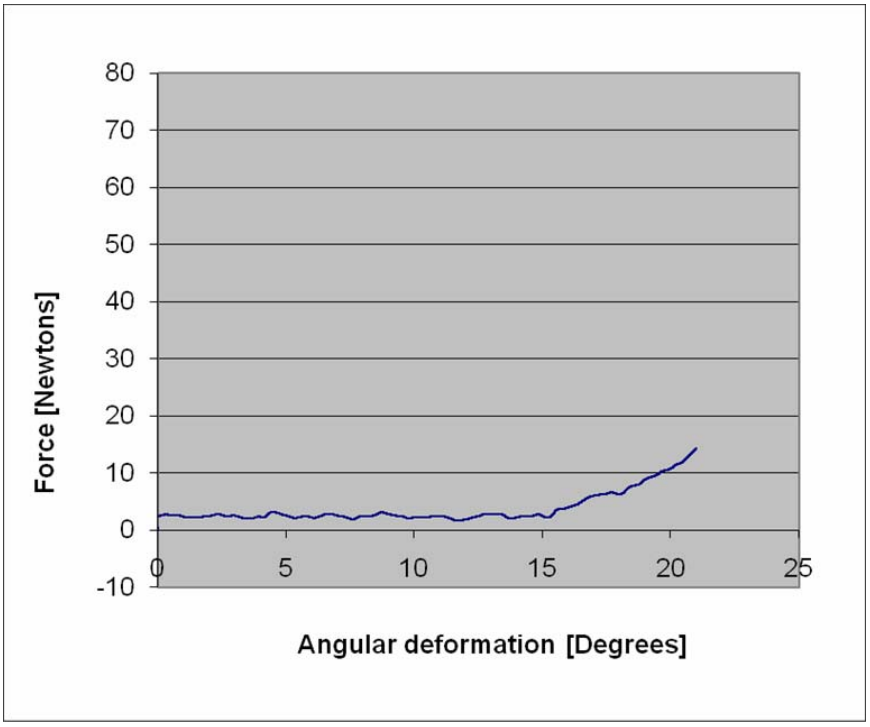

Figure A - 48: Load Deformation Curve – Specimen 26 (Group 2): Ventroflexion Post-modification.

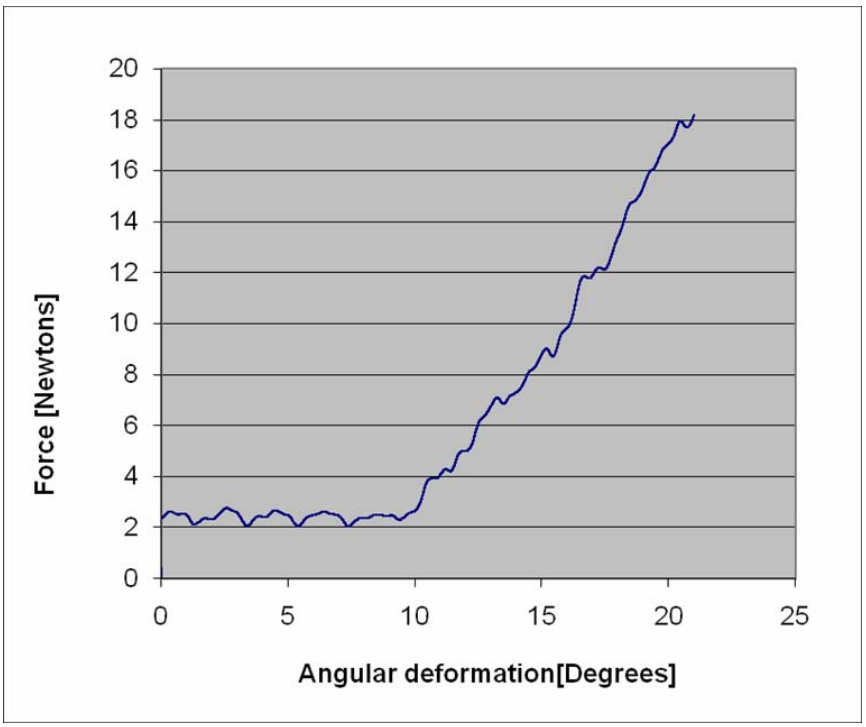

Figure A - 49: Load Deformation Curve – Specimen 9 (Group 3): Dorsiflexion Pre-modification.

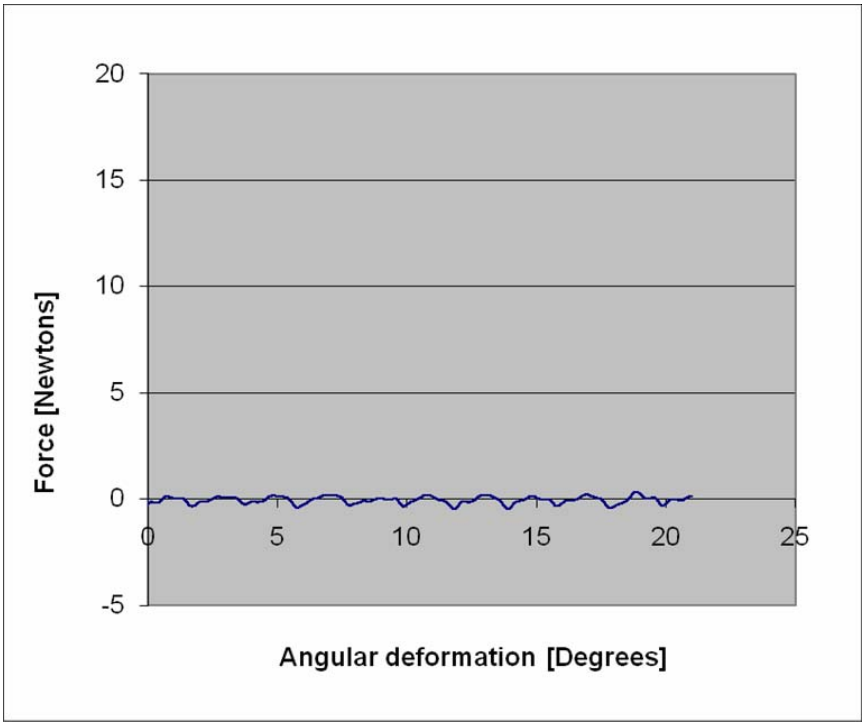

Figure A - 50: Load Deformation Curve – Specimen 9 (Group 3): Dorsiflexion Post-modification.

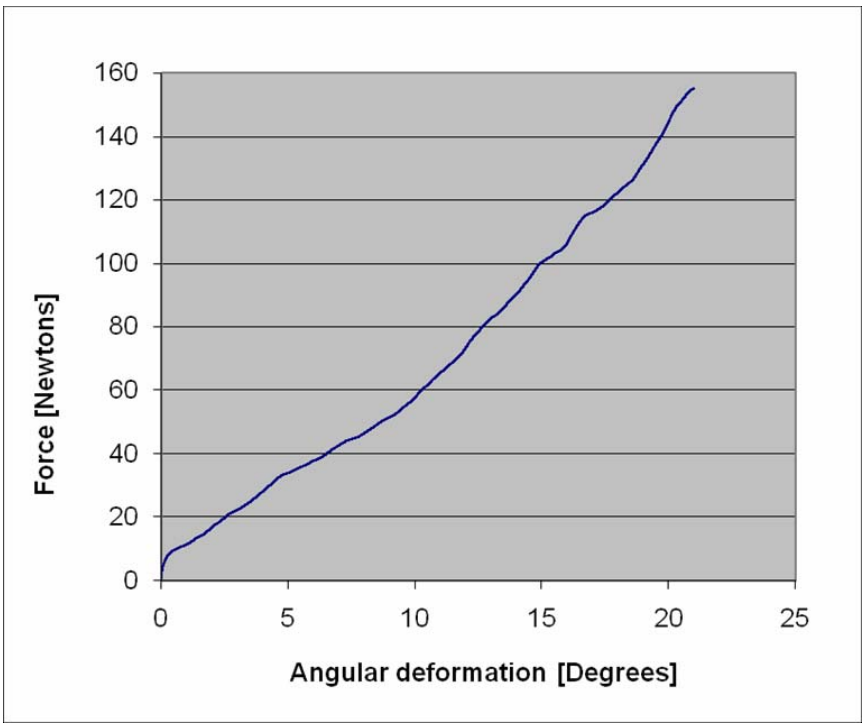

Figure A - 51: Load Deformation Curve – Specimen 9 (Group 3): Ventroflexion Pre-modification.

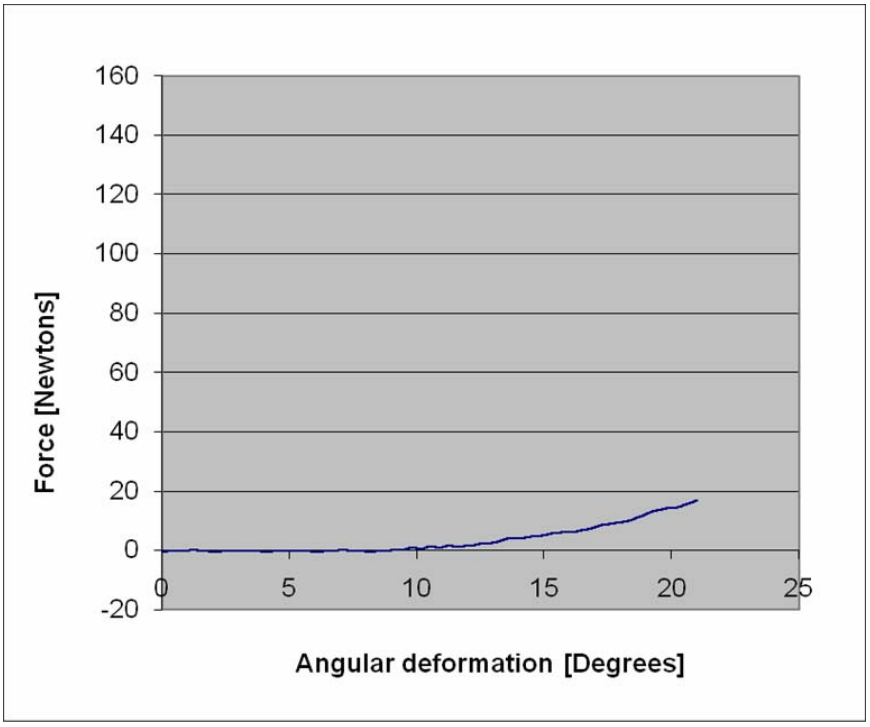

Figure A - 52: Load Deformation Curve – Specimen 9 (Group 3): Ventroflexion Post-modification.

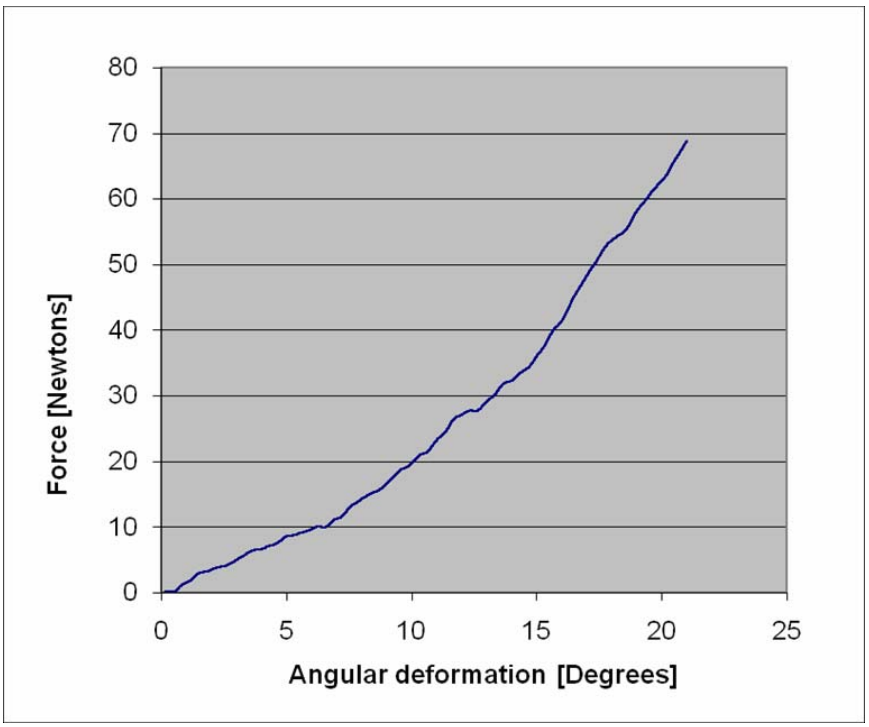

Figure A - 53: Load Deformation Curve – Specimen 11 (Group 3): Dorsiflexion Pre-modification.

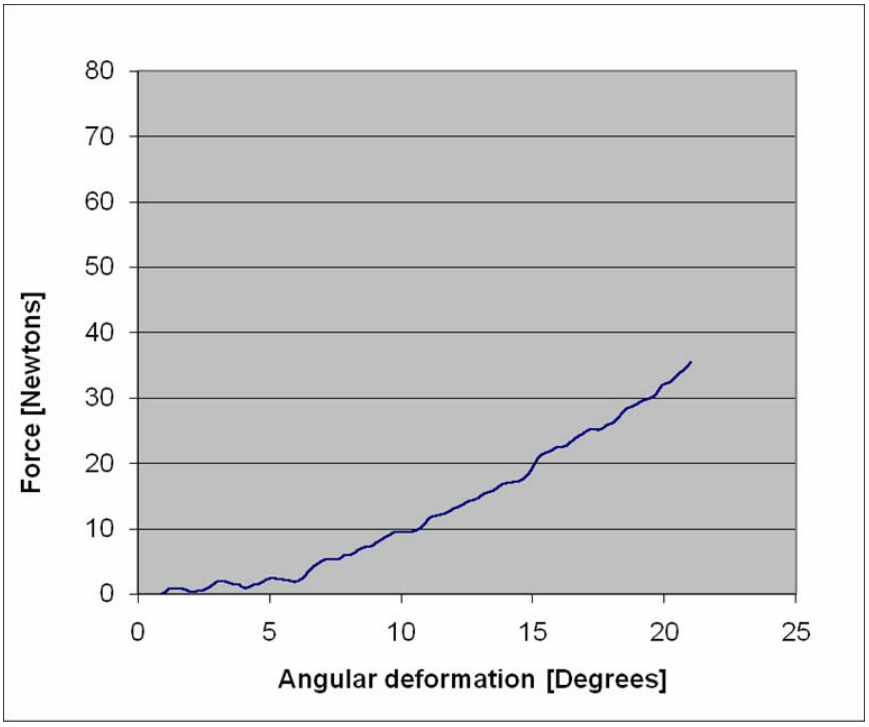

Figure A – 54: Load Deformation Curve – Specimen 11 (Group 3): Dorsiflexion Post-modification.

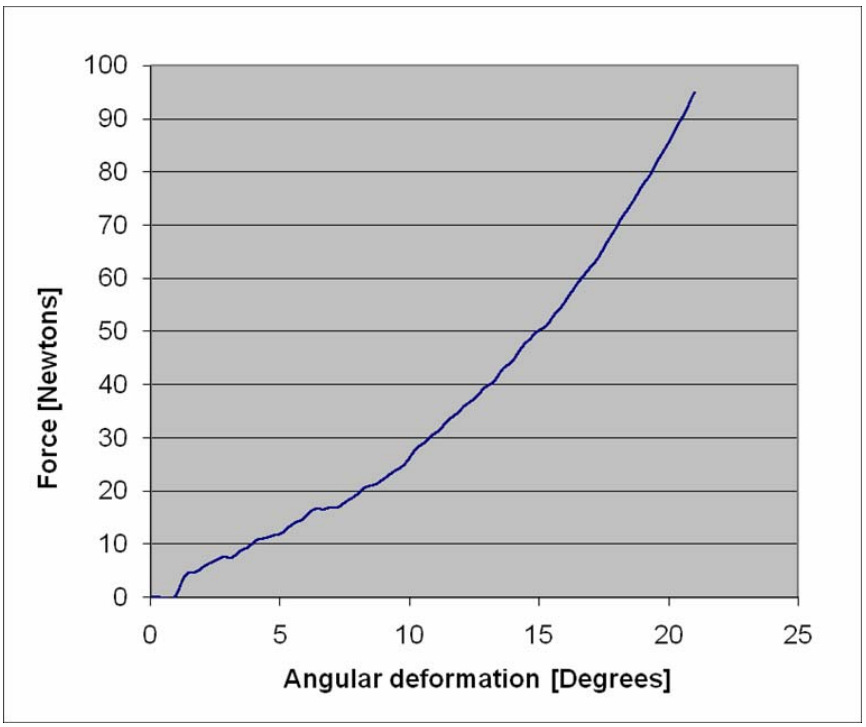

Figure A – 55: Load Deformation Curve – Specimen 11 (Group 3): Ventroflexion Pre-modification.

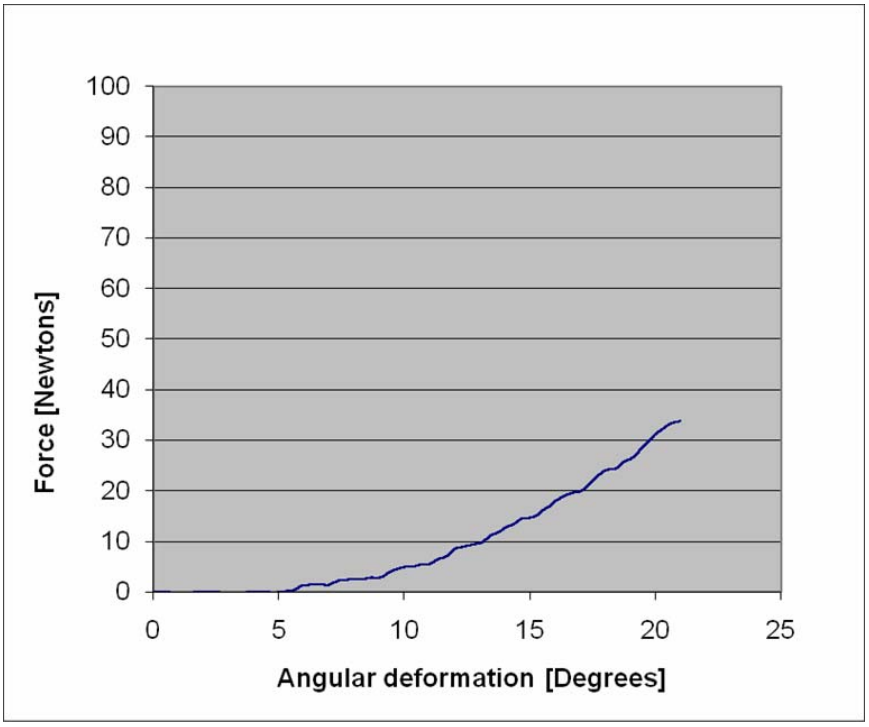

Figure A – 56: Load Deformation Curve – Specimen 11 (Group 3): Ventroflexion Post-modification.

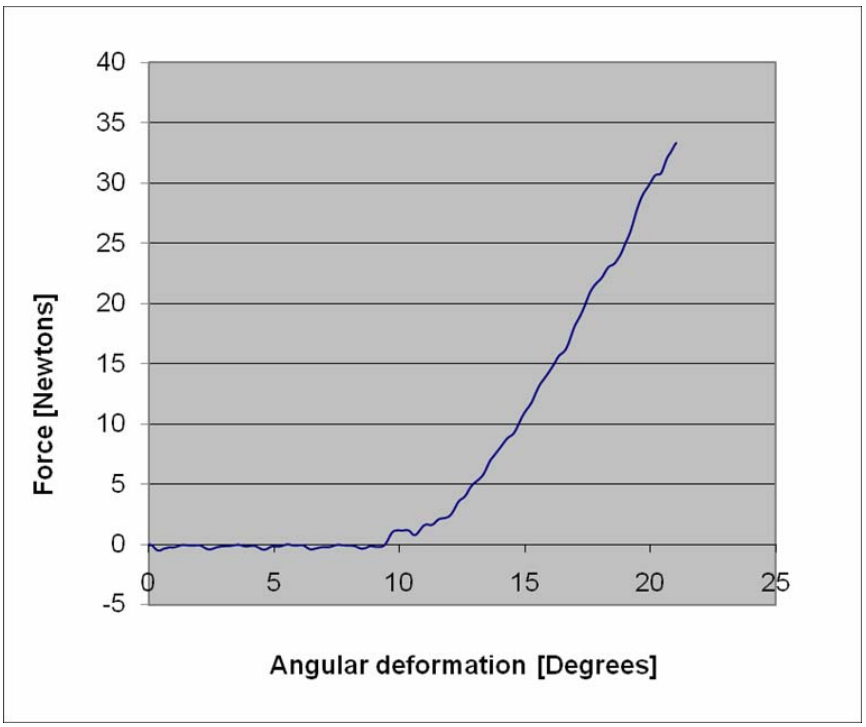

Figure A – 57: Load Deformation Curve – Specimen 18 (Group 3): Dorsiflexion Pre-modification.

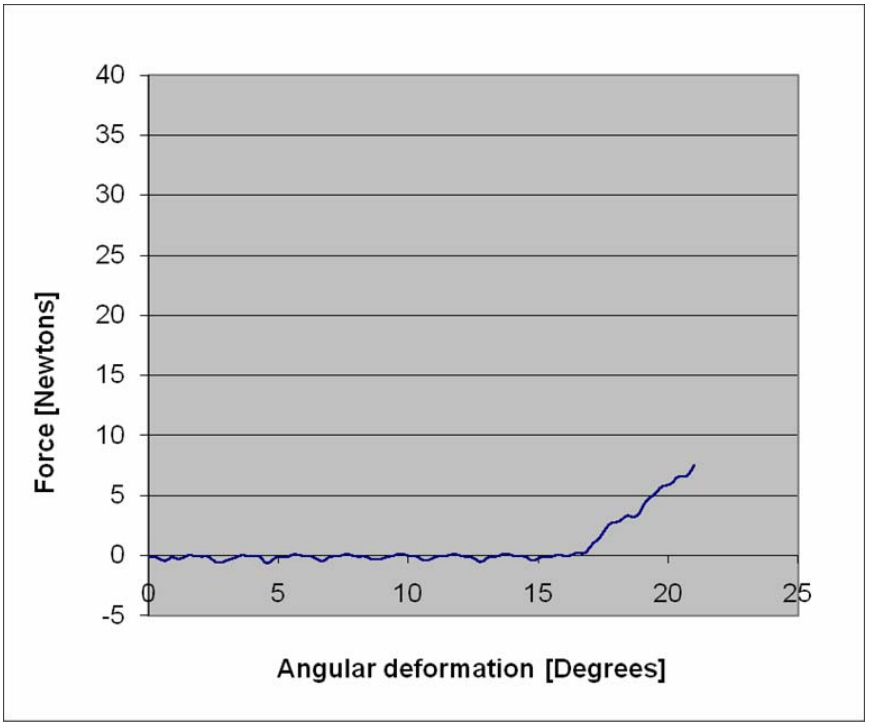

Figure A – 58: Load Deformation Curve – Specimen 18 (Group 3): Dorsiflexion Post-modification.

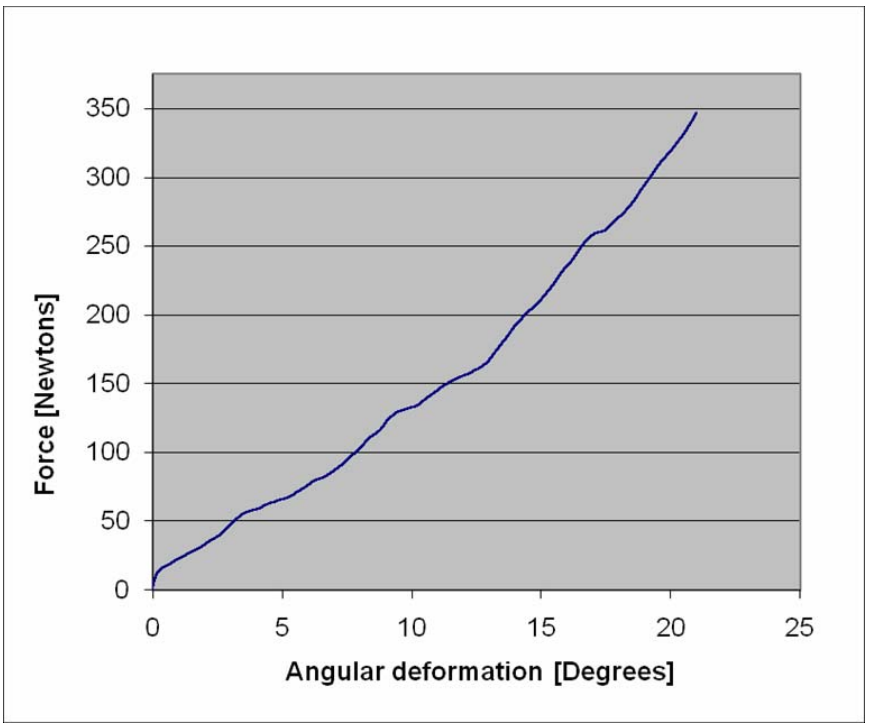

Figure A – 59: Load Deformation Curve – Specimen 18 (Group 3): Ventroflexion Pre-modification.

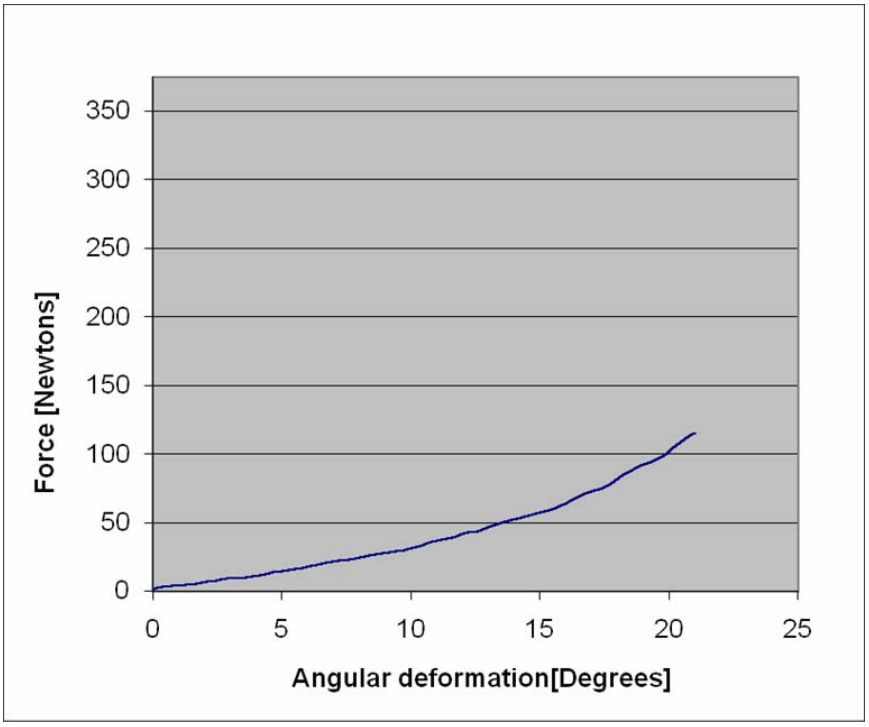

Figure A – 60: Load Deformation Curve – Specimen 18 (Group 3): Ventroflexion Post-modification.

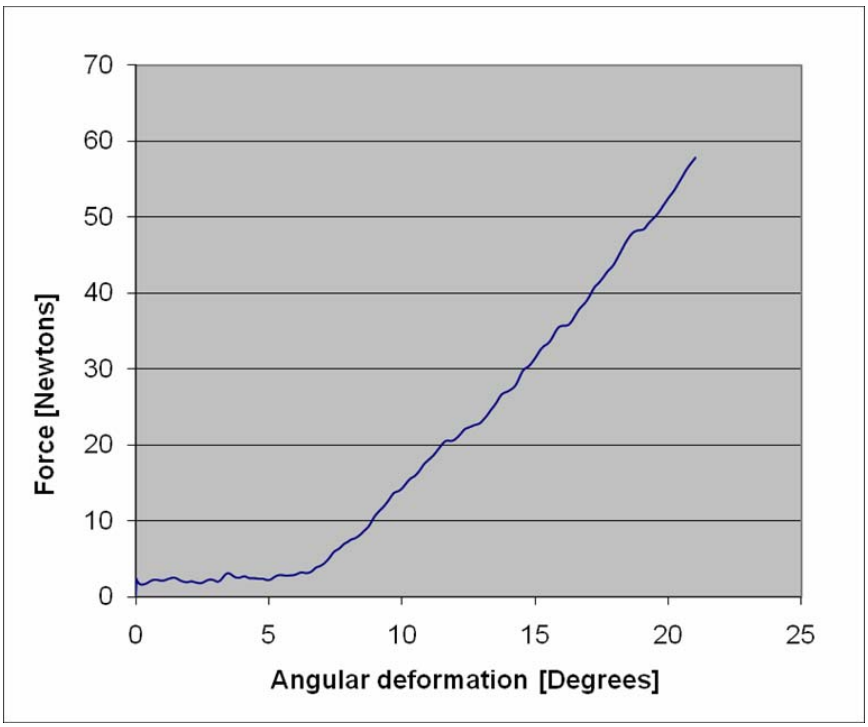

Figure A – 61: Load Deformation Curve – Specimen 21 (Group 3): Dorsiflexion Pre-modification.

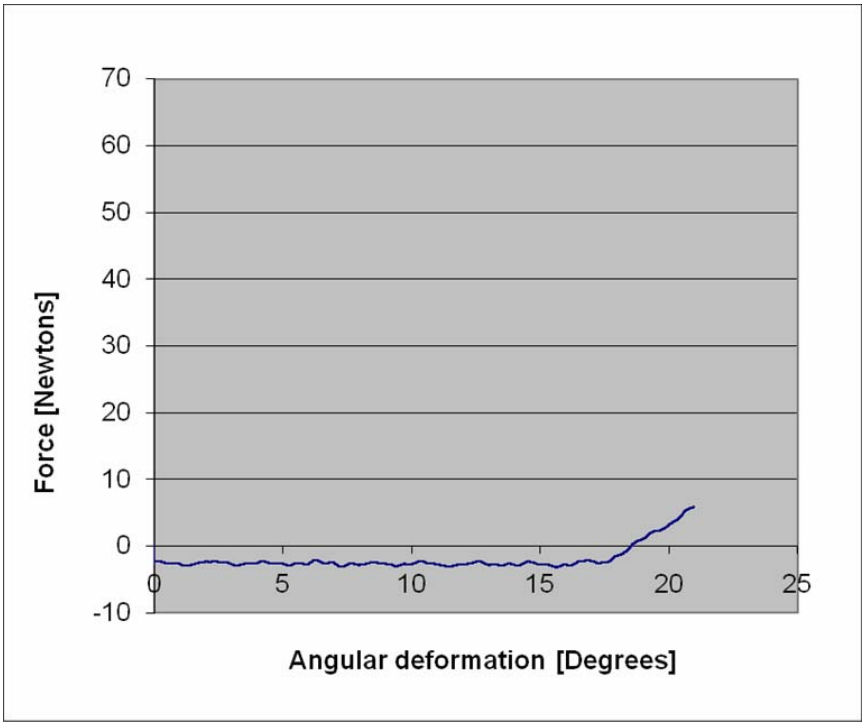

Figure A – 62: Load Deformation Curve – Specimen 21 (Group 3): Dorsiflexion Post-modification.

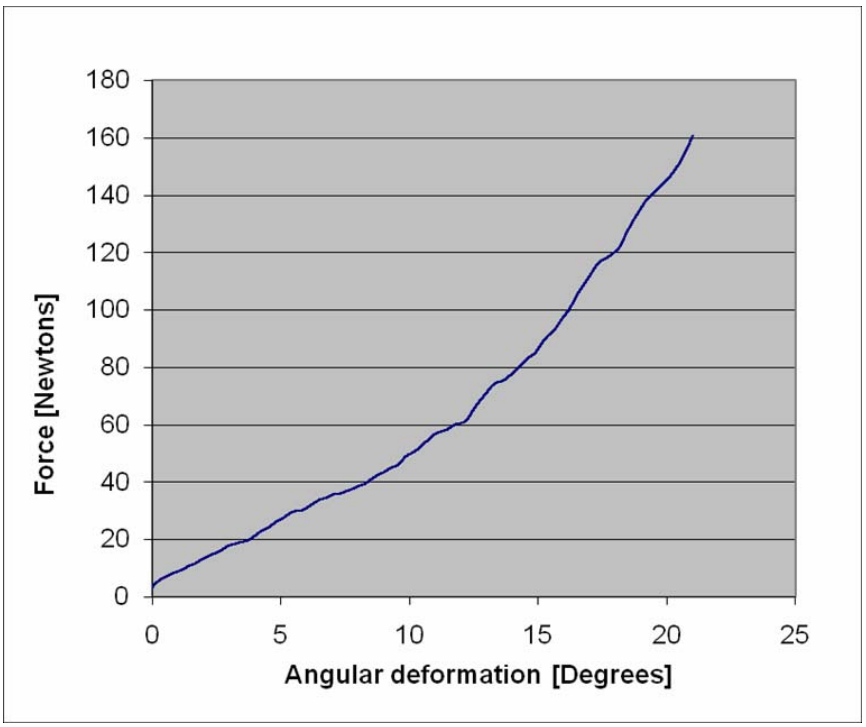

Figure A – 63: Load Deformation Curve – Specimen 21 (Group 3): Ventroflexion Pre-modification.

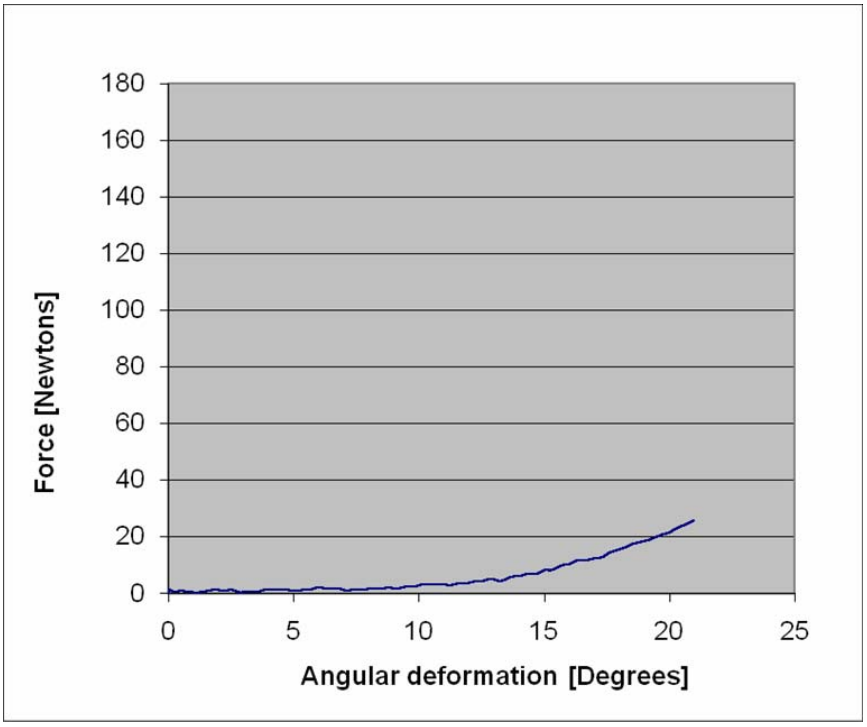

Figure A – 64: Load Deformation Curve – Specimen 21 (Group 3): Ventroflexion Post-modification.

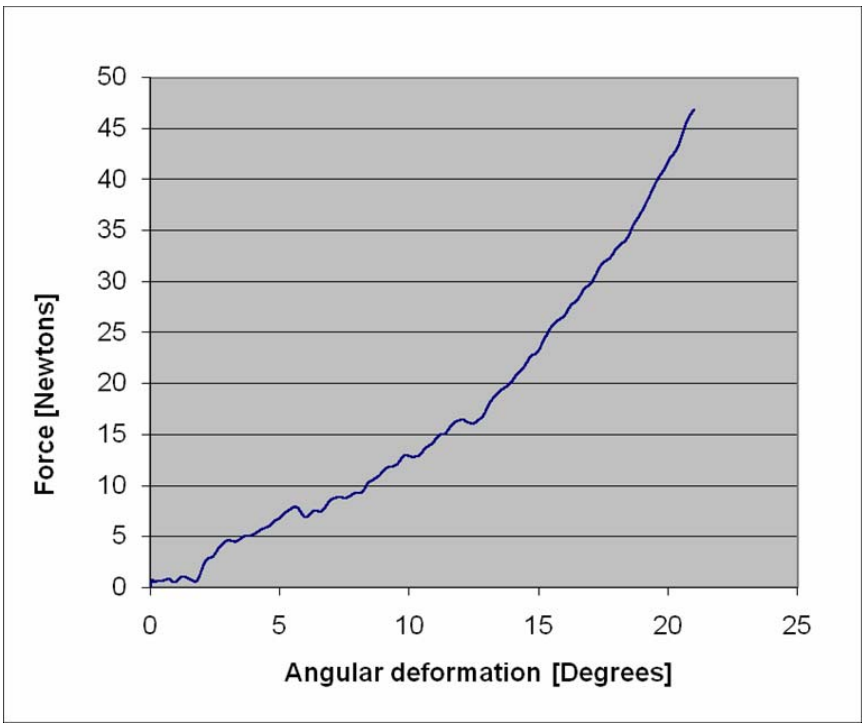

Figure A – 65: Load Deformation Curve – Specimen 22 (Group 3): Dorsiflexion Pre-modification.

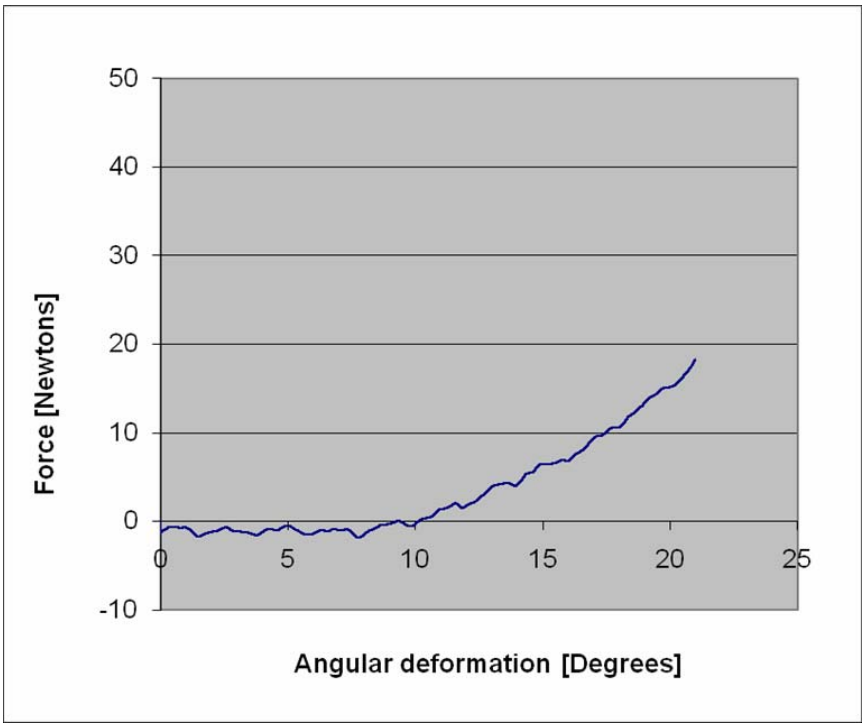

Figure A – 66: Load Deformation Curve – Specimen 22 (Group 3): Dorsiflexion Post-modification.

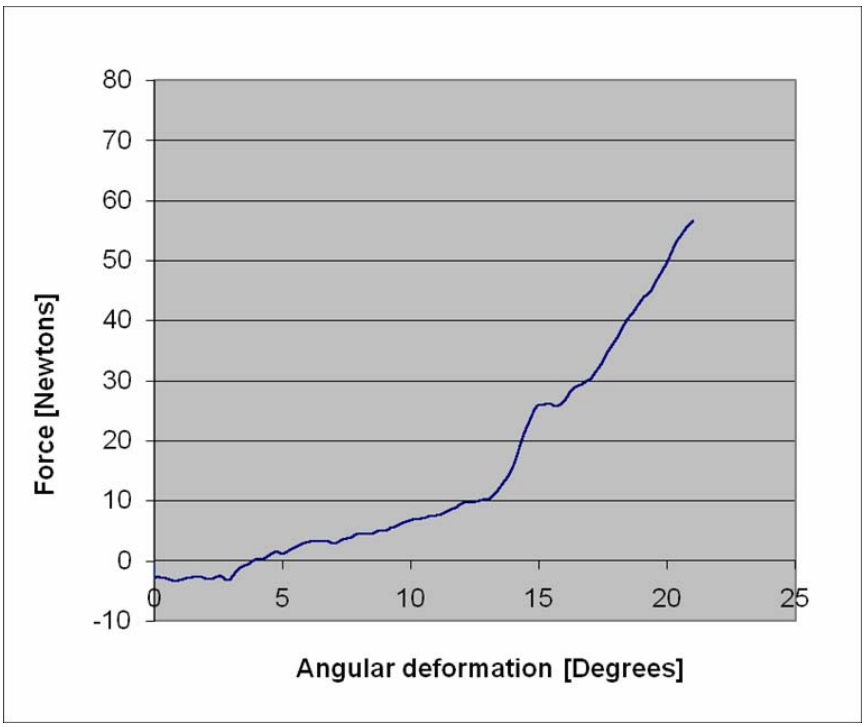

Figure A – 67: Load Deformation Curve – Specimen 22 (Group 3): Ventroflexion Pre-modification.

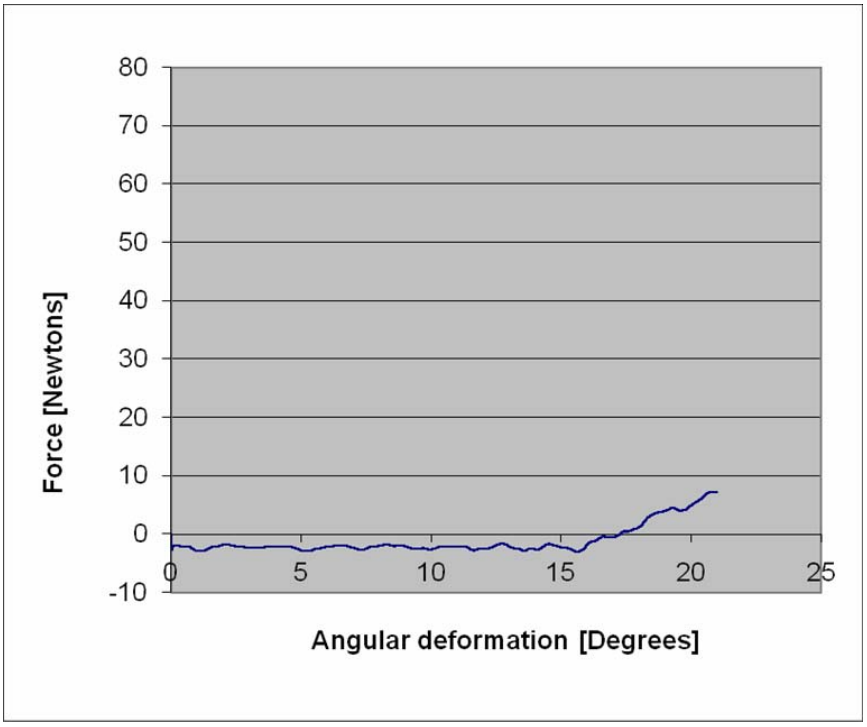

Figure A – 68: Load Deformation Curve – Specimen 22 (Group 3): Ventroflexion Post-modification.

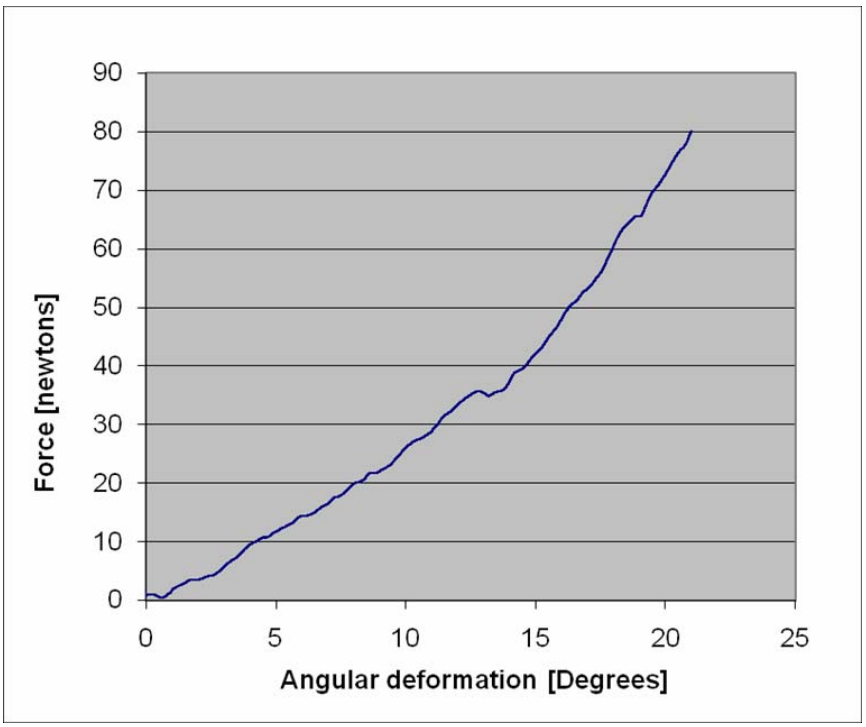

Figure A – 69: Load Deformation Curve – Specimen 27 (Group 3): Dorsiflexion Pre-modification.

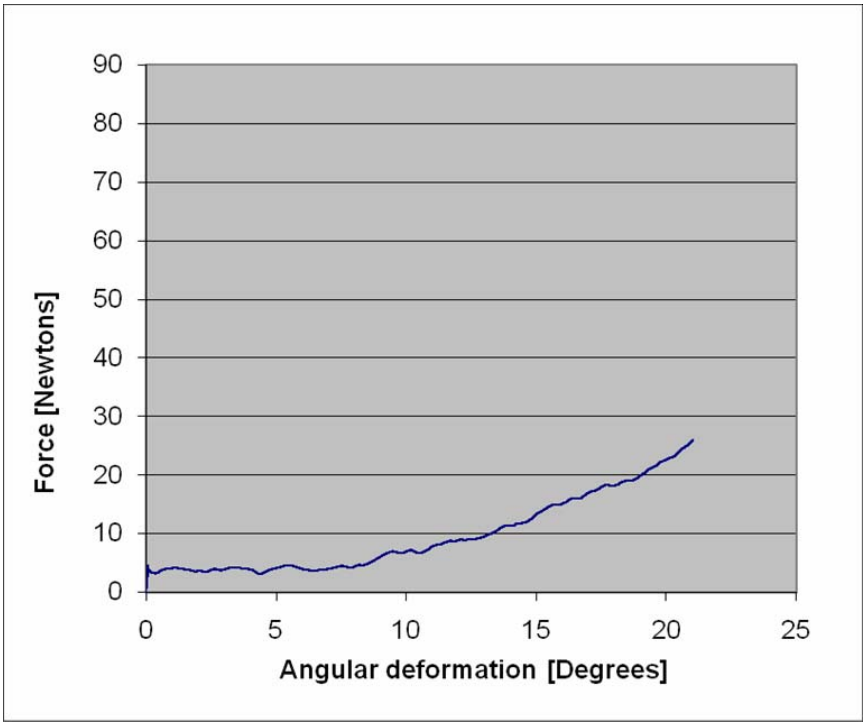

Figure A – 70: Load Deformation Curve – Specimen 27 (Group 3): Dorsiflexion Post-modification.

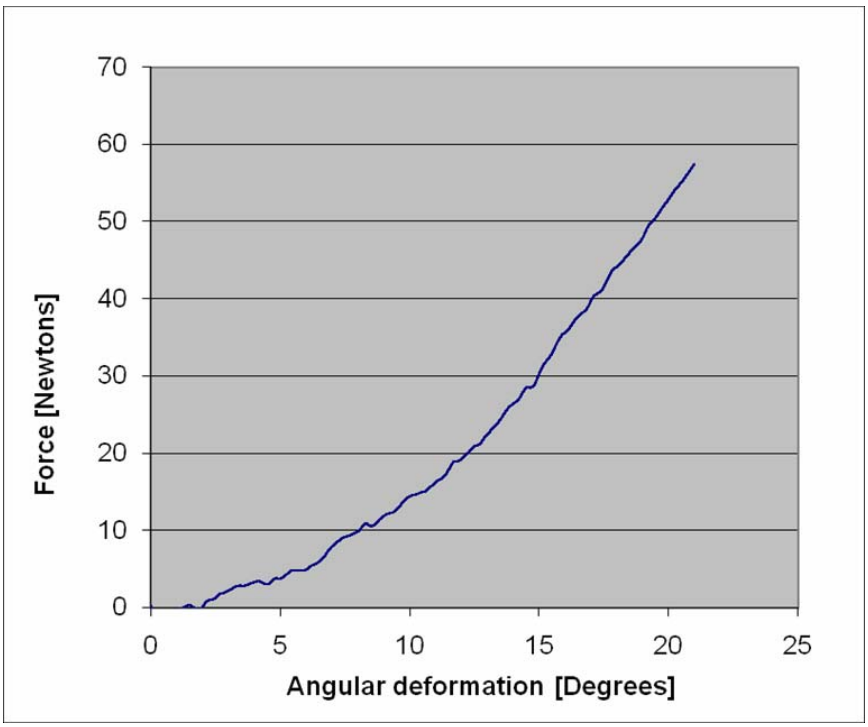

Figure A – 71: Load Deformation Curve – Specimen 27 (Group 3): Ventroflexion Pre-modification.

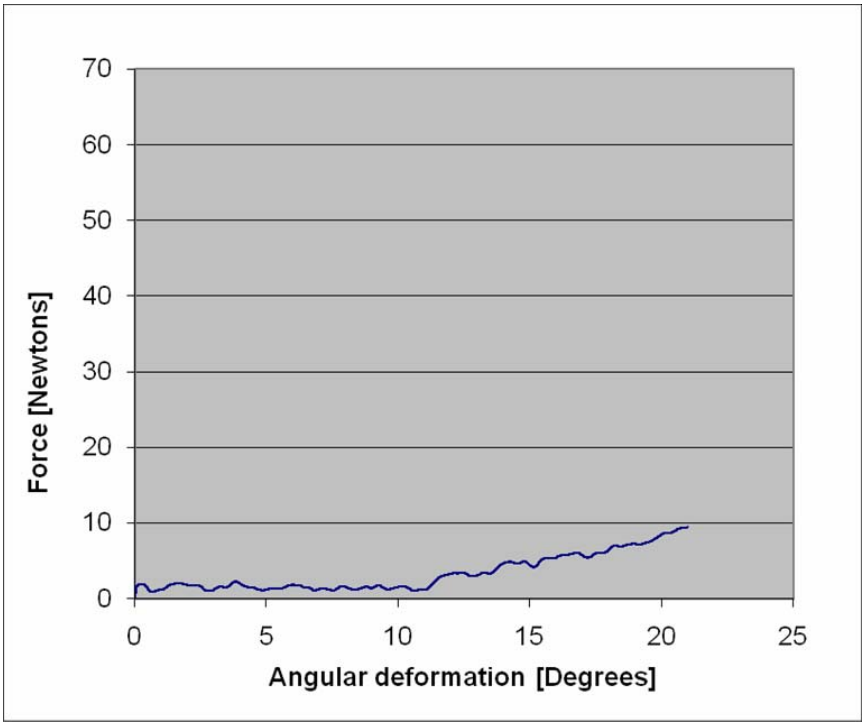

Figure A – 72: Load Deformation Curve – Specimen 27 (Group 3): Ventroflexion Post-modification.

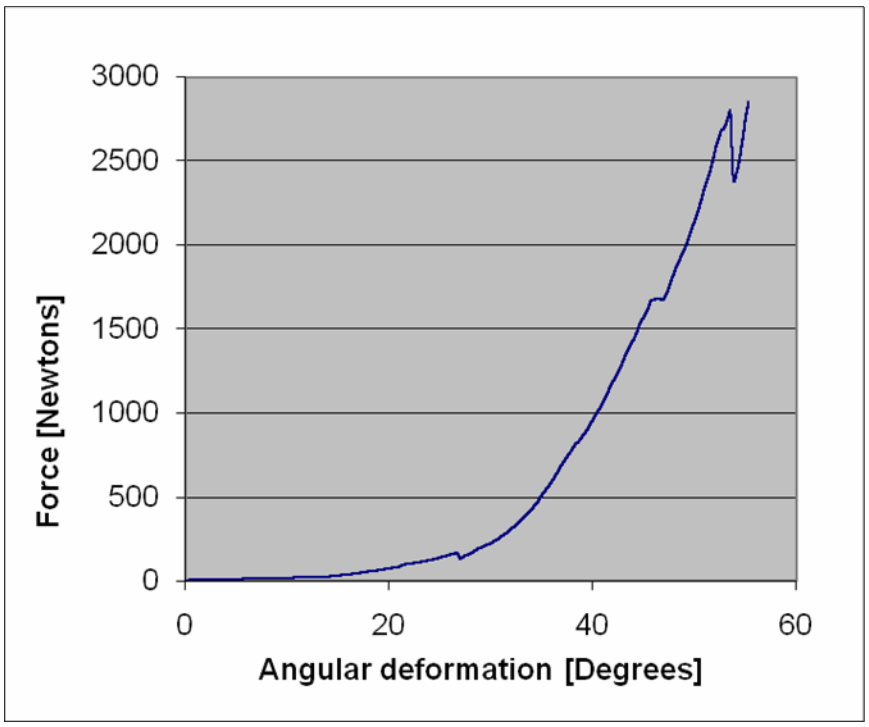

Figure A – 73: Load Deformation Curve – Specimen 10 (Group 1): Ventroflexion to failure.

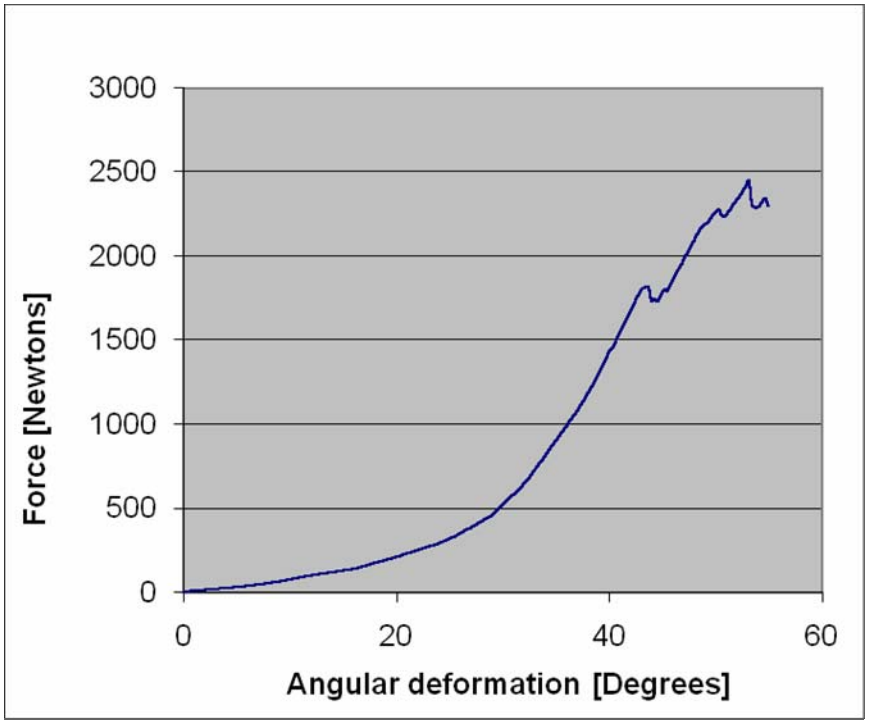

Figure A – 74: Load Deformation Curve – Specimen 12 (Group 1): Ventroflexion to failure.

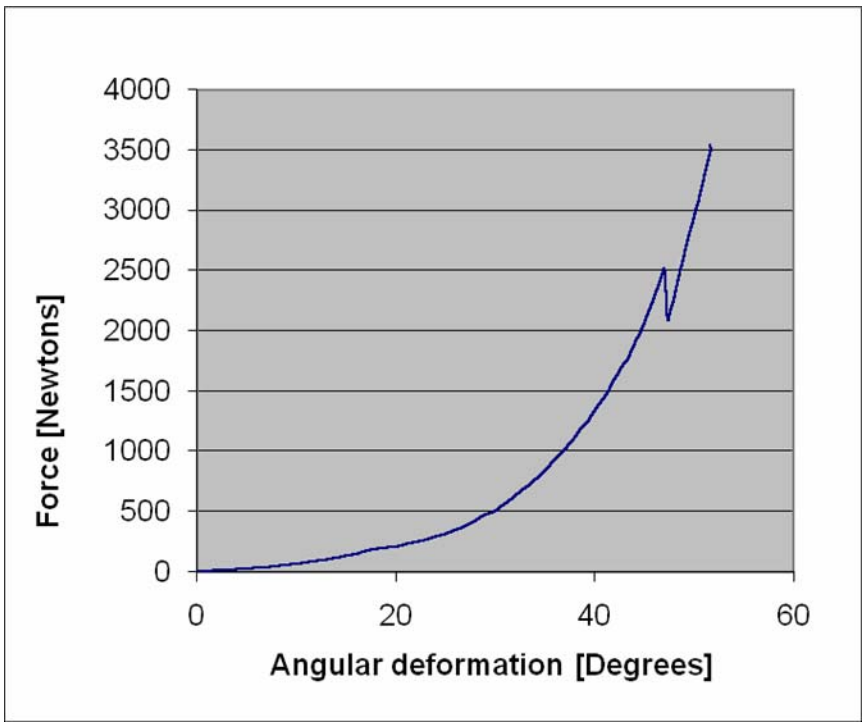

Figure A – 75: Load Deformation Curve – Specimen 13 (Group 1): Ventroflexion to failure.

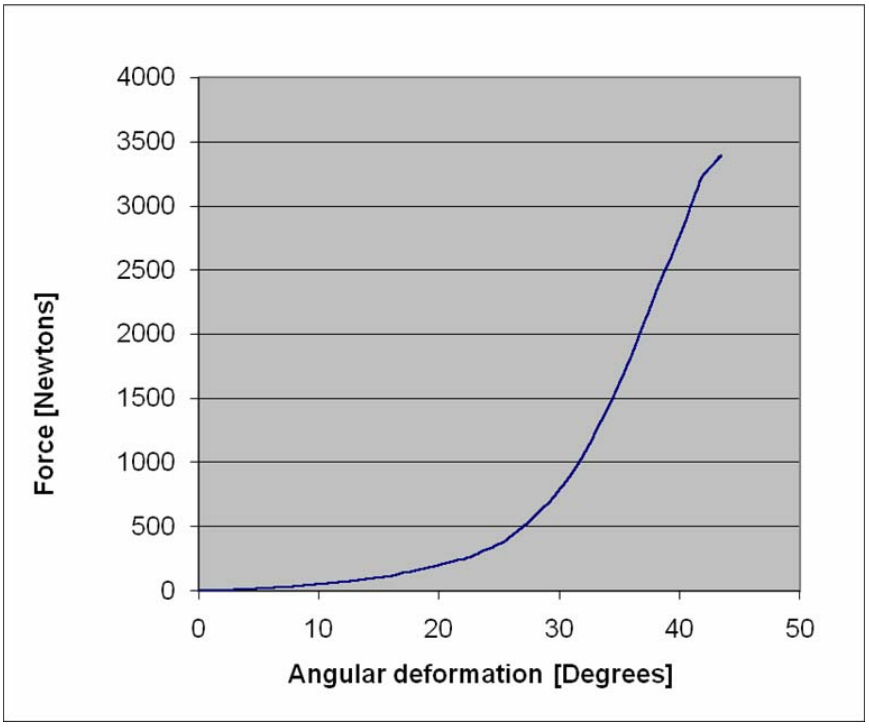

Figure A – 76: Load Deformation Curve – Specimen 16 (Group 1): Ventroflexion to failure.

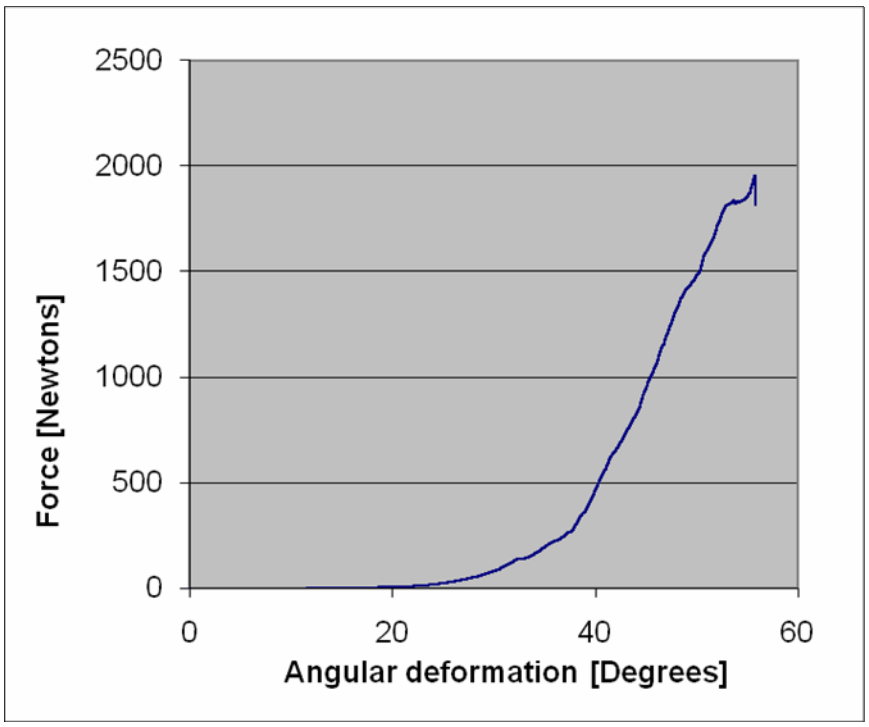

Figure A – 77: Load Deformation Curve – Specimen 6 (Group 2): Ventroflexion to failure.

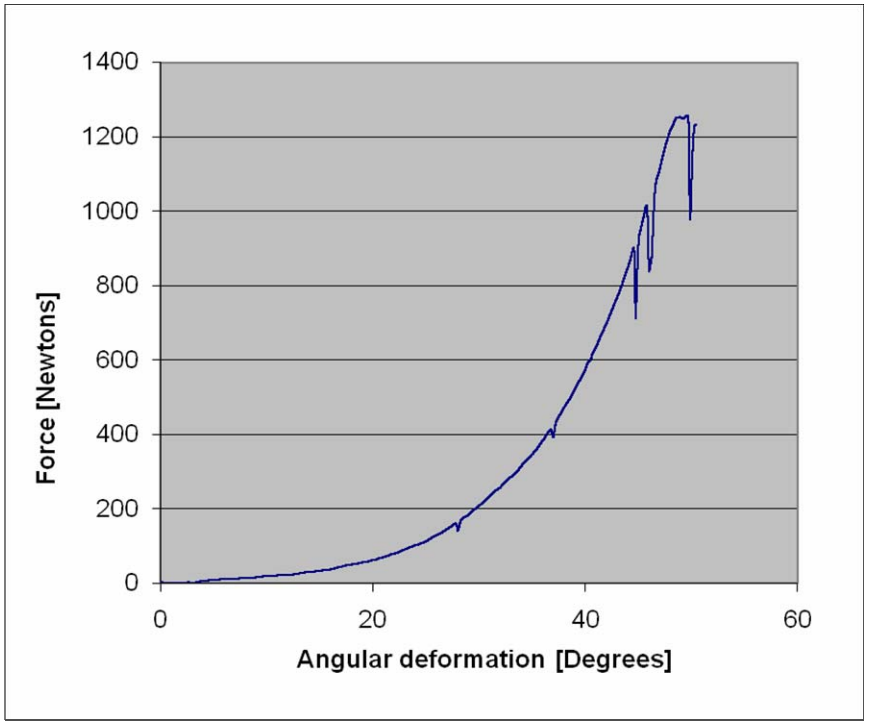

Figure A – 78: Load Deformation Curve – Specimen 7 (Group 2): Ventroflexion to failure.

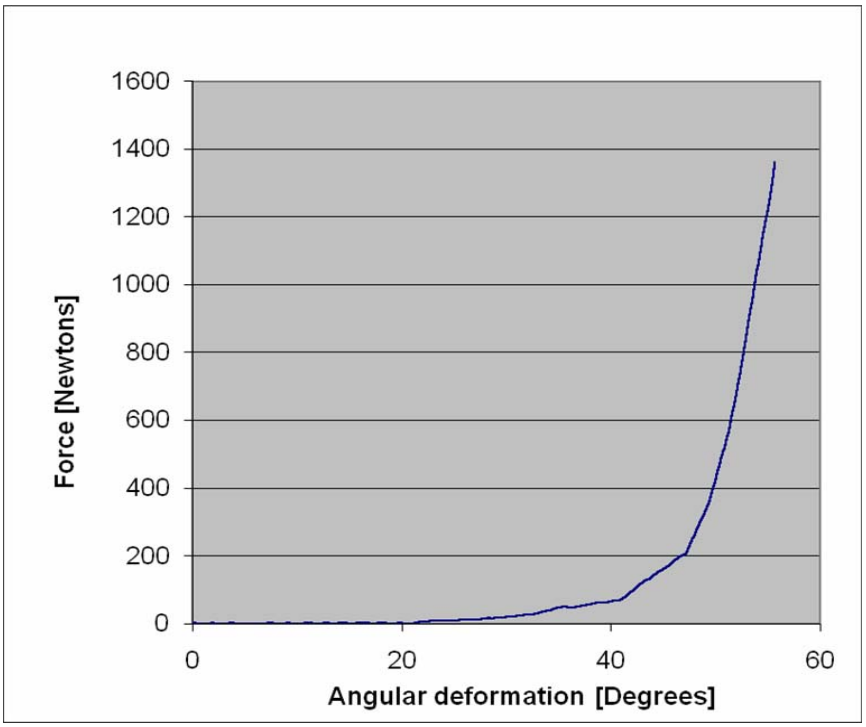

Figure A – 79: Load Deformation Curve – Specimen 15 (Group 2): Ventroflexion to failure.

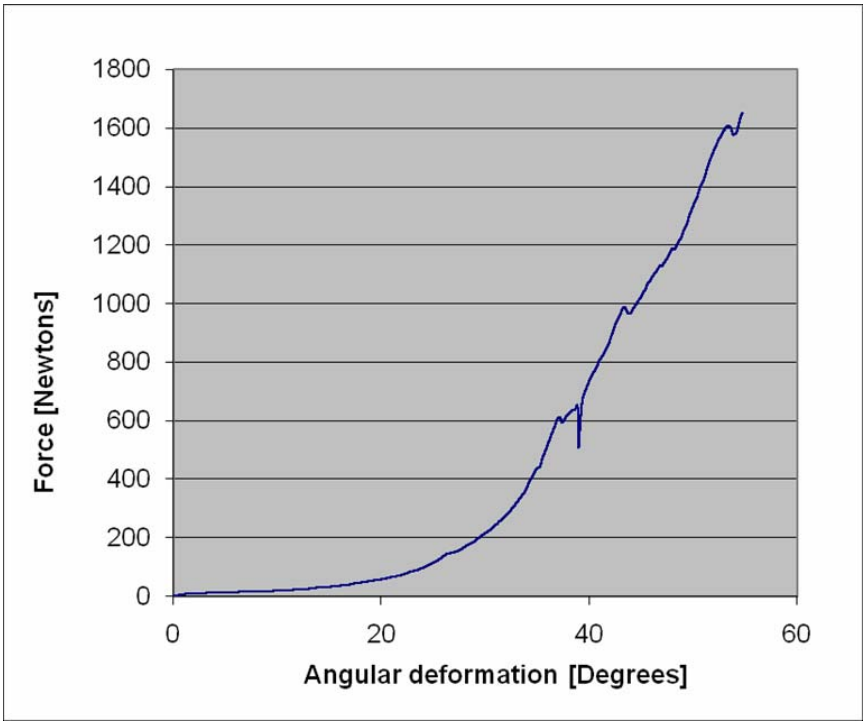

Figure A – 80: Load Deformation Curve – Specimen 17 (Group 2): Ventroflexion to failure.

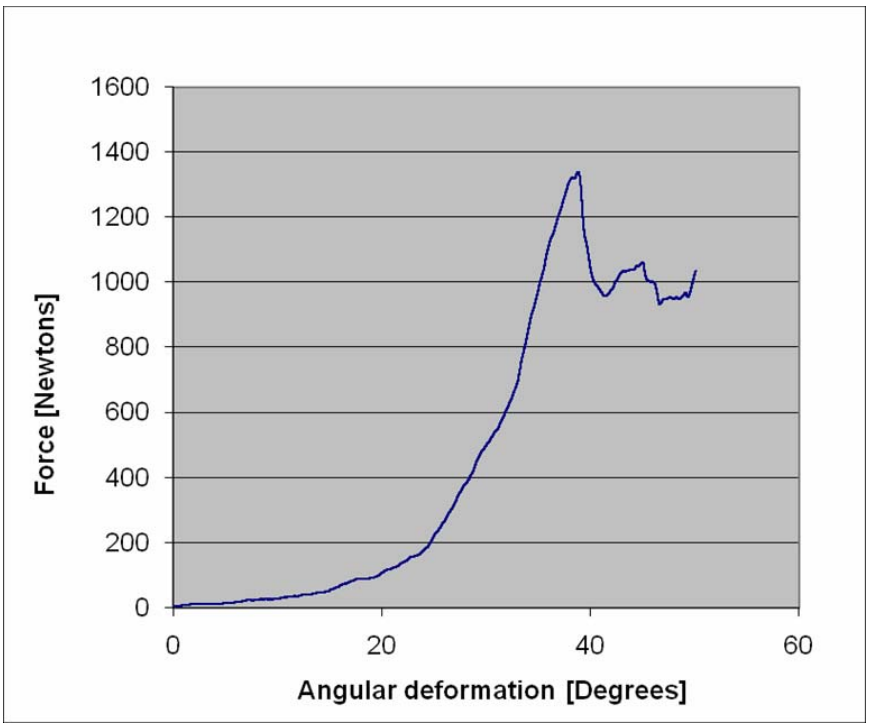

Figure A – 81: Load Deformation Curve – Specimen 19 (Group 2): Ventroflexion to failure.

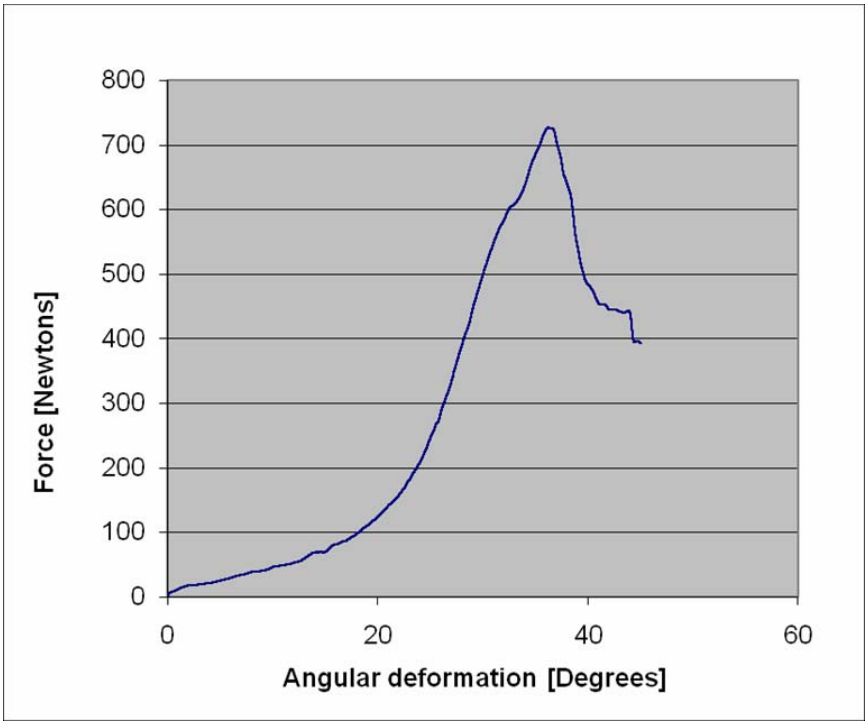

Figure A – 82: Load Deformation Curve – Specimen 20 (Group 2): Ventroflexion to failure.

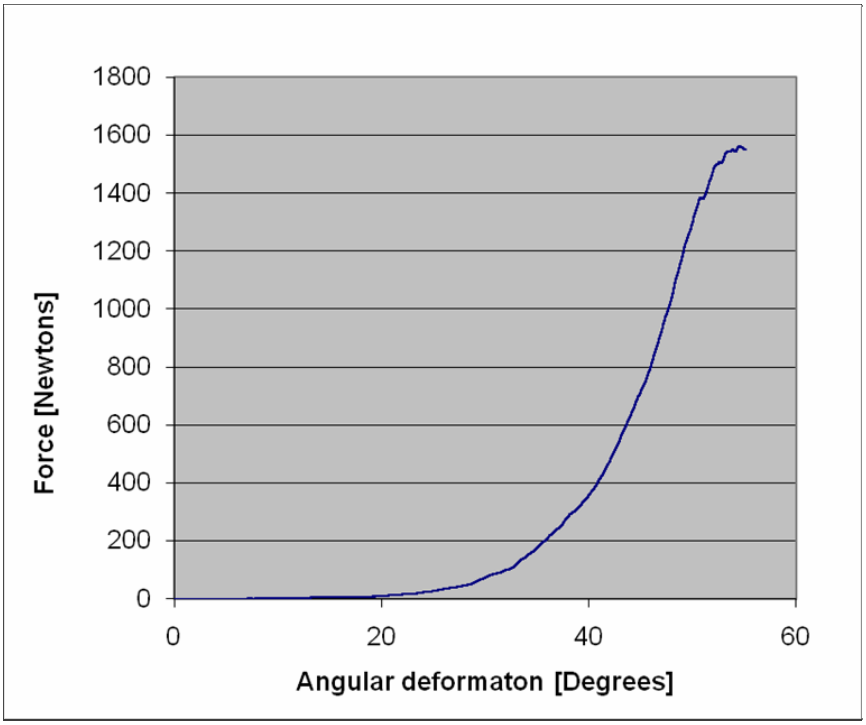

Figure A – 83: Load Deformation Curve – Specimen 26 (Group 2): Ventroflexion to failure.

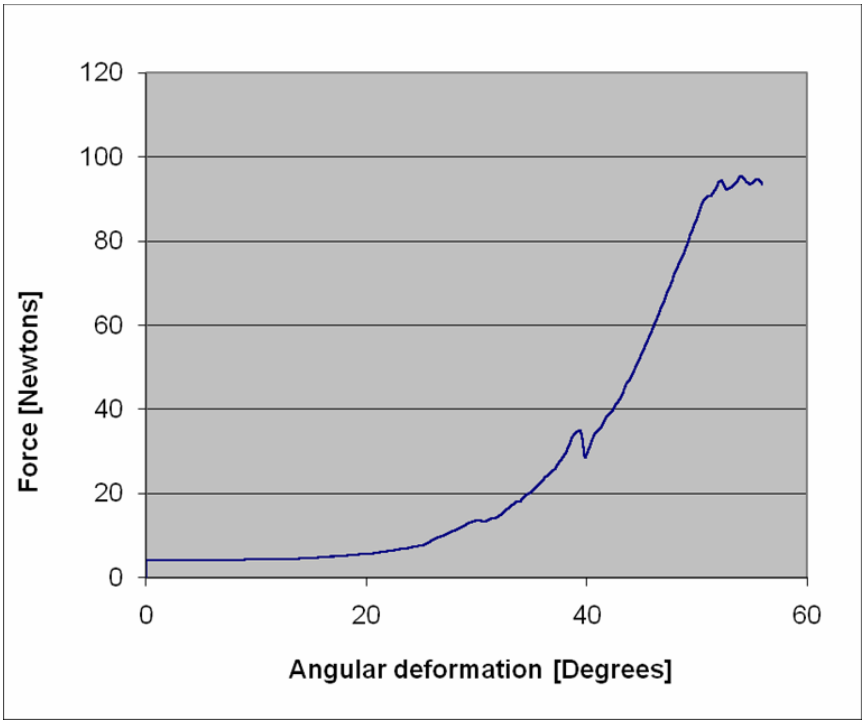

Figure A – 84: Load Deformation Curve – Specimen 9 (Group 3): Ventroflexion to failure.

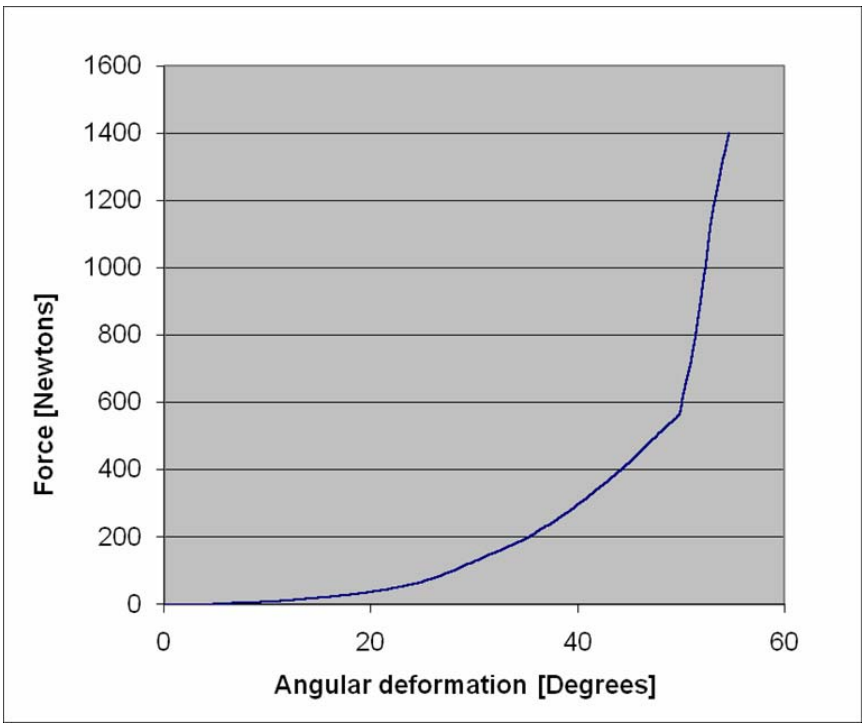

Figure A – 85: Load Deformation Curve – Specimen 11 (Group 3): Ventroflexion to failure.

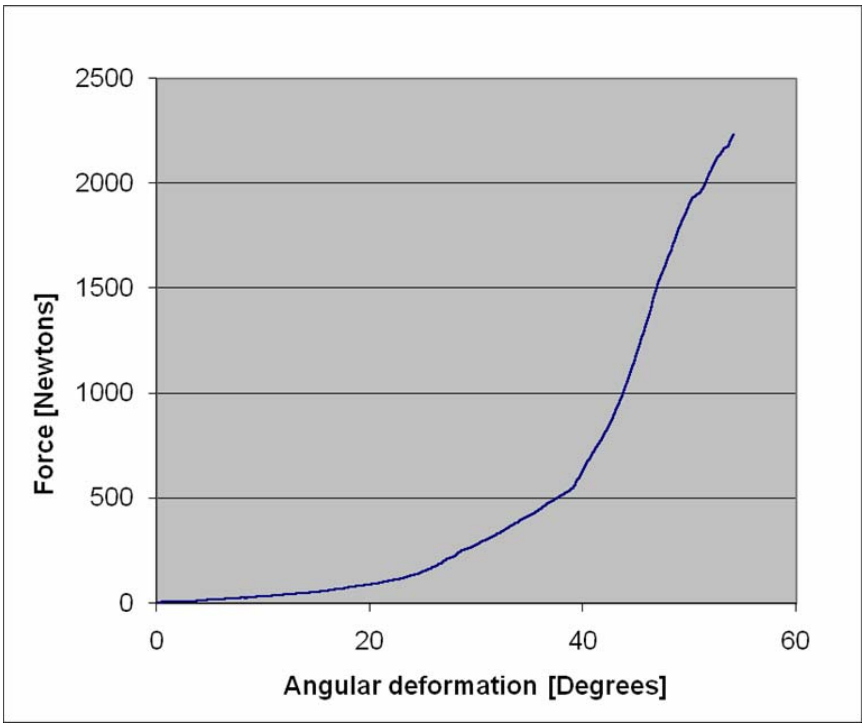

Figure A – 86: Load Deformation Curve – Specimen 18 (Group 3): Ventroflexion to failure.

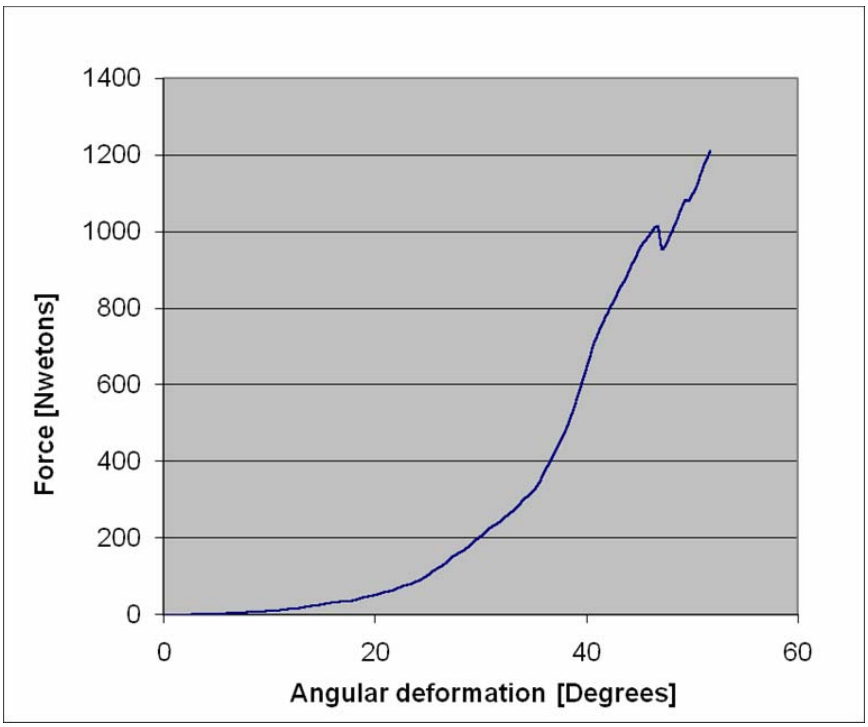

Figure A – 87: Load Deformation Curve – Specimen 21 (Group 3): Ventroflexion to failure.

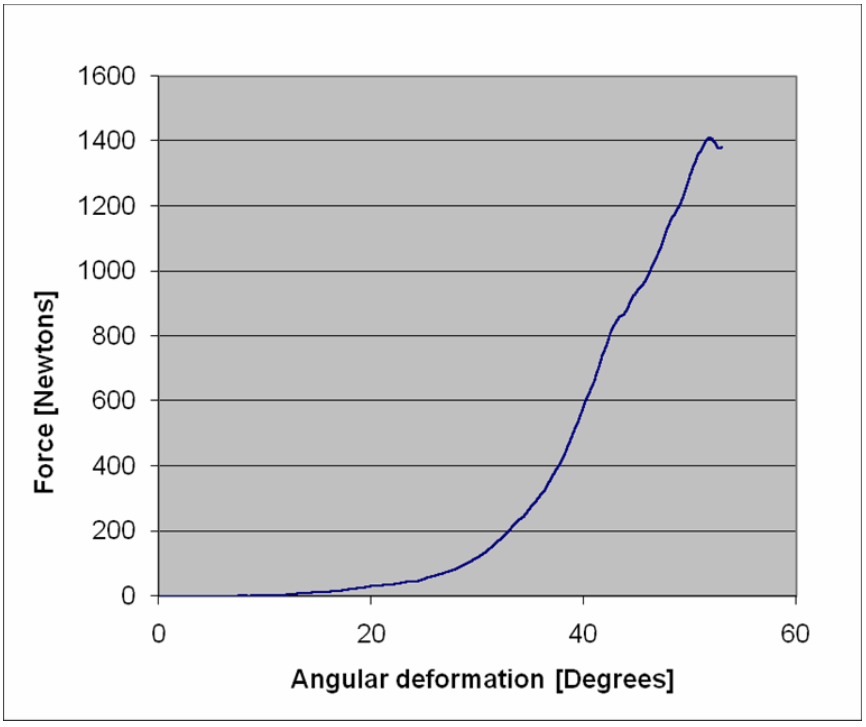

Figure A – 88: Load Deformation Curve – Specimen 22 (Group 3): Ventroflexion to failure.

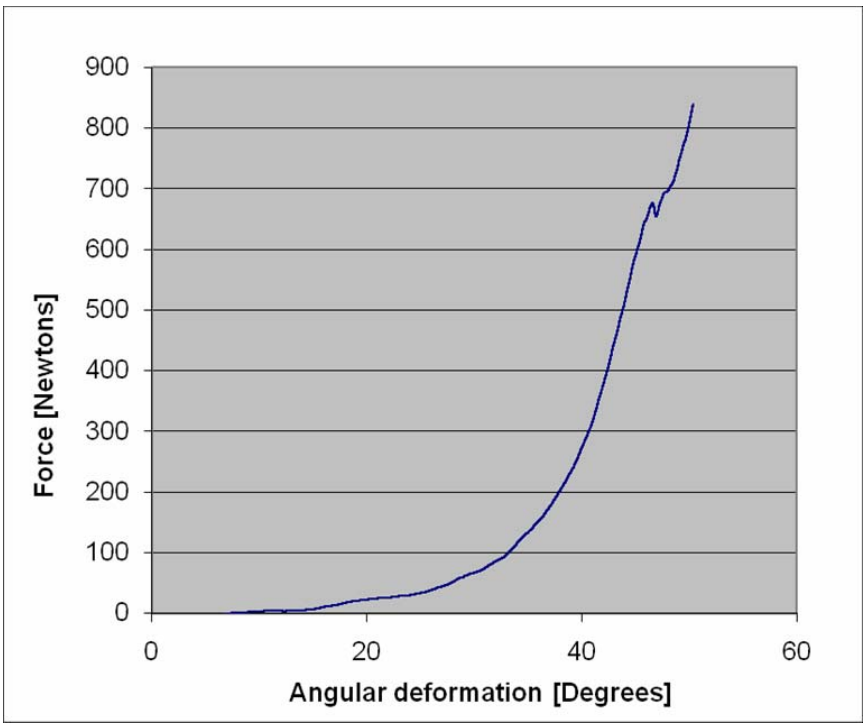

Figure A – 89: Load Deformation Curve – Specimen 27 (Group 3): Ventroflexion to failure.

	Specimen No.	Pre-modification stiffness*	Post-modification stiffness*	% change in stiffness
Group 1	5	1.359	1.866	-37.31
	10	1.754	1.883	-7.35
	12	0.733	0.559	23.74
	13	1.185	1.703	-43.71
	16	2.665	1.890	29.08
Group 2	6	5.513	2.581	53.18
	7	5.286	2.864	45.82
	15	6.116	0.992	83.78
	17	4.337	0.983	77.30
	19	4.179	1.058	74.68
	20	4.161	1.842	55.73
	26	2.610	0.143	94.52
Group 3	9	0.000	0.000	0.00
	11	2.294	2.006	12.55
	18	0.000	0.000	0.00
	21	2.212	0.000	100.00
	22	1.195	0.000	100.00
	27	2.776	0.193	93.05

Table A-1: Stiffness in dorsiflexion at 6°- 8° angular displacement. * stiffness in N/°, change in stiffness expressed as a percentage (positive values indicate decreased stiffness, negative values indicate increased stiffness).

	Specimen No.	Pre-modification stiffness *	Post-modification stiffness*	% change in stiffness
Group 1	5	5.481	6.070	-10.75
	10	3.554	2.810	20.93
	12	1.957	1.359	30.56
	13	2.938	3.526	-20.01
	16	4.413	3.413	22.66
Group 2	6	11.450	7.641	33.27
	7	12.221	7.375	39.65
	15	14.751	6.119	58.52
	17	8.117	2.336	71.22
	19	8.155	2.624	67.82
	20	7.345	3.576	51.31
	26	5.495	2.998	45.44
Group 3	9	1.206	0.029	97.60
	11	3.520	2.364	32.84
	18	3.025	0.000	100.00
	21	3.723	0.000	100.00
	22	2.562	1.265	50.62
	27	3.673	1.555	57.66

Table A-2: Stiffness in dorsiflexion at 12°- 16° angular displacement. * stiffness in N/°, change in stiffness expressed as a percentage (positive values indicate decreased stiffness, negative values indicate increased stiffness).

	Specimen No.	Pre-modification stiffness *	Post-modification stiffness*	% change in stiffness
Group 1	5	12.452	6.351	49.00
	10	5.020	3.903	22.25
	12	1.368	2.032	-48.54
	13	5.300	5.378	-1.47
	16	7.166	5.242	26.85
Group 2	6	19.891	14.209	28.57
	7	21.597	17.952	16.88
	15	28.618	19.824	30.73
	17	12.883	4.447	65.48
	19	9.737	6.582	32.40
	20	27.197	5.023	81.53
	26	7.196	6.006	16.54
Group 3	9	1.838	0.070	96.19
	11	4.561	2.998	34.27
	18	4.028	1.547	61.59
	21	4.226	2.288	45.86
	22	4.281	2.286	46.60
	27	6.142	2.237	63.58

Table A-3: Stiffness in dorsiflexion at 18°- 20° angular displacement. * stiffness in N/°, change in stiffness expressed as a percentage (positive values indicate decreased stiffness, negative values indicate increased stiffness).

	Specimen No.	Pre-modification stiffness *	Post-modification stiffness*	% change in stiffness
Group 1	5	6.516	4.246	34.84
	10	0.035	0.243	-594.29
	12	3.492	3.915	-12.11
	13	4.352	4.062	6.66
	16	6.695	1.331	80.12
Group 2	6	0.867	0.000	100.00
	7	5.473	4.714	13.87
	15	1.666	0.001	99.94
	17	3.042	1.329	56.31
	19	4.675	2.165	53.69
	20	5.864	1.979	66.25
	26	2.469	0.133	94.61
Group 3	9	4.357	0.119	97.27
	11	2.133	0.598	71.96
	18	13.414	3.347	75.05
	21	3.736	0.000	100.00
	22	0.731	0.072	90.16
	27	2.468	0.000	100.00

Table A-4: Stiffness in ventroflexion at 6°- 8° angular displacement. * stiffness in N/°, change in stiffness expressed as a percentage (positive values indicate decreased stiffness, negative values indicate increased stiffness).

	Specimen No.	Pre-modification stiffness *	Post-modification stiffness*	% change in stiffness
Group 1	5	15.230	12.230	17.12
	10	3.364	2.731	18.82
	12	6.714	7.745	-15.36
	13	10.720	7.220	32.65
	16	9.810	11.260	-14.78
Group 2	6	2.542	1.089	57.16
	7	15.455	3.378	78.14
	15	2.775	1.861	32.94
	17	7.292	1.349	81.50
	19	10.288	3.047	70.38
	20	8.081	4.212	47.88
	26	4.137	0.522	87.38
Group 3	9	8.267	1.173	85.81
	11	5.043	2.350	53.40
	18	19.986	5.542	72.27
	21	9.327	1.653	82.28
	22	4.301	0.176	95.91
	27	4.090	0.536	86.89

Table A-5: Stiffness in ventroflexion at 12°- 16° angular displacement. * stiffness in N/°, change in stiffness expressed as a percentage (positive values indicate decreased stiffness, negative values indicate increased stiffness).

	Specimen No.	Pre-modification stiffness *	Post-modification stiffness*	% change in stiffness
Group 1	5	19.557	20.142	-2.99
	10	5.776	7.007	-21.31
	12	16.468	11.586	29.65
	13	16.600	13.542	18.42
	16	23.317	12.639	45.79
Group 2	6	6.409	1.513	76.39
	7	23.036	7.804	66.12
	15	3.755	2.056	45.25
	17	16.947	0.946	94.42
	19	21.702	8.445	61.09
	20	14.800	10.181	31.21
	26	7.006	2.246	67.94
Group 3	9	11.207	2.453	78.11
	11	7.885	3.458	56.14
	18	24.052	10.054	58.20
	21	12.459	3.021	75.75
	22	6.486	1.865	71.25
	27	4.366	1.030	76.41

Table A-6: Stiffness in ventroflexion at 18°- 20° angular displacement. * stiffness in N/°, change in stiffness expressed as a percentage (positive values indicate decreased stiffness, negative values indicate increased stiffness).

	Specimen No.	Force (N) at yield point	Angle (°) at yield point
Group 1	5	No data	No data
	10	2760.926	53.32
	12	1758.737	42.58
	13	2438.766	46.64
	16	3308.324	42.62
Mean (Group 1)		2566.688	46.29
SD (Group 1)		647.277	5.06
Group 2	6	1799.252	52.76
	7	856.805	44.14
	15	191.099	46.32
	17	611.654	42.62
	19	1311.107	37.98
	20	712.893	35.70
	26	1483.763	54.14
Mean (Group 2)		995.225	44.81
SD (Group 2)		559.394	6.92
Group 3	9	286.648	38.68
	11	547.889	49.30
	18	513.554	37.98
	21	981.981	45.94
	22	1377.815	51.16
	27	656.878	46.12
Mean (Group 3)		727.461	44.86
SD (Group 3)		391.163	5.43
Mean (Groups 1, 2 and 3)		1270.476	45.18
SD (Groups 1, 2 and 3)		898.599	5.69

Table A-7: Force and angular displacement at failure. (Force in newtons and angle in degrees).

SD = Standard deviation.