172845

131808

# EPIDURAL STEROID THERAPY AND ULTRASOUND MASSAGE FOR THE MANAGEMENT OF PARAPLEGIA IN DOGS

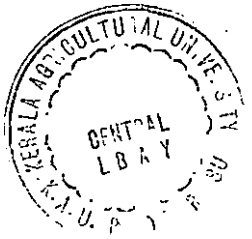
K. M. SEENA

Thesis submitted in partial fulfilment of the requirement for the degree of

## Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University, Thrissur

2008




Department of Veterinary Surgery and Radiology
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY, THRISSUR-680651
KERALA, INDIA

# DECLARATION

I hereby declare that the thesis entitled **"EPIDURAL STEROID THERAPY AND ULTRASOUND MASSAGE FOR THE MANAGEMENT OF PARAPLEGIA IN DOGS"** is a bonafide record of research work done by me during the course of research and that this thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

Mannuthy,
13-08-2008.

Seena

**SEENA. K. M**

# CERTIFICATE

Certified that this thesis entitled **"EPIDURAL STEROID THERAPY AND ULTRASOUND MASSAGE FOR THE MANAGEMENT OF PARAPLEGIA IN DOGS"** is a record of research work done independently by **Seena. K. M** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.

**Dr. T. Sarada Amma**

(Chairman, Advisory Committee)
Professor and Head
Department of Veterinary Surgery and Radiology
College of Veterinary and
Animal Sciences, Mannuthy

Mannuthy,
13-08-2008

# CERTIFICATE

We, the undersigned members of the Advisory Committee of **Seena. K. M,** a candidate for the degree of **Master of Veterinary Science in Surgery,** agree that the thesis entitled **"EPIDURAL STEROID THERAPY AND ULTRASOUND MASSAGE FOR THE MANAGEMENT OF PARAPLEGIA IN DOGS"** may be submitted by **Seena. K. M** in partial fulfilment of the requirement for the degree.

13/8/08

**Dr. T. Sarada Amma**
(Chairman, Advisory Committee)
Professor and Head,
Department of Veterinary Surgery and Radiology,
College of Veterinary and Animal Sciences,
Mannuthy.

13/8/08

**Dr. K. Rajankutty**
Professor,
Department of Veterinary Surgery and Radiology,
College of Veterinary and Animal Sciences, Mannuthy.
(Member)

13/8/08

**Dr. K. M. Jayakumar**
Associate Professor and Head,
Department of Clinical Medicine,
College of Veterinary and Animal Sciences, Mannuthy.
(Member)

13/8/08

**Dr. Syam. K. Venugopal**
Associate Professor,
Department of Veterinary Surgery and Radiology,
College of Veterinary and Animal Sciences, Mannuthy
(Member)

18/11/08

EXTERNAL EXAMINER
Dr. T. P. Balagopalan
ASSOCIATE PROFESSOR
DEPARTMENT OF VETERINARY
SURGERY AND RADIOLOGY
RAGACOVAS
PONDICHERRY - 605 005

# ACKNOWLEDGEMENT

*My feelings are always on the look-out for proper words, to place on record my deep and everlasting obligation to Dr. T. Sarada Amma, Professor and Head, Department of Veterinary Surgery and Radiology for her extraordinary consideration with which she always tried to lead me to the gateway of success. I would like to express my sincere gratitude and indebtedness to her, for her affectionate guidance, valuable suggestions, keen interest and patient correction of the thesis. It gives a great sense of pride to be her ward and without her support and cooperation the successful completion of this work would not have been possible.*

*I humbly express my sincere gratitude to Dr. K. Rajankutty, Professor, Department of Veterinary Surgery and Radiology, for his generous encouragement and invaluable guidance in the pursuit of this research work rendered to me as a member of my advisory committee.*

*With a deep sense of gratitude and respect I express my heart felt thanks to Dr. Syam. K. Venugpal, Associate Professor, Department of Veterinary Surgery and Radiology, for his comprehensive suggestions, inspiring advice and keen interest shown right from the beginning of the research work.*

*I place on record my respect and deep sense of gratitude to Dr. K. M. Jayakumar, Associate Professor and Head, Department of Clinical Medicine, for his kind nature, valuable suggestions, readiness to help and constructive review of my manuscript.*

*I am indebted to Dr. K. D. John Martin, Assistant Professor (ss), Department of Veterinary Surgery and Radiology, for his earnest help especially for conducting myelography in my study.*

*My heartfelt gratitude to Dr. M. K, Narayanan, Assistant Professor (ss), Department of Veterinary Surgery and Radiology, who always encouraged and supported me with his valuable suggestions through out my work.*

*I extend my heartfelt thanks to Dr. C. B. Devanand, Associate Professor, Department of Veterinary Surgery and Radiology for his supporting attitude and pleasant cooperation.*

*I am thankful to Smt. K, Indira Devi, Radiographer, Department of Veterinary Surgery and Radiology for the help rendered to me during the period of my study.*

*I am grateful to Dr. E. Nanu, Dean, Faculty of Veterinary and Animal Sciences, for providing me the facilities for the study.*

*I gratefully acknowledge Smt. K, S. Sujatha,and Smt K, M. Mercy for the help rendered in statistical analysis.*

*I am grateful to Dr. T. A. Raji, Teaching Assistant, Department of Veterinary Surgery and Radiology, for her support and help given to me during the period of my study.*

*The mental support and encouragement given by my colleagues Drs. Remya, Sheeja, Resmi and Ganesh is something that words or deeds cannot express. Its my privilege to accentuate my sincere thanks to Remya for her exuberant attention and support through out the study period.*

*I express my gratitude to my beloved seniors Dr. Tessy Mathew, Dr. N. S. Jinesh kumar and Dr. Somya Ramankutty for the support and help rendered, especially to Dr. Jinesh for understanding my difficulties and offering a helping hand whenever I needed.*

*I thankfully remember the help rendered to me by my junior colleagues Drs. Unnikrishnan. K, Archana.A, lakshmi. V and Priya.P who joined me during the later stages of my work.*

*A special bouquet of thanks to my friends Drs.Jessy, Seena, Rajeswari Pramod and Midhun for their warm friendship and priceless help.*

*I express heartfelt gratitude to my roommates Kavitha, Nimisha and Elso for their warm company, mental support, and sustained encouragement and for helping me to take easy strides during the period of study.*

*It gives immense pleasure to thank Dr. Dinesh. P. T, Veterinary surgeon, District Veterinary Hospital, Kozhikode for his brotherly affection, encouragement and support provided to me whenever I was in need.*

*I am grateful to the final year students of 2002 and 2003 batch for their generous support, help and co-operation extended to me.*

*I sincerely acknowledge the non-teaching staff of our Department and Veterinary hospital of Mannuthy and Kokkalai, for their timely help.*

*I am thankful to Kerala Agriculture University for providing me the fellowship for the postgraduate study.*

*I gratefully remember all those who directly and indirectly helped me in the preparation of thesis.*

*Words are inadequate to express my sense of gratitude and love to my beloved parents and brother for their love, care, moral support, encouragement and prayers.*

*Above all I bow before the Almighty God for all the blessings showered on me, thanking Him for all that I have and don't..... and helping me in completing this work successfully.*

*Seena K. M.*

# CONTENTS

## LIST OF TABLES

## LIST OF FIGURES

# *Introduction*

# 1. INTRODUCTION

Paraplegia is one of the most commonly presented neurologic problems to the veterinarians and is characterized by the absence of voluntary movement of pelvic limbs (Shell, 1996a).

Rapidly applied concussive, compressive, or distractive forces either alone or in combination are responsible for acute spinal cord trauma. Paraplegia / posterior paralysis in dogs may occur subsequent to many spinal cord diseases including compressive, infectious, degenerative, congenital, and neoplastic conditions. Intervertebral disc herniation is the most common cause of acute spinal cord injury in dogs (Thomson *et al*, 1989).

Establishing the cause of paralysis involves interpretation of the animal's history, signalment, physical and neurological examination, biochemical and serological data, results of CSF analysis, plain and contrast radiography and electro diagnostic techniques (Thomson *et al*, 1989). Neurological examination was used to localize the lesion to a spinal cord segment. Plain radiography will allow diagnosis of the majority of the conditions involving vertebrae and intervertebral disc. Myelography is indicated in cases where lesions can not be visualized on plain radiography, such as tumours not involving bone (Griffith, 1982).

There are both surgical and non surgical treatments for paraplegia in dogs. Corticosteroids are used to treat brain and spinal cord disorders usually those associated with trauma. These mainly reduce oedema and inflammation (Calvert and Cornelius, 1990b). Corticosteroids of varying potencies are available in a number of formulations with differing onset and duration of action (Cohn, 1997).

The gastrointestinal side effects are more with dexamethasone administration than with methyl prednisolone (Wheeler, 1997). Methyl prednisolone differs from prednisolone by the addition of a methyl group to C-6. This addition slightly boosts the anti-inflammatory activity (Calvert and Cornelius, 1990d). Epidural administration of methyl prednisolone acetate was reported to be effective for the treatment of paraplegia in dogs (Chandy and Vasanth, 2006). The local effect of methyl prednisolone acetate on the epidural space may reduce the inflammation and subsequent changes associated with the injury.

Ultrasound physiotherapy is beneficial for epaxial muscles that experiencing muscle spasm. In non surgical patients with acute spinal cord disease and neuromuscular spasm, ultrasound is applied to the epaxial muscles to manage pain and muscle spasm (Olby *et al.*, 2005).Ultrasound massage has both thermal and non thermal effects. Therapeutic ultrasound increases blood flow, heat in deep tissues, reduces pain, muscle spasm and accelerates wound healing (Taylor, 2001).

On screening the available literature, the systematic evaluation on the effect of epidural administration of methyl prednisolone acetate on paraplegia and the effective use of ultrasound massage in such cases are found scanty.

Hence the study was undertaken with an objective to evaluate the effectiveness of epidural steroid therapy with and with out ultrasound massage for the management of paraplegia in dogs

# *Review of Literature*

# 2. REVIEW OF LITERATURE

## 2.1. ANATOMY AND BIOMECHANICS OF DOG VERTEBRAL COLUMN

### 2.1.1. Anatomy

The vertebral column of dog is composed of approximately 50 vertebrae according to the formula C7, T13, L7, Cy20. A typical vertebra consists of a body and an arch, between them is the large vertebral foramen with which the foraminae of the other vertebrae forms the canal for the spinal cord. The arch consists of two pedicles attached to the sides of the body and a lamina forming the dorsal and dorsolateral walls of the vertebral canal. The spinous process project dorsally from the lamina while the transverse processes leave the body where it is joined to the arch. The spinal nerves and vessels left the vertebral canal through intervertebral foramina situated between the pedicles of adjacent vertebrae. Movement between the vertebrae is made possible by bilateral articular processes at each end of the vertebrae and the intervertebral disc between the bodies of adjacent vertebrae. (Gage and Horlein, 1974).

A number of ligaments unites the vertebrae and support the column. The tips of the spinous processes are connected by the supraspinous ligaments. The dorsal longitudinal ligament unites the vertebral bodies and lies on the floor of the vertebral canal. The ventral longitudinal ligaments run along the ventral surfaces of the vertebrae. Another ligament of importance is the intercapital, which extends from the head of one rib over the dorsal part of the disc to the head of the opposite rib. This ligament is absent at the 1$^{st}$ pair and the last two pairs of the ribs. It is the thick collagenous bands that usually prevent dorsal disc herniations from the second through tenth thoracic vertebrae. Short ligaments of significance include the interspinous ligament

that connects adjacent transverse processes of the lumbar vertebrae. (Toombs and Waters, 2003).

The basic movements of the vertebral column are: flexion, extension, straightening, or ventral arching of the spine, lateral flexion, extension and rotation. The size of the vertebral canal reflexes quite accurately the size and shape of the contained spinal cord, since there is only a small amount of epidural fat in the dog. The spinal cord is that part of the central nervous system which lies in the vertebral canal from the level of the foramen magnum to the junction of the sixth and seventh lumbar vertebrae. The spinal cord may be divided into segments, each segment being that portion of the spinal cord where fibers in the rootlets of a pair of spinal nerves enter and leave the cord. The spinal nerves are all formed by two roots from the spinal cord: (1) a ventral root/ motor root, made up of fibers carrying impulses from the spinal cord to the effector muscle and gland cells and (2) a dorsal root / sensory root, made up of fibers carrying impulses to the spinal cord (McClure, 1964).

The meninges are the fibrous membranes which surround and protect the spinal cord and the brain. They are composed of three membranes: the duramater, the arachnoid, and the piamater. The spinal duramater consists of only one layer, the meningeal layer. It is separated from the periosteum of the vertebrae by the epidural cavity. The epidural cavity is filled by a semi fluid fat and by the vertebral venous sinuses. The spinal duramater is continuous with the meningeal layer of the cranial duramater at the foramen magnum. The spinal duramater is in the form of a long tube surrounding the spinal cord. It has lateral tubular extensions which cover the spinal nerve roots and extends to the intervertebral foramina. The capillary space between the duramater and the arachnoid is the subdural cavity, which contains a small amount of fluid. The

spinal arachnoid is a thin, transparent tube which envelops the spinal cord and has tubular extensions surrounding the dorsal and ventral spinal nerve roots. The spinal subarachnoid is connected to the spinal piamater by connective tissue trabeculae which pass through the subarachnoid cavity. The subarachnoid cavity is the space between the spinal piamater and the arachnoid membrane. It is filled with CSF which pushes the arachnoid peripherally, and holds it in contact with the spinal duramater. The subarachnoid cavity extends the spinal nerve roots for variable distances and blends with the epineurium of the nerves, prior to fusing with the dura. The CSF serves to cushion and protect the spinal cord. The spinal piamater is a tough, highly vascularised membrane that intimately adheres to the spinal cord and roots of the spinal nerves. It contains all the blood vessels immediately peripheral to the spinal cord (McClure, 1964).

The spinal cord ends at the level of lumbar vertebrae L6 to L7 in the adult dog. A transverse section of the spinal cord reveals a butter- fly shaped central core of gray matter. The wings of the gray matter are referred to as dorsal and ventral horns and consist mostly of cell bodies of neurons. White matter surrounds the gray matter and consists primarily of nerve fibre tracts. The spinal cord is supplied by three arteries that run throughout its length. These are the ventral spinal artery which follows the surface of the ventral fissure of the cord and supplies the gray and white matter. And two paired dorsolateral spinal arteries which run close to the furrow from which the dorsal roots of the spinal nerves arise. In addition there are vertebral venous plexuses which consist of paired channels and also run the entire length of the vertebral column ventral to the cord. (Campoy, 2004).

Shell (1996a) stated that limb weakness caused by a problem in the upper or lower motor neuron system. Cell bodies or nuclei for the upper motor neuron system were with in the brain and were responsible for initiating

voluntary movement. Axons from these cell bodies form tracts (rubrospinal, corticospinal, and vestibulospinal tracts) that descend from the brain to synapse with interneurons in the spinal cord. Interneuronal axons then synapse with the large (alpha) motor neurons in the ventral gray matter of the spinal cord. These large motor neurons were the cell bodies of origin for the lower motor neuron system, which mediate spinal reflexes. Collections of lower motor neurons in the cervical and lumbar intumescences were necessary to innervate limb muscles. Axons from these large motor neurons form the ventral nerve roots, the spinal nerves, and ultimately the peripheral nerves that innervate the limb muscles.

### 2.1.2. Biomechanics

Bray and Burbridge (1998) reviewed that intervertebral disc disease was recorded most frequently at the most mobile region of the vertebral column in all animals. The intervertebral disc subjected to five possible loading conditions: axial compression, tension, bending, shear, and torsion. When a compressive load is passed through the normal intervertebral disc, hydraulic pressure is generated with in the gelatinous nucleus pulposus. This is radiated in all directions and absorbed by the annulus fibrosus. The compressive force is converted into tensile force within the annular fibers which resist collapse of the intervertebral disc space. The tensile force was largest in the outer layers of the annular fibrosus and most injuries are sustained in this area.

## 2.2. PARAPLEGIA

### 2.2.1. Etiology

McKee (1990) observed that 9.8 pre cent of 51 dogs and cats sustained spinal trauma due to unknown reasons. The author also reported that 58.82 per cent of spinal trauma in dogs and cats occurred due to automobile accidents.

Lobetti (1994) reported a case of subarachnoid abscess as a complication of discospondylitis in a dog. It was opined that haematogenous spread of bacteria to the intervertebral disc was the main cause of discospondylitis.

Shell (1996b) reviewed the differential diagnosis for acute onset paraparesis. The etiology for acute onset were commonly intervertebral disc herniation, trauma, and fibrocartilagenous embolism and less commonly discospondylitis, neoplasia, distemper myelitis, rickettsial meningomyelitis, *mycosis, parasitic migraton and rabies myelitis. The author further opined that* trauma was commonly caused by motorized vehicles, heavy falling objects, gunshot, and bites during dog fights.

Shell (1996c) described the differential diagnosis of progressive onset paralysis. The causes of progressive onset were type II intervertebral disc herniation, degenerative myelopathy, discospondylitis, degenerative lumbosacral stenosis, neoplasia affecting the spinal cord, and meningomyelitis.

Dhaliwal and Mirakhur (2001) stated that spinal cord injury was produced by both external and internal factors. Gunshot injuries, falls, fight with other animals, automobile accidents etc were the external factors. The internal factors included intervertebral disc extrusion, pathologic fractures, congenital vertebral anomalies and instability.

Kinzel *et al.* (2005) noted intervertebral disc disease as the most common cause of neurological trauma in dogs.

### 2.2.2 Incidence

#### *2.2.2.1. Breed*

Scott (1997), in a study of thoracolumbar disc disease in 40 dogs reported that, eighteen (45 per cent) were Miniature dachshunds and most of the others were small chondrodystrophoid breeds or their crossbreeds.

According to Smith *et al.* (1997) Dachshunds represented 66 per cent of all dogs with Hansen type I disc extrusion.

Wheeler (1997) opined that cervical and thoracic disc disease was a frequent disorder of small breeds of dog, particularly those with chondrodystrophoid characteristics.

Fitch *et al.* (2000) reported that Dachshunds were most commonly affected with cervical intervertebral disc disease, followed by mixed breed-dogs, Pekingese, Poodles, Cocker spaniels and Yorkshire terriers.

McKee (2000) reported that chondrodystrophoid breeds, spaniels, terriers and their crosses were more affected with intervertebral disc herniation. Dobermans, German Shepherd dogs, Labrador Retrievers and Rotweilers were typical of the large breeds affected.

Cherrone *et al.* (2004) in a case study reported the occurrence of Hansen type I cervical intervertebral disc extrusion in medium to large non-chondrodystrophoid breeds.

Mayhew *et al.* (2004) conducted a study on thoracolumbar intervertebral disc herniation in dogs and reported Dachshund as the most common breed affected.

Ranganath *et al.* (2006) opined that Hansen type I disc herniation was commonly seen in chondrodystrophoid breeds like Dachshund and Pekingese and Hansen type II disc herniation in non- chondrodystrophoid breeds like Doberman and Great Dane.

*2.2.2.2. Age*

Shell (1996b) reported that congenital, hereditary, and inflammatory disorders were more common in younger dogs.

Scott (1997) reported that the age of the dogs affected with thoracolumbar disc disease ranged from two to fourteen years (mean 6.5).

The average age of dogs affected with Hansen type I disc extrusion was five years as reported by Smith *et al.* (1997).

Wheeler (1997) opined that most patients with cervical disc disease were two years old or more, with a mean age of six years. Thoracolumbar disc diseases reaching a peak incidence between three and six years of age. Dogs of non-chondrodystrophoid breeds, when affected, tend to be middle aged or older.

McKee (2000) reported that disc disease was uncommon in dogs with less than two years of age.

Mayhew *et al.* (2004) in a study of thoracolumbar disc herniation revealed that middle aged dogs were most commonly affected with intervertebral disc disease.

*2.2.2.3. Sex*

Scott (1997) conducted a study of thoracolumbar disc disease in 40 dogs. The author reported that of the affected dogs, 26 were males and 14 were females.

Smith *et al.* (1997) recorded the details of 40 dogs with Hansen type I disc extrusion in which 26 dogs were females and 24 were males.

Wheeler (1997) reported no sex predilection in the occurrence of intervertebral disc herniation in dogs.

According to Mayhew *et al.* (2004) observed that disc herniation was commonly seen in females than in males. Of the 229 dogs studied, 120 were females and 109 were males.

*2.2.2.4. Site of lesion.*

Shell and Schueler (1996) opined that about 80 per cent of acute intervertebral disc extrusion in chondrodystrophoid breeds occurred between T11 through L3 vertebrae.

Scott (1997) opined that the commonest site of disc extrusion was T12 to T13 followed by T13 to L3 in dachshunds and chondrodystrophoid breeds. .

Smith *et al.* (1997) in a study of intervertebral disc extrusion reported that the most common site affected was T12 to T13 with T13 to L1 the next most commonly affected site.

According to Wheeler (1997) over half of all thoracolumbar disc lesions occurred at the T12/13 and T13/L1 discs, and over three-quarters occurred between T11/12 and L1 and L2 inclusive.

Lanz *et al.* (2000) stated that fracture / luxations usually occurred near the junction of a mobile and an immobile vertebral segment. These areas included lumbosacral, thoracolumbar, cervicothoracic, and atlantoaxial and atlanto-occipital junctions.

Mayhew *et al.* (2004) in a study of thoracolumbar intervertebral disc disease found out that T12 – T13 was the most common site affected.

### 2.2.3. Clinical signs and Symptoms

Shell (1996a) stated that upper motor neurons and their axons have an inhibitory influence on the lower motor neurons that maintains normal muscle tone and normal spinal reflexes. When upper motor neuron or their axons were damaged spinal reflexes were no longer inhibited or controlled. Thus reflexes become exaggerated or hyperreflexic. If the lower motor neurons or their processes (peripheral nerves) were injured, spinal reflexes were either reduced (hyporeflexic) or absent (areflexic). Since the lower motor neuron system was vital to skeletal muscles, damage to this system resulted in rapid and severe skeletal muscle atrophy, called neurogenic atrophy. Disuse atrophy was often seen with injury to the upper motor neuron system, but the onset was not as rapid as that of neurogenic atrophy.

Shell (1996b) opined that hyperpathia was most commonly associated with disc herniation, trauma, bony or meningeal tumours, and discospondylitis

Shell and Schueler (1996) opined that an upper motor neuron lesion proximal to the sacral spinal cord segments caused increased tone to the urinary sphinctor muscle and resulted in a large, firm urinary bladder.

Wheeler (1997) described the clinical symptoms of thoracolumbar disc disease. Spinal hyperesthesia and neurological deficits were seen in the pelvic

limbs. With more severe lesions urinary dysfunction might be present. The dog might show kyphosis and reluctance to run or jump, and discomfort could usually be elicited by deep palpation in the thoracolumbar region. Neurological deficits ranged from mild ataxia and paresis to paraplegia which might be accompanied by depressed or absent deep pain sensation caudal to the lesion.

Bergman *et al.* (2000) explained the Schiff-Sherrington posture, which was seen in severe cases of thoracolumbar spinal cord trauma in dogs. The posture was described as rigid forelimb extension with paraplegia of the pelvic limbs. It was thought to result from injury to the border cells or to the border cell pathways, which were located on the dorsolateral aspect of the ventral gray column from spinal cord segments L1-L7. These cells inhibit forelimb extension, and their disruption or injury causes the clinical signs associated with the Schiff- Sherrington syndrome. Such clinical signs might last for one or two weeks.

Apparent spinal pain associated with degenerative disc lesions may result from compression or ischemia of the meninges and / or spinal nerve roots. In addition, extruded nuclear material may incite an inflammatory reaction which may cause pain and result in fibrous adhesion between the duramatter and the disc material. Pain could also be produced from stimulation of sensory nerve endings in the annulus fibrosus and dorsal longitudinal ligament (McKee, 2000).

Mayhew *et al.* (2002) stated that cauda equina compression resulted in lumbosacral pain, paresis, lameness, urinary and faecal incontinence and dysesthesias (abnormalities of skin sensation). Disease of the cauda equina might be due to idiopathic stenosis, discospondylitis, trauma, neoplasia, inflammatory disease, vascular compromise, and congenital abnormalities.

Degenerative lumbosacral stenosis was the most common disease of the cauda equina in large breed dogs.

## 2.3. PATHOPHYSIOLOGY

Janssens (1991) stated that the main pathological phenomena that occurred in acute spinal cord trauma were rupture of cell membranes and microvessels. Microvessel rupture leads to free radical formation to thrombocyte aggregation and vessel obstruction. Cell oxygenation becomes impaired and eventually cell death occurs. Cell membrane rupture leads to $Ca^{2+}$ influx, neurotoxic firing with glutamate release and the activation of the arachidonic acid cascade. This results in production of thromboxane, prostaglandins, and prostacyclins. These actuate thrombocyte aggregation, serotonin release, vasoconstriction, and oedema and lead to cell anoxia and cell death. All processes were completed within 24 hours after which regeneration occurs.

Coughlan (1993) opined that secondary injury mechanism in spinal cord trauma mainly associated with ischemia, raised intraneuronal calcium and free radical induced lipid peroxidation. The derangements occurring in cell metabolism due to trauma or ischemia lead to increased production of oxygen free radicals, which could overwhelm natural defenses. The double bonds of membrane phospholipids were vulnerable to free radical attack, resulting in membrane disruption and the formation of lipid peroxides.

Bergman *et al.* (2000) described the mechanisms of spinal cord injury, the susceptibility of different areas of the spinal cord to injury, the prognosis for recovery of neurologic function, and the clinical syndrome associated with spinal cord trauma. Acute spinal cord trauma resulted in injuries by two mechanisms: the primary injury followed by the secondary injury. Primary

injury involved the actual forces that cause the mechanical injury, such as compression, shear, laceration, bending, and distraction. There were three categories of secondary injury: systemic, extra cellular, and intracellular.

## 2.4. DIAGNOSIS

### 2.4.1. History

Griffith (1982) stated that majority of animals with spinal disease presented with locomotor disturbances and / or spinal pain. In both situations a detailed clinical history was essential. This included such details as the duration, rate of onset and progression of signs, presence of bladder control, as indicated by the ability to pass urine in a stream or incontinence, and the general present and past health of the animal. The author opined that in many instances the history was the clue to diagnosis.

McKee (2000) described various historical features suggestive of spinal pain such as reluctance to jump or climb, arching of the back, low head carriage, yelping, reluctance to lower heads to eat, reluctance to look upwards, reluctance to turn in tight circles, tense neck, back, and / or abdomen, restlessness and panting.

### 2.4.2. Physical examination

Fossum (2002) mentioned about the importance of physical examination in neurologic disease. According to the authors a complete general physical examination should be performed in patients with possible neurologic disease. Some metabolic, cardiovascular, and musculoskeletal disorders mimic the clinical appearance of neurologic disorders. (e.g., Addison's disease, toxic pyometra, cardiovascular insufficiency, bilateral cruciate rupture).

Griffith (1982) opined that non-neurological disease should be considered before proceeding to a more detailed neurological examination. Pelvic fractures, bilateral limb fractures, or ruptured anterior cruciate ligaments could produce signs superficially suggestive of paraplegia.

### 2.4.3. Neurological examination

Shell (1996a) stated that paraparetic dogs with changes in mentation or cranial nerves were more likely to have a multifocal or diffuse disease rather than disc herniation. In the absence of cranial nerve signs, the lesions could be located below the foramen magnum.

McKee (2000) opined that the two main objectives of neurological examination were accurate localization of the lesion and assessment of the severity of the spinal cord injury. Assessment of motor function, proprioception, spinal reflex arcs, and spinal pain aided localization of the lesion. Spinal cord injury might be assessed according to the degree of motor and bladder dysfunction and the presence or absence of deep pain perception caudal to the lesion.

Lorenz and Kornegay (2004) described in detail the neurological evaluation of dogs. The authors stated that the examination based on mental status, posture, gait, postural reactions and spinal reflexes was sufficient in assessing the degree of damage to the spinal cord.

#### *2.4.3.1. Gait*

De Lahunta (1997) opined that an early sign in dogs with ascending myelomalasia associated with an acute intervertebral disc extrusion might be a hesitant, stumbling, awkward gait in the thoracic limbs. The severity of

advanced pelvic limb weakness was evaluated best by holding the patient suspended at the base of the tail and observing its gait.

Jeffery (2001) stated that a clumsy gait with exaggerated stepping was suggestive of a lesion in the cerebellum or its associated circuitry.

#### *2.4.3.2. Posture*

Shell (1996a) considered weakness of voluntary movements of pelvic limbs as paraparesis where as paraplegia was defined as complete absence of voluntary movements.

Jeffery (2001) defined paresis as weakness in voluntary movement and ataxia as incordination in a limb or the trunk.

#### *2.4.3.3. Postural reactions*

Shell (1996a) stated that if postural reactions and spinal reflexes were normal for the thoracic limb, the abnormality could be below T2 or T3 spinal cord segments.

Jeffery (2001) reported that there were two types of placing reactions: visual and tactile. Visual placing reaction assessed the integrity of the visual system, cerebellum and its pathways and motor control system. The second one was a test of fine tactile sensation and the motor systems that control movement.

#### *2.4.3.4. Spinal reflex*

Shell (1996a) opined that evaluation of spinal reflex was best performed with the animal in lateral recumbency and in a relaxed state. The grading scale used for spinal reflex was as follows:

0 = absent

1 or 1+ = depressed

2 or 2+ = normal

3 or 3+ = hyperactive or exaggerated

4 or 4+ = clonus (a sustained after contraction or quivering)

The author also stated that patellar reflex was the most reliable spinal reflex. This reflex was mediated by the femoral nerve, whose cell bodies of origin were found in spinal cord segments L4 through L6, located in L3 and L4 vertebrae. An exaggerated or clonic patellar reflex usually indicates an upper motor neuron lesion (*i.e.* above the L4 cord segments). a reduced or depressed patellar reflex indicate a lower motor neuron lesion in the quadriceps muscle, peripheral nerve, nerve roots, or cell bodies in cord segments L4 through L6. He opined that an exaggerated patellar reflex could also be observed if the sciatic nerve or its cell bodies at cord segments L6 to S1 were injured. This exaggerated patellar reflex was called a false localizing sign. The sciatic nerve and its branches mediate the cranial tibial muscle, gastrocnemius muscle, sciatic and flexor reflexes. Cell bodies for this nerve were located at L6 through S1 cord segments near L4 and L5 vertebrae. The most reliable sign of sciatic nerve damage was a reduced or absent flexor response. Increased or clonic reflexes mediated by the sciatic nerve were caused by a lesion proximal to the L6 cord segments.

Jeffery (2001) stated that the panniculus reflex can be impaired either because of a lesion affecting the afferent input through the sensory branches of the spinal nerves from T1-L1 or a lesion affecting the efferent output originating in spinal cord segments C8-T1.

### *2.4.3.5. Lesion localization*

Braund (1995) described about four spinal cord syndromes for localizing spinal cord lesions. Lumbosacral syndrome produced by lesions involving spinal cord segments L4 and L5 through S1 to S3. Spinal cord segments between T3 and L3 produced the thoracolumbar syndrome and C1-C5 produced the cervical syndrome. The cervicothoracic spinal cord segments that extend from C6 through T2 produced cervicothoracic syndrome.

Shell (1996a) divided the neurological examination into sections such as head and cranial nerve signs, thoracic limb signs, pelvic limb signs and tail signs. If head signs, (e.g. seizure activity, head tremors, and behavioral changes) and cranial nerve signs (e.g. nystagmus, facial weakness, and dilated pupil) were absent, the abnormality was most likely located below the foramen magnum. If the postural reactions and spinal reflexes were normal for the thoracic limbs, the abnormality must be below T2 or T3. By evaluating pelvic limb spinal reflexes, the abnormality could be localized to a site between T3 and L3 or between L4 and S1 cord segments. Evaluation of anal reflex and tail tone determined whether the S2 through coccygeal cord segments were affected.

### 2.4.4. Haematology

Moore and Withrow (1982) noted that loss of blood into the gastrointestinal tract could probably cause anemia in spinal patients.

Calvert and Cornelius (1990a) stated that exogenous glucocorticoids raised the number of polymorphonuclear leukocytes and lower the number of lymphocytes, eosinophils, and basophils. Both an increase in the rate of release of polymorphonuclear leukocytes from the bone marrow into the blood and a diminished rate of their removal were thought to be responsible for the steroid

induced granulocytosis. Lymphopenia resulted from the action of glucocorticoids on lymphoid tissue.

Shell and Schueler (1996) described the diagnosis for six cases of posterior paralysis. The complete blood cell count taken in all six animals at the time of clinical examination were found to be in the normal range.

Cohn (1997) reported that corticosteroid administration produced stress leukogram. Mature neutrophilia was a characteristic component of stress leukogram. Lymphopenia was another classic component. Animals could be divided into steroid-sensitive and steroid-resistant species. In steroid-resistant species lymphopenia resulted from a redistribution of circulating lymphocytes. Blood concentration of eosinophils and monocytes were altered. In dogs, steroid induced variable degree of monocytosis.

Kent (2004) stated that glucocorticoid administration resulted in inhibition of leukocyte migration to the site of inflammation, decreased leukocyte activation at the site of inflammation, and decreased secretion of cytokines that contribute to the inflammatory processes. They inhibited interleukin, specifically IL-1, IL-2, and IL-6 which affect lymphocyte differentiation and proliferation thus resulted in reduction of circulating lymphocytes.

### 2.4.5. Serum biochemistry

Moore and Withrow (1982) reported an increase in serum Alkaline phosphatase (ALP) activity in dogs having intervertebral disc disease treated with dexamethasone.

Huang *et al.* (1999) studied about 28 dogs with iatrogenic hyperadrenocortisism.The most common clinical signs were cutaneous lesions,

polydipsia, polyuria, and lethargy. The most common predominant findings on biochemical profile were elevated Alkaline phosphatase, and Alanine amino transferase, hypercholesterolemia, and elevated Aspartate amino transferase and elevated triglycerides.

Ginel *et al.* (2002) studied the effect and the duration of increased serum Alkaline phosphatase (ALP) activities induced by therapeutic doses of prednisone, methyl prednisone acetate and dexamethasone. Significant increase in serum Alkaline phosphatase activity was observed after methyl prednisone acetate administration for three weeks and returned to basal levels by 28 days.

Chandy (2006) in a study of traumatic paraplegia in dogs reported that the serum calcium and phosphorous values were found to be within the normal range at the time of presentation and after treatment with ultrasound therapy and epidural administration of methyl prednisolone acetate.

### 2.4.6. Radiography

#### *2.4.6.1. Survey radiography*

Lanz *et al.* (2000) described the three compartment theory for evaluation of instability of vertebral bodies. The dorsal compartment contains the articular facets, laminae, pedicles, spinous processes, and supporting ligamentous structures. The middle compartment contains the dorsal longitudinal ligament, dorsal annulus fibrosus, and dorsal vertebral body. The ventral compartment contains the remainder of the vertebral body, lateral and ventral aspects of the annulus fibrosus, nucleus pulposus, and ventral longitudinal ligament.

According to McKee (2000), significant findings on survey radiographs included narrowing of the intervertebral disc space, decreased size of intervertebral foramen, reduced joint space between the dorsal articular facets, and mineralized material in the vertebral canal. Mineralized material in the canal might be visualized as a discrete mass or a hazy increase in opacity of the intervertebral foramen.

Somerville *et al.* (2001) reported that the accuracy of survey radiographs to localize the correct site of disc protrusion ranged from 53-67 per cent. There was 58 per cent accuracy for single compressive lesions and 70 per cent for multiple disc extrusions. Calcification of disc material in the intervertebral foramen or opacity in the vertebral canal and narrowing of disk space were the radiographic changes they selected for evaluation.

Mayhew *et al.* (2004) studied the recurrence of clinical signs associated with thoracolumbar intervertebral disc herniation in dogs. The survey radiography was evaluated based on the characteristics such as disc space opacity- calcification (present or absent), disc space size (normal or narrowed), disc space wedging (present or absent), facet joint degenerative joint disease (present or absent), facet joint size (normal or increased) foramen opacity (normal or increased), foramen size (normal or decreased); and spondylosis (present or absent).

### *2.4.6.2. Myelography*

Wright (1984) described various indications of myelography as paresis, ataxia, lumbar pain, paraplegia, forelimb knuckling, quadriparesis, cervical pain, and quadriplegia. The contrast medium of choice was iohexol at the rate of 0.15-0.3ml/kg body weight (300mgI/ml).According to the author the interpretation of normal myelogram consisted of the following points:

(1) The width of the subarachnoid space was greatest at C2.

(2) Slight indentations of the ventral contrast column, at C2-3 in particular, and also C3-4 and C4-5, were normal phenomena.

(3) The ventral contrast column was quite a distance away from the floor of the neural canal from cranial C5 to C7.

(4) In the lumbar area the ventral columns was slightly elevated, or show a slight break at the intervertebral disc.

(5) Occasionally a small area of increased density occurred around the origin of the nerve roots in the lumbar area.

(6) The spinal cord was seen to begin to narrow at caudal L5 and tapered rapidly to end in the first sacral segment.

(7) Streaking of the column within L6 and L7 represented the cauda equina.

The author also described the following points as diagnostic signs on the myelogram,

(1) Obstruction – partial or total.

(2) Dorsal uplifting of the ventral column only, or both dorsal and ventral columns together.

(3) Stricture of the column, dorsally and ventrally or laterally.

(4) Deviation to the right or left.

(5) A break in the column – dorsal, ventral, or lateral.

(6) Narrowing of the column – dorsal, ventral, or lateral.

(7) A filling defect, i.e., failure of the contrast medium to fill part of or an area on serial films.

(8) Expansion of the column – dorsally.

Puglisi *et al.* (1986) compared the contrast agents metrizamide and iohexol for cisternal myelography in dogs. The study concluded that (1) iohexol was less epileptogenic than metrizamide, (2) isotonic iohexol was similar to isotonic metrizamide in diagnostic quality (3) metrizamide caused a significantly higher increase in CSF micro protein concentration 24 hours after intrathecal injection..

Lewis and Hosgood (1992) reported seizure as the main complication associated with Iohexol myelography in dogs. Seizures were significantly more prevalent when body weight was $\geq$ 29kg, when $\geq$ 2 injections of contrast medium were administered or when two injections of contrast medium were given at the cisterna magna. Other adverse effects reported were bradycardia, vomiting, transient apnoea, and exacerbation of neurologic abnormalities. They opined that hyperosmolality and inherent chemotoxicity were the factors contribute to a medium' neurotoxicity. Iohexol has large, bulky, hydrophilic side chains to shield its iodine atoms, therefore, reducing its neurotoxicity.

Widmer *et al.* (1992) studied the CSF changes after iopamidol and metrizamide myelography. Iopamidol (200mgI/ml) and metrizamide (170mgI/ml) were administered by cerebellomedullary injection at dosage of 0.45ml/kg body weight. In both groups post myelographic CSF changes included high specific gravity, Pandy score, protein concentration, and WBC count. The authors concluded that iopamidol was less irritating to the leptomeninges and was less neurotoxic than mertizamide.

Fatone *et al.* (1997) opined that the most common responses during administration of contrast medium were increase in cardiac and respiratory rate. Fasciculation of temporal and facial muscles and rigidity of the cervical musculature noticed during recovery from anaesthesia. Side effects were higher with cervical administration than with lumbar administration.

Scott (1997) performed plain radiography and myelography in dogs with thoracolumbar disc disease under general anaesthesia. For myelography, iohexol (300mgI/ml) at the rate of 0.3 to 0.5 ml /kg body weight of the animal was injected into the ventral subarachnoid space at L5-L6 junction. The author was able to accurately localize the site of disc involvement in all 40 dogs with thoracolumbar disc disease by myelography. However, lateralization of the lesion was possible only in 18 dogs (45 per cent).

Penderis *et al.* (1999) reported subdural injection of contrast medium as a complication of myelography. Contrast medium had a smooth border on dorsal aspect and an undulating border on spinal cord aspect.

McKee (2000) stated that myelography mainly indicated for confirmation of diagnosis, detection of multiple disc lesions, assessment of the degree of spinal cord compression and characterization of the lesion.

Kaur and Singh (2004) studied iohexol myelography in 13 dogs suffering from various spinal affections.Cisternal myelography was performed using 0.45ml/kg body weight of iohexol (300mgI/ml). The contrast agent readily mixed with CSF and reached up to cauda equina within 5-10 minutes after injection. In cases with partial compression, there was partial impairment to the flow of the agent, while complete obstruction to the flow indicated complete severance of the spinal cord. The author emphasized that the hydration status of the animal should be checked and dehydration should be

corrected before undertaking myelography. Dehydration decreased the renal clearance of the contrast agent, thus enhancing adverse effects of the agent on cardiovascular system.

Kumar *et al.* (2004) conducted myelographic studies on 12 healthy dogs using iohexol and iopamidol. Both contrast agents were administered at the rate of 0.3ml/kg body weight (300mgI/ml) by cisternal puncture. The lateral and ventrodorsal myelograms were obtained at 5,15,30,60, and 90 minutes after contrast medium injection.Myelograms appeared to be uniform and of superior radiographic quality and were of diagnostic value up to 60 minutes in both groups.

Devecioglu *et al.* (2006) conducted iohexol and iopamidol myelography in 12 dogs. Iohexol resulted in higher image quality than iopamidol. They stated that iohexol was a water soluble, non- ionic monomeric and an iodinated compound. It had the lowest chemotoxicity among other monomeric non-ionic, low-osmolarity agents. Iohexol was the least effective on animal behavior after subarachnoid injection compared to other agents.

Ranganath *et al.* (2006) reported that myelography was effective for accurate determination of disc material in cervical and thoracolumbar disc herniation and the lateral myelography was more beneficial in delineating the lesion. Narrowing and dorsal deviation of ventral contrast column at the site of protrusion was the most common myelographic finding in intervertebral disc disease. Blockage for flow of contrast media caudal to the obstruction was seen in case of acute onset spinal cord swelling

Riyaz *et al.* (2006) described the use of cisternal myelography to diagnose spinal lesions, other than fracture or dislocation, causing constriction of spinal canal. The contrast material used was iohexol (300mgI/ml) at the dose

rate of 0.3ml / kg body weight. For myelographic evaluation of spinal lesions in dogs they used diazepam as preanaesthetic at the rate of 0.5mg/kg body weight.Anaesthesia was induced and maintained with thiopentone sodium at the rate of 25mg/kg body weight, intravenously.

Bos *et al.* (2007) suggested that ventrodorsal myelographic view was useful in predicting the side of extruded disc material in dogs with thoracolumbar intervertebral disc extrusion. Dogs with gap in the contrast columns of unequal lengths, disc material was found to be located on the side with the shorter, rather than the longer, gap, a phenomenon described as paradoxical contrast obstruction. This occurred because lateralized extruded disc material displaced the spinal cord toward the opposite side, compressing the contra lateral subarachnoid space over a greater distance than the length of the extrusion itself.

In a case study, Carstens and Kitshoff (2007) reported epidural leakage of contrast medium as a complication of myelography. Increased permeability due to meningeal pathology resulted in leakage and made the accurate evaluation of subarachnoid space impossible. It was opined that incorrect needle placement during contrast injection may also leads to this condition. The authors used L5-L6 intervertebral site for lumbar myelography.

### *2.4.6.3. Other imaging techniques*

Barthez *et al.* (1994) used discography and epidurography for evaluation of cauda equina syndrome in 21 dogs. On survey radiographs, the most common findings were spondylosis, malalignment of the sacrum to the last lumbar vertebrae, collapse of the lumbosacral disc space, stenosis of the vertebral canal at the lumbosacral junction, and transitional vertebral segments. Discograms were performed by spinal needle introduced in a saggital plane into

the lumbosacral disc space under fluoroscopic guidance. Epidurograms were performed following discography by injecting contrast medium after repositioning the tip of the needle into the ventral epidural space at the level of the lumbosacral junction. Discography was considered of diagnostic quality in 90 per cent of the dogs, showing disc protrusion in 67 per cent. Epidurography was of diagnostic quality in 100 per cent dogs, showing abnormal findings in 78 per cent. Combination of survey radiographs, discoepidurography was correctly positive in 89 per cent. It was concluded that discography associated with epidurography was a valuable procedure for evaluation of the lumbosacral junction in dogs.

Lanz *et al.* (2000) opined that magnetic resonance imaging (MRI) offered superior soft tissue definition as compared with computed tomography (CT), which is helpful for evaluation of spinal cord.

McKee (2000) stated that MRI might provide valuable information in lumbosacral and foraminal disc cases. Nerve root compression at intervertebral foramina might be evident on transverse views, a diagnosis that can not be made myelographically.

George (2002) evaluated the comparative efficacy of ascending coccygeal venography and epidurography in locating the site and type of lesions in dogs suffering from paraplegia. The author opined that epidurography possessed a distinct advantage over ascending coccygeal venography in consistency and technical and diagnostic feasibility.

Dickinson (2005) stated that a variety of imaging modalities were available for the imaging of small animal patients with neurological disease like computed tomography (CT), magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), magnetic imaging spectroscopy (MRS),

positive emission tomography (PET), angiography, scintigraphy, and ultrasonography.MRI provided better soft tissue resolution than CT . MRI allowed transverse images of spinal cord which was not possible with myelography. Structures beyond the subarachnoid space such as nerve roots and vertebral foramina could also be imaged.

Costa *et al.* (2006) opined that MRI was more accurate in predicting the site, severity, and nature of spinal cord compression when compared to myelography.

**2.4.7. CSF Analysis**

Griffiths (1982) stated that for traumatic injuries of the spinal cord like fracture / dislocation, traumatic disc, cord concussion, and vertebral collapse CSF analysis was not usually performed for diagnostic purposes. However, useful information for the diagnosis of inflammatory diseases could be obtained. Although a multitude of tests were possible, total and differential cell counts and estimation of total protein levels and a Pandy test were the most useful examinations.

Thomson *et al.* (1989) studied 118 CSF samples from dogs with intervertebral disc disease. The fluid was removed from the cerebellomedullay cistern, lumbar cistern or both. The samples were analyzed within 30 minutes of collection, as cells in the fluid degenerated rapidly. Changes in CSF were identified in 84.7 per cent of the lumbar samples, compared with only 37.1 per cent of the cerebellomedullary samples. More pronounced pleocytosis and protein level increases were seen in dogs with acute and clinically severe lesions. This result indicated that marked protein and white blood cell count elevations could occur in association with intervertebral disc extrusion.

Wheeler (1997) stated that analysis of CSF was useful to eliminate inflammatory disease. Results of CSF analysis might be abnormal in disc disease, but elevations of protein and cells in these patients were usually mild.

McKee (2000) stressed that CSF analysis should be performed prior to myelography to rule out inflammatory diseases. Normal CSF was clear and colorless. Subarachnoid haemorrhage secondary to intervertebral disc extrusion might result in pink discoloration or xanthochromia if more than 48 hours had elapsed following haemorrhage. CSF analysis should include a quantitative estimation of protein content. Cytological studies should be completed within 30 minutes of fluid collection since the cells degenerate rapidly. A total white blood cell count should be performed on non- concentrated fluid and cell morphology and a differential cell count performed in a concentrated sample. Degenerative disc disease might cause a mild elevation of cerebrospinal fluid protein.

## 2.5. NEUROLOGICAL GRADING

Griffiths (1982) proposed a simple scheme for grading the severity of spinal cord damage as follows:

Group 1 - paretic.

Group 2 - paraplegic, intact bladder control and pain sensation.

Group 3 - paraplegic and loss of bladder control, some pain sensation present.

Group 4 - paraplegic with loss of bladder control and pain sensation.

Wheeler (1997) graded dogs with thoracolumbar disc disease as follows:

Grade 1 – pain only

Grade 2 – ataxia, conscious proprioceptive deficit, paraparesis

Grade 3 – paraplegia

Grade 4 – paraplegia with urinary retention and overflow

Grade 5 – paraplegia, urinary retention and overflow and loss of deep pain sensation

The author reported that the prognosis for a functional recovery was very good for dogs with grade 1,2,3 when compared to 4.

McKee (2000) graded dogs with thoracolumbar disc disease according to the degree of neurological dysfunction as follows:

Grade 1 : spinal pain, no paresis.

Grade 2 : ambulatory parapresis. (Mild, moderate or severe)

Grade 3 : non- ambulatory paraparesis

Grade 4 : paraplegia

Grade 5A: As for grade 4, plus absence of superficial pain perception.

Grade 5B: as for grade 5A, plus absence of deep pain perception.

Grade 5C: as for grade 5B, plus evidence of progressive myelomalacia.

Tartarelli *et al.* (2005) assigned different grades to dogs with thoracolumbar disc extrusion as follows:

Grade I – spinal hyperesthesia only

Grade II – ambulatory paraparesis

Grade III – non-ambulatory paraparesis

Grade IV – paraplegia

Grade V – paraplegia with urinary incontinence

Grade VI – paraplegia, urinary incontinence and absence of deep pain sensation

## 2.6. PROGNOSIS

Shell (1996a) stated that the most important prognostic tool in the neurologic examination of a paralyzed patient was the assessment of deep pain perception. It was performed by applying a painful stimulus to the digits or tail. When haemostats or forceps were used to pinch the toes, the animals withdraw the pelvic limb if sciatic nerve was intact.

Dhupa *et al.* (1999) opined that complete sensorimotor deficit was a significant predictor of poor outcome in dogs with caudal lumbar disc herniation.

Bergman *et al.* (2000) stated that tissues of the spinal cord vary in their susceptibility to injury because they differ in their need for oxygen. Gray matter was considerably more susceptible to ischemic injury than white matter because of its increased metabolic needs. So injuries to the cervical or lumbar intumescences, which contain a larger amount of gray matter, had a more guarded prognosis.

Lanz *et al.* (2000) opined that the prognosis was guarded in case of animals with no deep pain perception. But animals with loss of deep pain

perception secondary to disc herniation have 50 per cent chance of recovery after surgical intervention.

Ruddle *et al.* (2006) reported that intervertebral disc herniation resulted in either lower motor neuron (UMN) or upper motor neuron (LMN) lesions. Disc disease was the most common cause of caudal paresis in dogs. Upper motor neuron signs in the rear limbs, which were thought to have a more favourable prognosis compared to the lower motor neuron signs created by herniation of disc material. Due to potential disruption of the lumbar intumescences, lower motor neuron signs have been reported as having a less favourable prognosis.

## 2.7. MANAGEMENT OF SPINAL CORD COMPRESSION

### 2.7.1. Medical management

Moore and Withrow (1982) noted that there was highest prevalence of gastrointestinal hemorrhage and pancreatitis in dogs that received dexamethasone treatment after spinal surgery

Calvert and Cornelius (1990a) reported that glucocorticoids suppressed inflammation by first inhibiting edema, fibrin deposition, capillary dilation, migration of leukocytes and phagocytic activity. Later in the processes, they inhibit capillary and fibroblast proliferation, collagen deposition, and cicatrisation. They might inhibit the release of arachidonic acid from phospholipids, thus decreasing the formation of prostaglandins, leukotriens, and thromboxanes, all which were important in chemotaxis and inflammation.

Calvert and Cornelius (1990b) described in detail the indications of corticosteroid hormone therapy in allergic conditions, immune mediated diseases, neoplasia, and metabolic disorders. Corticosteroids were used to treat

brain and spinal cord disorders, usually those associated with trauma. Oedema and inflammation accompanying neoplasia or infection have also been treated with corticosteroids.

Calvert and Cornelius (1990c) described in detail, the side effects of glucocorticoid hormone therapy. Two categories of toxic effects could be observed with the therapeutic use of glucocorticoid hormones- those resulting from withdrawal and those resulting from prolonged use of large doses. A sudden withdrawal of glucocorticoids after prolonged therapy resulted in acute adrenal insufficiency. The characteristic signs with glucocorticoid withdrawal were malaise, vomiting, anorexia, and weakness. Short and intermediate acting glucocorticoids administered to dogs and cats at the appropriate doses for one to two weeks did not produce prolonged suppression of hypothalamo-pituitary-adrenal (HPA) axis. When daily treatment lasted more than two weeks, it should be discontinued by reducing the dosage by 50 per cent every five to seven days over a two to three weeks period. .

Calvert and Cornelius (1990d) reported that methyl prednisolone differs from prednisolone by the addition of a methyl group to C-6. This addition slightly boosts the anti-inflammatory activity and reduced the sodium retaining potential relative to prednisolone. The acetate and acetonide esters released the glucocorticoid slowly and had a duration of action ranging from days to weeks. The recommended dosage for methyl prednisolone acetate was 0.5 to 2mg/kg body weight.

Cornelius and Calvert (1991) reported that long term corticosteroid therapy at pharmacologic doses produced suppression of hypothalamic-pituitary- adrenal axis (HPA) and the abrupt withdrawal of exogenous corticosteroids resulted in signs of glucocorticoid insufficiency.

Coughlan (1993) emphasized the importance of early treatment of a patient with spinal cord injury with methyl prednisolone sodium succinate to help improve neuronal outcome. Corticosteroids act on the cell membrane receptor lead to the formation of phospholipase $A_2$ inhibitory proteins. The author suggested the following protocol for the administration of methyl prednisolone sodium succinate for spinal injury patients. First the drug had to be given at a dose rate of 30mg/ kg body weight intravenously, given over several minutes. This had to be followed by a further dose of 15mg/kg body weight every hour to six hours. The drug had to be administered within eight hours of trauma and the treatment should not exceed 24 hours. There was poor neurological recovery when the protocol was begun after eight hours or if continued for longer than 24 hours.

Significant suppression of cortisol and a trend toward suppression of ACTH was observed after one week of methyl prednisolone administration in cats in a study conducted by Cranger *et al.* (1994). The hypothalamic-pituitary- adrenal axis quickly recovered from suppressive effects of methyl prednisolone, one week after administration of the steroid was discontinued.

Shell (1996c) recommended medical management of paralyzed animals under the following conditions. 1) the dog was experiencing its first episode of pain, ataxia, or mild paresis; 2) the dog had been paralyzed without perception of deep pain stimuli for longer than 24 hours; or 3) the dog was paralyzed with intact deep pain perception, but the owners didn't consider surgical intervention an option. Management of these cases consisted of supportive care: keeping the dog clean of urine and faeces to avoid dermatitis, padding the cage to help prevent decubital ulcers, and assisting with urination and defaecation. If voluntary urination was not present, the bladder must be manually emptied or catheterized three to four times daily. Warm water enema might be necessary if

defaecation does not occur. Topical application of a zinc oxide ointment waterproofs the preputial or vulvar areas and helped to prevent urine scalding. Restricted patient activity was mandatory for a minimum of four to six weeks.

Cohn (1997) stated that corticosteroids of varying potencies were available in a number of formulations (oral, parentral, topical) with differing onset and duration of actions. Soluble esters released their steroid component quickly, allowing a rapid onset of action. Moderately insoluble esters released steroids slowly over days to weeks. Poorly soluble esters acted as repositols and released steroids over a period of weeks to months.

Wheeler (1997) described about the importance of cage rest in corticosteroid treatment. The corticosteroid relieved the dog's discomfort and made it much more active. This rendered the dog very susceptible to further herniation of disc material and subsequent development of severe neurological deficits. The animal must rest quietly in a confined space for at least two weeks, during which time it should only be allowed out to defaecate and urinate. The author opined that the gastrointestinal side effects were more with dexamethasone administration than with methyl prednisolone. Combined use of steroids and NSAID's were contraindicated because of high incidence of side effects.

Chandy and Vasanth (2006) reported that epidural administration of methyl prednisolone acetate at the dose rate of 2mg/kg body weight was effective for the treatment of paraplegia in dogs. The drug was administered at the lumbosacral junction.

Levine *et al.* (2008) described about the complications and neurological outcomes associated with dexamethasone administration to dogs with surgically treated thoracolumbar intervertebral disc herniation. Dexamethasone group

dogs were more likely to develop complications like diarrhoea and urinary tract infection compared with non- treatment group dogs.

*2.7.1.1. Epidural injection*

Jones (2001) described in detail the technique of epidural injection in dogs and cats. Injection usually carried out at the lumbosacral junction. The site was located by using the external angle of ilia and the dorsal spinous processes of the seventh lumbar vertebra and the sacrum as anatomical landmarks. Loss of resistance test was used for ensuring the needle placement in the epidural space. Sacrococcygeal space also used in case of larger dogs. Epidural injection had a localized and intense effect than systemic administration.

The author opined that infectious skin disease particularly that which involved sepsis, in the region of the lumbosacral area, was an absolute contraindication for epidural drug administration. Anatomical abnormalities which might be congenital or which arised as a result of trauma and made access to the lumbosacral space either extremely difficult or impossible were also absolute contraindications. It was difficult to locate the anatomical landmarks and lumbosacral space in case of fatty animals.

Campoy (2004) used Hanging drop test and Loss of resistance test to ensure correct positioning of the needle in the epidural space. In the Hanging drop test, a drop of saline was placed in the hub. As the needle entered the epidural space, due to the sub atmospheric pressure within the space the drop would be sucked in. Loss of resistance test might be carried out by injecting a small amount of saline to verify the ease of injection by determining the lack of resistance encountered.

### 2.7.2. Physiotherapy

Physiotherapy was the treatment of disease or injury with physical agents such as cold, heat, light, electricity, sound, and mechanical agents such as massage and exercise. Physiotherapy resulted in improvement in physical strength, range of motion, and coordination, as well as decreasing inflammation, edema, and pain (Hodges and Palmar, 1993).

#### *2.7.2.1. Therapeutic ultrasound*

Bromiley (1991) opined that the distress demonstrated by an animal during ultrasound massage may be the result of pain due to incorrect setting (W/cm2) and / or may be the result of an ability to register a previously unknown sound. The author opined that the pulsed setting minimized the thermal effects created at the vertebral body surface. The shear stress waves created during continuous ultrasound may increase the local heat and since the periosteum is avascular no cooling occurs and the excessive heat will cause the pain.

Ultrasound is a mechanical vibration produced at frequencies beyond the audible range of the human ear. An important characteristic of ultrasound-wave transmission is reflection, which occurred at interfaces between tissues of different acoustic impedance. Burning of tissues and gaseous cavitations of tissues as a result of reaction with the dissolved gases within the tissues were the two disadvantages (Hodges and Palmar, 1993).

Clark and McLaughlin (2001) described in detail about therapeutic ultrasound massage. Ultrasound used therapeutically to heat deeper tissues. As sound waves are being absorbed in muscle, some of the energy is converted to heat. A depth of 5cm could be reached, elevating the temperature of that tissue to between 104 and 113F. Ultrasound was thought to promote healing by

stimulating enzyme activity; increasing cellular metabolism, improving circulation and increasing the strength and pliability of tendons. Non- thermal effects included increased cell membrane permeability, increased calcium transport, removal of blood cells from the interstitial space, and increased phagocytic activity of macrophages. The units have a continuous and pulsed mode. Continuous ultrasonography leads to unstable cavitation, a phenomenon in which bubbles of dissolved gas form and grow, but this does not seems to be a clinical problem as long as the appropriate time and power settings were used. Rate of energy delivered per unit area ($w/cm^{2)}$ was the intensity and 1-2 $w/cm^2$ is required to raise tissue temperature to 104-113F.

Taylor (2001) stated that therapeutic ultrasound was commonly used to increase collagen extensibility, enhance collagen remodeling and production, increase heat in deep tissues, blood flow, and range of motion, reduce pain and muscle spasm, and accelerate wound healing. The two most commonly used US frequencies were 1.0 MHZ and 3.3 MHZ. The 1MHZ penetrated more deeply and was used for heating tissues from 2-5cm in depth. The 3.3MHZ was used to heat tissues to a depth of 1-2 cm. A coupling agent was needed to connect the US head with the skin in order to maximize transfer of the US energy in to the tissues. The hair must be clipped over the desired treatment area. It interfere with the absorption of the US energy and it may burn the skin if this not done. The hair absorb US energy and little will be available to heating deeper tissues.

Olby *et al.* (2005) opined that ultrasound might be beneficial for epaxial muscles that were experiencing muscle spasms. Its use was contraindicated over exposed spinal cord, and continuous mode ultrasound was not recommended in postoperative neurological patients. In non-surgical patients with acute spinal cord disease and neuromuscular spasm, ultrasound might be

applied to the epaxial muscles to help manage pain and muscle spasm. This also improved the blood supply.

## 2.8. RECOVERY AND RECURRENCE RATE

In a case study of thoracolumbar intervertebral disc disease Scott (1997) concluded that dogs with evidence of multiple disc degeneration as evidenced by the presence of a least two calcified intervertebral disc spaces between T10 and L5 would appear to be most at risk of recurrence. The interval from initial treatment to recurrence ranged from 7-25 months.

Dhupa *et al.* (1999) conducted a study to determine the postoperative outcomes of dogs with caudal lumbar intervertebral disc herniation. The potential predictors of functional recovery they selected were age, body weight, severity of preoperative pelvic limb neurological deficits and surgical procedure employed. They reported that the median time to walking in dogs with caudal lumbar disc herniation was 13 days, compared to 7 days with thoracolumbar disc herniation.

Mayhew *et al.* (2004) reported that number of opacified disc was a significant risk factor for recurrence. Risk increased with number of opacified discs in an almost linear manner; each opacified disc increased risk by 1.4 times. No significant differences in recurrence rates observed between the chondrodystrophic breeds and non-chondrodystrophic breeds. Also recurrence rate were not influenced by age or sex.

## 2.9. REHABILITATION

Hodges and Palmar (1993) described about the usage of slings and carts in the management of posterior paralysis in dogs. The carts increased patient mobility and helped to prevent decubital ulcers and abrasions. The authors

discouraged the use of carts for small dogs during the first 3-4 months after disability because patients depend on the cart and were less enthusiastic about their physical therapy. But if disability extended beyond 3-4 months carts could be of tremendous help. Mobile slings were helpful in supporting large breed dogs. Placing the patient in the sling several times a day helped to prevent decubital ulcer formation.

# *Materials and Methods*

# 3. MATERIALS AND METHODS

## 3.1. SELECTION OF CASES

The study was conducted in fourteen clinical cases of dogs of different age, sex, breed, and body weight presented to the Surgery unit of Veterinary Hospital Mannuthy and University Veterinary Hospital Kokkalai with paraplegia.

## 3.2. BROAD OUTLINE OF WORK

The cases were randomly divided into two groups of seven dogs each, Group I and Group II.

The Group I consisted of seven cases of paraplegic dogs serially numbered from $I_1$ to $I_7$. These animals were subjected to epidural administration of methyl prednisolone acetate[1] at the rate of 2mg/kg body weight on the day of presentation and repeated after twenty four hours and maintained with oral administration of prednisolone acetate[2] starting at a dose rate of 2mg/kg body weight for five days, tapered to 1mg/kg body weight for next five days, and then 0.5mg/kg body weight for the following five days. (Fig.1)

Group II consisted of seven cases of paraplegic dogs serially numbered from $II_1$ to $II_7$. In addition to the steroid treatment as in Group I, Group II animals were subjected to ultrasound massage of the affected dorsal spinal

---

[1] Depo-medrol™ –Methyl prednisolone acetate 40mg/ml, Pfizer Products India Pvt Ltd., Mumbai

[2] Wysolone – Prednisolone acetate 10mg tablets, Wyeth Ltd., Goa

region for five days continuously and later at weekly intervals for five days till improvement noticed or for a maximum of six weeks.

All the dogs were subjected to supportive medicines like neurotropic drug (methyl cobalamine[3]) and antibiotics (amoxicillin-cloxacillin[4]) during the period of observation. Ranitidine[5] was given orally during the period of steroid administration. The clients were strictly advised to adhere to the following managemental care such as:

1. Strict cage rest

2. Maintain the dog in a sanitary environment, with soiled bed removed regularly

3. Alternatively change the recumbency position in right and left to prevent ulcer formation and

4. Manual expression of bladder, if possible in case of urine retention.

## 3.3. OBSERVATIONS

### 3.3.1. Anamnesis and signalment

History and observations narrated by the owner were recorded to determine whether the onset was acute or chronic and also to determine the possibility of involvement of the spinal cord. General condition, age, sex, breed, and body weight of the dogs were observed and recorded.

---

[3] Met- Neurobion – Methyl Cobalamine 500mcg/ml, Merck Ltd., Thane

[4] Novaclox – Amoxicillin-Cloxacillin 250mg tablets, Okasa Pvt Ltd., Haridwar

[5] Rantac – Ranitidine 150mg tablets, Torrent Pharmaceuticals, Makhena, Baddi

### 3.3.2. Physiological parameters

Respiratory rate (per min), pulse rate (per min), rectal temperature ($^0$C), and colour of visible mucous membrane were recorded.

### 3.3.3. Clinical symptoms

A thorough physical examination was conducted prior to neurological examination to rule out non-neurological diseases. The vertebral column and bones of the limbs and pelvis were palpated to detect fracture and / or other abnormalities.

The urinary bladder function was assessed through abdominal palpation of the bladder. Cystoplegic dogs were graded as having lower motor neuron (LMN) bladder or upper motor neuron (UMN) bladder based on whether urine was freely relieved or not when the bladder was compressed through the abdominal wall. Presence of voluntary urination with occasional dribbling or normal urination was also noted.

### 3.3.4. Neurological examination

All the dogs were subjected to a detailed neurological examination to localize the site and type of lesion as mentioned by Lorenz and Kornegay (2004) and the observations were correlated with radiography to confirm the diagnosis.

*3.3.4.1. Mental status, Posture and Gait:* The mental status of the dogs was recorded as alert, depressed, or comatose depending up on its level of consciousness. Posture assessed by the dogs when left on the floor was noted and recorded as sitting / recumbent, or standing with or without help. The dogs were allowed to move on their own to detect abnormalities in gait and recorded as dragging of hindquarters, ataxia, occasional ataxia or normal gait.

*3.3.4.2. Postural reactions*

3.3.4.2.1. Wheel barrowing reaction

The dog was supported under the abdomen with all the weight on the thoracic limbs and made to walk. The ability of the animal to walk forward and sideways with coordinated movements of both thoracic limbs noted and recorded as present or absent. (Fig.2)

3.3.4.2.3. Proprioceptive positioning reaction

The dogs were supported under abdomen and their paws turned backwards so that the dorsal surface of the paws to touch the ground. The ability of the animal to return the paws to the normal position was assessed and recorded as normal, delayed or absent. (Fig.3)

**3.3.5. Spinal reflexes**

The spinal reflexes were graded as 0, 1, 2, 3 and 4 representing absence of reflex, depressed reflex, normal reflex, hyperreflexia and clonus respectively (Shell, 1996a).

*3.3.5.1. Patellar reflex:* With the dog on lateral recumbency, the limb was supported under femur with the left hand and the stifle was flexed slightly. The straight patellar ligament of the stifle joint was struck crisply with the knee hammer. The normal response was a single, quick extension of the stifle. The test was repeated on the other side with the dog lying on lateral recumbency on the opposite side. The reflex was recorded as absent, depressed, normal or increased. (Fig.4)

*3.3.5.2. Flexor reflex:* The dogs were restrained on lateral recumbency and the upper hind limb was held in a relaxed position and the toes pinched with fingers. The normal response was flexion of the entire limb including the hip, stifle, and hock. The reflexes were recorded as increased, normal, depressed, or absent. (Fig.5)

*3.3.5.3. Deep pain sensation:* Deep pain sensation was assessed by pinching the toes with an artery forceps. Based on the behavioural response of the dog, deep pain sensation was recorded to be present or absent. (Fig.6)

*3.3.5.4. Tail wag reflex:* Observed the tail wagging and recorded as normal, reduced, or absent.

*3.3.5.5. Hyperpathia*: Hyperpathia was noted when pressure applied to spinous processes and paraspinal muscles of the thoracic and lumbar region and transverse processes and paraspinal muscles of the cervical region resulted in pain and a behavioural response. The response was observed and recorded as present or absent. (Fig.7)

*3.3.5.6. Perineal reflex:* The perineal and peri anal skin was pinched gently using artery forceps and the ability of the external anal sphinctor to contract was recorded as normal, depressed, or absent. (Fig.8)

**3.3.6. Radiographic evaluation**

*3.3.6.1. Survey radiography:* Lateral and / or ventrodorsal view radiographs of regions of the vertebral column suspected to have lesions were obtained. Lateral view radiographs of the vertebral column were obtained with the dogs on lateral recumbency, with the fore and hind limbs parallel to each other and stretched cranially and caudally respectively. Ventrodorsal view radiographs

were obtained with the dogs on dorsal recumbency with the fore and hind limbs parallel to each other and stretched cranially and caudally respectively.

*3.3.6.2. Myelography:* After survey radiography, two dogs were subjected to myelography. The contrast agent used was Iohexol[6] at the dose rate of 0.3ml/kg body weight (90mg I/kg body weight).

General anaesthesia was induced using atropine sulphate[7] @ 0.045mg/kg body weight intramuscularly, followed by xylazine hydrochloride[8] @ 1mg/kg body weight intramuscularly and diazepam[9] @ 0.2mg/kg body weight intravenously.

3.3.6.2.1. Cisterna magna injection

The hair was clipped from the area overlying the occipital crest and the first cervical vertebrae to the width of the palpable wings of the atlas. The site was prepared asceptically.

The dog was placed on lateral recumbency near the edge of the table with the head kept into extreme flexion. The following landmarks were identified: external occipital protuberance with the middle finger, the edge of the wing of the atlas with the thumb and the region overlying the atlanto-occipital space with the index finger. The spinal needle or hypodermic needle (20G) was introduced into the midline and directed into the skin parallel with the line of the lower jaw. Its point was advanced slowly through the skin

---

[6]Contrapaque™- Iohexol 300mgI/ml, J.B. Chemicals &Pharmaceuticals Ltd, Ankleshwar

[7]Atropin - Atropine Sulphate 0.6mg/ml, Hindustan Pharmaceuticals, Barauni

[8]Xylaxin – Xylazine hydrochloride 23.3mg/ml, Indian Immunological Ltd.,Gguntur

[9]Calmpose – Diazepam 10mg/ml, Ranbaxy Laboratories Ltd., Mumbai

Resistance was felt as the tip penetrates the ligamentum flavum, and might be followed by a "pop" as it enters the subarachnoid space.

Aspirated CSF in equal quantity to that of the volume of the contrast material to be injected. A small volume of contrast material was injected. If there was no adverse response and the injection was unimpeded, the remainder was injected slowly.

Lateral and ventrodorsal radiographs were taken immediately, 5, 10, and 15 minutes following injection. The head should be kept raised at all times following injection to minimize the amount of contrast flowing forward into the cranial cavity

3.3.6..2.2. Lumbar injection

The dog was positioned on lateral recumbency. The hind limbs were pulled cranially, so arching the lumbar region increasing the space between the neural arches. Introduced the spinal or hypodermic needle (20G) into the spinal canal through the lumbosacral space. Injected the contrast material slowly. The hind limbs were elevated to favour anterior flow of the solution. Lateral and ventrodorsal radiographs were taken immediately, 5, 10, and 15 minute following injection (Wright, 1984; Lewis and Hosgood 1992; Krishnamurthy and Singh, 1994).

### 3.3.7. Haematological parameters

Wet film examination of blood was conducted to rule out blood parasites.

Blood smears were prepared and blood samples were collected in

EDTA[10] for estimation of haemoglobin concentration (g/dl), packed cell volume (per cent), total leukocyte count ($10^3$/cu.mm.), and differential leukocyte count (per cent of individual cells). (Meinkoth and Clinkenbeard,2000; Benjamin, 2005). The samples were collected on the day of presentation and at fortnight intervals up to six weeks.

### 3.3.8. Serum biochemistry

Blood samples were collected, separated the serum and subjected to estimation of serum inorganic calcium (mg/dl), phosphorous (mg/dl), and serum enzyme alkaline phosphatase (IU/L). The samples were collected on the day of presentation and at fort night intervals up to six weeks.

Serum calcium, phosphorous, and alkaline phosphatase were estimated by using standard Agappe diagnostic kit[11].

## 3.4. NEUROLOGICAL GRADING OF PATIENTS

Based on the severity of the clinical signs animals were graded as follows (Tartarelli *et al*, 2005):

Grade I – (+) spinal hyperesthesia only

Grade II – (+ +) ambulatory paraparesis

Grade III – (+ + +) non-ambulatory paraparesis

Grade IV – ( + + + + ) paraplegia

---

[10]EDTA – EDTA Disodium salt (Nice Laboratory Reagent), New India chemical Enterprises, Kochi

[11]Agappe Diagnostic kit – Agappe Diagnostic Pvt Ltd, Ernakulam, Kerala

Grade V – (+ + + + + ) paraplegia with urinary incontinence

Grade VI – (+ + + + + + +) paraplegia, urinary incontinence and absence of deep pain sensation

## 3.5. EPIDURAL INJECTION

### 3.5.1. Site of injection

The lumbosacral space from the seventh lumbar vertebrae (L7) to the first sacral vertebrae (S1) was the preferred site for injection. The site for needle placement was clipped cleaned and applied Tr. Iodine.

### 3.5.2. Procedure

All animals in both the groups were placed either on their sides in right or left lateral recumbency or in sternal recumbency.If in lateral recumbency, the hind limbs were pulled forward or, if in sternal recumbency, the hind limbs were "tucked" under the animal. The site was located by using the external angles (wings) of ilia and the dorsal spinous processes of the seventh lumbar vertebrae and the sacrum as anatomical landmarks.

The external angles of ilia were palpated with the thumb and middle finger of one hand, the index finger was directed caudally. The lumbosacral space was located by palpation of the depression immediately caudal to the dorsal spinous processes of the seventh lumbar vertebrae (Fig.9). An insensitive skin weal was made with 0.5 ml of lignocaine hydrochloride[12] at the site of epidural injection.

---

[12]xylocaine 2% – Lignocaine injection I. P., Astra-IDL, Bangalore

The spinal / hypodermic needle (20G) was inserted through the skin at midline slowly at an angle of $90^0$ to the spine. The needle was directed downwards and forwards at an angle of $45^0$ and advanced to pierce the interarcuate ligament with a distinct "poping" sensation felt at the fingers.

The hub was inspected for blood (indicating that the needle has entered the ventral venous plexus). Loss of resistance test was used to ensure the needle placement in epidural space (Hall, 1971; Jones, 2001; Campoy, 2004).

## 3.6. THERAPEUTIC ULTRASOUND MASSAGE

All the dogs of Group I were subjected to ultrasound massage. Pulsed ultrasound (1:1) was given on either side of the dorsal aspect of the affected spinal region at 1.5 watts /square centimeters for 10 minutes (Fig.10).

Placed the animal on lateral recumbency or in sitting position. The hair from the dorsal aspect of the affected spinal region was clipped and shaved. Ultrasound gel[13] was used as the coupling agent for better transfer of energy. Placed the transducer head over the area to be treated at $90^0$ angle to the surface. Moved the transducer head slowly over the gelled area throughout the treatment in a circular fashion. Washed off and dried the treated area. (Bromiley, 1991; Taylor, 2001; Chandy and Vasanth, 2006).

## 3.7. POST INJECTION OBSERVATION

All the observations were repeated at fortnight intervals till recovered or up to a period of six weeks.

---

[13]Gel – Ultrasound gel 5000 mL Medichem India, Pondichery

## 3.8. COMPLICATIONS

Complications of paraplegia (*viz.*urinary tract infection, ulcer formation and wounds), myelography, and steroid therapy, if any, were noted.

## 3.9. RECURRENCE

The recurrence of clinical cases if any was also observed during the period of study.

## 3.10. STATISTICAL ANALYSIS

The data recorded were subjected to statistical analysis using Paired t test (Snedecor and Cochran, 1994).


**Fig. 1. Drugs & Contrast agents used.**

1. **Depo - medrol**
2. **Wysolone**
3. **Met - neurobion**
4. **Contrapaque 300**

**Fig. 2. Testing for Wheel barrowing reaction. (Dog $I_1$)**

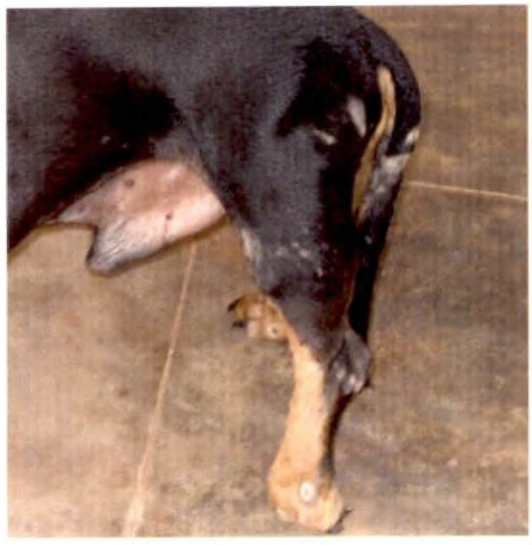


Fig. 3. Testing for Proprioceptive positioning reaction. (Dog $II_2$)

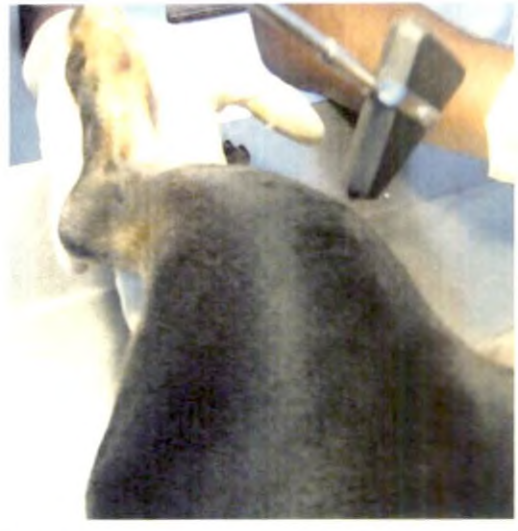


Fig. 4. Testing for Patellar reflex. (Dog $II_2$)

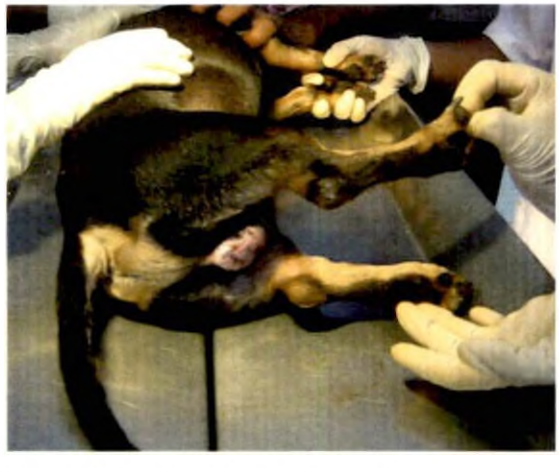


Fig. 5. Testing for Flexor reflex. (Dog $II_2$)

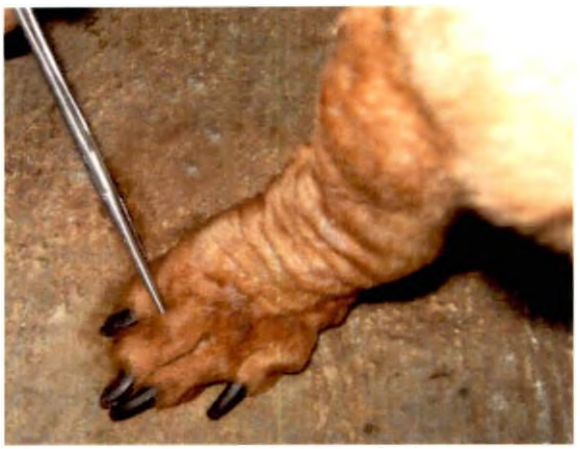


Fig. 6. Testing for Deep pain sensation (Dog $I_1$)

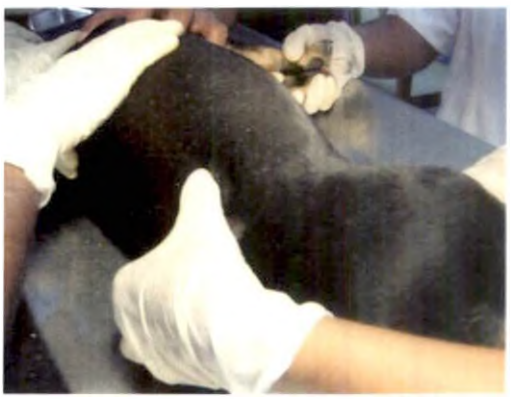


Fig. 7. Testing for Hyperpathia (Dog $II_2$)

Fig. 8. Testing for Perineal reflex (Dog $II_2$)

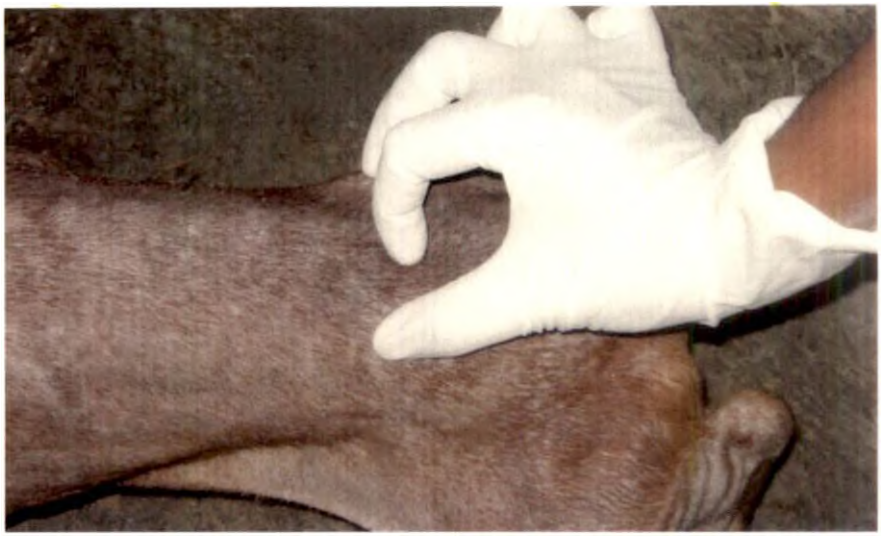


**Fig. 9. Locating the site for Epidural injection. (Dog $I_1$)**

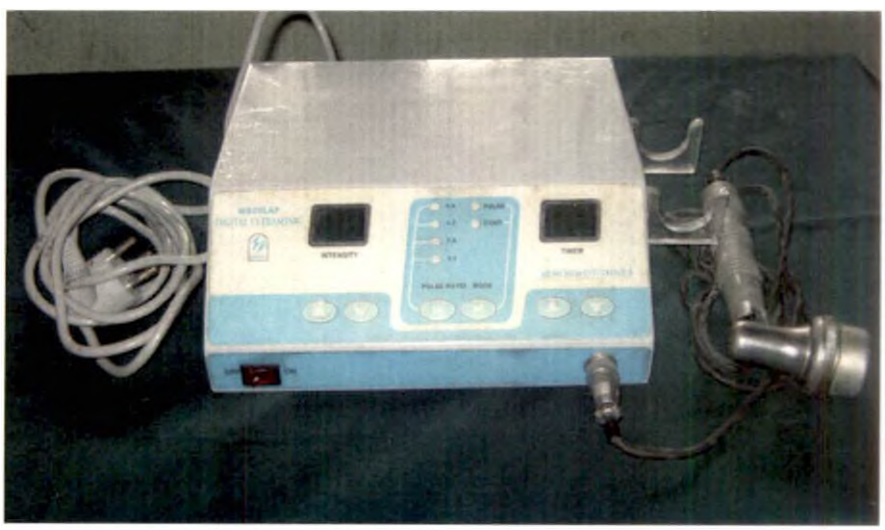


**Fig. 10. Therapeutic ultrasound machine. (Dog $I_1$)**

# *Results*

# 4. RESULTS

The study was conducted in fourteen clinical cases of dogs of different age, sex, breed and body weight brought to the Surgery unit of Veterinary Hospital Mannuthy and University Veterinary Hospital Kokkalai with paraplegia. The cases were randomly divided into two groups of seven dogs each, Group I and II.

Preliminary clinical, neurological and radiological examinations were conducted in all dogs. The dogs of Group I were subjected to epidural administration of methyl prednisolone acetate 2mg/kg body weight initially and oral administration of prednisolone acetate in a tapering dosage for 15 days and that of Group II were subjected to ultrasound massage of the dorsal spinal region in addition to the steroid administration as in Group I. The efficacy of both treatment procedures were compared by evaluating the recovery period and neurological status of animal.

## 4.1. ANAMNESIS AND SIGNALMENT

All the dogs in both groups were alert but not able to bear weight on hind limbs when presented. Dribbling of urine was reported in 10 cases. All the dogs were regularly vaccinated against rabies. No previous occurrence was reported in any of these cases. Three dogs were treated in the nearby veterinary hospital with B- complex vitamins before presented. The data of the dogs included in the study in the two groups is given in Table 1.

### 4.1.1. Breed

Of the fourteen cases of posterior paralysis studied, eleven were Daschunds (79 per cent), two were non-descripts (14 per cent), and one was Lhasa apso (seven per cent). (Fig.11).

### 4.1.2. Age

The age of the dogs ranged from three months to seven years and five months (mean 4.7years). Nine dogs (63 per cent) were between four to six years of age, three (21 per cent) were between six to eight years of age and one each (eight per cent) between zero to two and two to four years of age.(Fig.12).

### 4.1.3. Sex

Half of these dogs were males and half were females.

### 4.1.4. Body weight

The body weights of the dogs ranged from 6.5 kg to 13.5 kg (mean 11kg).

### 4.1.5. Etiology

Among the fourteen dogs, one dog was ($I_2$) injured in an automobile accident, three dogs ($I_1$, $I_3$, $II_6$) due to falling from height and one dog ($I_7$) by malicious injury caused due to blow by neighbour.The cause was unknown for the remaining nine cases.

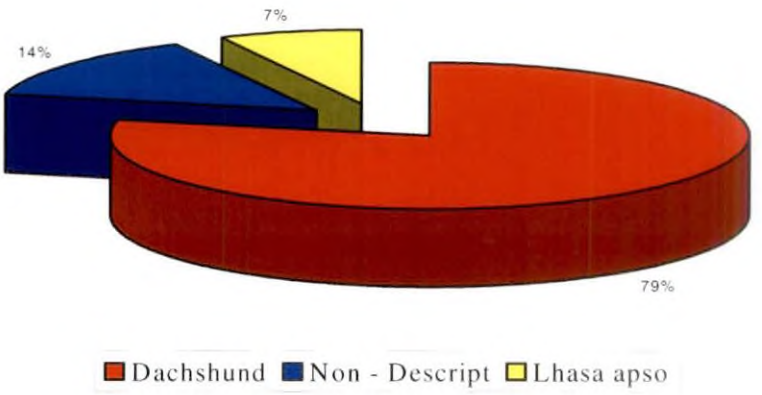



**Fig 11. Breed - wise distribution of paraplegia in dogs.**

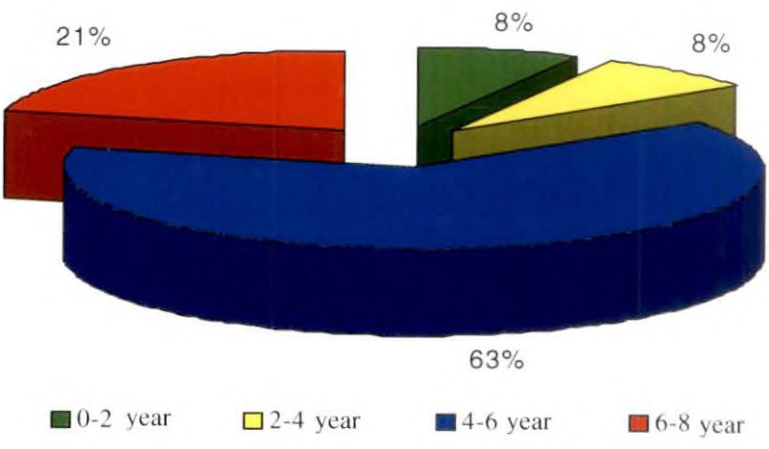



**Fig 12. Age - wise distribution of paraplegia in dogs.**

### 4.1.6. Duration of illness

In Group I, out of the seven dogs, three dogs ($I_1$, $I_2$, $I_7$) were presented on the next day of onset of illness, another three ($I_3$, $I_4$, $I_5$) after two days and the remaining one ($I_6$) two months after the onset.

In Group II, out of the seven dogs, three dogs ($II_1$, $II_2$, $II_3$) were presented two days of onset of illness, another two ($II_4$, $II_5$) after four days and remaining two ($II_6$, $II_7$) after seven days of onset of illness.

## 4.2. GROUP I

### 4.2.1. Physiological parameters

The observations are presented in Table 2.

The mean value of the respiration rate (per min) ranged from 27.16 ± 1.88 to 28.00 ± 2.39 during the study period. The variations observed were marginal and within the normal range.

The mean value for the rectal temperature ($^0$C) ranged from 39.18 ± 0.21 to 38.18 ± 0.15 during the study period. The variations observed were marginal and within the normal range.

The mean value for the pulse rate (per min) ranged from 95.00 ± 5.80 to 109.16 ± 3.72 during the study period. The variations observed were marginal and within the normal range.

### 4.2.2. Clinical symptoms

All the dogs were not able to bear weight on hind limbs when presented. Dribbling of urine was noticed in four dogs ($I_3$,$I_4$,$I_5$,$I_7$). The vertebral column and bones of the limbs were palpated to detect fracture and / or other

abnormalities. Pain could be elicited on palpation of the affected part of the spine in all dogs of Group I. Gross distortion of the vertebral column could be palpated in two dogs ($I_2$ and $I_7$). There was swelling and oedema at the site of trauma and pain on palpation at the site of injury in one dog (I7).

*4.2.2.1. **Bladder function:*** The data obtained with respect to bladder function during the study period is depicted in Table 3.

In Group I, one dog had UMN bladder at the time of presentation ($I_4$), while three had LMN ($I_3$, $I_5$, $I_7$) and the rest ($I_1$, $I_2$, $I_6$) had normal bladder function.

The dog $I_4$ had returned to voluntary urination with occasional dribbling at second week and to normal bladder function at sixth week. The dog $I_3$ had returned to voluntary urination with occasional dribbling at second week and to normal bladder function at fourth week. The dog $I_5$ had returned to voluntary urination with occasional dribbling at fourth week and to normal bladder function at sixth week. The dog $I_7$ died on the second day.

### 4.2.3. Neurological examination

Detailed neurological examination of all the dogs was performed and the following data were obtained.

*4.2.3.1. **Mental status, posture and gait:*** The findings in mental status, posture and gait in dogs of Group I are recorded in Table 4.

Six dogs were alert while one was depressed at the time of presentation. All the dogs of this group were non-ambulatory and could not stand or bear weight on the hind limbs, and dragged their hindquarters on the day of presentation.

*4.2.3.2. Postural reactions*

4.2.3.2.1. Wheel barrowing reaction

A normal response of symmetrical forelimb movement was observed in five dogs (Table 5). The test was not performed in dogs $I_2$ and $I_7$ due to instability of the vertebral column.

4.3.2.2.2. Proprioceptive positioning reaction

The findings of conscious proprioception in dogs of Group I are presented in Table 5.

Conscious proprioception was absent in the hind limbs of all dogs on the day of presentation. By sixth week three dogs had regained delayed and two had regained normal conscious proprioception in the hind limbs.

***4.2.3.3. Deep pain sensation:*** The observations of deep pain sensation are presented in Table 6. All dogs had deep pain sensation in the hind paws at the time of presentation and throughout the study period.

***4.2.3.4. Hyperpathia:*** Hyperpathia was noticed in the affected spinal region of all dogs (Table7).

***4.2.3.5. Spinal reflexes:*** The forelimb spinal reflexes were normal in all the seven dogs. Spinal reflexes of the hind limbs were then examined and described individually. The findings are tabulated in Table 6 and Table7.

Dog $I_1$ - The spinal reflexes of the hind limbs were found to be depressed. The patellar and flexor reflexes were found to be depressed on the day of presentation and became normal on second week. The perineal reflex and bladder function were normal through out the study period. The tail wag reflex

was absent on the day of presentation and became normal on fourth week. Deep pain sensation was intact in both the hind limbs through out the study period. The lesion was localized between L4 and S2 cord segments.

Dog $I_2$ - The patellar reflexes were normal on the day of presentation, but found to be depressed on second week. The flexor reflexes and tail wag reflexes were normal initially but found to be depressed after twenty four hour. Deep pain sensation was intact through out the study period. Also perineal reflex and bladder function were found to be normal through out the study period. The lesion was localized between L4 and S2 cord segments.

Dog $I_3$ - The spinal reflexes of the hind limbs were found to be depressed. The patellar and flexor reflexes were sluggish on the day of presentation. Tail wag reflex, perineal reflex and deep pain sensation were intact through out the study period. The bladder was flaccid with overflow incontinence. The lesion was localized between L4 and S2 cord segments.

Dog $I_4$ - The animal showed a spastic bladder i.e. it was firm and difficult to express and there was brief intermittent spurting of urine. Tail wag reflex and perineal reflex were normal through out the study period. The patellar reflexes were found to be exaggerated on the day of presentation and tend to be normal on second week. The flexor reflex was absent for right limb and depressed for left limb. The reflexes were found to be normal on fourth week. Deep pain sensation was intact through out the study period. Lesion was localized between T3 and L3 cord segments.

Dog $I_5$ - The bladder was flaccid with overflow incontinence on the day of presentation and become normal on sixth week. The tail wag reflex was found to be depressed initially and become normal on second week. Perineal reflex was normal through out the study period. The patellar reflexes were depressed

and flexor reflexes were absent on the day of presentation. Deep pain sensation was intact through out the study period. The lesion was localized between L4 and S2 cord segments.

Dog $I_6$ - The patellar reflexes were found to be depressed. Flexor reflexes were depressed on the day of presentation and became normal on fourth week. Tail wag reflex and perineal reflex were normal through out the study period. Deep pain sensation was intact through out the study period. Lesion was localized between L4 and S2 cord segments.

Dog $I_7$ - The bladder function was flaccid with overflow incontinence on the day of presentation. Tail wag reflex and patellar reflexes were found to be depressed. A perineal reflex was normal. Flexor reflexes were found to be normal on the day of presentation, but depressed on second day. Deep pain sensation was intact through out the study period. The lesion was localized between L4 and S2 cord segments.

### 4.2.4. Radiographic evaluation

*4.2.4.1. Survey radiography:* Survey radiography was used to locate the site of lesion and the radiographic findings were correlated with the neurological examination findings. The observations of the cases were recorded individually and given in Table 8.

Dog $I_1$ . Protrusion of third and fourth lumbar intervertebral disc in to the spinal canal and calcification of the fifth and sixth lumbar intervertebral disc were noticed. Narrowing of tenth, eleventh, twelth and thirteenth thoracic intervertebral disc spaces were observed (Fig.13).

Dog $I_2$ . Luxation of fourth lumbar vertebra was noticed (Fig.14).

Dog $I_3$ - Calcification of intervertebral disc between fifth and sixth lumbar vertebrae. Reduction and calcification of last thoracic intervertebral disc space and protrusion of third lumbar intervertebral disc into the spinal canal (Fig.15).

Dog $I_4$ - Calcification of the twelth thoracic intervertebral disc was noticed (Fig.16).

Dog $I_5$ - Narrowing and protrusion of the second, third, fourth and fifth lumbar intervertebral discs in to the spinal canal and calcification of the sixth lumbar intervertebral disc were noticed. Bladder distension and faecal stasis were also noticed.

Dog $I_6$ – Narrowing of tenth and eleventh thoracic intervertebral disc space and third, fourth, fifth and sixth lumbar intervertebral disc spaces were noticed (Fig.17)

Dog $I_7$ – Complete longitudinal fracture of body of last thoracic, first, second and third lumbar vertebrae were noticed (Fig.18)

*4.2.4.2. **Myelography:*** The dog $I_7$ was subjected to myelography using Iohexol at the dose rate of 0.3ml/kg body weight (90mg I / kg body weight). Both Cisterna magna puncture and lumbar puncture were used for the procedure.

The anaesthesia was sufficient for restraining the dog for myelography. The 20G, 1.5 inch long hypodermic needle was ideal for puncture. The needle had to be retracted and re-introduced into the cisterna magna as it had punctured dural vessels in the first attempt.

The quantity of iohexol used in the case provided good contrast for demarcation of the spinal cord. Keeping the dog in an elevated position with the

head up promoted caudal flow of iohexol. Myelography was useful in determining the extent of spinal cord compression.

Radiographs were taken at zero and fifth minute following lumbar puncture. Complete stoppage of the dorsal contrast column and narrowing of ventral contrast column at the level of third lumbar vertebrae noticed (Fig.19)

Serial radiographs were taken at zero, fifth and tenth minutes following cisterna magna puncture. The zero minute radiograph showed no contrast medium flow in the spinal canal. Flow of contrast material noticed up to the level of third lumbar vertebrae in the fifth minute radiograph. No further improvement in the flow of contrast medium was noticed in the tenth minute radiograph (Fig.20)

### 4.2.5. Correlation of radiographic pattern and neurological findings

Lesion was localized between L4 and S2 cord segments by neurological examination in all dogs except $I_4$. Calcification and protrusion of the intervertebral disc and narrowing of disc space in the lumbar region were the changes noticed in these dogs on survey radiography. Lesion was localized between T3 and L3 cord segments in dog $I_4$ and calcification of twelfth thoracic intervertebral disc was noticed on survey radiography.

### 4.2.6. Haematological parameters

The results of haematological studies are presented in Table 10.

Wet film examination of blood was negative in all the seven dogs.

The mean value for haemoglobin concentration (g/dl) ranged from 14.90 ± 0.71 to 13.73 ± 0.80 during the study period. The variations observed were marginal and within the normal range.

The mean value for packed cell volume (per cent) ranged from 42.66 ± 0.98 to 39.66 ± 0.49 during the study period. The variations observed were marginal and within the normal range.

The mean total leukocyte count ($10^3$/cu.mm) were 14.93 ± 1.16, 29.11 ± 3.08, 25.48 ± 2.28 and 19.36 ± 1.99 on day of presentation, second week, fourth week and sixth week respectively. The total leukocyte count was within the normal range initially. The values showed a significant increase on second week and showed a reduction on fourth week and returned towards normal levels by the end of the study period. (Fig.21)

The mean value for neutrophil count were (per cent) 73.66 ± 0.84, 84.5 ± 1.47, 84.5 ± 1.05 and 77.66 ± 1.81 on the day of presentation, second week, fourth week and sixth week respectively. The neutrophil counts were within the normal range initially. The values increased on second and fourth week and lowered to normal levels by the end of the study period.

The mean lymphocyte counts (%) were 24.50±0.80, 14.16±1.37, 13.50±0.95 and 20.16±1.77 on the day of presentation, 2nd week, 4th week and 6th week respectively. The lymphocyte count was within the normal range initially. The values decreased on 2nd and 4th week and returned to normal level by 6th week and the variations were corresponding to the value of neutrophil count.

The mean value for monocyte counts (per cent) ranged from1.50 ± 0.22 to 1.33 ± 0.42 during the study period. The variations observed were marginal and within the normal range.

The mean value for oesinophil counts (per cent) ranged from 0.83 ± 0.16 to 0.33 ± 0.21 during the study period. The variations observed were marginal and within the normal range.

### 4.2.7. Serum biochemical evaluation

The results of serum biochemical evaluation are presented in Table11.

The mean serum calcium values (mg/dl) ranged from 9.96 ± 0.26 to10.45 ± 0.42 during the study period. The variations observed were marginal and within the normal range.

The mean serum phosphorous values (mg/dl) ranged from 4.13 ± 0.41 to 4.25 ± 0.29 during the study period. The variations observed were marginal and within the normal range.

The mean values for alkaline phosphatase (IU/L) were 114 ± 8.73, 449.16 ± 28.65, 194.83 ± 5.81 and 102.66 ± 4.84 on the day of presentation, second week, fourth week and sixth week respectively. There is an increase on second week, but from third week onwards it lowered and became normal by the end of the study period. (Fig.22)

### 4.2.8. Neurological grading of patients

The details of neurological grading are given in Table 9.

Four dogs ($I_3$, $I_4$, $I_5$, $I_7$) were graded V and three dogs ( $I_1$, $I_2$, $I_6$ ) were graded IV on the day of presentation. On sixth week, three were ($I_3$, $I_4$, $I_5$) graded II and two were ($I_1$, $I_6$) graded I, while one ($I_2$) was graded IV. Dog $I_7$ died on second day.

### 4.2.9. Treatment

The method adopted for locating the site for epidural injection was accurate. The 20G, 1.5 inch long hypodermic needle was ideal for giving

epidural injection. For ensuring needle placement in epidural space loss of resistance test was used and found to be effective.

Out of seven dogs in Group I, five ($I_1$, $I_3$ $I_4$, $I_5$, $I_6$) became ambulatory at the end of the study period. Four dogs ($I_1$, $I_4$, $I_5$ $I_6$) had occasional ataxia. The dog $I_2$, which had luxation of fourth lumbar vertebra, remained as paretic till the end of the study period.

The progressive improvement of dogs of Group I is shown in Fig.23 to 26.

4.3. GROUP II

**4.3.1. Physiological parameters**

The observations are presented in Table 2.

The mean value for the respiratory rate (per min) ranged from 26.66 ± 1.54 to 23.66 ± 1.02 during the study period. The variations observed were marginal and within the normal range.

The mean value for the rectal temperature ($^0$C) ranged from 38.61 ± 0.17 to 38.63 ± 0.11 during the study period. The variations observed were marginal and within the normal range.

The mean value for the pulse rate (per min) ranged from 104.16 ± 3.62 to 98.83 ± 4.65 during the study period. The variations observed were marginal and within the normal range.

**4.3.2. Clinical symptoms**

All the dogs were not able to bear weight on hind limbs when presented. Dribbling of urine was noticed in 6 dogs ($II_1$, $II_2$, $II_4$, $II_5$, $II_6$, $II_7$). The vertebral

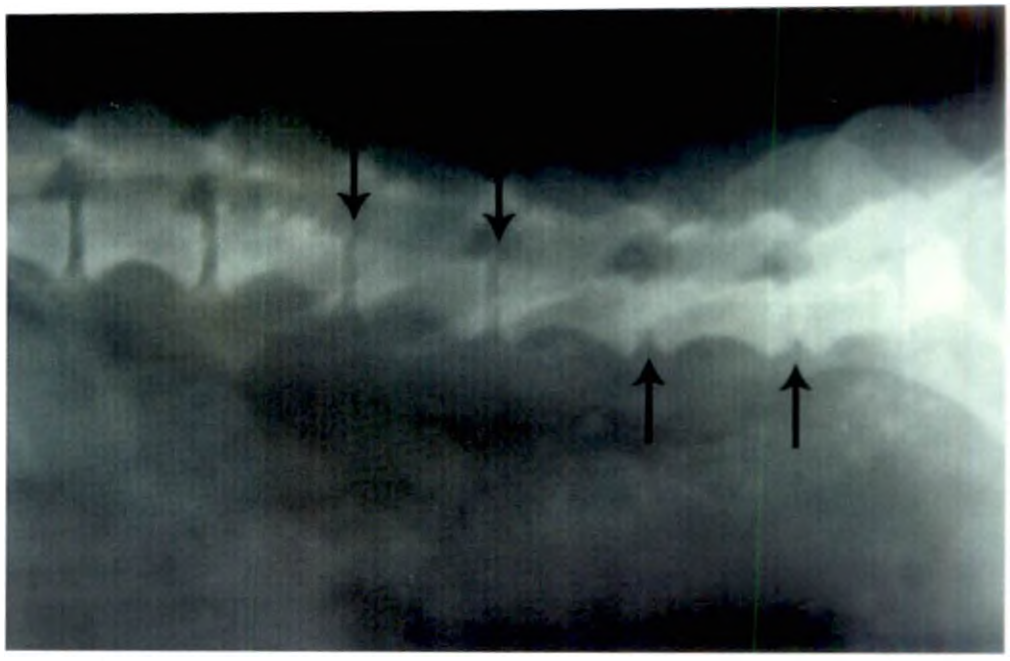


**Fig. 13. Skiagram showing protrusion of 3rd & 4th and calcification of 5th & 6thlumbar intervertebral discs. (Dog $I_1$)**

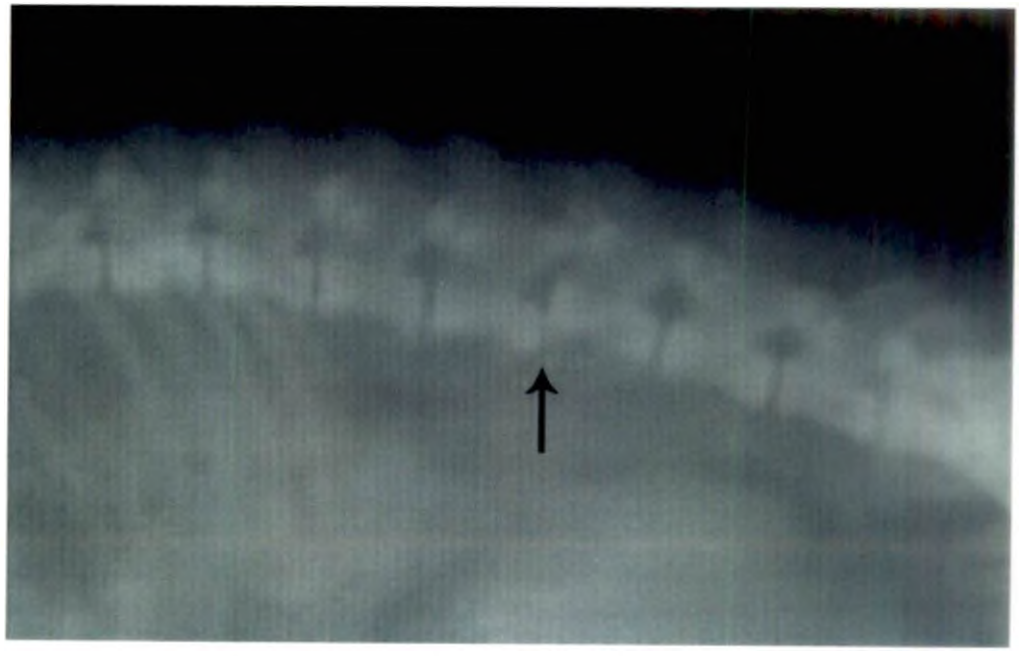


**Fig. 14. Skiagram showing luxation of 4th lumbar vertebra. (Dog $I_2$)**

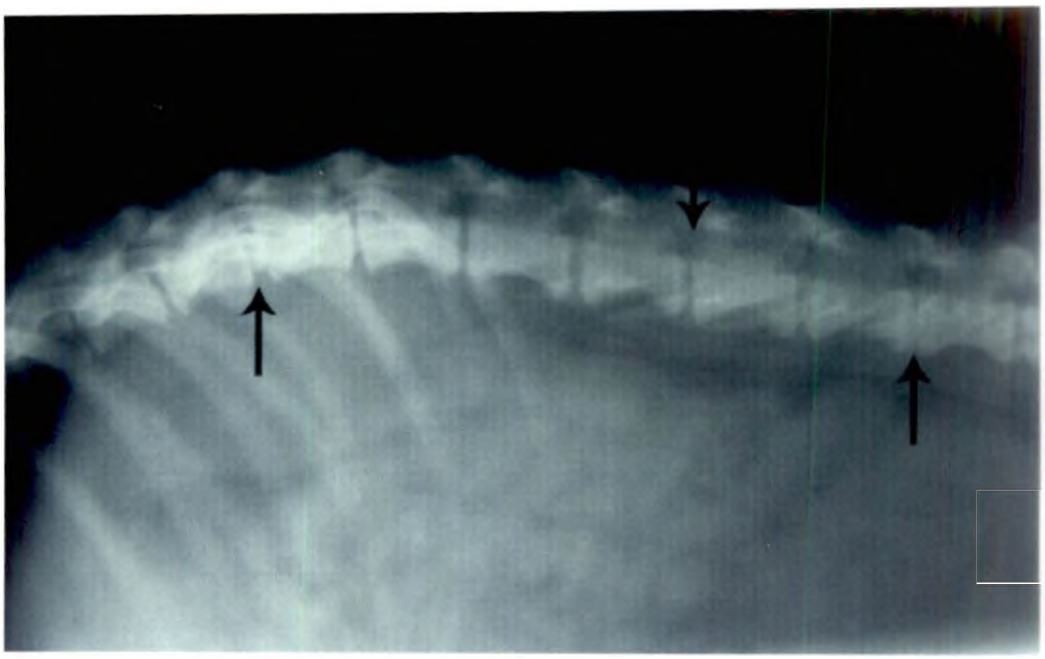


Fig. 15. Skiagram showing calcification of last thoracic, 5th lumbar and protrusion of 3rd lumbar intervertebral disc. (Dog $I_3$)

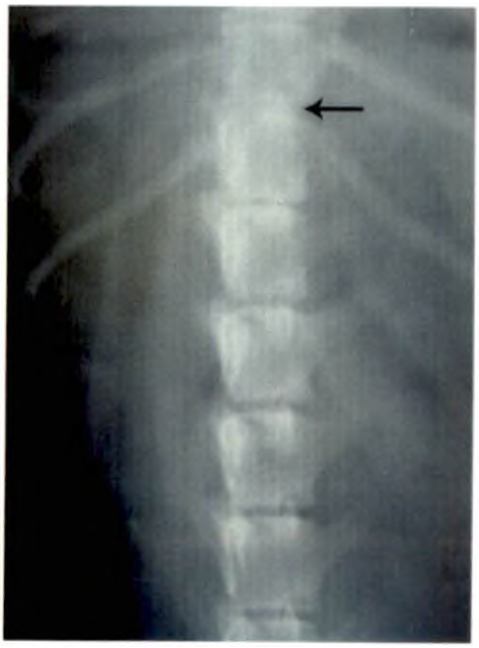


Fig. 16. Skiagram showing calcification of 12th thoracic intervertebral disc. (Dog $I_4$)

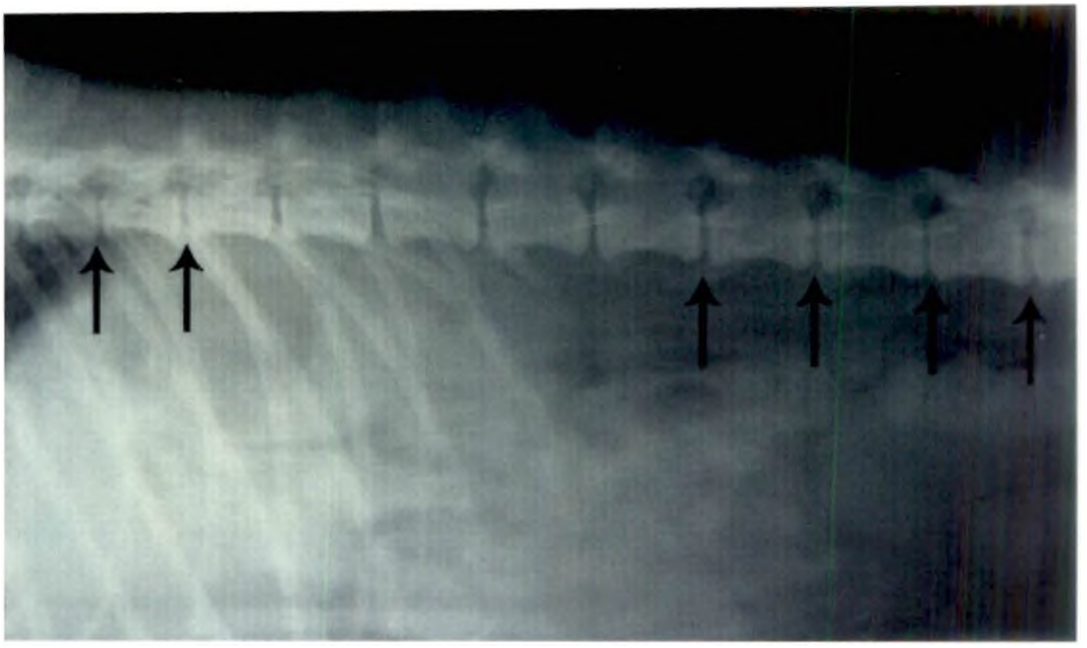


Fig. 17. Skiagram showing narrowing of 10th & 11th thoracic & 3rd, 4th, 5th & 6th lumbar intervertebral disc spaces. (Dog $I_6$)

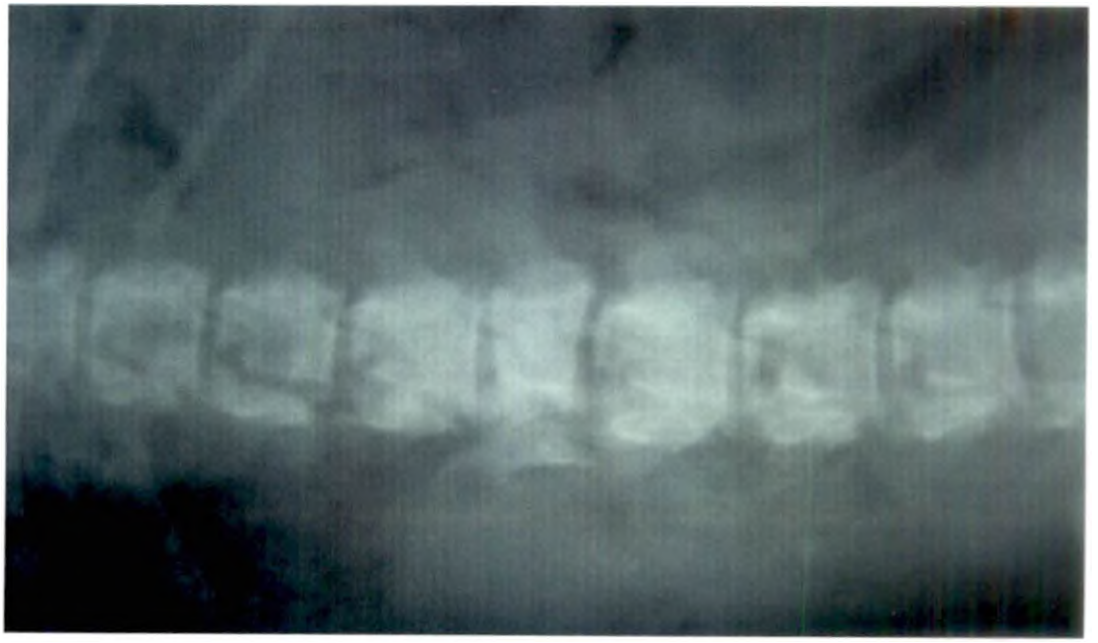


Fig. 18. Skiagram showing fracture of last thoracic, Ist, 2nd & 3rd lumbar vertebrae. (Dog $I_7$)

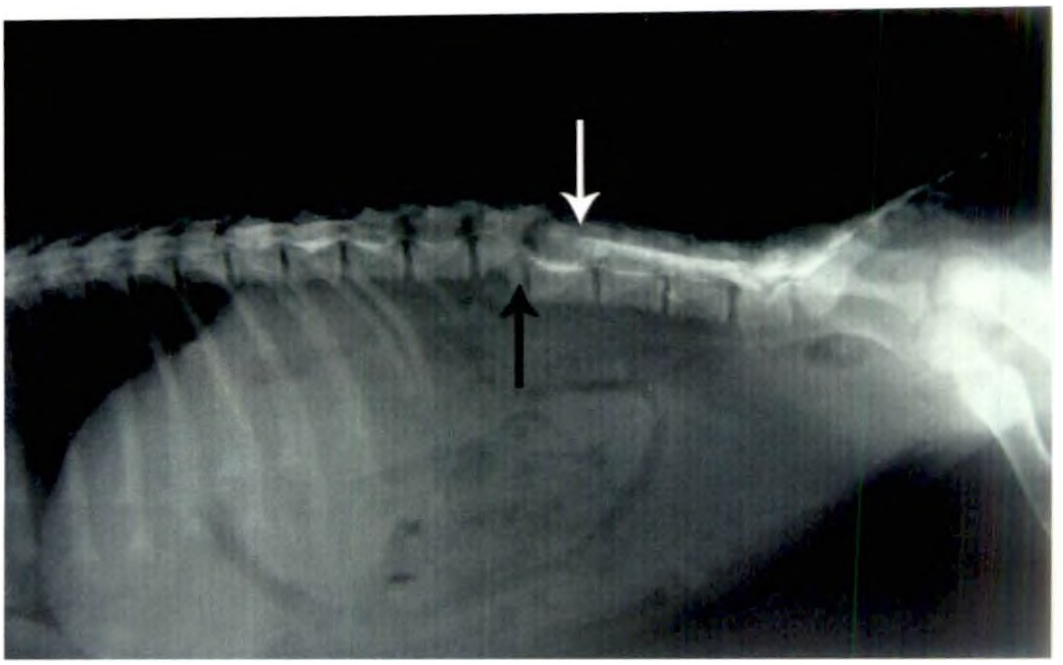


**Fig. 19. Skiagram of the lumbar myelography of Dog $I_7$ at o minute showing complete obstruction of dorsal contrast column & narrowing of ventral contrast column at the level of 4th & 3rd lumbar vertebrae.**

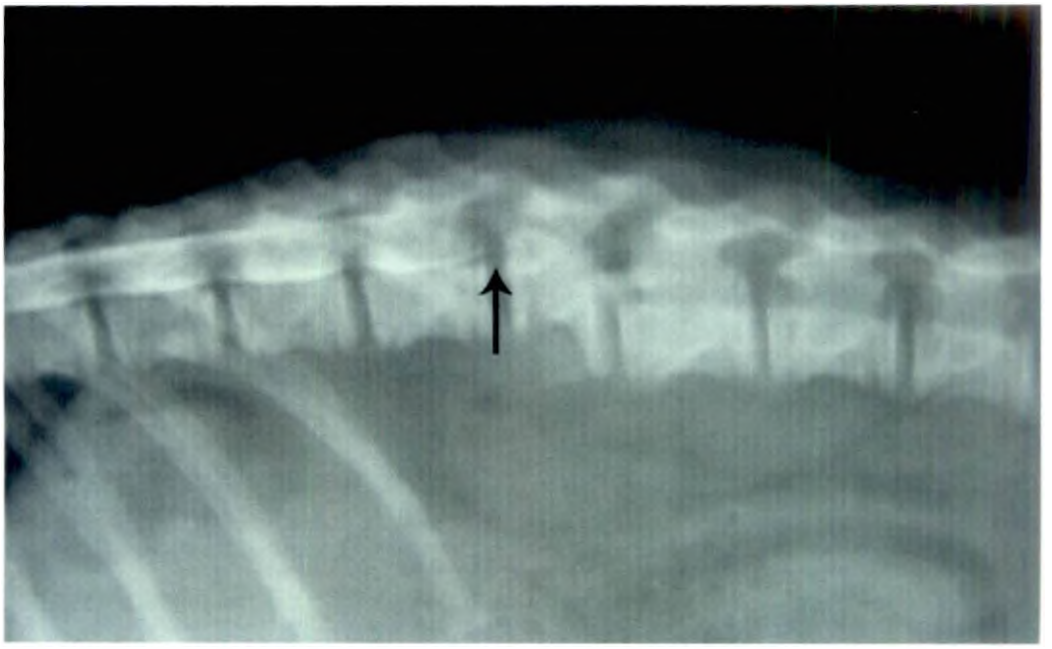


**Fig. 20. Skiagram of the cisternal myelography of Dog $I_7$ at 10th minute showing complete obstruction of contrast column at the level of 3rd lumbar vertebra.**

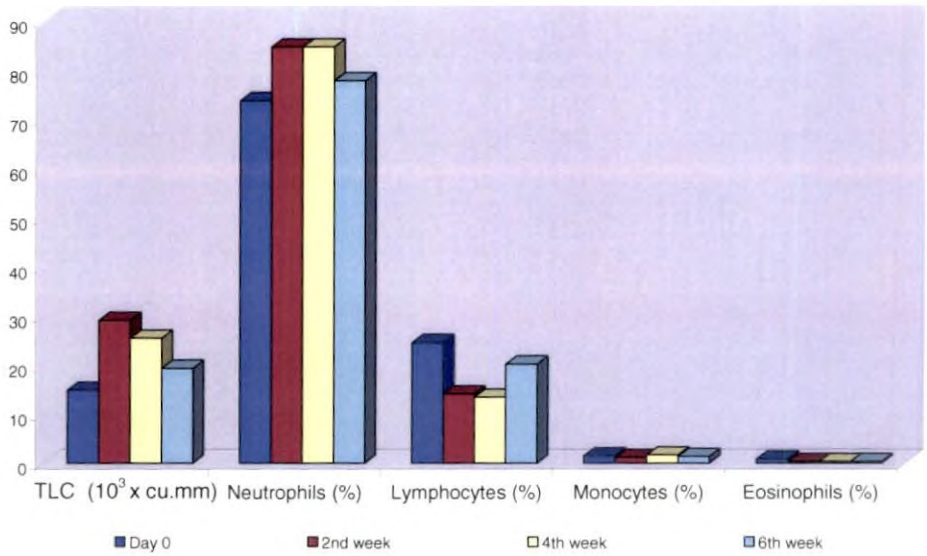



**Fig.21. Variation in TLC & DLC during different stages of study period in Group I dogs**

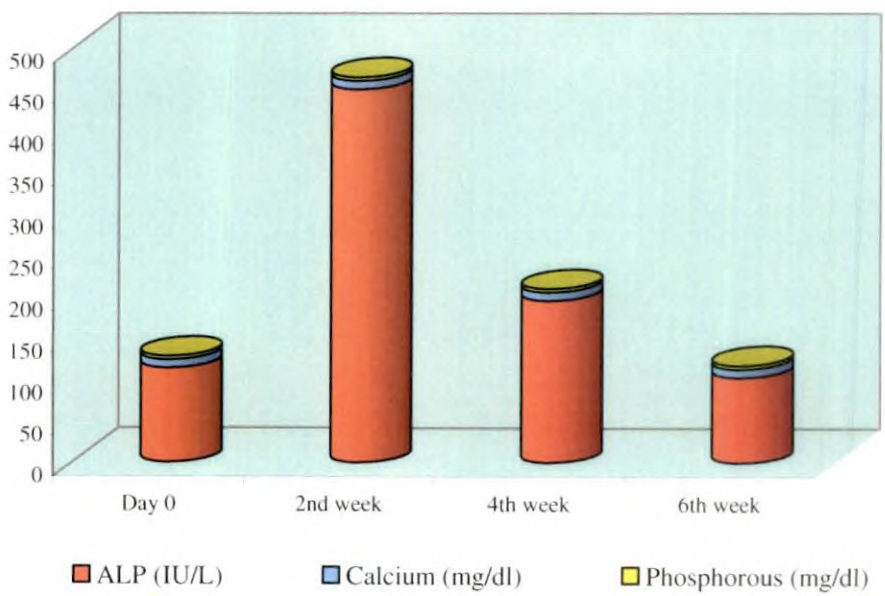



**Fig 22. Variation in serum calcium, phosphorous & alkaline phosphatase in Group I dogs.**

Fig. 23. Paraplegic dog on the day of presentation. (Dog $I_3$)

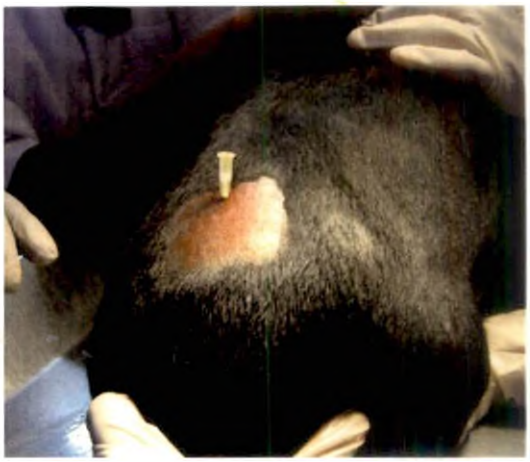


Fig. 24. Dog $I_3$ undergoing Epidural Injection. (Day 0)

Fig. 25. Dog $I_3$ able to stand on its own. ($4^{th}$ week)

Fig. 26. Dog $I_3$ walking normally. ($6^{th}$ week)

column and bones of the limbs were palpated to detect fracture and / or other abnormalities. Pain could be elicited on palpation of the affected part of the spine in all dogs of Group II.

*4.3.2.1. Bladder function:* The data obtained with respect to bladder function during the study period is depicted in Table 3.

In Group II, three dogs ($II_1$, $II_4$, $II_7$) had UMN bladder at the time of presentation, while three had LMN ($II_2$, $II_5$, $II_6$) and one had normal bladder function ($II_3$).

Dogs $II_1$, $II_2$ and $II_5$ had returned to voluntary urination with occasional dribbling at $4^{th}$ week and to normal bladder function at $6^{th}$ week. The dogs $II_4$ and $II_7$ had returned to voluntary urination with occasional dribbling at second week and to normal bladder function at fourth week. The dog $II_6$ died on the second day.

**4.3.3. Neurological examination**

Detailed neurological examinations of all the dogs were performed and the following data were obtained.

*4.3.3.1. Mental status, posture and gait:* The findings in mental status, posture and gait in dogs of Group II were recorded in Table 12.

Six dogs were alert at the time of presentation while one was depressed. All the dogs of this group were non-ambulatory and not able to stand or bear weight on the hind limbs, and dragged their hindquarters on the day of presentation.

*4.3.3.2. Postural reactions*

4.3.3.2.1. Wheel barrowing reaction

A normal response of symmetrical forelimb movement was observed in all the seven dogs (Table 13).

4.3.3.2.2. Proprioceptive positioning reaction

The observations of conscious proprioception in dogs of Group II are presented in Table 13

Conscious proprioception was absent in the hind limbs of all dogs on the day of presentation. By sixth week six dogs had regained conscious proprioception (delayed) in the hind limbs.

***4.3.3.3. Deep pain sensation:*** The observations of deep pain sensation are presented in Table 14.

Six dogs had deep pain sensation in the hind paws at the time of presentation and throughout the study period. The deep pain sensation was absent in the hind paws of dog $II_6$ on the day of presentation.

***4.3.3.4. Hyperpathia:*** Hyperpathia was noticed in the affected spinal region of all dogs (Table 15).

***4.3.3.5. Spinal reflexes:*** The forelimb spinal reflexes were normal in all the seven dogs. Spinal reflexes of the hind limbs were examined and described individually are tabulated in Table 14 and Table 15.

Dog $II_1$ - The spinal reflexes of the hind limbs were found to be depressed. The patellar reflex and flexor reflex were absent on the day of presentation. Tail wag reflex, perineal reflex and deep pain sensation were intact through out the

study period. The animal showed a spastic bladder on the day of presentation i.e. it was firm and difficult to express and there was brief intermittent spurting of urine. The lesion was localized between L4 and S2 cord segments.

Dog $II_2$.- The spinal reflexes of the hind limbs were found to be depressed The patellar reflex and flexor reflex were absent on the day of presentation. The patellar reflex was noticed at sixth week but sluggish. The flexor reflexes were found to be normal towards the end of the study period. Deep pain sensation was intact through out the study period. The tail wag reflex, which was found to be depressed on the day of presentation, became normal on fourth week. Perineal reflex was normal throughout the study period. The bladder was flaccid with overflow incontinence on the day of presentation. The lesion was localized between L4 and S2 cord segments.

Dog $II_3$ - The dog had normal bladder function. The patellar and flexor reflexes were sluggish on the day of presentation and became normal on second and fourth week respectively. The tail wag reflex, perineal reflex and deep pain perception were intact through out the study period. The lesion was localized between L4 and S2 cord segments.

Dog $II_4$ - The dog showed a spastic bladder on the day of presentation i.e. it was firm and difficult to express and there was brief intermittent spurting of urine. Urination became normal on fourth week. Patellar reflexes were sluggish on the day of presentation. Flexor reflexes were sluggish initially and became normal from second week onwards. The animal had normal perineal and tail wag reflexes. Deep pain sensation was intact throughout the study period. Lesion was localized between T3 and L3 cord segments.

Dog $II_5$ - The flexor and patellar reflexes which were found to be sluggish initially became normal on second and fourth week respectively. The bladder

was flaccid with overflow incontinence on the day of presentation and found to be normal on sixth week.Tailwag reflex; perineal reflex and deep pain sensation were intact through out the study period. The lesion was localized between L4-S2 cord segments.

Dog $II_6$ - The bladder was flaccid with overflow incontinence on the day of presentation. Patellar and flexor reflexes were absent on both hind limbs. Absence of deep pain perception. Normal tail wag and perineal reflexes. The lesion was localized between L4 and S2 cord segments.

Dog $II_7$ - The dog showed a spastic bladder on the day of presentation i.e. it was firm and difficult to express and there was brief intermittent spurting of urine. Urination became normal on fourth week. Patellar reflex was sluggish on the day of presentation. Deep pain perception and perineal reflexes were intact throughout the study period. Depressed tail wag and flexor reflexes on the day of presentation became normal on second week. The lesion was localized between L4 and S2 cord segments.

### 4.3.4. Radiographic evaluation

*4.3.4.1. Survey radiography:* The observations of cases have been recorded individually. (Table 16)

Dog $II_1$ – Calcification of intervertebral disc between tenth and eleventh & eleventh and twelth thoracic vertebrae.

Dog $II_2$ – Calcification of the sixth lumbar intervertebral disc. Varying degrees of calcification noticed in the second, third, fourth and fifth lumbar intervertebral disc spaces (Fig.27 & 28).

Dog $II_3$ . Narrowing of last thoracic, thoracolumbar and first lumbar intervertebral disc space. Vertebral lipping noticed. Moderate calcification of the $6^{th}$ lumbar intervertebral disc space (Fig.29).

Dog $II_4$ – Narrowing of eleventh and twelth thoracic intervertebral disc spaces. Calcification of the sixth lumbar intervertebral disc space.Ostitic changes noticed on the thoracolumbar region.

Dog $II_5$ . Calcification of the second lumbar intervertebral disc (Fig.30).

Dog $II_6$ – Calcification and narrowing of first, second and third lumbar intervertebral disc spaces (Fig.31).

Dog $II_7$ . Calcification of eleventh and twelth thoracic intervertebral discs.

*4.3.4.2. **Myelography:*** The dog $II_6$ was subjected to myelography using Iohexol at the dose rate of 0.3ml/kg body weight (90mg I / kg body weight). Both cisterna magna puncture and lumbar puncture were used for the procedure.

The anaesthesia was sufficient for restraining the dog for myelography. The method adopted for locating the sites was accurate in achieving a successful puncture. CSF sample collected was clear and without visible contamination with blood.

Serial radiographs were taken at zero, fifth and tenth minutes following cisterna magna puncture. The zero minute radiograph showed no contrast medium flow in the spinal canal. Flow of contrast material noticed up to the level of tenth thoracic vertebrae in the fifth minute radiograph. No further improvement in the flow of contrast medium was noticed in the tenth minute radiograph.

Radiographs were taken at fifth, tenth and fifteenth minute following lumbar puncture. Upward elevation of the ventral contrast column at third, fourth and fifth lumbar intervertebral disc, and deviation of the dorsal contrast column and narrowing of ventral contrast column at the level of fourth lumbar intervertebral disc were noticed in the fifth minute radiograph. No further changes were noticed in the tenth and fifteenth minute radiograph (Fig.32).

### 4.3.5. Correlation of radiographic pattern and neurological findings

Lesion was localized between L4 and S2 cord segments by neurological examination in all dogs except $II_4$.Calcification and protrusion of the intervertebral disc and narrowing of disc space in the lumbar and thoracic region were the changes noticed in these animals on survey radiography. Lesion was localized between T3 and L3 cord segments in dog $II_4$ and narrowing and calcification of the thoracic and lumbar intervertebral disc were noticed on survey radiography.

### 4.3.6. Neurological grading of patients

The details of neurological grading are given in Table 9.

Six dogs ($II_1$, $II_2$, $II_4$, $II_5$, $II_6$ $II_7$) were graded V, while one ($II_3$) was graded IV on the day of presentation. On sixth week, four were ($II_1$, $II_2$, $II_5$, $II_7$) graded II and two were ($II_3$, $II_4$) graded I. Dog $II_6$ died on second day.

### 4.3.7. Haematological parameters

The results of haematological studies are presented in Table 10.

Wet film examination of blood was negative in all the seven dogs.

The mean value for haemoglobin concentration (g/dl) ranged from 15.73 ± 0.25 to14.76 ± 0.26 during the study period. The variations observed were marginal and within the normal range.

The mean value for packed cell volume (per cent) ranged from 44.33 ± 1.70 to 40.66 ± 0.61 during the study period. The variations observed were marginal and within the normal range.

The mean total leukocyte counts ($10^3$/cu.mm) were 14.23 ± 0.66, 29.96 ± 3.23, 26.45 ± 2.13 and 18.46 ± 0.68 on the day of presentation, second week, fourth week and sixth week respectively. The total leukocyte count was within the normal range initially. The values showed a significant increase on second and reduced on fourth week and returned towards normal level by the end of the study period.(Fig.33)

The mean value for neutrophil counts was (per cent) 73.50 ± 1.28, 84.83 ± 1.10, 83.66 ± 0.42 and 77.66 ± 0.84on the day of presentation, second week, fourth week and sixth week respectively. The neutrophil count was within the normal range initially. The values were increased on second and fourth week and lowered to normal level by the end of the study period.

The mean lymphocyte counts ($10^3$/cu.mm) were 26.50 ± 1.87, 13.83 ± 1.01, 14.00 ± 0.36 and 18.83 ± 1.60 on the day of presentation, second week, fourth week and sixth week respectively. The lymphocyte count was within the normal range initially. The values decreased on second and fourth week and returned to normal level by sixth week and the variations were corresponding to the variations in the value of neutrophil count.

The mean value for monocyte counts (per cent) ranged from 1.00 ± 0.25 to1.83 ± 0.16 during the study period. The variations observed were marginal and within the normal range.

The mean value for oesinophil counts (per cent) ranged from 0.50 ± 0.22 to 0.16 ± 0.16 during the study period. The variations observed were marginal and within the normal range.

**4.3.8. Serum biochemical evaluation**

The results of serum biochemical evaluation are presented in Table11.

The mean serum calcium values (mg/dl) ranged from 9.95 ± 0.22 to 9.93 ± 0.17 during the study period. The variations observed were marginal and within the normal range.

The mean serum phosphorous values (mg/dl) ranged from 4.55 ± 0.41 to 4.41 ± 0.35 during the study period. The variations observed were marginal and within the normal range.

The mean values for alkaline phosphatase (IU/L) were 115.83 ± 7.54, 425.66 ± 29.37, 179.5 ± 6.51 and 138.83 ± 5.14 on the day of presentation, second week, fourth week and sixth week respectively. There is an increase on second week, but from third week onwards it lowered and became normal by the end of the study period. (Fig 34)

**4.3.9. Treatment**

Therapeutic ultrasound massage was given on either side of the dorsal spinal region of all dogs of Group II. Two dogs ($II_1$, $II_5$) had developed allergic reaction while using gel as the coupling agent.

Out of the seven dogs in group II, six dogs ($II_1$, $II_2$, $II_3$, $II_4$,$II_5$ $II_7$) became ambulatory at the end of the study period. Two animals ($II_3$, $II_4$) had normal gait while two ($II_5$, $II_7$) had occasional ataxia and two ( $II_1$, $II_2$) had ataxia on walking.

The progressive improvement of animals of Group II is shown in Fig.35 to 42.

## 4.4. COMPLICATIONS

Almost all the dogs of Group I and Group II had developed wounds at paws and hock joints due to the dragging as a result of neurological deficit. Wound dressing with zinc oxide ointment and bandaging was done in these dogs. Allergic reaction to gel was noticed in two dogs which was subsided when the use of the gel was stopped (Fig.43).

## 4.5. RECURRENCE

Recurrence of paraplegia was noticed in two dogs ($I_1$, $II_2$). The condition recurred in dog $I_1$ after four month and in dog $II_2$ after eight month. Epidural injection of methyl prednisolone acetate (single) was given to these two dogs. The dog $I_1$ recovered within one week, but no improvement was seen in case of dog $II_2$.

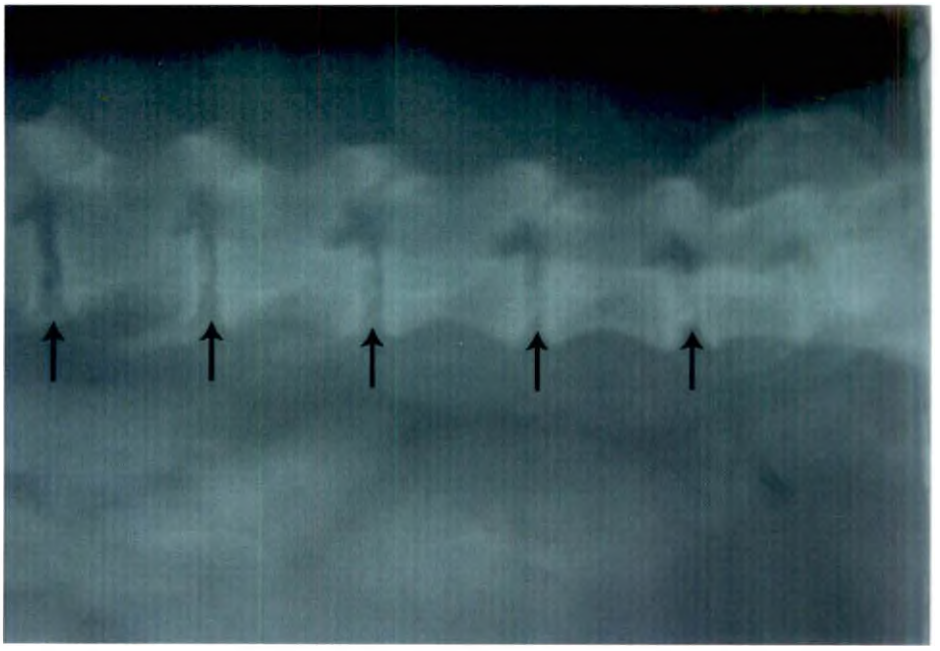


**Fig. 27. Skiagram showing varying degrees of calcification of 2nd to 6th lumbar intervertebral discs. (Dog $II_2$, day 0)**

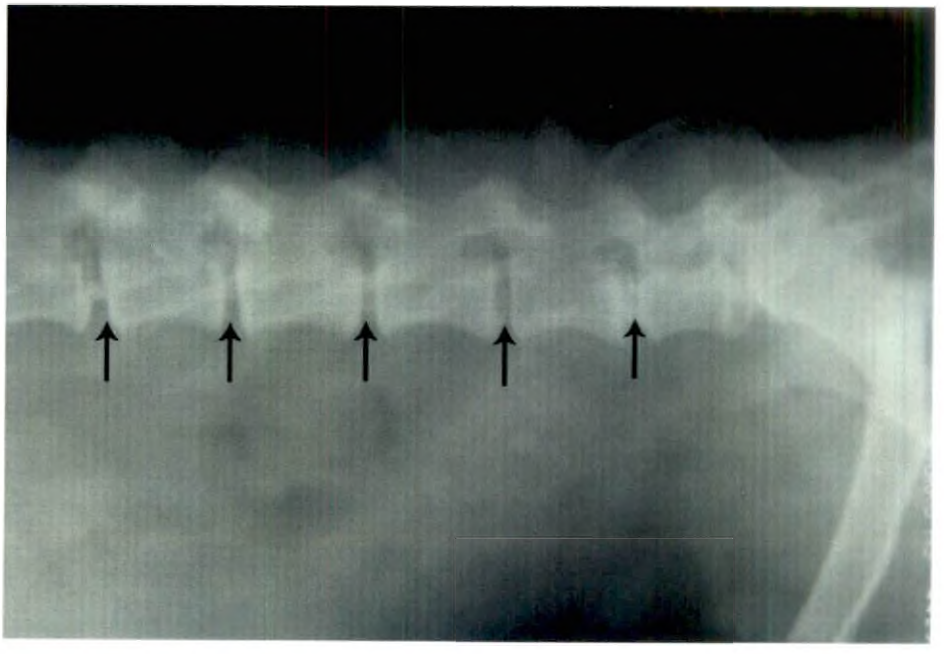


**Fig. 28. Skiagram showing varying degrees of calcification of 2nd to 6th lumbar intervertebral discs. (Dog $II_2$, 6th week)**

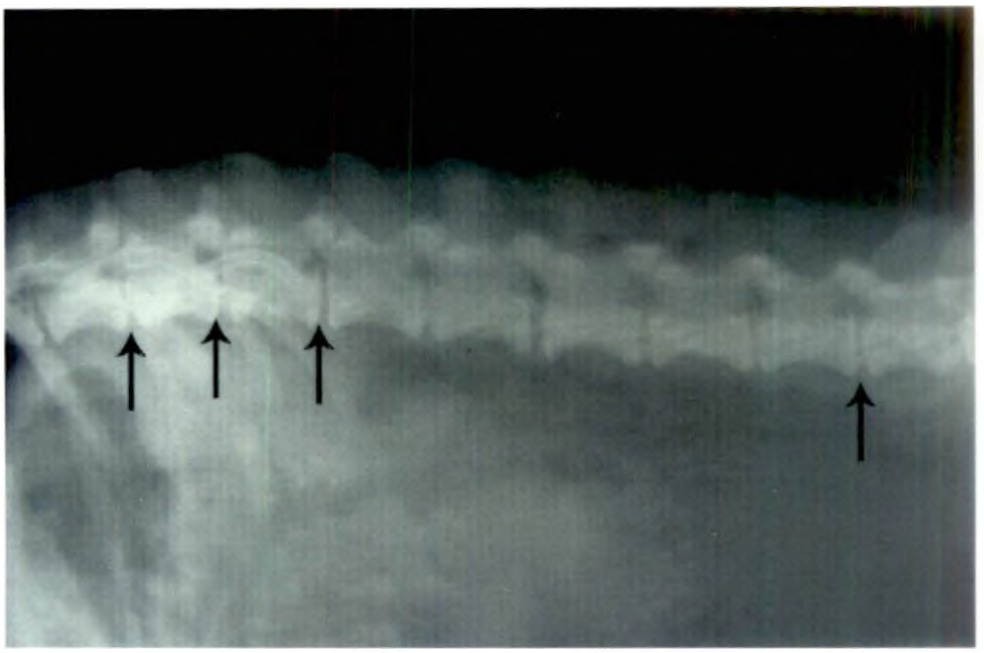


**Fig. 29. Skiagram showing narrowing of last thoracic, thoracolumbar and I$^{st}$ lumbar intervertebral disc. (Dog $II_3$)**

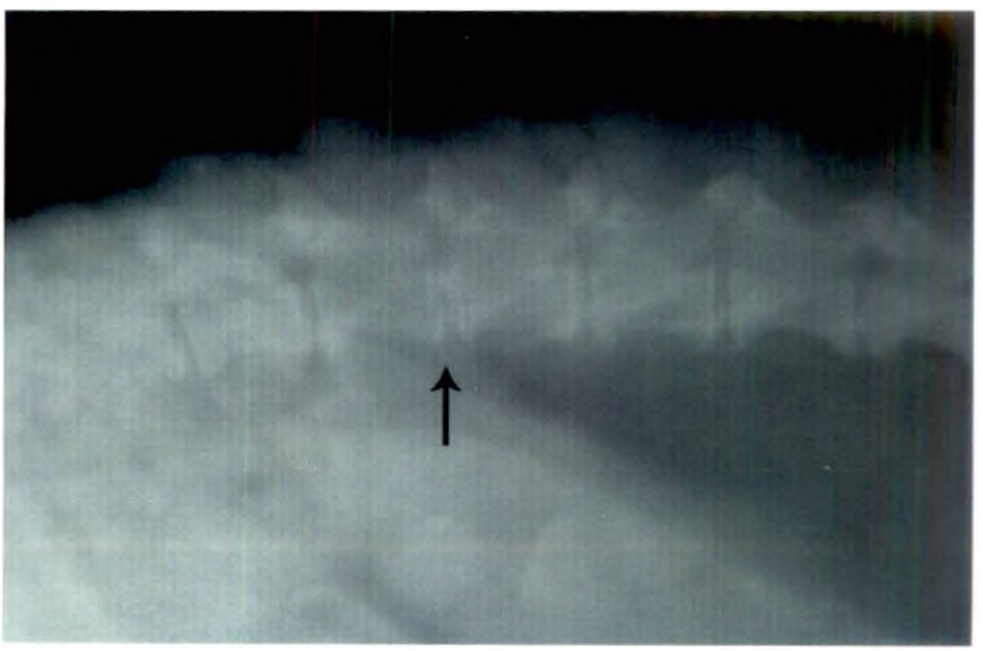


**Fig. 30. Skiagram showing calcification of 2$^{nd}$ lumbar intervertebral disc. (Dog $II_5$)**

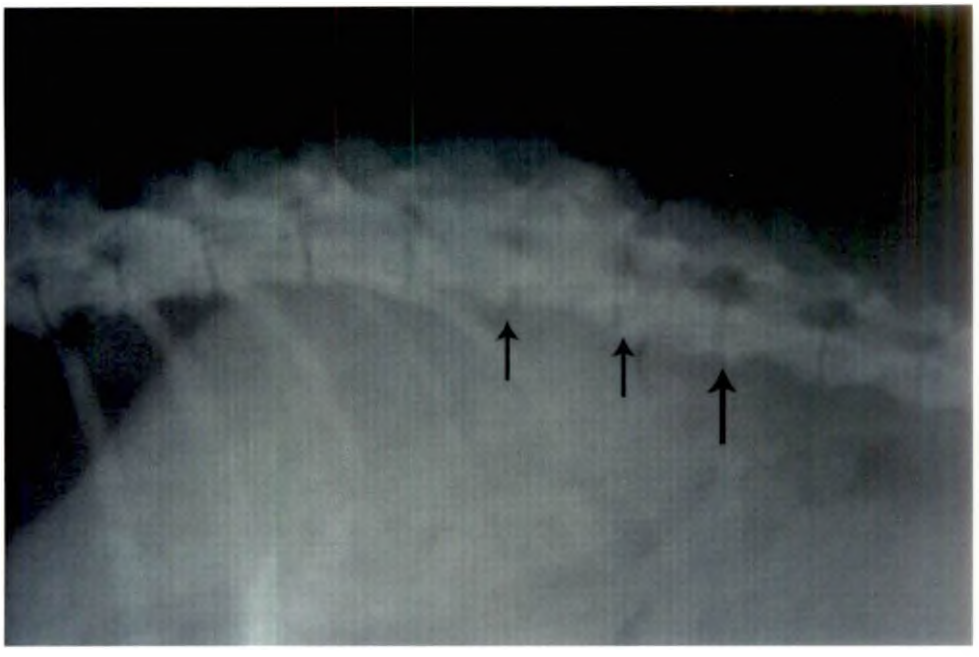


Fig. 31. Skiagram showing calcification & narrowing of I$^{st}$, 2$^{nd}$ and 3$^{rd}$ lumbar intervertebral disc spaces. (Dog $II_6$)

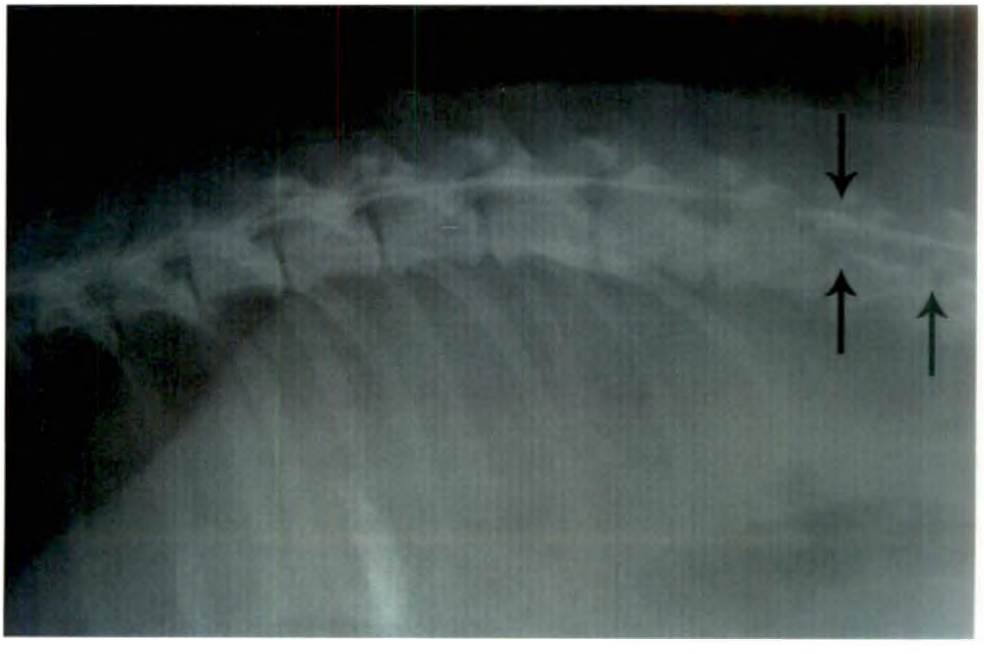


Fig. 32. Skiagram of the lumbar myelography of Dog $II_6$ at 10$^{th}$ minute showing deviation of dorsal contrast column and elevation & narrowing of ventral contrast column at the level of 2$^{nd}$ & 3$^{rd}$ lumbar intervertebral disc.

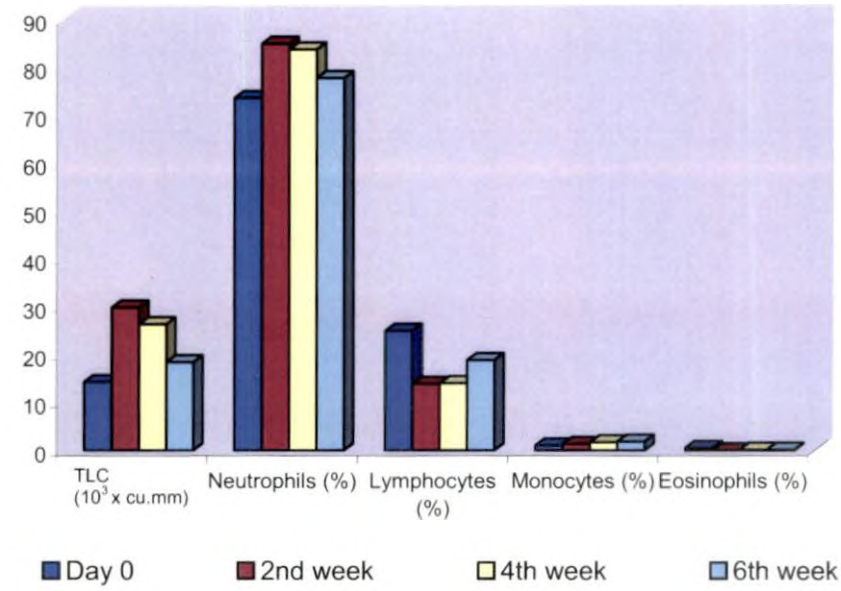



**Fig 33. Variation in TLC & DLC during different stages of study period in Group II dogs.**

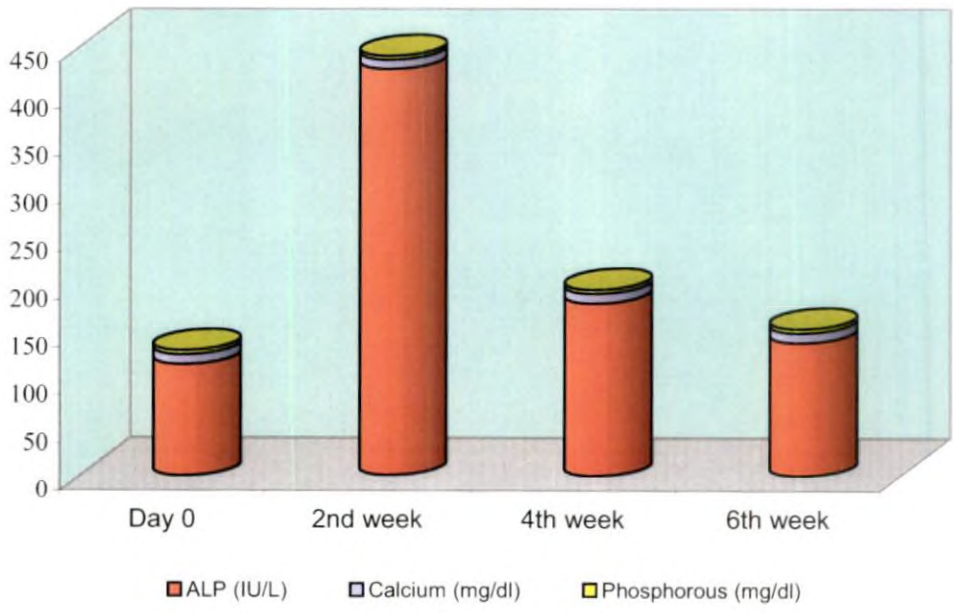



**Fig. 34 Variation in serum calcium, phosphorous & alkaline phosphatase in Group II dogs.**

Fig. 35. Paraplegic dog on the day of presentation. (Dog $II_2$)

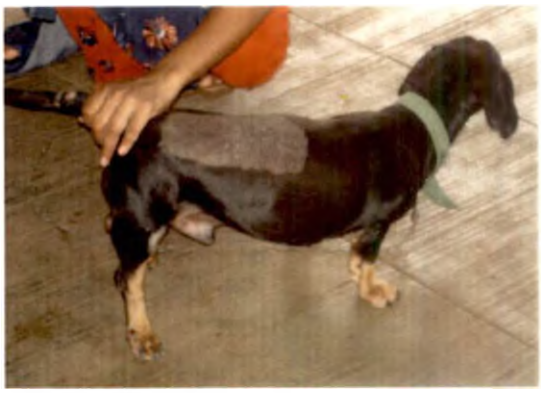


Fig. 36. Dog $II_2$ able to stand with help. ($4^{th}$ week)

Fig. 37. Dog $II_2$ trying to raise its hind quarters. ($5^{th}$ week)

Fig. 38. Dog $II_2$ able to stand on its own (towards the end of $5^{th}$ week).

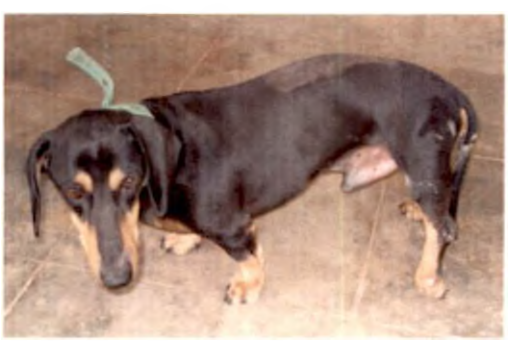


Fig. 39. Dog $II_2$ with occassional pro prioceptive deficit. ($6^{th}$ week)

Fig. 40. Dog $II_2$ able to climb the steps (towards the end of $6^{th}$ week)

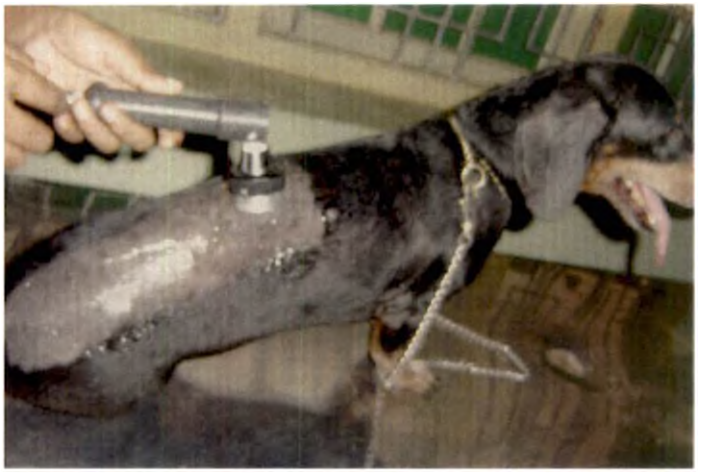


**Fig. 41. Ultrasound massage in progress on the day of presentation. (Dog $II_3$)**

**Fig. 42. Weight bearing of Dog $II_3$ at $6^{th}$ week**

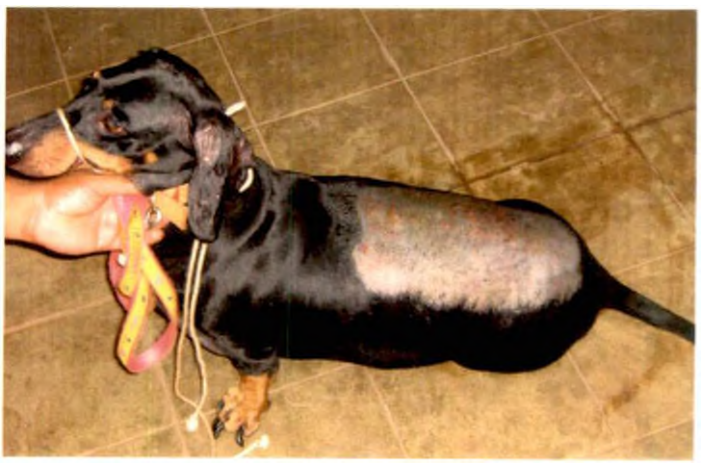


**Fig. 43. Allergic reaction to ultrasound gel (Dog $II_1$)**

Table 1. Observations on breed, age, sex, body weight, duration of illness and etiology in dogs of Group I and II

| Serial number | Animal number | Age ( years ) | Breed | Sex | Body weight ( kg) | Duration of illness ( days ) | Etiology |
|---|---|---|---|---|---|---|---|
| 1 | $I_1$ | 4.5 | Dachshund | Male | 10 | 1 | Fallen from sofa |
| 2 | $I_2$ | 7 | Lhasa apso | Male | 11.6 | 1 | Automobile accident |
| 3 | $I_3$ | 4 | Dachshund | Female | 10 | 2 | Fallen from cage |
| 4 | $I_4$ | 5 | Dachshund | Male | 10 | 2 | Unknown |
| 5 | $I_5$ | 4 | Dachshund | Female | 8 | 2 | Unknown |
| 6 | $I_6$ | 5 | Non descript | Female | 13.5 | 2 month | Unknown |
| 7 | $I_7$ | 3 month | Non descript | Female | 6.5 | 1 | hitten by neighbour |
| 8 | $II_1$ | 7 | Dachshund | Male | 17 | 2 | Unknown |
| 9 | $II_2$ | 7.5 | Dachshund | Male | 12 | 2 | Unknown |
| 10 | $II_3$ | 3 | Dachshund | Female | 10 | 2 | Unknown |
| 11 | $II_4$ | 6 | Dachshund | Female | 10 | 4 | Unknown |
| 12 | $II_5$ | 4 | Dachshund | Female | 10 | 4 | Unknown |
| 13 | $II_6$ | 5 | Dachshund | Male | 12 | 7 | Fallen from sofa |
| 14 | $II_7$ | 4 | Dachshund | Male | 13 | 7 | Unknown |
| Average age - 4.7 | | | Average body weight - 11 | | | | |

Table 2. Pre and post injection observations on physiological parameters of GroupI and Group II dogs (Mean ± SE) n=6

| Parameter | Group | Pre injection period | Post injection period | | |
|---|---|---|---|---|---|
| | | | $2^{nd}$ week | $4^{th}$ week | $6^{th}$ week |
| Respiratory rate (per min) | I | 27.16±1.88 | 28.50±1.64 | 25.83±1.57 | 28.00±2.39 |
| | II | 26.66±1.54 | 30.33±1.89 | 29.16±1.04 | 23.66±1.02 |
| Pulse rate (per min) | I | 95.00±5.80 | 108.33±5.07 | 106.50±3.55 | 109.16±3.72 |
| | II | 104.16±3.62 | 98.50±5.84 | 102.83±3.84 | 98.83±4.65 |
| Rectal temperature ($^0$C) | I | 39.18±0.21 | 38.80±0.12 | 38.75±0.12 | 38.18±0.15 |
| | II | 38.61±0.17 | 38.90±0.13 | 38.70±0.10 | 38.63±0.11 |

Table 3. Observations on bladder function in dogs with paraplegia (Group I and Group II)

| Animal number | On the day of presentation | At 24 hour | 2nd week | 4th week | 6th week |
|---|---|---|---|---|---|
| $I_1$ | +++ | +++ | +++ | +++ | +++ |
| $I_2$ | +++ | +++ | +++ | +++ | +++ |
| $I_3$ | - | - | ++ | +++ | +++ |
| $I_4$ | + | + | ++ | ++ | +++ |
| $I_5$ | - | - | - | ++ | +++ |
| $I_6$ | +++ | +++ | +++ | +++ | +++ |
| $I_7$ | - | - | * | * | * |
| $II_1$ | + | + | + | ++ | +++ |
| $II_2$ | - | - | - | ++ | +++ |
| $II_3$ | +++ | +++ | +++ | +++ | +++ |
| $II_4$ | + | + | ++ | +++ | +++ |
| $II_5$ | - | - | - | ++ | +++ |
| $II_6$ | - | - | * | * | * |
| $II_7$ | + | + | ++ | +++ | +++ |

\- = constant dribbling, empty bladder (LMN)
\+ = constant dribbling, full bladder (UMN
++ = voluntary urination with occasional dribbling
+++ = normal urination * Not available (Animal died)

Table 4. Observations on mental status, posture and gait of dogs in Group I

| Animal number | Mental status | | | | | Posture | | | | | Gait | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | At 24 hour | 2nd week | 4th week | 6th week |
| $I_1$ | A | A | A | A | A | + | + | +++ | +++ | +++ | + | + | ++ | +++ | +++ |
| $I_2$ | A | A | A | D | D | + | + | + | + | + | + | + | + | + | + |
| $I_3$ | A | A | A | A | A | + | + | +++ | +++ | +++ | + | + | +++ | +++ | ++++ |
| $I_4$ | A | A | A | A | A | + | + | ++ | +++ | +++ | + | + | + | ++ | +++ |
| $I_5$ | A | A | A | A | A | + | + | ++ | +++ | +++ | + | + | + | ++ | +++ |
| $I_6$ | A | A | A | A | A | + | + | ++ | +++ | +++ | + | + | + | ++ | +++ |
| $I_7$ | D | D | * | * | * | + | + | * | * | * | + | + | * | * | * |

Mental status

A - Alert
D - Depressed
C - Comatose
* Not available (Animal died)

Posture

\+ = Recumbent/sitting
++ = Stand with help
+++ = Stand on its own

Gait

\+ = Dragging of hindquarters
++ = Ataxia
+++ = Occasional ataxia
++++ = Normal gait

Table5. Observations on postural reactions in dogs in Group I

| Animal number | Wheel barrowing reaction | | | | | Proprioceptive positioning reaction | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | | At 24 hour | | 2nd week | | 4th week | | 6th week | |
| | | | | | | R | L | R | L | R | L | R | L | R | L |
| $I_1$ | P | P | P | P | P | - | - | - | - | ± | ± | ± | ± | + | + |
| $I_2$ | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| $I_3$ | P | P | P | P | P | - | - | - | - | - | - | ± | ± | + | + |
| $I_4$ | P | P | P | P | P | - | - | - | - | - | - | ± | - | + | ± |
| $I_5$ | P | P | P | P | P | - | - | - | - | - | - | - | - | ± | ± |
| $I_6$ | P | P | P | P | P | - | - | - | - | - | - | - | - | ± | ± |
| $I_7$ | - | - | * | * | * | - | - | * | * | * | * | * | * | * | * |

Present - P
Absent - A

+ = Normal
± = Delayed
- = Absent

R - Right
L - Left

* Not available (Animal died)

Table6. (a) Observations on spinal reflexes of dogs in Group I

| Animal number | Patellar reflex | | | | | | | | | | Flexor reflex | | | | | | | | | | Deep pain sensation | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 0 | | At 24 hour | | 2nd week | | 4th week | | 6th week | | Day 0 | | At 24 hour | | 2nd week | | 4th week | | 6th week | | Day 0 | At 24 hour | 2nd week | 4th week | 6th week |
| | R | L | R | L | R | L | R | L | R | L | R | L | R | L | R | L | R | L | R | L | | | | | |
| $I_1$ | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P |
| $I_2$ | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | P | P | P | P | P |
| $I_3$ | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P |
| $I_4$ | 3 | 3 | 3 | 3 | 2 | 2 | 2 | 2 | 2 | 2 | 0 | 1 | 0 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | P | P | P | P | P |
| $I_5$ | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 0 | 0 | 0 | 0 | 1 | 1 | 2 | 2 | 2 | 2 | P | P | P | P | P |
| $I_6$ | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | P | P | P | P | P |
| $I_7$ | 1 | 1 | 1 | 1 | * | * | * | * | * | * | 2 | 2 | 1 | 1 | * | * | * | * | * | * | P | P | * | * | * |

0 - Absent
1 - Depressed
2 - Normal
3 - Hyperreflexic
P - Present
A – Absent
* Not available (Animal

Table7. (b) Observations on spinal reflexes of dogs in Group I

| Animal number | Tail wag reflex | | | | | Hyperpathia | | | | | Perineal reflex | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | At 24 hour | 2nd week | 4th week | 6th week |
| $I_1$ | 0 | 0 | 1 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $I_2$ | 2 | 2 | 1 | 1 | 1 | P | P | P | P | P | P | P | P | P | P |
| $I_3$ | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $I_4$ | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $I_5$ | 1 | 1 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $I_6$ | 1 | 1 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $I_7$ | 1 | 1 | * | * | * | P | P | * | * | * | P | P | * | * | * |

0 - Absent
1 - Depressed
2 - Normal
3 - Hyperreflexic
P - Present
A – Absent

* Not available (Animal died)

Table 8. Correlation of lesions identified with neurological examination and radiography in dogs of Group I

| Animal number | Neurological examination and correlation | Radiographic findings |
|---|---|---|
| $I_1$ | Localized between L4-S2 cord segments. Positively correlated with survey radiography | Protrusion of 3rd and 4th lumbar intervertebral disc in to the spinal canal. Calcification of 5th and 6th lumbar intervertebral disc. |
| $I_2$ | Localized between L4-S2 cord segments. Positively correlated with survey radiography | Luxation of the 4th lumbar vertebra. |
| $I_3$ | Localized between L4-S2 cord segments. . Positively correlated with survey radiography | Calcification of intervertebral disc between 5th and 6th lumbar vertebrae. Reduction and calcification of last thoracic intervertebral disc space and protrusion of 3rd lumbar intervertebral disc into the spinal canal. |
| $I_4$ | Localized betweenT3-L3 cord segments. Positively correlated with survey radiography | Calcification of 12th thoracic intervertebral disc. |
| $I_5$ | Localized between L4-S2 cord segments. Positively correlated with survey radiography | Protrusion of the 2nd, 3rd, 4th and 5th lumbar intervertebral disc into the spinal canal. Calcification of the 6th lumbar intervertebral disc. |
| $I_6$ | Localized between L4-S2 cord segments. Positively correlated with survey radiography | Narrowing of 10th and 11th thoracic intervertebral disc space and 3rd ,4th,5th and 6th lumbar intrvertebral disc spaces were noticed. |
| $I_7$ | Localized between L4-S2 cord segments. Positively correlated with survey radiography | Complete longitudinal fracture of body of last thoracic, 1st ,2nd and 3rd lumbar vertebrae. On myelography partial obstruction of contrast column at the level of 3rd lumbar vertebrae. |

Table 9. Neurological grades of dogs with paraplegia during different stages of study period

| Group I | Day 0 | 2nd week | 4th week | 6thweek | Group II | Day 0 | 2nd week | 4th week | 6th week |
|---|---|---|---|---|---|---|---|---|---|
| $I_1$ | ++++ | ++ | ++ | + | $II_1$ | +++++ | +++++ | ++++ | ++ |
| $I_2$ | ++++ | + +++ | ++++ | ++++ | $II_2$ | +++++ | +++++ | ++++ | ++ |
| $I_3$ | +++++ | ++ | ++ | ++ | $II_3$ | ++++ | +++ | ++ | + |
| $I_4$ | +++++ | +++ | ++ | ++ | $II_4$ | +++++ | ++++ | ++ | + |
| $I_5$ | +++++ | +++++ | +++ | ++ | $II_5$ | +++++ | +++++ | ++ | ++ |
| $I_6$ | ++++ | +++ | + | + | $II_6$ | +++++ | * | * | * |
| $I_7$ | +++++ | * | * | * | $II_7$ | +++++ | +++ | ++ | ++ |

Grade I - (+) spinal hyperesthesia only
Grade II - (+ +) ambulatory paraparesis
Grade III - (+ + +) non-ambulatory paraparesis
Grade IV - (+ + + +) paraplegia
Grade V - (+ + + + +) paraplegia with urinary incontinence

* Not available (Animal died)

Table 10. Pre and post injection observations on haematological parameters. (Mean ± SE)
n=6

| Parameter | | Group | Pre injection period | Post injection period | | |
|---|---|---|---|---|---|---|
| | | | | 2nd week | 4th week | 6th week |
| Haemoglobin concentration (g/dl) | | I | 14.90±0.71 | 13.90±0.43 | 14.33±0.57 | 13.73±0.80 |
| | | II | 15.73±0.25 | 13.70±0.31 | 14.16±0.35 | 14.76±0.26 |
| Packed cell Volume (%) | | I | 42.66±0.98 | 39.33±1.22 | 41.00±0.81 | 39.66±0.49 |
| | | II | 44.33±1.70 | 41.66±0.95 | 41.16±0.79 | 40.66±0.61 |
| Total leukocyte count ($10^3$/cu.mm) | | I | 14.93±1.16 | 29.11±3.08* | 25.48±2.28 | 19.36±1.99 |
| | | II | 14.23±0.66 | 29.96±3.23* | 26.45±2.13 | 18.46±0.68 |
| Differential leukocyte count (per cent) | N | I | 73.66±0.84 | 84.50±1.47 | 84.50±1.05 | 77.66±1.81 |
| | | II | 73.50±1.28 | 84.83±1.10 | 83.66±0.42 | 77.66±0.84 |
| | L | I | 24.50±0.80 | 14.16±1.37 | 13.50±0.95 | 20.16±1.77 |
| | | II | 26.50±1.87 | 13.83±1.01 | 14.00±0.36 | 18.83±1.60 |
| | M | I | 1.50±0.22 | 1.16±0.30 | 1.66±0.42 | 1.33±0.42 |
| | | II | 1.00±0.25 | 1.33±0.33 | 1.66±0.21 | 1.83±0.16 |
| | E | I | 0.83±0.16 | 0.33±0.21 | 0.33±0.21 | 0.33±0.21 |
| | | II | 0.50±0.22 | 0±0.00 | 0.16±0.16 | 0.16±0.16 |

P < 0.05

Table 11. Pre and post injection observations on serum biochemical evaluation
(Mean ± SE), n = 6

| Parameter | Group | Pre injection period | Post injection period | | |
|---|---|---|---|---|---|
| | | | 2nd week | 4th week | 6th week |
| Calcium (mg/dl) | I | 9.96±0.26 | 10.30±0.37 | 10.25±0.29 | 10.45±0.42 |
| | II | 9.95± 0.22 | 9.78±0.35 | 10.11±0.33 | 9.93±0.17 |
| Phosphorous (mg/dl) | I | 4.13±0.41 | 4.40±0.40 | 4.08±0.24 | 4.25±0.29 |
| | II | 4.55±0.41 | 4.20±0.38 | 4.11±0.34 | 4.41±0.35 |
| Alkaline phosphatase (IU/L) | I | 114.0±8.73 | 449.16±28.65 | 194.83±5.81 | 102.66±4.84 |
| | II | 115.83±7.54 | 425.66±29.37 | 179.5±6.51 | 138.83±5.14 |

Table 12. Observations on mental status, posture and gait of dogs in Group II

| Animal number | Mental status | | | | | Posture | | | | | Gait | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | At 24 hour | 2nd week | 4th week | 6th week |
| $II_1$ | A | A | A | A | A | + | + | + | ++ | +++ | + | + | + | + | ++ |
| $II_2$ | A | A | A | A | A | + | + | + | ++ | +++ | + | + | + | + | ++ |
| $II_3$ | A | A | A | A | A | + | + | ++ | +++ | +++ | + | + | + | ++ | ++++ |
| $II_4$ | A | A | A | A | A | + | + | ++ | +++ | +++ | + | + | + | ++ | ++++ |
| $II_5$ | A | A | A | A | A | + | + | ++ | +++ | +++ | + | + | + | ++ | +++ |
| $II_6$ | D | D | * | * | * | + | + | * | * | * | + | + | * | * | * |
| $II_7$ | A | A | A | A | A | + | + | ++ | +++ | +++ | + | + | + | ++ | +++ |

Mental status

A - Alert
D - Depressed
C - Comatose
* - Not available (Animal died)

Posture

+ = Recumbent/sitting
++ = Stand with help
+++ = Stand on its own

Gait

+ = Dragging of hindquarters
++ = Ataxia
+++ = Occasional ataxia
++++ = Normal gait

Table 13. Observations on postural reactions in dogs - Group II

| Animal number | Wheel barrowing reaction | | | | | Proprioceptive positioning reaction | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | | At 24 hour | | 2nd week | | 4th week | | 6th week | |
| | | | | | | R | L | R | L | R | L | R | L | R | L |
| $II_1$ | P | P | P | P | P | - | - | - | - | - | - | ± | ± | ± | ± |
| $II_2$ | P | P | P | P | P | - | - | - | - | - | - | - | - | ± | ± |
| $II_3$ | P | P | P | P | P | - | - | - | - | - | - | - | - | ± | ± |
| $II_4$ | P | P | P | P | P | - | - | - | - | - | - | - | - | ± | ± |
| $II_5$ | P | P | P | P | P | - | - | - | - | - | - | - | - | ± | ± |
| $II_6$ | P | P | * | * | * | - | - | - | - | * | * | * | * | * | * |
| $II_7$ | P | P | P | P | P | - | - | - | - | - | - | - | - | ± | ± |

Present - P
Absent - A

\+ = Normal
± = Delayed
\- = Absent

R - Right
L – Left

* Not available (Animal died)

Table 14. (a) Observations on spinal reflexes of animals in Group II

| Animal number | Patellar reflex | | | | | | | | | | Flexor reflex | | | | | | | | | | Deep pain sensation | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 0 | | At 24 hour | | 2nd week | | 4th week | | 6th week | | Day 0 | | At 24 hour | | 2nd week | | 4th week | | 6th week | | Day 0 | At 24 hour | 2nd week | 4th week | 6th week |
| | R | L | R | L | R | L | R | L | R | L | R | L | R | L | R | L | R | L | R | L | | | | | |
| $II_1$ | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 2 | 2 | P | P | P | P | P |
| $II_2$ | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 1 | 2 | 2 | P | P | P | P | P |
| $II_3$ | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | P | P | P | P | P |
| $II_4$ | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P |
| $II_5$ | 1 | 1 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P |
| $II_6$ | 0 | 0 | 0 | 0 | * | * | * | * | * | * | 0 | 0 | 0 | 0 | * | * | * | * | * | * | A | A | * | * | * |
| $II_7$ | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | 1 | 1 | 1 | 1 | 2 | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P |

0 - Absent
1 - Depressed
2 - Normal
3 - Hyperreflexic

P - Present
A – Absent

* Not available (Animal died)

Table15. (b) Observations on spinal reflexes of dogs in Group II

| Animal number | Tail wag reflex | | | | | Hyperpathia | | | | | Perineal reflex | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | At 24 hour | 2nd week | 4th week | 6th week | Day 0 | At 24 hour | 2nd week | 4th week | 6th week |
| $II_1$ | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $II_2$ | 1 | 1 | 1 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $II_3$ | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $II_4$ | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $II_5$ | 2 | 2 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |
| $II_6$ | 2 | 2 | * | * | * | P | P | * | * | * | P | P | * | * | * |
| $II_7$ | 1 | 1 | 2 | 2 | 2 | P | P | P | P | P | P | P | P | P | P |

0 - Absent
1 - Depressed
2 - Normal
3 - Hyperreflexic
P - Present
A – Absent

* Not available (Animal died)

Table 16. Correlation of lesions identified with neurological examination and radiography in dogs of Group II

| Animal number | Neurological examination and correlation | Radiographic findings |
|---|---|---|
| $II_1$ | Localized between L4-S2 cord segments. Could not be correlated with survey radiography | Calcification of disc space between $10^{th}$& $11^{th}$ and $11^{th}$ &$12^{th}$ thoracic vertebrae. |
| $II_2$ | Localized between L4-S2 cord segments. Positively correlated with survey radiography | Calcification of $6^{th}$ lumbar intervertebral disc. Varying degrees of calcification of $2^{nd}$, $3^{rd}$, $4^{th}$ and $5^{th}$ lumbar intervertebral disc. |
| $II_3$ | Localized between L4-S2 cord segments. Positively correlated with survey radiography | Narrowing of last thoracic, thoracolumbar and $1^{st}$ intervertebral disc space. Vertebral lipping and moderate calcification of the $6^{th}$ lumbar intervertebral disc. |
| $II_4$ | Localized betweenT3-L3 cord segments. Positively correlated with survey radiography | Narrowing of $11^{th}$ and $12^{th}$ thoracic intervertebral disc. Calcification of $6^{th}$ lumbar intervertebral disc. |
| $II_5$ | Localized between L4-S2 cord segments. Could not be correlated with survey radiography | Calcification of $2^{nd}$ lumbar intervertebral disc. |
| $II_6$ | Localized between L4-S2 cord segments. Positively correlated with survey radiography | Calcification and narrowing of $1^{st}$, $2^{nd}$ and $3^{rd}$ lumbar intervertebral disc. On myelography partial obstruction of contrast column at the level of $3^{rd}$ lumbar vertebrae. |
| $II_7$ | Localized between L4-S2 cord segments. Could not be correlated with survey radiography | Calcification of $11^{th}$ and $12^{th}$ thoracic intervertebral disc. |

# *Discussion*

# 5. DISCUSSION

The study was conducted in fourteen clinical cases of paraplegic dogs of different age, sex, breed and body weight. The dogs were randomly divided into two groups of seven animals each, Group I and II. Preliminary clinical, neurological and radiological examinations were conducted in all dogs. The dogs of Group I were subjected to epidural administration of methyl prednisolone acetate initially and oral administration of prednisolone acetate in a tapering dosage for 15 days and that of Group II were subjected to ultrasound massage of the dorsal spinal region in addition to the steroid administration as in Group I.

## 5.1. ANAMNESIS AND SIGNALMENT

All the dogs in both groups were alert but not able to bear weight on hind limbs when presented. Dribbling of urine was reported in 10 cases. All the dogs were regularly vaccinated against rabies. No previous occurrence was reported in any of these cases. Three animals were treated in the nearby veterinary hospital with B- complex vitamins before presented.

### 5.1.1. Breed

Of the fourteen dogs with paraplegia studied, eleven were Dachshund (79 per cent), two were non-descript (14 per cent) and one was Lhasa apso (seven per cent). The incidence was more in Dachshunds in this study and is in agreement with Scott (1997), Smith *et al.* (1997), Fitch *et al.* (2000), Mayhew *et al.* (2004) and Ranganath *et al.* (2006). The authors reported that Dachshund and other members of chondrodystrophoid breeds were more prone to disc herniation. McKee (2000) and Cherrone *et al.* (2004) reported the occurrence of disc disease in non-chondrodystrophoid breeds.

### 5.1.2. Age

The age of the animals ranged from 3 months to seven years and five months with a mean of 4.7 years. The maximum numbers of cases were with in the age group of 4-6 years. This is in accordance with the observations of Scott (1997), Smith *et al.* (1997), Wheeler (1997) George (2002) and Mayhew *et al.* (2004). They reported that the middle aged dogs were most commonly affected with disc disease. According to Smith *et al.* (1997) the average age of dogs affected with Hansen type I disc extrusion was 5 years.

### 5.1.3. Sex

No sex predilection was observed as seven were males and remaining were females. Similar findings were recorded by Wheeler (1997).

### 5.1.4. Body weight

The body weights of the affected dogs studied ranged from 6.5 to 13.5 Kg (mean 11Kg).

### 5.1.5. Etiology

Automobile accident, falls and malicious injury were the reported cause of paraplegia in this study. The cause was unknown for most of the cases. McKee (1990) observed that 9.8 per cent of 51 dogs and cats sustained spinal trauma due to unknown reasons. The author also reported that 58.82 per cent of spinal trauma in dogs and cats occurred due to automobile accidents. Kinzel *et al.* (2005) noted that intervertebral disc disease was the most common cause of neurological trauma in dogs and was characterized by sudden onset of paraplegia.

### 5.1.6. Duration of illness

In the present study dogs were presented one day to two months after the onset of illness.

## 5.2. PHYSIOLOGICAL PARAMETERS

The mean values for respiratory rate, rectal temperature and pulse rate were with in the normal range throughout the study period in both the groups. This indicated that paraplegia or the type of treatment had no effect on physiological parameters. This was in agreement with the findings of George (2002) and Chandy (2006).

## 5.3. CLINICAL SYMPTOMS

All the animals were paraplegic when presented and dribbling of urine was noticed in 10 cases studied. Pain elicited on palpation of the affected part of the spine in all animals. Similar observations were noticed by Wheeler (1997) in animals with thoracolumbar disc disease.

Apparent spinal pain associated with degenerative disc lesions may result from compression or ischemia of the meninges and / or spinal nerve roots. In addition, extruded nuclear material may incite an inflammatory reaction which may cause pain and result in fibrous adhesion between the duramatter and the disc material. Pain could also be produced from stimulation of sensory nerve endings in the annulus fibrosus and dorsal longitudinal ligament (McKee, 2000).

### 5.3.1. Bladder function

Of the fourteen dogs with paraplegia studied in two groups, four dogs ($I_4$,$II_1$.$II_4$,$II_7$ had UMN bladder (28.50 per cent), six dogs ($I_3$,$I_5$,$I_7$,$II_2$,$II_5$,$II_6$) had

LMN bladder (42 per cent) and the remaining ($I_1$,$I_2$,$I_6$,$II_3$) had normal bladder function (28.50 per cent) at the time of presentation.

All the dogs except $I_7$ and $II_6$ returned to normal bladder function at the end of the study period. It was found that improvement in bladder function was seen in 75% of dogs with UMN bladders on second week while only 25% of dogs with LMN bladder showed improvement in bladder function on second week. According to Ruddle *et al.* (2006) UMN signs had a more favorable prognosis compared to LMN signs.

Of the total animals with abnormal bladder function 66.6% of animals in Group I and 40% of dogs in Group II showed improvement in bladder function on second week. This reduced percent in Group II may be due to prolonged duration of illness of the dogs at the time of presentation when compared to Group I, more dogs had LMN bladder and more dogs were graded V based on neurological study on the day of presentation.

## 5.4. NEUROLOGICAL EXAMINATION

The method adopted for neurological evaluation of the dogs as suggested by Lorenz and Kornegay (2004) based on mental status, posture, gait, postural reactions and spinal reflexes was sufficient in assessing the degree of damage to the spinal cord. The evaluation of spinal reflexes was very useful in localization of lesion and assessing the neurological recovery of dogs throughout the study period.

All the dogs in both the groups except $I_7$ were alert on the day of presentation. Dogs in both the groups were non-ambulatory and not able to bear weight on hind limbs. None of the dogs on either group revealed any cranial nerve deficits and had normal postural reactions for both the forelimbs.

Based on the above observations, lesions in brain were ruled out in both the groups and the site of lesion was localized below the level of the foramen magnum. This was in accordance with the method followed by Shell (1996a).

The spinal reflexes for both the forelimbs in all dogs of both the groups were normal and there was full weight bearing as well as normal voluntary activity. This as well as normal wheel barrowing reaction ruled out the presence of lesion up to the T3 spinal cord segments. This was in agreement with the reports of Braund (1995) and Shell (1996a).

Based on the type of pelvic limb reflexes (UMN / LMN), the lesion was localized either between T3 and L3 or L4 and S2 spinal cord segments. The localization of lesion so made was in agreement with the reports of Braund (1995) and Shell (1996a).

Thus in Group I and II lesions were localized between L4 and S2 cord segments in six dogs each and between T3 and L3 cord segments in one dog each.

Absence of proprioceptive positioning reaction was noticed in the hind limbs of all dogs in both groups on the day of presentation. Lorenz and Kornegay (2004) opined that proprioceptive pathways were sensitive to compression, and abnormalities in proprioceptive positioning might occur before motor dysfunction could be detected.

Hyperpathia was noticed on the affected spinal region of all dogs studied. Shell (1996b) stated that hyperpathia was usually associated with disc herniation.

All the dogs except $II_6$ had deep pain sensation throughout the study period by which the prognosis of the condition was assumed favourable for the

treatment. Lanz *et al.* (2000) opined that the prognosis was guarded in case of dogs with no deep pain sensation.

## 5.5. RADIOGRAPHIC EVALUATION

### 5.5.1. Survey radiography

Lateral and / or ventrodorsal radiographs were taken in all dogs in Group I and II. Intervertebral disc space reduction, calcification and protrusion of the disc into the spinal canal, vertebral lipping, vertebral body fractures and luxation were the lesions that could be identified on survey radiographs of the spine of the dogs included in the study. These are in agreement with McKee (2000) and Somerville *et al.* (2001) who observed narrowing of intervertebral disc space and mineralized material in the vertebral canal in the case of intervertebral disc disease.

A positive correlation was obtained in 78.5 per cent of survey radiographic findings with neurological examination. There was 21.42 per cent accuracy for single compressive lesions and 57.14 per cent for multiple lesions. Somerville *et al.* (2001) reported that 53-67 per cent of accuracy for survey radiograph to localize the correct site of disc protrusion.

Out of the three cases where single compressive lesions noticed, two were fracture and luxation and the remaining one was calcification of intervertebral disc. Somerville *et al.* (2001) reported 58 per cent accuracy for single compressive lesions in case of disc disease. The author also opined that the accuracy rates were higher when calcification of the disc was present.

### 5.5.2. Myelography

Myelography was performed in two cases. The method adopted for location of the sites (both cisterna magna and lumbar puncture) were accurate in achieving a successful puncture.

Iohexol at the dose rate of 0.3ml/kg body weight (90mgI/kg) used in the study provided good contrast for demarcation of the spinal cord on myelograms. The drug had been used in similar dose by Wright (1984), Scott (1997), Kumar *et al.* (2004) and Riyaz *et al.* (2006).

Keeping the dogs in an elevated position with the head up as suggested by Lewis (1992) promoted caudal flow of iohexol and prevented its flow into the brain.

The technique was adequate in identifying the sites of compression. No seizures were noticed in both cases studied. Lewis et al. (1992) reported seizure as the main complication associated with Iohexol myelography in dogs.

Narrowing, deviation and upward elevation of the contrast column and partial obstruction to the flow were the myelographic changes that could be identified in this study. Wright (1984) reported these changes as diagnostic signs on myelogram. The narrowing of contrast column indicated partial compression as reported by Kaur and Singh (2004). According to Ranganath *et al.* (2006) narrowing and dorsal deviation of ventral contrast column at the site of protrusion was the most common myelographic finding in intervertebral disc disease.

## 5.6. CORRELATION OF RADIOGRAPHIC PATTERN AND NEUROLOGIC FINDINGS

Lesion was localized between L4 and S2 cord segments in twelve dogs and between T3 and L3 cord segments in two dogs. Out of the fourteen dogs studied, the neurological examination findings were positively correlated with survey radiography in eleven cases. Of the eleven cases, eight were multiple lesions and three were single compressive lesion. The result indicated that the neurological examination followed was very useful for localization of lesion in such cases. The same procedure was followed by Shell (1996a) and Braund (1995) for localizing the lesions.

## 5.7. NEUROLOGICAL GRADING OF PATIENTS

The method of neurological grading used based on motor activity of hind limbs and urinary bladder function was very effective for assessing the neurological status and evaluating the progressive improvement in dogs. Similar methods of grading of patients with spinal cord injury have been used by many authors. (Griffith, 1982; McKee, 2000).

Dog $I_2$, which had luxation of fourth lumbar vertebrae, remained as grade IV till the end of the study period. This indicated that the type of treatment adopted may not be effective for traumatic paraplegia in dogs with instability of the vertebral column.

Seventy five percent of dogs graded IV on the day of presentation returned to Grade I at the end of the study period. Seventy percent of dogs graded V on the day of presentation returned to Grade II and ten per cent returned to grade I at the end of the study period. The dog graded IV showed better neurological improvement when compared to dogs graded as V. This indicated that the evaluation of bladder function was found to be sufficient in

assessing the neurological status of dogs with paraplegia and the finding was in agreement with the observations of Wheeler (1997).

With regards to the neurological grading, severity was more in Group II when compared to Group I. Fifty seven percent of animals in Group I and 85 per cent of dogs in Group II were graded as V on the day of presentation. Even then the outcome of result was similar in both groups studied. Also one dog in Group II ($II_4$), which was graded V at the time of presentation became Grade I at the end of the study period. The result indicated that the therapeutic ultrasound massage has some favourable effect in paraplegic dogs.

## 5.8. HAEMATOLOGICAL PARAMETERS

The mean values of Hb and PCV were with in the normal range in all stages of the two groups. This indicated that either paraplegia or the type of treatment did not produce any significant changes in Hb or PCV. Similar finding were recorded by Griffiths (1982) and Chandy (2006). The authors did not list any possibility of abnormality in the blood picture in fractures and luxation of the vertebral column in dogs. Moore *et al.* (2000) and Sanders *et al.* (2002) reported that the results of a complete blood count were normal in dogs with spinal cord injury of different etiology.

The mean values of TLC were with in the normal range in all the dogs except $I_2$ and $I_6$ on the day of presentation. The dogs $I_2$ and $I_6$ with fracture and luxation showed moderate leukocytosis on the day of presentation. In dog $I_2$, the values continued to increase till the end of the study period. In all other dogs the mean values showed a significant increase on second and lowered towards normal range at the end of the study period.

The mean neutrophil, lymphocyte, monocyte and eosinophil counts were with in the normal physiological range on the day of presentation.

Neutrophilia and lymphopaenia were noticed in both groups on second and fourth week. The mean values returned to normal range at the end of the study period. The monocyte and eosinophil counts were within the normal range throughout the study period.

Shell and Schueler (1996) reported that the complete blood cell count was found to be in the normal range in paraplegic dogs. The presence of stress leukogram during initial stage of the study period may be associated with steroid administration. Similar observations were reported by Calvert and Cornelius (1990a) and Cohn (1997).

## 5.9. SERUM BIOCHEMICAL EVALUATION

The mean values of serum calcium and phosphorous were found to be with in the normal physiological range in all stages in the two groups. This indicated that paraplegia / the modalities of treatment adopted did not interfere with the serum calcium and phosphorous. These findings were in agreement with the observations made by Chandy (2006).

The mean value of serum alkaline phosphatase (ALP) was found to be with in the normal range on the day of presentation in the two groups. There was increase in serum ALP values on the second week in both the groups after which the values lowered at the end of the study period. Ginel *et al.* (2002) observed a significant increase in ALP activity after methyl prednisolone acetate administration for three weeks and returned to normal level by $28^{th}$ day. A non-significant increase in ALP values was noticed by Chandy (2006) in paraplegic dogs after epidural administration of methyl prednisolone acetate.

## 5.10. TREATMENT

The schedule of steroid administration used in this study was found to be effective for the treatment of paraplegia in dogs. The dose rate of methyl prednisolone acetate used in this study was 2mg/kg body weight. The same dosage of drug was used at weekly intervals by Chandy and Vasanth (2000). No complication of steroid administration was noticed during the study period. The steroid was discontinued by reducing the dosage by 50 per cent every five days over a period of 15 days. This may be the reason for the absence of complications and this was in agreement with the findings of Calvert and Cornelius (1990c).

Ranitidine was given throughout the period of steroid administration in this study. Moore and Withrow (1982) reported that intestinal protectants in conjunction with antacids or $H_2$ antagonists might help to reduce the complications of steroid.

The severity of neurological deficit was more in Group II dogs when compared to Group I dogs. Even then the outcome of the results was similar in both groups studied. The result indicated that the therapeutic ultrasound has some favourable effect in paraplegia indogs. According to Olby *et al.* (2005) in patients with acute spinal cord disease and neuromuscular spasm, ultrasound might be applied to the epaxial muscles to help manage pain and muscle spasm. This also improved the blood supply.

The pulsed ultrasound was given on the dorsal aspect and on either side of the vertebral body for ten minutes in this study. The pulsed setting was selected because it minimized the thermal effects created at the vertebral body surface. The shear stress waves created during continuous ultrasound may increase the local heat.

None of the dogs in Group II showed any distress / uneasiness during the ultrasound massage. Bromiley (1991) opined that the distress demonstrated by an animal during ultrasound massage may be the result of pain due to incorrect setting ($W/cm^2$) and / or may be the result of an ability to register a previously unknown sound. The intensity 1.5w/ $cm^2$ used in this study didn't result in any difficulty to the dog.

At the end of the study period, 71.4 per cent of dogs in Group I and 85.7 per cent of dogs in Group II became ambulatory. The stability of vertebral column was not affected in any of the dogs in Group II. In Group I two dogs had vertebral instability due to fracture and luxation of the vertebrae, which may be the reason for the reduced success rate in these dogs.

## 5.11. RECURRENCE

Recurrence of paraplegia was noticed in two dogs $I_1$ and $II_2$. Multiple lesions were observed on survey radiography in these two dogs. Calcification of the fifth and sixth lumbar intervertebral disc was the radiographic finding in dog $I_1$. Varying degrees of calcification was noticed in almost all lumbar intervertebral discs in case of dog $II_2$. This was in agreement with the observations of Scott (1997) and Mayhew *et al.* (2004). According to Mayhew *et al.* (2004) risk of recurrence increased with number of opacified discs in an almost linear manner; each opacified disc increased risk by 1.4 times.

From the study it is found that,

Epidural administration of methyl prednisone acetate followed by oral administration of prednisolone acetate was effective for the treatment of

172845

paraplegia in dogs due to intervertebral disc disease, but was not effective for traumatic paraplegia in dogs with instability of the vertebral column.

The therapeutic ultrasound massage along with steroid therapy enhanced the neurological recovery in paraplegic dogs. .

Since standardized procedures are not available with regards to the intensity and duration of the application of ultrasound massage technique, more studies are required in therapeutic ultrasound massage for the treatment of paraplegia in dogs.

# *Summary*

## 6. SUMMARY

The study was conducted in fourteen paraplegic dogs in two groups (Group I and II) brought to the Surgery unit of Veterinary Hospital Mannuthy and University Veterinary Hospital Kokkalai. The objective of the study was to evaluate the effectiveness of epidural steroid therapy with and without ultrasound massage for the management of paraplegia in dogs.

The Group I dogs were subjected to epidural administration of methyl prednisolone acetate at the rate of 2mg/kg body weight on the day of presentation and repeated after twenty four hours and maintained with oral administration of prednisolone acetate starting with a dose rate of 2mg/kg body weight for five days, tapered to 1mg/kg body weight for next five days, and then 0.5mg/kg body weight for the following five days. In addition to the steroid treatment as in Group I, Group II dogs were subjected to ultrasound massage of the affected dorsal spinal region for five days continuously and later at weekly intervals till improvement noticed or for a maximum of six weeks.

All the dogs were administered supportive medicines like neurotropic drug (methyl cobalamine) and antibiotics (amoxicillin-cloxacillin) during the period of observation. Ranitidine was given orally during the period of steroid administration.

The incidence was highest in Dachshunds (79 per cent), followed by non-descript (14 per cent) and Lhasa apso (seven per cent). The age of the animals ranged from three months to seven years and five months. Of the fourteen dogs studied seven were males and the remaining were females. The etiology was unknown for most of the cases studied (nine cases). Automobile accident, fall from height and malicious injury were the other causes.

Total leukocyte count and differential leukocyte count indicated stress leukogram during the initial stage of the study period. Alkaline phosphatase values

showed an increase on second week and lowered to normal at the end of the study period.

Detailed neurological examination revealed localized lesion between L4 and S2 cord segments in twelve dogs and between T3 and L3 in the remaining two dogs.

The urinary bladder function was assessed through abdominal palpation of the bladder. Cystoplegic dogs were graded as having lower motor neuron (LMN) bladder or upper motor neuron (UMN) bladder based on whether urine was freely relieved or not when the bladder was pressed through the abdominal wall. Of the fourteen cases of paraplegia studied in two groups, four dogs ($I_4$,$II_1$.$II_4$,$II_7$) had UMN bladder (28.50 per cent), six dogs ($I_3$,$I_5$,$I_7$,$II_2$,$II_5$,$II_6$) had LMN bladder (42 per cent) and the remaining (28.50 per cent) had normal bladder function at the time of presentation. All the dogs except $I_7$ &$II_6$, which died on second day, returned to normal bladder function at the end of the study period.

Survey radiograph was taken and the findings were correlated with the neurological examination findings. Intervertebral disc space reduction, calcification and protrusion of the disc into the spinal canal, vertebral lipping, vertebral body fractures and luxation were the lesions that could be identified on survey radiographs. A positive correlation was obtained in 78.5 per cent of survey radiographic findings with neurological examination.Narrowing, deviation and upward elevation of the contrast column and partial obstruction to the flow were the myelographic changes that could be identified in two dogs studied.

Based on motor activity of pelvic limbs and urinary bladder function the dogs were graded from Grade I (spinal hyperesthesia only) to Grade VI (paraplegia, urinary incontinence and absence of deep pain sensation) during the study period. Three dogs of Group I and five dogs of Group II were graded as V and the rest of the dogs were graded as IV on the day of presentation. Seventy five percent of the patients graded IV returned to Grade I at the end of the study period. Seventy

percent of the dogs graded V returned to Grade II and ten per cent returned to Grade I at the end of the study period.

At the end of the study period, 71.4 per cent of dogs in Group I and 85.7 per cent of dogs in Group II became ambulatory. The stability of vertebral column was not affected in any of the dogs in Group II. In Group I two dogs had vertebral instability due to fracture and luxation of the vertebrae, which may be the reason for the reduced success rate in these animals.

From the study the following conclusions were drawn,

The incidence was more in Dachshunds. No sex predilection was observed. The maximum number of dogs were within the age range of four to six years. The etiology was unknown for most of the cases studied.

Physiological parameters were not affected by paraplegia or the type of treatment adopted.

Hb, PCV, Serum calcium and phosphorous values were within the normal physiological range in both groups during the study period. Total leukocyte count and differential leukocyte count indicated stress leukogram during the initial stage of the study period. Alkaline phosphatase values showed an increase on second week and lowered to normal at the end of the study period.

Evaluation of bladder function was very useful for assessing the neurological status of the dogs.

The neurological examination was effective for the localization of lesion and assessing the neurological recovery of dogs throughout the study period.

Absence of deep pain sensation indicated poor prognosis for recovery

The method of neurological grading used based on motor activity of hind limbs and urinary bladder function was very useful for assessing the neurological status and evaluating the progressive improvement in animals

A positive correlation was obtained in 78.5% of survey radiographic findings with neurological examination. There was 21.42% accuracy for single compressive lesions and 57.14% for multiple lesions.

The myelography using Iohexol at the dose rate of 0.3ml/kg body weight (90 mg I/kg) body weight was adequate in identifying the site of compression.

Epidural administration of methyl prednisone acetate followed by oral administration of prednisolone acetate was effective for the treatment of paraplegia in dogs due to intervertebral disc disease, but was not effective for traumatic paraplegia in dogs with instability of the vertebral column.

The therapeutic ultrasound massage along with steroid therapy enhanced the neurological recovery in paraplegic dogs.

No standardized procedure available with regards to the intensity and duration of the technique. However more studies are required in usage of therapeutic ultrasound massage to arrive at a conclusion.

# *References*

## REFERENCES


Barthez, P.Y., Morgan, J.P.and Lipsitz, D. 1994. Discography and Epidurography for evaluation of the lumbosacral junction in dogs with cauda equina syndrome. *Vet.Radiol. Uitrasound.* 35:152-157

Benjamin, M. M. 2005. *Outline of Veterinary Clinical Pathology*. Third edition. Kalyani Publishers, New Delhi, 351

Bergman, R., Lanz, O. and Shell, L. 2000. Acute spinal cord trauma: mechanisms and clinical syndromes. *Vet.Med.* 95:844-849

Bos, A.S., Brisson, B. A., Holmberg, D.L. and Nykamp, S.G. 2007. Use of the ventrodorsal myelographic view to predict lateralization of extruded disc material in small-breed dogs with thoracolumbar intervertebral disc extrusion. *J. Am. Vet. Med. Assoc.* 230:1860-1865

Braund, K.G. 1995. Using neurologic syndrome to localize lesions in the spinal cord.*Vet.Med.* 24: 157-160

Bray, J.P. and Burbidge, H.M. 1998. The Canine Intervertebral Disc Part one: Structure and Function. *J.Am Anim Hosp Assoc.* 34:55-63

Bromiley, M.W. 1991. *Physiotherapy in Veterinary Practice*. Blackwell scientific publications, London, 135p.

Calvert, C. A. and Cornelius, L.M. 1990a. Corticosteroid hormones: Endogenous regulation and the effects of exogenous administration. *Vet. Med.* 85:810-823

Calvert, C. A. and Cornelius, L.M. 1990b. The most common indications for using corticosteroid hormones in veterinary practice. *Vet. Med.* 85:826-845

Calvert, C. A. and Cornelius, L.M. 1990c. Avoiding the undesirable effects of glucocorticoid hormone therapy. *Vet. Med.* 85:846-856

Calvert, C. A. and Cornelius, L.M. 1990d. The pharmacodynamic differences among glucocorticoid preparations. *Vet. Med.* 85:860-865

Campoy, L. 2004. Epidural and spinal anaesthesia in the dog. *In pract.*15: 262-266

Carstens, A. and Kitshoff, A.M. 2007. Imaging Diagnosis-Postmyelographic Epidural leakage due to increased meningeal permeability. 48: 554-556

Chandy, G. 2006. Studies on spinal fixation techniques with or without decompression for traumatic posterior paralysis in dogs. PhD thesis. Karnataka Veterinary, Animal and Fisheries Sciences University, Bidar, 238p.

Chandy, G. and Vasanth, M.S. 2006. Efficacy of Spinal Fixation versus Medical Management in Dogs with Traumatic Posterior Paralysis. National symposium on "Surgical Techniques for the Benefit of Livestock Farmers and Companion Animals", 2nd- 4th November, Bidar, Indian Society for Veterinary Surgery, Izatnagar. *Abstract*: 44

Chandy, G. and Vasanth, M.S. 2006. Epidural use of methyl prednisolone acetate in paraplegic dogs: a report of five cases. *Indian J. Vet.Surg.* 27:122-124

Cherrone, K.L., Dewey, C.W., Coates, J.R.and Bergman, R.L. 2004. A Retrospective Comparison of Cervical intervertebral Disc Disease in Non-Chondrodystrophic Large Dogs Versus Small Dogs. *J.Am Anim Hosp Assoc.* 40:316-320

Clark, B. and Mclaughlin, R.M. 2001. Physical Rehabilitation in Small-Animal Orthopaedic Patients. *Vet Med.* 32:234-246

Cohn, L.A. 1997. Glucocorticoids as immunosuppressive agents. *Seminars in Veterinary Medicine and Surgery (small animal).* 12:150-155

Cornelius, L.M. and Calvert, C.A. 1991. Four case studies in the use and misuse of corticosteroid therapy. *Vet.Med.* 25: 1186-1198

Costa, K.C.D., Parent, J., Dobson, H., Holmberg, D. and Parlow, G. 2006. Comparison of magnetic resonance imaging and myelography in 18 Doberman pinscher dogs with cervical spondylomyelopathy. *Vet.Radiol. Uitrasound.* 47: 523-531

Coughlan, A.R. 1993. Secondary injury mechanism in acute spinal cord trauma. *J.Small Anim. Pract.* 34:117-122

Cranger, C. S., Kemppainen, R.J., Brewer, W.G. and Angarano, D. W. 1994. Adrenocorticotropic hormone and cortisol concentrations after corticotropin- releasing hormone stimulation testing in cats administered methyl prednisolone. *Am. J. Vet. Res.* 55: 704-709

De Lahunta, A. 1997. *Veterinary Neuroanatomy and Clinical Neurology.* W.B.Saunders Company, Philadelphia; 439p.

Devecioglu, Y., Guzel, O., Aktas, M., Duzgun, O. and Akbar, M. 2006. Comparison of Iohexol and Iopamidol in cysternal myelography in dogs. *Indian Vet. J.* 83: 398-401

Dhaliwal, T.P.S. and Mirakhur, K.K. 2001. Clinical management of neurological disorders in dogs- A review of 26 cases. *Indian J. Vet.Surg.*22:34-37

Dhupa, S., Glickman, N.W. and Waters, D.J. 1999. Functional outcome in dogs after surgical treatment of caudal lumbar intervertebral disc herniation. *J.Am Anim Hosp Assoc.* 35: 323-331

Dickinson, P. n .d. 2005. *Neuroimaging.* (On line). International Veterinary Information Service, Available: http//www.IVIS.org (25 June 2008)

Fatone, G., Lamagna, F., Potena, A. and Brunetti, A. 1997. Myelography in the dog with non-ionic contrast media at different iodine concentrations. *J.Small Anim. Pract.* 38: 292-294

Fitch, R. B., Kerwin, S. C. and Hosgood, G. 2000. Caudal cervical intervertebral disc disease in the small dog: Role of distraction and stabilization in ventral slot decompression. *J.Small Anim. Pract.* 36:68-74

Fossum, T.W. 2002. *Small Animal Surgery.* Third edition.Mosby, Inc., Missouri, 1400 p.

Gage, E.D. and Horlein, B.F. 1974. Intervertebral disc. *Canine Surgery.* (ed. Archibald, J.). Second edition. American Vety Pub, California, pp.862-898

George, J. 2002. Ascending Coccygeal Venography in Evaluation of Paraplegia in Dogs. MVSc thesis. Kerala Agricultural University, Trichur, 118p.

Ginel, P.J., Lucena, R. and Ferandez, M. 2002. Duration of increased serum alkaline phosphatase activity in dogs receiving different glucocorticoid doses. *Res. Vet. Sc.* 72:201-204

Griffith, I. 1982. Spinal disease in the dog. *In Pract.* 4:44-52

Hall, L.W. 1971. *Wrights Veterinary Anaesthesia and Analgesia.*Seventh edition, Bailliare Tindall, London, 499p.

Hodges, C.C. and Palmar, R.H. 1993. Post operative physical therapy. *Surgical complications and wound healing in the small animal practice.* (ed.Harari, J.).Second edition.W.B.Saunders company, Philadelphia, pp.389-405

Huang, H., Yang, H., Liang, S., Lien, Y.and Chen, K. 1999. Iatrogenic hyperadrenocortisism in 28 dogs. . *J.Am Anim Hosp Assoc.* 35:200-207

Janssens, L.A.A. 1991. Mechanical and pathophysiological aspects of acute spinal cord trauma. *J.Small Anim. Pract.* 32:572-578

Jeffery, N. 2001. Neurological examination of dogs 1. Techniques. *In Pract.* 210: 118-130

Jones, R.S. 2001. Epidural analgesia in the dog and cat. *The Vet. J.* 161:123-131

Kaur, A. and Singh, S.S. 2004. Myelography for spinal affections with iohexol in dogs. *Indian J.Vet.Surg.* 25:69-71

Kent, M. 2004. Therapeutic options for neuromuscular diseases. *Vet Clin Small Anim.* 34:1525-1551

Kinzel, S., Wolff, M., Buecker, A., Krombach, G.A., Stopinski, T., Afify, M., Weiss, C.and Kupper, W. 2005. Partial percutaneous discectomy for the treatment of thoracolumbar disc protrusion: retrospective study of 331 dogs. *J.Small Anim. Pract.* 46:479-484

Krishnamurthy, D. and Singh, A.P. 1994. Contrast Techniques. *Veterinary Radiology* (eds. Singh, A.P. and Singh, J.). CBS Publishers and Distributors, Delhi, pp.184-215

Kumar, P., Ranganate, L., Ranganath, B.N., Vasanth, M.S. and Jayadevappa, S.M. 2004. Myelographic studies in dogs using iohexol and iopamidol. *Indian J.Vet.Surg.* 25:72-74

Lanz, O., Bergman, R. and Shell, L. 2000. Initial assessment of patients with spinal cord trauma. *Vet.Med.* 23: 851-853

Levine, J. M., Levine, G. J., Boozer, L., Schatzberg, S.J., Platt, S.R., Kent, M., Kerwin, S.C. and Fosgate, G.T. 2008. Adverse effects and outcome associated with dexamethasone administraton in dogs with acute thoracolumbar disk herniation: 161 cases (2000-2006). *J. Am. Vet. Med. Assoc.* 232: 411-417

Lewis, D.D. and Hosgood, G. 1992. Complications associated with the use of iohexol for myelography of the cervical vertebral column in dogs: 66 cases (1988-1990). *J. Am. Vet. Med. Assoc.* 200: 1381-1384

Lobetti, R.G. 1994. Subarachnoid abscess as a complication of discospondylitis in a dog. *J.Small Anim. Pract.* 35:480-483

Lorenz, M. D. and Kornegay, J.N. 2004. *Handbook of Veterinary Neurology.* Fourth edition. Saunders, USA, 468 p.

Mayhew, P.D., Kapatkin, A.S., Wortman, J.A.and Vite, C.H. 2002. Association of cauda equina compression on magnetic resonance images and clinical signs in dogs with degenerative lumbosacral stenosis. *J.Am Anim Hosp Assoc.* 38:555-562

Mayhew, P.D., Mclear, R.C., Ziemer, L.S., Culp, W.T.N., Russel, K.N., Shefer, F.S., Kapatkin, A.S. and Smith, G.K. 2004. Risk factors for recurrence of clinical signs associated with thoracolumbar intervertebral disc herniation in dos: 229 cases (1994-2000). *J. Am. Vet. Med. Assoc.* 225: 1231-1236

Meinkoth, J. H. and Clinkenbeard, K. D. 2000. Normal Haematology of the Dog. *Schalm's Veterinary Haematology.* (ed. Feldman,B. F., Zinkel,J. G. and Jain, N. C.). Lippincott Williams and Wilkins, A Wolters Klumen Company, Philadelphia, 1344p.

McClure, R.C. 1964. The spinal cord and meninges. *Anatomy of the Dog.* (ed.Miller, M.E.). W.B.Saunders Company, Philadelphia, pp.533-543

McKee, M. 2000. Intervertebral disc disease in the dog 1. Pathophysiology. *In Pract.* 22: 35-369

Mckee, W.M. 1990. Spinal trauma in dogs and cats: a review of 51 cases. *Vet Rec.* 126:285-289

Moore, G.E., Mathey, W.S., Eggers, J.S. and Estep, J.S. 2000. Osteosarcama in the adjacent lumbar vertebrae in a dog. *J. Am. Vet. Med. Assoc.* 217: 1038-1040

Moore, R. W. and Withrow, S. J. 1982. Gastrointestinal haemorrhage and pancreatitis associated with intervertebral disk disease in the dog. *J. Am. Vet. Med. Assoc.* 15: 1443-1447

Olby, N. Halling, K. B. and Glick, T. R. 2005. Rehabilitation for the neurologic patient. *Vet Clin Small Anim.* 35: 1389-1409

Penderis, J., Sullivan, M., Schwarz, T. and Griffith, I.R. 1999. Subdural injection of contrast medium as a complication of myelography. *J.Small Anim. Pract.* 40: 173-176

Puglisi,T.A., Green,R.W., Hall,C.L., Read,W.K., Green,R.A., Tangner,C.H., Mann,F.A.and Phil Hebson, H. 1986. Comparison of metrizamide and iohexol for cisternal myelographic examination of dogs. *Am. J. Vet. Res.* 47:1863-1869

Ranganath, L., Ranganath, B.N. and Kumar, P. 2006. Myelographic evaluation of intervertebral disk disease in dogs. *Indian J. Vet.Surg.* 27:125-126

Riyaz, D.B., Vasanth, M.S., Ranganath, L. and Ranganath, B.N. 2006. Myelography for evaluation of spinal lesions in dogs. *Indian J. Vet.Surg.* 27:127

Ruddle,T.L., Allen,D.A., Schertel,E.R., Barnhart,M.D., Wilson,E.R., Lineberge,J.A.,Klocke,N.W. and Lchenbauer,T.W. 2006. Outcome and prognostic factors in non-ambulatory Hansen type I intervertebral disc extrusions: 308 cases. *Vet Comp Orthop Traumatol.* 19:29-34

Sanders, S.G., Bagley, R.S. and Gavin, P.R. 2002. Intramedullary spinal cord damage associated with intervertebral disc material in a dog. *J. Am. Vet. Med. Assoc.* 221:1594-1596

Scott, H.W. 1997. Hemilaminectomy for the treatment of thoracolumbar disc disease in the dog: a follow-up study of 40 cases. *J.Small Anim. Pract.* 38:488-494

Shell, L. G. 1996a. The basics of pelvic limb weakness. *Vet. Med.* 91:220-228

Shell, L. G. 1996b. Differential diagnosis of acute-onset paraparesis. *Vet. Med.* 91:230-239

Shell, L. G. 1996c. Differential diagnosis of progressive-onset paraparesis. *Vet. Med.* 91:240-253

Shell, L.G. and Schueler. 1996. Six case studies of paraparesis. *Vet. Med.* 91: 254-262

Smith, J.D., Newell, S.M., Budsberg, S.C. and Bennet, R.A. 1997. Incidence of contra lateral versus ipsilateral neurological signs associated with lateralized Hansen type I disc extrusion. *J.Small Anim. Pract.* 38: 495-497

Snedecor, G.W .and Cochran, W.G. 1994. *Statistical Methods*. Eight edition. Iowa State University Press, Ames, Iowa, 564 p.

Somerville, M. E., Anderson, S.M., Gill, P.J., Kantrowitz, B.J.and Stowater, J.L. 2001. Accuracy of Localization of Cervical Intervertebral Disk Extrusion or Protrusion Using Survey Radiography in Dogs.*J.Am Anim Hosp Assoc.* 37:563-572

Tartarelli, C.L., Baroni, M.and Borghi, M., 2005. Thoracolumbar Disc Extrusion Associated With Extensive Epidural Haemorrhage: a retrospective study of 23 cases. *J.Small Anim. Pract.* 46:485-490

Taylor, R. n. d. 2001. *Physical therapy in Veterinary Medicine.* (Online). International Veterinary Information Service, Available:http//www. IVIS.org (25 June 2008)

Thomson, C.E., Kornegay, J.N., and Stevens, J.B. 1989. Canine intervertebral disc disease: changes in the cerebrospinal fluid. *J.Small Anim. Pract.*30:685-688

Toombs, J.P.and Waters, D.J. 2003. Intervertebral disc disease. *Textbook of Small Animal Surgery.* (ed.Slatter, D.). Fourth edition.W.B.Saunders Company, Philadelphia, pp.1193-1208

Wheeler, S. 1997. Decision making in the management of canine intervertebral disk disease. *In Pract.* 22: 72-81

Widmer, W.R., Denicola, D.B., Blevins, W.E., Cook, J.R., Cantwell, H.D. and Teclaw, R.F. 1992. Cerebrospina fluid changes after iopamidol and metrizamide myelography in clinically normal dogs. *Am.J.Vet.Res.* 53:396-401

Wright, J.A. 1984. Myelography in the dog. *In Pract.* 17: 25-27

# EPIDURAL STEROID THERAPY AND ULTRASOUND MASSAGE FOR THE MANAGEMENT OF PARAPLEGIA IN DOGS

K. M. SEENA

**Abstract of the thesis submitted in partial fulfilment of the requirement for the degree of**

## Master of Veterinary Science

**Faculty of Veterinary and Animal Sciences**
**Kerala Agricultural University, Thrissur**

**2008**

**Department of Veterinary Surgery and Radiology**
**COLLEGE OF VETERINARY AND ANIMAL SCIENCES**
MANNUTHY, THRISSUR-680651
KERALA, INDIA

## ABSTRACT

The study was conducted in fourteen clinical cases of paraplegic dogs with an objective to evaluate the effectiveness of epidural steroid therapy with and without ultrasound massage for the management of paraplegia in dogs.

The dogs were randomly divided into two groups of seven animals each, Group I and II. Preliminary clinical, neurological and radiological examinations were conducted in all dogs. The dogs of Group I were subjected to epidural administration of methyl prednisolone acetate (2mg/kg body weight) initially and oral administration of prednisolone acetate in a tapering dosage for 15 days and that of Group II were subjected to ultrasound massage of the dorsal spinal region in addition to the steroid administration as in Group I.

The incidence was more in Dachshunds. No sex predilection was observed. The maximum number of dogs was within the age range of four to six years. The etiology was unknown for most of the cases studied.

The physiological parameters were within the normal range. Total leukocyte count and differential leukocyte count indicated stress leukogram during the initial stage of the study period. Hb, PCV, serum calcium and phosphorous values were within the normal range. Alkaline phosphatase values showed an increase on second week and lowered to normal at the end of the study period.

The neurological examination was effective for the localization of lesion and assessing the neurological recovery of dogs. Absence of deep pain sensation indicated poor prognosis for recovery. Evaluation of bladder function was very useful for assessing the neurological status of the dogs.

A positive correlation was obtained in 78.5 per cent of survey radiographic findings with neurological examination. The survey radiography had 57.14 per cent accuracy for identifying multiple lesions and 21.42 per cent accuracy for single compressive lesions. The myelography was sufficient for identifying the site of compression and iohexol at the dose rate of 0.3ml/kg body weight (90mgI / kg body weight) provided good contrast for demarcation of the spinal cord.

Neurological grading system based on the motor activity of the hind limbs and urinary bladder function was useful for evaluating the progressive neurological improvement in dogs during the course of study.

The outcome of treatment was good in both groups studied. Epidural administration of methyl prednisone acetate followed by oral administration of prednisolone acetate was effective for the treatment of paraplegia in dogs due to intervertebral disc disease. But it was not effective for traumatic paraplegia in dogs with instability of the vertebral column. The therapeutic ultrasound massage enhanced the neurological recovery in paraplegic dogs.