SERIAL RADIOGRAPHIC AND HISTOLOGICAL CHANGES AS A RESULT OF A DISC CURETTAGE IN CHONDRODYSTROPHIC CANINES

by

Stanley D. Wagner, DVM

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APPROVED BY:

Hugh C. Butler, DVM, MS
Major Professor

H. Rodney Ferguson, DVM, PhD
Major Professor

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INTRODUCTION

Thoracolumbar pain has affected man and the canine for years. Once it was established that the intervertebral disc has a causal relationship to pain, the disc itself became the object of investigative attention.[43,44,50-52,70,78] Pain can be of two types; discogenic which is associated with degeneration of the disc material without extrusion of the disc material into the spinal canal or radicular which is due to compression of a nerve root.[76] The literature has concentrated on the clinical signs, neuroradiography,[2,4,19,33,64,74] pathophysiology,[20-23,26,41,43,44,50,51,52,58,73,76,78] medical and surgical management of thoracolumbar disc disease. Recently a better understanding of the mechanical function and biochemical composition of the anulus fibrosus and nucleus pulposus has been investigated.[1,3,14,28,29,35,36,45,46,47,54,66-69,82,83,85]

Several medical and surgical modalities have been advocated for treatment of thoracolumbar disease in the canine.[50] The use of disc curettage for discogenic pain is controversial. The surgical results have been heralded[5,8,24,33,62,73,79,88] and derided[13,76] in the literature. Proponents cite the surgery for its prophylactic and therapeutic benefits. Technically the proponents feel that it is a safer procedure than other intraspinal operations. In addition since more than one thoracolumbar disc space is curetted the causative disc space need not be identified.[8,24,33,62,73] Opponents feel that painful animals can be successfully managed medically because curettage will not remove disc material that may be compressing a spinal nerve root. Also multiple disc fenestrations are unnecessary because multiple disc herniations are uncommon.[13,76] Recurrence of neurological deficits has been reported at 14.8 per cent following thoracolumbar disc fenestration.[63]

The purpose of this study was to evaluate disc space curettage for treatment of discogenic pain. The study attempted to answer several questions:

1. does removal of the nucleus pulposus cause a collapse of the operated disc space when compared to an untreated disc space?

2. how complete is disc material evacuated by curettage?

3. what histologic tissue if any can be seen under light microscopy in the reparative process following curettage?

4. does removal of the nucleus pulposus place additional mechanical stress on adjacent disc spaces?

LITERATURE REVIEW

Incidence of Disease

Moat studies report that degenerative disc disease is highest in the Daschund breed.[13,43,44] The animala tend to be middle age with an age range of 3-6 years.[13,33,44,50] Some studies report a higher incidence in males than females.[50] Pain and rear leg paresis are the most common presenting signs,[13,50] signs vary with the amount of spinal cord compression. Disc space T12-13[13,50] is the most commonly involved followed by T13-L1.[13,50]

An anulus fibrosus that is partially ruptured while its outer layer is stretched but still contains the nucleus is called a disc herniation.[50] Ruptured, extruded or prolapsed disc all refers to a complete rupture of the anulus with loss of the nucleur material.[50]

Varying degrees of an ischemic cord myelopathy due to interference of blood flow to the parenchyma result from disc protrusion. Cord ischemia leads to demyelination, axonal degeneration, focal hemorrhage and myelomalacia in the grey and white matter.[23] A release of vasoactive substances such as norepinephrine, serotonin and dopamine may occur with disc protrusion to account for the autodestruction of the spinal cord which is observed.[76]

Prognosis with degenerative disc disease is based on the degree of paresis, spinal reflex function and cerebral response to pain.[23]

Anatomy and Function

The canine possesses 27 vertebrae between the skull and the sacrum. With the exception of the first and second cervical vertebrae, all the others have a hydroelastic cushion interposed between them.[81] The disc and its adjacent vertebrae form an amphiarthrosis or fibrocartilagenous joint. The limited flexibility of this joint enables the spine to bend and twist under muscular control.[28] A diarthrodial joint also is present between cranial and caudal articular facets of the adjacent vertebrae. The discs in the thoracic region are oval shaped while the lumbar discs are been shaped.[60] Grossly the disc can be divided into two distinct parts, an outer fibrous ring the anulus fibrosus, and an eccentrically located gelatinous inner part, the nucleus pulposus.[27] On cut surface the ventral portion of the disc is thicker than the dorsal portion. The dorsal to ventral thickness has been estimated by Hansen to be 1:3,[43,44] while Miller lists 1:2.[27] Smith and King record a 1:1.1 - 3.0[81] ratio in various breeds. Sagittal sections of the disc demonstrate a very thin layer of hyaline cartilage separating the disc from the endplate of cancellous vertebral bone.[43] The epiphyseal surface of the adjoining vertebral bodies have a depression which conforms to the nucleus pulposus.[60] The intervertebral disc is a highly specialized composite of several connective tissues.[83] The anulus consists of coarse collagen fibers which form circular sheets or lamellae that interconnect to the adjacent vertebral bodies which stain red with eosin.[28,48] There are fewer lamellae dorsally than ventrally, the exact number of lamellae has been reported to vary from 10-30[43,44] to 25-40.[60] At the periphery of the disc in man and dog the coarse fibers of the outer lamellae are anchored directly to the vertebral bone by Sharpey's fibers.[44] Deeper, these fibers merge with the hyaline cartilage of the vertebral endplate. Fibroblasts with elongated compressed nuclei are present between collagen fibers, in degenerated disc cartilage cells may be present.[26] In addition the outer lamellae are fused to the wide ventral longitudional ligament which courses from the axis to the sacrum.[27,59] The outer lamellae are fused to the dorsal longitudional ligament along its wide midline but laterally this ligament thins.[59] Two other ligaments, the ligament of the head (radial) and the intercapital, are associated with the lamellae of the anulus in the thoracic region. The former passes from the ventral edge of the articular head of each rib to attach laterally to the anulus and each adjacent vertebral.[27,60] The intercapital ligament, courses dorsally over the anulus but under the dorsal longitudional ligament, is absent from the first, twelfth and thirteenth rib pairs.[27] It is reported that the intercapital ligament is absent or small at the eleventh pair of ribs.[27,60] The intercapital ligament is thickest and widest at the fifth pair of ribs while thinning and narrowing as it diverges from this point.[59] The T11-L3 region is subjected to the greatest mechanical load and this area clinically has the largest number of prolapses.[4,13,50] Spinal movement however is greatest at the L7-S1 articulation.[10]

The various canine breeds have been classified into two categories, chondrodystrophoid and nonchondrodystrophoid, based on the altered histologic morphology of endochondral ossification of their epiphyseal growth plates and intervertebral disc.[9,43,44] The histology of the newborn canine nucleus is a loose aggregation of low differentiated cells scattered throughout a homogenous intercellular substance. The border between the anulus and nucleus is indistinct and blends to form a fibrocartilaginous layer or transitional layer.[34] The collagenous lamellae become more mature toward the periphery. In the nucleus pulposus of the chondrodystrophoid breeds there is an early transformation at two to four months of age of the matrix cells into calcified fibrocartilaginous tissue (chondroid) with few notochordal cells present.[9,43] In the nonchrondrodystrophoid breeds the nuclear cells are gradually transformed with increasing age into fibrocytes (fibroid).[9,37,43] Presence of notochordal cells vary between species. These cells are present in man until about 10 years of age, are seen in aged cats but decrease throughout the life span of the dog.[15,16] In man disc degeneration is characterized by progressive fibrous change in the nucleus, loss of nuclear distinction from the anulus and loss of anular organization.[20-22] In addition there is a loss of ground substance, the unmasking of collagen fibrils and the gathering together of fibrils into broader fiber bundles.[26,28,47]

Hansen described the histology of the disc by layers that include the nucleus pulposus, perinuclear layer, inner layer and outer layer of anulua fibrosus.[43,44] Braund used a similar claasification for the histology of the canine disc but used the term transitional zone as synonymous for Hansen's perinuclear layer.[9] Both authors described this zone as noticeablely wider in the chondrodystrophoid breed than in the nonchondrodystrophoid animal.

Scanning electron microscopy of adult dogs, rats and human show that the nuclear collagen fibrils are oriented in a loose irregular pattern that develop into the regular mature collagen lamellae of the anulus fibrosus.[54] Freeze fracture studies of these anular lamellae show the individual fibers run parallel to one another or in a helical arrangement. These fibers are at an angle of about 60° to the spinal axis and 120° to the fibers of adjacent lamellae.[28] During flexion there is a ventral narrowing and a dorsal widening of the disc space. Dorsal anular fibers fail when extended beyond 4 per cent of their original length leading to concentric rupture.[45,46] Radiating fissures occur in the anulus fibrosus in the most centrally situated lamellae and extend outward to the periphery.[48,64] These radiating fissures progressively weaken the anulus resistance to nuclear herniation.

About half the dry weight of the disc is collagen, the collagen types in the porcine and human disc are type I in the well defined lamellae of the outer anulus, type II in both the transitional zone and healthy nucleus.[29,68] Information about the canine is not reported. The strength of the disc depends on the concentration and stability of the covalent bonds between the collagen fibrils.[28] The outer anulus is the most collagenous part of the disc and collagen falls as a percentage of dry weight from the anulus to its lowest level in the nucleus.[28] Studies on whale nucleus pulposus show the very center of their nucleus contains a constant amount of collagen throughout life. The transitional zone contains several times more collagen which increases in amount relative to the nucleus during aging.[28]

Proteoglycans are the second most abundant component of the intervertebral disc in the young after collagen nad is theorized to play a role in both disc degeneration and repair.[14,28,35,36,37,66,67] Proteoglycans account for at least half the dry weight of the nucleus and diminish toward the outer edge of the anulus to roughly 10 per cent of its dry weight.[28] Proteoglycans have been well studied in hyaline cartilage.[28] In cartilage the function of proteoglycans is to retain water and therefore resist compressive forces, a similar function occurs in the disc. Proteoglycans consist of a protein core with linkage regions for the sulfated glycosaminoglycans. Proteoglycans of intervertebral disc are similar to cartilage but are small because of a shorter protein core[82] but contain more protein than cartilage proteoglycans.[28] Chondroitin sulfate and keratan sulfate compose the glycoaaminoglycans in disc from both chondrodystrophoid beagles and nonchondrodystrophoid greyhounds.[9,14,35,36] These two sulfates are composed of repeating disaccharide units, chondroitin sulfate has two negative charges and keratan sulfate has one negative charge.[85] It is the fixed array of negatively charged groups that determines equilibrium in the tissues and therefore the capacity of the nucleus to bind water.[28,69,85] The collagen of the disc is either embedded in a proteoglycans gel or is covered in some way by the material.[85] Interactions between collagen fibrils and their surrounding proteoglycans is poorly understood. The fiber morphology of connective tissue does appear to be related to the type of proteoglycan present.[68,85]

Proteoglycans can exist in the aggregated form which is the association of many glycosaminoglycans subunits with a hyaluronic acid molecule, or in the unaggregated form. In the human nucleus the glycosaminoglycans exist mainly in the unaggregated form while the anulus has 60-65 per cent in the aggregated form.[66,67,75,85] Aggregation function is unknown but may serve to immobilize the proteoglycana in the collagen network and offer partial protection against proteolytic degradation.[66] Aggregated proteoglycans appear to be a sign of healthy cartilage, a reduction is seen in osteoarthritis and degenerative disc disease.[66] The nucleus of the nonchondrodystrophoid breed contains larger amount of glycosaminoglycans than the nuclear region of the chondrodystrophoid breed.[9,14,35,36,37] This may be genetically controlled in some breeds.[38]

With aging the composition of the nucleus approaches that of the inner anulus. Keratan sulfate is initially not in the nucleus or transitional zone but with age it increases while chondroitin sulfate decreases.[35,36,37] With degenerative disc disease there is a loss of water content, the ratio of chondroitin sulfate changes and an increase in the collagen levels.[35,36,37] In prolapsed disc material glycosaminoglycans content has been found to be low and the collagen content higher than normal. It is unknown if a defect in proteoglycans initiates disc degeneration. Lipson has shown that proteoglycans respond in experimental disc herniation by an attempt to repair the damaged disc.[66]

The pressure developed in the nucleus under a compressive force, both in vivo and in vitro, was measured to be 1.5 times higher than the pressure initially applied.[45] The pressure produced within the nucleus allows the disc to maintain height under axial compression.[35,36,45] When the disc is loaded the nucleus transfers the compressive forces in all directions away from the nuclear center. The anulus experiences two internal stressed, one in the radial direction, the other in the tangential direction.[1] The tangential stress was estimated to be 3.5 times the compressive stress.[71] The nucleus is kept under constant compressive pressure by the tension applied by the tension applied by the anular fibers and the intervertebral ligaments.[69]

Degenerative discs have a higher rate of deformation than normal discs.[56]

The intervertebral disc is the largest avascular structure in the body. The endplates are partly permeable to solutes due to contact between the vertebral body marrow cavity and the hyaline cartilage.[53] The central portion of the cartilage endplate is more permeable than the periphery. The anulus is always permeable.[85] Continuous daily exercise for at least two hours daily was found to increase aerobic metabolism in the outer part of the anulus and in the central part of the nucleus pulposus, resulting in a reduction of lactate.[53]

Nerves are present in the connective tissue and fat on the dorsal surface of each disc and superficial layers of the anulus fibrosus. Nerves have not been detected in the deep layers of the anulus or within the nucleus. The dorsal longitudional ligament is well innervated.[31] Dogs exhibit pain because the degenerated disc puts pressure on the dorsal longitudional ligament.[76] In man the dorsal longitudional ligament is innervated by the sinuvertebral nerve but this has not been confirmed in the canine.[31]

THORACOLUMBAR DISC DISEASE

Mixture and Barr (1934) reported in man that the common condition of sciatica was not due to chondromatous tumors arising from the disc space as previously thought but was a result of prolapsed or protruded disc material.[70] Disc protrusions can be dorsal lateral, dorsal medial, ventral or intravertebral. Clinical signs due to dorsal medial or dorsal lateral protrusions in veterinary medicine are reported.[50,76,78] Mixture and Barr's treatment of prolapsed disc material consisted of an intraspinal approach to the disc space by a dorsal laminectomy, gentle retraction of the neural tube and removal of the prolapsed disc material.[70] Riser (1946) demonstrated that these same compressions occur in the thoracolumbar spine of the canine and cited several veterinary authors who had previously reported the condition.[78] Riser gave an excellent summary of the clinical signs of disc disease and the gross spinal cord pathology.[78] Riser left the question of surgical correction open to the application of human surgical techniques to the canine.[78] Hansen (1951) in Europe also gave an in-depth review of the canine disease known as enchondrosis intervertebralis.[43,44,73] Hansen demonstrated, like Riser, that the extradural compressions were of discogenic origin and not neoplastic. Hansen histologically and grossly charted the transformation of the nuclear disc substance from its normal gelatinous nature to a cartilaginous state. Hansen observed that disc deterioration occurred in all breeds of canine but at a younger age in three specific breeds (Dachshund, French Bulldog, and Pekinese).[43,44]

Hansen described two types of disc prolapse following disc degeneration, the first (Type I) is a total rupture of the anulus with extrusion of nuclear material into the spinal canal.[43,44] The second (Type II) is a bulging of the anulus without nuclear extrusion, this type is seen in older nonchondrodystrophic breeds[43,44] and causes less severe sensory motor disturbances. Other authors have added subgroups to Hansen's classifications.[8,33]

Independently Greene (1951),[40] Redding (1951)[77] and Olsson (1951)[73] reported that the intraspinal approach to the disc used by Mixture and Barr was feasible in the canine. In Redding's study using normal dogs, all operated animals had temporary paresis following surgery for one to six days.[77]

Keyes and Compere (1932) curetted the lumbar disc of dogs by the intraspinal approach to develop a surgical model for disc protrusion.[58] Surgical incision of the anulus caused immediate prolapse of nuclear material in the normal disc, the result was a collapse of the disc space in relation to the amount of nucleus removed. In a follow up study Key and Ford with young nonchondrodystrophic canines did an intraspinal approach to study four methods of discectomy.[57] With the first two methods the disc space was curetted; one vigorously in an attempt to rupture the cartilage endplates resulting with bleeding. In the other method no attempt was made to distupt the endplate. In the third method only the anulus fibrosis was incised and the last method consisted of perforating both the anulus and nucleus with a twenty gauge needle. All four methods caused dorsal displacement of nuclear material and varying degrees of disc space collapse.[57]

Olsson (1951) demonstrated an alternative surgical approach to the disc space which was first mentioned by Lindblom.[73] It was called fenestration which is the creation of an incision into the anulus fibrosus and nucleus pulposus outside the vertebral canal.[73] Following the surgical incision the nucleus is evacuated with a curette or needle. Olsson felt fenestration to be safer than the intraspinal approach to the disc space because of less manipulation of the neural tube. Because of the differences in the location of the cauda equina in the canine and man, further trauma to the spinal cord would most likely be rendered with the intraspinal approach to the canine intervertebral disc space. Fenestration is less likely to cause bony structural instability than the intraspinal approach. Fenestration is ineffective for removal of prolapsed disc material dorsal to the vertebral body. The objective of fenestration is to relieve the pressure within the disc, thus elimination of the dynamic factor a prolapsed disc has on the spinal cord.[72]

Olsson used a dorsolateral approach with the animal in ventral recumbency to fenestrate the disc spaces caudal to T12-T13. This was accomplished by removal of the epaxial muscles from the dorsal arches and body of the involved vertebrae to the level of the rib or transverse process. For the spaces rostral to T12-T13 a ventrolateral fenestration was made by a transthoracic approach with the animal in lateral recumbency. Olsson advocated fenestration of more than one disc space or at least the disc spaces that had a higher incidence of prolapse.[73]

Leonard advocated a ventral approach for all thoracolumbar disc fenestrations.[62] With the patient in lateral recumbency both an intercostal and paracostal incision was used to expose the thoracolumbar disc spaces. Several individuals have modified Leonard's approach.[8] A dorsolateral approach by separating the multifidus muscles medially and the longissimus muscles laterally can be used.[88] A lateral approach to the disc space which elevates the longissimus dorsi upward or dorsomedially from the transvere vertebral processes or ribs[11,24,30,79] has been described.

Hoerlein (1978) noted the types of spinal surgery used by veterinary neurosurgeons.[49] Most surgeons use the intraspinal approach; laminectomy or hemilaminectomy to decompress the spinal cord.[51,52] The most frequently employed method was hemilaminectomy combined with prophylactic fenestration.[49] Others advocate nuclear removal by means of laminectomy, bilateral facetectomy and foraminotomy of the involved disc space.[76]

Flo and Bojrab question the value or need to do any type of decompressive spinal surgery other than fenestration.[8,30] These authors feel that the damage to the cord by the concussive force occurs at the time of disc expulsion and disc material in the spinal canal should not cause further damage.[8,30,65,72]

A percutaneous lateral approach similar to fenestration has been described and used in a limited number of clinical cases in man.[55] A hollow cannula is positioned into the nucleus pulposus under image intensification and the nuclear contents are evacuated by negative pressure from the involved disc space.[55] In man the intraspinal approach to the disc space by a laminectomy or hemilaminectomy is the preferred method.[25] Recently the use of microlumbar discectomy using an operating microscope has been reported.[39,87] With this technique the ligamentum flavum is incised, there is no laminectomy or curettage of the disc space, only the prolapsed disc material causing nerve root compression is removed.[39,87] The use of intradiscal steroids or chemonucleolysis has had variable results in man and dogs.[7,34]

Haas (1946) removed the disc by curettage which caused subsequent fibrous and some osseous bridging in older animals with complete osseous bridging in younger animals.[42] This has not been reported with clinical use of fenestration in veterinary surgery. In man there is not total agreement as to the benefits of discectomy alone or concurrent with vertebral body fusion.[1,32,61,84]

Filippi, Smith and Walmsley, Key and Ford, and Lipson and Muir have used rabbits and dogs as histopathological models to study nucleus pulposus prolapse.[66,67,80] Nuclear prolapse was created by an incision into the ventral portion of the anulus fibrosus.

In man structural failure for whatever reason produces radiographic findings of disc space narrowing, reactive sclerosis, lipping of adjacent vertebral bone and osteoarthrosis of the articular facets.[2,19,74] In the canine, changes in length or appearance of disc space, changes in adjacent vertebral endplates or alignment, masses within the intervertebral foramina and decrease in the disc length dorsally to give a wedge shaped appearance are used radiographically to assess the disc space.[4] Other radiographic findings include calcification of nucleus with or without annular calcification and calcified material in the spinal canal.[50] In one study neuroradiographic assessment was the most accurate means of localizing the pathological extruded disc and correlated with surgical location of the lesion in 75.4 per cent of the cases. Correlation of neurological exam to surgical site was 40 per cent.[13] Spinal myelography has also been used to localize disc lesions when the radiographic examination demonstrated no lesion or when more than one lesion is suspected.[13,33,50] Discograms are used in man to demonstrate tearing of the anulus without nuclear protrusion into the spinal canal[76] but have not been widely used in small animal surgery because of the small disc space.

Material and Methods

Six aged chondrodystrophic dogs[9] (Beagles) were obtained for the study, five intact females and one male all weighing between 25-30 pounds. Each dog's age was estimated to be greater than 8 years. All dogs were vaccinated[a], treated for intestnal parasites[b] and had physical examinations especially noting neurological function prior to surgery. Except for all dogs having extreme amounts of dental tartar, obese body conditions and one animal having a calcified umbilical hernia all others systems were normal. Serum chemistries, complete blood counts and Knotts tests were done. The animals were maintained in individual runs of three by six feet, fed a commercial dry dog food[c] ad libum with no supplementation and allowed unrestricted exercise. Two dogs because of housing limitations were housed for approximately four weeks in three by three foot wire cages.[d]

The dogs were divided into two surgical groups each with three members. Groups were assigned the following treatment regime according to disc space:

T10-T11 Disc 1	T11-T12 Disc 2	T12-T13 Disc 3	T13-L1 Disc 4	L1-L2 Disc 5	L2-L3 Disc 6	L3-L4 Disc 7	L4-L5 Disc 8
Groups I - 3 dogs							
C	T	C	T	C	T	C	T
Group II - 3 dogs							
T	C	T	C	T	C	T	C

C - no surgery T - treated, fenestrated

A total of 48 disc spaces were approached surgically, 24 of those had disc curettage, the rest had no surgery other than surgical exposure of anulus fibrosus.

Preoperative lateral and ventral dorsal thoracolumbar radiographs were taken of the anesthetized animals to rule out congenital or acquired lesions. The radiographic beam was centered on the vertebral body of T11, L2 and L4 for the lateral views and at T11 and L4 in the ventral dorsal view for a total of five radiographic projection (Table 3). It was difficult to keep the beam consistently centered at T11. The film focal distance was 40 inches, using radiographic film[e] in a screen with a 10:1 grid.[f] Radiographic studies were obtained immediately following surgery, at two, four, eight, twelve and sixteen weeks. The radiographic films were independently viewed by the surgeon and two radiologists. The radiologists were given a questionnaire to fill out concerning the preoperative and postoperative radiographs.

All surgical procedures were done by a dorsolateral muscle-separation approach on the animal's left side. Following surgery all animals were observed daily and had weekly physical assessments. Two animals had electrodiagnostic[g] examinations at eight weeks following surgery and two were examined at 12 weeks following surgery. The electromyography was done under gas[cc] anesthesia using a 26 gauge concentric electrode for recording a ground electrode which was 0.35 mm in diameter.[g]

Necropsies were scheduled at two, four, eight, and sixteen weeks postoperatively. All necropsies were done within fifteen minutes following intravenous euthanasia.[h] Grosa examination alone was performed on all thoracic and abdominal viscera. Gross and histopathological examinations were performed on the entire spinal cord and the thoracolumbar disc spaces that were in the surgical protocol.

After disarticulation of the skull between the occiptal condyles and the first cervical vertebra, the blade of a bone rongeur[i] was introduced into the vertebral foramen with the spinal cord medial and the bony lamina lateral. The bony lamina was transected on each vertebra down to its body from the cervical through the sacral vertebra. The spinal cord was examined grossly before its removal. The dura mater was clamped with forceps and the cord was removed by bilateral severance of the dorsal nerve roots as they entered the intervertebral foramen. No adhesions were noted around any of the nerve roots. The spinal cord was placed in 10 per cent buffered neutral formalin.

The dorsal longitudinal ligament was examined grossly to see if it had been ruptured. The spinal column was divided vertically along the midline with a butcher band saw[j] using the dorsal longitudinal ligament as a guide. The T10-11 rib was left intact to serve as an identification landmark. The intervertebral disc spaces were photographed and examined grossly. The right and left halves of the spinal column were placed in 10 per cent buffered neutral formalin for 2 days then transferred to a commercial decalcification solution[k] for 30 days. At that time the vertebral columns were removed from the decalcification solution. If the sections could be bent and incised with a razor blade knife without noise they were further processed. Those that were not completely decalcified were returned to the solution for an additional 7 days. Midsagittal sections of the decalcified vertebrae were taken to include the intervertebral space and vertebral bone rostrally and caudally (Table 4). The sections were 4 millimeters in thickness. These specimens were placed in numbered plastic crickets, and washed under cold running tap water for 2 hours. Following washing the specimens were dehydrated, embedded in paraffin[l], sectioned[m], mounted and stained with Masson's trichrome and hematoxylin and eosin (H + E).

Two different methods were used to measure disc length from the radiographs. One method was by direct measurement of disc length and the other was a ratio of disc length/caudal vertebral length X 100. Measurements to the nearest tenth of a millimeter were made using a caliper. The approximate center of the disc space was found and the calipers were placed on the endplates of the adjacent vertebral bodies to record the disc space length.

The disc spaces were assigned a number and the measurements recorded.

Disc Space	T10-11	T11-12	T12-13	T13-L1	L1-L2	L2-3	L3-4	L4-5
	Disc 1	Disc 2	Disc 3	Disc 4	Disc 5	Disc 6	Disc 7	Disc 8

For the measurements the serial ventral dorsal radiographs were not used, only the lateral radiographs were measured. At the vertebral body where the radiographic beam was centered, two disc space lengths on either side were recorded. In some cases three disc spaces had to be recorded. The same measurement system was used for the ratio method with the addition of the vertebral body length.

A two sample independent T test was used to analyze the data. Four time periods (M2-M5) following treatment were chosen in which to place the data immediately, two weeks, four weeks and eight weeks post treatment. There was insignificant data to include the twelve and sixteen week post treatment animals. Statistical analysis was used to determine if treatment had any effect on disc length. (Table 1 and Table 2)

SURGICAL TECHNIQUE

Following a twelve hour fast each animal had an indwelling catheter[n] placed into a cephalic vein. Atropine sulfate (0.2 mg/pound)[o] was given intramuscularly ten minutes prior to the induction of general anesthesia. General anesthesia was achieved by administration of intravenous sodium thiamyl[p] (8 mg/pound); then an endotracheal catheter was passed and anesthesia maintained with halothane[q] vaporized into oxygen and delivered through a semiclosed circle system.[r] All animals had preoperative and postoperative thoracolumbar radiographic examinations as previously described.

With the anesthesized animal in ventral recumbency the hair coat was clipped with a number 40 blade[s] and the surgical area prepared with iodophore detergent followed by a mist of iodophore solution which was allowed to dry.[t] The margins for the surgical field were cranial from the fifth thoracic vertebrae to four centimeters caudal to the crest of the ilium. Laterally the margins extended 10 centimeters on either side of the dorsal midline. The animal was placed in ventral recumbency on the surgical table with a vacuum positioner[u] so that the thoracolumbar spine was in dorsal flexion. Once positioned in the surgical suite, lactated Ringer's solution[v] (10 ml/pound/hour of surgery) was administered via the cephalic catheter.

All the dogs had the discectomy performed from their left side. After four corner draping with barrier towels and application of a fenstrated drape, a dorsal midline skin incision was made extending from the dorsal spinous process of the eighth thoracic vertebrae to two centimeters distal to the crest of the ilium. Skin barrier towels were applied to the edges of the incised skin. The multifidus muscles (Multifidus lumborum et multifidus thoracis) were divided medially and the longissimus (Longissimus lumborum and Longissimus thoracis) laterally.[88] These muscles were separated by blunt dissection with a periosteal elevator in the thoracic region. All disc spaces from T10-11 through L4-5 had the left lateral side of the anulus fibrous surgically exposed. Starting with T13-L1 and moving caudally all disc spaces were identified by palpating the transverse process of the vertebra directly caudal to it; the transverse process was then dissected free with periosteal elevators. Rostral to the intersection of the transverse process and the vertebral body is the disc space location. Care was taken to retract the ventral branch of the spinal nerve with a periosteal elevator[w] held in the nondominant hand while tunneling down to the disc space. It is difficult to identify the disc spaces rostral to T12-T13 because of their location rostral medial to the rib head. The exposure to these disc spaces is much better if first the origin of the levatores costrum muscle is partially severed.

The individual disc spaces were fenestrated according to patient assignment to Group I or Group II. A lateral section of the anulus fibrosus was removed with a number eleven scalpel blade[x] and the nucleus pulposus vigorously curetted with a series of dental tartar scrapers[y] which had been filed laterally to accommodate the size of the disc space. Following curettage and hemostasis, the surgical area was flushed with saline solution. The multifidus muscle was resected with Metzenbaum scissors where it had been devitalized. The thoracolumbar fascia was closed with a simple continous suture pattern using braided polyglycolic acid[z] or braided polyglactin 910[aa] suture material. The same suture pattern and material was used to close the subcutaneous layer, this layer was also attached to the underlying thoracolumbar fascia to eliminate serum accumulation. The skin edges were apposed with nylon suture[bb] using a simple interrupted suture pattern. Body bandages were not applied postoperatively. After anesthetic recovery the animals were returned to their runs without any additional medications or exercise restriction.

RESULTS

Clinical Findings

All the animals were able to stand and ambulate following recovery from general anesthesia. Four of the dogs initially had scoliosis of the operated side for 7-10 days. All the surgical incisions healed by first intention. One animal, dog 2, had bilateral alopecia of the surgical site which never became fully covered by hair.

Radiographic Evaluations

The radiographic readers had both ventral dorsal and lateral radiographs to view but found the lateral radiographs with the beam centered at L2 to be the most informative.

Preoperative films showed that 4 of 6 dogs had disc space narrowing at T11-12, T12-13, T13-L1, L1-2, L2-3 and L4-5. Narrowing was based on visual observation not measurement. One dog (Dog 2) had only twelve pairs of ribs.

Dog	Preoperative narrowed disc spaces	Spondylosis
1	T11-12, L1-2*, L2-3	L2-3, L7-S1
2		L7 - S1
3	T13-L1*	
5	T12-13*	
6	L2-3, L4-5	

*fenestrated

Two readers reported that the collapsed disc spaces were evident with the immediate postoperative films. The third reader reported the earliest detectable changes were at two weeks. In one dog the changes were not evident until 8 weeks.

Reader	1	2	3
Dog 1 (16738)	Immediate Postoperative	Immediate Postoperative	2 weeks
Dog 2 (7997)	" "	" "	2 weeks
Dog 3 (J8)	" "	" "	2 weeks
Dog 4 (8062)	" "	" "	2 weeks
Dog 5 (8267)	" "	" "	2 weeks
Dog 6 (E-30)	" "	" "	8 weeks

The primary radiographic change was a narrowing of the disc space. There were no changes involving the vertebral bodies or diarthrodial joint.

Electromyography Study

Dog 3 and 6 had an electromyographic examination 8 weeks following surgery under gas[cc] anesthesia. The area examined was the epaxial muscles from T8 region to the sacrum. With dog 3 the right side of the dog was quiescent. On this animal's left side at T10-11 region there were occasional positive waves and fibrillation potentials. At the L4 level a few small potentials of 2-4 msec duration and < 500 uv amplitude were seen. Dog 6 on the right and left side had fairly large potentials of apparent voluntary origin from T7 to L4 region even though the animal was under general anesthesia. These potentials tended to persist for 30-45 seconds after placement of the recording needle and were 2-5 msec duration with peak to peak amplitude of 3.5-4 mv. In addition the left side showed fibrillation potentials in the T10-T11 area. In the T10 to L4 region there were some small potentials of 2-3 msec duration and < 500 uv in amplitude. Also in the T10 to L4 region there were recorded some bizarre small multiphasic potentials with 7-8 peaks of < 200 uv amplitude with 7-8 msec duration.

Dogs 1 and 2 had recordings taken at 12 weeks following surgery. On dog 1 the right side was quiescent except for some voluntary activity above T13 which correlated with respiratory movements. On the left side a rare fibrillation potential was found at T11-L1 region. At the T 11 to L4 region some spontaneous small potentials of up to 400 uv in amplitude and 2-5 msec in duration were recorded. Dog 2 on the right side was quiescent except for a few rare fibrillation potentials at the T13-L1 level. Some voluntary activity above L1 was seen to correlate with respiratory movement. On the left side of dog 2 fibrillation potentials at the T11-12 level were seen.

Gross Post Mortem Observations

There was no evidence of adhesions between the dura mater and the bony spinal column upon removal of the spinal cord. No adhesions were noted around any of the spinal nerve roots. The dorsal longitudinal ligament was intact. After the spinal column was divided along the dorsal longitudinal ligament there did appear to be some differences between the treated and controlled disc spaces. All disc spaces were white in appearance and material thought to be nucleus pulposus bulged from the cut surfaces of the untreated disc. There was no bulging from the treated disc. Grossly one could not tell if there were differences in disc length. These findings were observed at 2,4,8, and 16 weeks.

HISTOLOGY OBSERVATIONS

Spinal Cord

One dog (dog 5) had evidence of wallerian degeneration of the spinal cord at disc space T10-11 on the left side. The wallerian degeneration involved the white matter of the lateral and ventral funiculus. Gitter cells and axon swelling was observed.

Control Disc

On sagittal section the ventral portion of the disc space was funnel shaped and the skeletal epaxial muscle attachments seen. Dorsally the lamellae of the anulus fibrosis were wavy and fissured, occasional nests of cartilage cells were observed between the lamellae. The severed bundles of the spinal nerves could be seen for orientation. There was a marked indentation of the cancellous vertebral bone as it conformed to the nucleus. A thin line of hyaline cartilage separated the vertebral bone from the disc. Some disc nuclei appeared coagulated, some had basophilic staining calcified disc material present. No inflammatory cells were present. There was a separation between the nucleus and anulus which might represent a loss of material or shrinkage during processing. All 24 nonoperated disc spaces demonstrated some degree of degeneration. Presence of a transitional zone was not seen in these mature discs; only an anulus fibrosus and nucleus pulposus was evident.

Treated Disc -- 2 weeks (Table 4)

There was a definite cavitation of the nucleus pulposus; the cavity contained disc material that was incompletely curetted. Remnants of disc material composed of amorphous ground substance with a few cartilage cells was observed. At the vertebral body endplate, a cement line was observed between osteon and noncalcified tissue. Chondrocytes were present in lacunae which had well defined rims; some lacunae contained a single cell while other contained two to four. The chondrocytes appeared to be proliferating, especially at the junction between the annular-nuclear border and the vertebral body. No inflammatory cells, blood vessels, or granulation tissue were observed.

Treated Disc -- 4 weeks

There was an advancing line of acidophilic ground substance and mesenchymal cells that were attempting to bridge the nuclear deficit. The proliferating line appeared to be hyperplastic cells that originated at the junction where the vertebral body adjoins the annular-nuclear border. Some of the mesenchymal cells were differentiating into chondrocytes.

There were defects in the hyaline cartilage vertebral endplates that occurred due to vigorous curettage. Because there was little nuclear material present none hernitated into the cancellous vertebral body as is the case with a Schmorls nodule. Fibrous tissue from the nuclear area did fill the deficit. The changes occured in Dog 1 at disc spaces T12-13 and L1-2.

Treated Disc -- 8 weeks

There was a proliferation of chondrocytes from the outer perimeter advancing on the nuclear deficit. Chondrocytes were forming columns; nests of chondrocytes were present. The defects in the cartilage plate were filled with chondrocytes from the nucleus.

Treated Disc -- 16 weeks

The defect in the cartilage plate had been repaired. The surgical defect in the nucleus pulposus was filled with fibrocartilage. Under polarized light, mature collagen was observed filling the nuclear area.

Statistical Analysis

A two sample independent "t" test was used to evaluate the effect of treatment versus no treatment on the eight disc spaces. Two methods of disc length measurement were used as previously described.

Disc 1, 2, 4, 6, and 8 showed differences between the mean of the treated and control disc with direct measurement. Disc 3, 5, and 7 did not always show mean differences and in some cases the mean of the treated disc was larger than the control.

Disc 1, 2, 4, 6, and 8 all showed significant differences at the .05, .10, and .15 levels at some postoperative time (M), but it was not consistent with increasing time periods (M_2 to M_5). (Table 1).

With the ratio of disc space/vertebral body length disc 1, 2, 4, 6, and 8 show mean differences between treated and control discs. Disc 3, 5, 7 did not show mean differences as great as the other discs and in some cases the mean of the treated disc was greater than the control disc (Table 2).

DISCUSSION

The dorsolateral muscle-separating approach provided adequate exposure of the eight thoracolumbar disc spaces through a single operative incision with minimal disruption of the muscular attachments to the vertebrae.[88] Some permanent electromyographic changes did occur with the surgical approach but clinically this did not present a problem nor was there a muscular depression where the surgery had been performed. No electromyographic studies could be found in the literature for the other surgical approaches. The scoliosis was due to neurapraxia of the dorsal branch of the spinal nerves and the ventral branch of the lumbar nerves that innervate the epaxial and sublumbar muscles.[5] In all cases this was a temporary condition which did not encumber the affected animals. The wallerian degeneration of dog 5 was an unexpected finding. This degenerative change occurred without clinical signs and with no gross necropsy evidence that the dorsal longitudinal ligament was breached. It is unknown if clinical signs would have developed had the animal survived for a longer time frame. This occurred in one of the first dogs operated on and demonstrates the problems that can arise by surgical inexperience. The damage could be explained if the curettage instrument was inadvertently introduced into the intervertebral foramen.

Radiographically disc space collapse in some cases was noted immediately following the surgical procedures. Once collapsed the narrowing did not seem to be progressive. With the use of nuclear chymopapain injections in nine month old beagles there was an initial narrowing, then a significant rewidening of the disc length over a two year period.[34,86] With this sixteen week study no disc space rewidening was observed.

With iatrogenic damage to the cartilage endplate no additional changes occurred at the interspace on plain serial radiographs. Histologically these cartilage defects reacted similar to a Schmorl's node. Schmorl's node has been described as the herniation of disc substance through the cartilage plate into the cancellous bone of the adjacent vertebra.[22] A reactive change occurs in the bone around the prolapsed nucleus which is seen radiographically and histologically in the form of bony sclerosis.[22]

In man, with loss of the nucleus pulposus, the axis of motion is shifted to the articular facets leading to osteoarthrosis of this joint.[3,58] This was not observed radiographically to occur in any of the operated or unoperated disc spaces. If the axis of motion was changed it was not severe enough to damage the diarthrosis. Another explanation would be the fact that bipedal stature puts more stress on the diarthrodial joint than quadruped stature.[47]

Experimental studies that have limited or fused the intervertebral disc spaces in immature dogs have resulted in significant compositional changes of the discs encompassed by the fusion and those adjacent to it.[84] All histological changes seen in the control disc spaces throughout this study were thought to be normal age changes, no changes seemed related to discectomy of an adjacent disc space.

Absolute measurements of disc length may be misleading. There are problems associated with measurement or visual estimation of disc length:

a. difference in specimen orientation with respect to the central roentgen beam. As the distance from the central beam increases there is an artificial narrowing of the disc space with an elongation of the vertebral body. Lateral bending and longitudinal axis rotation toward or away from the roentgen beam and radiographic plate will affect disc length.[2]

b. vertebral bodies do not have well defined corners due to overlying gas and ingesta patterns. Some judgements must be made by the reader.

Visual recognition of space collapse was noted early following surgery and was noted in all operated disc spaces. Direct and ratio measurements established trends of narrowing in Disc 1, 2, 4, 6 and 8. Trends were never established for Disc 3, 5 and 7. No explanation can be given for this latter finding since all three of the latter were collapsed radiographically in the treated animals. This inconsistency must be due to error in measurement from the radiographs. Disc 3 (T12-13) is reported to have the highest incidence of protrusion. Disc 5 (L1-2) is in the range where the largest incidence of protrusion occurs.

The data for statistical analysis was reviewed in several ways. A split-plot analysis of variance where the dogs were the main plot and time was the subplot was considered. Because of repeated measurements on the same dog, observations over time within the same dog were likely to be correlated thus violating the assumption of a variance analysis over time.

Hence, it was deemed that the "t" test for control and treated disc spaces for each time period would be the most viable. A paired sample "t" test and a two sample independent "t" test for each time period was considered. With the paired sample "t" test the preoperative measurement was compared to the mean of the postoperative measurements. Using this method no significant differences were shown. The averaging of the postoperative time periods destroyed the differences and had fewer degrees of freedom.

The two sample independent "t" test was more sensitive to uncover differences between treated and control discs but it also may claim differences when there were none.

There was disc collapse even when incomplete nuclear evacuation was performed. In man there are opposing views on disc removal. One group prefers to remove as much disc material as possible, the other group removes only the prolapsed portion.[39,87] This latter group also stress blunt not sharp incision of the anulus fibrosus. By blunt probing the integrity of the anulus is maintained and the anular wound heals to lessen the possibility of further nuclear herniation.[39,87] Markopf did a series of static test (load versus displacement, creep and load relaxation) on cadaver discs which had undergone discectomy. After an initial readjustment the discectomy specimens had comparatively equal compressive stiffness as did intact cadaver disc. Markopf feels the reparative powers of the anulus are great and responsible for viscoelastic and static stiffness.[69] In this study the majority of anulus was left intact except for the operative incision.

Capanna calculated the total disc volume by measuring disc material removed and the amount of contrast material that could be injected into a disc at the time of discectomy. The average percentage of disc material removed surgically was six per cent. There was no correlation found between percentage of disc removed and the patient's recovery.[17] Unmeasured amounts of disc material were removed from every operated disc in this study.

The histologic changes in the curetted canine interspace resemble those reported for the rabbit, following ventral incision of the anulus and nuclear proplase.[66,67,80] Key and Ford reproduced disc protrusion in the dog by an intraspinal approach and curettage of the disc space. They did not achieve fibrosis of the nucleus. Only the anulus healed at its superficial layer. The nucleus remained a fluid filled cavity.[57] No attempt was made to mechanically evacuate the nucleus in these experimental models.

Filippi described a regeneration of the anulus by a proliferation of fibrocartilage from each vertebral plate. The anulus was restored after 40 days and at 100 days fibrocartilage occupied the nucleus pulposus.[80]

Smith and Walmsley observed that the superficial fibers of the anulus heal within 3 weeks with fibrous proliferation.[80] There is a proliferation of cartilage cells around the superficial part of the wound which calcified, then ossified. The ossification occurs between the adjacent vertebra leading to ankylosis of the vertebrae by one year. The deeper areas of the anulus were slowly healing over one year with chondrification. Deeper layers of the anulus heal slower because of the avascularity.[80] The nucleus showed a rapid proliferation of chondrocytes during the first six months. At nine weeks chondrocytes were in a linear arrangement and found in alignment with the long axis of the vertebral column. At 21 months the nucleus contained few cells and was composed of dense collagen.

Lipson and Muir showed that the chondrocytes formed fibrocartilage and within 6-8 weeks the fibrocartilage was less cellular. After this period, coarse fibrous tissue with clefts occupied the nuclear area.[66,67] Taylor feels that fibrocartilage, like hyaline cartilage has a limited and abortive capacity to repair a defect. This is based on findings in man when a defect in the disc space may persist following prolapse.[83]

In this study the anulus healed more rapidly than in the other reported studies because the prolapsed nucleus was removed and not left between the cut edges of the anulus. When the nuclear material was removed, no osteophyte formation was observed. There did not appear to be a continual nuclear material prolapse following surgery as others have reported[30] because the anulus sealed itself very quickly.[69] Curettage did not cause any lipping or ankylosis of adjacent vertebral bodies as it did in the rabbit models.

Electron microscopy of chondrocytes has been described at the articular epiphyseal complex.[12,18] Ultrastructurally two chondrocytes habe been described, a dark and light chondrocyte.[12] The light cells are more numerous and a recent study suggests three distinct types. The functional significance of these three light cells is unknown.[12]

The chondrocyte response may be due to mechanical forces exerted on the disc space.[66,67] Mechanical factors, particularly combined with variations in oxygen tension, have been demonstrated to be crucial in the behavior of mesenchymal tissue in culture conditions.[6] From this study these mechanical forces appear to be concentrate at the annular nuclear border and the vertebral body endplate.

SUMMARY AND CONCLUSION

This study demonstrated that the dorsal lateral muscle-separating approach gave a limited but adequate exposure of the lateral anular disc wall. This approach was used in 48 thoracolumbar disc spaces of six dogs. Anatomical knowledge of this area is mandatory because it can result in clinically undetected damage to the spinal cord. While the surgical approach left no visual defects or locomotor dysfunction, electromyographic findings were abnormal in at least two dogs. Perhaps this technique should be compared electromyographically to the other surgical approaches.

Radiographically all operated disc spaces showed evidence of collapse. Histologically the chondrocytes were the main cell involved in a reparative process.

Conclusions drawn from this study include:

1. Radiographically disc space collapse was complete not just a dorsal wedging of the adjacent vertebral bodies. Disc space collapse was evident immediately postoperatively in the majority of the operated disc spaces. Disc space collapse was the only radiographic change seen in this study, there was no evidence of ventral bridging by bone. The disc space collapse did not appear to be progressive with time.

2. Subjective visual estimation of disc space collapse was more accurate than statistical analysis from radiographic measurements. Radiographic positioning, subjective decisions about disc boundaries, and the difficulty in detecting small changes were most likely the source of this problem.

3. Disc space collapse was evident even when not all the disc material was excavated at the time of surgery. Presence of old disc material could be detected histologically.

4. Histologically the body responded within two weeks to curettage by a reparative attempt to replace the nucleus. Few anular reparative changes were noted leading one to believe that the anulus seals almost immediately following trauma. There were no indications that nuclear material of the operated disc space continued to prolapse from any anular defect.

5. Chondrocytes appear to be the main cells involved in the reparative process. Chondrocyte response may have been the result of added stresses which were once applied to the intact nucleus. If chondrocytes are the main reparative cells it is surprising to find that chondrocyte activity was not present in the unoperated degenerated discs. Apparently there must be actual loss of nuclear material to initiate a cellular reaction.

6. Removal of the nucleus didnot appear to place additional stress on the diarthrodial vertebral joint of either the operated or unoperated disc space. This conclusion was based on radiographic examination of the diarthrodial joints.

Further studies over a longer time period need to be done to observe the long term reparative process in nuclei which have undergone curettage.

TABLE 1

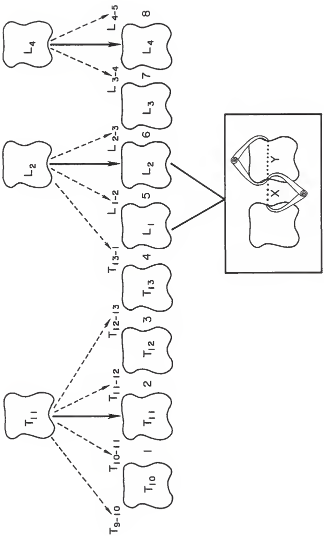
Direct Space Measurement - Disc #1

	TRT	N	MEAN	STD. DEV.	T	DF	PROB > /T/
M_2	C	3	2.06666667	0.11547005			
	T	3	1.43333333	0.40414519	2.6099	4.0	0.0594
M_3	C	1	2.00000000				
	T	3	1.66666667	0.57735027	0.5000	2.0	0.6667
M_4	C	3	1.93333333	0.11647005			
	T	2	1.90000000	0.14142136	0.2828	3.0	0.7868
M_5	C	2	1.50000000	0.70710676			
	T	2	2.00000000	0.28284271	-0.9285	2.0	0.4512
Disc #2							
M_2	C	3	1.86666667	0.11547005			
	T	3	1.33333333	0.41633320	2.1381	4.0	0.0993
M_3	C	5	1.80000000	0.40000000	0.5222		
	T	3	1.60000000	0.52915026	0.5222	4.0	0.8291
M_4	C	2	1.90000000	0.14142136			
	T	3	1.00000000	0.00000000	12.0748	3.0	0.0012
M_5	C	2	2.10000000	0.14142136			
	T	2	1.20000000	0.00000000	9.0000	2.0	0.0121
Disc #3							
M_2	C	3	1.86666687	0.11547005	0.1562		
	T	3	1.83333333	0.35118846	0.1562	4.0	0.8835
M_3	C	3	2.06666667	0.11547005			
	T	3	1.80000000	0.60000000	0.7559	4.0	0.4918
M_4	C	3	1.86666667	0.23094011			
	T	2	2.20000000	0.84652814	-0.6956	3.0	0.6367
M_5	C	2	1.80000000	0.28284271			
	T	2	1.90000000	0.14142136	-0.4472	2.0	0.6885
Disc #4							
M_2	C	3	2.46666667	0.50332230			
	T	3	1.70000000	0.55677644	1.7692	4.0	0.1518
M_3	C	3	2.46666667	0.46186022			
	T	3	1.53333333	0.46189022	2.4749	4.0	0.0886
M_4	C	2	3.00000000	0.00000000			
	T	3	1.33333333	0.41633320	5.3709	3.0	0.0128
M_6	C	2	3.00000000	1.13137085			
	T	2	1.10000000	0.14142136	2.3587	2.0	0.1425
Disc #5							
M_2	C	3	2.06666667	0.11647005			
	T	3	1.93533333	0.23094011	0.8944	4.0	0.4218
M_3	C	3	2.33333333	0.41633320			
	T	3	2.40000000	0.69282032	-0.1429	4.0	0.8933
M_4	C	5	2.26666667	0.11547005			
	T	2	2.80000000	0.56568542	-1.0742	3.0	0.3615
M_5	C	2	2.20000000	0.28284271			
	T	2	2.10000000	1.27279221	0.1085	2.0	0.9235
Disc #6							
M_2	C	3	2.56666667	0.81445278			
	T	3	1.93333333	0.11547005	1.3336	4.0	0.2632
M_3	C	3	2.73333333	0.46166022			
	T	3	1.80000000	0.52915026	2.3018	4.0	0.0828
M_4	C	2	2.40000000	0.56666542			
	T	3	1.66666667	0.57735027	1.4008	3.0	0.2558
M_5	C	2	2.60000000	0.56566542			
	T	2	1.60000000	0.28264271	2.2361	2.0	0.1648
Disc #7							
M_2	C	3	2.33333333	0.41633320			
	T	3	2.20000000	0.72111026	0.2774	4.0	0.7953
M_3	C	3	2.40000000	0.34641018			
	T	3	2.06666667	0.30550505	1.2500	4.0	0.2784
M_4	C	5	2.06666667	0.11547005			
	T	2	2.40000000	0.84852814	-0.7319	3.0	0.6172
M_5	C	2	2.40000000	0.28264271			
	T	2	2.30000000	0.70710678	0.1857	2.0	0.8696
Disc #8							
M_2	C	3	2.26666667	0.84291005			
	T	3	1.93333333	0.11547006	0.8838	4.0	0.4287
M_3	C	3	2.26666667	0.64291005			
	T	3	1.33333333	0.41633320	2.1108	4.0	0.1024
M_4	C	2	2.40000000	0.56566542			
	T	3	1.26666667	0.46188022	2.4885	3.0	0.0886
M_5	C	2	2.10000000	0.70710678			
	T	2	1.40000000	0.56566542	1.0932	2.0	0.3884

TABLE 2

Disc #1 - Disc space/vertebral body length

	TRT	N	MEAN	STD. DEV.	T	DF	PROB > /T/
M_2	C	3	12.77000000	0.92989247			
	T	3	8.35686667	2.31647002	3.0624	4.0	0.0376
M_3	C	1	11.76000000				
	T	3	9.75666667	3.35792098	0.5167	2.0	0.6568
M_4	C	3	12.13666667	0.45456939			
	T	2	11.17000000	0.83436600	1.7413	3.0	0.1800
M_5	C	2	8.95500000	4.77297077			
	T	2	11.69000000	1.56977705	-0.4883	2.0	0.6738
Disc #2							
M_2	C	3	10.61000000	1.08678425			
	T	3	8.03000000	2.73622002	1.5178	4.0	0.2037
M_3	C	3	10.30666667	1.99560393			
	T	3	8.65000000	3.11614047	0.3072	4.0	0.7740
M_4	C	2	11.14000000	0.69296465			
	T	3	6.12333333	0.15534907	13.0936	3.0	0.0010
M_5	C	2	12.02500000	1.29400541			
	T	2	7.32000000	0.25455844	5.0454	2.0	0.0371
Disc #3							
M_2	C	3	9.86333333	2.76625456			
	T	3	8.95333333	2.14311300	-0.0445	4.0	0.9666
M_3	C	3	12.02000000	0.67549981			
	T	3	8.99666667	3.33500123	1.0299	4.0	0.3612
M_4	C	3	10.54333333	1.11073549			
	T	2	12.31000000	6.37401154	-0.5987	3.0	0.5818
M_5	C	2	10.27000000	1.37176716			
	T	2	10.55500000	0.78488853	-0.2550	2.0	0.8225
Disc #4							
M_2	C	3	12.64333333	2.87273969			
	T	3	8.88333333	2.96466412	1.5776	4.0	0.1898
M_3	C	3	12.12666687	2.64643786			
	T	3	8.36000000	2.67761518	1.7660	4.0	0.1522
M_4	C	2	15.39500000	0.65661436			
	T	3	7.19666667	2.07051523	5.2182	3.0	0.0137
M_5	C	2	16.62500000	6.89019948			
	T	2	5.96000000	0.98994949	2.2884	2.0	0.1483
Disc #6							
M_2	C	3	10.36666667	0.55075705			
	T	3	8.35866667	1.06610926	1.4385	4.0	0.2242
M_3	C	3	12.28333333	2.19135422			
	T	3	11.37666667	3.25146634	0.4005	4.0	0.7083
M_4	C	3	11.65000000	0.66787725			
	T	2	12.84500000	2.80620887	-0.8018	3.0	0.4813
M_5	C	2	11.51500000	1.39300038			
	T	2	10.31000000	8.42052957	0.2594	2.0	0.8198
Disc #6							
M_2	C	3	11.67333333	3.66061926			
	T	3	8.52333333	0.82561088	0.9824	4.0	0.3772
M_3	C	3	12.72333333	1.56771324			
	T	3	8.93333333	2.61023626	2.1488	4.0	0.0981
M_4	C	2	11.80000000	2.94156421			
	T	3	8.20000000	2.77126129	1.3165	3.0	0.2796
M_5	C	2	11.99500000	2.14253355			
	T	2	7.96500000	1.46371104	2.1884	2.0	0.1592
Disc #7							
M_2	C	3	11.23000000	2.06856410			
	T	3	10.06000000	3.25815899	0.5238	4.0	0.6282
M_3	C	3	11.56666667	1.54078964			
	T	3	9.40666667	1.44638630	1.7703	4.0	0.1514
M_4	C	3	9.90333333	0.50836339			
	T	2	10.92000000	3.64666089	-0.4928	3.0	0.6559
M_5	C	2	11.63500000	1.05358910			
	T	2	10.40500000	3.11834091	0.5286	2.0	0.6600
Disc #8							
M_2	C	3	9.97333333	2.99072455			
	T	3	8.94666667	0.32807519	0.5910	4.0	0.5863
M_3	C	3	10.27000000	3.19016606			
	T	3	8.24666667	1.72865073	1.9206	4.0	0.1272
M_4	C	2	10.69500000	2.39709199			
	T	3	8.00666667	2.06163413	2.3430	3.0	0.1008
M_5	C	2	9.40000000	2.96984646			
	T	2	6.54500000	2.52437121	1.0359	2.0	0.4091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L2
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T9-10
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4
L1
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X
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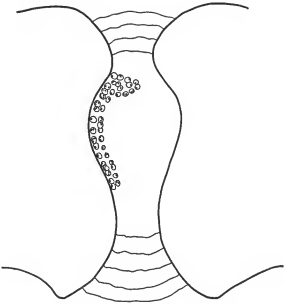
TABLE 4



FOOTNOTES

a) Vanguard CA_2P + CPV. Norden Laboratories, Inc. Lincoln, NE 68501

b) Strongid T. Pfizer Inc. New York, NY 10017

c) Science Diet. Hill's Pet Products, Inc. Topeka, KS 66601

d) Shor-Line. Kansas CIty, MO

e) Cronex X-ray Film. E.I. Dupont DeNemours and Co. Photo Products Dept. Wilmington, DE 19898.

f) Quanta III. E.I. Dupont De Nemours and Co., Photo Products Dept. Wilmington, DE 19898

g) TECA Electromyograph Model M. TECA Corporation. Pleasantville, NY 10570

h) T61. American Hoechst Corporation, Animal Health Division, Somerville, NJ 08876

i) Ruskin Rongeur. Miltex Instrument Co. Lake Success, NY 11042

j) Model 5216-DHS. Hobart MFg. Co., Troy, OH

k) 5-sulfosalicylic Acid. MCB Mfg. Chemist, Inc. Associate of E. Merck. Darmstadt, Germany

l) Autotechnicon. The Technicon Co. Chauncey, NY

m) AO Spencer model 900 table microtome, VWR Scientific Co., P.O. Box 3200, San Fransisco, CA 94119

n) Sovereign Indwelling Cather. Monoject, St. Louis MO 63103

o) Atropine Sulfate Injection USP Med-Tech Inc., Elwood, KS 66024

p) Bio-tal. Bio-Ceutic LAboratories Inc. St. Joseph, MO 64502

q) Fluothane. Ft Dodge Laboratories, Inc. Ft. Dodge, IA 50501

r) Fluotec MK III. Distributed by Fraser Harlake. Orchard Park, NY 14127

s) Oster, Milwaukee, WI

t) The Purdue Frederick Co. Norwalk, CT 06856

u) Olympic Vac-Pac Olympic Medical Seattle, WA

v) Lactated Ringers Injection USP. Travenol Laboratories, Inc. Deerfield, IL 60015

FOOTNOTES (Cont)

w) Adson Curved Periosteal Elevator. American V. Mueller. Chicago, IL 60648

x) Bard-Parker surgical Blade. Becton Dickinson and Co., Lincoln Park, NJ 07035

y) Clawtype canine tartae scraper, Arista surgical Supply Co., Inc.

z) Dexon "S" American Cyanamid Co., Pearl River, NY 10965

aa) Coated Vicryl, Ethicon Inc., Somerville, NJ 08876

bb) Ethilon, Ethicon Inc., Somerville, NJ 08876

cc) Metofane, Pitman Moore, Inc., Washington Crossing, NJ 08560

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APPENDIX

A	B
C	D
E	

Figure 1

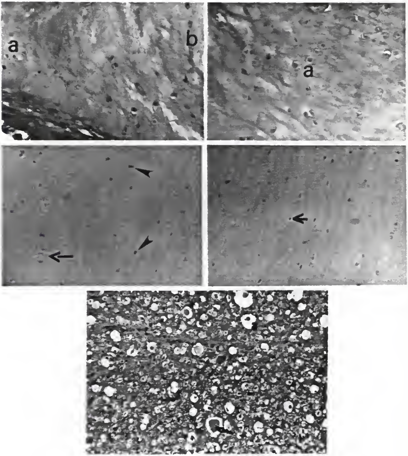
A. Nontreated disc. Notice disc reveals age changes, notice nest of cartilage (a) and single cartilage cells (b)

B. Nontreated disc section was taken close to center of disc see single chondrocytes.

C. Nontreated disc. Section is from periphery of disc notice arrangement of chondrocytes in column (open arrow) and single chondrocytes toward the center of the disc (closed arrow).

D. Nontreated disc. Section is from center of disc, single chondrocytes in cavity (arrow).

E. Section of spinal cord from animals left side showing Wallerian degeneration. Notice Gitter cell (a) and swollen axon (arrow).


a
b
a

A	B
C	D
E	F

Figure 2: Two weeks following curettage of the nucleus. H + E stain; X120.

A. Cavity at top created by curettage.

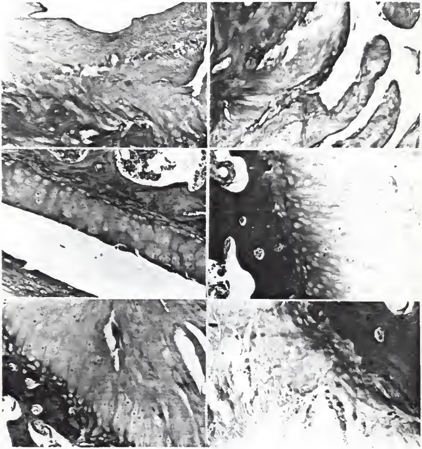
B. Bone at top left with frayed cartilage remnants of nucleus pulposus.

C. Proliferation of chondrocytes on the sides of the gap created by curettage.

D. Cement line with osteon to the left and chrondrocytes to the right. Chondrocytes are beginning to form columns.

E. Proliferation of chondrocytes in columns.

F. Nonoperated disc space with degenerated nucleus pulposus.



A	B
C	D
E	F
G	H

Figure 3: Four weeks following curettage of the nucleus. H + E stain; X120.

A. Clusters of proliferating chondrocytes (a), area of chondrocytes (b), cavity created by surgery (c).

B. Area of proliferation of chondrocytes at junction between anulus and nucleus (a).

C. Cartilage debris from surgery remains where nucleus was curettage (a). Proliferating zone of chondrocytes (b).

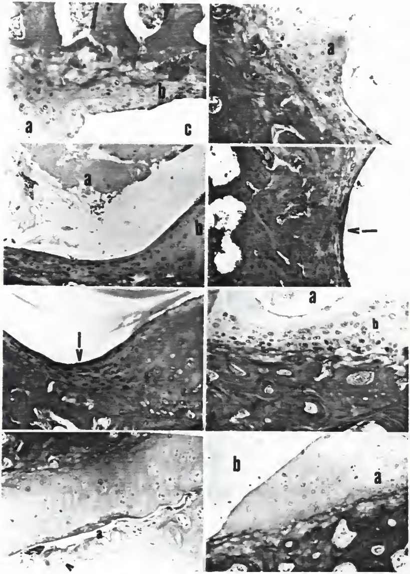
D. Arrow points to superficial defect in cartilage endplate do to surgical curettage.

E. Arrow points to deep defect in cartilage endplate do to surgical curettage.

F. Debris remaining in surgically created defect (a). Proliferating zone of chondrocytes (b).

G. Arrow points to nest of chondrocytes that surrounds a narrow surgically created deficit (a). It is obvious that not enough nucleus was removed.

H. Proliferating zone of chondrocytes are shown (a) along with the deficit created in the nucleus (b).


a
b
c
a
a
b
a
b
b
a

A	B
C	D
E	F

Figure 4: Eight weeks following curettage of the nucleus. H + E stain; X120.

A. Arrow pointing out chondrocytes nest around cluster of other chondrocytes. Gap from initial curettage is present with debris (a).

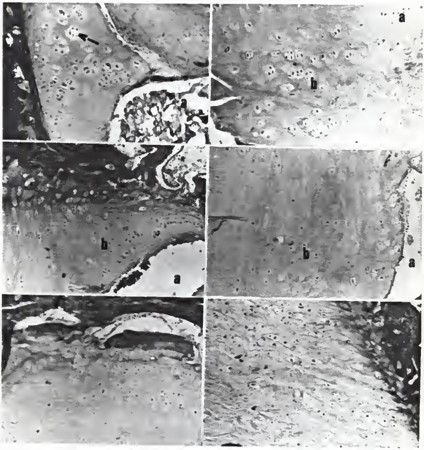
B. Chondrocytes in a nest (b) that are arranged in columns (a).

C. Surgical defect (a) with proliferation of chondrocytes near junction between anulus and nucleus (b).

D. Proliferation of chondrocytes (b) to fill in the surgical defect (a).

E. Continuation of filling the deficit.

F. Chondrocytes in columns on right side with the advancing edge of chondrocytes on the left side.


a
b
b
a
b
a

G	H
I	J
K	

Figure 4 (continued): Eight weeks following curettage of the nucleus.

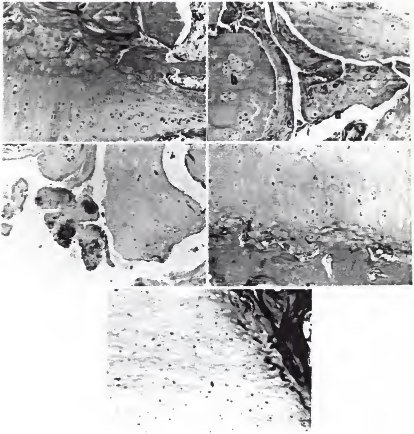
G. Proliferating zone of chondrocytes

H. Surgically created defect with debris (a) beening filled in by the proliferating chondrocytes with nests of cells (b).

I. Dead debris in the curetted gap with advancing chondrocytes.

J. Junction between vertebral body bone at the bottom and the proliferating chondrocytes at the top.

K. Trichrome stain showing nonossified area on the left with ossified area on the right.



A	B
C	D
E	F

Figure 5: Sixteen weeks following curettage of the nucleus.

A. Bone is shown at the bottom on the left side with the advancing chondrocytes which has almost filled the surgical deficit.

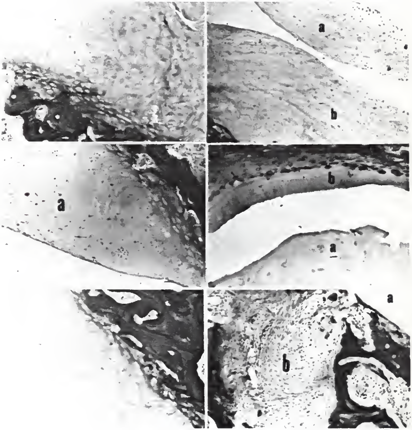
B. Cartilage (a) remains in the deficit while proliferation of chondrocytes continues (b). Edge of bone can be seen at bottom left.

C. Large area of proliferating chondrocytes (a).

D. Large area of old cartilage that was incompletely removed (a) and new proliferating chondrocytes (b).

E. Closer view of proliferating chondrocytes on left side and vertebral body endplate right.

F. View of the surgical deficit (a) and core of fibrosus tissue (b) similar to Schmorls node that replaces boney deficit in vertebral body endplate do to surgical curettage.


a
b
a
b
a
a
b

G	H
I	J
K	

Figure 5 (continued): Sixteen weeks following curettage of the nucleus pulposus.

G. Arrow points to boney deficit that has filled in with a fibrosus core similar to Schmorls node. The proliferating chondrocytes are also shown (a).

H. View of proliferating chondrocytes in nucleus (a), the vertebral bone is shown (b) and the area (c) where a fibrous core of tissue is responding to the boney deficit created.

I. Bone on the left side with advancing chondrocytes to the right.

J. Proliferating zone of chondrocytes (a) advancing on the deficit filled with debris.

K. Chondrocytes filling the deficit (a), vertebral body endplate (b).

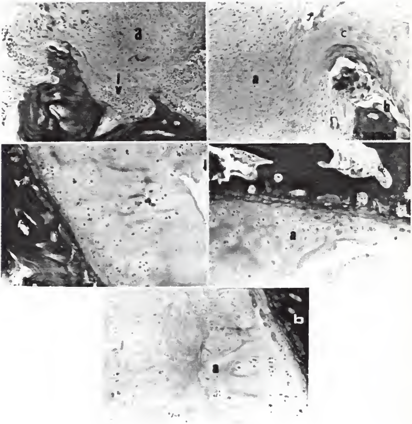

a
a
c
b
a
a
b

Figure 6. Dog 1, Immediately following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4. Note ventral spondylosis of L2-3 and a bony bridging at L7-S1. Preoperatively this animal had evidence of disc space collapse at T11-12, L1-2 and L2-3.

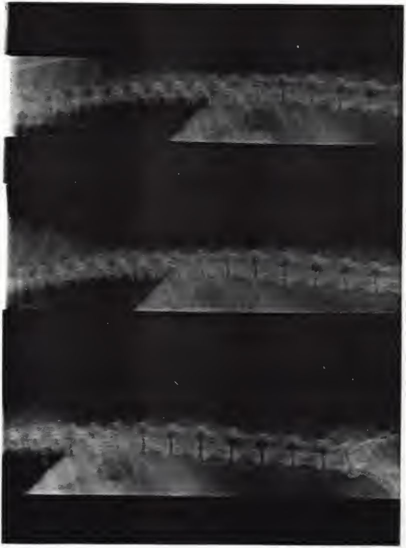


Figure 7. Dog 1, two weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.

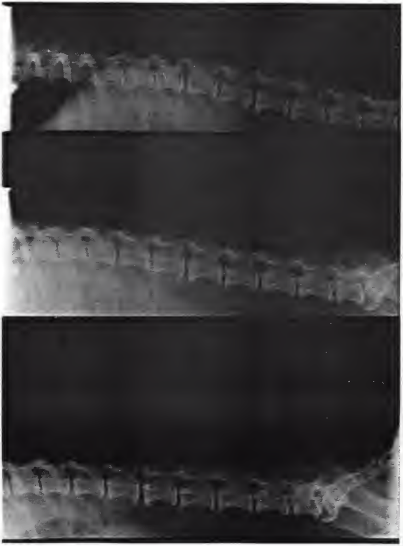


Figure 8. Dog 1, four weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.

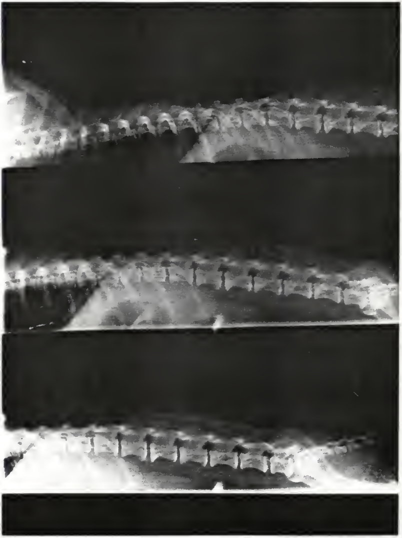


Figure 9. Dog 1, eight weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.

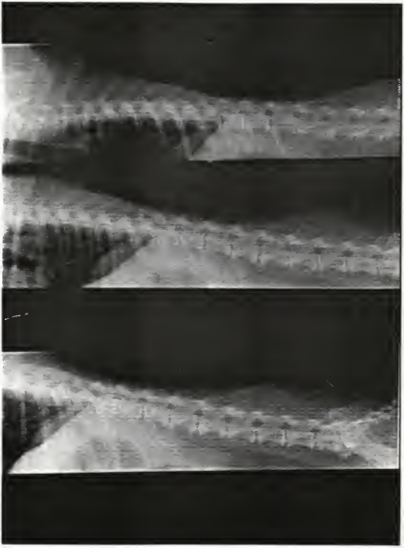


Figure 10. Dog 1, twelve weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.

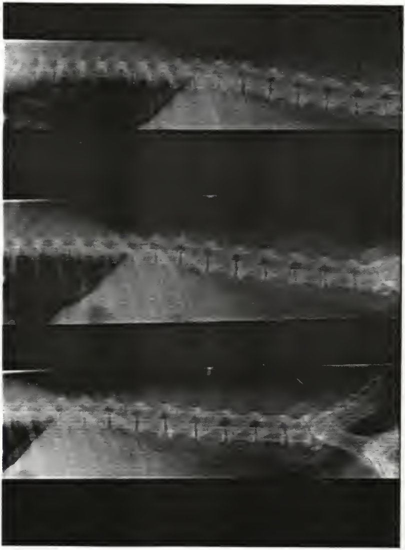


Figure 11. Dog 1, sixteen weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.

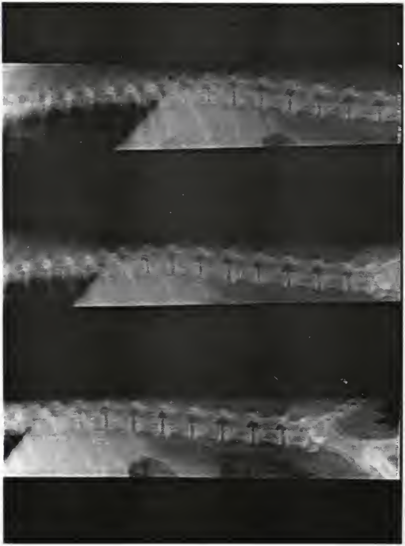


Figure 12. Dog 2, immediately following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5. Note ventral spondylosis at L7-S1.

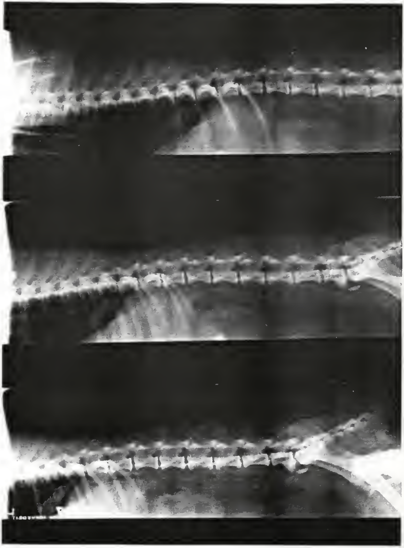


Figure 13. Dog 2, two weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

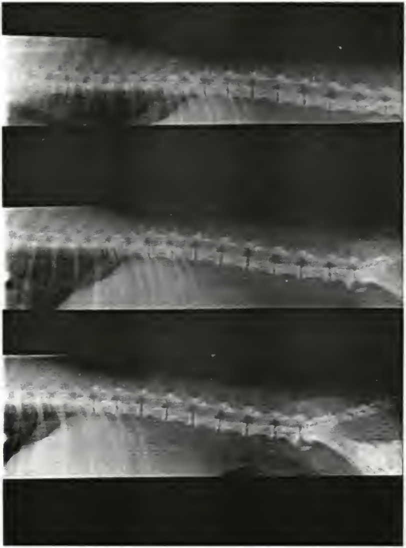


Figure 14. Dog 2, four weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

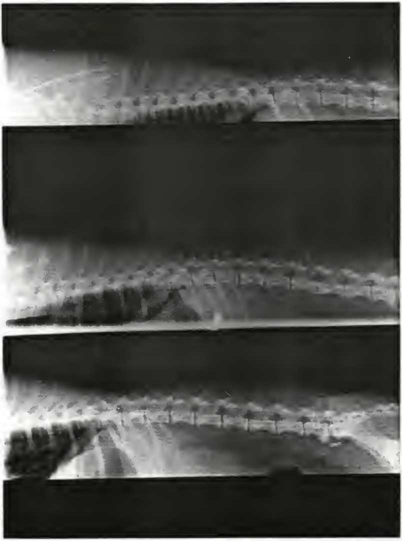


Figure 15. Dog 2, eight weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

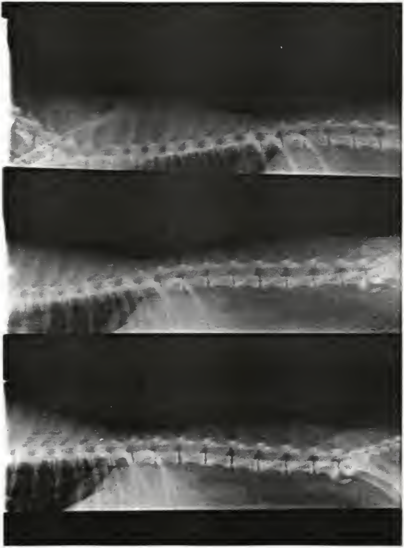


Figure 16. Dog 2, twelve weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

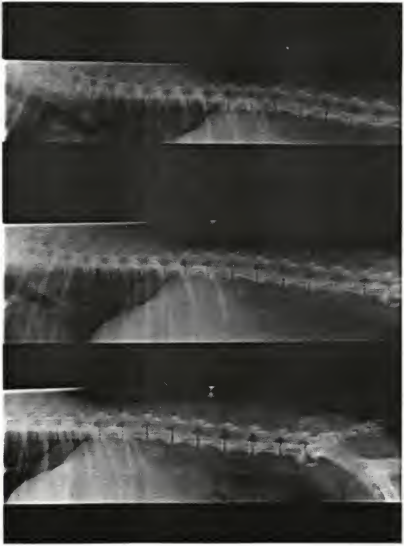


Figure 17. Dog 2, sixteen weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

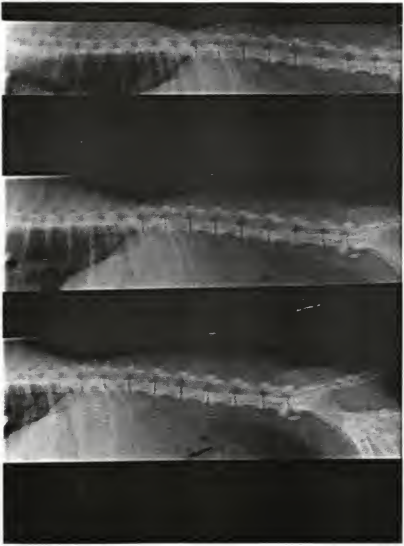


Figure 18. Dog 3, immediately following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5. T13-L1 was narrowed prior to surgery.

Figure 19. Dog 3, two weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

Figure 20. Dog 3, four weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

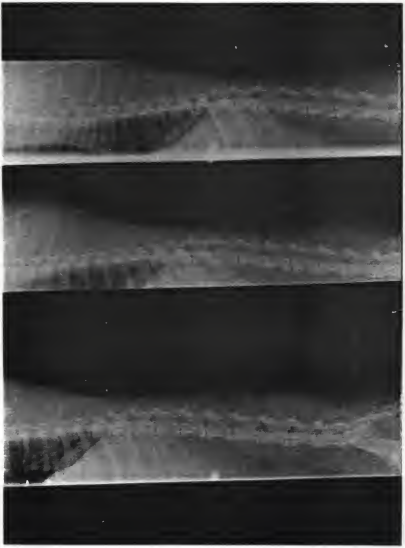


Figure 21. Dog 3, eight weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

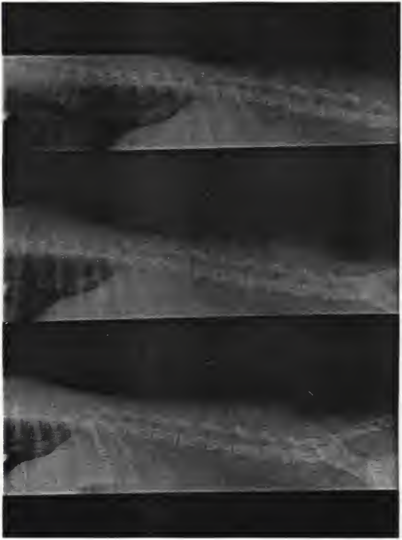


Figure 22. Dog 4, immediately following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

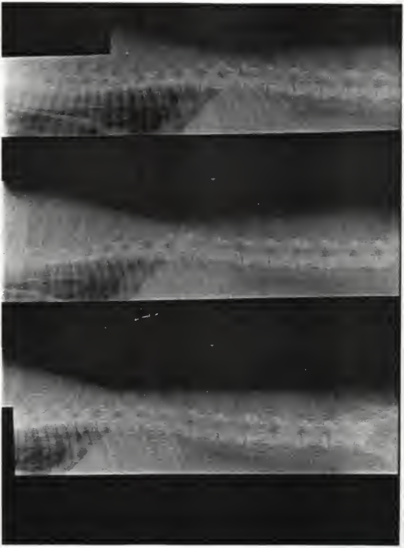


Figure 23. Dog 4, two weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

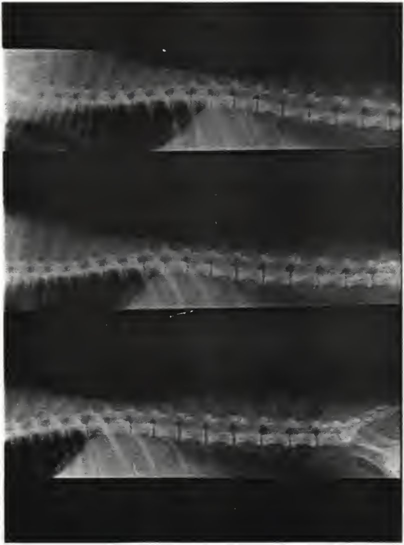


Figure 24. Dog 4, four weeks following treatment of disc spaces T11-12, T13-1, L2-3 and L4-5.

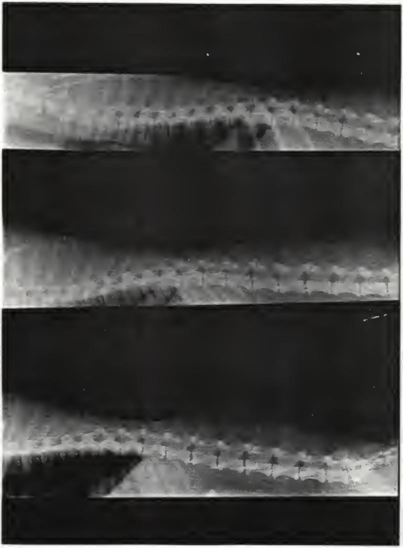


Figure 25. Dog 5, immediately following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4. Prior to surgery this animal had a narrowed T12-13 disc space.

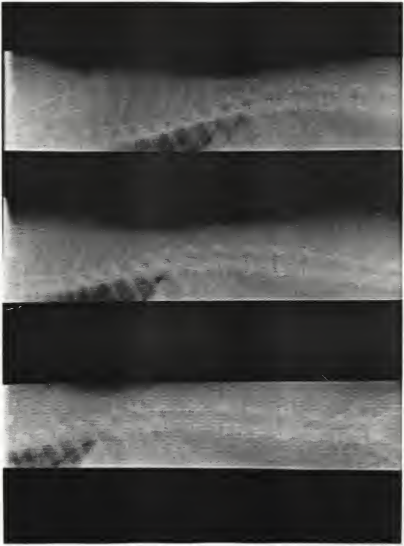


Figure 26. Dog 5, two weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.

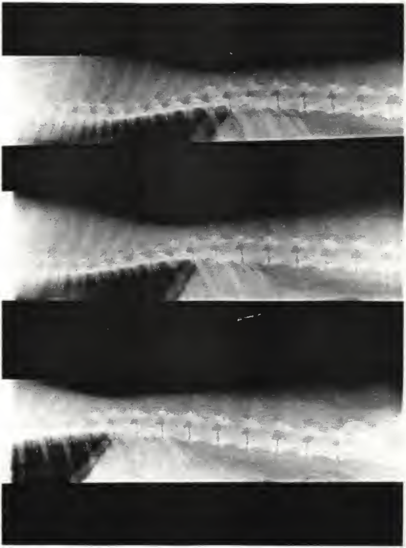


Figure 27. Dog 6, immediately following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4. Prior to surgery this animal had narrowed L2-3 and L4-5 disc spaces.

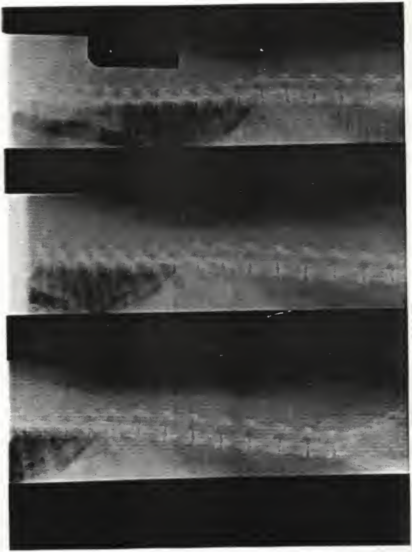


Figure 28. Dog 6, two weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.

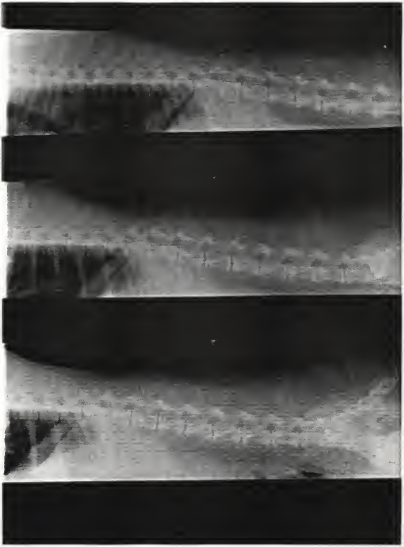


Figure 29. Dog 6, four weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.

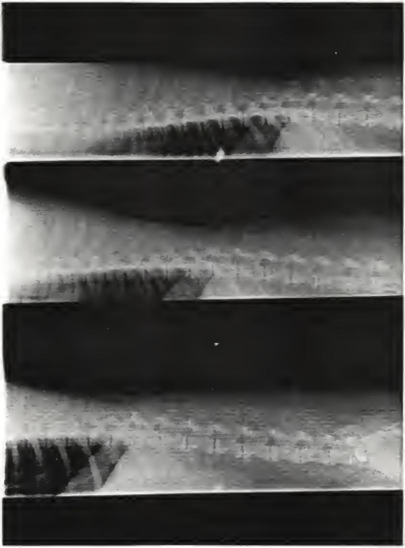
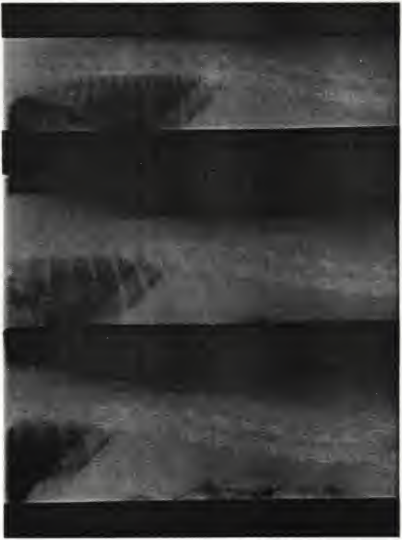


Figure 30. Dog 6, eight weeks following treatment of disc spaces T10-11, T12-13, L1-2 and L3-4.



SERIAL RADIOGRAPHIC AND HISTOLOGICAL CHANGES AS A RESULT OF A DISC CURETTAGE IN CHONDRODYSTROPHIC CANINES

by

Stanley D. Wagner, DVM

Doctor of Veterinary Medicine

Purdue University, 1974

An Abstract of a Master's Thesis

submitted in partial fulfillment of
the requirements for the degree

Master of Science

Department of Surgery and Medicine
Kansas State University
Manhattan, Kansas
1985

ABSTRACT

Six mature chondrodystrophic dogs had eight thoracolumbar disc spaces approached surgically. A dorsal lateral muscle-separating technique was used to expose the anulus fibrosus. At alternating disc spaces the nucleus pulposus was curetted with a modified dental instrument.

All six animals survived the surgery, all wounds healed without complication and no animal outwardly displayed any permanent disability. All animals were followed clinically and radiographically for 2 to 16 weeks following surgery. Four animals were also followed by electromyography. The animals were subjects of postmortem examination of both the spinal cords and intervertebral disc spaces. One animal underwent wallerian degeneration of the spinal cord as a result of the surgical procedure, this change was not detected antemortem. Two animals demonstrated fibrillation potentials along their incision sites.

All animals demonstrated radiographically, gross and histologic alterations at the operated disc spaces. Collapse of the disc space was the sole radiographic finding. Visual estimation of disc space collapse from the radiographs was better than actual metric measurement. When two different measurement systems were employed spaces T12-13, L1-2 and L3-4 did not show significant collapse of the disc space even though collapse could be subjectively visualized.

Gross examination of sagittal disc spaces revealed no nuclear protrusion at the operated sites. Grossly it was difficult to detect length differences between operated and unoperated sites. Histologically all

48 disc had evidence of disc degeneration. The response to discectomy appeared within two weeks of surgery and was mainly a chondrocytic response. At eight to sixteen weeks mature collagen had replaced the surgically created deficit.