THE PREVALENCE OF SUBCLINICAL GASTRODUODENAL ULCERATION IN DACHSHUNDS WITH INTERVERTEBRAL DISC PROLAPSE

By

SARA-ANNE MARGARET DOWDLE

Submitted in partial fulfilment of the requirements for the degree

M.Med.Vet. (Surg)

FACULTY OF VETERINARY SCIENCE

UNIVERSITY OF PRETORIA

ONDERSTEPOORT

October, 2003

THE PREVALENCE OF SUBCLINICAL GASTRODUODENAL ULCERATION IN DACHSHUNDS WITH INTERVERTEBRAL DISC PROLAPSE

BY

SARA-ANNE MARGARET DOWDLE

ABSTRACT

Endoscopy was used to determine the prevalence of subclinical gastroduodenal ulceration in 30 Dachshunds undergoing decompressive surgery for acute intervertebral disc prolapse. The endoscopy was performed on the day of admission and on the third or fourth day after surgery. Three regions of the stomach (cardia, corpus and pyloris) and the proximal duodenum were visually inspected and biopsy samples were sent for histopathology. The combination of visual and microscopic changes present, were then used to determine the prevalence of gastroduodenal ulceration in this population. An overall prevalence of 76% was calculated from these findings. Ulcerogenic medication administered prior to admission, did not appear to influence the prevalence. The results show that veterinarians need to be aware of gastroduodenal ulceration and that the use of prophylactic anti-ulcer medication in spinal surgery patients is warranted.

Director of studies: Dr K. E. Joubert

TABLE OF CONTENTS

SUMMARY

THE PREVALENCE OF GASTRODUODENAL ULCERATION IN DACHSHUNDS WITH INTERVERTEBRAL DISC PROLAPSE

by

SARA-ANNE MARGARET DOWDLE

PROMOTOR: Dr K.E. Joubert, Department of Small Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria

CO-PROMOTOR: Dr R.G. Lobetti, Department of Small Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria
Current address: Bryanston Veterinary Hospital, P.O. Box 67092, Bryanston 2021, South Africa.

DEPARTMENT: Department of Small Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort 0110, South Africa

DEGREE: M.Med.Vet. (Surg)

The primary objective of this study was to determine the prevalence of subclinical gastroduodenal ulceration (GDU) in a population of Dachshunds undergoing decompressive surgery for acute intervertebral disc prolapse (IVDP). Secondary objectives included determining the extent to which treatment with ulcerogenic drugs, prior to admission, and the severity of spinal injury, would influence the prevalence of GDU. It aimed to further establish patient risk profile components (for example: age, weight, sex or concurrent disease conditions) that might influence the prevalence of GDU and make suggestions as to whether all dogs presenting with acute IVDP should be treated with anti-ulcerogenic drugs on admission for decompressive surgery.

Thirty Dachshunds were admitted into the study after a diagnosis of acute IVDP was confirmed based on clinical signs and lumbar myelography. Full histories were obtained from both the owners and the referring veterinarians. Information obtained included any previous history of spinal disease, duration of clinical signs, rate of deterioration of clinical signs and drugs administered prior to admission. A full clinical and neurological examination was performed on each patient at admission. Blood and serum chemistry samples were taken to rule out any concurrent diseases that may have contributed to the development of GDU.

Three regions in the stomach (cardia, corpus and pyloris) as well as the proximal duodenum were visualised using gastroduodenoscopy. The following criteria were assessed: colour and appearance of mucosa, presence and distribution of erosion, ulceration and/or submucosal haemorrhages. These results were combined with those obtained from histopathology of pinch biopsy samples, taken from the same four regions, in order to obtain the overall prevalence. Criteria used to assess histopathology of the

regions included: disruption of the epithelial lining, haemorrhage into the submucosa and infiltration of neutrophils or macrophages into the submucosa. All dogs were scoped and biopsy samples taken on the first day of admission and three to four days post-surgery.

Seventy six percent of Dachshunds that presented with IVDP had visual and/or histopathologic evidence of GDU. The highest overall prevalence of GDU was shown to occur in the pyloric region of the stomach both pre- and post-surgery. No significant correlation was found between the prevalence of GDU and the administration ulcerogenic drugs prior to admission. Similarly, no correlation was found between GDU and the severity of neurological signs, the duration of clinical signs prior to admission, the length of the procedure (general anaesthesia or surgery), age weight or sex. No significant statistical difference was found between the pre- and post-surgery results.

Veterinarians should be aware of this potentially serious complication when dealing with Dachshunds with acute IVDP. The judicious use of ulcerogenic drugs and early use of prophylactic anti-ulcer medication is recommended in all patients presenting with this condition.

ACKNOWLEDGEMENTS

I gratefully acknowledge the help of the following people and institutions, without whose support and assistance this project would not have been possible:

From the **Department of Companion Animal Clinical Studies**, Faculty of Veterinary Science, University of Pretoria:

Dr K.E. Joubert for his advice, guidance and patience and for his services as my promoter

Prof R.G. Lobetti for his expert help with the protocol preparation and with all endoscopic procedures

Prof N. E. Lambrechts for his guidance and support as my mentor, through all stages of this project and the masters degree for which it is intended

Dr P.H. Turner for affording me the facilities and equipment to conduct this work and for his inspiration and guidance throughout the project

Hospital staff of the **Onderstepoort Veterinary Academic Hospital** for their invaluable help in collecting in the data required fro this project

Sr H. Mentz for her endless help and expertise with the surgical procedures performed on all patients in this study

DR A. D. Pardini (Equine Research Centre, Faculty of Veterinary Science, University of Pretoria) for histopathology done on all biopsy samples taken

From the **Section of Clinical Pathology:** Thanks to **Mrs E. Myburg** for her help with all blood testing and serum chemistry work

From the **Section of Diagnostic Imaging:** Thanks to **Prof R. Kirberger** and his staff for their help with the myelogram procedures that were performed on each dog

From the biostatistics unit of the **Medical Research Council**:

Dr P. Becker for his work with the processing and analysis of all data

Dr **E.A. Buffa** for affording me the time off to complete the writing up of this dissertation

The **South African Veterinary Foundation** for their generous contribution to the funding of the project

The **University of Pretoria** for the financial support and outstanding facilities, which helped to make this project possible

CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

Intervertebral Disc Prolapse (IVDP) is the most common neurological condition seen in dogs, particularly in the chondrodystrophic breeds (eg. Dachshund, Pekingese, Beagle)[3,11,13,14,16,25,27]. It is a degenerative condition of the intervertebral disc, which is characterised by the dessication of the nucleus pulposus and, in some cases, the herniation of disc material into the vertebral canal[4]. In the chondrodystrophic breeds, this process is known as chondroid metaplasia. The Dachshund reportedly has a 10- to 12-fold greater risk of IVDP than all other breeds' combined[13].

Presenting clinical signs usually have an acute onset and occur between two and six years of age. They include spinal hyperaesthesia, ataxia, paresis and paralysis. In severe cases, loss of deep pain sensation to the pelvic limbs may occur[13]. The degeneration and prolapse of intervertebral disks is more common in the region of the thoracolumbar junction with 70% of all clinical cases occurring between the twelfth thoracic and second lumbar vertebra[4]. The resultant pain originates from either dessication of the nucleus pulposus, degenerative pathology of the annulus fibrosis, inflammatory changes in the dorsal longitudinal ligament and/or as a result of entrapment and neurovascular ischaemia of the spinal nerve root[21].

The correlation between central nervous system injuries and GDU, as well as the tendency to misdiagnose the latter complication, has been recognised for many years[15,17,19,20,22,23,25,26]. Hoerlein reported various non-neurological complications following decompressive spinal cord surgery in dogs[11]. He made special note of the

occurrence of gastric, duodenal and colonic ulceration in these patients. Toombs *et al.* reported the presence of colonic perforation following neurosurgical procedures with concurrent corticosteroid treatment. They concluded that the antemortem diagnosis of perforation was difficult and that a prophylactic approach to these complications was warranted in high-risk patients[25]. In a retrospective study based on clinical signs, Moore showed that the prevalence of gastrointestinal complications (pancreatitis, gastrointestinal haemorrhage, ulceration and perforation) was as high as 15 %, in dogs with intervertebral disc prolapse. He suggested that approximately 2 % would die from these complications[19]. Neiger *et al*. published the results of an endoscopic examination, which showed that the incidence of GDU in dogs with acute IVDP and concurrent corticosteroid use, was greater than 75 %[20].

Gastric erosions are defined as superficial mucosal defects that do not penetrate the *lamina muscularis mucosae*, while gastric ulcers penetrate into this layer and may even perforate it. If mucosal damage exceeds the reparative process, then erosions can progress to ulcers[9]. Early lesions manifest as mucosal hyperaemia and submucosal haemorrhages, reflecting the underlying inflammatory process.

Most authors agree that the aetiology of gastrointestinal ulceration is multifactorial and is usually secondary to systemic disease, trauma, stress, hypovolaemia or administration of ulcerogenic drugs[5,19,20,25,28]. In addition, it is postulated that in IVDP cases, autonomic dysfunction caused by the spinal cord compression may lead to hypersecretion of gastric acid and pepsin with resultant mucosal ulceration [5,15,22].

Ulcerogenic drugs like corticosteroids and non-steroidal anti-inflammatory drugs (NSAID) are commonly used in patients with spinal injury as they are beneficial in reducing local oedema, providing analgesia and limiting the "autoinflammatory" cycle that may lead to progressive spinal cord injury, including ascending haemorrhagic necrosis[27].

Endogenous prostaglandins are important in maintaining gastrointestinal mucosal integrity by enhancing the secretion of bicarbonate and mucus, reducing acid production and possibly increasing mucosal blood supply. Most NSAID and corticosteroids inhibit prostaglandin formation and thus increase the likelihood of GDU by affecting prostaglandin-mediated defence mechanisms of the bowel wall[2,5,17,18,24,25,26,28].

In previous studies, clinical signs such melaena, intermittent vomition, abdominal pain and decreased haematocrit, were used as indicators of the presence of ulceration and haemorrhage[17,25,26,28]. The rapid onset of gastrointestinal complications, together with the fact that very few clinical signs may be present, mean that this condition is often recognised too late for appropriate treatment protocols to be implemented effectively[1,7]. In the past, an accurate antemortal diagnosis of gastro-intestinal complications was only made in 25 % of cases[6,25].

Endoscopic examination provides a safe, non-invasive and accurate technique for the diagnosis of gastric mucosal lesions[8,10,12,22]. In addition, it permits evaluation of the colour and integrity of the gastric mucosa, without the increased risk of dehiscence or peritonitis associated with a celiotomy[8]. Biopsy samples of the gastric mucosa are

easily performed using the biopsy channel of the endoscope and are useful in diagnosing the clinical and subclinical stages of GDU. In a study based on 42 dogs, gastroscopy was reported to be superior to gastrography in the detection of gastritis or gastric ulceration, however the final diagnosis was dependant on the correct histopathological interpretation of the biopsy samples[12].

A review of the literature revealed the following deficiencies regarding GDU in IVDP cases:

1. The prevalence of GDU in Dachshunds with acute IVDP that did not receive daily ulcerogenic drugs, during the hospitalisation period, has not been recorded.
2. Risk profiles for dogs with acute IVDP that may be more susceptible to GDU have not been formulated.
3. Successful strategies for decreasing the incidence of GDU in hospitalised Dachshunds with acute IVDP have not been identified.
4. The prevalence of GDU in dogs with neurological disease that have not received any form of ulcerogenic drug treatment has not been reported.
5. Endoscopy has only been used before in one study to determine the prevalence of GDU in cases presenting with acute IVDP[20].

The aim of this study was to determine the prevalence of GDU in dogs with acute IVDP and to increase our knowledge of patient risk profiles that would help clinicians decide whether or not prophylactic anti-ulcerogenic treatment should be given to all dogs or selected dogs with acute IVDP.

PUBLISHED RESEARCH ARTICLE

The prevalence of subclinical gastroduodenal ulceration in Dachshunds with intervertebral disc prolapse

S M Dowdle[a*], K E Joubert[a], N E Lambrechts[a], R G Lobetti[a**], A D Pardini[b]

ABSTRACT

Endoscopy was used to determine the prevalence of subclinical gastroduodenal ulceration in 30 Dachshunds undergoing decompressive surgery for acute intervertebral disc prolapse. The endoscopy was performed on the day of admission and on the third or fourth day after surgery. Three regions of the stomach (cardia, corpus and pylorus) and the proximal duodenum were visually inspected and biopsy samples were sent for histopathology. The combination of visual and microscopic changes present, were then used to determine the prevalence of subclinical gastroduodenal ulceration in this population. An overall prevalence of 76% was calculated from these findings. Ulcerogenic medication administered prior to admission, did not appear to influence the prevalence. This result identifies a need for veterinarians to be aware of this potentially severe complication and warrants the use of prophylactic anti-ulcer medication in spinal surgery patients.

Key words: gastric ulcer, spinal cord, intervertebral disc disease, canine, dog, endoscopy.

Dowdle S M, Joubert K E, Lambrechts N E, Lobetti R E, Pardini A D **The prevalence of subclinical gastroduodenal ulceration in Dachshunds with intervertebral disc prolapse.** *Journal of the South African Veterinary Association (2003).* Department of Companion Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

[a] Department of Companion Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.
*Author for correspondence.
Current Address: Ridgemall Veterinary Hospital, P.O. Box 1311, Randpark Ridge, 2156 South Africa.
E-mail: sdowdle@mweb.co.za
** Current address: Bryanston Veterinary Hospital, P.O. Box 67092, Bryanston, 2021 South Africa.
[b] Equine Research Centre, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

INTRODUCTION

Intervertebral disc prolapse (IVDP) is the most common neurological condition seen in the dog, particularly in the chondrodystrophic breeds[3,11,13,14,16,21,25,27]. In human medicine, the correlation between spinal cord injury and gastroduodenal ulceration (GDU), as well as the tendency to misdiagnose the latter, has been recognised for many years[15,23]. Gastric erosions are defined as superficial mucosal defects that do not penetrate the lamina muscularis mucosae, while gastric ulcers penetrate into this layer and may even perforate it. If mucosal damage exceeds the reparative process, then erosions can progress to ulcers[9]. Less severe lesions that occur in the early

development of an ulcer manifest as inflammation of the mucosa causing hyperaemia and submucosal haemorrhages.

Gastroduodenal ulceration is reported to frequently accompany IVDP in the dog[4,7,15,19,25,26], however, until recently, the actual prevalence and severity of this complication has not been determined[20]. A 2% mortality rate has been proposed in cases of IVDP associated with gastrointestinal complications[19].

Most authors concur that the aetiology of GDU, in general, is multifactorial. It is usually secondary to systemic disease, trauma, stress, hypovolaemia or administration of ulcerogenic drugs[5,19,20,25,28]. In IVDP cases, it is postulated that autonomic dysfunction caused by the spinal cord compression leads to hypersecretion of gastric acid and pepsin with resultant GDU [5,15].

Ulcerogenic drugs such as corticosteroids and non-steroidal anti-inflammatory drugs (NSAID) are commonly used in patients with spinal injury and have been shown to be beneficial in reducing local oedema, providing analgesia and limiting the "autoinflammatory" cycle that may lead to the progression of ascending haemorrhagic necrosis of the spinal cord[27]. However, corticosteroid therapy predisposes the patient to GDU and perforation by inhibiting the action of prostaglandins, which are important in maintaining gastroduodenal mucosal integrity by enhancing secretions of bicarbonate and mucus[2,5,6,17,20,24]. Similarly, NSAID increase the likelihood of gastroduodenal haemorrhage and ulceration by affecting prostaglandin-mediated defence mechanisms of the bowel wall[2,5,17,18,24,25,26,28].

In the past, an accurate antemortal diagnosis of gastrointestinal complications was only made in one out of every four cases presented[6,25]. Clinical signs such as melaena, intermittent vomition, abdominal pain and decreased haematocrit, were used as indicators of the presence of gastric ulceration and haemorrhage[17,25,26,28]. The rapid onset of gastrointestinal complications, together with the fact that very few clinical signs may be present, mean that this condition is often recognised too late for appropriate treatment protocols to be implemented effectively[1,7]. For this reason, a prophylactic approach to gastroduodenal complications in high-risk patients, such as those presenting with spinal cord injury, has been recommended[5,25].

Endoscopic examination provides a safe, non-invasive and sensitive technique for the early diagnosis of gastric mucosal lesions (submucosal haemorrhage and hyperaemia) as well as obvious ulcerations[8,10,12]. In addition, it facilitates visualisation of the colour and integrity of multiple sites of the gastric mucosa, without the risk of dehiscence or peritonitis associated with surgical exploration[8]. It has also been shown to be more accurate for detecting subclinical GDU than diagnostic imaging modalities like ultrasonography or contrast gastrography[12].

The purpose of this study was to determine the prevalence of subclinical GDU in patients with IVDP, before and after undergoing decompressive spinal surgery, in order to formulate a risk profile for these animals.

MATERIALS AND METHODS

The Animal Use and Care Committee of the Faculty of Veterinary Science, University of Pretoria, has approved the research reported in this project.

Thirty Dachshunds admitted to the Onderstepoort Veterinary Academic Hospital (OVAH) with clinical signs suggestive of acute thoracolumbar or lumbar IVDP, were assigned to this study after myelographic confirmation. This sample size was chosen using statistical analysis (SPSS, SPSS Inc.) based on a sample population of 3000 Dachshunds with a 90 % confidence interval and an estimated expected prevalence of 30 % and a 10 % accuracy range.

Dogs of any age, sex or weight were accepted into the trial. A full clinical and drug history was obtained. The duration of clinical signs before admission, treatments administered prior to admission and any previous history of IVDP were recorded. Clinical examination and serum chemistry (urea, creatinine, alkaline phosphatase (ALP), alkaline transaminase, total serum protein, albumin and globulin levels) were performed to rule out any concurrent diseases that may have predisposed the dogs to GDU. A full neurological examination including assessment of ambulation, flexor and extensor reflexes, deep pain sensation, tail response to vocal stimulation and proprioception was performed to assess the severity and location of the spinal cord injury.

Following premedication with diazepam (0.2 mg/kg) (Valium, Roche Pharmaceutical, Isando) and morphine (0.4 mg/kg) (Morphine Sulphate, Micro HealthCare,

Bethlehem), anaesthesia was induced with propofol (6 mg/kg) (Diprivan, Astra-Zeneca, Sandton). Intravenous Ringer's lactate (Sabax, Adcock Ingram, Johannesburg) was administered throughout the procedures to maintain blood pressure. Anaesthesia was maintained with halothane (Fluothane, Astra-Zeneca, Sandton) delivered in oxygen through an out-of-circuit, precision vaporiser and a non-rebreathing anaesthetic circuit. The fresh gas flow was maintained at twice the minute volume. Heart rate, respiratory rate and pulse oximetry (Satellite plus, Datex, Helsinki) were monitored continuously throughout the procedure. A lumbar myelogram was performed within the first 6 hours of admission to the hospital, in order to confirm and locate the compressive spinal cord lesion[14].

Gastroduodenoscopy was performed immediately after the myelogram, but prior to surgery. A 1m long, flexible endoscope (Olympus GIF XQ200) was used with a 2.8mm biopsy channel and a mobile tip. In each case, the cardia, corpus, pylorus and proximal duodenum were evaluated, using the following criteria: colour and appearance of mucosa, presence and distribution of submucosal haemorrhages (erosions) and/or obvious ulceration. Three to four random, pinch biopsies were taken through the biopsy channel from each of these anatomical locations and placed in 10% buffered formalin. Samples were dehydrated, embedded in paraffin, sectioned and stained with haemotoxylin and eosin (H&E) for light microscopy.

The following histopathological changes were sought: infiltration of neutrophils and macrophages (indicating inflammation), disruption of the mucosal lining and haemorrhage into the submucosa (indicating erosions) and obvious ulcerations (lesions disrupting the mucosa and penetrating the muscularis layer). Patients with

evidence of erosions and/or obvious ulceration were then designated as having (positive) evidence of ulceration, whilst those without were considered (negative). Results from gastroduodenoscopy and histopathology were combined to give an overall prevalence of GDU for this population. All gastroduodenoscopy procedures were videotaped and photographs taken of areas of interest.

Following gastroduodenoscopy, corticosteroids (methylprednisolone (30 mg/kg), Solu-cortef, Upjohn Pharmacia, Isando) and antibiotics (amoxicillin (20 mg/kg), Amocillin, Caps Pharmaceuticals, Isando) were administered intravenously. Decompression of the spinal cord was then performed following the technique described by Lubbe[16]. Post-operative amoxicillin (20mg/kg) and morphine (0.2mg/kg) were administered at 4 and 12 hours post surgery.

All patients remained hospitalised until the follow-up gastroduodenoscopy. This was repeated on the third or fourth day after surgery, following the induction of anaesthesia with propofol. The same four areas of interest that were stated earlier were examined and biopsied.

Data were divided into two groups, positive or negative, based on the above criteria. The student T-test and the Mann-Whitney Rank Sum test were used to determine differences between the two groups. These differences included severity of spinal cord injury, duration of injury prior to admission, treatment with ulcerogenic drugs, length of general anaesthesia, age, weight and sex. For correlation between variables and the prevalence of gastric ulceration, the Fisher's exact test was used. McNemar's test was used for non-parametric data. P was set at ≤ 0.05. The data were statistically

analysed using a computer software program (Sigma Stat V4, Jandel Scientific, Milwaukee).

RESULTS

No dogs were excluded from this study, but one dog did not receive a post-operative endoscopic examination as it presented an undue anaesthetic risk. Thirteen animals were male (3 neutered) and 17 female (12 neutered). Median age and weight were recorded as 5.6 ± 1.9 years and 6.8 ± 1.7 kg, respectively. Six dogs had suffered a previous incidence of IVDP. Twenty-one dogs had received treatment prior to referral with corticosteroid and/or a NSAID (See table 1). The median duration of clinical signs prior to admission was 11.4 ± 18.9 hours. No dogs were showing clinical signs of GDU at time of admission. The median duration of anaesthesia from induction for the myelogram until the completion of surgery was 160.8 ± 31.2 minutes, while surgery lasted 78.5 ± 20.5 minutes.

Drug or Drug Combination	Number of patients (n=18)
Prednisolone	6
Dexamethazone	3
Dexamethasone - phenylbutazone	2
Flunixin meglumine	1
Flunixin meglumine-prednisolone	1
Phenylbutazone	1
Ketoprofen	1
Meloxicam	1
Methylprednisolone	1
Prednisolone-dexamethazone	1

Table 1: Potentially ulcerogenic drugs administered to patients prior to admission.

Neurological deficits based on criteria recommended by Lubbe[16], were present in 21 dogs. The other 9 dogs presented with thoracolumbar or lumbar spinal pain only. Four

of the animals were considered to have severe neurological deficits, that is, limited or no deep pain sensation was present in either pelvic limb. A summary of the anatomical location of IVDP lesions is given in **Error! Reference source not found.** 2. These results concur with those locations indicated by previous studies[16]. The results of the gastroduodenoscopy and histopathology examinations are presented in **Error! Reference source not found.** 3.

Disc Space	Number (n = 30)	Prevalence (%)
T11 – T12	2	6.67
T12 – T 13	8	26.67
T13 – L1	10	33.33
L1 – L2	5	16.67
L2 – L3	2	6.67
L3 – L4	2	6.67
L4 – L5	0	0
L5 – L6	1	3.33

Table 2: Distribution of intervertebral disc prolapse lesions.

Endoscopic lesions identified ranged from mucosal hyperaemia (Fig. 1 and 4), submucosal haemorrhage (Fig. 1 and 2) and obvious ulceration (Fig.3). The incidence of these lesions is presented in Table 4.

Sampling stage	Cardiac		Corpus		Pylorus		Duodenum	
	(n)	(%)	(n)	(%)	(n)	(%)	(n)	(%)
Pre-Surgery (n = 30)								
Gastroduodenoscopy	6	20	5	16.7	15	50	1	3.3
Histopathology	7	23.3	7	23.3	15	50	6	20
Post-Surgery (n = 29)								
Gastroduodenoscopy	4	13.8	5	17.3	14	48.2	1	3.3
Histopathology	7	24.1	10	34.5	12	41.4	8	27.6
Overall Incidence of Ulceration								
Pre-Surgery	11	36.7	10	33.3	21	70	6	20
Post-Surgery	8	27.6	12	41.4	17	58.6	8	27.6

Table 3: The prevalence of ulcerations at various locations of the gastrointestinal tract. This table provides the number and percentage of patients with evidence of GDU at the various anatomical sites assessed within the gastrointestinal tract by either gastroduodenoscopy or histopathology. The overall incidence was calculated from pre- and post-surgical results, when a positive result was found with either one of the methods used to assess GDU.

Parameter	**Number**	**%**
Pre-Surgery (n = 30)		
Obvious ulceration	1	3.3
Submucosal haemorrhage	12	40
Hyperaemia	5	16.7
Normal	12	40
Total	**30**	**100**
Post-Surgery (n = 29)		
Obvious ulceration	3	10.3
Submucosal haemorrhage	9	31.0
Hyperaemia	5	17.2
Normal	12	41.5
Total	**29**	**100**

Table 4: The incidence of specific endoscopic lesions identified in both pre- and post-surgery procedures. This table provides the number and percentage of patients with specific endoscopic findings related to gastroduodenal ulceration.

No correlation was found between the prevalence of GDU and the administration of ulcerogenic drugs prior to the admission. Similarly, no correlation was found between GDU and the severity of neurological signs, the duration of signs prior to admission, the length of the procedure (general anaesthetic or surgery), age, weight or sex. An overall prevalence of 76% of dogs that were positive for GDU was calculated.

DISCUSSION

In 1975, Hoerlein[11] reported various non-neurological complications following decompressive spinal cord surgery in dogs, especially the occurrence of gastric, duodenal and colonic ulceration. Toombs[25,26] reported the presence of colonic perforation following neurosurgical procedures with concurrent corticosteroid treatment. They concluded that antemortal diagnosis in these cases was difficult and that a prophylactic approach to these complications was warranted. Moore[19] showed in a retrospective study based on clinical signs, that the prevalence of gastrointestinal complications (pancreatitis, gastrointestinal haemorrhage, ulceration and perforation) was as high as 15 % in dogs with IVDP, with a mortality rate of approximately 2 %.

In the current study, the overall prevalence of subclinical GDU was calculated at 76% by using results indicating either endoscopic or histopathological evidence of ulceration at any one of the four above-mentioned regions of the gastrointestinal tract. Histopathology identified mucosal changes and ulcerations that were not visualised during gastroduodenoscopy, thus increasing the sensitivity of detecting mucosal changes.

The highest overall prevalence of ulceration was shown to occur in the pyloric region of the stomach both before and after surgery. Histopathology results of the proximal duodenum revealed that in this region, endoscopic visualisation was much less sensitive for detecting mucosal pathology than histopathology. The apparent lower prevalence of GDU seen in the post-surgery groups was not shown to be statistically

significant. In cases where ulcers were visualised, but histopathology was negative, it is explained by the fact that biopsy samples were taken randomly from the four identified regions, thus visualised mucosal changes were not necessarily biopsied.

In a similar study, Neiger[20] used endoscopic examination to determine the incidence of GDU in dogs with acute intervertebral disc disease and concurrent corticosteroid use. All dogs enrolled in this study received dexamethasone (2mg/kg IV) on day one of admission followed by prednisilone (1mg/kg PO) daily for the duration of hospitalization. Histopathology was not performed and their results indicated an overall prevalence of 76% of dogs positive for GDU. There was no significant decrease seen in the incidence of GDU with concurrent anti-ulcer treatment. Their conclusion was that either anti-ulcer medication was instituted too late to prevent mucosal lesions from forming or that the drugs and dosages used were not effective in acute intervertebral disc disease cases.

The prevalence of GDU in the current study was similar to that described by Neiger[20], despite the use of histopathology. This could imply that endoscopic visualisation alone is a sufficiently sensitive diagnostic modality. However, it could also imply that the incidence in Neiger's study would have been higher if a histopathological examination had been included.

No correlation was found, in this study, between the use of ulcerogenic drugs and the prevalence of ulceration or between severity and duration of spinal cord compression with GDU. This may be an indication of insufficient sample size. More importantly, it indicates that ulcerogenic drugs probably play a lesser part in the pathophysiology of

GDU in dogs with IVDP and autonomic dysfunction caused by compression of the spinal cord, could play a more dominant role than has previously been reported.

The prevalence of gastroduodenal haemorrhage and ulceration in dogs with neurological disease that have not received any form of ulcerogenic drug treatment has not yet been reported[22]. In this study, 11 dogs had not received any form of ulcerogenic drug prior to admission. Of these, 6 presented with submucosal haemorrhage and/or obvious ulceration. Of the 19 dogs that received ulcerogenic drugs, 13 showed visual signs of submucosal haemorrhages and/or obvious ulceration. This supports the proposal that GDU in IVDP cases is not primarily related to ulcerogenic medication.

Future research should be directed at strategies to reduce the incidence of gastric ulcers in dogs with IVDP. This may involve judicious use of ulcerogenic drugs in the pre-surgical period and earlier surgical intervention in these cases. The early use of prophylactic anti-ulcer medication is recommended in all patients presenting with IVDP.

ACKNOWLEDGEMENTS

Funding received from the Pet Memorial Fund of the South African Veterinary Foundation and the department of Companion Animal Clinical Studies is gratefully acknowledged. Dr Piet Becker, the statistician from the Biostatistics unit of the Medical Research Council, is thanked for his contributions. Our gratitude is also

extended to the staff of the Onderstepoort Veterinary Academic Hospital for their help and support in collecting the data required for the completion of this project.

REFERENCES

1. Bellah J R 2000 Colonic perforation after corticosteroid and surgical treatment of intervertebral disk disease in a dog. *Journal of the American Veterinary Medical Association* 9: 1002-1003

2. Bolton J P, Cohen M M 1979 The effect of prostaglandin E2, 15-Methyl prostaglandin E2, and metiamide on established canine gastric mucosal barrier damage. *Surgery* 333-338

3. Braund K G 1981Canine intervertebral disc disease. In Bojrab M J (ed) *Pathophysiology in Small Animal Surgery* First Ed. Lea and Febiger, Philadelphia: 739-754

4. Bray J P, Burbridge H M 1998 The canine intervertebral disk. *Journal of the American Animal Hospital Association* 34: 55-63

5. Cosenza S F 1984 Drug-induced gastroduodenal ulceration in dogs. *Modern Veterinary Practice* 12: 923-925

6. Crawford L M, Wilson R C 1982 Melaena associated with dexamethasone therapy in the dog. *Journal of Small Animal Practice* 23: 91-97

7. Davies M 1985 Pancreatitis, gastrointestinal ulceration and haemorrhage and necrotising cystitis following the surgical treatment of degenerative disc disease in a dachshund. *Veterinary Record* 116: 398-399

8. Grooters A M, Johnson S E, Sherding R G 1994 Endoscopy case of the month: Chronic vomiting and weight loss in a dog. *Veterinary Medicine* 3: 196-199

9. Hall J A 2000 Diseases of the stomach. In Etthinger S J (ed) *Textbook of Veterinary Internal Medicine* Fifth Ed. WB Saunders, Philadelphia: 1154-1184

10. Happe R P, Van der Gaag I 1983 Endoscopic examination of esophagus, stomach, and duodenum in the dog. *Journal of the American Animal Hospital Association* 19: 197-206

11. Hoerlein B F 1975 Non-neurological complications following decompressive spinal cord surgery. *Archives of the American College of Veterinary Surgeons* 4: 11-16

12. Jakovljevic S 1988 Gastric radiology and gastroscopy in the dog. *Veterinary Annual* 28: 172-182

13. Jerram R M, Dewey C W 1999 Acute thoracolumbar disk extrusion in dogs – part 1. *Compendium for Continuing Education* 21: 922-930

14. Kirberger R M, Roos C J, Lubbe A M 1982 The radiological diagnosis of thoracolumbar disk disease in the Dachshund. *Veterinary Radiology and Ultrasound* 33: 255-261

15. Lu W Y, Rhoney D H, Boling W B, Johnson J D 1997 A review of stress ulcer prophylaxis in the neurosurgical intensive care unit. *Neurosurgery* 41: 416-425

16. Lubbe A M, Kirberger R M, Verstraete F J M 1994 Pediculectomy for thoracolumbar spinal decompression in the Dachshund. *Journal of the American Animal Hospital Association* 30: 233-238

17. Matz E M 1995 Gastrointestinal ulcer therapy . In Kirk R W (ed) *Current Veterinary Therapy XII, Small Animal Practice* Twelfth Ed. WB Saunders, Philadelphia: 706-710

18. Menguy R, Masters Y F 1963 Effects of cortisone on mucoprotein secretion by the gastric antrum of dogs - pathogenesis of steroid ulcer. *Surgery* 54: 19-28

19. Moore R W, Withrow S J 1982 Gastrointestinal haemorrhage and pancreatitis associated with intervertebral disk in the dog. *Journal of the American Veterinary Medical Association* 180: 1443-1447

20. Neiger R, Gaschen F, Jaggy A 2000 Gastric mucosal lesions in dogs with acute intervertebral disc disease: Characterization and effects of omeprazole or misoprostol. *Journal of Veterinary Internal Medicine* 14: 33-36

21. Prata R G 1981 Neurosurgical treatment of thoracolumbar disks: The rationale and value of laminectomy with concomitant disk removal. *Journal of the American Animal Hospital Association* 17: 17-26

22. Rohrer C R, Hill R C, Fischer A, Fox L E, Schaer M, Ginn P E, Casanova J M, Burrows C F 1999 Gastric haemorrhage in dogs given high doses of methylprednisilone sodium succinate. *American Journal of Veterinary Research* 60: 977-981

23. Tanaka M, Motoaki U, Motoo K 1979 Gastro-duodenal disease in chronic spinal cord injuries. *Archives of Surgery* 114: 185-187

24. Taylor L A, Crawford L M 1968 Aspirin-induced gastrointestinal lesions in dogs. *Journal of the American Veterinary Medical Association* 152: 617-619

25. Toombs J P, Caywood D D, Lipowitz A J, Stevens J B 1980 Colonic perforation following neurosurgical procedures and corticosteroid therapy in four dogs. *Journal of the American Veterinary Medical Association* 177: 68-72

26. Toombs J P, Collins L G, Graves G M, Crowe D T, Caywood D D 1986 Colonic perforation in corticosteroid-treated dogs. *Journal of the American Veterinary Medical Association* 188: 145-150

27. Trotter E J 1978 Canine intervertebral disk disease. In Kirk R W (ed) *Current Veterinary Therapy* Sixth Ed. WB Saunders, Philadelphia: 841-848

28. Wallace M S, Zawie D A, Garvey M S 1990 Gastric ulceration in the dog secondary to the use of non-steroidal anti-inflammatory drugs. *Journal of the American Animal Hospital Association* 26: 467-472

LIST OF CAPTIONS TO FIGURES

FIGURE 1: Endoscopic view of the corpus of the stomach showing multifocal, mucosal hyperaemia, with more severe, submucosal haemorrhages occurring in the pylorus.

FIGURE 2: Severe, diffuse, submucosal haemorrhages in both the corpus and pyloric regions of the stomach of a dog, as seen using endoscopy on the day of admission.

FIGURE 3: Endoscopic view of the minor incisure of the corpus of the stomach showing obvious, gastric ulceration, with complete penetration of the mucosal layer.

FIGURE 4: Severe, diffuse, mucosal hyperaemia noted on endoscopy of the pyloric region, on the third day after surgery.

CHAPTER 2

OBJECTIVES

This studies primary objective was to determine the prevalence of subclinical gastroduodenal ulceration in a population of Dachshunds undergoing decompressive surgery for acute intervertebral disc prolapse. Secondary objectives were to identify potential risk factors for the development of GDU. This information would provide the following clinical benefits:

1. Determine the extent to which treatment with ulcerogenic drugs, prior to admission, influences the prevalence of GDU.

2. Determine if the degree of GDU is related to the severity of clinical signs, neurological signs or signalment (age, weight, sex or length of general anaesthetic) in Dachshunds with acute IVDP.

3. Determine if the degree of GDU is related to the duration of spinal cord injury prior to surgical decompression.

4. Determine whether there is a significant difference between the prevalence of GDU at time of admission as opposed to three to four days post-surgery.

5. Rationalise the use of ulcerogenic drugs and anti-ulcer medication in the peri-operative period and possibly influence the degree of GDU occurring in IVDP patients during hospitalisation.

CHAPTER 3

MATERIALS AND METHODS

The Animal Use and Care Committee of the Faculty of Veterinary Science approved the research protocol used in this project.

3.1 MODEL SYSTEM

This study was an observational trial using 30 Dachshunds admitted to the Onderstepoort Veterinary Academic Hospital (OVAH) with clinical signs suggestive of acute IVDP in the thoracolumbar or lumbar regions of the spinal cord. Dachshunds of any age, sex or weight were accepted into the trial. This sample size was chosen using statistical analysis (SPSS, SPSS Inc.) based on a sample population of 3000 Dachshunds with a 90 % confidence interval, an estimated expected prevalence of 30 % and a 10 % variance.

3.2 EXPERIMENTAL PROCEDURES

3.2.1 History and clinical examination

Full clinical and drug histories were obtained from each dog's owner and the referring veterinarians. Any previous medical conditions for example chronic renal failure or liver disease, which are known to cause GDU, were noted. Body weight was recorded in kilograms (kg) to one decimal place. This was followed by thorough physical and neurological examinations and collection of blood, urine and faecal samples.

3.2.2 Neurological examination

A detailed neurological examination was performed on each dog included in this trial in order to provide an indication of the severity of the spinal cord injury. The system, suggested by Lubbe *et al*[16], is currently in use in the OVAH and was used in this study for grading the severity of clinical signs related to acute IVDP.

The following parameters were evaluated and recorded according to guidelines provided by Lubbe[16].

F = Presence of a flexor (withdrawal) reflex in the pelvic limbs

D = Presence of deep pain sensation in the pelvic limbs

T = A tail response to vocal stimulus

A = Ambulatory status (see later)

P = Presence of bilateral conscious proprioception in the pelvic limbs

The ambulatory status was evaluated and recorded according to guidelines provided by Prata[21].

0. No deficits in the pelvic limbs
1. Ambulatory with pelvic limb paraparesis
2. Non-ambulatory with pelvic limb paraparesis, with voluntary movement in the pelvic limbs still present
3. Non-ambulatory with paraparesis, without voluntary movement in the pelvic limbs
4. Complete pelvic limb paraplegia

Patients in category 4, that had bilateral loss of deep pain sensation, were admitted into the trial if the owners elected to continue with decompressive surgery, despite having been given a poor prognosis. Loss of deep pain sensation and no tail response to vocal stimulus, were considered to be the most important indicators of a poor prognosis[16].

For statistical analysis, neurological status was classified according to the following scheme:

0 – No neurological deficits present

1 –Moderate neurological deficits present (no conscious proprioception, ambulation and/or tail response)

2 – Severe neurological status (no deep pain sensation present)

3.2.3 Blood and serum chemistry

Serum and EDTA blood samples were collected aseptically from each dog. Samples were taken on the day of admission and prior to the second gastroduodenoscopy procedure on the third or fourth day after surgery.

The serum sample was used to determine the following parameters:

Urea, Creatinine, Alkaline Phosphatase (ALP), Alkaline Transaminase (ALT), Total Serum Protein (TSP), Albumin (alb) and Globulin (glob) levels.

Values that fell outside of the normal ranges were used to indicate of the presence of concurrent disease conditions that could contribute to the development of GDU, so as to exclude these animals from the trial.

The EDTA blood tube was used to obtain a Complete Blood Count (CBC). The Packed Cell Volume (PCV) and Thrombocyte Count (TCC) of the patients were also recorded. These values were again used to rule out the presence of any concurrent diseases and to possibly help formulate the patient risk profiles at a later stage.

3.2.4 Urine and faecal examination

A urine sample was obtained from each dog via cystocentesis. Urine specific gravity (SG), the pH and urine protein were recorded. The purpose of these values was intended to aid in assessing the overall health of the patient and to rule out concomitant renal disease.

A 5 milligram faecal sample was collected from each dog. A faecal occult blood test was performed on this. The faeces was diluted with sufficient water to make a 1:5 dilution. The blood patch on standard urine dipstick was used and considered a positive result if the patch turned dark green immediately on being dipped into the dilution. Only strongly positive results were considered relevant. This precaution was taken to try and eliminate false positive results that occur due to the cross reaction that occurs with myoglobin from the dog's diet.

3.2.5 General anaesthesia

A standardised anaesthetic regimen was used. It was not necessarily identical for each dog, as it depended on individual health status and requirements. Following premedication with diazepam (0.2 mg/kg) (Valium, Roche Pharmaceutical, Isando) and morphine (0.4 mg/kg) (Morphine Sulphate, Micro HealthCare, Bethlehem), anaesthesia was induced with propofol (6 mg/kg) (Diprivan, Astra-Zeneca, Sandton).

Anaesthesia was maintained with halothane (Fluothane, Astra-Zeneca, Sandton) delivered in oxygen through an out-of-circuit, precision vaporiser and a non-rebreathing anaesthetic circuit. The fresh gas flow was maintained at twice the minute volume. Heart rate, respiratory rate and pulse oximetry (Satellite plus, Datex, Helsinki) were monitored continuously throughout the procedure.

All dogs were placed on intravenous Ringers Lactate (Sabax, Adcock Ingram, Aeroton, Johannesburg) at 10mg/kg/hr for the period of anaesthesia. This was done in order to maintain circulating blood volume and counteract fluid loss during the subsequent procedures.

3.2.6 Gastroduodenoscopy

A 1m long, flexible video endoscope (Olympus GIF XQ200, Japan) with a 2.8mm biopsy channel and a mobile end tip was used for all gastroduodenoscopy procedures. Each procedure took no longer than 30 minutes to perform. All efforts were made to ensure that the endoscope settings were kept constant and that photographs were printed of the areas of interest. Dachshunds were starved for at least 12 hours prior to gastroduodenoscopy. In each case, the entire mucosal lining was evaluated from images on the monitor, however particular attention was given to the following four sites: cardia, corpus, pylorus and proximal duodenum.

Each region of the stomach/duodenum was graded as follows:

0 = Normal appearance of mucosa

1 = Hyperaemia of the mucosa without submucosal haemorrhage or erosions

2 = Submucosal haemorrhage and mild erosions of the mucosal layer present

3 = Obvious ulceration present

Three to four random biopsy samples were taken at each site regardless of whether lesions were visible or not. These were placed in marked bottles containing 10% buffered formalin and submitted for histopathology. Focal, visual lesions were not biopsied.

3.2.7 Histopathology

Mucosal biopsy samples were sent to an independent histopathologist for evaluation. Samples were dehydrated, embedded in paraffin, sectioned and stained with haemotoxylin and eosin for light microscopy. The following histopathology changes were sought: infiltration of neutrophils and macrophages into the mucosal and submucosal layers (indicating the presence of inflammation), disruption of the mucosal lining and haemorrhage into the submucosa (indicating erosions) and disruption of the mucosal layer with penetration into the muscularis mucosae layer (indicating obvious ulceration).

The following grading system was applied to the results:

0 – Normal mucosa

1 – Inflammation of the mucosa

2 – Submucosal haemorrhage or erosions

3 – Obvious ulceration

3.2.8 Lumbar myelogram

This was performed according to a recognised standard protocol[14]. It followed immediately after the gastroduodenoscopy procedure and was performed by the section of Diagnostic Imaging, Department of Companion Animal Clinical Studies (CACS) at the Onderstepoort Veterinary Academic Hospital (OVAH). The myelogram was used to confirm and locate any compressive spinal cord lesions and these findings were then recorded.

3.2.9 Surgical decompression

Spinal cord decompression was performed at the level of the spinal cord that was indicated by the lumbar myelogram, using a recognised pediculectomy procedure [16]. Antibiotics administered prior to surgery consisted of amoxicillin given intravenously at a dose rate of 20mg/kg (Amocillin, Caps Pharmaceuticals, Isando). This was repeated at 4 and 12 hours post-surgery. A single intravenous injection of hydrocortisone sodium succinate (Solu-cortef, Upjohn Pharmacia, Isando) was also administered, prior to surgery, at a dose rate of 30mg/kg. Patients were admitted to the intensive care unit for recovery following surgery.

3.2.10 Repeat blood count

On the third or fourth day following surgery, a second EDTA blood tube was drawn from each patient. This was used to obtain a follow-up CBC, PCV and TCC in order to assess if any significant changes had occurred. Any abnormalities outside of accepted reference ranges were recorded.

3.2.11 Repeat gastroduodenoscopy

Gastroscopy was repeated on the third or fourth day after surgery, following the induction of anaesthesia with propofol. The same process as previously discribed, was followed and biopsy samples taken. Anaesthesia was terminated immediately after the procedure.

3.3 DATA ANALYSIS

3.3.1 Data preparation

Data were recorded onto Microsoft Excel (Microsoft, Redmund, CA) spreadsheets for computerized data analysis at a later stage. The following data was recorded for each dog:

- Age, sex, weight
- Duration of clinical signs prior to admission
- Previous history of renal or liver disease
- Previous history of IVDP
- Previous treatments (for this episode of IVDP) with ulcerogenic drugs and the dosages used
- Neurological status in both left and right pelvic limbs including: Ambulatory status, conscious proprioception, tail response to vocal stimulus, flexor reflex strength, deep pain sensation. Patients were then allocated to a neurological group as described on page 34
- Gastroduodenoscopy results from first and second procedures
- Histopathology results from biopsy samples taken at first and second procedures
- Initial serum chemistry results and blood counts from both sampling stages

- Faecal occult blood and urinanalysis results
- Total general anaesthetic time required for the first procedures (gastro-duodenoscopy and decompressive spinal surgery)
- Total surgical time required for decompressive spinal surgery

3.3.2 Data processing

All data were converted from nominal to ordinal data in order to allow statistical analysis. All patients were then designated into one of two groups based on a combination of the results from the gastroduodenoscopy and histopathology. The groups consisted of patients with evidence of ulcers (positive) and those without evidence of ulcers (negative).

3.3.3 Statistical analysis

Descriptive statistics were calculated for:

- Age, weight and sex of patients
- Duration of clinical signs prior to admission
- Previous incidents of IVDP
- Treatment with potentially ulcerogenic drugs prior to admission
- Neurological status at time of admission
- Ambulatory status
- Blood and serum chemistry results
- Urinanalysis and faecal occult blood test results
- Distribution of affected disc spaces
- General anaesthetic and surgical times
- Gastro-duodenoscopy and histopathology results

The student T-test and the Mann-Whitney Rank Sum test were used to determine statistical differences between groups (with ulcers and without ulcers) for the recorded variables. These variables were:

Severity of spinal cord injury

Duration of clinical signs prior to admission

Treatment with ulcerogenic drugs prior to admission

Length of general anaesthesia and surgery

Age, weight and sex

For correlation between variables and the prevalence of gastric ulceration, the Fisher's exact test was used. McNemar's test was used for non-parametric data. P was set at $\leq$ 0.05. The data were statistically analysed using a computer software program (Sigma Stat V4, SPSS, Chicago). These correlations included: use of ulcerogenic drugs, neurological status and duration of clinical signs, relative to the degree of GDU that was present. All calculated percentages, mean and medians were rounded off to one decimal place, while standard deviations and statistical significance were rounded off to three decimal places.

CHAPTER 4

RESULTS

Descriptive statistics were calculated for age, weight, sex, duration of clinical signs prior to admission, previous history of IVDP, previous treatments with ulcerogenic drugs, neurological status, ambulatory status, blood and serum chemistry results, urinanalysis and faecal occult blood test results, distribution of affected disc spaces, general anaesthetic and surgery times and gastro-duodenoscopy and histopathology results.

Data were divided into two groups, those without ulcers (negative) and those with ulcers (positive). The student T-test and the Mann-Whitney Rank Sum test were used to determine differences between the two groups. These differences included severity of spinal cord injury, duration of clinical signs prior to admission, treatment with ulcerogenic drugs, length of general anaesthesia, age, weight and sex.

For correlation between variables and the prevalence of gastric ulceration, the Fisher's exact test was used. McNemar's test was used for non-parametric data. P was set at $\leq$ 0.05. Details of the results and data analysis are presented below. A brief summary is provided at the end of this chapter.

4.1 PATIENT SIGNALMENT

4.1.1 Patient age and body weight

The summarised statistics of patient age and body weight are presented in Table 4.1.1. The individual patient signalment details are recorded in Appendix 1 (Pg.67).

TABLE 4.1.1

PATIENT AGE AND BODY WEIGHT

VARIABLE (n=30)	MEAN	MEDIAN	STANDARD DEVIATION	RANGE
Age (yrs)	5.6	6.5	1.882	2 - 11
Weight (kg)	6.839	6.7	1.704	4.1 - 9.3

These results for age and weight were similar to those found in other studies of IVDP in Dachshunds[16].

4.1.2 Gender

The summarised statistics of patient gender are presented in table 4.1.2. The individual patient signalment details are recorded in Appendix 1(Pg. 67).

TABLE 4.1.2

SUMMARIZED PATIENT GENDER

GENDER	FREQUENCY (n=30)	PERCENTAGE (%)
Female intact	5	16.67
Female spayed	12	40.00
Male intact	10	33.33
Male neutered	3	10.00
TOTAL	30	100.00

No sex prevalence was found to predominate in the above results. This is a similar finding to that of other studies.

4.2 CLINICAL HISTORY

4.2.1 Duration of clinical signs

The summarised statistics of duration of clinical signs are presented in table 4.2.1. The individual patient details are recorded in Appendix 2 (Pg. 68).

TABLE 4.2.1

SUMMARIZED DURATION OF CLINICAL SIGNS

VARIABLE (n=30)	MEAN	MEDIAN	STANDARD DEVIATION	RANGE
DURATION OF CLINICAL SIGNS (days)	11.383	5	18.886	0.25 - 90

It is noted that a very wide range for the duration of clinical signs is reflected by the above results. This is explained by the fact that patients affected by intervertebral disc disease may suffer from discogenic pain prior to the acute onset of neurological signs associated with an IVDP[21].

4.2.2 Previous history of intervertebral disc prolapse

The summarised statistics of previous history of clinical intervertebral disc prolapse are presented in table 4.2.2. The individual patient details are recorded in Appendix 2 (Pg. 68).

TABLE 4.2.2

PREVIOUS HISTORY OF INTERVERTEBRAL DISC PROLAPSE

PREVIOUS HISTORY	FREQUENCY (n=30)	PERCENTAGE (%)
NO	24	80.00
YES	6	20.00
TOTAL	30	100.00

These findings relied on good previous histories and owner's observational abilities, which means that accuracy is questionable. Findings, however, were still thought to give a useful indication.

4.2.3 Treatment prior to admission with potentially ulcerogenic drugs

The summarised statistics of treatments given to patients of potentially ulcerogenic drugs are presented in table 4.2.3. The individual patient details are recorded in Appendix 3 (Pg. 69).

TABLE 4.2.3

TREATMENT PRIOR TO ADMISSION WITH POTENTIALLY ULCEROGENIC DRUGS

PREVIOUS TREATMENT	FREQUENCY (n=30)	PERCENTAGE (%)
YES	18	60.0
NO	12	40.00
TOTAL	30	100.00

From these results it can be seen that the majority of patients presented for acute IVDP, had received some form of ulcerogenic drug prior to admission.

4.2.4 Potentially ulcerogenic drugs administered prior to admission

The summarised statistics of potentially ulcerogenic drugs administered to patients prior to admission are presented in table 4.2.4. The individual patient details are recorded in Appendix 3 (Pg. 69).

TABLE 4.2.4

POTENTIALLY ULCEROGENIC DRUGS ADMINISTERED TO PATIENTS PRIOR TO ADMISSION

DRUG OR DRUG COMBINATION	FREQUENCY n=18	PERCENTAGE (%)
PREDNISILONE	6	33.3
DEXAMETHASONE	3	16.7
DEXAMETHASONE-PHENYLBUTAZONE	2	11.1
FLUNIXIN MEGLUMINE	1	5.6
PREDNISILONE+FLUNIXIN MEGLUMINE	1	5.6
KETOPROFEN	1	5.6
MELOXICAM	1	5.6
METHYLPREDNISILONE	1	5.6
PREDNISILONE+DEXAMETHAZONE	1	5.6
PHENYLBUTAZONE	1	5.6

Combination drugs are indicated by a hyphen (A-B). Two drugs used simultaneously are indicated by a plus sign (A+B). From the above table it is noted that

administration of Prednisilone and Dexamethasone predominate. This finding reflects the personal preference of private practioners referring patients to OVAH.

4.3 CLINICAL SIGNS

The summary of neurological status, ambulatory status, blood and serum chemistry results, urinanalysis and occult faecal blood tests are presented in the tables 4.3.1-4.3.6. Individual patient details regarding these parameters appear in Appendix 4,7 and 8 (Pg. 70, 73 and 75).

TABLE 4.3.1

SUMMARY OF NEUROLOGICAL STATUS

NEUROLOGICAL DEFICITS	FREQUENCY (n=30)	PERCENTAGE (%)
0	9	30.00
1	18	60.00
2	3	10.00
TOTAL	30	100.00

TABLE 4.3.2

SUMMARY OF AMBULATORY STATUS ON PRESENTATION

AMBULATORY STATUS	FREQUENCY (N=30)	PERCENTAGE (%)
0	9	30.00
1	6	20.00
2	7	23.33
3	7	23.33
4	1	3.33
TOTAL	30	100.00

TABLE 4.3.3

SUMMARY OF BLOOD RESULTS

PARAMETER	MEAN	MEDIAN	STANDARD DEVIATION	STANDARD ERROR	RANGE
PCV PRE (l/l)	0.502	0.504	0.050	0.009	0.378 - 0.601
PCV POST (l/l)	0.452	0.449	0.042	0.008	0.374 - 0.518
TCC PRE ($10x10^9$/l)	408.86	386.50	93.447	17.061	230.00 - 599.00
TCC POST ($10x10^9$/l)	361.20	352.00	67.776	12.586	244.00 - 501.00
WCC PRE ($10x10^9$/l)	12.027	11.050	4.743	0.866	3.600 - 26.600
WCC POST ($10x10^9$/l)	12.506	11.400	5.586	1.037	5.600 - 29.400

PCV PRE – Packed cell volume pre-surgery

PCV POST – Packed cell volume 3-4 days post - surgery

TCC PRE – Thrombocyte count pre-surgery

TCC POST – Thrombocyte count 3-4 days post-surgery

WCC PRE – White cell count pre-surgery

WCC POST – White cell count 3-4 days post-surgery

TABLE 4.3.4

SUMMARY OF SERUM CHEMISTRY RESULTS

PARAMETER	MEAN	MEDIAN	STANDARD DEVIATION	STANDARD ERROR	RANGE
ALT (U/l)	37.433	26.500	35.556	6.492	13.000 - 147.000
ALP (U/l)	56.267	43.000	54.051	9.868	12.000 - 271.000
UREA (Mmol/l)	4.437	3.800	2.522	0.461	1.800 - 13.000
CREATININE (µmol/l)	80.833	79.000	18.229	3.328	45.000 - 135.000
Albumen (g/l)	41.070	41.250	3.946	0.720	32.600 - 49.800
Globulin (g/l)	29.167	27.100	6.987	1.276	20.600 - 54.700
Alb:Glob ratio	1.434	1.505	0.346	0.063	0.099 - 1.840

TABLE 4.3.5

SUMMARY OF URINANALYSIS RESULTS

PARAMETER (n=30)	MEAN	MEDIAN	STANDARD DEVIATION	STANDARD ERROR	RANGE
SG	995.90	1022.0	188.14	34.350	1.038 - 1048.0
PH	6.450	6.500	0.855	0.156	5.000 - 8.000
Prt (mg/dl)	54.333	30.000	93.077	16.993	0.000 - 500.00

TABLE 4.3.6

SUMMARY OF FAECAL OCCULT BLOOD TESTS

PREVIOUS TREATMENT	FREQUENCY (n=30)	PERCENTAGE (%)
YES	22	73.3
NO	8	26.7
TOTAL	30	100

4.4 DISTRIBUTION OF INTERVERTEBRAL DISC PROLAPSE

The summarised statistics of the distribution of intervertebral disc prolapse based on myelogram findings are presented in table 4.4. Details of individual patients myelogram results are presented in Appendix 2 (Pg. 68).

TABLE 4.4

SUMMARY OF DISTRIBUTION OF INTERVERTEBRAL DISC PROLAPSE

DISC SPACE AFFECTED	FREQUENCY (n=30)	PERCENTAGE (%)
t11-12	2	6.67
t12-13	8	26.67
t13-l1	10	33.33
l1-l2	5	16.67
l2-3	2	6.67
l3-4	2	6.67
l4-5	0	0
l5-6	1	3.33
TOTAL	30	100

4.5 GENERAL ANAESTHESIA AND SURGICAL TIMES

The summarised statistics of the general anaesthetic and surgical times are presented in table 4.5. Details of individual times are presented in Appendix 9 (Pg.76).

TABLE 4.5

SUMMARY OF GENERAL ANAESTHETIC AND SURGICAL TIMES

PARAMETER	MEAN	MEDIAN	STD DEVIATION	MIN	MAX
GA TIME (min)	160.8	160	31.2	100	260
SURG TIME (min)	78.5	77.5	20.5	45	125

4.6 GASTRODUODENOSCOPY

The summarised statistics of the prevalence of gastroduodenal ulceration identified by gastro-duodenoscopy procedures is presented in table 4.6. The individual patient details are recorded in Appendix 5 (Pg. 71).

TABLE 4.6

PREVALENCE OF GASTRODUODENAL ULCERATION IDENTIFIED BY GASTRODUODENOSCOPY

SAMPLING STAGE	CARDIA		CORPUS		PYLORUS		DUODENUM	
	(n)	(%)	(n)	(%)	(n)	(%)	(n)	(%)
PRE-SURGERY (N=30)	6	20	5	16.7	15	50	1	3.3
POSTSURGERY (N=29)	4	13.8	5	17.3	14	48.2	1	3.3

Pre-surgery = at time of admission
Post-surgery = 3-4 days after surgery

4.7 HISTOPATHOLOGY

The summarised statistics of the prevalence of gastroduodenal ulceration identified by histopathology analysis is presented in table 4.7. The individual patient details are recorded in Appendix 6 (Pg. 72).

TABLE 4.7

PREVALENCE OF GASTRODUODENAL ULCERATION IDENTIFIED BY HISTOPATHOLOGY

SAMPLING STAGE	CARDIA		CORPUS		PYLORIS		DUODENUM	
	(n)	(%)	(n)	(%)	(n)	(%)	(n)	(%)
PRESURGERY (N=30)	7	23.3	7	23.3	15	50	6	20
POSTSURGERY (N=29)	7	24.1	10	34.5	12	41.4	8	27.6

4.8 OVERALL PREVALENCE

The summarised statistics of the prevalence of gastroduodenal ulceration was obtained by combining gastroduodenoscopy and histopathology analysis results. Dogs that were classified as either a grade 2 or 3, on either gastro-duodenoscopy or histopathology, were considered to be positive. Those with grade 0 or 1 were considered negative. These findings are presented in table 4.8. Twenty three out of thirty Dachshunds in this study were positive GDU on admission to OVAH, giving an overall prevalence of seventy six percent.

TABLE 4.8

OVERALL PREVALENCE OF GASTRODUODENAL ULCERATION

SAMPLING STAGE	CARDIA		CORPUS		PYLORIS		DUODENUM	
	(n)	(%)	(n)	(%)	(n)	(%)	(n)	(%)
PRE-SURGERY (N=30)	11	36.7	10	33.3	21	70	6	20
POST-SURGERY (N=29)	8	27.6	12	41.4	17	58.6	8	27.6

4.9 CORRELATIONS

The Fisher's exact test was used to determine whether statistically significant correlations existed between the variables listed below and the overall prevalence of gastroduodenal ulceration found in all the four regions at both time of admission (pre-surgery) and three to four days post-surgery. **P was set at ≤ 0.05**. The results are presented in table 4.9.1 – 4.9.3

4.9.1 Correlations between prevalence of ulceration and use of ulcerogenic drugs

TABLE 4.9.1

CORRELATION BETWEEN PREVALENCE OF ULCERATION AND USE OF ULCEROGENIC DRUGS

REGION	FISHER'S EXACT SCORE
CARDIA – PRE-SURGERY	0.687
CARDIA – POST-SURGERY	0.067
CORPUS – PRE-SURGERY	0.431
CORPUS – POST-SURGERY	0.234
PYLORIS – PRE-SURGERY	0.389
PYLORIS – POST-SURGERY	1.000
DUODENUM – PRE-SURGERY	0.329
DUODENUM – POST-SURGERY	0.209

4.9.2 Correlation between prevalence of ulceration and patient's neurological status

TABLE 4.9.2

CORRELATION BETWEEN PREVALENCE OF ULCERATION AND PATIENT'S NEUROLOGICAL STATUS

REGION	FISHER'S EXACT SCORE
CARDIA – PRE-SURGERY	1.000
CARDIA – POST-SURGERY	0.207
CORPUS – PRE-SURGERY	0.112
CORPUS – POST-SURGERY	1.000
PYLORIS – PRE-SURGERY	0.095
PYLORIS – POST-SURGERY	0.196
DUODENUM – PRE-SURGERY	0.814
DUODENUM – POST-SURGERY	0.480

4.9.3 Correlation between prevalence of ulceration and duration of clinical signs

TABLE 4.9.3

CORRELATION BETWEEN PREVALENCE OF ULCERATION AND DURATION OF CLINICAL SIGNS

REGION	FISHER'S EXACT SCORE
CARDIA – PRE-SURGERY	0.372
CARDIA – POST-SURGERY	0.305
CORPUS – PRE-SURGERY	0.633
CORPUS – POST-SURGERY	0.669
PYLORIS – PRE-SURGERY	0.049
PYLORIS – POST-SURGERY	1.000
DUODENUM – PRE-SURGERY	1.000
DUODENUM – POST-SURGERY	0.305

The pyloric region is the only site that happened to show marginal significance in the pre-surgical period. This probably reflects an incidental finding.

In summary, no correlation was found between the prevalence of GDU and the administration of ulcerogenic drugs prior to the admission or at post-operative follow-up on day 3 or 4. Similarly, no correlation was found between GDU and the severity of neurological signs, the duration of signs prior to admission, the length of the procedure (general anaesthetic or surgery), age, weight or sex. Seventy six percent of dogs were positive for GDU.

CHAPTER 5

DISCUSSION

IVDP, in chondrodystrophic breeds like the Dachshund, is a frequent reason for surgical admission to OVAH. GDU is reported to be more prevalent in dogs with IVDP than was previously identified at this facility[11,19,20]. Most cases of GDU are difficult to confirm clinically due to the subtle nature and late onset of the clinical signs, so actual prevalence is unknown and a rational, prophylactic approach is hard to formulate[25].

The primary objective of this project was to use a combination of gastro-duodenoscopy and histopathology in order to obtain an accurate assessment of the percentage of Dachshunds that suffer from GDU during presentation and treatment for IVDP. It was hypothesised that the results would reflect a higher prevalence than was previously obtained when assessing the dogs using only the clinical signs of intermittent vomition, melaena, abdominal pain and a decreased haematocit. Only one other case series looking at this problem is reported in the veterinary literature[20]. This study substantiated the assumption of a higher prevalence than was reflected by clinical signs alone.

Secondary objectives included assessing whether a significant difference existed between the prevalence of GDU on admission as opposed to three to four days post-surgery and formulating a risk profile for these dogs that would allow us to predict more accurately the likelihood of a patient with IVDP being affected by GDU. Factors that were considered for such a profile were: age, sex, weight, severity of clinical

signs, duration of spinal cord compression, length of general anaesthesia and previous treatments with ulcerogenic drugs. This information will hopefully allow clinicians to avoid the potential risk of subclinical GDU developing into more overtly clinical disease, leading to an increased morbidity and possibly mortality.

Ulcerogenic drugs like corticosteroids and NSAID are commonly used in patients with IVDP and have been shown to increase the likelihood of GDU. Confirming this suspicion would help clinicians rationalise the use of ulcerogenic drugs in the peri-operative period and thus influence the degree of GDU occurring in IVDP patients.

The process reported here was based on the subjective assessments of both the person performing the endoscopy and the histopathologist, making accuracy a concern throughout the project. By utilising the combination of endoscopy and histopathology we had hoped to improve the sensitivity more than by using any one of these procedures alone. In retrospect, combining these two results did not increase accuracy, as there was a more than 50% discrepancy between endoscopy and histopathology. It would have more beneficial to look for significant correlations between these modalities in the different regions of the gastrointestinal tract, both before and after surgery.

Faecal occult blood tests were performed in these patients and data were collected for interest sake. They proved however to be inconsistent and no statistical significance was found. It was concluded that faecal occult blood test is not a reliable test for detecting GDU in dogs due to the high proportion of myoglobin present in their diets.

Endoscopic examination permitted visualisation of the colour and integrity of multiple sites of the gastric mucosa, without the increased risk of perforation. The criteria used for evaluating the gastroduodenal mucosa during endoscopy relied on the accuracy of the observations of the person performing the endoscopy. All efforts were made to ensure that dogs were starved for at least 12 hours prior to the procedure and that monitor settings were kept constant and photographs recorded in order to provide comparisons and improve accuracy. The biopsy procedure was aimed at obtaining random samples and not specifically targeting mucosal defects that could be seen visually with the endoscope. This was performed in order to increase the accuracy of GDU prevalence determination, by identifying affected areas that were not visually significant.

Histopathology changes were often subtle and often difficult to categorise. Problems included having to differentiate between artefacts caused by the biopsy process (which would lead to false positive results) and genuine pathological changes. False negatives were obtained if isolated areas of the mucosa, which were not affected, were biopsied instead of affected areas. The random biopsy sampling process was again used to try and alleviate this.

Despite an extensive search of the literature, finding a grading system that was simple and yet effective for determining the criteria that would place a sample in a particular category was a further challenge. Ultimately it was decided, in consultation with the histopathologist, to develop a system that would suit the requirements of the project. Unfortunately, this system did not distinguish between patients with more than one lesion. It also made it difficult to categorise patients into one of two groups. Thus,

patients showing signs of only erosions and/or haemorrhage were classified in the same group as those with frank ulceration. With this point in mind, "gastroduodenal injury" would have been a more correct term to use throughout the project, than "gastroduodenal ulceration". This point should be noted if further trials are attempted.

Thirty dogs were admitted to the trial after confirmation of the presence of intervertebral disc prolapse based on clinical signs and lumbar myelographic results. In twenty-nine dogs, complete data was available. One dog was not available for follow-up gastroduodenoscopy and histopathology at four days post-surgery, as it presented an undue anaesthetic risk. No dogs were excluded based on the presence of concurrent diseases like renal or hepatic disease, which have been shown previously to predispose patients to GDU[5].

The results showed no correlation between the prevalence of GDU and the administration of ulcerogenic drugs prior to admission. Similarly, no correlation was found between the severity of clinical or neurological signs prior to admission, the length of the general anaesthesia and surgery times, age weight or sex.

Twenty three out of thirty Dachshunds (76%) were positive for GDU on either gastro-duodenoscopy and/or histopathology at the time of admission. The highest overall prevalence of ulceration was shown to occur in the pyloric region of the stomach both before and after surgery. The lowest prevalence occurred in the duodenum. Histopathology results of the proximal duodenum revealed that in this region, endoscopic visualisation was much less sensitive for detecting mucosal pathology

than histopathology. Gastric ulcers did not appear to be predictors for duodenal ulceration or visa versa.

The apparent lower prevalence of GDU seen in the post-surgery groups was not shown to be statistically significant. Cases where ulcers were visualised, but histopathology was negative may be explained by the fact that biopsy samples were taken randomly from the four identified regions, thus visualised mucosal lesions were not necessarily biopsied. Histopathology identified mucosal changes and ulcerations that were not visualised during gastroduodenoscopy, thus increasing the sensitivity of detecting GDU.

Neiger[20] used endoscopic examination to determine the incidence of GDU in dogs with acute intervertebral disc disease and concurrent corticosteroid use. They randomly assigned their patients into one of two treatment groups with anti-ulcer drugs or the control group. All three groups received dexamethasone at 2mg/kg on day 0, prednisilone at 2mg/kg on day 1, prednisilone at 1mg/kg on day 2 and then at 0.5mg/kg on all further days of the hospitalisation period. They found that there was no significant decrease seen in the incidence of GDU with concurrent anti-ulcer treatment. Histopathology was not performed and their results indicated an overall prevalence of 76% of dogs positive for GDU. Their conclusion was that either anti-ulcer medication was instituted too late to prevent mucosal lesions from forming or that the drugs and dosages used were not effective in acute intervertebral disc disease cases.

The prevalence of GDU in the current study was similar to that described by Neiger[20], despite the use of histopathology. This could imply that endoscopic visualisation alone is a sufficiently sensitive diagnostic modality. However, it could also imply that the incidence in Neiger's study would have been higher if a histopathological examination had been included. Significantly higher doses of corticosteroids were utilised in Neiger's study as opposed to this one. This could also account for the similarity in prevalence that they obtained, despite use of endoscopy only.

At OVAH dogs are carefully observed for any signs of GDU. If clinical signs are noticed, then all potentially ulcerogenic medication is withdrawn and anti-ulcerogenic medication instituted. It is also hypothesised that with concurrent surgery, which corrects the IVDP and alleviates the pain caused by this condition, we decrease the chance of subclinical GDU becoming a clinically overt disease. We speculated that if subclinical GDU is left untreated and the underlying problem is not corrected, then an increased risk exists of mild mucosal lesions becoming overt GDU with the possibility of penetration, increased morbidity and even patient mortality.

The proposed pathogenesis of GDU is multifactorial, however, in IVDP cases it is postulated that autonomic dysfunction caused by the spinal cord compression may lead to hypersecretion of gastric acid and pepsin with resultant GDU [5,15]. The current study, supports the suspicion that there may be a more involved pathophysiological processes occurring in patients with IVDP and that the administration of ulcerogenic medication is not the most important cause of the high percentage of mucosal lesions seen.

The prevalence of gastroduodenal haemorrhage and ulceration in dogs with neurological disease that have not received any form of ulcerogenic drug treatment has not yet been reported[22]. In this study, 11 dogs did not receive any form of ulcerogenic drug prior to admission. Of these, 6 presented with submucosal haemorrhage and/or obvious ulceration. Of the 18 dogs (60%) that received ulcerogenic drugs, 13 showed visual signs of submucosal haemorrhages and/or obvious ulceration. This again supports the proposal that GDU in IVDP cases is not primarily related to ulcerogenic medication.

Future research should be directed at assessing whether breed differences in the prevalence of GDU in IVDP patients occur and at strategies to reduce the incidence of gastric ulcers in dogs with IVDP. This may involve judicious use of ulcerogenic drugs in the pre-surgical period and earlier surgical intervention in these cases. The early use of prophylactic anti-ulcer medication is recommended in all patients presenting with IVDP, despite the lack of evidence that it reduces the morbidity[20].

REFERENCES

1. Bellah J R 2000 Colonic perforation after corticosteroid and surgical treatment of intervertebral disk disease in a dog. *Journal of the American Veterinary Medical Association* 9: 1002-1003.

2. Bolton J P, Cohen M M 1979 The effect of prostaglandin E2, 15-Methyl prostaglandin E2, and metiamide on established canine gastric mucosal barrier damage. *Surgery* 333-338.

3. Braund K G 1981Canine Intervertebral Disc Disease. In Bojrab M J (ed) *Pathophysiology in Small Animal Surgery* First Ed. Lea and Febiger, Philadelphia: 739-754.

4. Bray J P, Burbridge H M 1998 The canine intervertebral disk. *Journal of the American Animal Hospital Association* 34: 55-63.

5. Cosenza S F 1984 Drug-induced gastroduodenal ulceration in dogs. *Modern Veterinary Practice* 12: 923-925.

6. Crawford L M, Wilson R C 1982 Melaena associated with dexamethasone therapy in the dog. *Journal of Small Animal Practice* 23: 91-97.

7. Davies M 1985 Pancreatitis, gastrointestinal ulceration and haemorrhage and necrotising cystitis following the surgical treatment of degenerative disc disease in a dachshund. *Veterinary Record* 116: 398-399.

8. Grooters A M, Johnson S E, Sherding R G 1994 Endoscopy case of the month: Chronic vomiting and weight loss in a dog. *Veterinary Medicine* 3: 196-199.

9. Hall J A 2000 Diseases of the stomach. In Etthinger S J (ed) *Textbook of Veterinary Internal Medicine* Fifth Ed. WB Saunders, Philadelphia: 1154-1184.

10. Happe R P, Van der Gaag I 1983 Endoscopic examination of esophagus, stomach, and duodenum in the dog. *Journal of the American Animal Hospital Association* 19: 197-206.

11. Hoerlein B F 1975 Non-neurological complications following decompressive spinal cord surgery. *Archives of the American College of Veterinary Surgeons* 4: 11-16.

12. Jakovljevic S 1988 Gastric radiology and gastroscopy in the dog. *Veterinary Annual* 28: 172-182.

13. Jerram R M, Dewey C W 1999 Acute thoracolumbar disk extrusion in dogs – part 1. *Compendium for Continuing Education* 21: 922-930.

14. Kirberger R M, Roos C J, Lubbe A M 1982 The radiological diagnosis of thoracolumbar disk disease in the Dachshund. *Veterinary Radiology and Ultrasound* 33: 255-261.

15. Lu W Y, Rhoney D H, Boling W B, Johnson J D 1997 A review of stress ulcer prophylaxis in the neurosurgical intensive care unit. *Neurosurgery* 41: 416-425.

16. Lubbe A M, Kirberger R M, Verstraete F J M 1994 Pediculectomy for thoracolumbar spinal decompression in the Dachshund. *Journal of the American Animal Hospital Association* 30: 233-238.

17. Matz E M 1995 Gastrointestinal ulcer therapy . In Kirk R W (ed) *Current Veterinary Therapy XII, Small Animal Practice* Twelfth Ed. WB Saunders, Philadelphia: 706-710.

18. Menguy R, Masters Y F 1963 Effects of cortisone on mucoprotein secretion by the gastric antrum of dogs - pathogenesis of steroid ulcer. *Surgery* 54: 19-28.

19. Moore R W, Withrow S J 1982 Gastrointestinal haemorrhage and pancreatitis associated with intervertebral disk in the dog. *Journal of the American Veterinary Medical Association* 180: 1443-1447.

20. Neiger R, Gaschen F, Jaggy A 2000 Gastric mucosal lesions in dogs with acute intervertebral disc disease: Characterization and effects of omeprazole or misoprostol. *Journal of Veterinary Internal Medicine* 14: 33-36.

21. Prata R G 1981 Neurosurgical treatment of thoracolumbar disks: The rationale and value of laminectomy with concomitant disk removal. *Journal of the American Animal Hospital Association* 17: 17-26.

22. Rohrer C R, Hill R C, Fischer A, Fox L E, Schaer M, Ginn P E, Casanova J M, Burrows C F 1999 Gastric haemorrhage in dogs given high doses of methylprednisilone sodium succinate. *American Journal of Veterinary Research* 60: 977-981.

23. Tanaka M, Motoaki U, Motoo K 1979 Gastro-duodenal disease in chronic spinal cord injuries. *Archives of Surgery* 114: 185-187.

24. Taylor L A, Crawford L M 1968 Aspirin-induced gastrointestinal lesions in dogs. *Journal of the American Veterinary Medical Association* 152: 617-619.

25. Toombs J P, Caywood D D, Lipowitz A J, Stevens J B 1980 Colonic perforation following neurosurgical procedures and corticosteroid therapy in four dogs. *Journal of the American Veterinary Medical Association* 177: 68-72.

26. Toombs J P, Collins L G, Graves G M, Crowe D T, Caywood D D 1986 Colonic perforation in corticosteroid-treated dogs. *Journal of the American Veterinary Medical Association* 188: 145-150.

27. Trotter E J 1978 Canine intervertebral disk disease. In Kirk R W (ed) *Current Veterinary Therapy* Sixth Ed. WB Saunders, Philadelphia: 841-848.

28. Wallace M S, Zawie D A, Garvey M S 1990 Gastric ulceration in the dog secondary to the use of non-steroidal anti-inflammatory drugs. *Journal of the American Animal Hospital Association* 26: 467-472.

ABBREVIATIONS

A -Ambulation
Alb – Albumin
Alb/glob – Albumin/Globulin ratio
ALP – Alkaline Phosphatase
ALT – Alkaline Transaminase
CACS – Companion Animal Clinical Studies
CBC – Complete Blood Count
D – Deep pain sensation
Dex – Dexamethasone
Df – Dexafort
Dt - Dexatomonol
F – Flexor reflex
FI – Female Intact
FM – Flunixin Meglumine
FS – Female Spayed
GDU – Gastroduodenal ulceration
H - Histopathology
Hr/Hrs – Hour/Hours
IVDP – Intervertebral Disc Prolapse
Kg – Kilogram
Kpf – Ketaprofen
l – Lumbar Vertebra
MI – Male Intact
MS – Male Spayed
MAX – Maximum
Mel – Meloxicam
MIN – Minimum
Min – Minutes
M-pred - Methylprednisilone
NSAID – Non Steroidal Anti-inflammatory Drugs
OVAH – Onderstepoort Veterinary Academic Hospital
P – Conscious proprioception
Pg - Page
Pb – Phenylbutazone
PCV – Packed Cell Volume
Pred – Prednisilone
Prt – Protein
S – Scope (Gastroduodenoscopy)
SG – Urine Specific Gravity
T – Tail response
t – Thoracic vertebra
TCC – Thrombocyte Count
TSP – Total Serum Protein
Yr/Yrs – Year/Years

APPENDIX 1: Patient signalment

Patient	Age (yrs)	Sex	Body weight (kg)
1	5	MI	5.6
2	4	MI	6.5
3	8	FS	7.5
4	8	FS	4.2
5	6	MI	8.6
6	11	MI	6.0
7	6	MI	7.6
8	6	MI	8.4
9	5	MS	9.0
10	4	MI	9.2
11	2	FI	5.2
12	6	FS	6.0
13	5	FS	9.3
14	5	FI	7.2
15	4	MS	9.0
16	2	FI	6.4
17	5	MI	8.2
18	4	FS	7.7
19	6	FI	5.0
20	4	FS	7.76
21	5	FS	4.4
22	4	FS	6.16
23	8	MI	5.84
24	6	FS	9.3
25	5	FS	4.64
26	7	FS	8
27	5	MI	4.50
28	7.5	FI	4.10
29	7	FS	5.5
30	7.5	MI	8.38

APPENDIX 2: Duration of clinical signs, intervertebral disc affected and previous history of intervertebral disc prolapse

Patient	Duration (d)	Disc affected	Previous_History (Yes/No)
1	14	t12-13	Yes
2	56	t13-L1	No
3	0.75	t13-L1	No
4	1	t13-L1	Yes
5	1	t12-13	No
6	2	l3-4	Yes
7	14	t11-12	No
8	3	t12-13	Yes
9	14	l1-2	No
10	3	t13-L1	No
11	0.25	t13-L1	No
12	28	l1-2	No
13	5	l1-2	No
14	1	l2-3	No
15	3	t13-L1	No
16	90	l1-2	No
17	28	t12-13	No
18	7	t12-13	No
19	7	t12-13	No
20	5	t11-12	Yes
21	21	t13-L1	No
22	3	t12-13	No
23	4	l1-2	Yes
24	5	l5-6	No
25	2	t13-L1	No
26	6	t13-L1	No
27	4	t12-13	No
28	3	l3-4	No
29	10	l2-3	No
30	0.5	t13-L1	No

APPENDIX 3: Previous treatments with potentially ulcerogenic drugs

Patient	Yes/No	Drug
1	Yes	Dex
2	Yes	Pred
3	No	0
4	No	0
5	Yes	Pred + FM
6	No	0
7	No	0
8	Yes	Pred
9	Yes	Dex - Pb
10	No	0
11	No	0
12	Yes	Pred
13	Yes	Dex
14	Yes	Pb
15	Yes	Pred
16	No	0
17	Yes	Dex - Pb
18	Yes	Kpf
19	No	0
20	Yes	Pred + Dex
21	No	0
22	Yes	Pred
23	Yes	FM
24	Yes	Mel
25	Yes	Dex
26	Yes	Pred
27	No	0
28	Yes	Mp
29	No	0
30	No	0

KEY FOR APPENDIX 3:

Dex – Dexamethasone
Dex - Pb – Dexamethasone – Phenylbutazone combination
FM – Flunixin meglumine
Pb – Phenylbutazone
Pred – Prednisilone
Pred + FM – Prednisilone plus flunixin meglumine
Pred + Pb – Prednisilone plus phenylbutazone
Kpf – Ketaprofen
Mel – Meloxicam
Mp – Methylprednisolone

APPENDIX 4: Neurological status

Patient	F L:R	D L:R	T	A	P L:R.
1	1:1	1:1	1	1	0:0
2	1:1	1:1	1	2	0:0
3	1:1	1:1	0	2	0:0
4	1:1	0:0	0	2	0:0
5	1:1	1:1	0	3	0:0
6	1:1	1:1	0	3	0:0
7	1:1	1:1	1	1	0:0
8	1:1	1:1	1	2	0:0
9	1:1	1:1	0	3	0:0
10	1:1	1:1	0	2	0:0
11	1:1	1:1	1	2	0:0
12	1:1	1:1	1	0	0:0
13	1:1	1:1	0	3	0:0
14	1:1	0:0	0	3	0:0
15	1:1	1:1	0	3	0:0
16	1:1	1:1	1	0	0:1
17	1:1	1:1	1	0	1:0
18	1:1	1:1	1	0	1:0
19	1:1	1:1	0	2	0:0
20	1:1	1:1	1	0	1:1
21	1:1	1:1	1	0	1:1
22	1:1	0:0	0	4	0:0
23	1:1	1:1	1	0	1:1
24	1:1	1:1	0	3	0:0
25	1:1	1:1	1	0	1:1
26	1:1	1:1	1	1	0:0
27	1:1	1:1	1	1	0:0
28	1:1	1:1	1	1	1:0
29	1:1	1:1	1	1	0:0
30	1:1	1:1	1	0	0:0

KEY FOR APPENDIX 4:

A – Ambulation
D – Deep pain sensation
F – Flexor reflex
P – Conscious proprioception
0 = absent / abnormal
1 = present / normal

KEY FOR AMBULATION (A):

0 = Normal
1 = Ambulatory with pelvic limb paraparesis
2 = Non-ambulatory with pelvic limb paraparesis, with voluntary movement
3 = Non-ambulatory with paraparesis, without voluntary movement
4 = Complete pelvic limb paraplegia

APPENDIX 5: Gastroduodenoscopy results

Patient	Cardia		Corpus		Pyloris		Duodenum	
	S1	S2	S1	S2	S1	S2	S1	S2
1	2	2	2	1	2	2	0	0
2	0	0	0	0	2	3	0	0
3	0	0	0	0	1	0	0	0
4	0	0	0	0	0	1	0	0
5	0	0	0	0	0	0	0	0
6	0	0	0	0	0	0	0	0
7	0	0	1	2	2	2	0	0
8	0	0	0	0	1	0	0	0
9	0	0	0	0	2	2	0	0
10	2	2	2	1	2	2	3	0
11	0	0	0	0	0	1	0	0
12	0	0	0	1	0	0	0	0
13	0	0	2	0	2	1	0	3
14	2	1	2	3	3	2	0	0
15	0	0	0	0	0	0	0	0
16	0	*	0	*	0	*	0	*
17	1	1	1	2	2	2	0	0
18	0	0	0	1	2	0	0	0
19	2	3	1	3	2	2	0	0
20	0	0	0	0	3	1	0	0
21	0	0	0	0	0	1	0	0
22	1	2	1	1	0	2	0	0
23	0	0	0	0	0	0	0	0
24	0	0	0	0	1	1	0	0
25	1	0	1	2	2	2	0	0
26	0	0	1	0	2	2	0	0
27	2	1	1	0	1	2	0	0
28	2	0	2	0	3	2	0	0
29	0	0	0	0	0	0	0	0
30	0	1	0	1	2	2	0	0

KEY:

S = Scope / Gastroduodenoscopy
0 = Normal
1 = Hyperaemia / Inflammation
2 = Submucosal haemorrhages / Erosions
3 = Obvious ulceration
* = Sample not obtained (Dog 16 did not have a second scoping)

0 and 1 = negative for ulceration
2 and 3 = positive for ulceration

APPENDIX 6: Histopathology results

Patient	Cardia		Corpus		Pyloris		Duodenum	
	H1	H2	H1	H2	H1	H2	H1	H2
1	0	2	0	0	0	0	0	2
2	1	0	2	2	2	2	0	0
3	1	2	0	0	2	2	0	1
4	0	2	2	0	0	0	2	2
5	0	0	0	1	0	2	0	2
6	0	0	2	1	2	2	0	0
7	2	1	2	2	2	2	2	2
8	2	0	0	0	2	2	0	0
9	0	0	0	0	0	0	0	0
10	0	1	0	2	0	2	0	1
11	0	0	0	0	0	0	0	0
12	0	1	0	0	2	1	2	2
13	0	0	1	1	0	1	0	0
14	2	2	0	2	2	1	0	0
15	0	0	0	0	2	2	1	1
16	0	*	0	*	0	*	0	*
17	0	0	0	0	0	0	0	0
18	1	2	0	0	0	0	1	2
19	0	1	1	2	2	2	1	2
20	0	0	0	0	2	2	2	0
21	2	2	0	0	0	1	2	0
22	1	1	2	2	2	0	0	0
23	0	0	0	0	0	0	0	0
24	0	0	0	2	0	0	0	0
25	0	0	0	0	0	2	0	0
26	2	0	1	2	1	2	0	2
27	2	0	2	0	2	0	0	0
28	2	2	2	2	2	0	0	1
29	0	0	0	0	2	0	2	0
30	0	0	0	2	2	0	0	0

KEY:
H = Histopathogy results
0 = Normal
1 = Hyperaemia / Inflammation
2 = Submucosal haemorrhages / Erosions
3 = Obvious ulceration
* = Sample not obtained (Dog 16 did not have a second scoping)

0 and 1 = negative for ulceration
2 and 3 = positive for ulceration

APPENDIX 7: Blood and serum chemistry results

Patient	Ht (l/l)	TCC 10x^9 /l	WCC 10x^9 /l	ALP U/l	ALP U/l	Urea Mmol/l	Creatinine µmol/l	Alb g/l	Glob g/l	Alb/Glob
1	0.459 0.436	336 244	14.3 7.1	128	271	4.9	72	45.9	20.6	2:23
2	0.455 0.438	339 352	8.1 6.6	34	75	2.8	70	39.2	23.3	1.68
3	0.504 0.444	340 303	17.8 23.4	17	29	4.9	88	36.6	38.3	0.96
4	0.542 0.482	495 450	7.7 21.5	13	16	1.8	79	38.7	30.7	1.26
5	0.546 0.473	358 305	10 9.27	32	19	2.5	69	43.2	23.8	1.82
6	0.535 0.548	482 411	8.9 12.4	29	41	4.5	95	41.7	30.2	1.38
7	0.489 0.489	230 287	10.2 8.8	20	19	13	135	42.7	41.7	1.02
8	0.561 0.516	305 361	11.8 9.0	19	15	11.3	117	39.4	27.9	1.41
9	0.601 0.507	439 330	9.1 5.6	131	136	3.9	87	48.2	37.0	1.30
10	0.483 0.449	322 294	14.2 9.0	37	59	3.7	84	40.6	26.3	1.54
11	0.407 0.468	315 307	9.6 13.6	14	45	3.2	79	37.9	20.9	1.81
12	0.458 0.391	599 451	7.6 10.8	27	67	3.7	83	41.4	27.9	1.48
13	0.493 0.490	508 485	17.4 11.6	14	36	2.5	93	43.4	28.3	1.53
14	0.498 0.411	552 356	20.8 14.6	36	53	4.0	92	49.8	27.0	1.84
15	0.548 0.479	327 320	12.7 13.7	18	12	3.9	102	42.2	26.2	1.61
16	0.505 0.437	475 359	15 10.7	14	21	4.2	89	37.2	27.2	1.37
17	0.549 0.500	485 400	9.6 17.1	147	32	2.5	72	41.1	26.3	1.56
18	0.532 0.432	373 436	12.1 11.3	16	16	2.9	83	42.5	24.8	1.71
19	0.545 0.518	479 382	13.4 15.5	28	41	5.1	73	36.3	25.2	1.44
20	0.516 0.455	304 316	9.9 17.1	15	60	2.6	75	37.4	31.5	1.19
21	0.521 0.480	573 470	12.9 11.8	34	180	3.2	64	40.6	26.6	1.53
22	0.464 0.385	349 273	11.7 29.4	19	63	4.0	45	38.2	21.3	1.79
23	0.460 0.383	390 299	18.9 7.3	16	24	5.9	65	41.9	27.3	1.53
24	0.536 0.396	383 316	10.4 13.3	26	21	3.7	75	43.9	35.2	1.25
25	0.378 0.374	337 330	3.6 6.6	25	67	3.3	70	32.6	27.0	1.21
26	0.503 0.429	406 340	7.4 5.9	15	58	2.6	66	44.3	24.3	1.82

27	0.578 0.446	506 409	26.6 16.1	66	66	9.1	104	45.2	33.8	1.34
28	0.478 0.454	393 396	12.8 16.8	34	58	3.7	56	40.7	32.7	1.24
29	0.431 0.506	347 501	5.9 7.8	46	31	4.0	60	33.7	54.7	1.60
30	0.481 0.429	519 403	10.4 11.4	53	57	5.7	83	45.6	27.0	1.69

APPENDIX 8: Urinanalysis and faecal occult blood test results

PATIENT	Urine SG	Urine pH	Urine protein (mg/dl)	Faecal occult blood (positive / negative)
1	1038	7.5	100	positive
2	1028	6	100	positive
3	1022	7	200	positive
4	1038	7	100	negative
5	1042	7	30	negative
6	1028	6.5	30	negative
7	1020	5.5	30	negative
8	1018	5.5	30	positive
9	1012	7	30	positive
10	1032	5	15	positive
11	1034	8	0	negative
12	1038	5	30	positive
13	1047	8	60	positive
14	1030	5.5	30	positive
15	1036	6	30	positive
16	1042	7	30	negative
17	1048	6.5	15	positive
18	1028	5	30	positive
19	1038	6.5	15	positive
20	1034	5	30	positive
21	1030	7	30	positive
22	1023	6.5	30	positive
23	1040	6.5	30	positive
24	1026	6.5	0	positive
25	1035	7.5	30	positive
26	1028	6.5	30	positive
27	1018	7	500	positive
28	1020	6.5	15	positive
29	1031	6	0	negative
30	1010	7	30	negative

APPENDIX 9: General anaesthesia and surgical times

Patient	General anaesthetic time (min)	Surgery time (min)
1	175	85
2	160	80
3	150	75
4	135	75
5	180	90
6	145	70
7	140	75
8	170	80
9	180	75
10	165	90
11	145	50
12	130	55
13	135	60
14	140	65
15	200	90
16	195	80
17	200	120
18	260	125
19	165	75
20	190	90
21	145	55
22	175	105
23	170	80
24	150	90
25	140	60
26	160	110
27	110	55
28	100	50
29	180	100
30	135	45