Survival rates and outcomes in cats with thoracic and lumbar spinal cord injuries due to external trauma

Survival rates and outcome were retrospectively examined in 30 cats with traumatic spinal cord lesions to the thoracic and lumbar spine. Treatment included both surgical and non-surgical management. In cats with loss of deep pain sensation, a high incidence of myelomalacia was found during surgery or postmortem examination. The outcome in cats with grade II and III neurological dysfunction (11/18) was poor in two cases, functional in one and complete in eight. Recovery in cats with grade IV neurological dysfunctions (7/18) was poor in one case, functional in two and complete in four. The other 12 of the original 30 cats were euthanased within four days of presentation. The results of this study are compared with those reported in cats with spinal injuries due to spontaneous disc herniations and to those that have been achieved in cats with experimental transections of the spinal cord.

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INTRODUCTION

Spinal fractures, luxations and compressive lesions may result in different degrees of spinal dysfunction, ranging from pain to paraplegia. The most common causes of spinal trauma in cats include road traffic accidents and falls. In contrast to the dog, spontaneous disc herniations causing clinical problems are rarely seen (Kathmann and others 2000, Munana and others 2001) in cats. The outcome of spinal injuries has been reported in many articles describing studies of dogs (Brown and others 1977, Scott 1997) or of mixed populations of cats and dogs (Carberry and others1989, Selcer and others 1991). However, there is little clinical information focusing on cats with spinal cord injuries caused by external trauma.

The purpose of this study was to assess outcome and survival rates in cats with thoracic and lumbar spinal cord injuries related to external trauma. The results are compared with those achieved in cats with experimentally induced spinal cord lesions (Barbeau and Rossignol 1987, Lovely and others 1990, Edgerton and others 1991) and with those reported for cats with degenerative disc disease (Kathmann and others 2000, Munana and others 2001).

MATERIALS AND METHODS

Case selection

This study was carried out between 1996 and 2001 at the Department for Small Animals, University of Zurich. Clinical details were retrospectively examined for 30 cats with traumatic injuries to the thoracic and lumbar spinal cord. Clinical records were reviewed and cases were deemed eligible for the study when neurological and radiographical data, and treatment information were available. Neurological diagnosis was based upon radiographic, intraoperative and postmortem findings. Data obtained from the records included signalment, cause of injury, degree of neurological dysfunction, radiographic interpretation and mode of treatment (non-surgical/surgical). Follow-up information was obtained through phone calls and neurological re-evaluations at various intervals. A final examination was performed on each cat when owners reported that no visible progress had been observed for at least four weeks. The length of the follow-up period ranged from four weeks to two years. At the end of the study, cases were clinically classified as having a complete, functional or poor recovery.

The cats ranged in age from two months to 12 years (mean 3·6 years), and included 14 males and 16 females. Three were purebred cats, the others were domestic cats. Within the entire group, 22 cats suffered from compression of the spinal cord, caused by vertebral body and endplate fractures, or subluxations with or without articular facet involvement. Common causes were road accidents, falls, bites and gunshot wounds. Six cats had thoracolumbar disc extrusions as a consequence of a confirmed trauma without evidence of associated fractures. Two cats suffered from contusion of the spinal cord after falls. The cats were clinically graded into

Table 1. Grades used to group cats in the study, based on the severity of their neurological signs

Grade	Neurological dysfunction	No of cats
I	Back pain, no neurological deficits	0
II	Ambulatory paraparesis, normal micturition	6
III	Ambulatory paraparesis, urinary retention	7
IV	Non-ambulatory paraparesis/paraplegia, urinary retention, intact deep pain perception	9
V	Paraplegia, urinary retention, loss of deep pain perception	8

one of five groups based on the severity of their neurological dysfunction (Table 1).

Deep pain perception was assessed by pinching the extremities of the limbs and the tail with bone-holding forceps. Cats showing doubtful reactions to stimulation were considered to have no deep pain perception. In addition, cats were assigned to one of the two following categories: acute (onset of clinical signs in less than 48 hours, 20 cats) or chronic (onset of clinical signs in more than 48 hours, 10 cats).

Radiography

Plain radiography was performed in all cases. Thirteen cats underwent myelography under general anaesthesia. Iotrolan 0·25 ml/kg bodyweight (Isovist 240; Schering-Plough) was injected into the subarachnoid space by cisternal or lumbar puncture. Myelography was performed in cats when no evidence of cord compression was detectable on plain radiographs or at the veterinary surgeon's request. All cats with traumatic disc herniations were diagnosed with the aid of myelography.

Treatment

Eighteen cats could be followed after non-surgical or surgical treatment; 12 cats (grade II to V) were euthanased or died within four days of presentation. Six cats received non-surgical treatment. Criteria for conservative therapy included absence of unstable fractures or luxations and compressive lesions within the spinal canal as defined by myelography. Three cats were treated with steroids (prednisone) orally at a dose of 1 mg/kg twice daily for three to four days. Three cats received no steroidal medication. All cats were cage rested for a minimum of one week and remained under restricted movement for an additional four weeks. Physiotherapy was only applied when the animals were pain free and they could be handled without risking further damage to the injured sites.

Surgery was performed in cats with unstable fractures or luxations and/or compressive spinal cord lesions due to disc material or bony fragments. Six out of the 12 cats that underwent surgery were treated with prednisone or dexamethasone by the referring veterinarian. Four surgically treated cats were infused with methylprednisolone sodium succinate within eight hours of the accident, initially with a 30 mg/kg bolus, and with 15 mg/kg boluses at two and six hour intervals for a maximum duration of 24 hours. A standard dorsolateral hemilaminectomy was performed in two cases with traumatic disc extrusions. In 10 cats with fractures of the thoracolumbar spine, a spinal stabilisation was performed using a U-shaped Kirschner wire and figure-of-eight wiring around the dorsal and transverse spinal processes (Koch and Montavon 1998). In cats with absent deep pain perception, gross integrity of the spinal cord was visually assessed by a durotomy. If the cord showed evidence of malacia or transection, the cat was euthanased.

Urinary retention due to an upper motor neuron lesion was treated pharmacologically with diazepam (Valium; Roche), at a dose of 1 mg/kg three times daily, to relax the external urinary sphincter, and with prazosin (Minipress; Pfizer), at a dose of 0·1 mg/kg twice daily, to relax the internal urinary sphincter. Bladder contractility was enhanced with bethanechol chloride (Myocholin; Glenwood) at a dose of 2 mg/kg twice daily.

Surgically and non-surgically treated cats received rehabilitative care four to six times daily for 10 to 15 minutes, including massage, passive motion of the paralysed hindlimbs and assisted gait support, as soon as spontaneous motor activity was present. Cats were usually discharged within one to six weeks, as soon as bladder function returned to normal and the owner managed to manually void urine at home.

RESULTS

Clinical features

All of the 30 cats had varying degrees of pelvic limb upper or lower motor neuron dysfunction. Thirteen cats (43 per cent) were assigned as having grade II or III neurological dysfunction, nine cats (30 per cent) grade IV and eight cats (27 per cent) grade V. Urinary retention occurred in 23 cats (76 per cent). The most common site of spinal cord damage were vertebral bodies L2/3 and L4/5, followed by T12/13, T13/L1, T11/12 and L1/2. The vertebral bodies L2/3 and L4/5 accounted for 43 per cent of all injuries. The thoracolumbar area (T11 to L2) was affected in 45 per cent of cases.

Survival rates

At the end of the follow-up period, 15 of the 30 cats (50 per cent) were alive. Twelve cats (40 per cent) had been euthanased or had died within one to four days after presentation. Reasons for euthanasia included myelomalacia detected during surgery (six cats, 20 per cent), severe postoperative cardiovascular complications (three cats, 10 per cent) and owner's decision (three cats, 10 per cent). The last three cases were all assigned as having grade IV or V neurological dysfunction. Two of them had luxated, overriding vertebrae, detected by plain radiography, and a postmortem examination revealed focal myelomalacia in the third case. A poor response to treatment meant that three of the 30 cats (10 per cent) were euthanased between two weeks and two months after treatment at their owner's request. Persistent urinary retention (one cat) and persistent, severe gait abnormalities (two cats) were the reasons for euthanasia in these cases. Postmortem results were not available for these cats.

Table 2. Case details of 30 cats with traumatic spinal cord injuries

Signalment	Duration	Grade	Radiological diagnosis	Therapy/ intraoperative findings	Outcome	Time of recovery (days)
Persian, 8 y, mc	Acute	II	DH L1/2, M	Decompression, steroids	Excellent	7
DSH, 6 y, fs	Acute	II	DH T12/13, M	Decompression, steroids	Excellent	40
DSH, 1 y, fs	Acute	II	Intramedullary swelling, T12-13, M	Steroids	Excellent	24
Persian, 6 m, f	Chronic	II	Endplate fracture, luxation L4/5	Stabilisation	Excellent	21
DSH, 2 y, mc	Acute	II	Subluxation T13/L1	Stabilisation, steroids	Died postoperatively	
DSH, 12 y, fs	Acute	II	Endplate fracture L2/3	Stabilisation	Died postoperatively	
DSH, 5 y, fs	Acute	III	Subluxation L4/5	Stabilisation, steroids	Excellent	30
DSH, 1 y, mc	Chronic	III	Endplate fracture L4	Stabilisation	Excellent	28
DSH, 3 y, fs	Acute	III	DH T12/13, M	Steroids	Functional	40
DSH, 2 y, mc	Acute	III	Endplate fracture, subluxation L2/3, M	Stabilisation, steroids	Excellent	28
DSH, 4 y, fs	Acute	III	Endplate fracture L3/4	Stabilisation, steroids	Excellent	30
DSH, 1 y, fs	Chronic	III	Endplate fracture, subluxation T10/11, M	Stabilisation, steroids	Poor, euthanasia	22
DSH, 3 y, f	Acute	III	Endplate fracture, subluxation L1/2, M	Steroids	Poor, euthanasia	14
DSH, 3 y, fs	Acute	IV	Endplate fracture T12/13	Stabilisation, steroids	Functional	40
DSH, 2 y, fs	Chronic	IV	Endplate fracture L2	Stabilisation, steroids	Excellent	21
DSH, 2 m, f	Acute	IV	Intramedullary swelling L2-3, M	Cage rest	Excellent	112
DSH, 12 y, fs	Acute	IV	Endplate fracture, subluxation T12/13, M	Stabilisation, steroids	Functional	40
DSH, 1 y, f	Chronic	IV	Vertebral body fracture T12	Cage rest	Excellent	28
Siamese, 6 y, fs	Chronic	IV	Vertebral body fracture L2	Cage rest	Excellent	28
DSH, 1 y, mc	Acute	IV	Endplate fracture, subluxation L2/3, M	Stabilisation, steroids	Poor, urinary retention, euthanasia	28
DSH, 8 y, fs	Acute	IV	Vertebral body fracture L1, DH L1/2, M	Decompression, steroids	Died postoperatively	
DSH, 3 y, mc	Chronic	IV	DH L1/2, M	Focal myelomalacia (postmortem)	Euthanasia	
DSH, 3 y, m	Acute	V	Endplate fracture, subluxation T13/L1	Myelomalacia	Euthanasia	
DSH, 1 y, mc	Chronic	V	Vertebral body fracture L3	Myelomalacia	Euthanasia	
DSH, 6 y, fs	Acute	V	DH L4/5, M	Myelomalacia	Euthanasia	
DSH, 9 y, fs	Chronic	V	Endplate fracture T12/13	Myelomalacia	Euthanasia	
DSH, 2 y, mc	Acute	V	Luxation T13/L1	Myelomalacia	Euthanasia	
DSH, 1 y, fs	Chronic	V	DH L2/3, M	Myelomalacia	Euthanasia	
DSH, 10 y, fs	Acute	V	Luxation, overriding T12/13	–	Euthanasia	
DSH, 8 y, fs	Acute	V	Luxation, overriding L1/2	–	Euthanasia	

DSH Domestic shorthaired cat, m Male, mc Male castrated, fs Female spayed, f Female, DH Traumatic disc herniation, M Myelography performed, y Years, m Months

Clinical outcome

Overall, 18 of the 30 cats (60 per cent) were followed for a sufficient period of time after treatment to allow assessment of the clinical outcome. Poor outcome was defined as persistent difficulty in voiding urine and/or no improvement in gait after treatment. Functional recovery was defined as the cat becoming an acceptable household pet in terms of bladder control and the ability to undertake unassisted and pain-free ambulation. Complete recovery corresponded to normal urination and no gait and proprioceptive abnormalities.

The outcome in cats with neurological dysfunctions of grade II and III (11 of 18 cats) was poor in two, functional in one and complete in eight cases. Recovery in cats with grade IV neurological dysfunction (seven of 18) was poor in one, functional in two and complete in four cases. All cats classified as having grade V neurological dysfunction were euthanased.

Twelve cats underwent surgical treatment and six cats were treated non-surgically. Of those animals treated surgically, two (17 per cent) had a poor outcome, two recovered functionally and eight (66 per cent) recovered completely. Three cats undergoing surgery suffered from non-ambulatory paraparesis (grade IV neurological dysfunction) and all recovered functionally or completely.

Of the six non-surgically treated cats, four (67 per cent) recovered completely, one functionally and the outcome was poor in one. Prior to treatment, three cats in this group were non-ambulatory paraparetic and three had ambulatory paraparesis. Out of this group, three cats recovered without the use of steroids, by cage rest and rehabilitative care alone.

In 20 of the 30 cats, neurological signs had an acute onset (within less than 48 hours). Of 12 cats surviving the initial trauma or its sequelae, 10 (83 per cent) recovered functionally or completely between a period of seven and 112 days (mean 40 days). Ten of 30 cats had a chronic onset of neurological dysfunction at between two and 42 days (mean nine days). Five (83 per cent) of the surviving six cats recovered completely within 21 to 28 days (mean 25 days). Detailed information about each individual case is given in Table 2.

DISCUSSION

Spinal injuries due to trauma account for 13 per cent of cases of feline spinal cord disease (Marioni and others 2002). Management of these patients, including non-surgical and surgical treatment protocols, has been described in various articles (Mendenhall and Litwak 1976, Carberry and others1989, Selcer and others 1991, Koch and Montavon 1998, Bagley 2000). However, clinical data focusing on the outcome in feline cases are lacking in the veterinary literature. This is in contrast to experimental work that has been carried out in cats with spinal transections (Lovely and others 1986, Barbeau and Rossignol 1987, Roy and others 1992, Edgerton and others 1983). Data derived from these studies showed that a very high percentage of cats with artificially inflicted spinal lesions (complete transections) were able to walk with minimal assistance.

It was concluded from these studies that, with adequate training, the isolated lumbar spinal cord can develop a degree of spinal walking (see later), which is similar to the gait of control cats. A prerequisite for these results is intensive and regular training on a treadmill, which results in a near normal gait within four weeks after spinalisation.

Paraplegic cats with spinal cord injury due to spontaneous herniation of degener-

ated discs are reported to have a good prognosis, provided that timely decompression is carried out (Kathmann and others 2000, Munana and others 2001).

In this study, 15 of 30 cats (50 per cent) were euthanased as a direct consequence of the spinal trauma or secondary lesions that became life-threatening during surgery, or because of malacia of the spinal cord. Although recovery of function in the presence of focal myelomalacia has been described in small animals (Salisbury and Cook 1988, Duval and others 1996), the finding of myelomalacia (including one case with focal myelomalacia) was considered an indicator of a hopeless prognosis in this study. Scott (1997) and Wheeler and Sharp (1994) recommend visual assessment of gross spinal cord integrity as an indicator of possible 'restitutio in integrum'. Loss of deep pain sensation is not necessarily associated with myelomalacia. For that reason, absent deep pain may not be used as an objective test to assess anatomical cord integrity.

However, in this study the coincidence of loss of deep pain and myelomalacia has been striking. Similar observations have been reported in dogs and cats after spinal trauma by Feeney and Oliver (1980). The most likely explanation for this finding is the violent nature of the primary injury, resulting in irreversible damage to the spinal cord. In the more chronic cases reported here, myelomalacia may eventually have resulted from secondary mechanisms of spinal cord damage. On the basis of this result, it seems reasonable to perform a durotomy in cases with absence of deep pain sensation following spinal trauma, as they are likely to be suffering from myelomalacia.

Feeney and Oliver (1980) reported that none of the animals lacking deep pain sensation in the hindlimbs showed neurological improvement in the three years after injury. In contrast, unassisted ambulation in cats with experimentally induced complete cord transections is described by several authors (Lovely and others 1986, Barbeau and Rossignol 1987, Edgerton and others 1983). In agreement with prior studies (Feeney and Oliver 1980), the present study shows that damage to the spinal cord after external trauma seems to be more severe and widespread than an experimentally induced, focal lesion. Consequently, mechanisms of recovery, including immediate and delayed reorganisation within spinal cord synapses, may be more difficult to achieve (Jeffery and Blakemore 1999). An additional explanation for the poor recovery in cats with absent deep pain sensation in the clinical situation may be the lack of intensive rehabilitative training.

The effect of training on cats with spinal cord transection has been impressively demonstrated by Lovely and others (1986). Fourteen of 16 (87 per cent) cats in that study were capable of bearing the full weight of their hindlimbs while walking on a treadmill one month after transection (spinal walking). The authors point out that not only the presence of locomotor training, but also the precise method of training, with emphasis on correct paw placement and tail pinching to enhance muscle activation, are factors in the recovery of locomotor capabilities. The cats were trained for 30 minutes a day, five days a week (Lovely and others 1986). In contrast, Giuliani and others (1984) found that only seven of 21 untrained cats regained full weightbearing ability four months after transection. It is not stated whether spinal walking occurred in these animals. To date, it remains unclear if cats with myelomalacia as a consequence of traumatic lesions are capable of similar recovery with the aid of an intensive training protocol. This question should be addressed in future investigations.

In the present study, clinical outcome in the surviving cats was complete in 12 cases (grade II to IV), functional in three (grade III to IV) and poor in three (grade III to IV). There was no clear correlation between initial grade of neurological dysfunction and outcome in this relatively small population. However, it may be concluded that cats with spinal cord injury have a favourable prognosis if deep pain perception is intact. Cats with neurological dysfunctions classified as grade V were not among these 18 surviving animals. In agreement with reports of spinal injuries following spontaneous disc herniations (Kathmann and others 2000, Munana and others 2001), cats with a lower grade of spinal dysfunction due to external trauma (grade II to IV in this study) have a good to excellent prognosis.

Recommendations for the management of spinal injuries in animals remain controversial. Some authors suggest that similar results are obtained with both surgical and non-surgical therapies for spinal fractures, irrespective of the severity of spinal dysfunction (Carberry and others1989, Selcer and others 1991). Historically, surgery is indicated in cases with instabilities and spinal cord compression. However, recovery in animals with ambulatory paraparesis (grade II and III) may occur also in non-surgically treated cases (Carberry and others1989). Non-ambulatory animals are of greater concern. In this study, an equal number (three cases) of non-ambulatory cats was treated either surgically or non-surgically. In both groups, outcome was functional or excellent. However, the small number of cases and the biased application of treatment modality do not allow statistically valid conclusions to be drawn. The surgically semi-rigid technique for vertebral fractures used in the cats in this report is described in detail by Koch and Montavon (1998). It is beyond the scope of this paper to comment on it. Theoretically, any internal fixation technique can be applied (Wheeler and Sharp 1994).

The time frame in which complete recovery occurred in this study was broad and ranged from 21 to 112 days. This means that predicting the course of recovery at the moment of presentation will be difficult. The length of time required for recovery after spinal cord injuries depends on the severity (number of involved axons) and type of the lesion (compression versus contusion).

Contusion leads to irreversible damage to spinal cord function due to tissue

destruction triggered by secondary mechanisms mainly induced by ischaemia (Coughlan 1993). In experimental settings, no significant distinction in dysfunction exists between mild, moderate or severe spinal cord lesions within the first 24 hours. Differences become increasingly apparent though after seven days (Noble and Wrathall 1989). Deficits due to compression are attributed to blocking of impulse transmission caused by mechanical compression of axons (Tarlov and Klinger 1954) and the disruption of normal microcirculatory blood flow (Wright and Palmer 1969). Chronic compression leads to structural changes in blood vessels, loss of axons and demyelination of the remaining ones. That is to say, the longer the period of compression lasts, the greater the length of time required for recovery will be (Tarlov and Klinger 1954). Under clinical conditions, there is often a combination of contusion and compression. Differentiation between these two types by currently available clinical methods, such as neurological examination and diagnostic imaging procedures, is not fully possible.

Cats with chronic spinal dysfunction, as defined here, might be expected to have a longer recovery period. This was not confirmed in the current study. Although not statistically proven, this is in agreement with the observation of Funkquist (1962), who noted that the duration of the onset of clinical signs is not a reliable prognostic indicator. Therefore, cats with a history of chronic (defined as more than 48 hours in this study) spinal dysfunction and intact deep pain perception may not necessarily be expected to have longer recovery periods than those with an acute onset of clinical signs.

The authors acknowledge the limitations of this paper. Myelography or another advanced imaging method is needed to establish spinal compression in cases of spinal fractures and luxations (Bagley 2000). The amount of displacement or compression noted at the time of myelographic examination is the minimum that has occurred and therefore does not reflect the real situation at the time of injury.

As not every cat in this retrospective study underwent myelography, additional lesions not seen on survey radiographic examination may have been missed. The small sample size in this report did not allow valid statistical analyses. Therefore, absence of differences between outcome in surgically and non-surgically treated animals, as well as an absence of differences between the acute and the chronic group, may not hold true in a larger series.

Conclusions

In this population of cats with external spinal trauma, the survival rate was 50 per cent. Although this group of cats is heterogeneous in their degree of neurological dysfunction and treatment modality, it illustrates that outcome may be successful even in paraplegic cats with intact deep pain sensation. Mortality was mainly due to myelomalacia and life-threatening secondary lesions. In agreement with the results from reports of cats with spontaneous disc herniations, it was found that outcome was good to excellent in both cats with ambulatory and non-ambulatory paraparesis, and intact deep pain sensation. Based on the high incidence of myelomalacia in this series of cats, the authors suggest durotomy as a standard procedure in cats without deep pain sensation, if surgery is considered.

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