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'Bottom-hung window' trauma in cats: neurological evaluation and outcome in 71 cats with bilateral hindlimb injury

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ABSTRACT

Objectives Influence of neurological status on the mortality rate of paraparetic/paralysed cats presenting after entrapment in a bottom-hung window was evaluated. It was hypothesised that (1) loss of deep pain sensation at admission would not be a negative prognostic factor for regaining motor function and that (2) mortality rate would be influenced by the severity of neurological grade upon admission.

Methods Clinical and pathological data of affected cats that presented at our institution between 2001 and 2012 for this specific trauma were collected retrospectively: breed, age , sex, last contact with owner (<3 hours and >3 hours), whether the cat was suffering from monoparesis or paraparesis/paraplegia, duration of hospitalisation ,rectal temperature, surface temperature of the skin of the hindlimbs, femoral pulse and tone of the pelvic musculature. Neurological status was categorised according to Scott's classification scheme with neurological grade 1 representing spinal pain on manipulation down to neurological grade 5 representing paraplegia with no deep pain sensation.

Results Data were collected for 98 cats that had been caught in the thoracolumbar area. A total of 71 cats were included in the statistical analysis after excluding 27 cats for the following reasons: monoparesis/monoplegia by getting one limb caught only (15), no initial neurological examination (5), was dead on arrival (1), caught within the thoracolumbar area but no clinical or neurological abnormalities (6). The proportional mortality rate overall was 35% (25/71) with 65% of these (16/25) dying with clinical signs of respiratory distress. The proportional mortality rate of patients with a neurological grade of 5 on admission was 55% (17/31).

Conclusions Mortality was influenced by the severity of neurological grade on admission, with the majority of cats having a neurological grade of 5 (paraplegia with loss of deep pain perception) at admission dying (11/31) rather than undergoing euthanasia on admission at the owner's request (6/31). In the survivors' group, loss of deep pain perception was not a negative prognostic factor for regaining motor function although it was associated with mortality.

INTRODUCTION

Acute traumatic paralysis of the hindlimbs after getting caught in a 'bottom-hung window' has been reported in cats.[3] This trauma syndrome occurs when cats try to escape through the small V-shaped opening at the top of bottom-hung windows (Fig 1), but instead become trapped. Only a single retrospective study has been reported in the publicly available literature, and involved a group of 30 cats with bottom-hung window syndrome.[1] The animals had reportedly been lodged in the window between the last rib and the pelvis or caught by a single limb; when attempting to free themselves, they slid even further forward, ending up with clinical signs of ischaemia of the affected limbs. Clinical signs that can be related to ischaemia are coldness of the affected limbs, no palpable femoral pulses and low rectal temperatures.[1] Various neurological deficits, such as paraparesis, monoparesis, paraplegia and monoplegia, were reported in the previous study and a survival rate of 75% was observed.

The aim of the current study was to evaluate the association of clinical and neurological status with the outcome of cats that had presented to our university hospital with acute traumatic paralysis resulting from bottom-hung window trauma. We hypothesised that the proportional mortality rate would be influenced by severity of the neurological grade upon admission while pain perception on admission would not be a prognostic factor for regaining motor function.

MATERIALS AND METHODS

Case history and clinical data

The case history and clinical data of cats that presented at the Small Animal Surgical Clinic after being found trapped by the owners in bottom-hung windows between 2001 and 2012 were retrospectively evaluated. Exclusion criteria were as follows: cats with no initial neurological examination, cats that were dead on arrival, cats with entrapment of one leg only, cats that were trapped within the thoracolumbar area but without neurological deficits. The following clinical data were collected: breed, age, sex, last contact,

FIG 1 A bottom-hung window is a bottom-pivoting window that opens by tilting vertically, typically inside. It is also called a 'hopper window.'

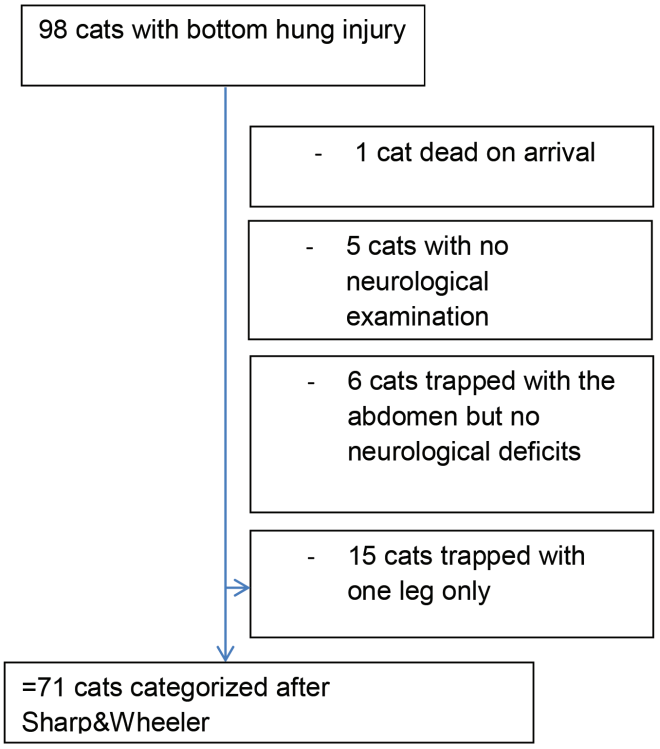

FIG 2 Exclusion criteria for evaluation of bottom-hung window trauma in cats; the final number of 71 cats represents cats that were trapped within the thoracolumbar abdominal area and with neurological deficits.

whether the cat was suffering from monoparesis or paraparesis/paraplegia, duration of hospitalisation, rectal temperature, surface temperature of the skin of the hindlimbs, femoral pulse and tone of the pelvic musculature. A rectal temperature of <38°C was defined as hypothermic and >39.3°C as hyperthermic. Surface temperature of the hindlimbs was subjectively evaluated by touch and graded as warm or cold skin surface. Femoral pulse quality was recorded as strong, weak (difficult to feel the pulse) or absent (no pulse detected at all). Muscle tone was classified as flaccid or spastic. Neurological status in paraparetic/paralysed cats was assessed based on deep pain perception, proprioceptive deficits, tibialis cranialis reflex, flexor reflex, patellar reflex, direct ischiatic reflex, anal reflex and muscular tail tone. All responses were categorised according to Scott's classification scheme: grade 1, pain on manipulation; grade 2, paraparesis and ambulatory; grade 3, paraparesis and non-ambulatory; grade 4, paraplegia; grade 5, paraplegia with loss of deep pain perception.[2] Neurological status was either assessed by the clinician at the time of admission and discharge or graded by the authors based on the neurological findings found in the patients' files. Follow-up data included neurological status at discharge, survival and incidence of euthanasia. Outcome (short term) was defined as neurological grade at 'time of discharge', euthanasia or death. No long-term outcome was obtained. The overall survival is reported for all cats presented with 'bottom-hung window' trauma in cats. Survival was defined as alive at discharge. Mortality was defined as either being euthanased or dying for different reasons. Causes of death are described.

Statistical analysis

All statistical analyses were performed with the SPSS Statistics software V.22 (SPSS Microsoft). Descriptive statistics, frequency tables and cross-classification tables with Fisher's exact tests were used to evaluate the relationships between clinical parameters (last contact with the owner, rectal temperature, surface temperature of the hindlimbs, femoral pulse, tone of the pelvic musculature) or results of neurological examination (neurological grade upon admission) and survival. Descriptive results are reported as median (minimum-maximum) values and proportions are reported with 95% CI where appropriate. Statistical significance was defined as $P<0.05$.

RESULTS

Ninety-eight cats diagnosed with 'bottom-hung window' trauma were found in the files. Cats with no neurological examination or diagnosis, dead on arrival and with monoparesis/monoplegia were removed, leaving a total of 71 cats (Fig 2) for analysis and classification after Sharp and Wheeler.[9]

Clinical characteristics

Cats evaluated were of two different breeds: European shorthair (n=69) and Persian (n=2). The median age was 2.4 years (0.16–19) with 52% less than two years. Sex/neuter status distribution was 19 entire females (27%), 16 spayed females (23%), 15 entire males (21%) and 21 neutered males (30%).

The time of owner's last contact with the cat was recorded for 27 (38%) cases, with 16 (23%) of those found within three hours. Median hospitalisation time was four days (0–13, Fig 2).

Rectal temperature was measured in 64 (90%) cats. Most cats (54; 84%) were hypothermic (<38.0°C). The median rectal temperature of the hypothermic cats was 35.1°C (32°C–37.8°C). The remaining 10/64 (16%) cats had a normal rectal temperature (≥38.0°C–39.3°C). Findings from surface temperature testing of the hindlimbs were subjectively recorded for 24 (34%) of the cats, and included warm surface temperature in seven (30%) and cold surface temperature in 17 (70%).

Femoral pulses were documented in 62 cats on admission, with 22 (35%) having normal femoral pulse, 20 (33%) having weak pulse and 20 (32%) having an absent femoral pulse. Findings from palpation of the hindlimb musculature were recorded for 16 cats (23%), with the majority deviating from normal as follows: spastic muscle tone, n=11 (69%); flaccid muscle tone, n=4 (25%); normal muscle tone, n=1 (6%).

Neurological findings and outcome

The abnormal spinal reflexes were located at the spinal cord segments L4-S2. The patellar reflex was the spinal reflex most commonly reduced or absent (38/51, 75%). The following reflexes were noted in the case files as absent upon admission: flexor reflex in 31/50 (62%); tibialis cranialis reflex in 30/40 (75%); proprioception in 37/51 (73%); anal reflex in 10/14 (71%); direct ischiatic reflex in 22/29 (76%); muscular tail tone in 6/11 (55%). The severity of injury in the 71 cats with paraplegia and/or paraparesis on admission was grade 5 in 44% of cats, grade 4 in 18%, grade 3 in 18%, grade 2 in 10% and grade 1 in 10% (Fig 2).

Cats with no deep pain perception at admission (grade 5, n=31) exhibited outcomes varying from death to full clinical and neurological recovery. Of the 17 cats (grade 5) that did not survive, six (19%) were euthanased at the owners' request on admission based on the clinical condition while 11 (36%) died within 1–96 hours of admission (median=four hours) with clinical signs of respiratory distress. The proportional mortality rate for cats with a neurological grade of 5 at admission was 55% (95% CI 36 to 72).

Of the 13 cats diagnosed with a grade 4 neurological status on admission, five did not survive (39%, 95% CI 15 to 68): one was euthanased on admission in accordance with the owners' request, one was euthanased after four days in the hospital (with pancreatitis) in accordance with the owner's request, one died within two hours of admission (with clinical signs of respiratory distress), one died after two days in the hospital (with clinical signs of respiratory distress), and one died after seven days in the hospital (with renal failure).

Of the 13 cats with grade 3 neurological status on admission, three died (23%, 95% CI 6 to 54) within four hours (two with clinical signs of respiratory distress; one because of renal failure). None of the cats with a grade 2 (n=7) or grade 1 (n=7) died.

A total of 17 cats died and eight underwent euthanasia at the owner's request, giving an overall mortality rate of 35% (95% CI 25 to 48). Sixteen of the 25 (65%) deaths were the result of respiratory distress of unknown origin: 11 cats (grade 5); two cats (grade 4); three cats (grade 3). Seven cats were euthanased on admission at the owners' request: six with neurological grade 5 and one with neurological grade 4. The Fisher's exact test shows a significant (P=0.007) relation between neurological grade and non-survivors (Table 1).

Table 1 shows the cross tabulations between the clinical parameters (femoral pulse, rectal temperature, last contact with the owner, warm surface temperature) and non-survivors. An abnormal quality of the femoral pulse, a low rectal temperature turned out to be a negative predictive value. Neither the 'last contact with the owner' nor the subjectively evaluated 'surface temperature of the

TABLE 1 Cross tabulations between non-survivors and clinical parameters. Significance was defined as P value <0.05.

Parameter		Survivors	Non-survivors	P value (Fisher's exact test)
Femoral pulse	Absent/weak	20	20	0.004
	Normal	19	3	
Rectal temperature	Low	30	24	0.006
	Normal	10	0	
Last contact with owner	<3 hours	13	3	0.279
	>3 hours	7	4	
Warm surface temperature	Cold	6	11	0.122
	Normal	5	2	

hindlimbs' is not significantly associated with non-survival.

DISCUSSION

The neurological grade upon admission significantly influenced the survival rate, with 46 (n=71; 65%) of the cats presenting with paraparesis/paraplegia surviving. All but two of the cats with neurological grade 5 that survived (12/14, 86%) improved to a better neurological grade at the time of discharge (median hospitalisation time: four days, range: 2–13). In this study, loss of deep pain perception upon admission was not a negative prognostic indicator for neurological recovery, but was for survival alone. The owner's decision for euthanasia was unlikely influenced by the potential of poor prognosis as the cats' condition worsened while in the hospital.

To our knowledge, this retrospective study is the first to be performed with a large number of cats that had presented at a single institution for this specific injury. Findings are in agreement with previous results and highlight potential mechanisms underlying the deterioration of affected animals. The following clinical signs were observed and confirm previous observations: low rectal temperature in 86% (54/71) and reduced or absent femoral pulse in 65% (40/71).[1] The paraplegia or paraparesis may have a multifactorial origin: ischaemia of the spinal cord due to compression of the main abdominal blood vessels and/or direct compression of the nerves and adjacent blood vessels leading to neuropraxia. The clinical presentation of this type of injury is similar to that of cats with systemic arterial thromboembolism. In both conditions, the blood supply to the spinal cord is interrupted and similar clinical signs develop (cold hindlimbs, no palpable femoral pulse, low body temperature), but the pathophysiology and extent of ischaemia is different.[3] Simple ligation of the distal aorta at the level of the iliac bifurcation has been shown in various experimental studies to have no effect on locomotion, indicating that paralysis depends on the presence of a blood clot in the aorta.[4] Experimental injection of 5-hydroxytryptamine (serotonin), a degradation product of the platelets, was also shown to lead to paralysis and other clinical signs similar to aortic embolism.[4] In another experimental study, changes in blood flow, oxygen tension, action potentials and vascular permeability were evaluated after ligating the aorta or the caudal vena cava in dogs.[5] When the aorta was clamped (ischaemic model), the blood flow in the grey and white matter of the spinal cord recovered within one hour. The blood flow after clamping the caudal vena cava (congestion model), however, did not recover and remained significantly reduced in grey and white matter ($P<0.05$). Feline spinal cords have been shown to tolerate approximately 30 minutes of severely reduced blood flow before recovery of energy metabolism is significantly impaired upon restitution of blood flow.[6] Based on outcome, entrapment in a bottom-hung window most closely resembles the experimental model of congestion induced by clamping of the vena cava. Within the time span of this retrospective study, one histopathological examination of the spinal cord was performed with evidence of spinal cord oedema and haemorrhage. Another cat that was seen after the time span of the retrospective study, and dying of respiratory distress after bottom-hung window trauma also showed haemorrhage and signs of congestion within the spinal cord at the border between the grey and the white matter. These were the only two cases where the spinal cord underwent a histopathological examination. One reason for the lack of histopathological results is that the central nervous system must be fixed in formalin within a few hours after death, otherwise too many artefacts appear within the nervous tissue.[7] Because of the low explanatory power, these two cases were not mentioned in the Results section.

The direct or indirect ischaemia of the femoral nerve and the responding muscle fibres may lead to an absent patellar reflex. One histopathological examination of a cat that was presented to us after this study showed degeneration of lumbar muscle fibres and oedema and haemorrhage around the ischiatic nerve diagnosed as ischaemic neuromyopathy. It seems that the vena cava and the aorta were compressed during the window entrapment. In 32% of the cats in our study the femoral pulse was not detectable, which may be indicative of arterial perfusion deficiencies in the hindlimbs. The ischaemic neuropathy or neuropraxia may have two components. The first one is the direct compression of the blood vessels and nerves resulting reduced perfusion of the vasa nervorum. The second one refers to the congestion model with oedema within the spinal cord.[8]

Out of the 25 cats that died in our study population, the majority (16/25, 64%) presented clinical signs of sudden respiratory distress. The lung is one of the main target organs in multiple organ dysfunction syndromes or multiple system organ failures caused by severe injury.[9 10] A potential cause for such damage is the enhanced generation of oxygen radicals, which has been observed in the development of ischaemia-reperfusion injury.[10] Ischaemia-reperfusion of the lower extremities causes lung injury by sequestration of polymorphonuclear neutrophils in pulmonary microvasculature, increased endothelial permeability and interstitial oedema.[10] Because of the similarity of pathophysiology, it is possible that reperfusion injury may have played a significant role in the genesis of respiratory distress in the presented cats.[9]

Among the cats in our study that presented with no deep pain perception on admission (grade 5) and survived, 86% (12/14) improved neurologically. A neurological grade 5 according to the Scott's classification system can serve as a prognostic factor in intervertebral disc diseases and traumatic insults of the spinal cord in small animals. Previous studies have suggested that success, defined as regaining deep pain perception and ability to walk, was obtained in 11% of the cases after traumatic injury of the

spinal cord and 58% in the cases of intervertebral disc disease.[11] Our study corroborates the previous findings, in that the prognostic value of the neurological grade on admission shall take into account the underlying cause.

Due to the retrospective character of this study, clinical and neurological examinations have been performed by different clinicians with various levels of experience. Entitled as observational study issues of prognosis, aetiology and risk factors were addressed. Nevertheless, the status in terms of evidence-based medicine in observational studies is unclear.[12] In a number of cases with paraparesis or paraplegia the neurological grade after Scott had to be reconstructed. This caries a certain risk of bias and generalisability with it. The causes of euthanasia on the owners' request were not reproducible. No note was found if it was the bad clinical or neurological condition of the cat or if it was due to cost restraint of the owner. The reasons for discharge or euthanasia with a high neurological grade were not traceable. Another drawback is the short-term follow-up time of these patients. Recovery times and progress of the neurological status would have to be assessed with a long-term outcome. A prospective study on cats with bottom-hung window injuries to gain a better understanding of this rare injury is planned.

CONCLUSION

The prognosis of cats after entrapment in a bottom-hung window is fair, with an overall survival rate of 65%. Mortality rate was influenced by the severity of neurological grade upon admission, resulting in a mortality rate of 55% with neurological status grade 5. In the survivors' group, paraplegia with loss of deep pain perception at admission was not a negative prognostic factor for regaining motor function. Owners should be aware of sudden respiratory distress syndrome as one of the main causes of death with bottom-hung window trauma.

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Competing interests None declared.

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Data sharing statement No unpublished data from the study are available to a third party unless requested and with allowance of the head of the University of Veterinary Medicine.

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