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**Original Article****Somatosensory and motor evoked potentials in dogs with chronic severe thoracolumbar spinal cord injury**

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## Highlights

- Somatosensory (SSEPs) and motor evoked potentials can be recorded following ‘complete’ chronic spinal cord injury in dogs.
- Development of ‘spinal walking’ is neither positively or negatively associated with intact evoked potentials.
- Scalp-recorded SSEPs are more sensitive than those recorded over the spine.

## Abstract

Some dogs that become paraplegic after severe spinal cord injury regain ambulation on the pelvic limbs despite permanent loss of pelvic limb sensation, a phenomenon termed ‘spinal walking’. Plastic changes in spinal cord circuitry are thought to mediate this form of recovery but the precise circumstances that favour its development are not known. More information on this phenomenon would be helpful because it might be possible to coax more function in chronically paraplegic animals so improving their, and their owners’, quality of life. We analysed the correlation of ‘spinal walking’ and pelvic limb pain sensation with recordings of scalp and spinal somatosensory and transcranial magnetic motor evoked potentials. We prospectively examined 94 paraplegic dogs (including 53 Dachshunds) that had sustained T10 to L3 spinal cord injury (including 78 dogs with acute intervertebral disc herniation) at a median time of 12.0 months from injury.

Nine dogs exhibited ‘spinal walking’ and nine other individuals had intact pelvic limb pain sensation. Of 34 tested, 12 dogs had recordable scalp somatosensory evoked potentials. Fifty-three of 59 tested dogs had recordable spinal somatosensory evoked potentials, but only six had recordable potentials cranial to the lesion. Twenty-two of 94 tested dogs had recordable transcranial magnetic motor evoked potentials in the pelvic limb(s). There was no

apparent association between intact evoked potential recording and either spinal walking or intact pain sensation. We conclude that factors other than influence, or lack of influence, of input carried by spinal cord long tracts mediate recovery of spinal walking.

*Keywords:* Canine; Electrophysiology; Physical therapy; Recovery; Spinal walking

ACCEPTED MANUSCRIPT

## Introduction

Spinal cord injury is common in pet dogs, mainly resulting from intervertebral disc herniation, fractures and vascular lesions (Moore et al., 2017). A small proportion of dogs, estimated to be approximately 16% of cases presented with acute intervertebral disc herniation (Granger et al., 2014), become paraplegic and lose sensation in the hindquarters. Of these, between 20 to 40% remain permanently unable to walk and, usually, also unable to control urination and defecation (Scott et al., 1999; Olby et al., 2003; Ito et al., 2005, Jeffery et al., 2016). Not only is more information required to aid owners in providing optimal care but these chronically-injured dogs have many similarities to chronically-injured humans and so constitute a model in which novel therapies can be tested. Data on baseline function may also aid stratification of participants in future human or veterinary clinical trials.

One intriguing aspect of chronic spinal cord injured dogs is that some develop so-called ‘spinal walking’ in which they regain ambulation, despite absence of recovery of sensation in the pelvic limbs. In experimental dogs, it has been established that pelvic limbs can generate a gait pattern that allows locomotion despite complete thoracolumbar spinal cord transection (Handa et al., 1986; Nato et al., 1990). Because it is rarely possible to ascertain whether the spinal cord is truly transected in clinical injuries, spinal walking in these individuals is defined by the loss of ‘deep pain perception’ in association with the ability to walk for a potentially unlimited period and the ability to regain a standing posture from recumbency (Gallucci et al., 2017).

At present it is uncertain what factors are important in promoting development of spinal walking. It is known that most dogs with experimental transection of the spinal cord can acquire this activity, although it may be considerably delayed from the time of injury

(Handa et al., 1986). In clinical cases, the extent of injury to the spinal cord is rarely known and so there is an uncertain relationship between severity of injury and development of spinal walking. A recent analysis suggested that spinal walking is associated with intact conduction through the descending motor tracts, as assessed by transcranial magnetic motor evoked potential (TMMEP) recordings (Lewis et al., 2017).

In the course of carrying out two randomized controlled trials on novel therapies for chronic spinal cord injury in pet dogs (Granger et al, 2012; Hu et al, 2018), we have acquired plentiful baseline data from which we can examine various hypotheses regarding the relationships between spinal walking and spinal ‘long tract’ conduction. Spinal walking is thought to be a consequence of increased activity in the segmental reflex pathways within the pelvic limb central pattern generator (Pearson, 2000; Raineteau and Schwab, 2001), implying that any residual input from descending tracts might make it less likely for spinal walking to occur. Therefore, we hypothesized that (1) spinal walking would be associated with failure to record TMMEPs in the pelvic limbs. Second (2), because both somatosensory evoked (SEP) and TMMEPs imply conduction through the spinal cord, we considered that dogs with pain perception would be more likely to have TMMEPs recordable from their pelvic limbs or SEPs recordable over the brain or spinal cord. Lastly (3), we thought that dogs with evidence of longer regions of spinal cord loss would be more likely to exhibit spinal walking (because, as a corollary of hypothesis 1, they would less likely have interference of descending influence on pattern generators controlling pelvic limb movements).

## **Materials and methods**

### *Dogs*

Participants were pet dogs that had been prospectively enrolled with owner consent

into one of two clinical trials of novel therapy for chronic severe spinal cord injury. In both trials, dogs had to fulfil the same inclusion criteria: (1) weight < 25 kg; (2) had sustained acute, traumatic T10 – L3 spinal cord segment injury; (3) otherwise healthy; and, (4) had failed to regain either ‘voluntary ambulation’, pain sensation, or both, in their pelvic limbs by at least 12 weeks after injury. ‘Voluntary ambulation’ was defined as being able to walk 10 consecutive steps unaided *plus* having evidence of pain perception in the pelvic limbs. However, dogs with some ambulatory ability – as defined in the clinical assessment section below – were not excluded from these trials, so long as they *did not* show evidence of conscious pain perception in the pelvic limbs. For inclusion, on pre-enrolment examination, each dog also had to have intact pelvic limb reflexes and normal range of motion in the pelvic limb joints when manipulated. The location of the lesion in each dog was known from neurological examination, imaging studies and surgical reports at entry to the study. The location was stated as the intervertebral disc space forming the epicenter of the lesion, based on imaging and surgical findings (*i.e.* attributed to one spinal cord segment between T10 and L3), although the histopathological spinal cord lesion would, in most cases, have extended further cranially and caudally along several spinal cord segments.

The data described here were acquired from enrolled dogs *before* they underwent any of the planned interventions examined in Studies 1 and 2.

#### *Clinical assessment*

Dogs were categorized according to whether they: 1) could ambulate on the pelvic limbs without support; and, 2) exhibited evidence of conscious perception of stimuli applied to the pelvic limbs or tail, up to and including intensely noxious pressure applied by pliers to the digits and tail. To be considered ‘ambulatory’ a dog had to be able to walk 10 consecutive

unaided steps on a concrete floor without falling or the lateral aspect of any part of the foot or metatarsals touching the ground. Each dog that was able to ambulate in this way also had to have no evidence of pain perception in the pelvic limbs or tail and was therefore referred to as a 'spinal walker'. It was also recorded whether dogs could walk between 1 and (no more than) 10 steps, or if they had some pelvic limb movement but no ability to walk, or if they showed no pelvic limb movement at all. 'Deep pain' response was considered intact if the animal consistently vocalized, turned the head to the source of the stimulus, or attempted to bite, in response to stimuli applied to the pelvic limb digits or tail.

Electrodiagnostic procedures were performed under sedation with 0.005 mg/kg dexmedetomidine (Zoetis) IV and 0.2 mg/kg butorphanol (Zoetis) IV. During the procedure, each dog was placed in sternal recumbency and routinely monitored until the end of the procedure, when 0.05 mg/kg atipamezole (Zoetis) was given IM to reverse dexmedetomidine and the dog was fully conscious and recovered their normal mobility. Brief information on recording methods are included below; additional detail is available in *Supplementary Material*.

#### *Somatosensory evoked potentials*

Somatosensory evoked potentials were recorded from the sensory cortex or vertebral column using standard methods (Poncelet et al., 1993; Inglez de Souza et al., 2017). In both studies, each tibial nerve immediately proximal to the hock joint was stimulated individually with a subcutaneous electrode using just sufficient intensity to elicit a minimally perceptible movement in the pelvic limb digits (*i.e.* just above motor threshold). Repetitive, rectangular impulses of 0.2 ms duration were then applied to the nerve at a frequency of 3.1 Hz and intensities varying from 0.2 to 1mA.



For spinal SEP recordings, the reference monopolar electrode was placed in the epaxial muscle 1 cm lateral to the recording electrode placed on the vertebral lamina ipsilateral to the tibial nerve stimulation. Recordings were commenced on each side at the cranial aspect of L6 and progressed cranially in steps of one vertebra until potentials could no longer be recorded; location, amplitude and latency of the potential at the cranial-most vertebra on each side were recorded for analysis (Fig. 1). We used the location of the most cranial recording site to calculate the number of spinal cord segments from that segment to the lesion epicenter (*i.e.* number of spinal cord segments ‘below’ the lesion). If recordings could be obtained above the lesion epicenter, we then calculated the number of spinal cord segments between the lesion epicenter and the most cranial recording (*i.e.* number of spinal cord segments ‘above’ the lesion), providing information on conduction across the lesion epicenter. The location of the most cranial recording site and the lesion epicenter were coded by attributing a number to each and the difference calculated; the code was based on the following references: T10-11 = -3; T11-12 = -2; T12-13 = -1; T13-L1 = 0; L1-2 = +1; L2-3 = +2; L3-4 = +3; L4-5 = +4 and L5-6 = +5; L6-7 = +6 (Fig. 1).

#### *Transcranial magnetic motor evoked potentials*

Transcranial magnetic motor evoked potentials (TMMEPs) were elicited as described previously (Poma et al., 2002; Nollet et al., 2003; Granger et al., 2012; Lewis et al., 2017; Hu et al., 2018) by stimulating the motor cortex using a 1 ms pulse at 70% stimulation intensity for all the dogs (cases in Study 1 and Study 2) and at 80% stimulation intensity for some dogs in Study 2. A circular magnetic coil was used for all dogs (Magstim 200, Whitland) held 1–2 cm from the skull. The right and left motor cortices were stimulated in turn and recordings

were made from each contralateral *cranialis tibialis* muscle. Low and high frequency filters were set at 30 Hz and 10 kHz respectively.

### *Analysis*

Onset latency for both long tract potentials was defined as the time difference between stimulation and onset of deflection from the baseline in either positive or negative direction. Waveform sequential peak-to-peak amplitude was defined as the difference between the two largest peaks of reverse polarity following the initial deflection from baseline. For spinal SEPs, we measured the distance in ‘number of spinal cord segments’ between the lesion epicenter (as known from imaging or surgery) and the most cranially recorded SEP (Fig. 1).

All variables were summarized using the median as a measure of central tendency (because some variables were not normally distributed) and the range. First, variables that might be expected to vary with dog size, such as latency of scalp SEPs and latency of TMMEPs, were examined using graphs and Pearson’s correlation coefficient for association with bodyweight. Secondly, we assessed: (1) association between spinal walking (yes/no) and recordable TMMEPs (yes/no) using Fisher’s exact test; (2) association between pelvic / thoracic limb coordination score (derived in both studies using methods described in Hamilton et al., 2007; Granger et al., 2012; Hu et al., 2018) and recordable TMMEPs (yes/no) using the Mann-Whitney *U* test; (3) association between detectable pain perception in the pelvic limbs (yes/no) and recordable TMMEP (yes/no) using Fisher’s exact test; (4) the number of spinal cord segments above or below the lesion epicenter at which a spinal SEP could be recorded compared between spinal walking dogs and non-ambulatory dogs using the

Wilcoxon signed ranks test. For all tests,  $P < 0.05$  was taken to indicate a significant association.

## Results

### *Dogs*

Demographic information for participating dogs is included in *Supplementary Material* and Table 1.

### *Somatosensory evoked potentials*

Scalp SEP recording was performed on 34 dogs of which 12 (35.3%) had recordable potentials (Table 2); none of these 34 dogs displayed spinal walking. Of the 12, six had recordable potentials bilaterally and six unilaterally (four on the left and two on the right), resulting in a total of 18 data-points available for analysis. Three dogs within this group of 34 dogs had intact deep pain sensation, the remainder did not, and two of these three dogs had recordable scalp SEPs. Latency ranged between 18.8 ms and 32.6 ms with a median of 26.8 ms. The distribution of recorded amplitudes was right-skewed (*i.e.* asymmetric with a long right tail of higher values), ranging between 0.12  $\mu\text{V}$  and 1.07  $\mu\text{V}$  with a median of 0.34  $\mu\text{V}$ . In the 12 dogs with recordable potentials there was no correlation between bodyweight and latency of scalp SEPs (Pearson's correlation:  $r = -0.03$ ;  $P = 0.91$ ).

Spinal SEPs were recorded from sites along the vertebral column in 53 dogs of 59 attempted (89.8%): seven unilaterally and 46 bilaterally, providing a total of 99 data-points available for analysis (Table 2). In six dogs, the spinal SEP could be recorded from a site cranial to the lesion epicenter (Table 3); five of these 6 animals did not show spinal walking and one did; one of these 6 animals had present pain sensation. Latency ranged between 2.7

ms and 21.2 ms with a median latency of 3.7 ms, when all the recording sites from L6 up to T10 were included (Table 2). Amplitudes ranged between 0.37  $\mu$ V and 16.3  $\mu$ V with a median amplitude of 1.31  $\mu$ V (Table 2). The specific latency results for each most cranial spinal cord segment recording (from L6 to T10) are presented in Table 3. Recordings obtained from L2 to L6 (representing 47 dogs with a recording below the level of the lesion) had median latencies ranging between 3.5 and 3.9 ms. For the six dogs with a recording obtained above the level the latencies ranged between 11.75 and 21.2ms. No spinal SEPs were recorded in any dog above T10.

#### *Transcranial magnetic motor evoked potentials*

Transcranial magnetic motor evoked potentials were recordable from the right *extensor carpi radialis* muscle (the intra-individual positive control) in all 94 dogs. All 94 dogs included in this study were stimulated at 70% of maximum intensity and TMMEPs were recordable in the pelvic limbs of 22 dogs (23.4%): 12 bilaterally and 10 unilaterally (Table 2). Thirty-seven dogs (from Study 2) were also stimulated at 80% of maximum intensity and TMMEPs were recordable in the pelvic limbs of 3 dogs out of 37 dogs (8.1%): 2 bilaterally and 1 unilaterally (Table 2). For these 37 dogs, a response was seen in 5 dogs (13.5%) at 70% intensity, similar to the response rate at 80% intensity. The latency distribution for dogs stimulated at 70% was right-skewed with a median of 52.8 ms and a range of 27.8 to 90.0 ms. Latency of the three dogs in which potentials were recorded at a stimulation intensity of 80% had a median of 45.2 ms and a range of 28.0 to 52.1 ms. Transcranial magnetic motor evoked potential latency (for the 94 dogs stimulated at 70%) did not correlate with body weight (Pearson's correlation:  $r = 0.14$ ,  $P = 0.26$ ).

#### *Relationships between evoked long tract potentials and residual behavioral function*

For dogs in which we recorded spinal SEPs from sites along the vertebral column ( $n = 53$ ) there were nine dogs that exhibited spinal walking. Although there was variation within both groups in the ‘effective length’ of the lesion as measured by comparing the cut-off site for recording the SEP and the epicenter of the lesion, there was no overall difference between those showing spinal walking and those that did not (Fig. 3; Wilcoxon signed ranks  $P = 0.59$ ). However, the sample size of spinal walkers was small, limiting the power of the analysis. Six dogs had spinal SEPs recorded above the lesion site but only one exhibited spinal walking. The lack of association between spinal walking and recording SEPs above the lesion or at the scalp is shown in *Supplementary Table 1*.

Table 4 shows the numbers of animals that had positive TMMEP recordings from the pelvic limbs and those that exhibited spinal walking. There was no apparent association between these variables (Fisher’s exact test,  $P = 0.68$ ). Similarly, Table 5 shows the relationship between an intact TMMEP and intact pain sensation. Fisher’s exact test suggests that there was no apparent association between these variables ( $P = 0.43$ ).

Finally, the thoracic / pelvic limb coordination score was compared with the presence or absence of recordable TMMEP recordings and no association was found (Mann-Whitney  $U$ ,  $P = 0.878$ ). The mean cumulative lag, representing the strength of coordination between the thoracic and pelvic limbs, had a median value of 2.21 and ranged from 0.49 and 3.39.

## **Discussion**

Our data show that in some dogs, even those with apparently ‘complete’ spinal cord injury (*i.e.* that have no clinical evidence of transmission across the lesion site), there is persistent passage of electrophysiological stimuli across the lesion. This was supported by

finding scalp-recorded SEPs in 12 of 34 dogs and TMMEPs in 22 of 94 dogs. Further, in those dogs in which spinal SEPs were recorded above the lesion, the latency of these potentials was markedly increased compared to those below the lesion, suggesting conduction deficits within the lesion. These findings are not unexpected: it has been known for many years that, in humans with apparently clinically complete lesions, conduction through the lesion can be deduced based on electrophysiological examination (Dimitrijevic et al., 1992). This study, along with those of Granger et al. (2012), Lewis et al. (2017) and Hu et al. (2018), confirms that this also occurs in dogs with similar lesions. An interesting point of comparison is that both motor and sensory potentials are typically difficult or impossible to obtain following acute spinal cord injury in dogs, even when spinal cord injury is incomplete (Shores et al., 1987; Holliday, 1992; Sylvestre et al., 1993), possibly because of ‘spinal shock’ during the acute phase of injury (Smith and Jeffery, 2005).

In general, electrophysiological recordings from the spinal cord and brain, especially averaged SEPs, are challenging to record and the results vary with different operators, equipment, patient and environment. Our data also suggest that recording of SEPs from the vertebral column above the level of the lesion is more challenging than recording from the scalp in chronic spinal cord-injured dogs. This might perhaps be because of an amplification effect on ascending impulses as they pass through various processing centres between the spinal cord and cerebral cortex (*i.e.* the difference between field potentials recorded over the brain *versus* ascending (moving) potentials along the vertebral column).

Our data do not support our hypothesis that spinal walking is associated with loss of TMMEPs; in fact, our data suggest that a recordable TMMEP has neither a positive nor negative association with spinal walking, implying instead that it develops independently of

whether there are connections of the central pattern generator with higher motor centers or not. Although not what we expected, this result is consistent with the extensive plastic rearrangement that can occur following spinal cord injury and supports the notion that each central nervous system is uniquely rearranged after injury (Bareyre et al., 2004) and highly dependent on spared spinal cord tracts (Jeffery et al., 2011). The competition between local and descending input to the central pattern generator might perhaps be expected to sometimes favour spinal walking and sometimes not. Lewis et al. (2017) demonstrated that the H-reflex was systematically detected in chronic paraplegic cases with a lower detection threshold than control dogs, confirming re-organisation of the lumbar spinal cord circuitry below the lesion.

Lewis et al. (2017) report a different relationship: that spinal walking was positively associated with intact TMMEPs, although in that study, as in ours, there is a possibility of statistical error because of the small numbers of spinal-walking dogs (five dogs in Lewis et al. (2017), nine dogs in our series). Of note, Lewis et al. (2017) recruited cases that could be *both* ambulatory and have pain sensation in the pelvic limbs and, indeed, one of the four cases with detectable TMMEPs had these characteristics, suggesting a less complete injury. In contrast, we specifically excluded cases that could both walk *and* feel pain. Further, Lewis et al. (2017) also included dogs with higher thoracic lesions (T3 to T9 in two cases), concurrent spinal lesions (C7-T1 in one case with T3-L3 clinical signs) and four with unknown localisation. We restricted our cases to lesions of the T10-L3 spinal cord segments, again suggesting a need for caution when comparing results between studies. In particular, high thoracic lesions might have very different effects on re-organisation of local spinal cord circuitry from those affecting the T10-L3 spinal cord region. As examples of the importance of the lesion localisation and possible association with spinal walking, Handa et al. (1986) reported that all dogs with an experimental T9 or T10 transections recovered spinal walking.

Blauch (1977) found that that spinal walking did not occur if the lesion was cranial to T13 and Lewis et al. (2017) detected no relationship between lesion site and recovery of spinal walking. In our analysis the two cohorts of dogs originated from different countries and it is possible that owner management of chronically paraplegic dogs systematically varied between studies, leading to variations in the occurrence of spinal walking in between study cohorts. Other geographically-associated factors, such as genetic background of these cases, might also play a role. On the other hand, our suggestion that the relationship between lesion severity and development of spinal walking is inconsistent is also supported by our finding that lesion length (approximated by the size of the gap in SEP recordings along the vertebral column to lesion epicentre) does not appear to be related to the development of spinal walking.

We found no relationship between intact TMMEPs and intact pain sensation. Again, this is slightly surprising, since it might be supposed that if an animal has a surviving segment of spinal cord that supports TMMEPs then it might also have enough spinal cord to support pain sensation. However, it is clearly possible that this supposition is incorrect because the regions of the spinal cord that support these two modalities do not necessarily get injured or preserved together. Various regions of the cord are available to support these different modalities and may be separated by quite large (in spinal cord terms) distances (~2 to 5 mm across the cross-sectional area of the spinal cord).

In all, our results suggest that none of the examined aspects of spinal cord injury have a detectable relationship with the likelihood of developing spinal walking. One factor that is difficult to examine is the possibility that a training effect may have over-ridden any of the other possible constraints on recovery of spinal stepping. It is well-established that training



locomotor activity after spinal cord injury will promote recovery of stepping in cats (Lovely et al., 1986; Barbeau et al., 1987) and there is evidence that this can happen in clinically-injured dogs (Gallucci et al., 2017). It may be that the owners of spinal walking dogs cared for their animals in environments that were especially conducive to recovery of this function, such as opportunity for free-ranging attempts at walking. On the other hand, a meta-analysis of clinical data from humans with spinal cord injury that underwent either body-weight-supported or robotic-assisted body-weight-supported treadmill training did not gain superior recovery of locomotion compared to those undergoing conventional physiotherapy (Morawitez et al., 2013).

## **Conclusion**

The remaining electrophysiological conduction through the long tracts in dogs with chronic spinal cord injury is highly variable. In this study a surprisingly high proportion of dogs exhibited evidence of long tract conduction, although there was little evidence to suggest that this provides benefits in terms of improved functional outcome to those individuals. Our data do not support an association of intact TMMEPs and spinal walking function in chronic paraplegic dogs. The evidence of conduction across the lesion in the chronic spinal cord injured dogs of this report suggests that there is potential to improve outcome through interventions that can promote beneficial plastic changes in circuitry. Intraspinal cell transplantation and chondroitinase injection have already been shown to improve locomotor function in these dogs but there is a need to augment such interventions to produce more robust clinical impact.

## **Conflict of interest statement**

None of the authors has any financial or personal relationships that could

inappropriately influence or bias the content of the paper.

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### **Supplementary material**

Supplementary data associated with this article can be found, in the online version, at doi: ...

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**Table 1**Demographic summary of the  $n = 94$  dogs in the study cohort.

<i>Sex</i>	Female	41 (44 %)
	Male	53 (56 %)
<i>Breed</i>	Dachshund	53 (56 %)
	Others (see result section)	41 (44 %)
<i>Cause</i>	Intervertebral disc herniation	78 (83 %)
	Vertebral column fracture	16 (17 %)
<i>Treatment</i>	Surgery	71 (76 %)
	Conservative	23 (24 %)
<i>Ambulation</i>	Non-ambulatory	85 (90 %)
	Ambulatory	9 (10 %)
<i>Deep pain</i>	Absent	85 (90 %)
	Present	9 (10 %)
<i>Age (year)</i>	Median (range)	6.0 (0.5 – 14.0)
<i>Weight (kg)</i>		6.6 (2.2 – 23.0)
<i>Time after injury (month)</i>		12.0 (3.0 – 89.0)

**Table 2**

Summary of scalp, spinal somatosensory (SEP) and transcranial magnetic motor evoked potential (TMMEP) results. Recording of scalp sensory evoked potentials was attempted in 34 dogs and recording of spinal sensory evoked potentials was attempted in 54 other dogs. Transcranial magnetic motor evoked potential recording was attempted in 94 dogs.

Technique	Recordable response	Recordable data points	Variable	Median	Range
Scalp SEP	12 of 34 dogs	18 (6 dogs unilaterally and 6 dogs bilaterally)	Latency (ms)	26.8	18.8 - 32.6
			Amplitude ( $\mu\text{V}$ )	0.34	0.12 - 1.07
Spinal SEP	53 of 59 dogs	99 (7 dogs unilaterally and 46 dogs bilaterally)	Latency (ms)	3.7	2.7 - 21.2
			Amplitude ( $\mu\text{V}$ )	1.31	0.37 - 16.3
TMMEP (stimulation intensity 70%)	22 of 94 dogs	34 (10 dogs unilaterally and 12 dogs bilaterally)	Latency (ms)	52.8	27.8 - 90.0
TMMEP (stimulation intensity 80%)	3 of 37 dogs	5 (1 dogs unilaterally and 2 dogs bilaterally)	Latency (ms)	45.2	28.0 - 52.

**Table 3**

Latencies of the most cranial recorded spinal somatosensory evoked potentials at each spinal level in the 53 dogs in which a recording was possible; for potentials recordable above the lesions (L1 to T10) - which represented six dogs - the latency for each dog is given; for potentials recordable below the lesion (L5 to L2) - which represented 47 dogs - the median latency and range in parenthesis are given.

<b>Most cranial level of recording</b>	T10	T11	T12	T13	L1	L2	L3	L4	L5
<b>Number of dogs</b>	2	1	0	1	2	9	14	15	9
<b>Recorded obtain above or below the level of the lesion</b>	Above	Above	None	Above	Above	Below	Below	Below	Below
<b>Latency (ms)</b>	14.2 and 17.6	21.2	None	15.8	11.7 and 15.3	3.8 (3.2-7.3)	3.6 (2.6-7.9)	3.5 (2.7-7.6)	3.7 (2.7-18.2)

**Table 4**

Association between spinal walking and recordable transcranial magnetic motor evoked potential (TMMEP) in 94 dogs.  $P = 0.68$ .

	<b>TMMEP Present</b>	<b>TMMEP Absent</b>
<b>Spinal walker</b>	1	8
<b>Non-ambulatory</b>	21	64

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**Table 5**

Association between detectable pain perception in the pelvic limbs and recordable transcranial magnetic motor evoked potential (TMMEP) in 94 dogs.  $P = 0.43$

	<b>TMMEP Present</b>	<b>TMMEP Absent</b>
<b>Deep pain positive</b>	3	6
<b>Deep pain negative</b>	19	66

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## Figure legends

Fig. 1. Schematic diagram depicting measurement of distance between cranial-most site of somatosensory evoked potential recording and lesion epicentre site. Measurements were made in units of one vertebra, with each vertebral space allocated a code starting from T13-L1 = 0, corresponding to the sites at which consecutive recordings were attempted. SSEP – somatosensory evoked potential.

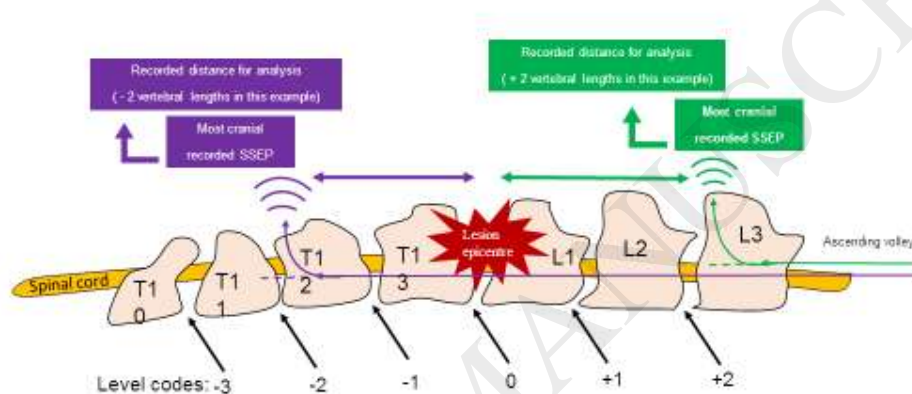
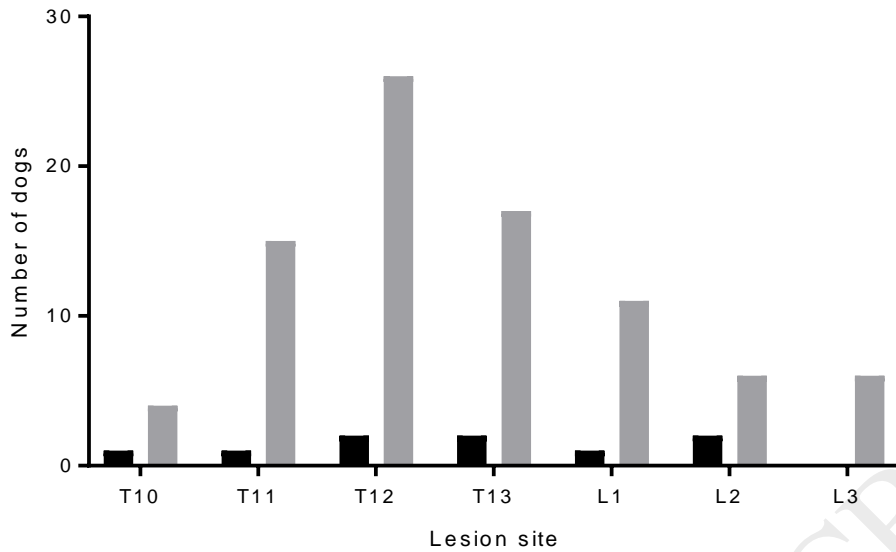
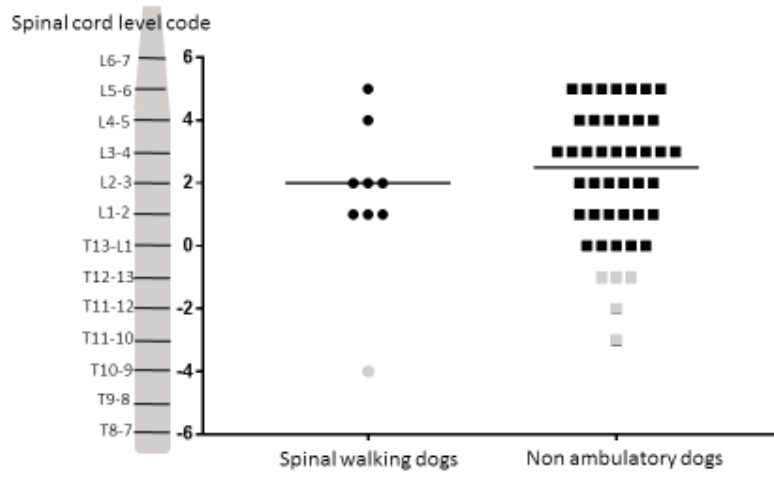


Fig. 2. Distribution of lesion epicentre in the 94 dogs, as determined from imaging and surgical observations. Grey bar indicates non-ambulatory dogs; black bars indicate dogs that exhibited 'spinal walking'.



**Figure 2**

Fig. 3. Comparison of the number of spinal cord segments above or below the lesion epicentre between spinal walking and non-ambulatory dogs. The '0' on the y-axis was allocated to the disc space T13-L1 which is depicted to the left of the graph by a sketch of the spinal cord with the caudal aspect of the cord at the top. The 'spinal cord level code' on the y-axis was obtained by subtracting the value attributed to the most cranial spinal somatosensory evoked potential obtained and the value attributed to the lesion epicentre. The dots depicted in grey represent the six cases where a spinal somatosensory evoked potential could be recorded above the letablesion epicentre.



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