

# Fibrocartilagenous embolism in 75 dogs: clinical findings and factors influencing the recovery rate

**The records of 75 dogs with fibrocartilagenous embolism of the spinal cord were evaluated retrospectively. The diagnosis was confirmed histopathologically in 21 dogs (group A) and remained suspected in 54 patients (group B). The two groups were compared. Particular emphasis was placed on the description of physiotherapy procedures, recovery rates and prognostic criteria. Results demonstrated that fibrocartilagenous embolism affected mainly middle-aged, large- or giant-breed dogs. Clinical signs were peracute in onset, non-progressive and often asymmetric. Cerebrospinal fluid analysis was normal in the majority of dogs. Intramedullary swelling was the only abnormality detected on myelograms of these patients. A positive correlation was found between a poor prognosis and the involvement of intumescences, symmetrical clinical signs and decreased deep pain sensation. However, physio/hydrotherapy instituted immediately after the diagnostic work-up seemed to have a major influence on the recovery rate.**

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*Journal of Small Animal Practice* (2003)  
**44**, 76–80

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## INTRODUCTION

Fibrocartilagenous embolism was reported for the first time in man in 1961 (Naiman and others 1961) and in the dog in 1973 (Griffiths 1973). Many additional canine cases have since been published in the veterinary literature (Zaki and others 1974, deLahunta and Alexander 1976, Hayes and others 1978, Chick, 1979, Bichsel and others 1984, Gilmore and deLahunta 1987, Penwick 1989, Dyce and Houlton 1993, Cauzinille and Kornegay 1996). Other species such as the cat (Zaki and Prata 1976, Bichsel and others 1984), horse (Taylor and Vandeveld 1977), and pig (Tessaro and others 1983) appear to be much less frequently affected.

Fibrocartilagenous embolism is definitively confirmed by neuropathological examination of the spinal cord. A characteristic finding is the presence of fibrocartilage in the lumen of spinal cord arteries or veins, which causes ischaemic or haemor-

rhagic infarction of the spinal cord. These particles of cartilage are histochemically identical to the nucleus pulposus (Bichsel and others 1984). Various theories have been proposed to explain the way in which occlusion of vessels occurs (Penwick 1989, Cauzinille 2000). Controversies exist about the pathophysiology of the process, and the exact mechanism is still poorly understood. The diagnosis of fibrocartilagenous embolism in cases which recover from acute paralysis, caused by a focal, non-progressive and non-traumatic lesion of the spinal cord, remains presumptive. The main characteristic finding in such patients is the lack of compressive features of the spinal cord during myelography.

The purpose of this study was to retrospectively evaluate the clinical, radiographic and neuropathological aspects of 75 dogs diagnosed with fibrocartilagenous embolism of the spinal cord. Particular attention was directed towards the physiotherapeutic management and follow-up of the patients.

## MATERIALS AND METHODS

The clinical records of 75 dogs with a diagnosis of fibrocartilagenous embolism of the spinal cord were retrospectively analysed. All of the patients had been referred to the Animal Neurology Section of the University of Bern between January 1978 and February 2001. The following parameters were evaluated: breed, age, gender, onset and course of the clinical signs, discomfort immediately after disease onset, level of motor dysfunction (paresis/paralysis), symmetry of the clinical signs, localisation of the lesion and the presence or absence of deep pain perception.

A complete blood cell count (CBC) and biochemical profile (electrolytes, phosphorus, glucose, cholesterol, total protein, albumin, blood urea nitrogen, creatinine, bilirubin and liver enzymes [creatinine kinase]) were performed in 66 dogs (88 per cent). Cerebrospinal fluid (CSF) analysis, including the Pandy reac-

**Table 1. Signalment of dogs with fibrocartilaginous embolism in groups A and B**

	Age	Gender		Size		
		Male	Female	Small breed (0-10 kg)	Medium breed (10-25 kg)	Large/giant breed (>25 kg)
Group A	5m – 9y (median 5y)	13 (61.9%)	8 (38.1%)	0 –	4 (19.1%)	17 (80.9%)
Group B	1y – 11y (median 6y)	34 [1 neutered] (62.9%)	20 [10 spayed] (37.1%)	8 (14.8%)	6 (11.1%)	40 (74.1%)

y Years, m Months

**Table 2. History of physical activity and pain at the onset of clinical signs and progression of clinical signs in dogs with fibrocartilaginous embolism**

	Physical activity		Pain		Progressive signs	
	Yes	No/not reported	Yes	No	Yes	No
Group A	9 (42.8%)	12 (57.2%)	1 (2.2%)	20 (97.8%)	8[6†] (38%)	13 (62%)
Group B	26 (48.1%)	28 (51.9%)	8[7*] (14.8%)	46 (85.2%)	6† (11.1%)	48 (88.9%)

\*Pain detected only immediately after the onset of the clinical signs  
 †Progression of signs within the first two hours

tion and total cell count as well as cytomorphological evaluation, was carried out in 58 cases (77 per cent). Survey radiographs of the spine and myelography were performed in 72 cases (96 per cent). The course of the disease and recovery rates were analysed. Dogs which were euthanased at the owner's request underwent postmortem examination and histopathological examination of the spinal cord. The spinal cord was removed and fixed in 10 per cent buffered formalin. Cross sections taken at several levels were embedded in paraffin wax, cut at 4 µm, stained with haematoxylin and eosin, and evaluated.

Dogs were divided into two groups: histopathologically confirmed cases (group A; n=21) and suspected cases without histopathological confirmation (group B; n=54). In all cases in group A, ischaemic or haemorrhagic spinal cord infarction was found on histopathological examination. The suspected cases were defined as follows: clinical signs compatible with acute, non-progressive and non-traumatic focal spinal cord disease and absence of a compressive lesion on myelography. Recovery was defined as the ability to regain spontaneous, physiological paw positioning while standing and walking. Occasional overknuckling and slight incoordination of the affected limbs were considered acceptable.

### Statistical analysis

Data were analysed by use of a commercial statistical software package (Statistica for Windows 4.5; StatSoft). The dichotomous variables (presence/absence of physical activity at the onset of the disease; presence/absence of discomfort immediately after disease onset; presence of paralysis/paresis; symmetry/asymmetry of the clinical signs; progression/non-progression of the clinical signs; upper motor neuron/lower motor neuron signs; presence/absence of deep pain sensation; presence/absence of intramedullary swelling; normal/abnormal CSF) were analysed using the McNemar Chi-square test in order to evaluate significant differences between group A and B.

Descriptive statistical analysis (mean± standard deviation [SD]) was made based on the follow-up findings in 49 dogs. Wald-Wolfowitz Runs test was performed to

compare the recovery time between the subgroups of dogs with different neuroanatomical localisations of the lesions (cervical, cervicothoracic, thoracolumbal and lumbosacral), with different severity of gait abnormalities (paresis and paralysis) and with the presence/absence of intramedullary swelling on myelograms. Differences were considered significant when P<0.05.

## RESULTS

### Signalment and history

Age, sex and breed data are summarised in Table 1. Severe gait abnormalities started peracutely in all cases. Strenuous physical activity immediately preceded the onset of clinical signs in less than half of the patients from both groups. The presence of brief, concomitant pain was noted more often in dogs of group B, but was not significantly different between the two groups. Progression of the clinical signs occurred mainly during the first two hours after their onset and was observed significantly more often in dogs of group A (P<0.01) (Table 2).

### Neurological examination

All dogs underwent a complete clinical and neurological examination. Motor dysfunctions were observed in all dogs but there was no statistical difference between the two groups (Table 3). Asymmetry of clinical signs was more often noted in group B (Table 4). Monoparesis or monoplegia were present only in group B (Table 3).

**Table 3. Impairment of motor function in dogs with fibrocartilaginous embolism**

	Group A		Group B	
	Number of dogs	Percentage	Number of dogs	Percentage
Monoplegia*	0	0	6	11.1
Paraplegia	7	33.4	12	22.2
Tetraplegia	5	23.8	2	3.7
Hemiplegia	2	9.5	5	9.3
Monoparesis*	0	0	7	13
Paraparesis	5	23.8	20†	37
Tetraparesis	2	9.5	2	3.7

\*Monoplegia and monoparesis in all cases refer to monoplegia or monoparesis of one hindlimb  
 †Eight dogs had asymmetric signs: paraparesis and monoplegia of one hindlimb

**Table 4. Neurological examination findings in dogs with fibrocartilaginous embolism**

	Symmetry of clinical signs*		Deep pain†		UMN/LMN signs	
	Symmetric	Asymmetric	Present	Decreased/absent	UMN	LMN
Group A	9 (45%)	11 (55%)	6 (33.3%)	12 (66.7%)	5 (23.8%)	16 (76.2%)
Group B	19 (35.2%)	35 (64.8%)	49 (90.7%)	5 (9.3%)	23 (42.6%)	31 (57.4%)

\*Not recorded in one dog from group A  
†Not recorded in three dogs from group A  
UMN Upper motor neuron, LMN Lower motor neuron

**Table 5. Neuroanatomical localisation of the lesion in dogs with fibrocartilaginous embolism**

	Group A		Group B	
	Number of dogs	Percentage	Number of dogs	Percentage
Cervical (C1-C5)	2	9.5	3	5.6
Cervicothoracic (C5-T2)	7	33.4	6	11.1
Thoracolumbar (T3-L3)	3	14.3	20	37
Lumbosacral (L3-S3)	9	42.8	25	46.3

Lower motor neuron signs (decreased or absent spinal reflexes) were more frequently, but not significantly, noted in group A (Table 4). Decreased or absent deep pain sensation was highly significantly associated with group A dogs ( $P < 0.0001$ ).

The lumbosacral intumescence (L3-S3) was most frequently affected in both groups of dogs (Table 5). Neuroanatomical localisation of the lesion to the cervical region or spinal cord intumescences was more often observed in group A dogs. Neuroanatomical localisation of lesions cranial to T2 was significantly more common in group A dogs ( $P < 0.05$ ) (Table 5).

### Clinical pathology and myelography

● **Group A.** No significant blood abnormalities were found in any of the dogs tested. CSF examination was performed in 12 patients and was abnormal in five cases (41.7 per cent). These dogs showed a marked positive Pandy reaction (2+ to 3+). Only one dog had mild mixed pleocytosis. Survey radiographs of the vertebral column were normal in all examined patients. Myelography revealed no abnormalities in 11 dogs (61.2 per cent) and thinning of the contrast columns (intramedullary swelling) in seven cases (38.8 per cent).

● **Group B.** No significant blood abnormalities were found. CSF examination was

performed in 45 dogs and was normal in 43 (95.6 per cent). Abnormal findings consisted of mild mixed pleocytosis with a slight positive Pandy reaction (1+). Survey radiographs of the vertebral bodies were normal or showed mild unrelated abnormalities in all dogs. Myelography was normal in 40 dogs (74.1 per cent). Intramedullary swelling at the presumptive site of the lesion was recorded in 14 cases (25.9 per cent).

CSF abnormalities were significantly more frequent in dogs of group A compared to group B ( $P < 0.001$ ). Myelographic findings did not differ significantly between the two groups.

### Physiotherapy and follow-up

Physiotherapy and hydrotherapy were instituted as soon as possible after the clinical work-up of the patients and a pre-

sumptive diagnosis of fibrocartilaginous embolism had been made. Physiotherapy was performed at the authors' clinic in the majority of cases. It was usually started within the first 24 to 48 hours after disease onset and the physiotherapy programme was tailored according to the needs of the individual patients.

Passive joint mobilisation (warming) was performed in lateral recumbency or in the standing position, using support from a chest/bellyband in plegic or severely paretic animals. Muscle tone regulation (reduction or increase) and massages followed, and lasted for at least 10 to 15 minutes, three to five times per day. In addition, sitting or laying in sternal recumbency or standing with support was always encouraged in such patients. All severely affected dogs underwent hydrotherapy for 10 minutes at least twice per day. In plegic patients, hydrotherapy was combined with passive limb movements in the water. Dogs with decreased or absent deep pain perception and lower motor neuron signs were treated with electrical stimulation of the affected limbs.

Dogs with mild paresis were taught to sit and stand in a physiological position, and physiological gait training with guiding support using the tail or with the help of a bellyband was performed five times per day. Manual massages and muscle tone regulation were always added when considered necessary.

All 21 dogs from group A were euthanased at the owners' request. Conversely 36 dogs (73.4 per cent) from group B showed a quick and almost complete

**Table 6. Group B follow-up: correlation between recovery time and lesion localisation, impairment of motor function and myelographic findings**

Recovery (days)	Total numbers of dogs (%)	Localisation		Gait abnormalities		Myelography	
		LMN	UMN	Paresis	Plegia	Normal	Swelling
0-7	20 (40.8%)	11 (55%)	9 (45%)	15 (75%)	5 (25%)	16 (80%)	4 (20%)
8-14	16 (32.6%)	10 (62.5%)	6 (37.5%)	5 (31.2%)	11 (68.8%)	9 (56.3%)	7 (43.7%)
>14	13 (26.6%)	7 (53.8%)	6 (46.2%)	7 (53.8%)	6 (46.2%)	10 (76.9%)	3 (23.1%)

LMN Lower motor neuron, UMN Upper motor neuron

**Table 7. Group B follow-up: correlation between recovery time and neuroanatomical localisation of the lesion**

	Number of dogs	Percentage	Recovery (days) (mean±SD)
Cervical (C1-C5)	3	6.2	22±5.6
Cervicothoracic (C5-T2)	5	10.2	14.2±9.3
Thoracolumbar (T3-L3)	18	36.7	8.6±5
Lumbosacral (L3-S3)	23	46.9	12.6±11.2

recovery within two weeks, while in 13 cases (26.6 per cent) it took from 15 to 45 days until an unassisted diagonal gait pattern was achieved (Table 6). One tetraplegic dog from group B was euthanased at the owner's request because no improvement was seen, but necropsy was not performed. Four dogs were lost to follow-up. The mean recovery time was 12 days (±9.77).

Lesion localisation, involvement of the intumescences and intramedullary swelling on myelography did not significantly influence the time of recovery of the 49 dogs for which follow-up results were available (Tables 6 and 7). Furthermore, the severity of motor dysfunction did not significantly correlate with the time of recovery (Table 8).

### Neuropathology

The lesions of fibrocartilaginous embolism are those of a spinal infarction, and definitive diagnosis depends on identification of cartilaginous thrombi in meningeal and/or spinal vessels. Since all the lesions noted in the present study were quite similar, they are reported here as general features.

In all the confirmed cases, the spinal cord grossly showed extensive necrotic areas, which, in many cases, were more severe on one side. The parenchyma was soft and pale grey or brownish-red. Histologically, white and grey matter as well as spinal nerve roots were involved. In selected cases, severe malacia with large macrophage sheets, embedded in a vascular network, was present. Haemorrhages may occur. In completely necrotic areas widely scattered parenchymal debris, even without macrophages, was left. The affected areas of spinal cord were sharply demarcated from less affected areas, which could show a myelopathy with swollen myelin sheaths and axons. The diagnostic hallmark was vessels in the meninges and/or the damaged parenchyma which contained compact emboli readily identifiable as cartilage.

**Table 8. Group B follow-up: correlation between recovery time and impairment of motor function**

	Number of dogs	Mean recovery time (days)
Monoparesis	7	6.9±5.40
Monoplegia	6	14.1±15.32
Paraparesis	17	11.2±10.16
Paraplegia	11	10.8±4.17
Hemiplegia	5	17±8.69

Two dogs with tetraparesis and one dog with tetraplegia are excluded from this table

## DISCUSSION

Comparisons of histopathologically confirmed and non-confirmed cases of fibrocartilaginous embolism have been made previously (Gilmore and deLahunta 1987, Cauzinille and Kornegay 1996). Patients seen by Cauzinille and Kornegay (1996) were compared with the data from previously published studies. By contrast, this study presents the results of a considerable number of cases of fibrocartilaginous embolism, with a complete work-up, all performed at the same institution. Other published reports usually refer to a single or a few histopathologically confirmed cases (Zaki and others 1974, deLahunta and Alexander 1976, Zaki and Prata 1976, Hayes and others 1978, Gill 1979, Doige and Parent 1983, Bichsel and others 1984, Penwick 1989, Dyce and Houlton 1993).

Fibrocartilaginous embolism is commonly reported in large- and giant-breed dogs. Neer (1992) stated that 71.8 per cent of all the confirmed cases reported in the veterinary literature up until 1992 were large-breed dogs. In the present study, 80.9 per cent of confirmed cases, and 74.1 per cent of suspected cases were large or giant breeds. One explanation for the prevalence of this condition in large-breed dogs could be that the nucleus pulposus remains soft for a longer period in these dogs and therefore may be more prone to mechanical vascular injection (Neer 1992). The prevalence of fibrocartilaginous embolism in adult and older patients (suspected, average age six years old; confirmed, average age five years old) supports the existence of other factors, such as ageing of the annulus fibrosus or its fissuration, which could promote intravascular herniation of material (Neer 1992, Cauzinille and Kornegay 1996).

A peracute onset of clinical signs in dogs with fibrocartilaginous embolism seems to be an important clue for the clin-

ician. The present case series supports this notion. Physical activity appears to play a considerable role in the development of the condition and was reported in almost half of the present cases. Most of the dogs, either confirmed or suspected, showed no discomfort or pain during the onset of clinical signs. Progression of the symptoms in these patients was recorded in the first one or two hours after the onset of the disease and was noted significantly ( $P<0.01$ ) more often in dogs which were later euthanased. Progression could be related to the expanding ischaemia and its sequelae (eg, oedema) in adjacent areas of the spinal cord. Nevertheless, progression of clinical signs per se did not indicate a poor prognosis, as shown by six cases in group B. Late progression of clinical signs was not observed in the present case series. Therefore, secondary ascending and/or descending myelomalacia seems to be unlikely in dogs with fibrocartilaginous embolism, even if severe damage to the spinal cord has occurred.

The authors' findings support previous observations that the neurological deficits are usually consistent with a focal lesion of the spinal cord. Moreover, asymmetry of clinical signs suggests partial ischaemic damage of the spinal cord at one level. This could be explained by the particular branching of the intrinsic spinal vasculature (Caulkins and others 1989). Interestingly, all the monoplegic dogs in this case series recovered. This data might suggest that unilateral spinal cord damage is connected with better recovery. Alternatively, transverse lesions and consequent symmetrical signs might suggest more severe involvement of the spinal cord.

Several authors have stated that lower motor neuron signs are related to a poor prognosis (Gilmore and deLahunta 1987, Neer 1992, Cauzinille and Kornegay 1996). Involvement of the spinal cord intumescences in a considerable number of the confirmed cases in the present series

supports this observation. Nevertheless, the frequency of upper and lower motor neuron signs was not statistically different in the two groups, and recovery occurred in dogs with both lower and upper motor neuron signs in this series. Extension of the lesion seems to be a more important prognostic factor than its localisation. Absence of deep pain perception was significantly associated with dogs which were later euthanased and was most probably associated with severe and bilateral damage of the grey and white matter. Absent deep pain perception was, in the authors' experience, the most important negative prognostic parameter observed.

The results of CSF examination and myelography confirm previous reports that both procedures offer low sensitivity in patients with fibrocartilaginous embolism (Dyce and Houlton 1993). Interestingly, CSF abnormalities were significantly related to group A dogs. Abnormal Pandy test results and pleocytosis could reflect a disruption of the blood-brain barrier and might be an indicator of more severe damage to the spinal cord. Intramedullary swelling was noted much less frequently in the present dogs than in previous reports (Dyce and Houlton 1993, Cauzinille and Kornegay 1996). Intramedullary swelling is most likely to be the result of spinal cord oedema or haemorrhage as a consequence of the embolism (Cauzinille 2000). If this is true, a larger swelling could mean a more severe spinal cord lesion. The present results do not confirm such a conclusion as intramedullary swelling of the spinal cord was noticed in almost similar proportions in groups A and B and was not significantly related to a longer recovery time. Although myelography is not specific for fibrocartilaginous embolism, it can rule out peracute compressive lesions in suspected cases. Magnetic resonance imaging offers more scope than myelography for defining the intramedullary lesions (Cauzinille 2000).

Several authors have reported that fibrocartilaginous embolism has a guarded to poor prognosis (Penwick 1989, Dyce

and Houlton 1993). In the present study, recovery occurred in 90.7 per cent of the suspected cases in group B, and in 65.3 per cent of the whole study population. Moreover, follow-up data indicate that a consistent percentage of dogs recovered within two weeks (73.4 per cent). The authors are convinced of the important role of physiotherapy in returning dogs affected with fibrocartilaginous embolism to a normal or near normal gait. The absence of surgical wounds and pain in dogs with this disease allows immediate, unlimited physiotherapy, free of complications, right from the onset of signs. In the authors' opinion, dogs with fibrocartilaginous embolism are ideal patients for the physiotherapist.

### Conclusions

In the past, fibrocartilaginous embolism was considered to be a more severe disease than it is now. Rather than being considered a disease that primarily involves the spinal cord intumescences or is associated with poor prognosis, the present results show that a patient with non-progressive, asymmetric signs, deep pain perception and no evidence of spinal cord swelling on myelography, has a relatively good prognosis and will most probably recover completely during the first weeks after the onset of signs. Conversely, signs associated with a guarded to poor prognosis might be symmetrical lower motor neuron signs, decreased or absent deep pain sensation and detection of CSF changes. The present results suggest that the absence of deep pain sensation is the most important prognostic factor.

Active nursing and intensive and early physiotherapy should be applied in as many cases of suspected fibrocartilaginous embolism as possible, as the authors are convinced that this plays a crucial role in the positive outcome of the disease. By aiding early recovery of dogs with symptoms suggestive of fibrocartilaginous embolism, the use of physiotherapy could be associated with lower treatment costs and fewer secondary complications.

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