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Listeriosis

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Listeriosis is a bacterial infection primarily caused by the serovar, *Listeria monocytogenes*. Most commonly, it affects cattle and small ruminants, but it is known to cause disease in humans, horses, swine, rabbits, and poultry.¹ Clinical signs are dependent on the clinical form of the disease. This issue of *Large Animal Veterinary Rounds* discusses the different forms, clinical signs, and diagnosis of listeriosis; in addition, cases are presented to illustrate these points.

Etiology and pathogenesis

There are seven species of *Listeria*: *Listeria grayi*, *L. innocua*, *L. ivanovii*, *L. monocytogenes*, *L. murrayi*, *L. seeligeri*, and *L. welshimeri*. Of these, only *L. monocytogenes* and *L. ivanovii* are of clinical importance. There are 16 serovars that are not species-specific.^{2,3} *Listeria* is a small, pleomorphic, Gram-positive rod. It is a facultative intracellular pathogen that multiplies within monocytes and macrophages. The organism is ubiquitous in the environment and is found in soil, silage, feed, manure, and as part of the normal flora in the distal gastrointestinal tract of humans and animals.^{2,3} *Listeria* proliferates at temperatures ranging from 3°C to 45°C in aerobic and microaerophilic environments and produces optimal growth at temperatures between 30°C and 37°C.³ It can grow within a pH range of 5.6–9.6;^{2,3} however, growth is absent in anaerobic environments and when the pH is < 5.6.³ *Listeria* can survive short-time pasteurization, is resistant to freezing, thawing, and desiccation, and survives for years in organic material with a neutral pH.

Mechanism of disease

There are two proposed mechanisms of disease:

- In the first mechanism, *Listeria* is ingested and, once in the intestinal tract, the organism multiplies without causing any clinical signs or it invades epithelial and phagocytic cells, multiplies within these cells and then invades neighbouring cells.² It can also multiply in hepatic and splenic macrophages using a hemolysin (listeriolysin O) to lyse the phagosomal membrane, thereby avoiding intracellular death.^{2,4}
- In the other mechanism, the organism enters through damaged mucosal surfaces, by inhalation, or by conjunctival contamination. It then invades the central nervous system (CNS) by traveling along the trigeminal nerve sheath.^{2,3,5,6} This mechanism was demonstrated by experimental injection of *Listeria* into the lips of goats.⁵ This second route of infection usually results in meningoencephalitis.⁶ However, there has been no evidence for tropism of the organism to neural tissue and it is unknown whether the organism can use nerves other than the trigeminal nerve to access the brain.³ The incubation period for *L. monocytogenes* infections is usually 2–6 weeks.^{1,3,6}

Transmission

Animals are exposed by ingestion, inhalation, or direct contact with the *Listeria* bacterium.^{2,4} The organism has been found in water, birds, bedding, animal feed, poorly prepared silage, feces, urine, aborted fetuses, uterine discharge, and milk.^{2,4} Food-borne transmission is the most common mode of transmission³ and *Listeria* is considered a food-borne pathogen for humans and animals.^{7,8} In animals, *Listeria* is most frequently transmitted by contaminated silage.^{9,10} It is most often found in silage that is poorly fermented with a pH \geq 5.6,^{9,10} in moldy silage,⁹ or in silage spoiled with soil.^{5,10}



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The organism is typically found near the top and sides of the silage pack where fermentation is inadequate² and these portions should not be fed to animals.

Listeriosis is a primary cause of disease in cattle and sheep. Young and old animals are often more severely affected, possibly due to impaired immune competency.² *Listeria* usually causes sporadic cases of disease, but outbreaks do occur when multiple animals are exposed to a single contaminated source.² Clinical listeriosis is seasonal in occurrence; most commonly occurring between January and May and, usually, secondary to management changes (eg, silage feeding, winter housing, and increased environmental contamination due to more confined housing).¹¹ The condition is rare in animals < 6-weeks of age. The morbidity rate of exposed ruminants is 0.2%–8%² and case-fatality rates for animals with clinical signs vary from 20%–100%.⁷ Reported mortality rates in silage-fed ewes exposed to *L. monocytogenes* are 3.1% to 12.2%, with almost 100% mortality in untreated cases. In humans, listeriosis is rare, but it can be severe with mortality rates approaching 50%.³

The on-farm prevalence of *L. monocytogenes* has been found to differ between small ruminant and cattle farms, revealing a higher prevalence on cattle farms.¹⁰ In one study, the prevalence of *L. monocytogenes* on cattle and small ruminant farms with clinical cases (case farms) was 27.3%, while on farms without clinical cases (control farms), the prevalence was 14.1%.¹⁰ In general, case farms had an increased prevalence in feces, soil samples, and feed samples when compared with control farms. The prevalence in cattle feces was found to be higher than in feed or soil, suggesting that cattle may serve to amplify the organism and may be a source of environmental contamination and a natural reservoir.¹⁰ Furthermore, it was found that the prevalence in soil was higher than in feed, suggesting that soil is a possible source of feed contamination.¹⁰

Cattle are frequently exposed to contaminated feed, but are less likely to develop disease unless they are compromised by stress (eg, pregnancy, overcrowding, transport, environmental conditions) or exposed to very high concentrations of *Listeria*.^{2,10} The serovars of listeriosis isolated from cattle farms in this study included those found in human listeriosis cases, suggesting that cattle are a possible reservoir for human infection.¹⁰ In general, on small ruminant farms, the prevalence in feed was higher than in feces, suggesting that small ruminants are less likely to amplify *Listeria*;¹⁰ however, after an outbreak, fecal shedding can continue for 3 months in sheep.⁵ Studies have found up to 50% of fecal samples from animals (cattle, sheep, goats, pigs, and poultry) without clinical listeriosis contain *L. monocytogenes*.¹⁰ In summary, *Listeria* is transmitted by the ingestion of contaminated feed with the subsequent amplification of the organism by clinical and nonclinical carriers, and is shed into the environment via feces.¹⁰

Clinical forms, diagnosis and treatment

Clinical listeriosis generally presents as 1 of 3 forms:

- neural (meningoencephalitis)

- visceral (septicemia)
- reproductive (uterine infections resulting in late term abortion).^{1,7,9,11,12}

Listeria can also cause mastitis^{1,11} and metritis,¹ as well as iritis and keratoconjunctivitis.¹¹ Encephalitis and reproductive disease forms are the most common clinical presentations.³

The neural form

The neural form of listeriosis is usually associated with *L. monocytogenes* and occurs sporadically, affecting a single animal in a herd or flock. It is referred to as a “circling disease” of cattle, sheep, and goats due to the effects of encephalitis and meningitis (see Cases 1 and 3).^{2,3} Clinical signs depend on the area of the CNS affected. In one study,¹² the most common clinical signs were:

- Goats: locomotion disorders, opisthotonus, and circling
- Sheep: locomotion disorders, saliva production disorders, and abnormal head position
- Cattle: locomotion disorders, saliva production disorders, and facial nerve paralysis.

Of the 42 animals (15 goats, 12 sheep, and 15 cows) in this study, 38% had locomotion disorders, 28.5% had saliva production disorders, 21.4% had facial nerve paralysis, and 21.4% were circling.¹² In small ruminants, disease progression to recumbency usually results in death within 2–3 days.^{2,3} In cattle, initial neurologic signs are usually unilateral, but the contralateral side will eventually become affected.⁶ The clinical course is usually 1–2 weeks, with eventual recumbency and death, if left untreated.^{3,6}

Diagnosis of the neural form of listeriosis is based on clinical signs of circling, lateral head tilt, facial nerve paralysis, tongue protrusion, and salivation.² There is no accurate antemortem diagnostic method to confirm listeric encephalitis.³ Diagnosis is confirmed by isolating the organism in the brain stem and anterior spinal cord on postmortem examination.² Culturing for *Listeria* using a “cold enrichment” technique improves the likelihood of growing the organism;^{2,3} however, isolating the bacteria from cerebrospinal fluid (CSF) is usually not successful.² A CSF analysis is generally not diagnostic, but can lend support to the diagnosis. In one study of clinical listeriosis,⁶ 77% of CSF samples had an elevated total white blood cell (WBC) count with a differential cell count of 56% monocytes and 38% lymphocytes, and 69% had an elevated protein concentration. In another study, CSF samples that were supportive of encephalitis secondary to *Listeria* infection had total WBC counts $>12 \times 10^6$ cells/L and a protein concentration >0.4 g/L.³ Gross postmortem examination rarely reveals any lesions. Histologic lesions are typically unilateral and most severe in the medulla oblongata and the pons.³ The characteristic lesion is the perivascular cuffing of lymphocytes³ and microabscesses in the midbrain, pons, and medulla oblongata.^{2,6} In one study,¹² most lesions were found to be localized to the brainstem (64.3%), and included perivascular cuffing (97.6%) and microabscesses (95.2%). This same study confirmed *L. monocytogenes* by immunohistochemistry in 80.9% of cases and 28.5% were positive on culture.¹²

Treatment: *Listeria* is sensitive to most antimicrobials except cephalosporins.³ Chlortetracycline (10 mg/kg IV q24h) for 5 days or penicillin (44,000 IU/kg IM q24h) for 7 days can be effective when treating encephalitis, but treatment may be required for up to 2 weeks.¹³ Recovery depends on the duration and severity of clinical signs, but prognosis for sheep and goats is guarded. In one study,⁶ 60 of 78 affected cattle capable of standing when treatment started, survived, whereas only 2 of 9 animals that were recumbent survived. This same study also found that animals in an excitable mental state were 5 times more likely to die, animals with an absent or weak menace response were 3 times more likely to die, and only 1 out of 5 animals with nystagmus recovered.⁶ If the animal is recumbent, comatose, or convulsing, survival is rare.²

The visceral form

The visceral form of listeriosis is clinically seen as septicemia. Acute septicemia is uncommon in adult animals, but occurs in monogastrics, including foals, young pigs, neonatal lambs, and neonatal calves (see Case 2).^{1,2} In adult sheep, it is seen as enteritis with abomasal hemorrhage and ulceration³ and, in pregnant sheep, as pyrexia and diarrhea.² In neonates, this form is usually an extension of intrauterine infection. The visceral form can cause secondary neural and reproductive tract infections.²

Diagnosis is by bacterial culture of liver, spleen, lung, or uterus. Histologic findings include multiple foci of hepatic necrosis with grey-white pinpoint lesions on the liver and possibly on the spleen.^{2,3}

The reproductive form

The reproductive form of listeriosis is seen clinically as abortions, stillbirths, and premature births. It is most common in ruminants and is caused by either *L. monocytogenes* or *L. ivanovii*, with *L. ivanovii* mainly affecting sheep.² Abortions in affected animals usually occur 5–12 days postinfection.^{2,3} Uterine infections are characterized by late-term abortions or septicemia in neonates¹⁰ due to hematogenous spread from the placenta.³

Diagnosis is by bacterial culture of the fetus, placenta, and cotyledons.^{2,3} Histologic lesions include placental lesions and pin-point yellow necrotic foci at the tips of villi on cotyledons with diffuse intercotyledonary placentitis.³ The fetus is usually autolyzed.

Other forms

Other forms of disease caused by *Listeria* include iritis, keratoconjunctivitis, mastitis, and atypical pneumonia in feedlot cattle. *Listeria* can cause eye infections and keratitis by direct inoculation of the eye.¹⁰ Eye infections are usually unilateral and seen during winter silage feeding.^{2,3} Clinical signs of ocular *Listeria* infection include a swollen, hyperemic conjunctiva, epiphora, photophobia, corneal clouding, scattered white corneal foci, blepharospasm, miosis, stromal edema, and vascular proliferation.^{1,9} Conjunctivitis is usually associated with meningitis and encephalitis.¹

Intramammary infection in cattle and sheep can cause subclinical mastitis with a high somatic cell count and persistent shedding of the bacterium in the milk.⁸ Intramammary infection can occur by hematogenous spread or by penetration of the teat canal, and can result in induration and atrophy of the mammary parenchyma. *Listeria* can often be isolated from the affected udder and regional lymph nodes.⁸

Atypical pneumonia in feedlot cattle is clinically seen as depression, listlessness, separation from the herd, fever, salivation, and diarrhea.² It usually occurs 60 days after animals enter the feedlot and presents pathologically as patchy bronchopneumonia with generalized interstitial pneumonia.²

Equine listeriosis: *L. monocytogenes* is a rare cause of disease in horses.⁴ However, there have been reports of *Listeria* causing abortion, septicemia in neonates (see Case 2), mature horses, and ponies, as well as, neurologic disease in foals⁴ and keratitis in adult horses.^{1,4} Listeriosis is a well-known disease in native Icelandic horses that are housed outside and fed grass silage during the winter.¹⁴ The most common signs are fever (39°–40°C), gastroenteritis, septicemia, diarrhea, inappetence, depression, and laboured respiration. Generally, only one or only a few horses are affected. Infection usually occurs by silage feeding, feeding poor-quality hay, or grazing on a flood-irrigated grass clover pasture.¹⁴ In Iceland, foals and young horses are more severely affected and large numbers of organisms are found in the feces of clinically affected horses.¹⁴ Listeriosis in horses is often fatal.

Zoonosis: Listeriosis is a zoonotic disease causing septicemic or cutaneous listeriosis in humans. Pregnant women may develop septicemia leading to abortion, stillbirth, or premature birth. Non-pregnant women and men may be affected by meningitis and septicemia with flu-like symptoms or gastrointestinal distress within 12 hours of consuming contaminated food.² Most affected adults have another predisposing condition (eg, pregnancy or a compromised immune system). Listeriosis can be transmitted by direct contact with eyes, skin abrasions, contaminated soil and feces, or by ingestion.² Outbreaks have been associated with ready-to-eat meat products, raw milk, pasteurized milk products, soft cheeses, poultry, and coleslaw.^{2,8,10}

Diagnosis in humans is by polymerase chain reaction used to identify the organism in CSF.⁴ Primary cutaneous listeriosis has been diagnosed in a veterinarian who was affected by a pustular rash, mild fever, myalgia, headache, and minor axillary lymphadenopathy.¹⁵ These signs occurred 7 days after delivering a dead calf with his bare hands. Bacterial cultures of the lesions revealed *L. monocytogenes*.

Treatment: With antimicrobial therapy, signs usually resolve in 1 week; however, without therapy, signs can persist for 1 month and can be fatal, with a reported mortality rate of 50%.³ Cutaneous listeriosis can be horizontally transmitted to humans and animals; however, this is very rare, since it is usually a localized cutaneous infection.^{10,15}

Prevention and control

Listeria is ubiquitous in nature and, therefore, prevention and control are difficult. Control measures include

limiting exposure of susceptible individuals, as well as the isolation and treatment of infected animals. Dead animals should be rendered or burned and buried in a pit with lime. If animals survive, they are chronic shedders and should be culled from the herd.² Control methods should be aimed at decreasing the likelihood of *Listeria* multiplication in silage by monitoring pH (maintaining pH at <5), as well as by reducing molds within the silage³ and not using feed from the top and sides of the silage pack. Silage fermentation can be assessed by EnviroTest Laboratories (1-800-667-7645) in Saskatoon, Saskatchewan. Currently, there is no commercially-available vaccine for listeriosis.

Case 1

A 10-year-old Charolais cow is presented to the Western College of Veterinary Medicine (WCVU) with a swollen face and circling. Three cows had died on the farm 5 days earlier after showing similar signs. This cow was fed 2-year-old barley silage for the past 6 weeks. The silage pit was evaluated 3 weeks prior and determined to have adequate fermentation.

On presentation, the cow is very aggressive, but not circling. The cow has an obvious mass on the right side of its face. Physical examination reveals fever (39.2°C) with a right-sided facial nerve paralysis that manifests as the following right-sided abnormalities: lip droop, partially chewed hay in the corner of her mouth (causing the observed swelling), ear droop, enophthalmos, ptosis, absence of menace response, reduced pupillary light reflex, dry eye, and an inability to clean the right nostril (Figure 1). The cow is also hypersalivating and has trigeminal nerve paralysis leading to a “loose” lower jaw. The cow can drink, swallow, and prehend, but has difficulty chewing feed.

A complete blood count (CBC) at presentation reveals a neutrophilia with a left shift and elevated total solids, which is interpreted as inflammation. A chemistry profile at presentation demonstrates elevated creatinine, creatine kinase (CK), and aspartate aminotransferase (AST), as well as hyperalbuminemia. These changes are interpreted as volume contraction and skeletal muscle leakage due to muscle injury, inflammation, or necrosis.

Figure 1: Case 1 – Cow with right-sided facial nerve paralysis



A venous blood gas at presentation reveals metabolic acidosis. The cow is treated with oxytetracycline (TetrajectLP,[®] 6.6 mg/kg IV q24h), topical eye ointment of bacitracin, neomycin, and polymyxin B (BNP[®] ointment, topically OD q12h) and topical atropine drops (generic, 1 drop OD q12h). Two days after presentation, a temporary tarsorrhaphy is performed and the eye is then treated with a topical formulation of penicillin G procaine, polymyxin B, and hydrocortisone (Special Formula,[®] 1/8 tube topically OD q12h). The cow is given 40 L of water containing alfalfa pellets, dairy ration, propylene glycol, oral electrolytes, and bicarbonate, twice daily by orogastric tube. A lumbosacral cerebrospinal tap performed 1 day after presentation yields 5 mL of clear, colourless fluid with an elevated nucleated cell count ($55 \times 10^6/L$), elevated red blood cells (RBCs: $75 \times 10^6/L$), and elevated protein concentration (0.83 g/L; Table 1).¹³ A differential cell count reveals predominately small and large mononuclear cells with 3% non-degenerate neutrophils, 1% eosinophils, free erythrocytes, but no infectious agents are present. The CSF changes are interpreted as a mononuclear cell pleocytosis with iatrogenic hemorrhage.

Clinical signs progress to include a head tilt, tongue protrusion, and decreasing rumen contractions. The cow's condition improves only slightly during

Table 1: CSF Analysis from Cases 1 and 3

| | Case 1 (bovine) | Case 3 (ovine) | Values supportive of encephalitis secondary to listeriosis ³ | Suggested CSF Analysis, normal reference range ¹³ |
|-------------------------|-----------------|----------------|---|--|
| NCC ($\times 10^6/L$) | 55 | 669 | >12 | <5 |
| Protein (g/L) | 0.83 | 2.18 | >0.4 | 0.23-0.66 |
| RBC ($\times 10^6/L$) | 75 | 3865 | n/a | 0 |
| Mononuclear pleocytosis | Yes | Yes | Yes | Yes |

NCC = nucleated cell count; RBC = red blood cell; CSF = cerebrospinal fluid

therapy and, due to poor prognosis, the owner elects euthanasia on day 7. The final diagnosis after post-mortem examination is subacute, locally extensive, non-suppurative, necrotizing meningomyeloencephalitis with multifocal microabscessation. There is severe inflammation in the caudoventral brain, sparing the cerebrum, but affecting the brainstem, parts of the cerebellum, and the cranial cervical spinal cord. A culture of brain tissue is negative for *Listeria*; however, the presumptive diagnosis based on clinical signs and histopathologic lesions is listeriosis.

Case 2

A 4-day-old Tennessee walking horse colt is presented to the WCVM because of an inability to stand for the past 12 hours. This foal was unattended at birth, but was observed nursing from the mare for a very short time. Since its birth, the colt seems “confused” about where to find the udder. On presentation, the foal is comatose and in lateral recumbency; it is hypothermic (36.6°C) with cold extremities, has a swollen left front fetlock, and a decreased suckle reflex. The mucous membranes are pale, icteric, and have a prolonged capillary refill time. The colt’s eyes are sunken and the sclera are icteric. He also has a prolonged skin tent that suggests an estimated 10% dehydration.

A CBC on presentation reveals leukopenia, neutropenia with a left shift and a 3+ toxic leukocyte change, anemia, decreased total solids, and hyperfibrinogenemia. Leukogram changes are interpreted as acute, severe inflammation. A chemistry profile on presentation reveals metabolic acidosis, azotemia, hypoglycemia, hyperbilirubinemia, elevations in glutamate dehydrogenase (GLDH), AST, and sorbitol dehydrogenase (SDH), as well as hypoproteinemia and hypoglobulinemia. These changes are interpreted as mild metabolic acidosis, mild azotemia, and mild hepatocellular injury with possible failure of passive transfer. A serum, enzyme-linked immunosorbent assay (ELISA) quantification reveals an IgG concentration of 400–800 mg/dL, indicating a partial failure of passive transfer. A joint fluid aspirate of the left front fetlock yields a thick, dark yellow fluid with an elevated nucleated cell count, an elevated protein concentration, and a few RBCs. The differential cell count shows 95% nondegenerate neutrophils and 5% mononuclear cells; no bacteria are present, leading to an interpretation of a non-septic suppurative inflammation. Treatment includes amikacin (Amiglyde-V[®], 30 mg/kg IV q24h), ampicillin (generic, 20 mg/kg IV q8h), ketoprofen (Anafen[®], 3 mg/kg IV q24h), IV fluid therapy, plasma administration, left front fetlock joint lavage, and feeding via a nasogastric tube. Over the next day, the foal seems to improve. He is able to

hold himself in sternal recumbency and has an improved suckle reflex. However, more joints become effusive and due to a poor prognosis, the owner elects to euthanize 1 day after presentation. Postmortem examination reveals necrotizing hepatitis, septic arthritis, cerebrocortical necrosis, icterus, and evidence of sepsis. A blood culture taken at presentation is positive for *L. monocytogenes*.

Case 3

A 4-year-old ewe is presented to the WCVM with a complaint of “not doing well.” The ewe has a history of not eating or drinking for 1 day, hypersalivation, and standing away from the flock. The flock was fed alfalfa hay and creep feed was available.

On presentation, the ewe is depressed, ataxic, and hypersalivating. She has a left ear droop, no menace response in the left eye, and no pupillary light reflexes bilaterally. Wheezes are heard on auscultation of the lung fields. A CBC on presentation reveals neutrophilia and hyperproteinemia. These changes are interpreted as mild inflammation with dehydration. A chemistry panel at presentation demonstrates elevated CK and hyperproteinemia. These changes are interpreted as mild myocyte damage with dehydration. A lumbosacral spinal tap yields CSF that is slightly cloudy with an elevated nucleated cell count ($669 \times 10^6/L$), an elevated RBC count ($3895 \times 10^6/L$), and an elevated protein concentration (2.18 g/L; Table 1).¹³ A differential cell count exhibits 81% large mononuclear cells, 12% nondegenerate neutrophils, and 7% small mononuclear cells with no visible infectious agents. Treatment includes thiamine (generic, 700 mg IV q24h), oxytetracycline (Tetraject LP[®], 6.6 mg/kg IV q24h), ketoprofen (Anafen[®], 3 mg/kg IV q24h) and IV fluids. The ewe progresses clinically by gaining a menace response on the left side, but still has decreased pupillary light reflexes bilaterally, hypersalivation, and becomes very depressed. Due to the poor response to therapy and a poor prognosis, the owner elects euthanasia 2 days after presentation. Postmortem examination reveals meningoencephalitis and bronchopneumonia. Histologic findings were suggestive of listeriosis and this was confirmed by immunohistochemistry.

Summary

Listeriosis is primarily a disease of cattle and small ruminants. It is seen most often associated with silage feeding. Successful treatment depends on the duration and severity of clinical signs. Prognosis is generally poor in small ruminants and horses, but some success can be expected when treating cattle. *Listeria* is a zoonotic disease and care should be taken by those in professions at high risk for exposure.

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Abstract of Interest

Clinical findings and treatment of 94 cattle presumptively diagnosed with listeriosis

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The clinical findings and treatment of 94 cattle with listeriosis are described. The general behaviour and condition of the animals were mostly moderately to severely disturbed. A common abnormality in posture was an exaggerated forward or sideward stance, and 11 of the animals were recumbent. More than half of the animals were ataxic and 22 circled. The most frequent cranial neurological signs observed were facial nerve paralysis,

salivation, strabismus, reduced or absent pupillary light reflex, reduced or absent tongue movement and head tilt. The haematological and biochemical findings did not contribute to the diagnosis of listeriosis, but they were useful indicators of dehydration and the acid-base status of the animal. Forty-four of 57 of the animals had high leucocyte counts in the cerebrospinal fluid (CSF), mostly mononuclear cells. Eighty-seven of the animals were treated with various antibiotics (penicillin G, oxytetracycline, amoxicillin, and amoxicillin and gentamicin combined), but there was no significant difference in the success rate of the different treatments. Only two of the nine recumbent animals that were treated survived. Univariable analysis suggested that animals that were recumbent, excited, with an absent or weak menace reflex, nystagmus, high numbers of leucocytes in the CSF, high serum concentrations of urea and calcium and high serum activities of aspartate aminotransferase and creatine kinase, and an acid-base deficit, had a smaller chance of surviving. When a logistic regression model was constructed, only recumbency, excitement and a weak or absent menace reflex remained significant factors affecting the likelihood of survival.

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52nd Convention of the American Association of Equine Practitioners (AAEP)

San Antonio, Texas, USA

Contact: E-mail: aaepoffice@aaep.org

Website: www.aaep.org

18-20 January 2007

16th Annual Conference of the Western Canadian Association of Bovine Practitioners

Saskatoon, SK

Contact: WCABP

Tel: 1-866-269-8387

E-mail: info@wcabp.com

Website: www.wcabp.com

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