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Polioencephalomalacia

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Polioencephalomalacia (PEM) is a descriptive term that refers to a softening (malacia) of the gray matter (polio) in the brain (encephalo).¹ PEM is a histological diagnosis with many different etiologies including excessive sulphur consumption, altered thiamine status, salt poisoning/water deprivation, and lead poisoning. This issue of *Large Animal Veterinary Rounds* reviews all four of these etiologies for PEM.

PEM occurs worldwide in cattle, sheep, goats, camelids, elk, and deer.² The condition may occur in pastured cattle with high dietary sulphur and, typically, is observed in late summer and fall; however, it is predominantly found in feedlots in the United States (US) within 3 weeks of a ration change.³ One study found that most cases occur after 15 to 30 days-on-feed (DOF) and 98% of cases occur within 60 DOF.⁴ Herd outbreaks have been associated with management changes, such as sudden feed changes and anthelmintic or amprolium use.⁵ PEM is seen primarily in cattle on high-concentrate rations and reported in cattle on a molasses-containing diet or a cobalt-deficient diet.⁵ In summary, PEM can be the result of:

- high sulphur intake
- altered thiamine status
- acute lead poisoning
- salt toxicity.

Sulphur-induced PEM

Sulphate (SO_4^{2-}) and elemental sulphur (S_2) are nonreduced, nontoxic forms of sulphur. Sulphide (S^{2-}) is the reduced form of sulphate and sulphur. Sulphide is toxic to cells due to its interference with normal energy metabolism at the cellular level.⁶

Sulphur metabolism and mechanism of action

Primarily, there are two metabolic pathways for rumen sulphur, the assimilatory and dissimilatory pathways.^{2,6} The assimilatory pathway is the reduction of sulphate to sulphide and its incorporation into sulphur-containing compounds (eg, cysteine and methionine) destined for use in microbial proteins. The dissimilatory pathway is the use of sulphate by rumen microbes as a terminal electron acceptor and results in the release of a sulphide ion. Sulphide-ion liberation is a normal occurrence and, typically, is used in hydrogen sulphide gas (H_2S) production.¹ H_2S can be absorbed through the rumen or eructated and possibly inhaled, resulting in absorption through the lungs.⁷ For sulphide to have toxic effects, it must bypass hepatic detoxification (oxidation to sulphate). Hepatic detoxification could be bypassed if the hepatic oxidation systems were overwhelmed or if the eructated H_2S were absorbed through the lungs, effectively bypassing hepatic circulation. However, absorption through the lungs has not yet been proven. The toxic effects of sulphide are believed to be due to its inhibition of cytochrome C oxidase, an enzyme of the electron transport chain that is involved in the production of adenosine triphosphate (ATP).

Factors affecting sulphide production

Many factors can affect sulphide production, including total sulphur intake, ruminal microbial populations, trace element concentrations, ruminal pH, and animal individuality.⁶ When determining the level of sulphur in a diet, all sources must be included both dietary and water intake. Sources of dietary sulphur may include supplements such as, methionine, elemental sulphur, sodium sulphate (Glauber's salt),



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Table 1: Suggested normal levels for ruminal gas cap hydrogen sulphide	
Ruminal gas cap H₂S	Level
Cattle	<0.695 mg/dL or <500 ppm ¹⁰
Healthy cattle on roughage	14-53 μM ⁴
Sheep	6-19 μM ⁴
Sheep fed <0.4% dietary sulfur	<200 μM ⁴
Beef cattle 21 DOF	175 ± 146 μM ⁴
Beef cattle 56 DOF	83 ± 117 μM ⁴

H₂S = hydrogen sulfide gas; DOF = days on feed; ppm = parts per million

magnesium sulphate (Epsom salt), ammonium sulphate, calcium sulphate (gypsum), and sulphur dioxide,⁸ or feedstuffs, such as, corn by-products (distillers grains), sugar cane or sugar beets and, due to sulphur-containing acidifying agents used in processing, molasses⁶ or fertilizers.^{1,2} Weeds that can accumulate sulphate include Canada thistle (*Cirsium arvense*), kochia (*Kochia scoparia*), and lambsquarter (*Chenopodium* spp).⁶ Cruciferous plants also contain high levels of sulphates.

When assessing sulphur intake, the total sulphur content of all matter consumed must be calculated. Total intake calculations are complicated by the lack of uniformity in reporting units between different substrates. Sulphate in water is expressed as parts per million (ppm), which equals mg/L. Sulphate is 1/3 sulphur, therefore, sulphate in mg/L ÷ 3 = sulphur in mg/L. An estimate for daily dry matter intake in mature cattle is 3% of body weight (bwt) in kg.⁶ Estimated daily water intake for beef cattle is 8%, 10%, and 18% bwt in kg at 4°C (40°F), 21°C (70°F), and 32°C (90°F), respectively.⁶ Daily water intake for lactating beef cows is estimated to be 10%, 15%, and 15% bwt in kg at 4°C, 21°C, and 32°C, respectively.⁶ Sulphur requirements for beef cattle are 0.15% dry matter with a maximum tolerable concentration of 0.4% on a dry matter basis.⁹ A “sulphur intake calculator” is available at <http://www.dlab.colostate.edu> under the special interests tab (Accessed: March 5, 2008).⁶

Ruminal sulphate reducing microbes

Microbial sulphide production has been shown to increase with high-dietary sulphur intake.^{1,7} There is an adaptation time for maximal H₂S production, with sulphur reduction increasing 10 days after the introduction of a high-sulphur diet.⁸ Ruminal flora typically adapt by 30 days after a high-sulphur diet is introduced.⁸ In experimentally-induced sulphur-associated PEM, both the ruminal fluid and the gas cap had elevated sulphide concentrations.² Rumen gas cap sulphide can be estimated using the H₂S detector-tube method.^{7,10} Rumenocentesis is performed in the left paralumbar fossa using an 18-gauge, 8.75 cm (3½ inch) spinal needle. Gas from the rumen gas cap is drawn into an H₂S detector tube and a concentration of H₂S >1,000 ppm indicates excessive sulphur consumption.² A ruminal H₂S level >2,000 ppm has been shown to precede the development of PEM (Table 1 indicates recommended normal H₂S levels).^{1,7} Sulphur-induced PEM seems to be independent of thiamine status.^{2,6,8,11}

In the case of an outbreak, assess the ruminal H₂S in herd mates, since the H₂S of those affected has usually returned to normal levels because they become anorectic and stop consuming excessive sulphur.⁸ The H₂S content of the rumen gas cap is affected by rumen microbe sulphide production, rumen pH, frequency of eructation, and ruminal absorption.^{1,7}

Trace elements

Sulphur availability can be diminished by the formation of insoluble metal sulphides of copper, iron, and zinc. The reverse is also true; eg, copper deficiency can result from high sulphur intake.^{6,8} Microbial sulphate reduction can also be hindered by molybdenum.⁶ The effect of trace elements on ruminal H₂S production has not yet been determined. Lesions may include dark ruminal ingesta or mucosa due to insoluble sulphide production.

Ruminal pH

The distribution of sulphide between the fluid and gas phases in the rumen is determined by ruminal fluid pH. Most sulphide ions (97.2%)² are in the gas phase as H₂S gas, with a rumen pH of 5.2.⁶ H₂S in the gas cap increases as rumen pH decreases.⁶ Assessment of ruminal sulphide production has demonstrated that there are more significant changes in ruminal gas-cap H₂S levels than in rumen fluid sulphide.⁷ Speculation suggests that this may be due to the separation of the gas cap from the rumen fluid sulphur cycle. Feed and water intake will decrease with clinical signs and result in a decrease in sulphur intake and, ultimately, a decrease in H₂S production. Therefore, sampling ruminal gas-cap H₂S in clinically affected animals may reveal normal levels, but clinically unaffected herd mates may have elevated levels.

Thiamine-associated PEM

Thiamine is water soluble, has a short half-life, and is stored mainly in liver and muscle. The principal function of thiamine is as cocarboxylase, a coenzyme in the Krebs cycle (also called the tricarboxylic acid [TCA] cycle or citric acid cycle) that provides energy to the body. The Krebs cycle is used in pyruvate metabolism; as a result, thiamine deficiency will cause blood elevations of pyruvate and lactate.³ Thiamine is also a coenzyme for transketolase, an enzyme in the pentose phosphate pathway (an oxidative pathway for glucose), found in erythrocytes and neurons. A decrease in thiamine causes a reduction in the adenosine triphosphate (ATP)-dependent sodium and water transport mechanisms of neurons, and results in intraneuronal swelling, increased intracranial pressure, and neuronal necrosis. Eventually, there is capillary endothelial swelling and proliferation, as well as macrophage infiltration of the cerebral cortex. Laminar cortical necrosis, the pathological change in PEM, is the final result of thiamine-related cell death, intraneuronal swelling, and increased intracranial pressure. Altered thiamine status can be due to:

- dietary thiamine deficiency
- thiaminase in the rumen
- administration of thiamine analogues.

In addition, there may be impaired absorption, increased fecal excretion, or decreased production of the active form, thiamine diphosphate.

Dietary thiamine deficiency

Ruminant thiamine is derived primarily from rumen bacteria and actual ingested thiamine represents only a small contribution. Dietary deficiencies are only seen in preruminants (young animals with undeveloped rumens).

Thiaminase in the rumen

In the rumen, thiaminase breaks down thiamine; low levels of thiaminase can be found in the ruminal contents and feces of normal animals.¹² Thiaminase can be the result of bacterial production that is associated with high-concentrate feeds, and it can be found in certain plants or secondary to drug usage. Two types of thiaminase are produced by bacteria in response to excessive grain intake.^{2,12} Thiaminase I, produced by *Bacillus thiaminolyticus* or *Clostridium sporogenes*, destroys thiamine and produces an analogue by a base-exchange reaction.^{2,3,12} Anthelmintics such as levamisole or thiabendazole can act as co-substrates for thiaminase I.¹² Thiaminase II, produced by *Bacillus aneurinilyticus*, destroys thiamine by cleavage.^{2,12} In one study, ruminal microbes in a pair of PEM-affected heifers resulted in decreased rumen thiamine levels when compared with a healthy steer.¹³ Another study found that normal rumen fluid at a pH of 6.8 had little thiaminase activity, but when it was “acid shocked” to a pH of 4.5, thiaminase was found.¹² Thiaminase-containing plants include bracken fern (*Pteridium aquilinum*; thiaminase I), horsetail (*Equisetum arvense*), and kochia weed (*Chenopodiaceae*). Sulphite ions (SO_3^{2-}) can also cleave thiamine and thus act as a thiaminase.^{1,12}

Thiamine analogues

Thiamine analogues competitively inhibit glycolytic reactions. Analogues can be produced by bacteria or plants (eg, bracken fern). As well, drugs such as amprolium, pyriithiamine, and oxythiamine may act as thiamine analogues.³

Thiamine status assessment

There are multiple methods to assess thiamine status.² A thiamine-dependent *Lactobacillus* bioassay can be used to determine the total blood thiamine content. Erythrocyte thiamine pyrophosphate concentration can be measured using high-performance liquid chromatography. Erythrocyte transketolase activity is considered a sensitive and specific measure of active thiamine status. A finding of thiamine deficiency should be interpreted with the understanding that an anorectic animal has decreased thiamine production in the rumen. Therefore, thiamine deficiency can be both the result and the cause of PEM. Decreased thiamine in body tissues, decreased transketolase activity (a thiamine-dependent enzyme) in the blood, and increased thiaminases in feces have been documented in cases of PEM.¹

Clinical forms, signs, and responses to PEM: sulphur or thiamine causation

There are two recognized forms of PEM: subacute and acute.^{2,6} The subacute form develops within hours to days. The affected animal will separate from the herd and reveal any of the following signs: anorexia, staggering, apparent blindness, a slight hypermetric gait, may exhibit excitement and hyperesthesia,

Table 2: Suggested normal levels for blood thiamine

Blood thiamine	Level
Cattle and sheep	75-185 nmol/L ¹⁵
Beef cattle 21 DOF	63 ± 13 µg/L ⁴
Beef cattle 56 DOF	61 ± 13 µg/L ⁴

DOF = days on feed

diarrhea, muscle tremors (eg, ear flicking or facial twitching), nystagmus, strabismus, or head tilt. Clinical signs progress to cortical blindness (no menace reflex, but intact pupillary light reflex [PLR]), head pressing, opisthotonus, dorsomedial strabismus, miosis, repetitive chewing, and ptyalism. Most animals respond favourably to aggressive therapy; however, it can progress to recumbency, tonic-clonic convulsions, and death.

The acute form manifests as blindness, recumbency, and seizure activity with intermittent tonic-clonic convulsions and sustained tonic activity between episodes. The animal may become comatose or may be found dead. The prognosis for acutely affected animals and those with very advanced subacute PEM is grave. Some animals that recover will have permanent decorication resulting in poor performance, ataxia, and blindness.

Diagnosis of PEM

Definitive diagnosis is based on histological findings; however, a presumptive diagnosis can be made based on history and clinical examination findings. Blood thiamine levels do not seem to be reliable in diagnosing PEM; however, thiamine levels from red blood cells and tissue may give a better indication (Table 2 indicates recommended normal blood thiamine levels).⁵ One study found no difference in blood thiamine levels for 5 cattle with PEM when compared with 11 cattle with other neurological diseases.¹⁴ Thiaminase levels in the rumen and feces may be elevated with PEM.⁵ There are increases in blood pyruvate levels, as well as pyruvate kinase in blood and urine.⁵ Cattle with PEM have reduced erythrocyte transketolase activity.⁵ Abnormalities of cerebrospinal fluid (CSF) are not specific for PEM, but may include mild pleocytosis (5–10 white blood cells [WBCs]/dL; normal <5 WBCs/dL)⁵ with an elevation in protein (>50 mg/dL; normal <40 mg/dL).^{2,5}

Postmortem findings

Gross findings indicate a soft, swollen brain with a flattening of the cerebral cortex gyri. Cerebellar coning may be present, due to increased intracranial pressure.⁶ In the subacute form, the initial histological lesions include shrunken neurons with homogeneous neuropil that has lost the normal dendritic marking pattern.⁶ This early stage reveals autofluorescence when observed with a 366 nm ultraviolet light (Wood's lamp).⁶ The autofluorescence is found in necrotic portions of the brain due to the presence of lipofuscin. Later in the disease, the necrotic tissue is removed by macrophages and results in cavitation on gross observation. At this later stage, autofluorescence is no longer visible.⁶ The acute form will have the above findings, as well as spongy changes indicating astrocyte swelling.⁶ Overall, the histological lesions include laminar necrosis of the grey matter, most prominently in the cerebral cortex; however, foci can be found throughout the brain. There will also be

intracellular edema, neuronal necrosis, neuronophagia, and gliosis.^{2,5} If an animal has recovered from a case of PEM or is chronically affected, cerebral atrophy with decortications may be present. Dark ruminal ingesta and mucosa may be seen with sulphur-associated PEM due to insoluble sulphide production.

Treatment of PEM

There are a number of treatment recommendations for PEM; however, a general recommendation is to treat with thiamine at 10 mg/kg intravenously (IV) or intramuscularly (IM) every 6 hrs for the first day, followed by daily treatment.⁵ Others report treating with thiamine at a dose of 10–20 mg/kg IM or subcutaneously (SC) every 8 hrs for at least 3 days.² Acute cases respond poorly to treatment; however, it is recommended to treat with 1 g/day injectable thiamine followed by 500 mg/day in the diet for 7 to 14 days.³ In calves and lambs, it is recommended to treat with thiamine at 100–400 mg/day IV or IM for 3 days.³ In sheep and cattle, treat with thiamine at 500–2000 mg/day for 3 days.³ In goats, a suggested dose is 6.6–11 mg/kg every 6 hrs for 1 day.³ Prophylactic recommendations for cattle include 5–10 mg/kg of dry matter with feeding such that each animal consumes 100–500 mg daily.³ If left untreated, death usually results in 1 to 4 days.³ Improvement is usually seen within 24 hrs and it may take up to 1 week for recovery.²

Thiamine deficiency and treatment in horses

Thiamine deficiency occurs when horses are fed plants containing thiaminases including bracken fern (*P aquilinum*) and horsetails (*E arvense*). It may also occur secondarily to treatment with amprolium, a thiamine analogue, at a 400–880 mg/kg dose.² Clinical signs include ataxia, proprioceptive deficits, bradycardia, blindness, weight loss, dysuria, muscle twitching, and posterior paralysis.^{2,8} Treatment with thiamine is reported to be effective at 100–200 mg/kg twice on Day 1, then daily for 7 to 14 days.^{2,8}

Prevention and control

When feeding a high-concentrate ration, animals should be slowly adapted to these rations and free access to grain must be avoided to prevent grain overload. Thiamine supplementation (brewer's yeast) should be considered. Thiamine supplementation at 3–10 mg/kg of feed has been recommended.⁵ Dietary sulphur should be limited to <0.4% of dry-matter intake; the total sulphur intake of affected herds should be assessed and all excess sulphur removed from the diet.

Salt poisoning

Salt poisoning, or sodium-ion toxicosis, is another cause of a histological diagnosis of PEM. Salt poisoning can be due to excess consumption of sodium, usually resulting in gastroenteritis and neurological signs, or due to water deprivation leading to cerebral edema and neurological signs after water reintroduction.^{16,17} High-salt sources include feeds such as whey and bakery by-

products, as well as oral electrolytes, some milk replacers, and drinking water.¹⁷

Mechanism of action

Normally, sodium diffuses passively from blood to CSF; however, sodium movement from CSF into blood requires energy. As blood osmolality increases, the available energy for sodium transport decreases, thereby “trapping” sodium in the CSF. When the plasma sodium level is returned to normal by the kidneys or due to free water access, an osmotic gradient is created with the CSF remaining hyperosmolar. This gradient draws water into the CSF and results in cerebral edema and neurological signs.

Sodium requirements

Sodium requirements in nonlactating beef cattle are 0.06% to 0.08% and lactating beef cows require 0.10% of sodium on a dry-matter basis.⁹ The maximum tolerable limit of dietary sodium is 9.0% on a dry-matter basis.⁹ Sodium in water is more toxic and should not exceed 7,000 ppm.^{2,9} Toxic doses of 2.2 g/kg of sodium chloride in cattle and horses, and 6 g/kg in sheep have been reported.²

Clinical signs, diagnosis, and postmortem findings

Clinical signs of salt poisoning include mucohemorrhagic diarrhea, colic, blindness, aggression, hyperexcitability, seizures, ataxia, head pressing, chewing movements, nystagmus, muscle twitching, coma, and death.^{2,17}

Diagnosis is based on elevated serum or CSF sodium concentrations, typically >160 mEq/L.² Normal serum sodium concentrations are 135–145 mEq/L and normal CSF sodium concentrations are 130–140 mEq/L.¹⁷ A CSF to serum sodium ratio >1 is suggestive of salt toxicity.² When assessing serum sodium concentrations, take into account any access to ion-free water, since the serum sodium concentration will be decreased. With the exception of cases due to excessive sodium intake, most affected animals have a history of water deprivation. Postmortem findings typically include inflammation of the upper gastrointestinal tract in those animals consuming excessive sodium and possibly an edematous brain.¹⁷ Histological evaluation reveals cerebral edema, flattening of cortical gyri, and laminar cortical necrosis.^{2,17}

Treatment

All sources of excessive sodium, including feed, mineral mixes, and water, should be removed. Free water should be offered in small amounts at frequent intervals. Severely affected animals may require hospitalization and IV fluid therapy.¹⁸

Acute lead poisoning

Lead poisoning, or plumbism, is a common cause of poisoning in cattle of all ages. Lead ingestion can be due to indiscriminant eating habits or contaminated feeds. Common lead-containing products include batteries, lead-based paints, and petroleum products.

Mechanism of action

Absorption of lead depends on the ingested form; metallic and sulphide forms are poorly absorbed, whereas acetate, phosphate, carbonate, and hydroxide salts are more readily absorbed. Most ingested lead forms insoluble lead complexes in the gastrointestinal tract and passes in the feces. Approximately 2% of ingested lead acetate is absorbed in the first 24 hrs and the rest is passed in the feces.¹⁹ In an acidic environment, such as in the abomasum or stomach, lead is ionized, allowing easier absorption.⁸ Absorbed lead is bound 90% to red blood cells and the remainder is distributed throughout other tissues, including bone, teeth, liver, lung, kidney, brain, and spleen.⁸

The maximum tolerable level of lead ingestion in beef cattle is 30 mg/kg.⁹ A single acute exposure of 400 to 600 mg/kg can be lethal in calves and 600–800 mg/kg is lethal in adult cattle.^{8,19,20} Chronic exposure (1–7 mg/kg/day) can also result in lead poisoning.^{8,19} Absorbed lead inhibits the use of iron and the synthesis of heme, resulting in fragile red blood cells, anemia, and red blood cell basophilic stippling. Lead localizes in endothelial cells resulting in cellular edema and microangiopathy. Central nervous system (CNS) degeneration is thought to be due to altered transport mechanisms, as well as microangiopathy, and peripheral nerves are affected by segmental demyelination.^{19,21} Ingested lead can also be irritating to the gastrointestinal tract, resulting in diarrhea. Lead can also cross the placenta and is excreted in urine, milk, and bile.

Clinical signs, diagnosis, postmortem findings

In general, acute ingestion of lead in large doses results in encephalopathy, ingestion of moderate doses leads to gastroenteritis, and chronic ingestion results in peripheral neuropathy. Cattle commonly develop encephalopathy and gastroenteritis. Clinical signs in cattle include depression, absence of rumen motility, muscle tremors, champing of the jaws, ataxia, blindness, seizures, bellowing, head pressing, aggression, diarrhea, and death. Horses more commonly demonstrate peripheral neuropathy with weight loss, dysphagia, laryngeal paralysis, facial nerve deficits, seizures, and death. A “lead line” (also called a “blue line” or “Burton’s line”) may be seen as a blue-black discoloration of the gums in horses, dogs, and humans, due to the deposition of lead sulphide.^{19,20} There is basophilic stippling of the red blood cells (can be a normal finding in ruminants) and anemia is common in lead toxicosis.

A whole-blood sample for lead concentrations should be collected with heparin as the anticoagulant because this will not chelate the lead in the sample.²¹ Whole-blood lead concentrations of 0.3–0.35 ppm are suggestive (Table 3) and concentrations of 0.6 ppm are diagnostic for lead toxicosis (normal range, 0.05–0.25 ppm).^{8,20} A urinary calcium ethylenediaminetetracetic acid (Ca-EDTA) postchelation test can be performed. Lead is measured in a 24-hr urine sample; Ca-EDTA is administered and followed by another 24-hr urine sample for lead. Lead toxicosis is indicated if the lead excretion from urine increases 10 times that of prechelation levels.

Table 3: Toxic tissue and blood lead concentrations⁸

	Blood	Liver	Kidney
Cattle	>0.30 ppm	5-300 ppm	5-700 ppm
Horses – chronic		4-50 ppm	4-140 ppm
Horses – acute		10-500 ppm	20-200 ppm
Most species	0.3-0.35 ppm is significant		
	0.6 ppm = toxic		

Postmortem examination commonly reveals laminar cortical necrosis and cerebral cortical swelling.^{8,19,21} Typically, there is also hepatic and renal tubular degeneration. Gastrointestinal contents should be submitted for lead concentrations.

Treatment

Without treatment, acute lead poisoning is usually fatal.²⁰ Treatment for lead poisoning includes nursing care, removal of the source of lead, and lead chelation using Ca-EDTA. Ca-EDTA acts by drawing lead out of bones, but it cannot cross cell membranes. Therefore, removal of soft-tissue lead is facilitated by an equilibration with lead in the bone as it is removed. Administration of Ca-EDTA may cause a worsening of clinical signs. One recommended Ca-EDTA dosing regimen for ruminants and horses is 73 mg/kg (1 mL/0.9 kg of a 6.6% solution of Ca-EDTA) divided into 3 IV doses for 3 to 5 days, followed by 2 days of rest, then repeated if necessary.^{8,19} Alternatively, give 110 mg/kg IV twice daily for 2 days, with 2 days rest, then repeated if necessary.^{8,21} Side effects of rapid Ca-EDTA administration include tachycardia, tachypnea, and tremors that can be avoided by slow IV administration. Other side effects associated with Ca-EDTA administration include hypocalcemia, hypotension, fever, anorexia, pain, and acute renal failure.¹⁹ Ca-EDTA will increase urinary lead excretion by 20 to 50 times the normal levels.¹⁹ Thiamine hydrochloride at a recommended dose of 250–1000 mg SC twice daily for 5 days will decrease lead deposition in soft tissues by lead chelation.^{19,20} Alternatively, thiamine hydrochloride can be administered at a dose of 75 mg/kg once or 25 mg/kg SC every 12 hours.²¹ Rumenotomy may be indicated to remove ingested lead.

Public health concerns

Currently, there is no recommendation for withdrawal times with cattle that recovered from lead poisoning. Recovered cattle should be monitored via blood lead concentration. Once blood lead concentrations are normal for 3 consecutive samplings, 2 weeks apart, the animal is thought to be safe for human consumption.²⁰ Reports in the literature suggest that a minimum of 6 months is necessary for lead concentrations to decline to normal levels.²⁰ Recommended milk and meat withdrawal times for Ca-EDTA are 2 days, due to the minimal body distribution.²²

Summary

PEM is a histological diagnosis with many etiologies, including excessive sulphate intake, altered thiamine status, salt poisoning or water deprivation, and lead poisoning. Prognosis is dependent on the etiology for those acutely affected; those with advanced subacute PEM have a grave prognosis.

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Upcoming Meetings

4 – 7 June 2008

American College of Veterinary Internal Medicine Forum

San Antonio, Texas

CONTACT: <http://acvim.org/>

19 – 22 July 2008

145th Annual Convention of the American Veterinary Medical Association

New Orleans, Louisiana

CONTACT: AVMA

Tel: 800 248-2862 ext. 6621

Fax: 847 925-1329

Website: <http://avmaconvention.org/>

17 – 21 September 2008

14th International Veterinary Emergency & Critical Care Symposium

Phoenix, Arizona

CONTACT: VECCS

Tel: 210 698-5575

Fax: 203 698-7138

Email: info@veccs.org

Website: <http://www.veccs.org>

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