Comparison of Common Toxicants That Cause Signs of CNS Disturbance by Interference with Oxygen Availability to Tissues

Toxicant	Physical characteristics	Sources	Mechanism	Toxicity	Color of Blood	Treatment
Cyanide	Solid, liquid or gas	Cyanogentic plants, varmint traps & baits	Anticytochrome oxidases	Acute—200 ppm in plants, 50-60 ppm as gas	Cherry red	Sodium nitrite & sodium thiosulfate
Nitrate (Nitrite) or Chlorate	Solid or liquid	Forages, fertilizer contaminated water (herbicide)	Methemoglobin	Acute—1% in forages, 750 ppm in water	Brown	Methylene blue
Carbon Dioxide	Gas—heavier than air	Poorly vented furnaces, complete combustion	Displaces O 2, tissue acidosis	10-15% in air, 40% lethal	Dark	Provide fresh air or O 2.
Carbon Monoxide	Gas—slightly lighter than	Incomplete combustion, poor tight buildings	Carboxyhemoglobin	0.01—1.0%—distress—death	Bright red	Carbogen
Hydrogen Sulfide	Gas	Manure pits	Cessation of respiration	400 ppm in air	Dark	Ventilation, gas traps over pits
Nitrogen Dioxide (Oxide)	Gas—yellow—brown, slightly heavier than air	Silos	Increased pulmonary resistance, inhibit pulmonary functions, slight methemoglobin	(LD 50 in rabbits) 8-12 ppm continous exposure for 12 wks	Dark or sightly brown	Methylene blue and Ca gluconate
Sulfur Dioxide (Trioxide) (H 2 SO 4)	Gases, liquid (H 2 SO 4)	Industrial effluents	SO 2 + H 2 O H 2 SO 4 Irritants, pulmonary	40 ppm SO 2 in air, 20 ppm H 2 SO 4 in mist	Dark	None specific

(1) Taken from the following publications:

a) Buck, W. B. et al. 1976. CLINICAL AND DIAGNOSTIC VETERINARY TOXICOLOGY. G. A. Van Gelder, ed. 2nd Ed. Kendall-Hunt Publishing Company, Dubuque, Iowa.

b) Buck, W. B. *Toxic Materials and Neulogic Disease in Cattle.* 1975. JAVMA 166: 222-226.

c) Howard, J. L., ed. 1981. CURRENT VETERINARY THERAPY FOOD ANIMAL PRACTICE. W. B. Saunders, Philadelphia.

Common CNS Diseases of Feedlot Cattle

Drs. R. Pierson and Stuart Young

Polioencephalomalacia

(PEM: Cerebrocortical necrosis)

Occurrence -

1. Feedlot and pastured animals .5-2 years old.
2. Summer and fall

Etiology - Deficiency of thiamine or altered thiamine metabolism

Predisposing factors:

1. Acidosis or other digestive upset - changes ruminal flora
2. B. thiaminolyticus may produce thiaminase in rumen
3. Brain with ischemic focal necrosis

Clinical signs - severe and mild forms

a. feedlot cattle - severe form

b. pastured cattle - mild form
1) depression
2) anorexia
3) blindness
4) ear twitching, limb fasiculations
5) circle walking, head push, and salivate
6) normal temperature

Location of Lesion -
Cerebral cortex of both cerebral hemispheres

Character of Lesion
Necrosis, softening and edema ("malacia") of brain tissue
Lesion limited to narrow zone of deep cortex
Lesion often more marked in sides and bottom of sulci

Gross Appearance of Lesion -
May be difficult to detect in fresh brain
Cerebral gyri slightly swollen, yellow and soft
Cut surfaces show line of discoloration in deep cortex.
Affected laminar zones more obvious after formalin fixation
Diffuse brain swelling may cause hemorrhages elsewhere
Cerebellum may be compressed in posterior fossa of cranium
Wood's U.V. lamp in a dark room may outline necrotic areas

Diagnosis -
1. History
2. Signs
3. Lesions and laboratory data

Differential diagnosis -
1. TEM
2. Listeriosis
3. Rabies
4. Brain abscesses
5. Lead poisoning

Comment -
No other lesions are consistently present
Blood pyruvate concentrationis increased
Acidosis often a predisposing condition in pasture cases
Morbidity up to 25% in some pens; mortality exceed 50%

Thromboembolic Meningoencephalitis (TEM)

Occurrence -
1. Cattle .5-2 years of age
2. All seasons — mainly fall and early winter
3. May follow respiratory diseases

Etiology - Hemophilus somnus (Microaeophilic, gram negative bacterial rod)

Transmission -
1. Contact with carrier cattle or their fomites
2. Bacteria enter either respiratory or digestive systems

Pathogenesis -
Septicemic bacterial (Hemophilus) infection
Often follows episodes of inclement weather
May follow a mild or severe episode of respiratory disease
Hemophilus infection may be widespread but sub-clinical in carriers

Clinical signs -
1. One or more animals found
2. Pyrexia 40 41° C and anorexia
3. Muscular weakness
4. Knuckling at the fetlock
5. Stiffness, lameness, swollen joints and tendon sheaths
6. CNS signs - ataxia, circle-walking, head pushing, prostration, paresis, opisthotonos, coma, and convulsions

Location of Lesion -
May be single, few or multiple in the brain at random
Sudden death may be related to single, large brainstem lesion
Longer clinical course may have multiple surface lesions
Other lesions are always present, particularly:

POLYARTHRITIS AND TENDOSYNOVITIS

Character of Lesion -
Acute infarcts of brain
Leptomeningeal vasculitis and thrombosis
Fibrinous polyarthritis and tendosynovitis

Gross Appearance of Lesions -
Circumscribed brown-ish zones of necrotic brain
Lesions may be small (1-10mm) and innumerable
Some lesions may be large (15-30mm), few, even single
Distribution of infarcts is random - surfaces or deep
CSF is turbid, yellow-red and abundant
Synovial fluid is turbid, yellow and increased in limb joints
Tendon sheaths of limbs contain organizing yellow fibrin
Multifocal yellow or reddish yellow lesions may affect kidneys

Diagnosis -

1. History
2. Clinical signs
3. Necropsy
4. Culture of organisms

Differential diagnosis -

1. PEM
2. BUSS disease
3. Listeriosis
4. Lead poisoning

LISTERIOSIS

(Circling disease, silage sickness)

Occurrence -

1. All breeds, sexes
2. Confined cattle .5-2.5 years of age with silage as part of diet
3. Any season — more commonly winter and spring

Etiology -

Susceptible cattle are exposed to contaminated silage, feed or water. Abrasions in mouth caused by poor quality feed, silage, caustic irritants — allow causative bacteria to penetrate tissues innervated by trigeminal and hypoglossal cranial nerves. Organisms enter branches of nerve and move eventually to brain — especially medulla and pons.

Clinical signs -

1. Fever, depression, disorientation
2. Lateral deviation of head
3. Unilateral facial paralysis (ear droops, eyelid closes, nostril immobilizes)
4. Loss of eye preservation reflexes
5. Circling, recumbency, coma, death

Location of Lesion -

Restricted to brain stem and associated cranial nerves
Medulla and pons are affected
Trunks of 5th cranial nerves (possibly 7th and 10th)

Character of Lesion -

Non-suppurative cranial neuritis - microscopic
Non-suppurative bulbar leptomeningitis - microscopic
Invisible focal microabscesses in deep tissues of brain stem

Gross Appearance of Lesion -

Lesions *can not* be detected by naked eye examination
Essential to include brain stem for laboratory confirmation
Sagittally hemisect entire brain stem
Submit one half for microbiological examination
Submit other half in formalin for histopathologic examination

Diagnosis -

1. History
2. Clinical signs
3. Histopathology
4. Culture of brain

Differential diagnosis -

1. Brain abscess
2. Meningitis
3. BUSS disease
4. PEM, TEM
5. Middle ear infection
6. Traumatic facial paralysis
7. Lead poisoning

Comment -

Often related to feeding spoiled silage (up to 3 weeks ago)
Listeria organism often difficult and slow to isolate
Histopathologic features are pathognomonic

OTITIS MEDIA

Occurrence -

1. Cattle .5-2 years of age
2. All seasons, mainly summer
3. Any time during feeding period - usually first 30-60 days

Morbidity -

1:3000

Etiology -

1. Contaminated dips
2. Foreign bodies (ear ticks, awns)
3. Pyogenic bacteria

Pathogenesis -

1. External infection of via the eusthachian tube

Clinical Signs -

1. Degree of anorexia
2. Head rotation
3. Unilateral facial paralysis
4. Intermittent exudate from external meatus

Diagnosis -

1. History
2. Clinical signs

Differential Diagnosis -

1. Listeriosis
2. Traumatic facial paralysis
3. Unilateral sinusitus
4. Cervical arthritis
5. Ear tick infestation
6. Chronic lead poisoning

Loction of Lesion -

Generally unilateral
Involvement may include external ear canal
Involvement may include eustachian tube
Inner surfaces of external ear matted with dried or moist exudate
Saw cuts exposing tympanic cavity necessary to reveal lesion

Character of Lesion -

Purulent exudate in tympanic cavity and external ear canal
Tympanic membrane and ossicles destroyed
Thickening of bone around tympanic cavity

Gross appearance of lesion -

Semi-solid plugs or cores of yellow-white exudate
Irregularly thickened necrotic bone around bulla
Purulent exudate may be detectable by odor
Spinose ear ticks or grass awn may be observed

Treatment -

1. Systemic antibiotics
2. Local treatment
3.

"Realize" animal

Reference -

Disiases of Feedlot Cattle — Jensen and Mackey, Lea & Febiger, 1979.

SPONDYLITIS/SPINAL ABSCESS

Occurrence -

1. Cattle .5-2 years of age
2. Mature cows and bulls
3. All seasons
4. Sporadic

Etiology -

1. Trauma
2. Abscesses - Corynebacterium pyogenes, Fusibacterium necrophorum

Pathogenesis -

1. Possible prior bacteremia or pyemia
2. Possible infection introduced by Hypoderma grubs
3. Local abscess of vertebral body
4. Progressive pressure on cord
5. Fracture of vertebra

Signs -

May occur suddenly or slowly; may be severe or mild

Mild Signs -

1. Fever
2. Difficulty in rising
3. Stiff gait
4. Ataxia
5. Dragging rear feet

Severe form -

1. Complete posterior paralysis
2. Absence of spinal reflexes

Diagnosis -

1. History
2. Clinical signs
3. Rectal exam (?)
4. Hematologic studies? (leukocytes)

Differential Diagnosis -

1. Listeriosis
2. PEM
3. Riding injury
4. Spinal neoplasm
5. Pesticide intoxication
6. Tick paralysis
7. Arthritis
8. Myopathy (MHD)
9. Tetanus

Prognosis -

1. Unfavorable

Location of Lesion -

Most commonly, thoracic vertebra
Occasionally, lumbar or sacral vertebra
Rarely, cervical vertebra
Clinical signs suggest the probably location

Character of Lesion -

Localized abscess of vertebral body and surrounding tissue
Possible fracture at site - pre-existing or pathologic
Size of abscess related to duration of course

Gross Appearance of Lesion -

Usually involves a single vertebra
May extend to vertebrae on either or both sides
Sagittally hemisected vertebral body is necrotic
Bone gray or yellow, granular or pasty in texture
Intervertebral disc brittle or dissolved
Abscess swelling of ventral periosteum and other tissue
Fluctuant and with yellow/green creamy pus (Corynebacterium)
Firm with gray/white semi-solid pus (Fusibacterium)
Extends to epidural fat and spinal dura
Vertebral canal may contain hemorrhage or pus

Spinal cord may be softened or liquified
Septic embolism reflected in kidneys, lungs, heart valves, etc.

COCCIDIOSIS-RELATED CONVULSIONS
(Hemorrhagic diarrhea)

Occurrence -
1. Animals 6-9 months of age
2. Mainly fall and winter

Etiology -
Eimeria spp. (E. bovis and E. zurnii, enteric protozoa)

Clinical signs - nervous form
1. Epileptiform seizures
   a. occur spontaneously
   b. induced by excitation
2. Between seizures - animal looks normal
3. May or may not be diarrhea

Location of Lesion -
Diffuse brain swelling
Often not related to concurrent-enteritis or diarrhea

Character of Lesion -
Edema of brain tissue

Gross Appearance of Lesion -
Brain generally enlarged, pale yellow or white
Brain tissue is very soft and watery
Displacements within cranium may occur
Cerebellum compressed in posterior cranial fossa
Median lobe (vermis) of cerebellum may obtrude into foramen magnum

Diagnosis -
1. History - concurrent outbreak of coccidiosis
2. Signs
3. Necropsy - not very often helpful

Differential diagnosis -
1. TEM, PEM
2. Rabies
3. Lead poisoning
4. Salmonellosis
5. Calculosis

Comment -
Relationship to enteric coccidiosis remains obscure
May result from hypoxemia (anemia, toxemia)
May result from dehydration and electrolyte imbalance
May result from acidiosis caused by bicarbonate loss
Dehydration-related hypomagnesemia may exist
Toxin may be produced by coccidia or other enteric microflora

RABIES
(Hydrophobia)

DEFINITION
An acute, bite-transmitted, inevitably fatal, nonsuppurative encephalomyelitis that occurs sporadically in cattle.

Occurrence -
1. All breeds, sexes and ages over 6 months
2. Low incidence in feedlot cattle
3. High incidence in cattle from Central and South America

Etiology -
1. Lyssavirus (Rhabdovirus group)

Clinical Signs -
(Signs are inconsistent)
1. Vampire bat transmitted rabies
   a. gradual ascending paralysis with flaccid tail
   b. recumbency
   c. posterior paralysis while eating
2. Nonvampire bat transmitted bovine rabies
   a. rumen atony - suggesting traumatic gastritis, or water deprivation
   b. erratic, violent, aggressive behavior
   c. *characteristic bellow,* an *"alert listening" ear position,* and *tenesmus*
   d. temp n - 104°F
   e. sexual excitement

Necropsy findings - Gross
1. Dehydration
2. Traumatized and abraded carcass

Histologically -
1. Nonsuppurative encephalomyelitis
2. Ganglioneuritis (fifth nerve ganglion)

Diagnosis -
1. Suspicious signs
   a. dysphagia
   b. intermittent raucous bellowing
   c. paralysis
2. Laboratory examination of fresh frozen brain tissue
   a. identifying rabies virus
   b. + FA test
   c. mouse inoculation tests
3. Histologic examination of fixed hemisected brain

Differential diagnosis - any CNS case
1. Esophageal choke
2. Nervous acetonemia
3. Polioencephalomalacia
4. TEME
5. neural IBR, pseudorabies
6. lead poisoning and organophosphage poisoning
7. plant poisonings

The Siteguard advantage.

Other products	Siteguard products	Additional Siteguard protection
2-way Blackleg (Blackleg, Malignant Edema)	Siteguard M (3 components)	Sord (Blackneck)
Triple Blackleg (Blackleg, Malignant Edema, Shipping Fever)	Siteguard M plus Pasteurella (5 components)	Sord (Blackneck)
3-way Blackleg (Blackleg, Malignant Edema, Novyi B)	Siteguard ML (5 components)	Sord Novyi D*
Novyi Bacterin (Novyi D*)	Siteguard L (2 components)	Novyi B
4-way Blackleg (Blackleg, Malignant Edema, Sord, Novyi B)	Siteguard ML (5 components)	Novyi D*
7-way Blackleg (Blackleg, Malignant Edema, Sord, Novyi B, Overeating Disease Types B, C & D)	Siteguard MLG (8 components)**	Novyi D*

*Known officially in U.S.A. as *Cl. haemolyticum*

**Certain toxins of *Cl. perfringens* Types C and D protect against Type B. True significance of Type B in U.S.A. unknown.

Unless you vaccinate with Siteguard,™ your calves are short on blackleg-type protection.

Your calves are not getting all the blackleg-type protection they need if you're still using a 2-way, a 4-way or a 7-way blackleg vaccine.

When you switch to a Siteguard™ vaccine, you can automatically upgrade your prevention program. Because Siteguard vaccines can give animals clostridial sudden death protection no other vaccines provide.

EXTRA PROTECTION. For example, you vaccinate against blackleg and malignant edema with a 2-way blackleg vaccine. But there's another disease in the muscle group that produces death even faster—*sord*. No 2-way vaccine covers *sord*. Siteguard M does.

And like Siteguard M, the Siteguard vaccine line offers extra blackleg-type disease protection. The reason is simple. Only in Siteguard vaccines are components grouped according to the three types of tissue in which clostridial infections are primarily triggered—muscle, liver and gut. So where other vaccines come up short, Siteguard covers each group completely.

This spring, provide the extra vaccine protection your calves may need. Ask for a Siteguard vaccine.

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