DIFFERENTIAL DIAGNOSIS OF CENTRAL NERVOUS SYSTEM TOXICOSES IN FEEDLOT CATTLE

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Toxicant	Occurrence	Etiology	Pathogenesis	Cl. Signs	Postmortem Findings	Diagnosis	Treatment
Lead	Seasonal; spring, summer and fall. Most common single toxicosis in cattle.	Paint, used oils, grease batteries machinery, Industrial contaminants, rubbish.	Chronic accumulation or acute; six mg/kg daily may lead to toxicosis; Poorly absorbed but toxic blood levels w/in 2-4 days; General cellular poison.	CNS Blindness, muscle twitching, head bobbing Hyperirritability, Depression Ataxia Circling Pushing G-I Grinding teeth, Excessive salivation, Anorexia, Tucked Abdomen diarrhea if oil source.	Acute: Nondescript, mild gastritis, focal hemorrhage in heart, GI Subacute: laminar cortical necrosis of cerebrum	Blood lead >0.35 ppm Liver>3.0 pp. Kidney>3.0 ppm Clinical signs present	EDTA, subcutaneously or slow IV; 1-2% in 5% dextrose at rate of 110mg/kg, twice daily for 2 days, skip 2 days followed by 2 days of treatment. Oral MgSO 1 lb./1,000 lb 4'½-animal (>1.0%)

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Organo-chlorine Insecticides	Improper sprays, dips; Accidental feed contamination. May cause toxicosis and/or meat residues	Soil/crop granules: aldrin dieldrin, Heptachlor, Endrin. Others: toxaphene Methoxychlor Lindane Mirex	Hyperstimulation or depression of the CNS; Onset of signs minutes to a few days, usually within 24 hours.	Tonic-clonic intermittent seizures; may be progressive or explosive, hyperirritable, belligerence; Fasciculations of facial, cervical muscles, eyelids, other body muscles; chewing movements, abnormal posturing; Some animals have severe CNS depression; Elevated body temp. 104-108°F	Nonspecific, Congestion of abdominal and thoracic organs, Parboiled appearance due to high fever.	Brain: 3-5 ppm. Variable concentrations in adipose tissue	Light anesthesia (pentobarbital) Remove dermal w/detergent. 1-2 lbs activated charcoal. Saline Cathartic.

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Salt/ Water Deprivation	Using salt to limit feed intake; Accidental contamination of feed or water deprivation	High NaCl (1.0%) in diet with water deprivation	High plasma Na+ in CSF passively; causes reduced brain glycolysis and energy production Na+ cannot escape CSF because energy required; Brain edema and malacia results	Thirst, hyperirritability, Epileptiform seizures CNS depression Knuckling of pasterns with muscle weakness	Edema and Malacia of cerebral cortex	Plasma and/or CSF Na^+ 150 mEq/l; Brain tissue greater 18 ppm. Na^+	Give fresh water carefully; if seizures, manitol diuretic may be beneficial

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Urea NPN	Unaccustomed animal placed on diet in which a major proportion of protein is from non-protein nitrogen, especially urea	Urea most hazardous of all NPN sources; 0.5 0.5 g/Kg b.w. may be lethal to unaccustomed animals	Urea hydrolyzed to ammonia (NH_4OH). Excess NH_3- that cannot be converted into urea by liver causes toxicosis	Muscle faciculations, weakness and tonic seizures; bloat; rapid death	Congestion of GI mucosa; congestion and edema of lungs	Rumen pH 7.0; Rumen fluid 80 mg/100ml Ammonia N; serum or plasma 2.0 mg/100 ml Ammonia N	Five-ten gallons of cold water orally 1-3 gal 5% acetic acid (vinegar)
Organo-phosphorus Carbamate Insecticides	Treatment for internal and external parasites; accidental contamination of feed	Systemic o.p. most common coumaphos, chlorpyrifos, Ronnel r, Ruelene r, others include Fenthion dichlorvos	Inhibition of acetylcholinesterase (AChE). May be recyclying of certain cpds through the rumen.	Central nervous hyperexcitibility or depression; muscle weakness, rumenatomy plus muscarinic signs in acute episodes.	Non-specific; Hyperemia and focal hemorrages in abdominal and thoracic organs, lung edema, excess fluid in rumen in some cases.	RBC/whole blood AChE at least 50-90%; chemical analysis for Op cpd in skin rumen contents, liver, kidney, adipose tissue.	Activated charcoal orally (1-2 lbs); atropine ¼mg/lb (IV and SQ) 2—PAM (10-20mg/kg SQ).
Soybean Overload	Excess consumption of raw soybeans or soybean Derivatives.	Ammonia Released; oil may form slate gray soap-like material.	Belligerent, hyperactivity, aggressive behavior; seizures rare; thirst and acute death	Rumen contents slate-gray swollen soybeans visible; excess fluid is rumen.	Characteristic rumen contents; pH 6.5 or greater	Rumenotomy or rumen lavage.	
Rumen overload (acidosis)	Excess grain (corn, wheat, barley, milo) to unaccustomed animals; may occur in animals that are temporarily off feed because of illness, inclement weather, etc. May occur with corn silage and other "non-grain" feeds; may occur due to monensin feed additive.	Not completely understood; excess rumen lactic acid formation; Gram negative rumen organisms disappear; replaced by Gram-Positive (e.g. Clostridia) May be endo-toxic shock caused by lysis of micro-organisms.	Rumen atony, inappetence, mild bloat, dehydration, increased thirst, decreased urine; incoordination and ataxia, weakness, CNS depression; anorexia, grinding teeth, fluid distention of abdomen; subnormal body temperature; weak followed by death.	Hemconcentration acid rumen, pH<4.5; excess rumen fluid and contents; blood pH<7.2; rumen micro-organisms predominantly Gram positive; prolonged cases: rumenitis, liver abcesses, laminitis.	Circumstantial evidence;pH of rumen fluid <4.5; clinical signs and course; blood pH<7.2; rumen, micro-organisms mostly Gram-negative	Poor prognosis in severe cases rumen lavage oral antacids correct dehydration and acidosis, antihistamines; oral thiabendazole to prevent mycotic rumenitis.	

Other toxicants and toxins that may require differential diagnosis:

1) Carbon tetrachloride/carbon difulfide (80/20) used as grain fumigant may cause severe gastro-intestinal pain, salivation, grinding teeth, tongue lolling, anorexia, and diarrhea, associated with severe hepatic damage.

2) Ergot alkaloids and tremoxigenic mycotoxin may contaminate certain small grain and grasses. They cause hyperactivity varying from fine muscle tremors to ataxia. Animals develop exaggerated high-stepping action which progresses to stumbling and falling with tonic-clonic seizure activity.

3) Mercury (inorganic and organic compounds) may occasionally contaminate cattle feeds. Severe subacute CNS involvement may occur, including depression, anorexia, fever, excessive salivation, depilation, eczema, pruritis, bronchial catarrh, lacrimation; there may be G-I mucosal hemorrhages and catarrh; swollen lymph nodes, nephrosis, hepatosis, and severe brain changes.

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