Diseases associated with inorganic and farm chemicals

Introduction

Suspicion of poisoning is aroused when illness occurs in a number of previously healthy animals; all affected at the same time and showing the same signs and necropsy findings, to the same degree of severity. These conditions may be similar to some infections, metabolic and nutritional deficiency diseases.
The distribution of poisonous plants often occurs in some a geographical area; particular industrial enterprises may create poison hazards in local areas; certain agricultural practices, including the spraying of orchards, the dipping or spraying of cattle for ectoparasites, and the use of prepared concentrate feed for pigs and cattle, may also lead to poisoning in groups of animals.
So many chemical agents are used in agriculture today that a section of miscellaneous farm chemicals likely to be associated with the poisoning of animals has been included. The appearance of clinical illness soon after feeding, after a change of ration, after medication or spraying, or after change to new pasture, is a common history in many outbreaks of disease associated with chemical agents.

The report which accompanies material for toxicological analysis should include:

- A full record of history
- clinical signs
- necropsy findings
- Particular results of a search of the environment for access to a poison.
- If the animal has been treated
- the drugs that were used
- The dates of administration should be given as they may create difficulties for the analyst.
- The poison or group of poisons suspected should be defined.

Specimens for analysis of suspected poisons should include:

- A sample of the suspected source material.
- Next most important is a specimen of alimentary tract contents, so that ingestion of the material can be proven
- A sample of tissue, usually liver, to prove that absorption of the poison has occurred.
- Kidney also provides a route for concentrating many toxicants for excretion and is an important specimen for chemical poisoning.
- Most toxic chemicals are ingested but percutaneous absorption and inhalation must be considered as possible portals of entry.
- One of the advantages of an examination of alimentary tract contents is that qualitative tests can be carried out and in many cases this determines whether or not further examination of tissues is necessary.
- Additional specimens required other than liver and alimentary tract and contents, vary with the poison and the following list is suggested for the common chemicals:

➢ Arsenic - kidney, skin, and hair

- Lead - kidney, liver, bones, and whole blood
- Phosphorus - kidney and muscle
- Mercury - kidney, brain if organomercurials are suspected
- Copper - kidney, liver, and blood
- Sodium chloride - alimentary tract and contents, brain, and serum
- Fluorine - bones, teeth, and urine,contaminated forages
- Hydrocyanic acid - ingesta in a filled and airtight container, blood and muscle
- Nitrate and nitrite - ingesta (plus chloroform or formalin) in an airtight, filled container, blood, ocular vitreous humor
- Strychnine - blood, kidney, and urine
- Insecticides - liver, kidney, brain, fat, ingesta. Careful packing of specimens is necessary to avoid loss of some poisons by escape as gas or conversion by bacterial fermentation, and to prevent contamination.

NB:

1. No preservative should be added except in the case of suspected nitrite poisoning.
2. If a preservative is necessary because of distance from the laboratory, packing in dry ice or ethyl alcohol (1 mLi g of tissue) is advisable
3. In the latter instance a specimen of the alcohol should also be sent.
4. Ingesta and tissues must be kept separate as diffusion is likely to occur between the two.
5. Specimens should be packed in glass or plastic to prevent contamination by lead in soldered joints of cans.

Collection of samples

1. A suitable amount of material should be submitted for analysis: 1 kg of ingesta, 1 kg of liver, 0.5 kg of kidney, and proportionate amounts of other viscera are suggested to cover all contingencies.
2. Urine (200 mL or whatever is available) may allow quick analysis of some toxicants.
3. Both blood and serum are helpful for rapid testing of some toxicants and for characterizing a potential poisoning through complete blood count and clinical chemistry.
4. Special action is needed when plant poisoning is suspected. Pica due to mineral deficiency or some other association may encourage toxic plant consumption
5. Deliberate or criminal poisoning is often suspected but is rarely proved. If there is a strong suspicion of criminal poisoning, or if litigation appears possible in accidental poisoning, specimens should be collected in duplicilte and placed in sealed containers in the presence of witnesses. A complete set of specimens should be available to both plaintiff and defending parties for independent analysis. Also, if litigation appears possible, the veterinarian should make detailed observations of the clinical, pathological, and epidemiological findings and "record them in detail. The taking of photographs of affected animals and the environmental surroundings is also recommended for future reference and documentation if necessary.

Mineral tolerance of animals

One of the very important aspects of toxicology as it applies to agricultural animals is the determination of levels of dietary constituents which the animals will tolerate for a limited period without impairing their performance and without producing unsafe residues in products destined for the human food chain. (The down table is an adapted summary of the information).

	Cattle	Sheep	Pig	Horse
Arsenic mg/kg				
inorganic	50	50	50	(50)
inorganic	100	100	100	(100)
Cobalt mg/kg	10	10	10	(10)
Copper mg/kg	100	25	250	(800)
Fluorine mg/kg				
dairy	40 breeding	60	150	(40)
mature beef	50 finishing	150		
finishing beef	100			
Iodine mg/kg	50	50	400	(5)
Iron mg/kg	1000	500	3000	(500)
Lead mg/kg	30	30	30	(30)
Mercury mg/kg	2	2	2	(2)
Molybdenum mg/kg	10	10	20	(5)
Phosphorus %	1	0–6	1–5	(1)
Selenium mg/kg	(2)	(2)	(2)	(2)
Silicon %	(0–2)	(2)	2	(2)
Sodium chloride %				
lactating	4	9	8	(3)
non-lactating	9			
Sulfur %	(0.4)	(0.4)	no data	no data
Zinc mg/kg	500	300	1000	(500)

Courtesy of National Research Council, USA (Figures in parentheses are extrapolation from data on other species)

The maximum tolerance levels of dietary minerals for domestic animals

Principles of treatment in cases of poisoning

There are certain principles which apply to all cases of poisoning. The three main principles are:

- Removal of the residual poison from the alimentary tract or skin
- Provision of chemical and physiological antidotes to the poison that has been absorbed.
- Effective supportive care, nursing, and convalescent care.

In farm animals:

A. gastric lavage and emetics are of little or no practical value and the removal of residual poison from the alimentary tract depends largely upon the use of adsorbents and purgatives. The only effective adsorbent is activated charcoal. The dose rate is 1-3 g/kg BW repeated as necessary. It adsorbs chlorinated hydrocarbons, organophosphorus compounds, mycotoxins and plant alkaloids, the common feed additives, antibacterial agents and bacterial toxins. It does not adsorb cyanide, heavy metals, halogens, nitrite, alcohols, caustics, sodium chloride, or chlorate.

B. A purgative is necessary to remove the combined adsorbent and poison; it can be administered simultaneously with the adsorbent. The use of irritant purgatives is not advisable when the poison is an irritant and has already been associated with gastroenteritis, and non-absorbable oily purgatives (e.g. mineral

oil) are preferable in these cases. Saline purgatives (sodium sulfate) are of value in the treatment of non-irritant poisons such as cyanogenetic glucosides.

C. Neutralization of residual poison in the alimentary tract includes use of Oxidizing agents or tannic acid preparations for precipitating alkaloids; proteins, including milk and eggs, are effective chemical antidotes for poisons that coagulate proteins; lead is precipitated by the addition of sulfates to the alimentary tract contents.

D. Poison that has already been absorbed can in some instances be inactivated or its excretion facilitated by the provision of chemical antidotes. For instance, sodium nitrite and sodium thiosulfate are effective systemic antidotes to hydrocyanic acid, and calcium versenate is an effective antidote against lead.

E. Treatment of the effects of a poison includes provision of physiological antidotes, e.g. the injection of a calcium salt in cases of overdosing with magnesium salts.

F. Ancillary or supportive treatment, including the provision of fluids in dehydration due to diarrhea, demulcents in gastroenteritis, sedatives in excitement, stimulants in cases of central nervous system depression, all treat the effects of poisoning.

G. It is essential when undertaking the treatment of animals for poisoning, especially those which are producing milk or which are destined to become meat in a short time, to take into account the possible unsuitability of the product for human consumption because of the presence of the poison or the antidote. Carefully planned sampling in concert with regulatory authorities can avoid unwanted contamination of the human food supply.

Diseases associated with inorganic poisoning

Lead poisoning (Plumbism)

Etiology

Accidental ingestion of lead or ingestion of feed or grazing pasture containing excessive lead.

Epidemiology Occurs in all age groups.

One of the most common poisonings of farm livestock especially in young calves after turn out in spring. In cattle, usually sporadic and due to ingestion of single source of lead but outbreaks occur when feed is contaminated. High case fatality rate if untreated. Sources include discarded lead batteries, lead-based

paints, industrial sources of lead, pastures near motor vehicle highways and smelters. Occurs in sheep and horses grazing contaminated pastures.

Pathogenesis

Regardless of the chemical form of the ingested lead, only a small proportion is absorbed because of the formation in the alimentary tract of insoluble lead complexes which are excreted in the feces. Of the lead absorbed, some is excreted in the bile, milk, and urine and the blood levels of lead provide a reliable indication of the lead status of the animal. Urine levels **may not be as reliable**. Deposition in tissues occurs, particularly in the liver and renal cortex and medulla in acute poisoning and in the bones in chronic poisoning: The deposition of lead in the brain is not high compared to other tissues but deposited lead is gradually liberated from tissues into the bloodstream and excreted via the bile and urine. Consideration must be given to these aspects of lead metabolism when assessing the results of chemical analyses of tissues. Although acute lead poisoning usually develops rapidly there may be a delay of several days after toxic material has been ingested before clinical signs appear.

Toxic effects of lead

The toxic effects of lead are manifested in three main ways:

- Lead encephalopathy
- Gastroenteritis
- Degeneration of peripheral nerves.
- Others

A. The blue **'lead-line'** at the gum-tooth junction, which is seen in man and the dog, does not commonly occur in ruminants because of failure to form tartar but present in severe poisoning in the horse and small ruminants. The 'lead line' is a deposit of lead sulfide formed by the combination of lead with sulfide from the tartar.

B. Lead is transferred across the placental barrier and high liver levels occur in the lambs of ewes fed more than normal amounts of lead.

C. Anemia may occur in chronic lead poisoning. The erythrocytes are microcytic and hypochromic, and reticulocytosis and basophilic stippling may be observed.

Clinical signs

Cattle: Acute - convulsions, blindness, tremors, charging, rapid death unless treated. Subacute - blindness, stupor, head-pressing, rhythmic ear tics, blepharospasm, rumen stasis and eventual death.
Sheep: Lambs on pasture with posterior paresis.
Horses: On pasture. Signs highly variable. Inspiratory dysnea, roughened hair coat, weight loss most commonly. Occasionally convulsions.

Clinical pathology

Lead levels in blood, feces, liver, kidney; elevated porphyrins in blood Lesions Encephalopathy, degeneration of liver and kidney; pale musculature, brain laminar cortical necrosis, intranuclear renal inclusion bodies.

Table 32.2 Lead levels in blood and feces of normal and poisoned animals

	Lead levels (ppm)	
Specimen	**Normal**	**Poisoned**
Whole blood (ruminants and horses)	0.05–0.25	More than 0.35 (deaths commence at 1.0)
Whole blood (pigs)	0.05–0.25	1.2
Feces (dry matter) (cattle)	1.5--35	Up to 1000
Pasture		350

Diagnostic confirmation Toxic levels of lead in blood and tissues.

Differential diagnosis list

Horses: See Table 22.1.
• Laryngeal hemiplegia
• Viral encephalomyelitides of West Nile virus
• Rabies
• Hepatoencephalopathy due to hepatotoxic plants
• Equine degenerative myeloencephalopathy
• Protozoal encephalomyelitis
• Equine motor neuron disease
• Horsetail poisoning
• Chronic weight loss
• Chronic upper respiratory tract disease
• Botulism
Cattle: See Table 32.3.
• Polioencephalomalacia
• Hypovitaminosis-A
• Ophthalmitis
• Hypomagnesemic tetany
• Nervous acetonemia
• Arsenic poisoning
• C laviceps paspali toxicity
• Meningoencephalitis
• Rabies

Sheep:
• Enzootic ataxia
• Polyarthritis
• Muscular dystrophy

Treatment

Calcium versanate and thiamin hydrochloride. Calcium versanate (calcium disodium ethylenediamine tetra-acetate, Ca EDTA) has been used successfully in cases of lead poisoning produced experimentally in calves and in natural cases in cattle. Ca/EDTA is available as a 6.6% solution for N administration.

The manufacturer's recommendations are to use 1 mL/kg BW per day given in divided doses 2-3 times daily over a period of 3-5 days.
Control: Prevent access of animals to sources of lead.

Table 22.1 Diseases of horses characterized by signs of intracranial or disseminated lesions of the central nervous system

Disease	Etiology and epidemiology	Clinical and laboratory findings	Treatment and control
Infection causes			
Viral encephalomyelitis (Western, Eastern, Venezualan: WEE, EEE, VEE)	Summer season. Insect vector, usually mosquitoes Young non-vaccinated horses at greatest risk, outbreaks may occur	Stage of slight hyperexcitability and mild fever initially, impaired eyesight, circling and walking. Stage of mental depression, somnolence, leaning, feed hanging from mouth, unsteady. Stage of paralysis, unable to swallow, weakness, recumbency; dies 2–4 days after onset. Serology for diagnosis	Supportive therapy, thick bedding. Recovery rate 60–75% Vaccinate foals at 6 months of age and other horses for the first time, twice 2 weeks apart and once or twice annually thereafter
Rabies	All age groups, knowledge of disease in area, wildlife. Usually single animal affected. Not common	Ascending paralysis, hyper-salivation, will bite. Ataxia and paresis of hindlimbs, lameness, recumbency, pharyngeal paralysis, colic, loss of tail and sphincter tone, fever. Dies in 1 week. Immunofluorescent antibody testing on brain for positive diagnosis	No treatment. All die. Vaccinate horses if anticipate outbreak
Herpesvirus myeloencephalopathy (EHV-1)	Can occur as outbreaks. Neurologic disease usually preceded by fever. Mature horses	Symmetrical ataxia and paresis, bladder paralysis, recumbency may occur, spontaneous recovery possible, CSF (hemorrhage or xanthochromia). Vasculitis with subsequent focal malacia in gray and white matter of brain and spinal cord	No specific therapy. Anti-inflammatory drugs may be useful. Use of corticosteroids is controversial.Recovery may occur spontaneously
West Nile encephalomyelitis (WNE)	West Nile virus. Late summer in temperate regions. Can occur as epizootics. Now enzootic in most of North America	Fever, muscle fasciculations, weakness, ataxia, depression, cranial nerve disease, recumbency. Prominent signs of spinal cord precede sign of intracranial disease in most cases	Supportive. Antiserum. Interferon. Anti-inflammatory. drugs including corticosteroids. Prevention by vaccination
Borna	Virus. Direct transmission. Germany and other European countries. Disease is recorded in Japan. Low morbidity, high case fatality rate	Pharyngeal paralysis, muscle tremor, flaccid paralysis, course 1–3 weeks. Viral encephalomyelitis with inclusion bodies	No treatment
Japanese encephalitis	Japanese encephalitis virus. Sporadic. Asia including Japan and China, parts of Oceania including New Guinea and Torres Strait. Pig is mammalian amplifying host. Vector mosquitoes, birds infected	Fever, lethargy, jaundice, dysphagia, incoordination, staggering, recovery in 1 week. Serology	Spontaneous recovery. Vaccination in endemic areas
Protozoal myeloencephalitis	*Sarcocystis neurona*. Single animal affected. Infectious but not contagious	Any central nervous system disorder. Usually causes ataxia but can cause cerebral and cranial nerve disease	Antiprotozoal medications (pyrimethamine + sulphonamide, ponazuril, or nitazoxanide). Vaccine available in USA but not recommended
Cerebrospinal nematodiasis (verminous encephalitis)	Migration of larval stages of *Strongylus vulgaris, Habronema* sp., and Filaroides. *Micronema deletrix (Helicephalobolus) deletrix*. Not common	Clinical signs referable to gray matter lesions are common. Hypalgesia, hyporeflexia, hypotonia, muscle atrophy and cerebral, cerebellar and cranial nerve involvement. Progressive encephalitis, incoordination, sensory deficits, blindness in one or both eyes, course of several days. Pleocytosis of CSF. Hemorrhage and malacia of thalamus, brain stem, cerebellum	Ivermectin or moxidectin at usual doses. High dose benzimidazole. Anti-inflammatory drugs. Parasite control
Brain abscess	Sporadic. Often a complication of strangles	Obtunded mentation, variable signs of intracranial disease. Leukocytosis. Variable pleocytosis and increased protein concentration in CSF. CT scan	Antimicrobials. Surgical drainage Prognosis is poor
Physical			
Traumatic injury to the brain	History of traumatic injury (falling, rearing-up and falling backwards)	Coma, depression, hemorrhage from nose and ears, blindness, cranial nerve deficits. Often rupture of longus capitus muscle	Anti-inflammatory drugs, mannitol. Fair to poor prognosis
Facial nerve paralysis	Associated with prolonged surgical recumbency and compression of facial nerve	Facial nerve paralysis lasting several days. Paralysis of ear, eyelid, lip, nostril on one side. No alteration in sensation or vestibular function	Supportive

Table 22.1 (*Cont'd*) Diseases of horses characterized by signs of intracranial or disseminated lesions of the central nervous system

Disease	Etiology and epidemiology	Clinical and laboratory findings	Treatment and control
Lightning strike	Observed lightning strike or history of recent thunderstorm activity	Death is most common. Horses that survive strike often have prominent signs of vestibular disease	Supportive. Recovery is possible
Fracture or arthritis of the temporal-stylohyoid articulation, otitis media	Sporadic in older horses	Acute onset circling, head tilt, nystagmus, unilateral facial paralysis, dysphagia	Antibiotics, anti-inflammatory drugs, supportive care
Intoxications			
Horsetail poisoning (*Equisetum arvense)*	Ingestion of plants mixed with hay. Not common	Incoordination, swaying from side to side, muscle tremor recumbency, bradycardia, cardiac arrhythmia	Thiamine parenterally. Good response
Equine leukoencephalomalacia (fumonisin toxicosis)	Horses eating moldy corn grain contaminated with *Fusarium moniliforme* fungus	Muscle tremor, weakness, staggering gait, dysphagia, depression	Nil
Hepatoencephalopathy associated with hepatotoxic plants (*Crotalaria, Senecio* and *Amsinckia*)	Horses on inadequate pasture forced to eat poisonous plants. More than one animal may be affected. Geographical distribution	Develops slowly, commonly ill for 2–3 weeks previously, depression, pushing, ataxia, hypertonic face and lips, yawning, compulsive walking, loss of weight, icterus, photosensitization occasionally. Serum liver enzymes elevated and liver function tests abnormal. Hyperammonemia. Gross and histopathological liver lesions	No treatment. Prevent access to poisonous plants
Lead poisoning	Grazing on pastures contaminated by atmospheric lead from nearby factories, not common now	Usually a chronic disease. Inspiratory dyspnea due to paralysis of recurrent laryngeal nerve. Pharyngeal paralysis, dysphagia, aspiration pneumonia, paralysis of lips, weakness and recumbency. Ingestion of large amounts causes subacute form similar to that seen in cattle	Calcium versenate
Yellow-star thistle poisoning (*Centaure* sp., anigropallidal encephalomalacia of horses)	Ingestion of yellow-star thistle in California and Australia. Summer months on weedy pasture	Difficult prehension, fixed facial expression with mouth held half open, hypertonic face and lips, persistent chewing movements and rhythmic protrusion of tongue, yawning and somnolence but easily aroused, aimless walking, slight stiffness of gait, high mortality Malacia of globus plants pallidus and substantia nigra	No treatment. Prevent access to poisonous plants
Botulism	Ingestion of preformed toxin of *Cl. botulinum* in decaying grass or spoiled silage, hay or grain. Sporadic in horses. Endemic in foals in some areas of North America	Flaccid paralysis of skeletal muscles leading to weakness, stumbling and recumbency. Mentation normal. Skin sensation normal. Paralysis of tongue and thoracic muscles. Die in 2–4 days. Some recover. Filtrates of intestinal tract into laboratory animals	Supportive therapy, antitoxins. Vaccination in enzootic areas. Prevent contamination of feed by animal carcasses
Tetanus	Wounds infected with *Cl. tetani*. Sporadic	Generalized tetany of all skeletal muscles. Fever, hyperesthesia, protrusion of third eyelid, trismus, recumbency followed by tetanic convulsions, die in 5–10 days	Prognosis unfavorable. Dark stall, penicillin, muscle relaxants, supportive therapy and antitoxin parenterally or into subarachnoid space Toxoid vaccination
Metabolic and idiopathic			
Lactation tetany	Lactating mares, suckling foals Hypocalcemia	Acute onset of generalized stiffness, trismus, no hyperesthesia, no prolapse of third eyelid, diaphragmatic flutter, soft heart sounds. Serum hypocalcemia	Rapid response to calcium borogluconate intravenously
Idiopathic epilepsy of Arabians	Single horse. first noticed from shortly after birth up to 6 months of age. Etiology unknown	Recurrent episodes of typical clonic tonic convulsions lasting 10–15 minutes, loss of consciousness, sweating, tachycardia, spontaneous defecation. No lesions	Control seizures with phenobartibal or potassium bromide. Spontaneous recovery as foals mature
Idiopathic epilepsy of adult horses	Sporadic disease. Unknown cause. Can be associated with brain lesions detectable on EEG or CT	Tonic-clonic convulsions. Variable periodicity and intensity	Control seizures acutely with diazepam and in long term with phenobarbital and/or potassium bromide. Spontaneous recovery unlikely

Table 22.1 (*Cont'd*) Diseases of horses characterized by signs of intracranial or disseminated lesions of the central nervous system

Disease	Etiology and epidemiology	Clinical and laboratory findings	Treatment and control
Cerebellar hypoplasia of Arabian and Swedish Gotland foals	Inherited. Signs noticeable from 2 to 6 months of age	Defective eye blinks, ataxia, headnodding, slight tremor of head and neck, intention tremor of the head, high-stepping gait, difficulty in rising, legs wide apart, difficulty in jumping over obstacles, fall backwards if dorsiflex head and neck. Cerebellar hypoplasia grossly or histologically	Eliminate carrier animals
Lower motor neuron disease	Associated with stabling and no access to pasture. Sporadic. North America and Europe. Low serum vitamin E concentrations	Weight loss, weakness, muscle fasciculations, maintained appetite. Normal mentation. Low serum vitamin E concentration. Diagnosis by muscle biopsy	No definitive cure. Some cases stabilized with administration of oral vitamin E. Poor prognosis for return to function

Note: *Other less common diseases affecting the nervous system of horses include: space-occupying lesions (cholesteatomas of old horses, tumors), intracranial myiasis due to migration of* Hypoderma bovis, *hydrocephalus in young horses, the accidental injection of an ataractic drug into the carotid artery and bacterial meningitis in young horses as a sequel to streptococcal infection.*

Differential diagnosis of lead poisoning in cattle

Table 32.3 Differential diagnosis of diseases of cattle with clinical findings referable to brain dysfunction

Disease	Epidemiology	Clinical findings	Clinical pathology and pathology	Response to treatment
Lead poisoning	All ages calves and cows on pasture with access to dumps. Discarded lead batteries, used crankcase oil, lead-based paint common sources. Case fatality rate high	Acute in calves. Blindness and 'chewing gum' champing of jaws, convulsions, charging, rapid death. Subacute in adults: blindness, stupor, head-pressing, grinding teeth, rumen static, protozoa dead	Blood and tissue for lead. Encephalomalacia	Will respond favorably to treatment in early stages if not too severe but most cases do not return to normal. Calcium versenate and thiamin hydrochloride. Must be concerned about disposition of meat and milk of treated animals
Polioencephalomalacia	Grain-fed rapidly growing feedlot cattle. May occur on pasture containing plants and water high in sulfates. Outbreaks occur	Sudden onset, blindness, tremors and shaking of head, twitching of ears, head-pressing, opisthotonos, nystagmus, strabismus, rumen contractions normal, CSF pressure increased	Blood biochemistry (see text). Brain for histopathology	Responds to thiamin in early stages. Those due to sulfate toxicity may not respond
Hypovitaminosis A	Calves 6–8 months of age most commonly but mature cows too off dry summer pasture (CSF form). Young rapidly growing cattle fed deficient ration for several months (ocular form)	CSF form: sudden on set; syncope and convulsions followed by recovery, eyesight and pupils normal. Nyctalopia. CSF pressure increased. Ocular form: blindness in daylight, pupils dilated and fixed, optic disc edema. Syncope and convulsions may also occur. Usually preceded by nyctalopia but missed by owner	Plasma and liver vitamin A. Optic nerve constriction. Squamous cell metaplasia of parotid ducts	CSF form: recover in 48 hours following treatment with vitamin A injections. Ocular form: will not recover because of optic nerve degeneration
Haemophilus meningoencephalitis (thromboembolic meningoencephalitis)	Feedlot cattle (8–12 months), outbreaks, preceded by respiratory disease in group. High case fatality if not treated early	Found down, fever common, ataxic, not usually blind, fundic lesions, irritation signs uncommon, weakness and paresis common, synovitis, laryngitis, pleuritis, May die in 8–10 hours. Myocardial abscesses may also occur	Neutrophilia CSF contains neutrophils. Typical gross lesions in brain. Pleuritis, pneumonia, synovitis, myocardial abscesses	Respond favorably to antimicrobials if treated early. Later, high case fatality rate
Listeria meningoencephalitis	Sporadic. Fed silage. Yearlings and adults	Unilateral facial paralysis, deviation of head and neck, mild fever, endophthalmitis, may be recumbent	CSF for cells. Brain for histopathology	Recovery may occur. Antimicrobials. Residual signs in survivors common
Nervous signs with coccidiosis (see text)	In 20% of young cattle affected with dysentery due to coccidiosis. Case fatality may exceed 50%	Tonic–clonic convulsions, normal eyesight, hyperesthesia, normal temp., dysentery, may live 2–4 days	Oocysts in feces	Unfavorable response to treatment. Must control coccidiosis
Rabies	Cattle exposed to wildlife, one or more affected, all ages, incubation 3 weeks to few months	Quiet and dull (dumb form) or excitable and easily annoyed (furious form). Bellowing, yawning, drooling, saliva, eyesight normal, tenesmus, ascending paralysis beginning with anesthesia over tail head, progressive course, dies in 4–6 days, usually no gross muscular tremors or convulsions, mild fever early	Hemogram normal. Brain for laboratory diagnosis	Nil
Bovine spongiform encephalopathy (BSE)	Mostly in dairy cattle; Epizootic began in Britain in 1986; long incubation period; caused by scrapie-like agent in protein concentrate made from sheep carcasses following change in processing procedures	Insidious onset, clinical course several weeks, change in behavior, hyperesthesia, ataxia, loss of body weight, stare, agnostic behavior, kick during milking, knuckling, falling, progressive weakness leading to recumbency	Nil	Nil
Pseudorabies	Disease of pigs transmitted to cattle by bites.	Intense, local pruritus at site of bite, excitement, bellowing, convulsions, paralysis, death 2–3 days	Tissues for injection into rabbit. Histopathology of brain	Nil
Hypomagnesemic tetany (lactation tetany)	Lactating dairy cows on lush pasture, late pregnant beef cows, cold, windy weather in spring. May be precipitated by long transportation or deprivation of feed and water. Outbreaks occur. Seen in yearlings too. Case mortality can be high	Acute: sudden onset of irritability, hyperesthesia; convulsions, recumbency, loud heart sounds, tachycardia, polypnea. Subacute: gradual onset (2–4 days), hyperirritable, difficult to handle, stilted gait, falling, stumbling, sudden movement may precipitate convulsion	Serum magnesium level slow	Responds to magnesium sulfate early

Table 32.3 (Cont'd) Differential diagnosis of diseases of cattle with clinical findings referable to brain dysfunction

Disease	Epidemiology	Clinical findings	Clinical pathology and pathology	Response to treatment
Nervous acetonemia	2–6 weeks postpartum. High-producing cow. Single animal	Sudden onset, bizarre mental behavior, chewing, licking, bellowing, hyperesthesia, sweating	Ketonuria, hypoglycemia	Responds to glucose parenterally and/or propylene glycol orally
Bovine bonkers (Bovine hysteria)	Mature cattle and calves consuming ammoniated feeds (lucerne hay, bromegrass hay, fescue hay, wheat hay, maize stalks or silage). May also occur when animals have access to molasses-urea-protein blocks. Toxic agent may be substituted imidazole formed by combination of soluble carbohydrates and ammonia. Usually occurs when high quality forage treated with ammonia concentrate of more than 3% dry matter by weight. Can occur in nursing cows fed ammoniated feedstuffs	Periodic episodes of hyperexcitability, bellowing, running, charging, circling, convulsions, weaving, episodes last 30 seconds and may recur every 5–10 minutes. Some die. Most recover following removal of feed	Information not available	Recover spontaneously following removal of feed source
Hepatic encephalopathy (i.e. ragwort poisoning)	Cattle with access to plants containing pyrrolizidine alkaloids Many cattle may be affected	Loss of body weight, gradual onset of aggressive behavior, ataxia, muscular tremors, recumbency, convulsions, tenesmus and bellowing	Hyperbilirubinemia, decreased excretion of BSP. Liver lesions	No treatment
Brain abscess	Sporadic, young cattle (6 months to 2 years of age) may have history of previous infections	Localizing signs, rotation or deviation of head and neck, loss of equilibrium, circling, mild fever, may be blind in one eye, nystagmus one eye	Neutrophilia, neutrophils in CSF	Unfavorable response to therapy
Enterotoxemia due to *Clostridium perfringens* type D	Calves 2–4 months of age sucking high producing cows grazing on lush pastures. Outbreaks occur. Uncommon	Peracute: found dead. Acute: bellowing, mania, convulsions, blindness, death in 1–2 hours. Subacute: dull, depressed, blind	Hyperglycemia (150–200 mg/dL), glycosuria marked. Smear intestinal contents. Recover toxin (mouse protection tests)	Hyperimmune serum. Most die. Vaccination effective
Whole milk hypomagnesemic tetany of calves	Calves 2–4 months of age on whole milk. Also in calves on milk replacers, concentrates and hay and occasionally in nursing calves on pasture	Sudden alertness, hyperesthesia, head-shaking, opisthotonos, muscular tremors, frothing at mouth, convulsions, heart rate 200–250/min	Serum magnesium levels usually below 0.8 mg/dL	Magnesium sulfate intravenously gives good response, must follow up daily because of previous depletion of bone reserves

Arsenic poisoning

Arsenic compounds likely to be encountered by large animals are as follows:
Inorganic compounds used as insecticidal dips or as herbicides

- Oxide, e.g. arsenic trioxide
- Trivalent, e.g. sodium arsenite
- Pentavalent, e.g. sodium arsenate.

Organic compounds

A. Aliphatic organic arsenicals:

- Pharmaceuticals, e.g. cacodylic and phenylarsonic acids
- Weedicides, e.g. monosodium,
- disodium methanearsonates (MSMA & DSMA) .

B. Aromatic organic arsenicals, used as pharmaceuticals:

- Trivalent phenylorganic arsenicals, e.g. thiacetarsamide and arsphencomplexamine

- Pentavalent phenylorganic arsenicals, e.g. arsanilic acid, roxarsone (4-hydroxy-3-nitrophenylarsonic acid), nitarsone (4 nitrophenylarsonic acid).

Relative toxicities

A. Inorganic and aliphatic organic compounds.
The organic pharmaceuticals are the least toxic, while the insoluble oxides of medium toxicity and the trivalent inorganic compounds are associated with the most severe syndrome. Toxic oral doses may range from 1 to 25 mg/kg for the arsenite, 30-100 mg/kg for the arsenate, cacodylic acid 25 mg/kg daily for 8-10 days, and 10-25 mg/kg for 5-6 days for the methanearsonates.
B. Aromatic organic arsenicals are toxic when the recommended cumulative dose is exceeded by 2-4 times the recommended dose, delivered by either exceeding the recommended percentage in the feed or feeding it for too long. Seven to 10 days feeding of Arsanilic acid at 500 mg/kg diet or 3-nitro A, hydroxyphenylarsonic acid at 250 mg/kg diet will be associated with toxicosis in swine; approximately twice these concentrations will result in poisoning of poultry
Epidemiology
Outbreaks due to accidental access to source, or due to use of excessive amounts as a dose rate or over time. Most cases result from ingestion but percutaneous absorption also possible.
Clinical signs
Ruminant gastroenteritis syndromes
Acute form: Enteric form a highly fatal gastroenteritis with diarrhea, dehydration Nervous form with incoordination and blindness, or a syndrome of incoordination, restlessness, squealing, convulsions.
Clinical pathology
High levels of arsenic in feces, urine, milk for 5 days (organic arsenicals), 1 0 days (inorganic arsenic). Chronic cases best assayed in hair or skin.
Necropsy lesions Gastroenteritis in enteric form, no lesions in nervous form.

Diagnostic confirmation

Higher than normal levels of arsenic in body fluids or tissues.
Treatment
Primary: 2, 3-dimercaptopropanol (BAL) or sodium thiosulfate. Supportive: fluids, electrolytes for dehydration.

Mercury poisoning

Etiology Rarely inorganic mercury, commonly organic preparations.

Epidemiology

Inorganic salts in preparations used as rubifacients. Mercury poisoning in farm animals occurred in the past almost exclusively as a result of accidental feeding of grain, pellets, or concentrate mixtures treated with organic mercurial antifungal agents (Organic preparations used in seed grain fed accidentally to livestock). Meat from animals poisoned by mercury is unsuitable for human

consumption. Milk is probably safe as little mercury is excreted in it. The toxicity of mercury compounds depends on their solubility and the susceptibility of the animals.

Clinical signs

Inorganic salts: Acute - vomiting, diarrhea, abdominal pain; Chronic - weight loss, depression, alopecia, scabby dermatitis, long course.
Organic preparations: blindness, incoordination, paralysis.

Clinical pathology

High levels of mercury in all tissues and fluids; high blood urea nitrogen, urine alkaline phosphatase in cases of nephrosis.

Necropsy lesions

• Inorganic salts: Acute - gastroenteritis; Chronic - nephrosis.
• Organic preparations: neuronal necrosis in brain and spinal nerves.
• Poisoning by mercury is associated with inflammation of the alimentary mucosa and damage to the kidneys. It is manifested clinically by gastroenteritis and terminally by signs of uremia.
• Diagnostic confirmation. High blood, urine, tissue levels of mercury.

Treatment

Primary: Sodium thiosulfate orally and parenterally; BAL by injection.
Supportive: Astringents orally, fluids parenterally. Control Care in the handling of agricultural and pharmaceutical mercurials.

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