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Section 13

TOXICOLOGY

Edited by F. W. Oehme and T. Tobin

in Robinson, N.E. (ed), Current Therapy in Equine medicine, Saunders, Philadelphia, 1983.

TOXICOSES COMMONLY OBSERVED IN HORSES

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A knowledge of the common poisons in the practice area, the frequency of various toxicities, and the incidence of these poisonings during certain seasons of the year or during various activities helps the practitioner to narrow the potential range of poisonings from which a diagnosis may be made. In suspected poisoning, it is better to concentrate on common toxicities than to devote diagnostic and therapeutic efforts on a wide variety of potential toxins.

While each practitioner must learn the type and frequency of the chemical exposures possible in the practice area, certain groups of chemicals or toxin-containing materials frequently cause poisoning in horses. Tables 1 through 5 summarize the signs and therapy of the common toxicoses causing gastrointestinal, nervous system, and hematologic signs and those causing skin problems.

Emergency Treatment

Urgency is of utmost importance in treating toxicoses. Three rules to follow are begin treatment promptly; retain samples of blood, urine, and feces for analysis; and keep the animal warm during therapy.

The practitioner must act to prevent further absorption of toxin. This can be simply accomplished by moving the horse to a different pasture or stall and supplying fresh food and water, i.e., preventing access to the toxin. In cases of skin exposure, a thorough washing with a mild detergent and plenty of water is necessary. In cases of ingested poisons a gastric lavage in the unconscious or anesthetized horse or a laxative of mineral oil (3 liters PO) should be used to empty the digestive tract.

Treatment should be followed by oral adminis-

TABLE 1. PRIMARY CLINICAL SIGNS ARE GASTROINTESTINAL

Toxin	Signs	Treatment
Acids	Corrosion of mucous membrane of upper GI tract; colic and purgation followed by acute shock	Milk of magnesia, 20–30 ml PO Flush externally with water; apply paste of sodium bicarbonate
Alkalis	As for acids	4–6 egg whites to 1 l. tepid water followed by a cathartic Flush externally with water
Arsenic	Acute: abdominal pain, staggering gait, extreme weakness, trembling, salivation, diarrhea, fast, feeble pulse Subchronic: depression, anorexia, watery diarrhea, increased urination followed by anuria, dehydration, ataxia, trembling, stupor, cold extremities	Tannic acid, strong tea, or protein (egg white) to absorb; d-penicillamine, 11 mg/kg qid for 7–10 days PO; or sodium thiosulfate, 8–10 gm of 20% solution IV, and 20–30 gm plus 300 ml water PO; or dimercaprol (BAL), 3 mg/kg IM Repeat every 4 hours for 2 days, then qid on day 3, then bid until day 10 Supportive fluid and electrolyte therapy
Carbon tetrachloride	Loss of appetite, dullness, staggering gait, gastroenteritis, bloody feces, constipation followed by diarrhea, collapse, and death	Empty stomach, give high protein and carbohydrate diet; maintain fluid and electrolyte balance Do not give epinephrine
Petroleum distillates	Immediate bloat, shivering, and incoordination Anorexia	Mineral oil, 3 l. PO; after ½ hr, 20% sodium sulfate, 250–1000 gm PO
Phenols and creosols	Gastroenteritis, painful abdomen, weakness and depression, sternal recumbency	Wash skin, apply sodium bicarbonate (0.5%) dressing Mineral oil, 3 l. PO Activated charcoal, 250–500 gm PO Symptomatic treatment, stimulants, blood transfusions, and fluid therapy
Plants: Oak (tannins)	Constipation, abdominal pain, hematuria, weakness	Symptomatic treatment
Ragwort (alkaloid)	Acute: dullness, weakness, abdominal pain, nervous excitement Chronic: prolonged poor condition, icterus, yawning, drowsiness, staggering gait	

TABLE 2. PRIMARY CLINICAL SIGNS ARE CENTRAL NERVOUS SYSTEM STIMULATION

Toxin	Signs	Treatment
Alkaloids	Nervousness, difficult breathing, loss of muscular control, excess salivation, convulsions	Potassium permanganate, 2–4 ml/kg (1:10,000 solution) gastric lavage or PO Physostigmine salicylate, 30–120 mg SC or IM
Insecticides: Carbamates	Profuse salivation, diarrhea, muscle fasciculation, hyperactivity, followed by posterior paresis	Atropine sulfate, 0.5–1.0 mg/kg IV or to effect (dry mucous membranes), repeat dose as needed
Chlorinated hydrocarbons	CNS stimulation, violent excitation, muscle fasciculations, cranial to caudal convulsions	External: wash thoroughly with soap and water Barbiturates or chloral hydrate to control seizures Activated charcoal 250–500 gm PO with 20% sodium sulfate, 250–1000 gm PO
Organophosphates	As for carbamates	Atropine sulfate, 0.5–1.0 mg/kg IV or to effect (dry mucous membranes) followed by pralidoxime chloride (2-PAM, protopam chloride), 2% solution, 25–50 mg/kg by slow IV, repeat as needed, usually every 8–12 hrs
Lead	Blindness, muscle twitching, ataxia, head pressing, convulsions; often appears as GI involvement (diarrhea, salivation, anorexia)	Activated charcoal, 250–500 gm PO with 20% sodium sulfate, 250–1000 gm PO Calcium disodium EDTA, 28.5 mg/kg qid for 5 days Initial dose IV, then SC as 10 mg/ml in 5% dextrose
Plants: Larkspur (alkaloid)	Hypersensitivity, muscular trembling, collapse, prostration, convulsions Constipation, bloat, excessive salivation sometimes noted	Physostigmine (2.2 grains) plus pilocarpine (4.4 grains) plus strychnine (1.1 grains) in 20 ml water given SC per 500 kg, use with caution
Locoweed (selenium plus others)	Very excitable and irritable, abnormal gait, separate from herd, head held peculiarly, disturbed vision, chronic loss of weight, weakness, prostration, convulsions	Laxative, sedatives, quiet
Lupine (alkaloid)	Nervousness, loss of muscular control, frothing at the mouth, convulsions	Sedatives, laxatives, see alkaloids
Oleander (glycoside)	Overstimulation of the vagus, abdominal pain, diarrhea, tremors, progressive paralysis, coma	Atropine sulfate, gastric lavage Symptomatic treatment
Poison hemlock (alkaloid)	Incoordination, salivation, abdominal pain, weakness, shallow, irregular respiration, coma	Laxatives, tannic acid, stimulants Supportive treatment
Water hemlock (resinoid)	Violent spasms resulting in rapid respiration and heart rate, coma	Symptomatic treatment Artificial respiration
White snakeroot (tremetol)	Marked trembling, incoordination, weakness, inability to stand Partial throat paralysis	Laxatives, stimulants
Yellow star thistle (unknown)	Lip twitching, involuntary chewing, mouth open, inability to swallow or hold food in mouth, mechanical damage to lips	Symptomatic treatment

tion of an activated charcoal slurry (250 to 500 gm in 2 to 4 liters warm water). Administer a specific antidote, if known; otherwise treat the horse symptomatically. Assist the patient's respiration if necessary, keep the patient warm, and observe the initial signs carefully.

POISONOUS PLANTS

In horses other than those continually stabled and fed hay and commercial feed, the risk from injury due to poisonous or harmful weeds is a serious one. Pastures contain a variety of plants, often unrecognized, and plants growing along fences are often protected from mowing while still being accessible to the horse. Weather conditions may reduce the available pasture while weeds thrive. The clinical signs may be acute or subacute, but the effects are usually the result of animals consuming the plant material for several days and eventually showing the effects in one or more body systems.

Gastrointestinal problems, characterized by colic and diarrhea, may develop from plants such as castor bean (p. 604), oleander (p. 602), and bracken fern (p. 600). Damage to the liver, usually the result of continued plant ingestion for many weeks, produces an altered temperament, a dummy-like attitude, loss of weight, and hepatic cirrhosis. Fiddleneck (Amsinckia) (p. 596), groundsel (Senecio) (p.

TABLE 3. PRIMARY CLINICAL SIGNS ARE CENTRAL NERVOUS SYSTEM DEPRESSION

Toxin	Signs	Treatment
Mercury	Muscle incoordination, ataxia, hyperesthesia, tremor, convulsions, and coma Can appear as GI involvement (diarrhea, anorexia, emaciation)	Activated charcoal, 250–500 gm PO Dimercaprol (BAL), 3 mg/kg IM; repeat every 4 hrs for 2 days, then qid on 3rd day, then bid for 10 days until recovery Supportive fluid and electrolyte therapy
Plants:		
Black locust (glycoside)	Anorexia, depression, weakness, posterior paresis, irregular pulse, labored breathing	Digitalis Symptomatic treatment
Crotolaria (alkaloid)	Acute: anorexia, gastric irritation, tenesmus, bloody feces Chronic: emaciation and depression	Supportive therapy
Death camas (steroid alkaloid)	Stiff-leggedness, hypersensitivity, anxious expressions, dyspnea, weakness, posterior paresis, convulsions	Atropine sulfate (4.4 mg) plus picrotoxin (17.6 mg) in 5 ml water given IV per 100 kg; repeat every 2 hrs for 2–3 injections
Horsetail (thiaminase plus unknown)	Weakness, diarrhea, rapid weight loss, incoordination, coma	Thiamine hydrochloride 100–200 mg SC daily for several days
Milkweed (resinoid)	Incoordination, depression, shallow respiration, inability to stand, coma	Symptomatic treatment
Bracken fern (thiaminase)	Emaciation, incoordination, marked progress to paralysis and inability to rise	Thiamine hydrochloride 100–200 mg SC daily for several days

TABLE 4. PRIMARY CLINICAL SIGNS ARE BLOOD ALTERATIONS

Toxin	Signs	Treatment
Chlorates, nitrites	Staggering, purging, abdominal pain, hematuria, hemoglobinuria, dyspnea, cyanosis Blood is dark brown	4% methylene blue 10 mg/kg IV; repeat at intervals of several hours
Cyanide Arrowgrass, corn, elderberry, prunus sp, sorghum sp	Initial excitement and muscle tremors followed by pronounced polypnea and dyspnea, salivation, lacrimation, and voiding of feces and urine Gasping for breath and clonic convulsions Blood is bright cherry red	20% sodium nitrite (10 ml) plus 20% sodium thiosulfate (30 ml), 0.09 ml/kg IV
Phenothiazine	Hemolysis, anemia, hemoglobinuria, weakness, anorexia, fever, icterus, colic, constipation, and diarrhea	Methylamphetamine 0.1–0.2 mg/kg IV for phenothiazine tranquilizers Symptomatic treatment

TABLE 5. PRIMARY CLINICAL SIGNS ARE EPITHELIAL DAMAGE

Toxin	Signs	Treatment
Plants:		
Horsebrush	Photosensitization	Topical ointments; symptomatic treatment Keep out of sun; graze at night
St. Johnswort	Photosensitization	See horsebrush
Foxtail	Mechanical injury	Symptomatic treatment
Cheatgrass	Mechanical injury	Symptomatic treatment
Needlegrass	Mechanical injury	Symptomatic treatment
Poverty grass	Mechanical injury	Symptomatic treatment
Crimson clover	Mechanical injury	Symptomatic treatment

596), and crotolaria (p. 596) are common plants that induce liver damage. Many horses with liver damage will exhibit central nervous system effects, assumed to result from the buildup of ammonia. This excitability and altered personality may be confused with direct central nervous system effects induced by another series of poisonous plants. Clinical signs of hyperexcitability, incoordination, paresis or paralysis, abnormal body movements or posturing, convulsions, and coma may result from yellow star thistle (p. 595), locoweed (p. 599), lupine, nicotine (p. 580), and the selenium-containing plants (p. 593). By the time central nervous system effects are observed, most horses are no longer treatable.

Sudden death may be due to cyanide-containing plants such as sorghums (p. 598) fed by owners unaware of the danger. A number of pasture plants contain awns and thistles that induce mechanical injury to the lips, gums, and tongues of consuming horses. Others, such as vines and coarse plants, may provide a digestive tract obstruction.

INSECTICIDES AND RODENTICIDES

The organophosphate insecticides (p. 582) and chlorinated hydrocarbon insecticides (p. 581) are common poisons of horses. Chlordane, heptachlor, aldrin, dieldrin, isodrin, endrin, toxaphene, lindane, methoxychlor, and the variety of organophosphate compounds that are continually growing in number and in ingenuity of naming are highly toxic compounds that horses are exposed to topically or via ingestion.

The rodenticides that may produce poisoning in horses include strychnine (p. 584), ANTU (p. 585), compound 1080 (p. 585), warfarin (p. 584), arsenic (p. 585), barium (p. 587), thallium (p. 586), phosphorus (p. 586), and zinc phosphide (p. 586).

MEDICATIONS

Because of the variety of "health products" provided to horses by their proud and enthusiastic owners, toxicoses due to drugs and chemical products intended for maintaining and improving equine health are not uncommon. Vitamins, stimulants, analgesics, anthelmintics, and tranquilizers may all cause problems due to misuse through overapplication or erroneous routes of administration. When several of these drugs are used concurrently, chemical interactions may occur, resulting in adverse drug reactions. The inherent sensitivity of the horse to foreign chemicals contributes further to the relatively high incidence of drug reactions in equine medicine.

SNAKE AND INSECT BITES

The sensitivity of horse skin and tissue, coupled with the environments in which many horses are housed or pastured, leads to a high incidence of insect (p. 558) or snake bites (p. 587). Localized and occasionally generalized reactions are common; fatalities occur if individual sensitivity is great or if bites evoke swelling that interferes with vital functions. Fortunately the incidence of snake and insect bite is seasonal and usually is restricted to specific regions.

MISCELLANEOUS TOXICOSES

Fungi are everywhere, and under appropriate conditions of moisture, temperature, and carbohydrate availability, they may grow in horse feeds. The presence of spoiled feed should remind the clinician of the possibility of fungal growth and the presence of mycotoxins. Aflatoxins produce pronounced liver damage, while other mycotoxins may induce colic, hemorrhagic gastroentertis, kidney dysfunction, blood coagulation defects, and interference with immune status.

Gases may be generated under a variety of housing or environmental conditions. Carbon monoxide, hydrogen sulfide, nitrogen dioxide, ammonia, sulfur dioxide, carbon disulfide, and hydrogen cyanide are all toxic to horses in confined and poorly ventilated situations.

Supplemental Reading

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GENERAL PRINCIPLES IN TREATMENT OF POISONING

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The treatment of any poisoning is based upon a sound diagnosis. Except in emergency treatments, in which case general antidotal therapy is employed, every attempt should be made to utilize the available diagnostic information to formulate the most specific treatment for the poisoning.

DIAGNOSIS

The diagnosis of poisoning is based upon an adequate history, clinical evaluation of the patient, and a necropsy if death occurs and other animals are still involved. Since few poisonings have pathognomonic clinical syndromes or necropsy lesions, the history is often a key to diagnosis. Observation, an adept questioning procedure, and utilization of the practitioner's knowledge of management practices and personality quirks of the client assist greatly in generating diagnostic clues. The clinical signs in poisoned horses may involve a variety of body systems, with the central nervous system, digestive tract, liver, and blood frequently being affected. General signs of poisoning are lack of appetite, depression, weight loss, dehydration, colic, and frequent and difficult respiration. Hyperexcitability, incoordination, muscular twitching, abnormal posturing and body movements, and convulsions leading to prostration and coma may be suggestive of primary central nervous system effects or can be secondary to liver or digestive tract disturbance.

Diagnosis is especially difficult in chronic, low-grade poisonings that may involve biochemical or metabolic "interference syndromes" or may reflect the gradual accumulation of chemicals in various body systems and the eventual expression of their toxicity. The history and general physical appearance of the patient will often suggest a long-term process that may be at variance with the client's insistence that the horse "just got sick."

Whenever possible, a complete postmortem examination should be performed on animals dying from poisons. Although very few poisonings provide pathognomonic necropsy findings, there are many horses thought to have been poisoned that upon necropsy have a strangulation or torsion of the digestive tract, a discovery that warrants the effort involved in performing field necropsies. Although laboratory studies are frequently expensive, they are often the only definitive procedures to identify the cause of an intoxication. Unfortunately, the laboratory is not able to perform an all-encompassing screening test, and the clinician must suggest the most likely or suspected poison for laboratory assay. Suggestions for sample collection are given in the article on the etiologic diagnosis of sudden death (p. 611). In the living patient, blood and urine, as well as samples of suspected contaminated material, may be submitted for analysis.

The clinician should not rely upon the laboratory assay and should not wait for histopathologic or laboratory results before initiating therapy and suggesting management changes. "Tincture of time" is applicable only if the patient can spontaneously deal with the disease process. In cases of overwhelming intoxications or in instances of continuing ingestion and accumulation of a toxic compound, not waiting even a matter of hours before initiating treatment may mean the difference between recovery or death. There will be numerous instances in which an absolute diagnosis is not confirmed, but circumstantial, clinical, and perhaps gross pathologic evidence suggests a general group of poisons or a specific intoxication. Treatment should then be initiated promptly to prevent further absorption. Apply specific antidotes where possible, hasten elimination of the circulating toxin, and provide supportive therapy to the animal.

TREATMENT PRINCIPLES

It is useful to follow a general set of objectives in dealing with poisonings in horses. These general steps are stabilization of the patient (if necessary), prevention of further exposure to or absorption of the toxin, application of specific antidotes or therapy, increasing elimination of the absorbed poison, and supportive therapy to counteract the specific organ effects of the poisoning. To respond to these objectives requires not only prompt action but also the availability of appropriate and necessary equipment and medications. Table 1 is a listing of the suggested components for an emergency poisoning kit. These items are fundamental to the treatment of equine poisonings and should always be available and fully stocked for immediate use by the clinician.

STABILIZATION OF THE PATIENT

Since a patient dying of respiratory failure is frequently not helped even by prompt administration of a specific antidote, it is most important to ensure

TABLE 1. EQUINE EMERGENCY POISONING KIT

Parenteral Solutions	Oral Medications	Equipment	Miscellaneous Items
Atropine sulfate	5% Acetic acid (vinegar)	Aspirator bulb	Mild detergent
Barbiturates (phenobarbital, pentobarbital)	Activated charcoal	Blankets	Oxygen
Calcium disodium EDTA	Albumin (diluted egg white)	Endotracheal tubes, several sizes	Sodium bicarbonate paste
23% Calcium gluconate	0.15% Calcium hydroxide	Enema kit	
Digitalis	20% Magnesium sulfate solution	Gauze rolls and tape	
Dimercaprol (BAL)	Milk of magnesia	Intravenous catheters and stylets	
Lactated Ringer's	Mineral oil	Mechanical respirator or compression bag	
4% Methylene blue	d-Penicillamine	Needles (hypodermic)	
Normal saline	1:10,000 Potassium permanganate solution	Stethoscope	
Physostigmine	20% Sodium sulfate	Stomach tubes, several sizes	
Picrotoxin	Tannic acid	Syringes	
Pilocarpine	Vegetable oils, lard	Thermometers	
Pralidoxine chloride (2-PAM, Protopam chloride)		Urinary catheters, various sizes	
Sedatives		Venotomy kit	
Thiamine hydrochloride			
1% Sodium nitrite			
20% Sodium nitrite			
20% Sodium thiosulfate			
Stimulants			
Strychnine			
Vitamin K_1			

that the horse does not die while the clinician is deciding upon the appropriate course of action. An adequate and patent airway should be ensured, and cardiac and respiratory function must be stabilized and maintained. Endotracheal intubation and artificial respiration may be coupled with cardiac stimulation to maintain these vital functions. Blood pressure should be adequate to ensure kidney perfusion and glomerular filtration. If in doubt, catheterization of the bladder should be performed and urinary flow monitored. Mechanical means may be used to stimulate vital signs and to maintain them, with drugs employed as necessary. Once the clinician has assured himself or herself that vital signs are stable, management of the poisoning may then continue.

Prevention of Further Exposure and/or Absorption

In a situation in which horses are being exposed to the toxic material, they should either be removed from that environment, or the toxic substance should be taken away from the patients. This may involve removal of the animals from a pasture or shed or may necessitate the cleaning of hay, grain, or water sources so that further consumption is halted. If the toxin has been applied to the skin, the animal should be washed with water and a mild detergent to remove the unabsorbed chemical. Abundant water should be used to wash the skin and to dilute any remaining toxin. Protective clothing should be worn by the veterinarian or animal handler during this process.

Absorption of toxins in the digestive tract may be limited by the use of adsorbents, such as activated charcoal, preferably of vegetable origin, used at a minimum of ½ lb (250 gm) for a foal, with up to 1½ lb (750 gm) used for an adult horse. Up to 1 gal (4 l) of warm water (depending upon the animal's size) should be used to make a slurry of the activated charcoal, which is then administered by stomach tube. The activated charcoal adsorbs many organic toxins but is relatively ineffective against inorganic and heavy metal poisons. The slurry should be left in the stomach for 20 to 30 minutes and then should be followed with a laxative to hasten removal of the charcoal and adsorbed chemical from the patient. Unless evacuated from the digestive tract, the poison may dissociate from the adsorbent and eventually may be absorbed by the patient. Although activated charcoal is probably the most effective adsorbent, other compounds such as bentonite, fuller's earth, and tannic acid may also be utilized to adsorb various toxic agents.

If no adsorbent is available, laxatives should be utilized to remove the toxic material from the digestive tract as soon as possible. Mineral oil (1 to 1½ gal, 4 to 6 l), 500 gm of magnesium sulfate or 1 mg of lentin (carbachol) may be administered to a mature horse. The sulfate laxatives (sodium or magnesium) are probably the most effective agents for evacuation of the digestive tract. If mineral oil is used initially, the use of a saline cathartic 30 to 45

minutes after oil administration will be an effective purgative. If the patient already has diarrhea due to the toxic syndrome, further administration of a purgative may add to the risk of dehydration.

Specific Antidotes

If the poisoning is identified early and an antidote available, it should be used early in the treatment regimen, immediately following stabilization of the patient and prevention of further exposure and absorption. There are, however, very few poisons with specific antidotes, and it is frequently not possible to identify the toxic syndrome until later in the management of the patient. The specific antidotes given in Table 1 may be applied for such poisonings as insecticides, arsenic, cyanide, nitrite, and others. In some cases, doses are critical, but in most the animal is being titrated with the antidote against the body burden of the toxin. For example, in insecticide poisonings, atropine is given to effect by intravenous administration. As the clinical signs abate, the rate of atropine administration is diminished. In the absence of specific antidotes, application of sound therapeutic principles and common sense in further managing the poisoned patient is critical.

Increased Elimination of the Absorbed Poison

General nonspecific detoxicants may be used in the absence of specific antidotes. Intravenous administration of 100 to 500 ml of 20 per cent calcium gluconate, 500 to 1000 ml of 10 to 50 per cent dextrose, or 150 to 500 ml of 25 per cent sodium thiosulfate solution is useful.

Since absorbed toxins are usually excreted by the kidneys, renal excretion may be enhanced by the use of large volumes of intravenous fluids (electrolytes, 5 per cent dextrose, or saline) or by the use of diuretics, which should be carefully managed to avoid dehydration. Adequate renal function and hydration of the patient are vital concerns. If a urinary flow of 0.1 ml per kg body weight per minute is not maintained by the patient, hydration of the affected horse should be improved.

Supportive Therapy

The final objective is to maintain the various body functions in a state compatible with detoxification of the poison and patient recovery. Central nervous system excitement may be managed by the use of sedatives, barbiturates, or combinations of tranquilizers, chloral hydrate, and magnesium sulfate. Convulsions are most effectively handled by pentobarbital administration, but care must be taken to ensure that respiration is not depressed. Although inhalation anesthetics are excellent for long-term management of central nervous system hyperactivity, prolonged anesthesia in horses is not without risk of gas exchange problems and muscle damage. Central nervous system depression is often complicated by respiratory depression, and both conditions must be managed. Artificial ventilation may support respiration while stimulants such as doxapram (5 to 10 mg per kg) pentylenetetrazol (6 to 10 mg per kg), or bemegride (10 to 20 mg per kg) may be administered intravenously to stimulate central nervous system activity. The action of the stimulants is of relatively short duration; hence, the clinician may wish to place more emphasis on artificial ventilation (p. 475), since adequate respiratory support frequently stimulates recovery from central nervous system depression.

Effective respiratory support requires an adequate patent airway, which may be obtained by an endotracheal tube or by performing a tracheostomy. A mechanical respirator is of great value, but manual compression of the bag of an anesthetic machine may also be utilized with equal efficiency. In the event of cyanosis, oxygen may be necessary, but under most conditions, environmental air is adequate. A mixture of 50 per cent oxygen and 50 per cent environmental air may also be employed.

Support of cardiovascular function requires adequate heart function, appropriate circulating blood volume, and appropriate acid-base balance. Fluid volume and cardiac activity are of most immediate concern. Heart rate may be aided by the use of closed-chest cardiac massage and by the administration of therapeutic agents intravenously or directly into the heart. The slow administration of calcium gluconate has been useful in some instances. Digoxin, 0.2 to 0.6 mg per kg intravenously, may also be effective. Clinical judgment is important to determine the type and extent of cardiac stimulation to be pursued.

For decreased circulating volume, whole blood administration is a valuable procedure. Hypovolemia due to water loss alone may be treated by administering lactated Ringer's solution, saline, or 5 per cent dextrose solutions. Administration of 2 to 10 mg dexamethasone per kg body weight intravenously is useful to prevent shock.

Acidosis is corrected by the administration of sodium bicarbonate, sodium lactate, or lactated Ringer's solution. Alkalosis is less commonly seen but may be reversed by the intravenous administration of physiologic saline (10 mg per kg) followed by 200 mg ammonium chloride per kg per day orally. Such therapy requires careful monitoring to ensure

the administration of appropriate concentrations and volumes.

Animals with severe diarrhea may require very careful monitoring of water and electrolyte balance. Fluid requirements may be given via stomach tube or intravenously. Symptomatic care of gastrointestinal disturbances includes protectants such as Kaopectate* or bentonite.

Body temperature should be maintained within normal limits by protection from environmental cold or heat, by providing heat lamps to prevent hypothermia, or cold water enemas, cold water baths, or ice bags to reduce hyperthermia. Constant monitoring of the animal's body temperature is necessary to ensure that vital biochemical and physiologic detoxification processes are able to proceed at optimal physiologic temperatures. Since horses are extremely sensitive to pain, control of pain is important.

Although it is optimal to have all the suggested procedures operational in each poisoned individual, practicality dictates that the clinician select those measures most appropriate to the case being managed. Careful attention to the application of these objectives in the poisoned horse will ensure maximal therapeutic effectiveness.

*Kaopectate, Upjohn Co., Kalamazoo, MI 49001

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INSECTICIDES

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Of all the chemicals to which horses might be exposed, insecticides constitute the largest and most potentially toxic group of compounds. They are chemicals that may either be intentionally applied for insect or parasite control in the animal, or they may be accidentally consumed via contamination of feed, forage, water, or the stable enviornment. These potentially hazardous situations make it imperative that the equine veterinarian be well informed of the dangers and safety of the various types of insecticides, and be prepared to diagnose and manage any instances of clinical intoxication.

There are three general groups of insecticide material to which horses may be routinely exposed: The plant-origin insecticides, the chlorinated hydrocarbon insecticides, and the organophosphorus and carbamate materials.

PLANT-ORIGIN INSECTICIDES

This group includes insect control agents derived from plant materials and some that are now synthesized rather than extracted from plants. Rotenone and pyrethrins are materials applied topically directly to the horse and are essentially nontoxic. Clinical cases of rotenone or pyrethrin poisoning are extremely rare and are always due to massive ingestion of these insecticides rather than topical application. The materials are not absorbed from the skin and may be clinically considered of no hazard.

NICOTINE

Nicotine is an extremely toxic chemical, but fortunately, it is used only for mite control in buildings. Nicotine sulfate is never directly applied to horses. Toxicity is, therefore, limited to accidental contamination of feeding materials or water or of horses being housed in recently sprayed stables.

CLINICAL SIGNS

If toxicity does result from nicotine sulfate contact, the signs of poisoning occur within a few minutes. They are characteristically those of central nervous system stimulation, producing marked excitement, rapid respiration and salivation. If the animal consumed the nicotine, irritation of the oral mucosa is seen, with increased peristalsis and diarrhea occurring with the ingestion of low to moderate doses. The initial stimulation period is followed by depression, with the horse becoming incoordinated and ataxic and having a rapid pulse with shallow and slow respiration. This leads rapidly to a flaccid pa-

ralysis, with coma and death occurring within a few hours. Death usually occurs during a terminal convulsive seizure from paralysis of the respiratory muscles. Recovery from sublethal doses is usually complete within four to six hours after exposure. No characteristic postmortem lesions are found with nicotine sulfate other than cyanosis and congestion of internal organs. With oral ingestion, congestion of the digestive tract mucous membranes, particularly the upper portion of the small intestine, may be seen.

THERAPY

Treatment of nicotine sulfate poisoning is usually not feasible because death occurs rapidly. However, spontaneous recovery may occur if only small amounts are ingested. Topical nicotine should be washed from the skin. The administration of laxatives, tannic acid, or potassium permanganate may help to eliminate ingested nicotine. General supportive care is aimed at prolonging life to allow biological detoxification.

CHLORINATED HYDROCARBON INSECTICIDES

Chlorinated hydrocarbon insecticides are slowly being removed from routine use as agricultural chemicals, but their application is still permitted on some non–food-producing animals, including horses. Although their environmental use has been reduced because of their biologic persistence, as a group these insecticides are effective agents. However, their slow metabolism and persistence in animal tissues causes biologic accumulation following repeated exposures. Horses may thus develop toxicity either from application of excessive concentrations or because of frequent, repeated applications of single recommended amounts. Because of their lipid solubility, all members of this class of insecticides are easily absorbed through the intact skin after topical application or close confinement of animals in recently sprayed housing areas.

CLINICAL SIGNS

The clinical signs of chlorinated hydrocarbon insecticide poisoning are intermittent, with colic and severe neurologic effects predominating. Hyperexcitability, hyperesthesia, and tonic-clonic convulsive seizures may alternate with periods of depression. The toxicity usually begins within an hour of application or exposure, with the animal initially being apprehensive. A period of hyperexcitability follows, characterized by exaggerated responses to stimuli and spontaneous muscle twitches and spasms. The muscle tremors usually originate in the head area and progress posteriorly to involve the neck, shoulder, back, and rear leg muscles. Early in the syndrome, the horse may develop these spasms while standing, but as they become more severe, the animal will collapse into lateral recumbency. The horse may have chewing movements, may twist or elevate its head, and may undergo abnormal posturing prior to the development of convulsions. Body temperature may be elevated during seizures. Intermittent respiratory paralysis occurs during the convulsions. The convulsions may last several hours, and the patient either dies during a severe seizure or undergoes gradual recovery, with the severity of each subsequent convulsion decreasing until body control and posture are once more regained. Recovered horses may have minor neurologic problems for a few days following recovery, with depression and partial loss of appetite remaining for three to five days. Most fatally affected horses will die within 12 hours after the onset of seizures. No characteristic postmortem lesions are observed in fatal cases other than those resulting from terminal convulsions and trauma due to the seizures.

THERAPY

Although there is no specific treatment for chlorinated hydrocarbon poisoning, conscientious attempts should be made to remove all unabsorbed insecticide from the patient's body. Washing of the skin with soap and warm water is very important. In instances of oral ingestion of chlorinated hydrocarbon insecticide, large amounts of activated charcoal may given by stomach tube to bind the unabsorbed material. Oily laxatives should be avoided, but magnesium sulfate and similar cathartics can be given following the activated charcoal to empty the digestive tract of the contained insecticide. The neurologic effects may be diminished by the use of sedatives or anesthetics. Barbiturates seem to provide most effective control of the centrally originating seizures, but chloral hydrate and tranquilizers may also be used. Repeated dosing is required to control the seizures, and animals that recover need smaller doses as the severity of the convulsions diminishes. Intravenous fluids may be given in severe or prolonged cases to maintain hydration. Generally supportive care will hasten recovery.

Since a variety of chlorinated hydrocarbon insecticides are available, it may be important to determine the specific chemical involved in any poisoning and to establish its source so that future cases can be prevented. The most common chlorinated hydrocarbons used on horses are toxaphene and lindane. Benzene hexachloride, aldrin, endrin, dieldrin, methoxychlor, heptachlor, and chlordane are other chlorinated hydrocarbon insecticides that may

be applied on and around horses. While each has its specific toxicity, individual horses may show hypersensitivity to the dose considered safe for the "average" horse. Young colts, weak and debilitated animals, and old horses with potential liver or kidney disease are especially at risk from exposure to the chlorinated hydrocarbon group of insecticides.

ORGANOPHOSPHORUS AND CARBAMATE INSECTICIDES

Unlike chlorinated hydrocarbons, the organophosphorus and carbamate insecticides have little environmental and biologic persistence and are, therefore, increasing in use. Their insecticidal properties depend upon an acute and overwhelming toxicity. Unfortunately, this same event often occurs in horses exposed to this group of chemicals.

As with the chlorinated hydrocarbon compounds, horses may be exposed by overzealous skin application, by spraying of the insecticide in confined areas containing horses, or by accidental contamination of forage, feed, or water. In addition, organophosphate compounds are also used for control of digestive tract parasites in horses, and toxicities occasionally result from this method of application. Horses with digestive tract lesions, animals with intestinal conditions that increase absorption (such as constipation or mucosal inflammation or irritation), or certain hypersensitive individuals are particularly likely to show clinical effects from the oral application of these compounds. The organophosphorus compounds include trichlorfon, dermeton, malathion, dichlorvos (DDVP), ronnel, Rulene, parathion, and diazinon. The carbamate group of insecticides is represented by carbaryl.

CLINICAL SIGNS

Both the organophosphorus and carbamate insecticides have their effect and cause their clinical signs by binding acetylcholinesterase, thereby permitting continuous cholinergic stimulation and excessive autonomic and muscular activity. Effects manifested within the first hour after exposure include frequent urination, increased peristalsis reflected as colic, and "patchy" sweating, particularly of the skin of the neck, shoulders, and rib cage of the affected horse. Salivation may be moderate to profuse, and the parasympathetic stimulation produces defecation, urination, and a general sense of anxiety or uneasiness in the patient. The heart rate is slow, respiratory efforts become exaggerated, and the animal may develop severe abdominal pains. A stiff-legged gait and muscle tremors of the face, neck, and other body muscles occur as the syndrome progresses. The muscular hyperactivity from organophosphorus and carbamate insecticides never develops into convulsions, as is so typical of the chlorinated hydrocarbon insecticides. Rather, the hyperactivity of the skeletal muscles is generally followed by muscle weakness, incoordination and ataxia, and prostration.

Respiratory failure is a sign of severe toxicity. Bronchoconstriction and pulmonary edema complicate respiratory efforts, and weakness of the respiratory muscles leads to difficult, frequent, and shallow respiratory efforts. Death is due to anoxia. Death may occur within minutes to several hours after the initial signs develop. Except in cases of oral absorption, when continuing absorption prolongs clinical signs, horses that do not die within 12 hours after exposure have a good chance of spontaneous recovery.

Some of the newer organophosphorus and carbamate insecticides are capable of producing variations in this clinical syndrome. All the described clinical signs may not be seen in any one horse, but several of the signs are usually present. In all instances, however, terminal muscle weakness and respiratory dysfunction are severe, and death is due to interference with and paralysis of respiratory efforts. If the diagnosis is in doubt, low blood cholinesterase activity will confirm the toxicity.

Interactions of organophosphorus and carbamate insecticides with other chemicals affecting the same enzyme systems are possible. Drugs working by this mechanism will have additive and sometimes synergistic clinical effects. Phenothiazine derivatives, such as the promazine tranquilizers, potentiate the effects of these cholinesterase-inhibiting insecticides. The administration of succinylcholine, carbachol, physostigmine, or neostigmine is contraindicated if horses have recently been exposed to organophosphorus or carbamate insecticides. The acetylcholinesterase inhibition persists for at least 14 days following organophosphorus insecticide exposure, and at least 30 days or more are required before normal circulating levels of acetylcholinesterase return in organophosphorus-exposed horses. The effects of carbamate insecticides are much shorter, but interaction with other anticholinesterase compounds is still possible if exposures occur with three to five days of each other.

Postmortem lesions associated with organophosphorus or carbamate poisoning are nonspecific. Excessive pulmonary fluids and evidence of excessive fluids in the mouth and digestive tract are supportive but not confirmatory for organophosphorus or carbamate poisoning. In some horses, the excessive peristaltic activity will result in pooling of the blood in "bands" in the small intestinal tract mucosa, and 1 to 7 cm wide areas of the mucosa of the small intestine will appear hyperemic. The bladder may be empty owing to excessive urination, and liquid

feces may be found in the rectum. Final confirmation of death due to organophosphorus or carbamate insecticides depends upon the detection of significant plasma, brain, liver, or kidney concentrations of the suspected chemical. Excessively depressed plasma and red blood cell cholinesterase activity may also support a diagnosis.

THERAPY

Fortunately for horses affected with organophosphorus or carbamate toxicity, an effective and specific treatment regimen is available. It involves providing respiratory assistance if death due to respiratory dysfunction is imminent, chemically antagonizing the signs produced by the excessive acetylcholine present at synapses, and aiding the dissociation of inhibited (complexed) acetylcholinesterase throughout the body.

All horses should be immediately treated intravenously with atropine sulfate. The approximate dosage of 1.0 mg per kg must be given to effect, with mydriasis and an absence of salivation used as end points. Since the atropine administered is being titrated against the absorbed organophosphorus or carbamate, the actual amount of atropine required in any case will be variable. After the initial atropine administration, repeated doses may be given every 1½ to 2 hours as required. Additional doses of atropine may be administered subcutaneously. Intravenous atropine administration is also a useful diagnostic tool. Horses not showing decreased clinical effects (decreased anxiety and less evidence of dyspnea and colic) when atropine is administered are probably not suffering from organophosphorus or carbamate insecticide poisoning.

Although atropine dramatically counteracts the parasympathetic signs within a few minutes after administration, it will only minimally reduce the skeletal muscle and nervous system effects. It also will not counteract the insecticide-acetylcholinesterase binding, which is relatively resistant to spontaneous hydrolysis.

Oximes are utilized to increase release of the inhibited enzyme. These compounds (2-PAM, pralidoxime chloride, TMB-4, and the commercially available Protopam chloride) are effective in binding the organophosphorus compound and freeing it from the enzyme-phosphorus complex. This releases the previously inhibited acetylcholinesterase to return to its normal physiologic functions. At least 20 mg Protopam chloride per kg are required, but occasionally as much as 30 to 35 mg per kg may be necessary to secure lasting results. The compound is given intravenously and is repeated every four to six hours. It is important that early treatment with the oximes be instituted. After 18 to 20 hours of organophosphorus exposure, a stabilized enzyme-insecticide complex has occurred. This complex is refractory to oxime therapy, which may need to be maintained for several days to be effective. The most effective results are observed with a combination of atropine and oxime treatment. In this way, the immediate clinical signs are treated, and the enzyme-organophosphorus complex is broken and directly antagonized.

Since oximes may have some deleterious effects in certain cases of carbamate poisoning, the routine use of oximes in cases not specifically known to be organophosphorus-produced is not recommended. In those instances, immediate treatment with atropine will provide life-saving effects. Since carbamate poisoning is short-lived owing to spontaneous dissociation of the complex and rapid biotransformation of the carbamate insecticide, repeated treatments with atropine are usually not necessary. Carbamate toxicity usually produces death within two hours, or spontaneous recovery occurs shortly thereafter. Provided that additional absorption of the carbamate insecticide does not occur through skin or the digestive tract, one or two treatments with atropine should be sufficient to manage carbamate toxicity. Rapid recovery should then follow.

To prevent additional absorption of the organophosphorus or carbamate insecticide, soap and water should be used to wash dermally exposed animals, and 1 to 2 lbs (0.5 to 1 kg) of activated charcoal should be administered orally in a water slurry to decontaminate the digestive tract of orally exposed horses. Osmotic laxatives should also be employed to empty the digestive tract.

The described specific therapy is usually quite adequate for routine cases of organophosphorus or carbamate toxicity. However, in severe instances or in episodes that are prolonged owing to delay in treatment or continuing exposure, clinical judgment must be used in the application of supportive therapy. Animals in life-threatening situations with severe respiratory embarrassment should be supported with artificial respiration where appropriate. Electrolyte and fluid therapy may be indicated in severely poisoned animals affected for several days. Whole blood and amino acid infusions may also be useful in ensuring the most effective management of these cases of insecticide toxicity.

Supplemental Readings

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RODENTICIDES

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Rodenticide toxicity in horses is relatively uncommon because most owners ensure that rodenticides are not placed in their horse's proximity. Should horses be exposed to rodenticides, the method of packaging and placement of bait usually result in the animal receiving less than a toxic dose. Since many of the commonly used anticoagulant rodenticides require several days of exposure, the potential for toxicity is further reduced.

In instances of feed contamination with highly toxic rodenticides, not only may a sufficient dosage be received, but also continual daily ingestion may result in accumulation of the toxic chemical. If the feed is being given to several horses, more than one animal may become ill, and a classic "outbreak" of poisoning may be seen. Environmental or feed contamination has produced poisoning from such rodenticides as warfarin and other anticoagulants, strychnine, ANTU, arsenic, fluoroacetate, zinc phosphide, and Vacor. In addition, potential toxic effects from phosphorus, thallium, barium chloride, and Castrix must also be considered whenever rodenticide toxicity in horses is discussed.

WARFARIN, PIVAL, AND OTHER ANTICOAGULANTS

Single doses of 75 to 100 mg per kg are toxic, but a dose of 2 mg or less per kg on a repeated daily basis is more likely and effective in producing poisoning. The anticoagulants antagonize vitamin K and produce coagulation system defects. Vascular shock may be seen with single large doses, but more commonly, mild to massive hemorrhages occur throughout the body. Nose bleeds, diarrhea with free blood in the stool, and subcutaneous hematomas (particularly over bony prominences and points of contact with hard surfaces) are typical signs. Lameness may occur due to hemorrhage into joint capsules, and soreness may be seen due to muscle hematomas. Occasionally, affected animals are first recognized by continual bleeding following owner or veterinary parenteral medication, and anemia may be observed upon examination of the mucous membranes or the performance of blood counts. The diagnosis is usually obvious upon the finding of elevated prothrombin times and clinical evidence of anemia and diffuse hemorrhagic foci. Occasionally in horses, acute death is seen due to massive hemorrhage into the thorax, abdominal cavity, or around the brain; these are obvious upon postmortem examination.

THERAPY

Treatment involves removal from the source of the anticoagulant, replacement of the inhibited coagulation factors, and supplying vitamin K to competitively antagonize the presence of the anticoagulant rodenticide. The horse should be removed from its immediate environment to prevent further exposure, or the source of the bait should be detected and removed. If ingestion of the anticoagulant has occurred within 24 hours, saline cathartics and activated charcoal may be administered. Whole blood (p. 325) will reverse anemia, will immediately replace missing coagulation factors, and will stabilize the horse against immediate life-threatening situations. Vitamin K_1 should be administered (1 mg per kg twice daily for five days). Synthetic vitamin K (menadione) is considerably less effective and should be reserved for less severe cases or for follow-up therapy by the owner (10 to 20 mg per kg for seven days orally or parenterally) following alleviation of the acute stage of the crisis. During the early stages of recovery, the horse should be kept quiet and should be handled as little as possible. Undue excitement and the potential for trauma carry the risk of producing internal or external hemorrhage that may be severe.

STRYCHNINE

At a dose of 0.5 to 1 mg per kg, horses may be poisoned with a single oral dose of strychnine. The rodenticide antagonizes inhibitory spinal cord neurons and allows excess activity of the central nervous system to cause convulsions and seizures in the patient. Acute onset of tetanus-like seizures is characteristic. The animal is hyperexcitable and hyperreflexic, so that wind, noise, and skin contact produce an aggravated reaction, often leading to muscle seizures, prostration, and tetanic convulsions. The syndrome is violent, with seizures progressing from moderate to fatal with 15 to 45 minutes. The seizure pattern is characteristic, and no significant postmortem lesions are found. A rapid onset of rigor mortis suggests strychnine poisoning.

THERAPY

No specific antidote is available, but seizures can be controlled with central nervous system depressants such as pentobarbital, chloral hydrate–magnesium sulfate combinations, or glyceryl guaia-

colate (5 per cent solution intravenously, 212 mg per kg). The seizures are controlled by administering these compounds to effect and as often as necessary to control muscle activity. Care must be taken to avoid depressing the respiratory center with the central nervous system depressant, and the availability of respiratory support may be vital to recovery. Activated charcoal (0.5 to 1 kg per animal) should be administered orally to bind unabsorbed strychnine in the digestve tract and to prevent continual absorption and recurrence of signs. A laxative should be administered upon clinical recovery to hasten digestive tract elimination of the charcoal-bound strychnine. Healthy horses usually recover within 24 hours if treatment is conscientiously applied.

ANTU (Alpha-Naphthyl ThioUrea)

Although toxicity is not seen frequently in horses owing to the limited availability of this agent, this rodenticide produces dramatic lethal effects if consumed. The toxic dose varies from 25 to 75 mg per kg and produces its toxicity by increased permeability of the lung capillaries. Pulmonary edema is the outstanding clinical sign. The affected horse develops moist rales, increased respiratory efforts, muscle weakness, and severe dyspnea as pulmonary edema and hydrothorax cause anoxia and rapidly developing cyanosis. As the condition progresses, foamy froth bubbles from the nose and mouth of the laboring animal. Death occurs within hours after the beginning of signs, with the animal prostrate and large volumes of white frothy foam emanating from the nose and mouth. Postmortem observations reveal hydrothorax, pulmonary edema, and frothy edematous fluid filling the air spaces of the lung, bronchioles, trachea, nose and mouth.

Therapy

No antidote is available to treat this rapidly fulminating condition. The animal should be kept as quiet as possible to reduce tissue oxygen demands, and sedation may be used if necessary. Oxygen should be administered. Osmotic diuretics (such as 50 per cent glucose or mannitol) and atropine administration (0.05 mg per kg) have been suggested to reduce pulmonary edema, but their effectiveness in field cases is not proven. Fluid balance should be ensured by monitoring skin tone and hemoconcentration. Secondary infections may be avoided by supplying broad-spectrum antibiotics during the stressful period of digestive tract irritation and inflammation.

FLUOROACETATE (Compound 1080)

This rodenticide is one of the most lethal based on a milligram dosage. The toxic dose ranges from approximately 0.25 to 1.5 mg per kg. The compound is converted to a metabolite that blocks the energy production cycle of the cell, producing cellular death due to lack of energy. When this affects the neurons of the central nervous system, dramatic major organ and central nervous system effects result. The chemical initially induces cardiac arrhythmias, a rapid, weak pulse, anxiousness, and hyperexcitability, with ventricular fibrillation, sudden excitement, and convulsions and death following quickly with sudden collapse. Because of the small quantity of toxin required to produce death, horses may consume sufficient amounts through feed or environmental contamination to become ill. The use of this chemical in range country for coyote and predator animal control may cause exposure to horses grazing native pastures. No significant postmortem lesions are found, and the rapidity of this compound's action may lead the owner to report "sudden death" as the only observation.

Therapy

No specific antidote is available, but seizures and excessive nervous activity should be controlled with short-acting barbiturates given to effect. Activated charcoal may be given orally to reduce further absorption of fluoroacetate. Glycerol monoacetate (0.1 to 0.5 mg per kg intramuscularly) may be given every hour for several doses, but its effectiveness has only been shown if given before or at the same time that clinical signs develop. The clinician must use common sense and knowledge of respiratory physiology to manage horses affected with ANTU poisoning. Recovery from this toxicity is not common once clinical signs have developed.

ARSENIC

This compound is used as a rodent and insect control agent, as well as being employed as a herbicide in agricultural weed programs. The toxic dose ranges from 2 to 7 mg per kg. Arsenic is a very irritating heavy metal and produces digestive tract irritation within hours after the ingestion of a toxic dose. Horses have signs of colic and excessive peristaltic activity and exhibit typical signs of digestive tract obstruction or irritation. After several hours, diarrhea develops, which is at first fluid and mucoid. After 12 hours, the diarrhea becomes blood-tinged owing to bleeding of the digestive tract. Affected

horses may become rapidly dehydrated from arsenic toxicity and may often be misdiagnosed as having colitis or a digestive tract obstruction. A careful physical examination will reveal congestion of most mucous membranes and obvious pain upon external and rectal palpation. The continuing excessive peristaltic activity followed by the appearance of bloody feces may help in the diagnosis.

Therapy

Dimercaprol (BAL) is the specific antidote for arsenic. It is given intramuscularly four times daily at the rate of 3 to 4 mg per kg. Sodium thiosulfate (20 per cent solution, 30 to 40 mg per kg intravenously) may be given two or three times daily in lieu of BAL. Since BAL is inherently nephrotoxic, it should only be given for three to four days. Sodium thiosulfate may then be administered until recovery. In severe acute cases, the combination of BAL and sodium thiosulfate may be valuable in providing additional sulfur for binding and detoxification of the arsenic. Since dehydration is a frequent complication of arsenic poisoning, the administration of electrolytes and glucose should be considered. Fluid balance should be ensured by monitoring skin tone and hemoconcentration. Secondary infections may be avoided by supplying broad-spectrum antibiotics during the stressful period of digestive tract irritation and inflammation.

ZINC PHOSPHIDE

With toxic doses of 20 to 40 mg per kg, zinc phosphide produces toxicity by being broken down in the acid of the stomach to phosphine. The toxicity of this compound is greater when the stomach is full owing to the additional acidity present during the digestive process. The phosphine generated produces irritation of the mucous membranes of the digestive tract, pulmonary edema, and cardiovascular collapse. The animal affected with toxicity becomes depressed, colicky and dyspneic. Occasionally horses will develop convulsive seizures. The digestive tract hyperemia is seen on postmortem examination together with excessive pulmonary fluid.

Therapy

The acidity of the intestinal tract may be neutralized by administering 2 to 4 l of 5 per cent sodium bicarbonate. This should be repeated as needed together with laxatives to purge the digestive tract of the unabsorbed zinc phosphide. Supportive treatment may be utilized, but no specific antidote is available.

VACOR

This rodenticide was very popular during the past several years but has recently been removed from the market owing to unexpected chronic adverse effects in dogs, cats, and humans. The toxic dose is in the range of 300 mg per kg. The compound is a general metabolic toxin and produces effects in the gastrointestinal tract and nervous system by apparently affecting biologic oxidation-reduction reactions. Affected horses may show colic, mental confusion and uneasiness, increased peristalsis, and some difficulty in vision.

Therapy

Because of the high dose required for lethal toxicity, most affected horses recover spontaneously, but affected animals should be treated with cathartics and supportive care to replace fluid loss and to control any nervous activity that might inflict damage to the horse or property. Nicotinamide is a specific antidote used in dogs and humans. It may be employed in horses by giving 1000 mg per kg every four hours for two days, followed by 1 gm of nicotinamide per day orally for seven more days.

PHOSPHORUS

Phosphorus is a systemic poison that has a toxic dose of 1 to 4 mg per kg. White and yellow phosphorus are the toxic forms capable of producing digestive tract, liver, and kidney damage. The early signs of toxicity are abdominal pain and a fluid, hemorrhagic diarrhea. After several days, generalized depression, icterus, and hepatorenal failure develop, followed shortly thereafter by death. A saline cathartic may be administered early in the syndrome to evacuate the digestive tract. Liver damage is obvious. Treatment is supportive and symptomatic, since no direct antidote is available. Glucose and lipotrophic agents (such as methionine) may be beneficial in hepatotoxic conditions (pp. 249 and 251).

THALLIUM

Although no longer used extensively as a rodenticide, thallium is notoriously toxic to all body systems. It has a toxic dose of 10 to 15 mg per kg, with about 50 mg per kg required to kill. Almost all body