A Survey of Poisonings Commonly Observed by the Bovine Practitioner*

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Poisonings comprise a significant portion of the total disease picture in cattle. Since cattle are almost entirely dependent upon man for providing the essentials of proper nutrition, housing, and total environment, poisonings appear to be even more important in bovine medicine than in human medicine.

Cattle have essentially become creatures of habit and have adopted the specific feeding, grazing, watering, and daily routine habits that man has imposed. Irregularities in diet and routine produced by mismanagement leave the animals with little alternative. They thus have become subject to man's whims, his attempts at efficiency or "cutting corners," and also to his stupidity and errors. The cow and bull are forced to feed and be housed where man wills. Feed, water, and therapeutic or prophylactic application of chemicals make feedlot cattle susceptible to errors in dosages, feed ingredients, faulty applications of chemicals, and general mismanagement, with the potential for poisoning always present.

It is common for poisonings to occur under widely varying circumstances. The epidemiological pattern of bovine poisonings is closely related to man's ability to properly manage his livestock. Following is a listing of the toxicities commonly observed by the bovine practitioner and brief discussions of several of the more important poisonings seen in cattle.

Poisonings Commonly Observed in Cattle

The variable husbandry practices in different geographic locations and types of cattle result in some variation in the commonly observed poisonings. In general, however, dairy cattle are more closely confined and exhibit insecticide or heavy metal intoxications most frequently. Beef or range cattle often encounter poisonous plants or dietary contaminants as the agents responsible for intoxications.

A listing of the common poisonings observed in cattle would include: Heavy metals—lead, arsenic, mercury, fluoride, molybdenum, copper, selenium; Insecticides and herbicides-fungicides—organophosphorus and chlorinated hydrocarbons, ammonium, phenolics, chlorates, arsenicals, plant-hormones, vehicles and solvents; Dietary and environmental chemicals—urea, nitrate-nitrate, cyanide, oil and salt contamination of environment, mycotoxins; Poisonous plants—bracken fern, horsetail, sweet clover, moldy corn, mycotoxins, ergot, fescue, thiaminases, nitrate- and cyanide-containing, selenium-containing, oak, algae, crotolaria; Miscellaneous poisons—rodenticides (strychnine, warfarin, phosphorus), botulism, snake and insect bites, hyperkeratosis and petroleum products, trichloroethylene-extracted soybean oilmeal, drugs (turpentine, adverse reactions).

Heavy Metal Poisons

Heavy metals are common in producing cattle poisonings because of their wide distribution and use as agricultural chemicals, components of greases and oils used on farm machinery, portions of waste materials found in garbage disposed on pastures, their presence in paints and as feed additives, and their role in environmental pollution. These compounds have the added hazard of being persistent in nature; hence their application to environments results in them being present for many years. Such problems have been demonstrated by attempts of livestock men to dispose of arsenic, lead, and mercury-containing materials by burning or dumping on open ground. The introduction of cattle to such areas in later years has resulted in severe losses.

Lead. Lead is the most common poisoning in

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cattle and is available through the ingestion of paint, lead solder or battery terminals, building materials, and various lead-containing sprays. The poisoning is usually acute and neurological in effect. Blindness, incoordination, and severe convulsive seizures are observed in affected animals. As cattle become older, the lead toxicity is usually expressed as a chronic syndrome with the convulsive pattern less pronounced. Although acute cases are treated successfully with chelating agents (calcium EDTA), the identification of a lead source for the clinical outbreak is a valuable diagnostic aid for the puzzled veterinarian. Adult cattle with lead poisoning respond well to removal from the source, good nutrition, and nursing care.

Arsenic. Arsenic is a less-frequent intoxication, but very dramatic when observed. It results from the topical application or oral absorption of arsenic-containing insecticides or orchard sprays. Severe hemorrhagic gastrointestinal pathology results in all affected animals, with a bloody diarrhea common. Deaths are acute. Treatment in the early stages with BAL (2 mg./lb.) every four hours and the application of digestive tract protectants is useful.

Copper. While the ingestion of copper usually leads to chronic accumulation of copper in the system, the signs of poisoning are acute and lead to death 36-72 hours after onset. Icterus and hemoglobinuria may be observed, but usually the animals are "just found dead." The necropsy examination is characteristic of a hemolytic crisis —generalized icterus, dark metallic-colored kidneys, swollen friable liver, and a bladder with brown-black urine. The blood is watery and often fails to clot normally. Treatment is to correct the copper-molybdenum diatary ratio by spraying 100 mg. ammonium molybdate and 1 G. sodium sulfate/head/day on the feed, adding one lb. sodium molybdate to every 200 lbs. of salt, or applying four ounces of molybdenum superphosphate/acre to the pastures.

Mercury. Fortunately, mercury poisoning due to fungicide-treated seed fed livestock is now uncommon. Occasionally, however, old mercury-treated seed may be included in animal feeds and can then produce toxicity. Mercury is a potent nephrotoxin and produces nephrosis and eventually uremia. Firm shrunken kidneys are obvious at necropsy. Although the Reinsch test may be used for laboratory confirmation of the presence of mercury in the feed, treatment is not of benefit due to the degree of damage present when a diagnosis is usually made.

Fluoride. An occasional but destructive intoxication is that produced by the chronic ingestion of fluoride salts. Most frequently this results from industrial effluents deposited on pastures and consumed with the forage. The condition is chronic and characteristically produces bone, teeth, and hoof abnormalities. Calves may be born with teeth or bone lesions if the mothers were exposed to fluorides during pregnancy. Severe cases of fluorosis will also exhibit a chronic diarrhea difficult to differentiate from other intestinal afflictions. No effective treatment is available, but the addition of aluminum to the diet will reduce the absorption of fluoride from the digestive tract.

Agricultural Chemicals

This group of compounds includes insecticides, herbicides, and fungicides; the latter two have the least potential for toxicity. The newer herbicides and fungicides are in general relatively safe, and only the use of some of the older more toxic chemicals or exposure to organic solvents used to carry the chemicals in their application are likely to produce poisoning.

The insecticides are a much more complicated and diverse group of foreign compounds. As causes of cattle poisonings, the chlorinated hydrocarbons and the organophosphorus and carbamate materials are hazardous. Fortunately, effective treatments are available for the organophosphorus and carbamate compounds; the chlorinated hydrocarbons are less effectively treated. The widespread use of insecticides around farm animals results in considerable hazard to these species. Poisoning usually occurs from accidental exposure via spray drift or due to the intestinal application of the insecticide to control livestock insects. Toxicity may result from improper dilution of the concentrated material or too frequent application of acceptable amounts. Both problems are diminished by users properly reading the labels of the respective products and abiding closely to the specified recommendations.

Chlorinated Hydrocarbons. These slowly metabolized insecticides owe their effectiveness largely to their persistence. This one vital factor has also led to their demise, since their very persistence in biological tissues and the environment has presented accumulation problems. The toxicity observed in cattle with this group of chemicals is largely neurological. Hyperexcitability, incoordination, muscular tremors and convulsions, and various bizarre postural attitudes are observed. The toxicity is acute and lasts several hours. Slow biological metabolism and excretion results in maintained plasma levels, and treatment is largely

aimed at reducing signs with sedation. Removal of affected animals from the source and the use of baths for topical applications and digestive tract lavages in instances of oral ingestion are widely used.

Organophosphorus and Carbamate Chemicals. Reduction in the use of chlorinated hydrocarbon insecticides has resulted in a great increase in the available number of organophorphorous and carbamate insecticides. These insecticides have the advantage of greatly reduced environmental and biological persistence, but their effectiveness depends upon acute and overwhelming toxicity. The public health hazard is obvious and has been demonstrated by the number of fatalities observed since the substitution in use has taken place.

The phosphate and carbamate insecticides exert their influence by reversibly or irreversibly binding acetylcholinesterase, thereby permitting continuous cholinergic stimulation. Toxicity is thus expressed as increased cholinergic function, excessive autonomic and muscular activity, and interference with respiratory function. Salivation, diarrhea, incoordination, muscle tremors and convulsions, and repiratory interference and paralysis is observed. Since the biochemical lesion associated with this group of insecticides is well known, specific treatment with atropine and one of the oxime compounds (such as 2-PAM) is very effective. Most animal toxicities are treated with atropine given to effect and repeated periodically until recovery. Cattle often only require repeated atropine therapy every 2-3 hours. Most effective results are observed if the combination of atropine and oxime is used. Of interest is the importance of early therapy. If initial treatment with the oxime is only initiated 24 or more hours after toxicity occurs, a stabilized acetylcholinesterase-organophosphorous complex may have occurred. This "aged" molecule will be relatively refractile to removal by the oximes.

Oximes should not be routinely employed in all cases of carbamate poisonings. In instances of certain carbamate toxicities, the application of 2-PAM has actually resulted in increased toxicity by developing a more stable enzyme-carbamate complex. Since carbamate poisonings are short-lived due to spontaneous hydrolysis of the acetylcholinesterase-carbamate complex and rapid biotransformation of the carbamate insecticide, treatment with atropine alone is recommended in such instances.

Herbicides. Cattle toxicity is most commonly observed from the pentachlorophenates, because of their lipid-solubility and rapid absorption, and the chloate salts, which produce toxicity by methemoglobin formation. The low cost of both these materials make them popular with local agencies for road weed control. Lesser, but occasional, toxicity is also observed from the phenoxy derivitives (2,4-D and 2,4,5-T), the dipyridyl compounds (diquat and paraquat), and organic arsenical materials, such as MSMA, DSMA, and cacodylic acid. Since no specific antidotes are available for most of these materials, general therapeutic principles, symptomatic treatment, and supportive therapy must be utilized in attempting to overcome clinical problems.

Fungicides. With the banning of organomercurials for fungicide purposes on seed, the incidence of animal toxicity due to this class of compounds has greatly subsided. The use of various toxic solvents for fungicides currently employed may lead to the assumption that the fungicide itself is toxic, but by-in-large the newer and currently used treatments are of limited hazard. Methyl bromide, captan, and dinitro-orthocresol are potentially toxic. Other toxicities may develop from the carbamate fungicides or the organotin materials.

Residue Problems. While direct toxicity to the exposed animals is a foremost concern, many of the agricultural chemicals are capable of accumulating in feed or food supplies following their application to the environment. Hence, the wide-scale use of insecticides to cattle on pasture may result in hay or feedstuffs growing on neighboring fields developing residues of these foreign chemicals. Likewise, if proper precautions in marketing the exposed cattle are not followed, the meat and by-products from such animals may contain high residues of the applied chemicals. Such matters are of vital interest to governmental regulatory agencies and provide a significant concern in their efforts to protect not only animal, but also human health. The Food and Drug Administration and the United States Department of Agriculture are diligent in their efforts to maintain the purity and safety of all foods by limiting the number and concentration of foreign chemicals present. In general, insecticides should not be used on meat-producing animals within 60 days of slaughter and should not be used on or around milk-producing animals at any time.

Dietary Chemicals

The addition of chemicals to livestock rations for the purpose of increasing feed efficiency and reducing disease is a characteristic unique to animal production. Although this practice has greatly

benefited the livestock economy, it is not without danger. Whenever foreign compounds are added to feeds, the possibility of error and resulting animal hazard increases. Due to improper mixing of the ration, incomplete following of feeding recommendations, faulty husbandry, or mismanagement, acute poisoning may result. One such common poisoning is that produced by excessive or improper feeding of urea.

Urea Toxicity. The factors associated with the development of urea poisoning in cattle are: (1) Only roughage fed before urea offered; (2) no previous urea fed; (3) "bully cattle" hogging feed; (4) cattle unusually hungry and overeating; (5) feed intructions not followed; (6) accidentally fed wrong mixture; and (7) improper or incomplete feed mixing. The production of ammonia from the urea by the ruminal microorganisms is normally followed by incorporation of the ammonia into bacterial protein; this protein is digested in the intestinal tract of the ruminant and serves as a source of nutrient protein. Under conditions of mismanagement, the production of ammonia becomes excessive and the rumen microorganisms are unable to utilize the ammonia in its entirety. Ammonia (i.e., urea) poisoning then results, with the onset of clinical signs within minutes and death often following in 1-2 hours. Rumen or blood ammonia levels are useful for diagnosis, as are the less accurate evaluations of blood urea nitrogen. The presence of an alkaline rumen (pH in excess of 8) in urea toxicity is useful for diagnosis in field situations.

Although early treatment of this condition is important for recovery, the use of calcium solutions and electrolytes, as well as supportive measures, are only slightly beneficial. Of more value are the use of rumen acidifiers (acetic acid) which serve to decrease enzymatic conversion of urea to ammonia and the administration of laxatives to remove the offending material promptly from the digestive tract. The best therapeutic results occur if treatment starts before the animal is prostrate and if complete emptying of the rumen, preferably through rumenotomy, is accomplished. Economics and medical judgement will dictate the treatment used in individual cases.

Environmental Chemicals

The occurrence of air, water, industrial, and other pollutions are just as much hazard for cattle as they are for humans. Good correlation may be observed between animal and human toxicity problems caused by chemicals polluting the environment; animal epidemiology is frequently identical to that observed for humans.

Air pollution usually results from industrial fumes released to the atmosphere, and cattle in the vicinity are exposed to sulfur and nitrogen oxides, heavy metals such as zinc and lead, hydrocarbons, and various forms of particulate matter. Since most industrial plants are located in suburban and rural areas, livestock grazing surrounding pastures are increasingly likely to assume body burdens of these chemicals or to exhibit biological responses to their inhalation. Other common hazardous gases and air contaminants are hydrogen sulfide, carbon monoxide, and fluorides.

Water pollution is a special problem for rural areas utilizing streams and wells as municipal water sources. The sewage discharge of upstream communities and industrial complexes and agricultural enterprises (feedlots, fertilization) may result in a variety of toxic materials being present in the water used by a downstream stockman or community. The same waters may enter wells supplying other farmsteads or communities. Recent interest in nitrate concentrations in water supplies has also resulted in increasing speculation as to the potential hazard to cattle of the continuous ingestion of low-level nitrate waters.

Nitrates-Nitrites. The problem of synthetic materials, such as nitrates and ammonia, and also naturally-occurring organic material, such as manure, contributing to cattle poisonings may seem remote. Unfortunately this is not so. The most obvious situation results from the excessive use of nitrate-containing fertilizers on crops and the accumulation of high nitrate levels in the harvested product. Even the application of manure to fields can result in high levels of nitrates developing in cash crops and weeds growing in such areas. An instance of a vacant feedlot that had grown a lush crop of weeds and was used to provide green pasture for cattle illustrates this problem. Within a few hours after a large group of feeder cattle were turned into the weed-covered feedlot, the owner found 17 cattle dead and numerous others in various stages of toxicity. Even though the cattle were promptly removed from the lot, approximately one-third of the animals died from acute nitrate poisoning. Analysis of the weeds growing in the area revealed concentrations of nitrate as high as 4.5%.

A complicating situation is the relationship between adverse growing conditions and the accumulation of nitrates in plant materials. Although levels of nitrate may be moderate in soils supporting grain crops (particularly corn and sorghum) or a variety of weeds, under the

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influence of a drought or the application of plant-hormone herbicides, these commonly-grown plants may accumulate excessively high and toxic concentrations of nitrate. The widescale losses in the middle 1950's due to cattle consuming drought-affected corn and sorghum were largely due to nitrate toxicity. The application of 2,4-D to weeds frequently permits these plants to develop transient toxic concentrations of nitrate; if consumed during this temporary phase, acute nitrate poisoning may result. Methemoglobin formation results in methemoglobinemia, but treatment with methylene blue is usually quite successful.

Of special interest is the occurrence of chronic nitrate intoxication due to long-term ingestion of low levels of nitrate from either water or feed supplies. While controversial, most veterinarians recognize a chronic condition in cattle that relates to the presence of increased nitrates in the diet. Clinical signs are non-specific, but often include decreased weight gain and feed efficiency, decreased milk production, poor reproductive capacity, and impaired health of epithelial tissues as expressed by digestive tract and respiratory disorders. Interference with the liver conversion of carotene to vitamin A, depression of thyroid activity by interference with iodine, and metabolic interference with vitamins A, E, D, B_{12} and K utilization have been suggested as possible mechanisms of action. Replacement therapy with quantities of vitamin A, iodine, and nutritional supplements are often of value in field cases, and response to this regimen supports the diagnosis.

While the influence of nitrates on cattle health has only partially been defined, there is little doubt that other foreign chemicals present in water supplies due to industrial and agricultural pollution are indeed capable of producing significant toxicity. These chemicals include arsenic, mercury, petroleum products, salt, insecticides, herbicides, and fungicides.

Other pollutants can produce toxicities and under circumstances that are frequently unique to the situation. Carbon monoxide is a special problem during winter weather in animals confined in tightly sealed quarters. Tractors or improperly vented heating equipment utilized within such facilities may produce lethal concentrations of this gas. Coal-tar (pitch, clay pigeon), chlorinated napthalene, and various phenols (creosote) are capable of entering the animal's body and producing poisoning because of their presence in the environment. The severe toxicity of the various rodenticides and their wide use presents a hazard for livestock and companion animals alike. As with most environmental problems, prevention of pollution is a matter of education and regulation, and prophylactic efforts are immeasurably more rewarding than the treatment of poisoned cattle.

Naturally-Occurring Toxins

Cattle poisonings due to a variety of poisonous plants are a significant part of bovine practice. The basic factor underlying most plant poisoning problems is mismanagement. When weather conditions are bad, cattle pastures may provide less forage than expected and limited plant material for animal consumption and overgrazing frequently results. Under conditions of repeated overgrazing, naturally-occurring desirable pasture grasses die out and weeds, many of them poisonous, take over the range. While such weeds are normally unpalatable, hungry livestock may be forced to consume them and thereby become poisoned. Although most owners recognize overgrazing and the resulting hunger that results in their animals, mismanagement aggravates the loss of native pasture grasses and speeds the introduction and multiplication of poisonous plants. When owners do not provide supplemental feeding for animals on such poor pasture land, the cattle are forced to consume the noxious weeds.

Cyanogenic Plants. The cyanide-producing forages are a common cause of cattle sicknesses. Usually the sorghums (Johnson grass, sudan grass, milo) are responsible for losses. In addition, arrowgrass (*Triglochin* spp.), elderberry (*Sambucus* spp.), wild cherry (*Prunus* spp.), and the pits of several common fruits (apple, peach, apricot) contain compounds with the potential of releasing cyanide upon ingestion. Toxicities usually result from ignorance on the part of the owners who feed such plant materials to their animals or who throw fence-row clippings into pastures to utilize the material for forage. Adverse weather conditions and wilting frequently increase the toxic potential from this group of poisonous plants.

The clinical condition results from the stabilization of the iron of cytochrome oxidase in the ferric state. Thus, cellular respiration is halted by blocking the transfer of oxygen from blood to the cells. The characteristic "cherry-red" appearance of blood is observed. Tissue anoxia is present and produces acute incoordination, respiratory and cardiac hyperactivity, convulsions, and death with the heart continuing to beat several minutes after respiration has stopped. Intravenous therapy with sodium nitrate and sodium thiosulfate is extremely effective and miraculous responses are observed in animals that no longer exhibit respiratory efforts. Several varieties of sorghums have been developed

that have limited cyanide-producing capability during normal growth conditions. On occasion, severe or unusual weather has introduced a hazard, but further refinements in plant genetics should greatly alleviate the once common threat of cyanide poisoning to livestock.

Locoism. Locoweeds (*Astragalus* spp. and *Oxytropis* spp.) are common and characteristic causes of cattle losses in the western states. The plants are not usually consumed, but under adverse growing conditions and inadequate natural forage, animals may be forced to consume them. Once livestock taste the plants they may develop a liking for their flavor and will then preferentially consume them, even if other feed is supplied. The clinical syndrome is one of weight loss and mental derangement. Animals become easily aggravated and undergo bizarre temperament changes. Deaths frequently result from self-inflicted injuries due to running through fences, falling down wells, or drowning in ponds or streams. The toxin has an affinity for the nervous system and characteristic microscopic lesions are detected in the neurons of the brain.

Perirenal Edema Syndrome. This interesting postmortem finding usually follows a 1-3 day clinical course of progressive weakness, abdominal distension, and coma and death. The blood urea nitrogen is extremely elevated and a tentative diagnosis of urinary tract obstruction and ruptured bladder is frequently suggested. The syndrome has been seen associated with pigweed (*Amarenthus retroflexus*) and oak (*Quercus* spp.) ingestion.

In addition to the marked edema seen around the kidneys on postmortem examination, hemorrhage and frank clotted blood may also be observed intermixed with edematous fluid in the perirenal area. The kidneys appear soft to the touch and microscopic study reveals extensive tubular necrosis with characteristic hyaline casts present in the lumen. Extensive ascites and hydrothorax (100 ml. to several liters) are common. Because of its insidious nephrotoxic nature, diagnosis of this syndrome is frequently only made on postmortem examination.

Selenosis. Selenium is found in certain rocks and hence specific types of soils. Plants growing on such soils may accumulate levels of selenium in their plant structure varying from only a few ppm to several thousand ppm. While any plant growing on soils containing selenium may build up low levels of this chemical, specific plants, such as poison vetch (*Astragalus* spp.) and woodyaster (*Xylorrhiza* spp.), have the ability to selectively take up and accumulate massive amounts of selenium. Some of these plants (goldenweed, *Oonopsis* spp., and princesplume, *Stanleya* spp., for example will only grow on soils high in selenium; hence, they are called "indicator plants," since they indicate the fact that selenium is present in the soils on which they are growing.

Toxicity due to selenium will produce varied clinical signs, depending upon the concentration of the chemical in the consumed plants. Low levels of selenium may result in weight loss, deformed hooves and hair loss, and the birth of deformed young. Larger amounts of selenium cause liver damage. A nervous syndrome, similar to that seen with locoism, commonly develops if large amounts of selenium are ingested. Treatment of the acute form of selenium poisoning is of no avail, but the addition of 37.5 ppm of sodium arsenite in salt or five ppm in water has been helpful in reducing the toxicity of long-term ingestion of low levels of selenium.

Acute Losses. Several other poisonous plants produce such rapid death that owners may report only that the animals were found dead. Waterhemlock (*Cicuta* spp.) grows in wet areas and contains the highest toxin concentrations in chambers just above the roots. Cattle become exposed by crushing the plant in the stream in which it is growing and them consuming the toxin-containing water, or they may consume one or more mouthfuls of the actual plant material. The toxin is so severe that violent muscle spasms may knock the animal off its feet and cause death within minutes. Cattle poisoned by waterhemlock have been found with walnut-size pieces of the plant still in their mouths.

Cocklebur (*Xanthium* spp.) is a weed capable of infesting almost any barren ground. Since the plant is so common, it is fortunate that its most potent state is during the early spring growth period, but unfortunately this is just when other green vegetation is limited. Cattle may be browsing pastures when early spring rains produce cocklebur sprouting. The resulting sprouts contain high concentrations of a potent toxin capable of producing massive liver necrosis and death within a few hours. The mature cocklebur plant is not palatable, but the burrs containing the seeds may sprout in the fall following a warm rain. Hence, poisoning not only occurs in the spring, but also in the fall when sprouting cockleburs again provide lush forage for grazing animals.

The Japanese yew (*Taxus cuspidata*) is an ornamental plant that recently has been shown to produce acute poisonings in animals consuming clippings. The plant is frequently installed around

fences and animals may become exposed to clippings or may browse the plant directly through the fence. The toxic principle has not yet been fully defined, but ruminants have been found dead by amazed owners following access to this plant.

The abundance of algal growth in various water sources indicate the widespread nature of this plant, but belie the acute and severe losses that may result from consumption of certain species of this group. The blue-green algae are especially toxic and under conditions of rapid growth are capable of developing concentrations of endotoxin that, upon ingestion, produce sudden and high losses. Weather conditions may result in accumulation of the algal growth in one area of the water supply. Cattle drinking from that area will then receive lethal quantities of toxin. While acute losses are most commonly seen, a variety of other syndromes may be associated with sublethal ingestions. Losses after two or three days due to hepatic damage and occasional cases of photosensitization have resulted. No effective treatment is known for the acute problem and symptomatic and supportive therapy is utilized in more chronic instances.

Mycotoxins. Some of the lower members of the plant family, the fungi, can produce a variety of toxins. The mycotoxins have become a focal point for scientific investigation during the past decade, and studies on aflatoxins have been especially productive. This latter group of mold poisons is produced by specific strains of *Aspergillus flavus* and *Penicillium* spp. The toxins are products of fungal growth and the mere presence of the fungus does not necessarily indicate that its particular toxin is also present. Conversely, the toxin may be present and viable fungi may no longer exist in the sample. Mold toxins commonly develop in stored grains and on certain feedstuffs subjected to unusual weathering or storage conditions. Although moldy feed is usually grossly identifiable, the spoiled feed may be mixed into a ration or otherwise offered for livestock consumption. Cattle will usually reject heavily spoiled feed, but well-diluted feeds or rations offered hungry cattle may result in acceptance and toxicity.

Most fungal toxins affect the liver and produce lesions varying from frank necrosis to interference with enzymes or blood-clotting mechanisms. Digestive tract disturbances, photosensitization, poor feed utilization, abortions, and reproductive failures have also been associated with mycotoxin consumption. Unfortunately, only a few of the fungal toxins have been isolated and identified. The experimental production of tumors by aflatoxins in some laboratory animals has resulted in concern over concentrations of this toxin in grains destined for human consumption.

The aflatoxins appear the most potent of the currently recognized mycotoxins. Concentrations in the diet as low as 50 ppb aflatoxin B_1 can cause hepatic damage in laboratory animals. In cattle, dietary levels of 300 ppb aflatoxin B_1 will result in decreased feed efficiency, and higher concentrations will reduce growth and produce illness. Feed levels of 1-2 ppm or more are capable of causing acute death. The constant threat of spoilage due to adverse weather conditions or faulty storage facilities increases the potential for aflatoxins occurring in the diets of cattle. Unfortunately, relatively sophisticated laboratory confirmation is necessary to determine the level of aflatoxins in feeds.

Fescue-Ergot-Freezing. A complex of etiologies is often presented to the veterinarian when a distal-gangrene condition in cattle is brought to his attention. Dry gangrene of one or more extremities, including the ears, tips of the tail, and mammary teats, may be observed due to poisoning from fescue grass (*Festuca arundinacea*), ergot (*Claviceps* spp.), or as a result of freezing during exposure to winter range conditions. The outward signs of all three are similar and without circumstantial evidence and the application of clinical judgment, a differential diagnosis is impossible.

Gangrene due to fescue is more common in cold weather and characteristically follows the onset of a cold spell by 2-3 days. Hind-feet gangrene is primarily observed with fescue poisoning, but gangrene of all extremities is also occasionally seen. Ergot toxicity may affect any extremity, including the tail and tip of the ears. It occurs at any time of the year and is associated with the finding of the dark sclerotium of ergot in the grain fed the affected cattle. Because of the vasoconstrictive properties of the ergot alkaloid, decreased blood flow to other areas of the body may also result in decreased milk production and semi-starvation in nursing animals. Freezing is always associated with extremely cold weather and exposure, and most commonly is observed in recently-born calves trapped in snow banks or caught by winter storms on the unprotected range.

Since all three conditions produce the same end-effect (i.e., dry gangrene of various extremities), treatment is identical and in early cases is aimed at re-establishing circulation to any still-viable tissues. All-too-often, owner neglect results in the gangrene being advanced and practical efforts must be aimed at salvaging the animal. Removal from the causative factor, local

treatment of the affected area and prevention of secondary infections, and good nursing care are helpful in permitting the owner to realize an economic return.

Miscellaneous Chemical Hazards

Petroleum Products. A variety of oils, greases, benzenes, hydrocarbons, and other petroleum products are used on and around cattle. Some are employed directly on machinery to which livestock have access; several are utilized as solvents for sprays and materials applied directly to the animals; others are formulated for application to buildings, and cattle lick or otherwise contact the applied product; or animals may directly consume the petroleum products by gaining access to storage areas housing opened containers of these materials. In certain areas of the United States, oil wells and oil storage tanks provide the potential for cattle consuming the crude petroleum product. Incomplete fencings of crude oil holdings or storage areas may permit inquisitive cattle to satisfy their curiosity.

Petroleum products produce a characteristic sequence of clinical signs. If applied to the skin, irritation and thickening commonly results. Photosensitization is frequent, especially in white-haired or light-skinned individuals. If consumed by mouth, the petroleum material produces digestive disturbances, may be inhaled with resulting pneumonia, and after several days can produce liver, kidney, and bone marrow dysfunction. Pregnant animals may abort and a poor-doing individual, continually losing weight and eventually dying is usual. Poisoning due to petroleum products is a complicated and varied intoxication. Its occurrence could largely be prevented by assuring that proper precautions were taken to prevent animal access to these materials.

Drugs and Other Therapeutic Agents. Errors in the choice of a therapeutic compound, in its route or mechanism of application, and in its dosage are common lapses of judgment that result in toxicities. The faulty selection of a worming preparation, the administration of a new chemical by an other-than-recommended route of administration, and the overdosage of an anesthetic are dangerous and frequently fatal errors. Not only are such errors subject to frank poisoning, but adverse reactions and a multitude of possible acute and chronic variations may also occur.

The oral and topical application of anthelmintics and insecticides can produce toxicity in cattle. In such instances, overdosing, misuse, or administration to debilitated animals are the usual causative factors. Careful reading of the product-container label and following of instructions is important. The label also contains a listing of contraindications and suggested therapy. In an emergency, the manufacturer will often be able to provide the most recently available therapeutic information.

Antibiotics, hormones, trace elements and minerals, vitamins, and parasiticides are added to rations to increase feed efficiency and/or animal health. Although these compounds seldom produce toxicity in consuming animals, they are prominent regulatory concerns if their use results in residues in animal products intended for human consumption.

Problems of Chemical Residues. Because of the Food and Drug Administration's standards for food purity, foreign chemicals are not permitted in animal and human foods in excess of guideline or tolerance levels. Residues of chlorinated hydrocarbon insecticides have been a common problem in past years and have led to the drastic reduction in their use on and around animals intended for human food production.

Chemical residues in foods are a hazard due to potential sensitivity reactions, the establishment and transfer of bacterial resistance, cancer-producing potential, effects upon the fetus, toxicity in more sensitive individuals, and various subtle adverse effects in select portions of the population. Residues may also affect biological response in other foreign chemicals by participating in chemical interactions and, among other mechanisms, contributing to enzyme induction or inhibition.

The government's concern with the accumulation of foreign compounds in not only foods, but also the general environment, has resulted in the consideration of restrictions on the use of several commonly used insecticides. An initial action was the banning of DDT from routine usage in the United States. This has been followed by the study of other chlorinated hydrocarbons preliminary to possible similar restrictions. Some of the heavy metal chemicals are also being re-evaluated because of their hazard and adverse environmental impact. Whether they will indeed be banned from use or only have restrictions placed on their applications remains to be seen.

Summary

Cattle are constantly exposed to a wide variety of foreign chemicals, many of which are potentially toxic. Ignorance can result in a number of unusual and sometimes fatal clinical syndromes. Pesticides are applied on or around animals and may be placed in areas without regard for

accessibility to cattle. Insecticides, herbicides, and fungicides are routinely and haphazardly applied to animal and environmental surfaces alike. Drugs are considered to have therapeutic effects; but disregard for recommended dosages can result in poisonings.

Failure to provide satisfactory storage facilities for animal feeds and the improper preservation and handling of feedstuffs allow the development of a variety of mycotoxins.

The dependence of animals upon their owners for the total environment makes these animals susceptible to environmental pollutants. Exposure to noxious gases, irritating and hazardous industrial materials and wastes, water contaminants, and casually discarded compounds of man's own use can result in illnesses and death. As long as such potentially toxic materials exist and are utilized, the hazards for cattle will be a prominent concern of the bovine practitioner.

Selected References

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enables us to find out exactly what the farmer is doing in his management, or conversely, to convey to the farmer exactly what we suggest he do in his management.

At an annual meeting with all the farmers in the group, the average performance of all herds is discussed, and an anonymous list of individual herd performances. Each farmer can see where he is in the efficiency order, learn what the potential is, and in the discussion, what are the techniques which are best used to achieve it. These are good meetings for us to measure consumer resistance to new procedures we would like to introduce, like a rise in fees.

Conclusions

Well, that is the system and I hope you were not too confused. It is a difficult subject to describe in detail in a few minutes, but I could see no point in discussing the subject only in generalities. Consideration of the detailed workings of a program such as this is one of the two important ways of conveying whether or not it is practicable. The other important way of demonstrating practicability is by demonstrating that the desired results can be achieved. I think I have done that in the mastitis paper and I hope to add to that in the talk on fertility tomorrow (see other paper). Those results should convey the impression that in our hands it is a practicable program. However, in spite of anything I may have said or may still say about its virtues, and I am inclined to exaggerate to make a point, the cold fact is that it is a provisional program and very much on trial in a full commercial situation.

We have every confidence in it in the rather narrow limits of a high-priced liquid milk production system.

Although we think it can be readily adapted to dairy herds producing milk for processing into other dairy products, especially butter, and to beef herds, we have not had enough experience in these areas to say how the adaptation should be done.

Editor's Note: For an extensive discussion of this and other programs, please refer to pages 13-26, Proceedings of the 1973 AABP Convention.

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