SCIENTIFIC REVIEWS

Toxicopathology of Oilfield Poisoning in Cattle: A Review

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ABSTRACT. The toxicologic pathology of petroleum and oilfield-related chemicals is reviewed, and a field gui for toxicopathologic evaluation of cattle is given. Cattle will voluntarily ingest petroleum and chemica used in the exploration, production and transportation of crude petroleum. Variability in chemical compositi of petroleum from different fields will alter the type and severity of lesions observed. When airborne poll tants are present, cattle are continually exposed and make excellent sentinel animals. The lung, kidney, li er, gastrointestinal tract, heart and brain are target organs for petroleum hydrocarbons. Exposure to eleme tal sulfur can produce pulmonary pathology. Sulfur-containing gases are irritating to the mucosa of the e and respiratory tract. Arsenic and lead cause lesions in the gastrointestinal tract, brain, liver and kidne Glycols are hepato-, nephro- and neurotoxic, and oral exposure to diethylene glycol produces corneal lesion Invert drilling fluids are fetotoxic. Nonpesticide organophosphate esters target the peripheral and centr nervous systems. Toxicopathy is a strategic tool in the diagnosis of intoxications occurring in cattle aft exposure to oilfield chemicals. Cattle are sensitive to oilfield pollutants and are a useful biomonitori species.

EXPOSURE

Exposure to intoxicating substances in oil and gas operations occurs under a variety of circumstances. Specifics of exposure conditions and clinical findings are discussed elsewhere (1), but cattle will voluntarily ingest various forms of petroleum and chemicals related to exploration, production and processing of crude petroleum (1,2). Exposure also occurs when feedstuffs, water and air are polluted from environmental incidents. Sour crude petroleum contains various sulfur compounds (3). Elemental sulfur and sulfur-containing gases are by-products of scrubbing sulfur-containing gases from sour petroleum; this process is also known as "sweetening". Cattle can be exposed to elemental sulfur and various sulfur compounds related to sour petroleum activities (3-6). Heavy metals are used in oilfield operations (7), and exposure of cattle occurs during access to drilling wastes and wellhead equipment. Pathology is one of the strategic tools used to link petroleum exposures to clinical effects in cattle.

PETROLEUM HYDROCARBONS

Toxicopathy in the Lung

In acute petroleum poisoning in cattle, especially with petroleum having a high content of volatile fractions, the lung is the target organ (8-10). Chemical (inhalation) pneumon occurs when droplets of oil are inhaled. Fo lowing oral ingestion, another cause of che ical pneumonia is aspiration of petroleum h drocarbons occurring during emesis and/ eructation. Disruption of protective reflex by the effects of hydrocarbons on the centr nervous system of cattle may contribute aspiration (1). Cattle can also inhale vol tile hydrocarbons during eructation by t eructation-inhalation reflex.

Lung lesions have been reported followi voluntary ingestion of petroleum by cattl Serosal petechiae and ecchymoses were o served in the lungs of cattle poisoned wi tractor vaporizing oil (11). Chemical pne monia was reported in cattle dead of dies fuel poisoning (12). Pneumonia and pulmona abscesses containing Gram-negative organis were found in cattle that died from drinki water contaminated with aviation turbine e gine fuel (13). Cattle that drank aviati turbine engine fuel were necropsied 124 da later. The lungs from most of the cattle we considered abnormal because of enlargeme and the presence of grayish-blue pulmona tissue with an abnormally firm consistenc However, histopathologic abnormalities we not observed. Infectious pneumonia often de velops rapidly after chemical injury to th lung. Cattle that survive chemical pneumon for 6 weeks or longer usually have marke loss of weight and chronic ill health, pleu ral adhesions and lung abscesses (8,9).

The characteristic lesions of oil-induce chemical pneumonia have been described (14) The pathogenesis and lesions can vary wit

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the cytotoxicity of the oil and concurrent bacterial infection. Generally, the cranioventral lobes of the lungs are affected first and may be observed to have oil-discoloration. Visible oil can be present in the lungs. In kerosene poisoning, ulceration of the ventral aspect of the trachea can occur, and the ulcers may be covered with a pseudomembrane (8, 9). Areas of the lung can be consolidated and discolored dark purple, reddened or mottled tan-gray. Consolidated areas can have a nodular appearance. Fibrinous pleural adhesions and serofibrinous pleural transudates may occur. Depending on the duration of the pneumonic process, abscessation can also be observed (8,9).

Histopathologic lesions from petroleum toxicosis are predominantly proliferative. Macrophages with foamy cytoplasm are found in and often fill the pulmonary alveoli (14). Pathogenic studies of oil toxicosis identified foamy macrophages in the lymphatics adjacent to blood vessels around bronchi. Fibrosis and proliferative type II macrophages are prominent features. Foamy-appearing macrophages may be incorporated into the alveolar septa by fibroblastic proliferation. Lipids, as detected by histochemistry and special stains, are both intracellular and extracellular. Oil generally can be identified in the alveoli. An acute necrotizing fibrinous bronchopneumonia may be observed in the early stages of chemical pneumonia resulting from oil. Neutrophils, lymphocytes and plasma cells generally surround the necrotic areas. Fibrinous exudate, inflammatory cells and amorphous eosinophilic and oily material can fill alveolar spaces. Empty spaces in tissues suggest that lipoidal liquids have been absorbed. Plant material from aspirated ingesta may be deposited in alveoli. Fibrin, hemorrhages and a mixed mononuclear cellular exudate can be observed on pleural surfaces. Extensive coagulative necrosis and suppuration may be found along with pulmonary consolidation. Pathogenic and saprophytic bacteria also may be present in necrotic areas and the alveoli.

Pulmonary lesions in experimental studies have been described (10). When cattle were given 20 to 60 ml of crude oil/kg of body weight and emesis occurred, the cattle aspirated oil. The results were an extensive black discoloration in the lungs (80% of the lung field in 1 animal) and visible oil in the cranioventral lobes. Pathologic changes included pulmonary consolidation and fibrinonecrotic pneumonia, pleuritis, emphysema, atelectasis, multifocal interstitial pneumonia, proliferative alveolitis, bronchopneumonia and alveolitis. In animals that survived 8 d, hyperplasia of type II pneumocytes was observed, and the alveoli were filled with large foamy alveolar macrophages.

Lipid emboli occur in the lungs of some animal species as a result of oil being absorbed from the gastrointestinal tract. Emboli of oil can block small blood vessels in the lungs and brain. Embolism in cattle poisoned with oil has not been reported.

Pulmonary lesions were observed in sheep after a 1-d exposure to water contaminated with natural gas condensate (15). Lesions at necropsy included bilateral consolidation of the lungs and tan to gray mottling of the ventral lobes. The histopathologic diagnosis was necrotizing bronchopneumonia. Extensive areas of coagulative necrosis bordered by a dense zone of neutrophils and lesser numbers of lymphocytes, plasma cells and macrophages were observed. The alveoli contained a fibrinous exudate and amorphous eosinophilic material. Plant material indicative of emesis and regurgitation-aspiration was observed in the lungs. Pleural surfaces were covered with fibrin; subpleural hemorrhages and monocular cells were observed. Pathogenic bacteria were isolated from some of the sheep lungs.

Toxicopathy in the Gastrointestinal Tract

The gastrointestinal tract has initial contact with petroleum when oral exposure occurs. Crude oil is irritating to the intestinal tract of cattle (16). Ulcers of the esophagus due to ingestion of kerosene have been reported (8,9). Pyloric ulcers were observed in the abomasum of cattle dosed with a sweet crude oil (10). In the same study, rectal lesions were described as mild congestion to mild hemorrhage with focal areas of inflammation. Mesenteric lymph nodes were discolored by oil. Petroleum can be observed in ingesta and its odor is detectable in ruminal and intestinal contents.

Ingestion of water contamined with natural gas condensate caused reddening of the gastrointestinal serosae of sheep (15). Hemorrhage was present in the intestinal lumen. Histopathologic observations were mucosal-submucosal congestion and mucosal-serosal hemorrhage. An inflammatory response was observed in all anatomic segments of the gastrointestinal tract. Superficial erosions, mild acute cryptitis, and neutrophilic lymphyocytic exudates were present. Multifocal necrosis of enterocytes was reported.

Toxicopathy in the Liver

Petroleum hydrocarbons are hepatotoxic in cattle and sheep. Fatty liver was observed in cattle that ingested tractor vaporizing oil (11). An abnormal-appearing liver in a heifer was attributed to drinking gasoline 15 mo previously (17). Fatty degenerative changes and focal areas of hepatitis were observed in cattle following ingestion of water contaminated with aviation turbine engine fuel (13). Sweet crude oil was hepatotoxic in cattle. Gross changes were swelling of the liver, increased liver weights, increased friability and centrilobular congestion (10). Hepatocytes contained numerous vacuoles that were interpreted as having contained oil. Hepatic changes in sheep poisoned by natural gas condensate were fatty degeneration and periportal infiltration of lymphocytes-plasmacytes. Biliary hyperplasia and periportal fibrosis were described (15).

Toxicopathy in the Kidney

Kidneys can also be target organs of petroleum hydrocarbon toxicosis. Renal lesions oc-

cur in cattle exposed to a variety of petroleum products. Tractor vaporizing oil was nephrotoxic in cattle (11). Tubular nephrosis, thrombi in renal vessels and interstitial nephritis were observed in cattle following ingestion of water contaminated with turbojet aviation fuel (13). Renal lesions were not seen in surviving animals slaughtered 124 d later. Oral exposure to an unweathered light crude oil induced renal lesions in cattle (10). These lesions were shrunken or collapsed glomeruli (2–5 in a 4x field), mild focal necrosis of epithelial cells in collecting ducts and inflammatory cells in the renal cortex. In sheep that drank natural gas condensate, tubular epithelial necrosis was observed (15). Granular eosinophilic casts (negative for hemoglobin) and protein droplets filled Bowman's spaces and many renal tubules. Inflammatory cells were around the tubular casts.

Toxicopathy in the Nervous System

Based on clinical signs, the physiology of the nervous system is altered by petroleum, especially petroleum high in volatile fractions (1,10,18). Lesions were in the brains of sheep poisoned by natural gas condensate (15). Mild perivascular hemorrhage was in the pia mater and white matter of the cerebellum and cerebrum. Increased separation between the pia and arachnoid membranes was prominent, and edema was observed in the stroma of the chorioid plexus.

Toxicopathy in the Heart

Lesions in the hearts of cattle poisoned by petroleum have not been reported; however, such lesions were described in sheep exposed to petroleum condensate (15). Gross pathologic lesions were epicardial hemorrhages, serosanguineous pericardial fluid, pale appearing myocardium and erythema of the endocardium. Histopathologic observations were segmental myocardial necrosis and calcification. Myofibrils had lost their cross striations and had a beaded appearance. Multifocal areas of the myocardium were congested and infiltrated with lymphocytes, macrophages and occasional neutrophils.

Toxicopathy in the Mammary Gland

Irritation of the mammary gland related to ingestion of hydrocarbons has been reported (20).

Clinical Pathology

Changes in clinicopathologic parameters can be used to aid the diagnosis of petroleum toxicosis in cattle. One week after exposure to turbojet aviation fuel, clinicopathologic changes in the sera of cattle consisted of elevated aspartate aminotransferase activity and increased concentrations of nonesterified fatty acids and blood urea nitrogen (13). Leukocytes were increased. The activity of γ-glutamyl transpeptidase was increased and the increased activity persisted for 6 w. In acute studies in cattle, sweet crude oil did not significantly alter hematologic parameters. There was a correlation between the severity of clinical signs and an increase in plasma fibrinogen. Hepatocellular enzymes in serum and blood glucose were not consistently increased after exposure to sweet crude oil. Exposure consistently decreased serum calcium and potassium and consistently increased bilirubin (10).

Rowe found that hematologic parameters, especially leukocyte counts, were elevated during the development of crude oil-induced chemical pneumonia (8,9). Leukocytic differential counts suggested an acute infection. A single dose of sour crude oil caused a constant decline in plasma glucose. A transient decline in plasma glucose was observed in calves given sweet crude oil or kerosene. Two days after they were dosed with sweet and sour crude oils, calves had increased serum α-globulin. Calves dosed with sweet crude oil had the most pronounced increase in serum α-globulin. Calves dosed with kerosene and sour crude oil had increased α-globulin by days 10 and 22, respectively. These observations suggest that acute phase reactive proteins may also be increased. Calves treated with sweet or sour crude oils or kerosene had decreased serum γ-globulins.

DRILLING FLUIDS

Drilling fluids are used in drilling oil and gas wells. The fluids are pumped down the drill stem and return to the surface in the space between the bore hole and the drill stem, carrying the cuttings to the surface. Drilling fluids are formulated to stabilize formations, lubricate the drill stem, scavenge sulfides and provide hydrostatic pressure. The chemical composition of drilling fluids can be variable. For example, potassium salts are used to increase the density of drilling fluids, a variety of chemicals may be added to adjust pH and viscosity, and the fluid may consist of petroleum hydrocarbons. Invert drilling fluid is a mixture of diesel oil and surfactant. Oil well sumps contain the drill cuttings and residual drilling fluid.

Ultrastructural pathology has been described in a calf exposed in utero to invert drilling fluids (21). Reduplication of the smooth endoplasmic reticulum was observed in the liver, and the endoplasmic reticulum of the renal tubular cells was swollen. Mitochondria in the liver and kidney were swollen, polymorphic and predominantly parabolic in shape.

SULFUR AND SULFUR-CONTAINING GASES

Elemental Sulfur

Cattle exposed to sulfur dust and fumes from an open-pit mine-source in Poland were poisoned by elemental sulfur (22). Since elemental sulfur from "sweetening" sour petroleum is released into the ambient environment (6), the Polish observations may have application to sulfur pollution from the oil and gas industry. Pathologic findings in exposed cattle consisted of gray-yellow tinged mucus in the respiratory tract, inflammation of the trachea, attenuation (decreased height) of bronchial epithelial lining cells almost to the point of squamous metaplasia, and interstitial inflammation. The lesions were worse in cattle with higher exposures.

Sulfur-containing gases, generally complex mixtures under environmental conditions, have also poisoned cattle. Sulfur dioxide caused histopathologic evidence of severe irritation of the respiratory tract and eyes (23). Manure gas poisoning (biogas with high concentrations of hydrogen sulfide) produced congestion and edema of the lungs, gastroenteritis and peritoneal effusions (23).

Detailed reports on the pathology of hydrogen sulfide (H_2S) and sour gas in cattle do not appear in the scientific literature. Calves continually exposed to H_2S by inhalation developed respiratory irritation, epistaxis, and corneal opacity (24,25). The interactive effects between H_2S and ammonia were given as synergistic.

HEAVY METALS

Chromate

Cattle have been poisoned by chromates from oilfield sources (26). Grossly visible lesions in an 8-mo-old calf were icterus, froth in the trachea, excessive pleural fluid, and serosal petechiae. Intestinal contents were black and covered with mucus. Edema and hemorrhage were observed in the mesentery and omentum. Hepatomegaly, yellowing of the liver, and edema of the gall bladder were observed. The kidneys were pale. Histopathologic findings were diffuse hepatocellular swelling, vacuolation of hepatocytes, inspissation of bile and moderate biliary hyperplasia. In the kidney, hemoglobinuric vacuolation was observed in the cortical tubular epithelium. Subepicardial hemorrhages and pulmonary proteinaceous exudate were observed. The chromium concentration in the liver was 14.8 ppm.

Arsenic and Lead

Cattle can be poisoned by arsenic and lead from oilfield sources. In some areas, arsenic is used as a rust inhibitor and herbicide. Lead is used in paints, pipe dope, sealant and waterproof greases (7,27). Cattle will ingest substances containing arsenic and lead (7). The gastrointestinal tract and nervous system are targets for inorganic arsenic and lead.

Inorganic arsenic affects the gastrointestinal tract, liver and kidney of cattle. Arsenic alters and inhibits mitochondrial enzyme pathways and thereby impairs tissue respiration. Death can occur in less than 24 h following ingestion. A lag of 24 to 48 h can occur between ingestion and onset of clinical signs (28). Although marked signs of gastrointestinal distress may be observed, lesions at necropsy in these tissues may be sparse (29). Vascular injury is common in arsenic poisoning. Hyperemia of the mucous membranes can occur and petechiae may be observed on serous membranes. Arsenic causes hyperemia, edema, and superficial erosions of the abomasal mucosa. The gastric rugae and plicae may be edematous (30). The intestinal contents may be a watery fluid, and contain blood, shreds of mucosa and mucus. Intermucosal and submucosal hemorrhages occur, and these can progress to massive erosions of the mucous membrane. Hemorrhagic typhlocolitis can also occur.

The liver, kidney, brain, heart and conceptus are also affected in arsenic poisoning (29). Congestion and swelling of the liver with microscopic lesions of fatty degeneration and hepatocellular necrosis occur. Renal edema and necrosis of renal tubular cells are observed. Marked subendocardial subserosal hemorrhages are seen. Brain lesions generally develop later than those in the viscera and are probably due to necrosis of cerebral vessels. The lesions are cerebral edema, petechiation and hemorrhage. Abortions and stillbirths can occur. This effect is also associated with vascular damage.

Lead affects the brain, liver, kidney and hemopoietic system of cattle (30). Cattle are sensitive to lead, and clinical signs of poisoning can occur after exposure to 2 mg of elemental lead/kg body weight for 28 days (32).

An excellent description of the toxicopathy of lead in cattle has been written (33). Macroscopic changes may or may not be present and, when present, vary in severity. Congestion of meningeal and cerebral blood vessels and cerebral edema may be present. Cerebral gyri may be flattened or depressed, and the color of the gyri may be pale to yellowish. On the cut surface, malacia varies from small foci to large cavitations. Cortical lesions are most visible at the tips of the gyri. Lesions in the occipital lobes develop first. The insula may also be affected.

Histopathologic changes vary with the duration of clinical illness (29,33). Swelling of endothelial cells and astrocytes are found in the cerebral cortex, and cytonecrosis of astrocytes can occur. Proliferation of endothelial cells and astrocytes occurs. This change is especially prevalent in the first to the third layers of the cerebral cortex. Spongiform change and necrosis are the result of cerebral swelling and are seen in gyri and sulci. Swelling of glial cells can be followed by proliferation. The leptomeninges can be infiltrated with eosinophils. In the pathogenesis of lead poisoning, lesions can develop in the thalamus, hypothalamus, medulla oblongata, other parts of the brain, and spinal cord. Subtle histopathologic alterations can be confused with postmortem autolysis.

There are some cellular biomarkers of lead poisoning in cattle. In chronic lead poisoning, acid-fast intranuclear inclusions may be observed in renal tubular cells and in hepatocytes. Basophilic stippling of red blood cells as a result of the anemia due to lead poisoning is most pronounced when sodium EDTA is used as an anticoagulant, the blood smears are air dried, and they are then stained with Wright-Leishman stain (33,34). Basophilic stippling is not pathognomonic for lead poisoning in cattle since it can also occur in anemia due to other causes (35).

Glycols

Drying agents and antifreezes are toxic to cattle (36). In cattle, diethylene glycol affects the eyes, liver, kidneys and nervous system. Gross pathologic changes after dosing with 1.5 ml diethylene glycol/kg were clouding of the corneas, swollen friable liver with prominent lobular pattern, enlarged friable kidneys, and massive perirenal edema. Histopathologic findings were centrilobular hydropic degeneration of hepatocytes, proteinaceous casts in renal tubules, and marked hydropic swelling of the renal tubular epithelium. The unique lesion observed in nerve tissue was diffuse ganglioneuronal atrophy and perineuronal amphicytic hypertrophy of the Gasserian ganglion.

Ethylene glycol is a commonly used antifreeze and is poisonous to cattle (1,37). Alcohol dehydrogenase metabolizes ethylene glycol to glycoaldehyde and then through intermediates to oxalic acid. The toxicopathy is primarily produced by the ethylene glycol metabolites. Oxalate crystals can be found in the liver and brain and in the renal tubules, where they cause nephrosis. Renal tubular lesions range from hydropic degeneration to cellular necrosis.

Ions

The toxicopathy of ion poisoning in cattle can be variable depending upon the species present (29,38-40). Ingestion of concentrated sodium ions produces marked congestion of the omasal and abomasal mucosa. Intestinal contents are fluid, and hemorrhages may cause dark discolorations. Edema of the skeletal muscles and hydropericardium occurs. Edema of the brain is also seen, and an eosinophilic infiltration of the Virchow-Robin spaces may or may not be observed in cattle. Lesions not unlike polioencephalomalacia can be present.

Potassium ions from drilling fluids can be fatal when ingested by cattle (1). Oral exposure to potassium salts can result in peracute intoxication. Few anatomic pathologic findings occur in cattle dying of this potassium intoxication. Analytic toxicologic results are elevated potassium in ruminal contents, serum and possibly in other body fluids.

Phosphate Esters

The toxicopathy of the nonpesticide phosphate esters has been discussed in another report (41). The primary compounds that produce delayed neuropathy in cattle are chemical mixtures given the trivial name of triaryl phosphates (41-45). Specific gross visible lesions are not observed during necropsy. However, irritation and hemorrhages in the intestines have been associated with triaryl phosphate poisoning (45).

Histopathologic lesions are in the peripheral nerves, spinal cord and brain (42-45). The histopathology varies with the time course of the disease and the chemistry of the tri-

Table 1. A guide for tissue collection in cattle suspected of being intoxicated by oilfield pollution.

Location	Tissue	Quantity	Fixative
Head and neck	Brain	All	Formalin[a]
	Cervical spinal cord	All	Formalin
	Gasserian ganglion-trigeminal nerve	One	Formalin
	Pituitary	All	Formalin
	Thyroid	All	Formalin
	Tongue	RS[b]	Formalin
	Salivary gland (parotid and submandibular)	RS	Formalin
	Tonsil	One	Formalin
	Lymph node (submandibular, retropharyngeal)	One	Formalin
	Eye	One (if representative)	Bouin's
Thorax	Esophagus	RS	Formalin
	Thymus	RS	Formalin
	Trachea	RS	Formalin
	Lungs	RS (each lobe)	Formalin
	Heart	RS (atria, septum, ventricle)	Formalin
	Aorta	RS	Formalin
	Lymph node (bronchial, mediastinal)	RS	Formalin
	Bone marrow (sternum)	RS	Formalin
Abdomen	Rumen	RS	Formalin
	Reticulum	RS	Formalin
	Omasum	RS	Formalin
	Abomasum	RS	Formalin
	Duodenum	RS	Formalin
	Spleen	RS	Formalin
	Pancreas	RS	Formalin
	Jejunum	RS	Formalin
	Ileum	RS	Formalin
	Cecum	RS	Formalin
	Colon	RS	Formalin
	Liver	RS	Formalin
	Lymph node (mesenteric, renal, hepatic)	one each	Formalin
	Gall bladder	RS	Formalin
	Kidney	RS	Formalin
	Adrenals	RS	Formalin
Pelvis	Ovary		Formalin
	Uterus (Fetus)	RS[c]	Formalin
	Urinary bladder	RS	Formalin
	Bone marrow (wing of ilium)	RS	Formalin
Other	Sciatic nerve	RS	Formalin

[a] 10% neutral buffered formalin
[b] Representative sample
[c] The fetus should also be given a detailed pathologic examination

aryl phosphates. Swelling of axons and myelin degeneration are prominent features of the axonopathy.

Swelling of the axonal cylinder (20x), with a transition from basophilic-to-acidophilic staining properties, were observed in the spinal cord of cattle inadvertently or experimentally exposed to triaryl phosphates. Ascending from lumbar to cervical, the lesions changed in pattern and severity. Histopathologic evidence of axonal degeneration was not seen in the L5 vertebral region, but a few swollen axons were observed at L2 and T12. A marked increase in degenerated axons appeared at T5 and at C7. At C3, the lesions were more severe near the tips of the dorsal and ventral horns. Axonal degeneration was not observed in the brain. Bilateral demyelination and degeneration of the ascending tracts in the cervical regions of the spinal cord were also seen.

Carbon Disulfide and Hexacarbon Axonopathy

Other oilfield related compounds that can induce axonopathy are carbon disulfide and hexacarbons (1). It can be assumed that cattle are equally sensitive to hexacarbons and carbon disulfide-induced axonopathy as they are to nonpesticide organophosphate esters.

COLLECTION OF SPECIMENS FOR HISTOPATHOLOGY

During the necropsy of cattle where oilfield pollution is the suspected cause of death, attention to details is absolutely necessary. Reducing the interval between death and necropsy increases the sensitivity of histopathology by limiting postmortem autolysis. All organ systems should be examined during the necropsy procedure. Abnormal tissues and tissues representing major organs, including the brain, should be preserved in appropriate fixative(s). Details of the necropsy must be recorded during and immediately following the process. Tissues can generally be fixed in 10% buffered formalin. Tissues requiring firm fixation (testicle, eye ball) should be fixed in Bouin's solution. A log should be kept of all specimens and samples collected. A guide for specimen and sample collection is given (Tables 1,2). So that there will be a minimum of errors and deletions, consultation with specialists in toxicology and pathology should be done before necropsy procedures commence.

Table 2. Guide for collection of body fluids from cattle suspected of being poisoned with oilfield pollution, and the collection of environmental samples.

Sample Type	Description	Quantity	Preservative
Body fluid	Rumen contents	500 ml[a]	Frozen[b]
	Foreign substances in the reticulum	As much as possible	Frozen
	Gastrointestinal contents	500 ml	Frozen
	Whole blood[c]	30 ml	Refrigeration
	Serum	20 ml	Refrigeration or freezing
	Urine	100 ml	Frozen
	Bile	100 ml	Frozen
	Cerebrospinal fluid	100 ml	Frozen
Tissue	Liver	500 g	Frozen
	Lung	500 g	Frozen
	Kidney	500 g	Frozen
	Brain	One sagittal section	Frozen
	Eyeball	One	Refrigeration
Environmental samples	Sump (sludge pit)	One liter	Refrigeration
	Water(well, dugout, stream)[d]	One liter[a]	Refrigeration
	Contaminated forage	500 g	Frozen
	Spilled substance(s)	One liter[a]	Frozen

[a] Ensure that the container does not contaminate the specimen or sample
[b] -20°C
[c] The anticoagulant will vary with analytical toxicologic testing required
[d] Water samples may require special preservatives

CONCLUSIONS

Pathology is a strategic tool in linking exposures to effects in cattle. The target organ(s) vary with the intoxicating substance. The respiratory tract is affected by volatile hydrocarbons, elemental sulfur, sulfur compounds and other inhalable substances. The gastrointestinal tract may be irritated by petroleum hydrocarbons, heavy metals and alcohols. Liver pathology can be induced by petroleum hydrocarbons, glycols and heavy metals. Renal pathology also occurs after exposure to petroleum hydrocarbons, glycols and heavy metals. Clinicopathologic profiles are used to detect renal and hepatic toxicity and to assess effects on the immune system. Cardiac function may be altered by potassium ions. The central nervous system is affected by sodium and potassium ions, and by diethylene glycol and alcohols. Timeliness and attention to detail are essential in conducting meaningful pathologic examinations of cattle.

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MODIFIER MAYHEM

In a guest piece appearing in the Ontario Globe & Mail, public relations manager Robertson Cochrane reminds us of "The Joy of Dangling Absurdities." He says he enjoys collecting misplaced modifiers and shares these examples taken from various publications:

- "To help reduce the beaver colonies, the Department of Natural Resources has even begun allowing novice trappers to learn to trap in the comfort of their own living rooms."
- A pest control company advertised this: "The electronic stake emits vibrations and sounds that are intensely annoying to rodents up to 10 feet in diameter." (Better not antagonize them!)
- From the National Geographic publications catalogue: "Bird-watching chart/map...poster of tips for attracting birds with migration map on back side." (Does that interfere with flying?)
- A Montreal newspaper said a woman "had moved into an apartment where she was killed a few weeks before her death."
- A Texas television newscaster said, "For the second time in two weeks, a Galena Park woman has been murdered." (Slow learner...)
- Cochrane's favorite, however, is "When stewed, I like prunes."

HOW TO SUCCEED IN BUSINESS

You've heard it before, and you'll hear it again: Start networking. Join a major organization and, if available, a local chapter.

Determine your definition of success. Is it working 70 hours a week and pocketing a six-figure salary? Is it earning $40,000 a year and having enough time to play golf during the week? Is it working part-time and spending afternoons with the kids?

Save money, at least 6 months' worth of expenses. Because of the recession, many independents are squirreling away up to a year's worth of expenses.

If you're up to relocating, choose an area with a large concentration of corporations or associations (Washington, New York, Chicago, major state capitals). The competition might be keen, but there are usually plenty of opportunities.

Build a client base by tapping into colleagues and former employers, and, if worse comes to worse, by cold-calling.

Read year-end roundups in newspapers and business magazines. These articles will help you identify bullish companies and industries that are likely to offer opportunities.

Get help with marketing and promotional materials. Half the battle is letting the world know you exist.