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Swine Toxicoses

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Swine toxicoses, while not involving a large portion of swine losses, are important as problems of differential diagnosis and as economic considerations in individual herds. The problem of poisoning is important also in that swine toxicities are not amenable to treatment. Therefore, diagnosis of the problem, prompt removal from the source, and proper preventive procedures and management are essential.

By far the three most prominent swine toxicology problems received by the Iowa Veterinary Diagnostic Laboratory are Salt Poisoning—Water Deprivation, Organic Arsenical Intoxication and Perirenal Edema as caused by redroot pigweed. A variety of other minor poisonings occur, and some of the more important ones will be discussed.

**ARSANILIC ACID**

The use of arsanilic acid as a feed additive in swine rations has become a common and widely accepted practice. The organic arsenicals are used not only for their therapeutic value in certain enteric and blood parasite diseases, but as low level feed additives as well. Low levels of arsanilic acid in swine reportedly have a growth promoting effect. When used therapeutically in the feed, levels of from 250 ppm to 400 ppm are fed for 4 to 5 days. Then the dosage is reduced to 100 ppm for a longer period of time. The commonly recommended long term feeding level is 100 ppm, and used at that level commonly causes no problems. In most instances, arsanilic acid intoxication arises when (1) therapeutic dosages are continued too long, (2) mistakes are made in the amount added to the ration and (3) when arsenical medicated drinking water is added to lots of hogs already on feeding or therapeutic levels of arsanilic acid.

Evidence indicates that upwards of 450 ppm arsanilic acid fed daily in the ration will produce symptoms of acute poisoning within one week. Pigs fed 10–15 times the feeding level develop toxic signs in 3 to 4 days. Acute intoxication produces signs most directly referable to the nervous system. Early in the course, a slight looseness of the stool is seen, but severe watery diarrhea does not develop. Affected animals may show posterior ataxia and incoordination as early signs. These progress cranially to involve the entire body. The pigs are extremely incoordinated and ataxic, often walking with a jerky, erratic weaving gait. They repeatedly fall on their sides and writhe frantically to regain their footing, only to stagger and fall again. The head may be cocked to one side and remain so for long periods of time. The animals are alert and completely aware of their environment. Appetite is excellent and intake of feed is limited only by
the ability of animals to reach the feeders. By the third or fourth day of clinical signs pigs may be blind. If feeding is continued, they may become laterally recumbent. Prediction of death is extremely variable, and it appears quite difficult to kill pigs quickly on arsanilic acid. If removed from the source soon after signs appear, a majority of affected pigs will recover completely and go to market on schedule. However, after a certain point, the problem appears to be irreversible and animals go on to die or suffer chronic sequela.

Arsanilic acid, fed at lower levels than those acutely toxic, can produce a more subtle chronic change. Poisoned animals may knuckle at the fetlocks, walking sometimes on the carpal joints, and showing weakness or sagging of the hocks. Slight lateral bowing of the hocks may also be observed, giving the legs a weak, almost rubbery appearance. Moderate overreaching or "goose stepping" with the rear legs is another manifestation of chronic intoxication.

At necropsy, no gross lesions are observed. Histologic examination has revealed demyelinization of peripheral nerves, accounting at least in part for the incoordination and ataxia observed.

Confirmation of the diagnosis may be made by chemical analysis of tissues for arsenic, particularly liver and kidney, since arsenic is rapidly excreted via the kidney.

Acute arsanilic acid poisoning has been confused with gut edema, salt poisoning and insecticide poisoning. Close observation of clinical signs and an accurate history are most important in establishing a tentative diagnosis. Chronic effects of arsanilic acid are sometimes diagnosed as arthritis or as pantothenic acid deficiency or mineral imbalance.

**SALT POISONING—WATER DEPRIVATION SYNDROME**

Pigs fed excessive amounts of salt (NaCl) with limited access to water may commonly develop a characteristic clinical syndrome formerly called "salt poisoning." However, recent evidence indicates that the lack of water, not high levels of salt, is perhaps the more important condition. In fact, instances of production of the syndrome in swine having only a 0.5% NaCl level with restricted water intake have been recorded under field conditions.

The pathogenesis of the condition, while still unclear, appears to be related to the affinity of the brain for sodium, thus allowing an osmotic gradient to develop and drawing water from the circulatory system into the brain substance with resultant cerebral edema. Other evidence indicates that the sodium ion inhibits anaerobic glycolysis in the brain, and thus may affect cerebral function. At any rate, fairly characteristic and recognizable clinical signs develop.

Within 24 to 72 hours after restriction of water, clinical signs develop. Affected pigs show thirst, pruritis and constipation. Many pigs appear blind and deaf one to five days after clinical signs develop. They may stand quietly as if in a stupor, or may wander aimlessly about the area, bumping into objects. Some pigs appear to follow fences or walls and when they encounter a corner will stand for long periods with the head pressed into the corner. Occasionally pigs may circle and pivot around one front or hind foot.

A common and very dramatic clinical sign is the occurrence of intermittent epileptiform seizures. These begin with twitching of facial muscles and ears, blinking of the eyes and cervical muscle contractions. These progress to opisthotonus, paddling of front limbs and complete tonic-clonic convulsive seizures. Lateral recumbency with paddling movements and lateral deviation of the head and neck are also seen. During these convulsive periods, pigs appear insensitive to any external stimuli. After a seizure, the animals lapse again into a stupor until the next convolution. In some cases pigs appear almost perfectly normal between seizures.

During convulsions, temperature rises and respiration is accelerated. Pigs may die from one to three days after onset of symptoms.

Clinical-pathologic work indicates that serum and cerebrospinal sodium levels are markedly increased. Some instances of a serum Na level of 175 mEq./L. to 290
mEq./L are recorded. Eosinophils disappear from the blood at onset of signs.

At necropsy, inflammation of the gastrointestinal mucosa and ulcers of the gastric mucous membrane are observed, but some pigs display no gross lesions.

Microscopic changes in the brain and meninges are considered diagnostic. There is meningoencephalitis with edema and infiltration of eosinophils into the meninges and blood vessels of the cerebral cortex. In early cases, eosinophils are few in number and may be found only in the meninges of the sulci. Polioencephalomalacia is seen along with vascular endothelial proliferation and cystic spaces in the affected tissue.

Clinical signs and history are nearly always sufficient evidence to diagnose saltwater intoxication. Confirmation is made on the basis of elevated serum and cerebrospinal fluid sodium, and the finding of characteristic eosinophilic meningoencephalitis in the cerebral cortex.

**IRON TOXICITY**

The occurrence of iron toxicity in swine is confined primarily to baby pigs treated for baby pig anemia. The hazard is particularly great when injectable iron forms are used, since the body has no means of eliminating excess iron given parenterally. Various forms of iron injectables are iron dextran, iron dextrin, iron sorbitol and ferric ammonium citrate. Iron toxicosis has been observed at normally recommended levels of one 100 mg. dosage of the injectable preparations.

The addition of 0.5% iron in the diet of pigs appears to cause precipitation of PO₄ in the tract and resultant rickets and poor growth. Large doses of oral ferrous sulfate may break down the selective iron absorption function of the intestine, allowing increased absorption leading to shock and liver injury.

The pathogenesis of iron toxicity is thought to involve an increase in capillary permeability, followed by reduction in plasma volume and vascular collapse. Excess iron is also toxic to the liver itself.

Drowsiness, vomiting, signs of circulatory shock, coma and rapid death (4–5 hrs.) are observed in pigs on excess iron.

Post mortem reveals a yellowish brown discoloration with edema of tissues, particularly at the site of injection. The liver, kidneys and lymph nodes are extremely dark and periportal necrosis of the liver may occur. Ulceration of the stomach may occur with excessive oral dosing.

An accurate history coupled with clinical signs and characteristic post mortem lesions is usually sufficient to establish a diagnosis of iron toxicity.

**MERCURY**

Mercury may produce poisoning in swine most commonly by its occurrence as a fungicide seed dressing. Such treated grains may gain access to swine rations either by accident or ignorance. Other possible sources include antiseptics and mercurial diuretics, but these are far less common. Swine may develop toxic signs when the mercury level comprises 2% of the feed in animals fed ad lib.

Mercury may first act by producing coagulative necrosis of the gastrointestinal system. Calomel (mercurous chloride) is especially noted for this. However, the primary action in swine is one of capillary degeneration in the kidneys and brain.

Acute mercury poisoning is manifest clinically as severe gastroenteritis with colic, diarrhea, vomition, weakness, prostration and death.

Subacute or chronic poisoning will produce death 5–10 days after onset of symptoms, depending on the amount consumed. Hogs are anorectic, weak and unsteady, walking with a swaying gait. Glossopharyngeal paralysis may cause inability to eat or drink. Partial or complete blindness occurs and swine become prostrate, exhibiting paddling movements. Signs referable to uremia develop and progress rapidly as death nears.

Lesions presented include a kidney which is firm, swollen and congested. Lungs and liver are congested and edema, hyperemia and hemorrhage of the GI tract are seen. Histologic evaluation of kidneys exhibits tubular necrosis with interstitial fibrosis. Neuronal necrosis is observed in the brain.

The diagnosis must tentatively be based
on evidence of availability of mercury correlated with symptoms and lesions. Confirmation may be made chemically by demonstration of mercury in kidney tissue at levels of 15 ppm or greater.

**LEAD**

While lead poisoning in swine is not a commonly diagnosed condition, the widespread availability of lead and its consideration in differential diagnosis make some mention of it appropriate. Paints, insecticide sprays (as lead arsenate) and storage batteries are common sources of lead.

Lead intoxication occurs only as a result of continued ingestion over a period of time. Signs may appear after one to three days, first as anorexia and diarrhea with blood stained tarry feces. Incoordination in the rear quarters, salivation, chewing of jaws, squelching, blindness and convulsions commonly occur. Temperature elevation occurs during and immediately after convulsions. Death rarely occurs prior to the fourth day of clinical signs. Chronic manifestations of lead poisoning are similar to those of acute, except overt signs may be less severe. Twitching of facial muscles, eyelids and ears is seen. Pregnant sows may abort and infertility may be a problem.

Hemorrhagic gastroenteritis is the prominent lesion as well as hyperemia and hemorrhage of the renal cortex.

Diagnosis is based on clinical signs, lesions, evidence of lead ingestion and significant levels of lead in liver, kidney and stomach contents.

**CLAY PIGEON, COAL TAR POISONING**

Poisoning from coal tar products used in clay pigeons, tarring roofing paper and pipe sealing cement is an interesting, although not common, cause of swine losses. These materials do not break down or leach away, so their persistence in an area may be a problem. This is especially true in sites of abandoned trap shooting areas where broken clay pigeons may be on top or turned just under the soil.

The results of clay tar pitch poisoning is often sudden death with little clinical evidence. In less acute cases, weakness, depression, "thumpy" dyspnea and abdominal tenderness are observed. There may be a secondary anemia with accompanying icteric mucous membranes. Most affected animals will go on to die.

Livers of dead pigs are enlarged, friable and characteristically mottled in the lobular pattern with color varying from red to copper. Abdominal lymph nodes are swollen and hemorrhagic and the kidneys are enlarged, turgid and pale. Hydroperitoneum is often observed. Generalized icterus may also complete the picture. The microscopic lesion is one of centrolobular hemorrhagic necrosis of the liver, with hemosiderin giving evidence of ante mortem blood destruction.

**RODENTICIDES**

**ANTU (Alpha-napthyl thiourea)**

ANTU is a white crystalline powder, insoluble in water and heat stable. It is usually prepared in 1–3% sausage or bread baits for use as a rodenticide. Domestic animals are susceptible, but often vomit before the toxin is absorbed, particularly if the stomach is empty.

A single dose of 25–50 mg./Kg. may be fatal.

ANTU affects pulmonary capillaries, causing severe pulmonary edema and marked hydrothorax.

The course of poisoning is rapid, most animals dying within a few hours after ingestion. Symptoms are referable to effects of severe pulmonary edema. Inspiratory and expiratory dyspnea are pronounced. Coughing is evident and moist rales are heard upon auscultation. The pulse is rapid and heart sounds are muffled. Cyanosis develops rapidly, temperature becomes subnormal, and the animals become comatose and die. Frothy edema fluid may actually exude from the nose and mouth of recumbent terminal animals.

Severe pulmonary edema, hydrothorax and a froth filled trachea are the outstanding gross lesions. Kidneys may be hyperemic and the liver is often pale and mottled.

A history of ingestion of ANTU followed by rapid onset of dyspnea, cyanosis and death with lesions of severe hydrothorax and pulmonary edema is suggestive
of ANTU poisoning. Chemical tests for thiourea must be conducted within 24 hours as ANTU is rapidly metabolized. Liver and stomach contents are preferred tissues for analysis.

**Sodium Fluoroacetate:**

Sodium fluoroacetate, also known as 1080, is a white, water soluble chemical. The pig is highly susceptible to fluoroacetate toxicity, as little as 0.3 mg/Kg being fatal. Through its use as a rodenticide, 1080 may be consumed by pigs via ingestion of dead rats poisoned by the substance.

The fluoroacetate ion is absorbed and converted to fluorocitrate. The fluorocitrate in turn inhibits the enzyme aconitase in the tricarboxylic acid cycle, and the animal dies due to inhibition of cellular respiration.

Nausea, vomition and frequent defecation are early signs beginning about 1/2 to 1 hour after ingestion. Central nervous signs are manifested as nervousness, tetanic muscle spasms, and wild running interrupted by periods of relative quiescence. Cardiac depression may occur. Death is rapid, occurring within 30 minutes to several hours.

Cyanosis and dark tarry blood are prominent features. The spleen and liver are swollen and congested and the heart is in diastole with many subepicardial hemorrhages. The stomach is usually empty.

A history of consumption of bait or of animals poisoned on Na fluoroacetate is helpful. Predominant signs of vomition, diarrhea, and wild, aimless running with periods of tetanic convulsions are characteristic of 1080. Chemical analysis of liver is preferred to confirm a tentative diagnosis, but negative results are insignificant.

**Thallium Sulfate:**

Thallium is a colorless, odorless and tasteless chemical incorporated in treated corn, oats or peanuts. A minimum lethal dose is from 10–25 mg/Kg. The action of thallium is unclear; it does appear to produce vascular endothelial damage and accumulates in and affects the hair follicles.

Effects may range from acute to chronic. In acute cases vomition, salivation, diarrhea, abdominal pain, dyspnea, tachycardia, hyperesthesia and convulsions are seen. Recovered animals may become blind and lose most of their hair. Chronic cases display loss of hair, reddening of skin and moist eczema around the mouth and eyes. The tongue and mouth may be hyperemic in acute cases. Ulcerative lesions in the mouth, stomach and intestines are seen in less acute cases. Degeneration of kidney tubules is present.

Observation of clinical signs, either acute or chronic, is essential. A confirmatory urine test may be of value. However, kidney and liver are preferred tissues for chemical analysis.

**Warfarin:**

Warfarin is a widely used rodenticide which is odorless and tasteless. It is closely related to the dicoumarin isolated from moldy sweet clover hay. Warfarin acts by depressing prothrombin production with resulting prolongation of clotting time.

Symptoms of Warfarin poisoning are slow to develop. Frequent ingestion of the toxin is necessary to exhaust the reserve of plasma prothrombin. Twenty-four to 48 hours may lapse before signs appear. Anemia, pale mucous membranes, weakness and lameness are seen. Large bruises or hematocysts may occur as a result of trauma, and bleeding from body openings is not uncommon.

The lesions most prominent are hemorrhage in body cavities, and in many tissues, particularly the lungs. The hematocrit is low and clotting times are increased.

**INSECTICIDES**

**Chlorinated Hydrocarbons:**

The chlorinated hydrocarbons may be discussed as a group since the clinical signs are very similar. These compounds, of which Lindane, Chlordane, Aldrin, Dieldrin, Endrin, and Heptachlor are examples, are fat soluble. Animals such as swine, which carry greater amounts of fat, require more chemical to produce toxicity. The fat absorbs much of the chemical and reduces the amount available to act sy-
temically. The subcutaneous fat of swine offers great protection against dermal applications of insecticide. However, rapid oral or dermal application of chlorinated hydrocarbons will poison swine.

The predominant effect of chlorinated hydrocarbon insecticides is one of central nervous stimulation. Convulsions are commonly intermittent. Characteristic spasms begin with fasciculation of facial muscles, blinking of eyes and twitching of ears. The animal often has an apprehensive expression which may signal the onset of a convolution. Champing of the jaws and drooling of saliva are commonly observed. The spasms of the facial muscles are followed by those of the cervical muscles, forequarters and then hindquarters. The head and neck is often drawn back in opisthotonus as the pig backs up and sits on its rear quarters. Affected animals may fall on their sides and show paddling movements of all the limbs. Hypersensitivity to external stimuli (touch, noise) is characteristic. Commonly the animals recover from a convolution and appear perfectly normal until another occurs. Body temperature during the convulsions may reach 112°F to 116°F. Severity of the convulsions is not of value in prognosis. Some animals die rapidly after only one or two convulsions while others survive many extended and severe seizures. In contrast to the typical signs of hypersensitivity and cranial to caudal convulsions, some swine display depression, anorexia and drowsiness for extended periods of time.

History of exposure or availability to insecticides and close observation of clinical signs are valuable aids in diagnosing chlorinated hydrocarbon poisoning. Liver, kidney, spleen and body fat are the most valuable tissues for chemical analysis. However, animals may be fatally poisoned by one exposure and have no demonstrable tissue levels. Likewise, continued sublethal exposure may build up tremendous tissues levels in the body fat.

Organic Phosphate Insecticides:

As a group, the organic phosphate insecticides have a similar mode of toxicity and manifest closely related clinical signs. This group acts by inhibiting the enzyme, cholinesterase, which normally inactivates acetylcholine. Since acetylcholine is not inhibited, nerve transmission, particularly of the parasympathetic system, is enhanced. Repeated exposure to organophosphates will lower the cholinesterase reserves and render an animal more susceptible to further organic phosphorus treatment.

Affected animals show signs quickly after exposure. Profuse, watery salivation and open mouthed dyspnea are characteristic and myosis is apparent. Stiffness, especially of the hindquarters, is often seen and animals assume a stilted, sawhorse type of walk. Diarrhea and symptoms of colic are not uncommonly seen.

Correlation of clinical signs with reduced blood cholinesterase levels is indicative of organic phosphorus effects. Organic phosphates, with the exception of Ronnel, are rapidly metabolized and demonstration of the compound in animal tissues is unlikely.

TOXIC PLANTS

Redroot Pigweed:

The redroot pigweed, *Amaranthus retroflexus*, is a common inhabitant of pastures and farmyards in the midwest. It grows rapidly in fertile areas, especially those heavily supplied with organic nitrogen, reaching a height of three to five feet by late July or early August. The coarse, erect stem is topped by a large, rough, flowering head.

Almost invariably, a history and course for swine poisoned on pigweed involves a sudden access of the animals to the plant. The disease is seen in pigs ranging from 25 to 150 lb. which have been previously kept in a concrete or drylot environment. Upon being given access to the plant, pigs eat it voraciously, stripping the leaves and often chewing the stem off to ground level. After five to ten days of ingestion of *A. retroflexus*, typical signs of weakness, trembling and incoordination develop. These signs progress rapidly to knuckling of pastern joints, weakness in the hindquarters, sternal recumbency and almost complete flaccid paralysis. When excited,
they try to rise and may walk a few feet with a crouching gait. Some pigs may be unable to rise at all. Affected animals are alert and the appetite remains generally good if feed is placed where they can reach it. No blindness is noted. Temperatures remain normal or subnormal. The abdomen may be distended and ventral abdominal edema is often observed. Affected animals become quiet and comatose and usually die within 48 hours after onset of symptoms. In rare cases, pigs may live 10 to 14 days.

Clinical pathologic examination reveals blood urea nitrogen levels from 90 mg% to 350 mg%. Potassium levels are markedly elevated, ranging from 10.0 mEq/L to 16 mEq/L where 5 to 6 mEq/L is considered normal. Experimentally, EKG changes have substantiated the cardiac effect of hyperkalemia. Urine albumin ranges from 30 to 2240 mg/100 ml, and urine glucose ranges upwards to 300 mg%. Specific gravity of urine is often quite low. Upon necropsy, the consistent and characteristic lesion is retroperitoneal edema of the connective tissue around the kidneys. This edema may be extensive and encompasses the folds of mesorectum, mesometrium, and lateral ligaments of the bladder. In many cases, subcutaneous and intermuscular edema of the abdominal wall is pronounced. In animals exhibiting clinical signs longer than 72 hours, considerable blood may be found in the edematous perirenal edema. Kidneys are pale in color and often display petechial or ecchymotic hemorrhages beneath the capsule. The thoracic and abdominal cavities may contain large amounts of straw colored transudate.

Histologic evaluation of tissues consistently reveals cloudy swelling and necrosis of tubular epithelium. Numerous protein casts are found in the tubules and many tubules are dilated. Interstitial and perivascular hemorrhage and edema are not uncommonly seen. Glomerular tufts appear shrunken and increased cellularity is noted in some cases.

Diagnosis of perirenal edema does not present great difficulty. A history of sudden exposure to *A. retroflexus* after drylot confinement correlated with lesions of perirenal edema as described is usually sufficient. Submission of kidney tissues in 10% formalin along with whole blood and serum for clinical pathologic examination will substantiate the diagnosis.

**Cocklebur:**

The cocklebur (*Xanthium pensylvanicum*) is a rough, coarse herb somewhat less than three feet tall when mature. The stout, erect stem and spreading, red-spotted branches bear a triangular to heart-shaped leaf. The plant is an annual and reproduces only by seed. The seedling stage, while not often recognized, is the most dangerous. The seedling has a slender, straight, whitish-green stem, which is one to three inches tall with two rectangular leaves at the top. The seedlings may be seen sprouting from the burs of the previous season.

Cockleburs occur throughout North America in cornfields, ditches, fence rows and overflowed land along streams. The seedling may heavily infest pastures as a result of being washed in from adjoining cropland.

The plant is easily controlled by intensive cultivation and common broadleaf weed sprays.

The principle danger from cockleburs is their ingestion in the more toxic seedling stage. The larger plant contains less of the toxic glucoside, *Xanthostrumarin*, and is highly impalatable.

Within 8–24 hours after ingestion, pigs develop signs of depression, nausea, weakness, ataxia, and subnormal temperature. Spasms of the cervical muscles, vomition and dyspnea may occur. Death occurs within several hours after onset of symptoms.

Lesions may include hyperemia of the gastrointestinal mucosa and petechia in the myocardium and kidney cortex.

Finally, diagnosis must be based on evidence of the availability and ingestion of the plant correlated with clinical signs.

**Nightshade:**

Nightshade (*Solanum nigrum*) is a spreading multi-branched annual from six inches to two feet tall. The shiny, blackish-green leaves are elliptical in shape.
taper pointed and often contain many holes. Clusters of black, juicy berries are produced, being ½ to ¼ inch in diameter when ripe. Nightshade is found in woods, permanent pastures and fence rows and will grow in areas of less fertile soil.

The leaves and green berries principally contain the alkaloid solanine. The plant is not palatable and is usually consumed under conditions of abundant growth and lack of other suitable forage.

Affected animals display anorexia, constipation, depression and incoordination. Poisoned pigs may vomit. Dilation of the pupils and muscular trembling are signs referable to the nervous system. Animals may be seen lying on their sides and kicking with all feet, progressing then to coma and death.

In acute cases no lesions are found. Swine which live for some time display perirenal infiltration of blood tinged serum and large blood clots may be found adjacent to the kidney.

The toxin is rapidly eliminated by the kidneys and submission of urine from a suspect animal would help to confirm the diagnosis.

Water Hemlock:

Water hemlock (Cicuta maculata) is a tall, branched plant with a heavy purple-streaked stem. The leaves are shaped like an arrowhead with serrated edges. The veins of the leaf end in the notches of the stem and reveal a hollow central cavity crossed by horizontal plates of pith. The plant grows frequently in low wet pastures, in fence rows, and along streams and ponds. The tuberous roots are the most toxic portion of the plant and the habit of swine for rooting may expose them to that part.

Poisoning by the resin-like fraction, cicutoxin, produces signs related to the nervous system. Frothing at the mouth is followed by nervousness, pawing and rooting. Muscle twitching, tremors and convulsions may follow. Vomition is seen and severe abdominal pain is apparent. Dilation of pupils may be observed. Temperature may rise to 106°F during convulsions. The animals die of respiratory failure.

Analysis of stomach contents for presence of cicutoxin may aid in diagnosis. A strong musky odor is characteristic of the ingested material.

Poison Hemlock:

Poison hemlock (Conium maculatum) is a tall, coarse biennial with a smooth, purple, spotted stem. The bruised leaves smell like parsnip and are extremely unpalatable. It is distinguished from water hemlock by the leaf veins which run to the tips of the serrations and the root which is long and not tuberous. Although less abundant than water hemlock, it occupies approximately the same habitat. All parts of the plant are poisonous, the toxic fraction being largely the alkaloid, conine.

Poisoned animals salivate profusely. There is muscle trembling, depressed respiration and rapid, weak pulse. Body temperature rises several degrees, and death results from respiratory failure. No pathological lesions are produced. Submission of stomach contents for chemical determination may be helpful if there is evidence of consumption of the plant.

**MISCELLANEOUS PLANTS**

St. John's Wort:

This is a widespread plant of North America. The toxic principles, hypericin and hypericin red, cause photosensitization and dermatitis of unpigmented skin. The effect is caused if eaten during the flowering stage and if there is subsequent exposure to sunlight. Lesions include dermatitis and blistering of the skin and some gastrointestinal irritation. Body temperature is often elevated to 105°F.

Buttercup:

*Ranunculus spp.*, the buttercup or tall buttercup, produces an acrid volatile agent, anemonal. The plant grows in woodlands and moist pasture areas. Affected animals may live for two to three days. They develop diarrhea and abdominal pain, twitching of the ears, nervousness, dyspnea, tachycardia and paralysis. Upon necropsy a hemorrhagic gastroenteritis is seen and congestion and petechia of the lungs are common.

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Crotalaria:

*Crotalaria spp.* are low growing legumes with an erect hairy stem. They are used as cover crops in southern climates, but may grow in northern areas before frost. The toxin, monocrotaline, is concentrated in the seeds. Tachycardia, polypnea, diarrhea and loss of hair are prominent signs. Anemia, lymphocytopenia and neutrophilia are characteristic. Lesions include ecchymosis of the myocardium, especially the right ventricle, and a soft flaccid heart. Circumscribed renal hemorrhages, hepatic congestion and cirrhosis, and hemorrhagic or necrotic gastritis are seen.

Corn cockle:

Corn cockle (*Agrostemma githago*) is a slender, erect branched plant with very narrow pointed leaves and a brilliant purple flower in May and June. It occurs in fence rows, ditches and waste areas and has been a heavy contaminant in grain fields. The green plant is not very dangerous, but the seeds contain large amounts of a glucoside, githagin, and a saponin, agrostemmic acid.

Pigs are highly susceptible and may be poisoned via grain contaminated with cockle seeds. Vomiting, colic and diarrhea with frothy, foul feces are characteristic symptoms. Spasms may occur prior to death. Finding of the dark brown-to-black wart covered seeds in the gastrointestinal tract correlated with the clinical signs may suggest corn cockle poisoning.

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