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Acorn Poisoning in Cattle

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DURING THE MONTH of October reports of acorn poisoning have been widespread through Iowa and neighboring states. Representative animals from some herds were presented to the clinic. Other herds were described by practitioners in telephone conversations. When the first cases were encountered there was understandable hesitation in making the diagnosis since the problem was occurring on pastures that had been supporting trouble free grazing for years. As evidence continued to mount, however, it appeared that the heavy acorn production, together with abundant rainfall, may have resulted in an unusual problem this year.

Although the American literature reports the occurrence of oak poisoning of cattle and sheep in the Southwest by buds, shoots and new leaves in the spring of the year, 1,2 there appears to be no description of poisoning from the acorn crop in the fall. Survey of the European literature,3,4,5,6 however, reveals reports that outbreaks of the so-called “Acorn Poisoning” occur quite commonly in years of outstanding acorn production and that history, symptoms and lesions show definite correlation with their counterparts in recent outbreaks here.

The cases reported in this area have principally involved beef calves five to eighteen months of age grazing on pastures with heavy stands of oak trees. The onset of symptoms, though rather abrupt, does not occur until sometime after the calves have been on the acorn littered pasture. Apparently the morbidity in a particular herd varies from only one to two animals to nearly the entire calf crop. The mortality rate of severely affected individuals exceeds 75 percent.7 There have been some reports involving adults.

Although pastured calves are not closely inspected, it appears that some affected calves may die suddenly. More often, they linger from several days to a month before terminating in death or recovery.

An initial period of constipation during which brown-black mucous and blood covered feces are passed is followed by a dark fetid or bloody diarrhea. Temperature readings are normal to slightly subnormal. There is some increase in pulse and respiratory rates if the condition persists. Complete anorexia with ruminal atony develops rather rapidly, but the animals still drink, usually in smaller amounts at more frequent intervals. The calves become dehydrated, exhibit nasolacrimal discharge, and have a foul, odorous breath. The mucous membranes become anemic in appearance. There is a frequent discharge of urine of low specific
Edema of the underline is common. Animals have sickened as long as a week after being removed from the troublesome pasture.

Post-mortem examination reveals the rumen to be filled with a foul-smelling decomposing mass in which many acorn hulls can be found. The colon, small intestine, and to a lesser degree, the abomasum are affected by hemorrhagic inflammation. Large volumes of blood may at times be found in the intestines and black tarry feces in the colon. Petechiation may be found in the mucosa of the digestive tract, on the kidneys, in the subcutis, and on most serous surfaces. Degenerative changes are noted in the kidneys and there is a strong ammonia odor in the tissues. Hydroperitonemium, hydrothorax, and hydropericardium are usually encountered, with edema of all loose connective tissue and the peri-renal fat. The larger swellings in the dependent areas of the carcass may appear gelatinous on incision. Edema of the lungs is common.

The toxic substance has not been identified. It has been suggested that tannic acid or its metabolic products gallic acid and pyrogallol could be responsible. No specific treatment has been reported. Mineral oil via stomach tube and glucose preparations intravenously are being commonly used together with an attempt to include high fiber roughage in the diet.

While ingestion of acorns is a common feature in all reported cases, there have been no reports of reproducing the disease under controlled circumstances. Such an effort is now being made at the clinic. Should definite findings result, a further report will be made.

REFERENCES
7. Sm.th, D. E. Personal Communication.