The "Honker Syndrome" in Feedlot Cattle : A Possible Etiology

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Among the many respiratory afflictions found in feedlot cattle is a syndrome which has come to be referred to as the "honker." The affected animal exhibits a dyspnea characterized by a loud, gutteral sound on inspiration which is much deeper in tone than that exhibited by an animal with diptheria.

Animals may be asymptomatic until they are walked for three to four hundred yards, at which time they will exhibit the characteristic sound. Other animals are found dead in the pen from suffocation without anyone observing the dyspnea. The incidence is much higher in summer months, and in animals which have been on feed for at least 60 days. Animals may be found on the slaughter floor with the typical pathological changes even though they appeared normal on antemortem inspection.

The basic pathological changes are found in the submucosa of the trachea. Grossly there is an edematous swelling of the dorsal lining of the trachea which varies in its thickness from 1 cm to 5 cm. In some animals the edematous area contacts the ventral surface of the trachea resulting in suffocation of the animal. The lesions are usually 20 to 30 cm long extending posteriorly from the mid-cervical area to the thoracic inlet and occasionally to the tracheal bifurcation. Some postmortem examinations reveal massive hematomas on the exterior of the trachea and/or hemorrhage within the edematous submucosa.

These lesions have been described as severe focal fibrinous tracheitis.* The inflammatory response is principally edema in the submucosa with fibrin depositions on the mucosal surface. Occasionally organized thrombi are seen in the lumen of the larger arteries of the submucosa. Cases of longer standing are characterized by the presence of fibrous connective tissue with the edema. The hemorrhage within the submucosa is variable.

Hypotheses abound as to the cause of this condition. Some of those which have been suggested are: (1) The final sequela of chronic feedlot cough, possibly associated with polypoid tracheitis. (2) Trauma to this area of the trachea as a result of leaning against a feed trough. (3) Decreased venous flow into the thoracic cavity as a result of fat deposition in the thoracic inlet. Recently, evidence has suggested another etiology: Samples of corn silage and high moisture corn were extracted with ethyl acetate. Extracts given orally to rats and mice produced dyspnea with pronounced râles within 12 to 24 hours and death often followed within 48 hours. There was edema of the laryngeal and tracheal mucosa, focal necrosis, and in some instances a proliferative fibrinous tracheitis that partially occluded the tracheal lumen. Though the toxic material in the extract was suspected to be a mycotoxin, chemical tests (i.e., thin layer chromatography) did not verify it as being one of the known mycotoxins. Attempts to associate the toxic material with mold species isolated from the silage were not successful.

Treatment is usually successful if medication can be administered without the animals dying from suffocation during treatment. If the weather is extremely hot, the treatment should be delayed until the cool portion of the day. Broad spectrum antibiotics and corticosteroids to reduce the inflammation are effective. Any attempted tracheotomy should be accomplished under tranquilization and the tracheal tube should be long enough to reach the tracheal bifuration and thus bypass the edematous submucosa.

**Necropsy examination conducted by Larry T. Jones, State Diagnostic Laboratory, College Station, Texas.*