



# Cattle Producer's Handbook

Animal Health Section

631

## Pulmonary Artery Pressure as a Tool for Managing Brisket Disease

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### Cause and Incidence

Brisket disease is a common name for pulmonary arterial hypertension in cattle that often results in congestive right heart failure. Ranchers also refer to this disease as high mountain disease or dropsy.

Signs of the disease include pulsating jugular veins and subcutaneous edema in the brisket region. To the rancher, the signs and symptoms seen are lethargy, weakness, bulging eyes, enlarged brisket region, difficult breathing, and often death. Standard treatment is movement to lower altitude.

Brisket disease is caused by hypertrophy (enlargement) of the right ventricle of the heart resulting from pulmonary hypertension (elevated pulmonary blood pressure). Although there is shunting of blood in all animals exposed to low oxygen conditions (hypoxia), cattle are more susceptible to pulmonary hypertension because of the anatomy of the bovine lung including its relatively small size.

Hypoxia is the primary cause of brisket disease, but it is aggravated by respiratory disease, parasite load, chronic cold stress, locoweed, asthma, and high levels of nutrition. The reason brisket disease is pronounced at altitudes above 5,000 feet is due to the fact that the oxygen dissociation curve (relating partial pressure of oxygen and saturation by hemoglobin) is not linear but is "S" shaped. This "S" shape translates to little difference in hemoglobin saturations at low altitude (below 5,000 feet). However, at altitudes above 5,000 feet, hemoglobin saturation drops in a precipitous fashion, and the impact of lower oxygen is magnified.

Data suggest that the incidence in Colorado based on National Animal Health Monitoring System (NAHMS) is about 1 percent in native cattle (Salman et al. 1991a). However, movement of cattle from low altitude (or use of semen from low altitude bulls) would be expected to

have higher incidence since the animals are non-adapted. Data from 407,000 feedlot steers in Colorado fed at 5,248 feet where all animals that died were necropsied (1988 animals) showed that 5.8 percent of all deaths were attributed to brisket disease (Jensen et al. 1976).

Brinks et al. (1976) found higher pulmonary artery pressure adversely affected weight and gain of cattle at high altitude. Schimmel et al. (1980) found negative correlations between pulmonary artery pressure and post-weaning gain. At the Colorado State University Beef Improvement Center (BIC) where both home-grown pulmonary artery pressure (PAP) tested sires and artificial insemination (A.I.) sires (not selected for PAP) were used, the incidence of PAP above 50 millimeters mercury (mm Hg) in calves was 3 percent vs. 20 percent for selected and non-selected sires, respectively.

Salman et al. (1991b) found that brisket disease related costs (including both veterinary costs and death loss) ranged from \$1.00 to \$18.93 per head in a Colorado survey. Brinks and LeValley (1978) speculated that economic loss from brisket disease might exceed the total for all other contagious diseases in cattle populations at high altitude in the intermountain region. The incidence of brisket disease is confounded by the fact that it is aggravated by and may be confused with other respiratory diseases suggesting that often brisket disease is misdiagnosed and simply counted as a part of the respiratory disease complex. This may result in low estimates for brisket disease.

Pulmonary artery hypertension resulting in brisket disease is genetically inherited (Will et al. 1975; LeValley 1978). This genetic relationship helps explain the belief that some breeds are more susceptible to brisket disease than others. Heritability estimates for pulmonary artery pressure have been reported by LeValley et al. (1978) using Hereford, Angus, and Red Angus, and more recently by Enns et al. (1992) using Angus cattle, with