



Bovine pulmonary hypertension: 100 years of heartache

By [Joe Neary, MA, MS, Colorado State University](#)

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COMMENTS



In response to requests and monetary donations from producers, George Glover and Isaac Newsom of the Colorado Agricultural College, now Colorado State University (CSU), set out to investigate a strange new disease of cattle first reported in South Park,



Bovine pulmonary hypertension, otherwise known as high-altitude disease or brisket disease, can occur at lower elevations.

Colo. The year was 1913 and the problem was right-sided congestive heart failure induced by bovine pulmonary hypertension (BPH). Within a few years Glover and Newsom determined the disease accounted for 1

to 2 percent mortality of all cattle over 8,000 ft.

The disease became known as brisket disease or high-altitude disease because it affected cattle at high altitudes and often led to subcutaneous fluid accumulation in the brisket region, a consequence of congestive heart failure. They concluded that some cattle do not have sufficient cardiac reserve to meet the demands of high altitude, leading to “exhaustion of the heart.”

They found that affected animals could be completely cured within as little as two weeks if they were taken down to 7,000 ft. in order to “strengthen the heart or lessen its work.” Up until the 1960s the disease was considered to be only problematic at altitudes over 8,000 ft. in *Bos taurus* cattle. The average herd incidence was approximately 2 percent but reached as high as 10 percent in some herds.

In the early 1960s, the focus of research switched from the heart to the pulmonary arteries. Arteriographic studies conducted by Archibald Alexander and Rue Jensen showed that “pruning” of the distal pulmonary arteries occurred in association with the disease and so an “emphasis was placed upon the small pulmonary arteries in subsequent histologic investigations.” In the 1970s, further research revealed that the wall thickness of the small pulmonary arteries was positively

correlated with mean pulmonary arterial pressure (mPAP).

It is now commonly believed that alveolar hypoxia due to low atmospheric oxygen tension at high altitude causes the pulmonary arteries to contract. This vasopressor response is greatest in cattle and pigs and absent in species such as llamas that have adapted to living in mountainous environments.

Vasoconstriction increases resistance to blood flow and so increases mPAP. If the hypoxic stimulus is removed after brief exposure the pulmonary arteries relax and mPAP returns to its pre-stimulus value. However, in response to chronic alveolar hypoxia (more than one week, depending on the strength of the hypoxic stimulus) the smooth muscle cells within the walls of the pulmonary arteries hypertrophy and proliferate. The resultant pulmonary artery wall thickening causes a narrowing of the vessel lumens and so increases resistance to blood flow, leading to pulmonary hypertension. Myocardial hypertrophy of the right ventricle is a normal adaptive response to chronic pulmonary hypertension. In order to compensate for the progressive contractile dysfunction, the chamber dilates in order to increase preload and maintain a normal stroke volume. Unless the underlying cause of the chronic pulmonary hypertension is removed, right ventricular contractile dysfunction continues until death from right-sided heart failure. Although useful, this pathway is overly simplistic and does not adequately explain the emerging epidemiology of this disease.

Management tools

In the late 1970s, a test was developed to measure mPAP in the field. Using this test, cattle with high mPAP (greater than 45 mmHg) can be identified and culled from breeding herds. Since mPAP has been estimated to be a moderately to highly heritable trait, the selective breeding of only low-mPAP bulls in a herd should reduce the occurrence of brisket disease within the calf crop. This selection method is still the only tool available to producers and has been very successful. However, it should be pointed out that what is considered to be a “low” mPAP for cattle would represent pulmonary hypertension in most other mammalian species.



In humans, pulmonary hypertension is defined as a mPAP over 25 mmHg at rest or over 30 mmHg during exercise. Out of 119 Angus feedlot steers, approximately 16 months old and sired by low-mPAP bulls, the lowest mPAP recorded at 4,250 ft. was 39 mmHg. The mean mPAP was 48 mmHg. Therefore, use of the term low mPAP should be used with this frame of reference in mind. A low mPAP today was considered a high mPAP in the 1960s. Yearling steers pressure tested at CSU at 5,000 ft. in the 1960s had mPAP values in the 20s. Such low mPAP values would not be found today.

A persistent problem

Despite the merits of breeding only low-mPAP bulls, our findings show that pulmonary hypertension can still be found in high-altitude herds that have bred only lowmPAP bulls for over 20 years. A field investigation conducted on one ranch in southern Colorado that has selected low-mPAP bulls for over 20 years revealed that approximately 5 percent of calves died from BPH between branding in the spring and weaning in the fall. The bigger and more profitable the calf, the greater the risk of BPH seems to be. Glover and Newsom determined that the disease was hard to recognize in calves; the same is true today.

Calves with pulmonary hypertension can show very mild signs of disease. Swelling of the brisket does not always occur, making correct diagnosis of this disease difficult. Many calves with pulmonary hypertension show signs consistent with pneumonia such as a rapid breathing rate, cough and open-mouth breathing. Rectal temperatures can be elevated both in calves with pulmonary hypertension and in calves with pneumonia, since the muscular effort involved in rapid breathing raises their temperature. Calves showing signs of “summer pneumonia” may be suffering from pulmonary hypertension, pneumonia or both. This presents

an immense and frustrating challenge to both producers and veterinarians.

Part 2 of this series, titled “High-altitude disease or high-production disease?” will describe how pulmonary hypertension has become more prevalent in cattle at lower altitudes and explore possible reasons behind the trend. The article will appear in the January issue of *Bovine Veterinarian*.

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