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Mean pulmonary arterial pressures in Angus steers increase from cow–calf to feedlot–finishing phases[1]

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ABSTRACT: Pulmonary arterial hypertension due to hypoxia-induced pulmonary vascular remodeling is the predominant cause of right-sided congestive heart failure (CHF) in cattle. Historically, heart failure was problematic only at altitudes over 2,134 m. However, anecdotal reports suggest that the incidence of heart failure is increasing in feedlot cattle at moderate altitude (800 to 1,600 m), with late-fed, or fat, cattle at greatest risk. The goal of this study was to evaluate pulmonary arterial pressures (PAP) in a cohort of male Angus calves from suckling to finishing to better understand why heart failure is particularly problematic in fat cattle. It was hypothesized that mean PAP would increase through the feeding period and that the calves with the greatest pressures at high altitude would have the greatest pressures as fat cattle. A total of 362 PAP measurements were obtained from 153 calves. Calves were tested at altitudes of 2,170 (4 and 6 mo old), 1,560 (13 mo old), and 1,300 m (13 and 18 mo old). Mean PAP were greater in 18-mo-old steers than any other age group (mean = 50.3 mm Hg; 95% confidence interval 48.2 to 52.4; $P < 0.05$). Calves that had the greatest mean pressure at 6 mo of age tended to have the greatest mean pressures at 18 mo of age ($r = 0.45$, $P < 0.001$). The increase in mean PAP with increasing age and adiposity likely predisposed the steers to an increased risk of CHF during the finishing phase.

Key words: age, body fat, congestive heart failure, pulmonary arterial pressure

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INTRODUCTION

Historically, congestive heart failure (**CHF**) has been problematic in cattle at altitudes over 2,134 m (Glover and Newsom, 1915; Hecht et al., 1962). However, this may no longer be true. The second leading cause of death, after respiratory disease, in Holstein dairy heifers at the moderate altitude of 1,600 m was CHF (Malherbe et al., 2012). Furthermore, CHF is becoming increasingly problematic in the feedlot industry with the majority of deaths occurring in cattle close to finishing, or "fat cattle" (J.M. Neary, unpublished data). The timing of CHF occurrence suggests that mean pulmonary arterial pressure (**mPAP**) is high throughout the feeding period but greatest in fat cattle close to finishing. Cattle with a mPAP over 49 mm Hg are considered to be at risk of CHF (Holt and Callan, 2007).

The goal of this study was to better understand why CHF is potentially problematic in the late feeding period by characterizing the changes in pulmonary arterial pressure (**PAP**) from calfhood into the confined feeding period. This information is most relevant to the U.S. beef feeding industry because the majority of operations are located at moderate altitudes (800 to 1,600 m) within the High Plains (USDA, 1997). It

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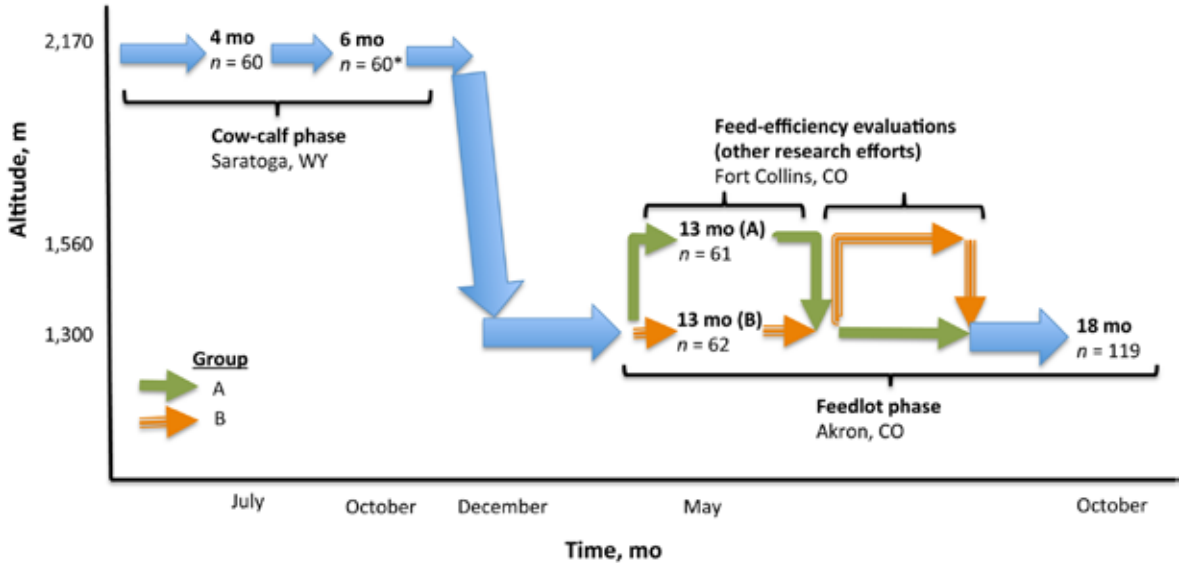

Figure 1. Flow diagram of the pulmonary arterial pressure testing (mean, systolic, and diastolic pressures) performed according to age, location, altitude, time of year, and the number of cattle tested on each occasion. At 6 mo of age, mean, systolic, and diastolic pressures were collected from the same 60 calves tested at 4 mo of age and mean pulmonary pressures were obtained from an additional 93 calves. In total, 31 steers were tested on 4 occasions: 18 steers were tested at 4, 6, 13 (Group A [A]), and 18 mo of age, and 13 steers were tested at 4, 6, 13 (Group B [B]), and 18 mo of age. *$P < 0.05$

was hypothesized that mPAP would increase during the feeding period and that the calves with the greatest mPAP at high altitude would have the greatest mPAP as fat cattle. The rationale was that factors such as increasing adiposity through the feeding period and ruminal engorgement following feeding could cause alveolar hypoventilation and thereby predispose cattle to CHF. If so, calves most susceptible to hypoxia-induced pulmonary hypertension at high altitude may also be the most susceptible to pulmonary hypertension as fat cattle.

MATERIALS AND METHODS

Study Overview

A cohort of male calves was evaluated from 4 to 18 mo of age. Their movements through the production system were not part of our experimental design but were the protocols of a single experimental research station (Fig. 1). Pulmonary arterial pressures were measured up to 4 times: twice before weaning at an altitude of 2,170 m (4 and 6 mo old) and twice after weaning at altitudes of 1,560 (13 mo) and 1,300 m (13 and 18 mo old). Right atrial pressure and pulmonary arteriolar wedge pressure were also measured in the late feeding period (18 mo old). This study received approval from the Colorado State University Animal Care and Use Committee before the testing or handling of any animals (protocol 12-3513A).

Study Sites and Cattle

The study population consisted of male Angus calves from the Beef Improvement Center of Colorado State University (Saratoga, WY). aThe calves were born between March and April at an altitude of 2,170 m and weaned in mid October in 2012. Approximately 50% of the calves were progeny of bulls with a yearling mPAP record < 40 mm Hg at an altitude of 2,170 m. The other 50% of the calves were progeny of industry relevant AI sires of unknown mPAP. Heifers with low mPAP (<40 mm Hg) were retained as herd replacements each year.

A cohort of sixty 4-mo-old male calves was randomly selected by testing every second calf that entered into the chute in July of 2012. The same cohort of calves was tested again at 6 mo of age in October of 2012 (Table 1; Fig. 1). That same day, all male calves within the herd had mPAP measured for genetic selection purposes of this experiment station. Therefore, in addition to the original 60 calves retested at 6 mo of age, mPAP measures were obtained from an additional 93 steers providing a total of 153 mPAP values from 6-mo-old male calves.

Thirty bull calves tested at 6 mo of age were sold before 13 mo of age and unsold bull calves were castrated. In mid December of 2012, all steers ($n = 123$) were transported to the Eastern Colorado Research Center (**ECRC**) of Colorado State University (Akron, CO). Cattle from cow–calf operations located in the Rocky Mountains are typically placed in feeding operations in the High Plains. The ECRC is a confined feeding operation located at an altitude of 1,300 m. The steers were fed silage, corn stalks, and dried distiller's grains until the summer months when they were turned out onto pasture. Cattle grazed native pasture until transported to the Agricultural Research, Development and Education Center (**ARDEC**) of Colorado State University (Fort Collins, CO) for feed efficiency evaluations (Fig. 1). At

Table 1. The date, altitude, age (±SD), and the number of male Angus calves tested according to age (mo)[1]

Age, mo	Altitude, m	Date	Number tested			Mean age ± SD, d	Body mass ± SD, kg
			Bulls	Steers	Total		
4	2,170	July 31, 2012	55	5	60	124 ± 18	NA[2]
6	2,170	Oct. 1, 2012	55	5	60	185 ± 19	NA
13 (A)[3]	1,560	May 2, 2013	0	61	61	398 ± 16	415 ± 31
13 (B)[3]	1,300	May 15, 2013	0	62	62	405 ± 19	384 ± 37
18	1,300	Oct. 8, 2013	0	119	119	554 ± 17	626 ± 62

[1]Mean pulmonary arterial pressures, but not systolic or diastolic arterial pressures, were recorded from additional 93 steers for genetic selection purposes at 6 mo of age. Therefore, 153 mean pulmonary arterial pressure values and 60 systolic and diastolic pulmonary arterial pressure values were obtained at 6 mo of age.

[2]NA = Not Applicable (Body mass not measured).

[3]A = 13 mo steers tested in Fort Collins, CO; B = 13 mo steers tested in Akron, CO.

the ARDEC, cattle were fed corn silage, whole corn, grass hay, and alfalfa. The ARDEC and the ECRC facilities were 206 km apart by road.

In April of 2013, half of the steers (n = 61) were transported to the ARDEC. The steers were PAP tested 10 d after arriving at the ARDEC (Group A; May 2, 2013); they returned to the ECRC in July of 2013. The remaining steers that were not PAP tested at the ARDEC (n = 62) were PAP tested 2 wk later at the ECRC (Group B; May 15, 2013). These steers had remained on grass pasture before testing. They were subsequently transported to the ARDEC in May of 2013 and returned to the ECRC in August of 2013. Each group remained at the ARDEC, located at an altitude of 1,560 m, for a period of 80 to 85 d. Steers were PAP tested in either Group A or Group B—not both. Group A steers were approximately 30 kg heavier than Group B steers even though they were approximately the same age at the time of testing (Table 1). Half of the steers in both groups were implanted with long-acting growth promotants containing estradiol and trenbolone acetate (Revalor-XS; Merck Animal Health, Madison, NJ) at the time of testing for a parallel and concurrent study.

When cattle returned from the ARDEC, they were transitioned onto a diet of dried rolled corn, distiller's grains, and hay, which was fed until finishing. The final test was performed at the ECRC between 2 and 10 wk before slaughter (18 mo old; October 8, 2013). The steers had been fed a finishing ration for either 117 (Group B) or 169 d (Group A) before the final test. Between 13 and 18 mo of age, 1 steer died from pneumonia and 1 steer was sold due to poor growth. Two healthy steers avoided testing at the final test. Therefore, 119 fat steers were tested in total. Of these, 31 had been tested on 4 occasions: at 4, 6, 13, and 18 mo of age. Eighteen of the 31 steers were in Group A and the remaining 13 steers were in Group B.

The dams of calves studied were given a prebreeding and precalving vaccination offering protection against viral agents of respiratory disease. Calves were vaccinated against the same respiratory pathogens at 4 to 8 wk of age and 2 to 4 wk before weaning. A record was kept of calf health and any treatments administered. On arrival at the ECRC, all cattle received a topical avermectin anthelmintic and vaccines against bacterial and viral agents of respiratory disease and clostridial diseases.

Pulmonary Arterial Pressure Testing

Systolic PAP (**sPAP**), diastolic PAP (**dPAP**), and mPAP were obtained from all cattle throughout the study except for 93 of the calves tested at 6 mo of age that were not part of the cohort previously tested at 4 mo of age. Only mPAP measurements were obtained from these 93 calves (Table 1). A full description of the equipment, materials, and facilities required for PAP testing is provided by Holt and Callan (2007). In brief, a 12-gauge, 8.9-cm needle was inserted into the jugular vein. Flexible, saline-filled, polyethylene catheter tubing was then fed through the needle, down through the right atrium, into the right ventricle, and then into the pulmonary artery. A pressure transducer connected the catheter to an oscilloscope (BM5Vet; Bionet America, Inc., Tustin, CA). The position of the catheter tip within the vascular system was determined from the pressure waveform on the oscilloscope. The jugular vein, right atrium, right ventricle, and pulmonary artery have distinct pressure waveforms (Holt and Callan, 2007).

Right atrial and pulmonary arteriolar wedge pressure measurements were obtained from a random subsample of steers during the late feeding period (18 mo old). The measurements were not collected from all cattle due to time restraints. Right atrial pressure, also known as central venous pressure, is an indirect measure of right ventricular function. A pulmonary arteriolar wedge pressure is an indirect measure of left ventricular end-diastolic pressure. Pulmonary arteriolar wedge pressures were obtained by advancing the catheter until the tip became lodged in an arteriolar vessel of sufficiently small diameter to occlude forward flow of blood. The pulmonary arteriolar wedge pressure has a waveform characteristic of a venous pressure trace with "a" and "v" waves produced by left-sided physiologic

Table 2. Adjusted cell means and 95% confidence intervals for mean pulmonary arterial pressures (mPAP), systolic pulmonary arterial pressures (sPAP), diastolic pulmonary arterial pressures (dPAP), and pulse pressure in clinically healthy nonimplanted, Angus steers according to age (mo)

Pulmonary pressure, mm Hg	Age,[1] mo				
	4	6	13 (A)	13 (B)	18
mPAP	38.6[a] (32.5 to 44.6)	41.7[a] (35.7 to 47.8)	40.9[a] (38.8 to 42.9)	43.3[a] (41.2 to 45.3)	50.3[b] (48.2 to 52.4)
sPAP	53.8[a] (45.3 to 62.3)	59.6[b] (51.1 to 68.1)	66.3[b] (63.5 to 69.2)	69.5[b] (66.6 to 72.3)	81.5[c] (78.6 to 84.4)
dPAP	23.6 (17.3 to 29.9)	24.4 (18.1 to 30.7)	19.4 (17.3 to 21.5)	16.0 (14.0 to 18.1)	19.8 (17.6 to 18.1)
Pulse pressure	30.9[a] (22.4 to 39.4)	35.9[a] (27.5 to 44.5)	47.1[b] (44.2 to 50.1)	53.4[c] (50.5 to 56.3)	61.8[d] (58.8 to 64.8)

[a,b]Within a row, values without a common superscript differ ($P < 0.05$). Within row, Type I error = 0.05.

[1]A = 13 mo steers tested in Fort Collins, CO; B = 13 mo steers tested in Akron, CO.

events. Arteriolar wedging of the catheter was verified by aspirating blood through the catheter to confirm that the blood-gas tensions were consistent with oxygenated blood from the venous side of the pulmonary circulation. Mean, systolic, and diastolic pressures were recorded after the pulmonary pressure waveforms stabilized.

Empty Body Fat Percentage

Cattle were slaughtered at a facility in Fort Morgan, CO, between October 25 and December 23, 2013. Empty body fat percentages were estimated from packing-plant carcass measurements using the following equation: empty body fat (%) = 17.76107 + (4.68142 × FT) + (0.01945 × HCW) + (0.81855 × QG) – (0.06754 × LMA), in which FT is 12th rib fat thickness in centimeters, HCW is expressed in kilograms, QG = quality grade (4 = Select, 5 = Choice–, 6 = Choice, 7 = Choice+, and 8 = Prime), and LMA = LM area (in cm^2). This equation was reported to explain 61% of the variation in the observed empty body fat percentage in a study of 1,352 feedlot steers and heifers (Guiroy et al., 2001).

Statistical Analyses

Statistical analyses were performed using STATA version 12 (Stata Corporation, College Station, TX). Statistics are provided as mean and 95% confidence interval (**CI**) of the mean unless otherwise indicated. Summary statistics are provided for empty body fat percentage, USDA quality grade, and age at slaughter.

Changes in PAP among tests were evaluated with generalized estimating regression equations while controlling for castration status (steer or bull) and growth implant status (implanted or nonimplanted). This regression method is robust to missing observations and accounts for lack of independence associated with repeated measures (Liang and Zeger, 1986; Zeger and Liang, 1986). An exchangeable correlation structure, which assumes the same correlation between all pairs of observations, was used. Mean PAP, sPAP, and dPAP, and pulmonary arterial pulse pressure were the outcome measures of interest. The adjusted means for each test were obtained for nonimplanted steers and statistical differences among group means evaluated.

Between-test pairwise correlation analyses were performed for each of the 4 PAP variables. A Bonferroni correction of probability values was applied so that the family-wise Type I error for all pairwise analyses within each of the 4 PAP variables was $P < 0.05$.

RESULTS

Overall Changes in Pulmonary Arterial Pressures

Mean PAP, sPAP, and pulse pressures were greater in steers at 18 mo of age than any other age ($P < 0.05$; Table 2). The mPAP of calves at an altitude of 2,170 m (4 and 6 mo old) were similar to those of 13-mo-old steers at altitudes of 1,560 (Group A) and 1,300 m (Group B; Fig. 2; Table 2). In contrast, sPAP and pulmonary arterial pulse pressure increased more uniformly with age (Fig. 2). Diastolic PAP did not vary with age (Table 2). The bulls sold before 13 mo of age had a mPAP at 6 mo of age of 45 mm Hg (95% CI 41 to 49 mm Hg).

Pairwise Correlation of Pulmonary Arterial Pressures

Pairwise correlation coefficients for mPAP were significant between ages 4 and 6 mo, 6 and 18 mo, and 13 (Group A) and 18 mo ($P < 0.05$; Table 3). Mean PAP at 6 mo tended to be correlated with mPAP at 13 mo of age (Group A; $P = 0.07$). In other words, calves with the greatest mPAP at 6 mo (2,170 m) and steers with the greatest mPAP in the early feeding period (Group A; 1,560 m) had the greatest mPAP as 18-mo-old fat cattle.

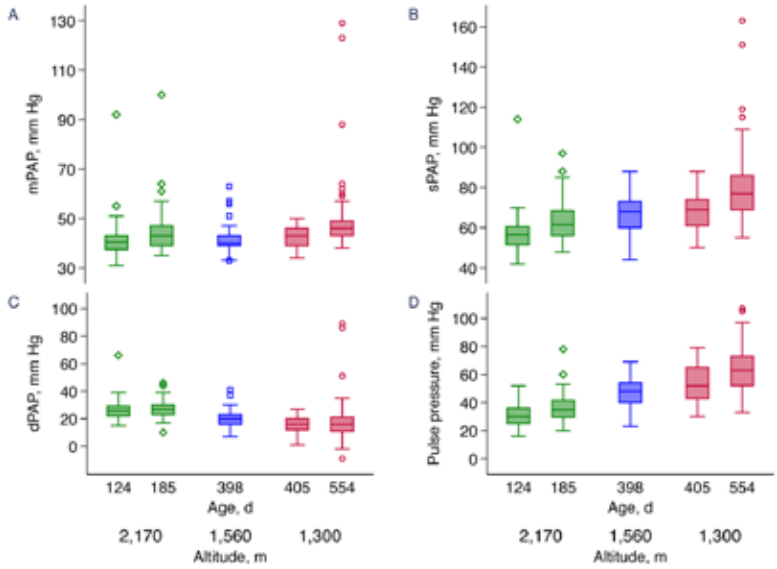

Figure 2. Box and whisker plot of A) mean pulmonary arterial pressures (mPAP), B) systolic pulmonary arterial pressures (sPAP), C) diastolic pulmonary arterial pressures (dPAP), and D) pulmonary arterial pulse pressures obtained from healthy male Angus calves according to age (d) and altitude (m). Measurements taken at altitudes of 2,170, 1,560, and 1,300 m are colored green, blue, and red, respectively. Hollow symbols represent outliers (observations that are greater than 1.5 times the interquartile range from the nearest quartile).

A single outlier with a mPAP of 123 mm Hg at 18 mo of age reduced the pairwise correlation in mPAP between 6 and 18 mo of age from 0.67 ($P < 0.001$) to 0.45 ($P < 0.001$). There were no significant pairwise correlations in mPAP, sPAP, or dPAP between 13 (Group B; 1,300 m) and 18 mo of age even though the tests were performed at the same altitude and the same facility. These results suggest that there were factors responsible for the significant pairwise correlation of mPAP in steers between 13 and 18 mo in Group A (13 mo; 1,560 m) but not Group B (13 mo; 1,300 m). Such factors may include greater hypobaric hypoxia (Group A), dietary differences (finishing ration versus pasture), a heavier body mass (+30 kg mean difference), or a combination of these factors with unexplained sources of variation experienced by cattle that moved from a cow–calf operation to a feedlot.

Steers that had the greatest mPAP at 6 and 13 mo (Group A) of age showed an increase in mPAP through the feeding period that was out of proportion to the increase in mPAP observed for the remaining steers. For illustrative purposes, the PAP of nonimplanted steers tested on 4 occasions at 4, 6, 13 (Group A), and 18 mo of age ($n = 8$) are shown in Fig. 3. Two of the 8 steers had pulmonary pressures that were distinctly greater than the other 6 steers at 18 mo of age (Fig. 3). The same 2 calves had greater than average mPAP at 6 and 13 mo of age (Fig. 3). The pattern in this illustrative subsample reflects the significant pairwise correlations in mPAP between 6 and 18 mo of age and between 13 (Group A) and 18 mo of age in the study population (Table 3). Pairwise correlation coefficients for dPAP were significant between 4 and 6 mo ($r = 0.40$, $n = 60$, $P = 0.009$) and between 13 (Group A) and 18 mo of age ($r = 0.64$, $n = 29$, $P < 0.001$). There were no significant pairwise correlations among tests for sPAP or pulse pressure.

Right Atrial and Pulmonary Arteriolar Wedge Pressures

The right atrial pressures and pulmonary arteriolar wedge pressures in fat cattle (18 mo old) were indicative of impaired right ventricular function and pulmonary venous hypertension, respectively. Mean, systolic, and diastolic right atrial pressures ($n = 53$) were 26.5 (95% CI 24.7 to 28.4 mm Hg), 80.8 (95% CI 76.4 to 85.3 mm Hg), and 3.0 (95% CI –1.5 to 7.5 mm Hg), respectively. Mean, systolic, and diastolic pulmonary arteriolar wedge pressures ($n = 36$) were 25.9 (95% CI 24.6 to 27.2 mm Hg), 51.7 (95% CI 48.1 to 55.2 mm Hg), and 4.0 mm Hg (95% CI 2.0 to 6.1 mm Hg), respectively.

Empty Body Fat Percentage

Mean empty body fat at slaughter was 31.6% (95% CI 31.3 to 32.0) and ranged from 27.2 to 36.1%. The majority of steers had a USDA quality grade of Choice (83%) followed by Prime (10%) and Select (7%). The mean age (±SD) of steers at slaughter was 591 ± 21 d.

DISCUSSION

The results of our study show that bovine pulmonary hypertension is not unique to high altitude. Mean PAP were greater in 18-mo-old feedlot steers at the moderate altitude of 1,300 m than they had been as suckling calves at an altitude of 2,170 m. Furthermore, the average mPAP of 50 mm Hg suggested that the

Table 3. Pairwise correlation coefficients for mean pulmonary arterial pressure in clinically healthy male Angus calves[1,2]

Age,[3] mo	Age, mo				
	4	6	13 (A)	13 (B)	18
4	–				
6	0.87** *n* = 60	–			
13 (A)	0.22 *n* = 18	0.34* *n* = 60	–		
13 (B)	–0.47 *n* = 9	0.01 *n* = 30	*n* = 0	–	
18	0.29 *n* = 17	0.45** *n* = 56	0.84** *n* = 29	0.32 *n* = 29	–

*Correlations with *P*-value between 0.05 and 0.10.

**Correlations with *P*-value < 0.05.

[1]Family-wise type I error rate for table = 0.05. Correlations followed by no superscript indicate *P* > 0.10.

[2]Tests at 4 and 6 mo of age were performed at an altitude of 2,170 m. At 13 mo of age, tests were performed at altitudes of 1,560 (A) and 1,300 m (B). At 18 mo of age, cattle were located at an altitude of 1,300 m.

[3]AA = 13 mo steers tested in Fort Collins, CO; B = 13 mo steers tested in Akron, CO.

majority of the fat steers were at risk of CHF (Holt and Callan, 2007). We speculate that body fat accumulation through the feeding period is the most plausible explanation for the PAP measured. In humans, multiple mechanisms have been proposed linking obesity to both pulmonary hypertension (Friedman and Andrus, 2012) and cardiac dysfunction (Alpert et al., 2014).

If the mPAP of steers in our study were attributable only to hypobaric hypoxia, then mPAP should have decreased following relocation to lower altitude. However, in our study, mPAP was greater in fat steers at an altitude of 1,300 m than it had been when the steers were 4 and 6 mo old at 2,170 m. This implies that alveolar hypoxia continued into the feeding period. Potential causes of alveolar hypoxia include hypobaric hypoxia, obstructive pulmonary disease, and hypoventilation. Given that the cattle studied were clinically healthy throughout the study, the most likely explanation is hypoventilation, operating in concert with moderate hypobaric hypoxia.

Hypoventilation has been previously proposed as a risk factor for CHF in feedlot cattle (Jensen et al., 1976), the rationale being that ruminal engorgement following feeding compresses the lungs, which leads to rapid shallow breathing, alveolar hypoventilation, and hypoxia-induced pulmonary hypertension (Jensen et al., 1976).

Another possible cause of hypoventilation in feedlot cattle is body fat accumulation. The steers in our study had estimated empty body fat levels ranging from 27 to 36% at slaughter. Cattle with a USDA quality grade of mid Choice or above have an empty body fat of 30% or more (Guiroy et al., 2001). This equates to a live animal total body fat of 27% or more (assuming that empty body weight is 90% of live BW; Owens et al., 1995). To put this value in perspective, nonlactating, nonpregnant mature Angus cows consuming a maintenance diet were reported to have an empty body fat of 17% (Williams and Jenkins, 1997). Healthy Holstein cattle early and late in their fourth lactation were reported to have an empty body fat of 11 and 17%, respectively (Andrew et al., 1994). Humans are diagnosed as obese when body fat exceeds 32% in women and 25% in men. Increased work of breathing, particularly when lying down, is one cause of alveolar hypoventilation in obese humans (Piper and Grunstein, 2011). The same may be true for fat cattle.

Several other major findings of this study could also be explained by body fat accumulation through the feeding period. These include the positive associations of sPAP and pulmonary arterial pulse pressure with age and the high pulmonary arteriolar wedge pressures in fat steers at 18 mo of age. In humans, sPAP has been positively associated with body mass index (McQuillan et al., 2001). This may be due to an obesity-associated increase in cardiac output or hypoventilation (Blankfield, 2002). The increase in cardiac output that occurs in association with obesity has been attributed to an increase in cardiac stroke volume, not heart rate (Alexander, 1964; de Divitiis et al., 1981). Therefore, although much of the age-related increase in pulse pressure observed in our study was likely attributable to normal, age-related increase in cardiac chamber size and stroke volume, it is feasible that body fat accumulation was a contributing factor. An alternative, or additional, explanation of the age-related increase in pulse pressure is pulmonary arterial stiffening, which has been previously reported in neonatal calves following hypoxia-induced pulmonary hypertension (Lammers et al., 2008). Stiffening of the normally elastic pulmonary artery could increase the risk of arterial rupture.

Body fat accumulation leading to left ventricular dysfunction may also explain the elevated pulmonary arteriolar wedge pressures observed in fat steers. In humans, obesity increases the likelihood of left ventricular dysfunction and pulmonary venous hypertension (de Divitiis et al., 1981; Galiè et al., 2009). Group 2 pulmonary hypertension, or pulmonary hypertension due to left heart disease, is diagnosed when mPAP is over 25 mm Hg at rest and pulmonary arteriolar wedge pressure is over 15 mm Hg (Galiè et al., 2009). The fat steers in our study had mPAP and wedge pressures that were approximately twice as great as these human diagnostic criteria and twice as great as the same measures obtained from healthy yearling Hereford steers at an altitude of 1,500 m over 50 yr ago (Reeves et al., 1962). Although cattle may ultimately die from right-sided CHF secondary to pulmonary arterial hypertension, we cannot rule out the possibility that pulmonary venous

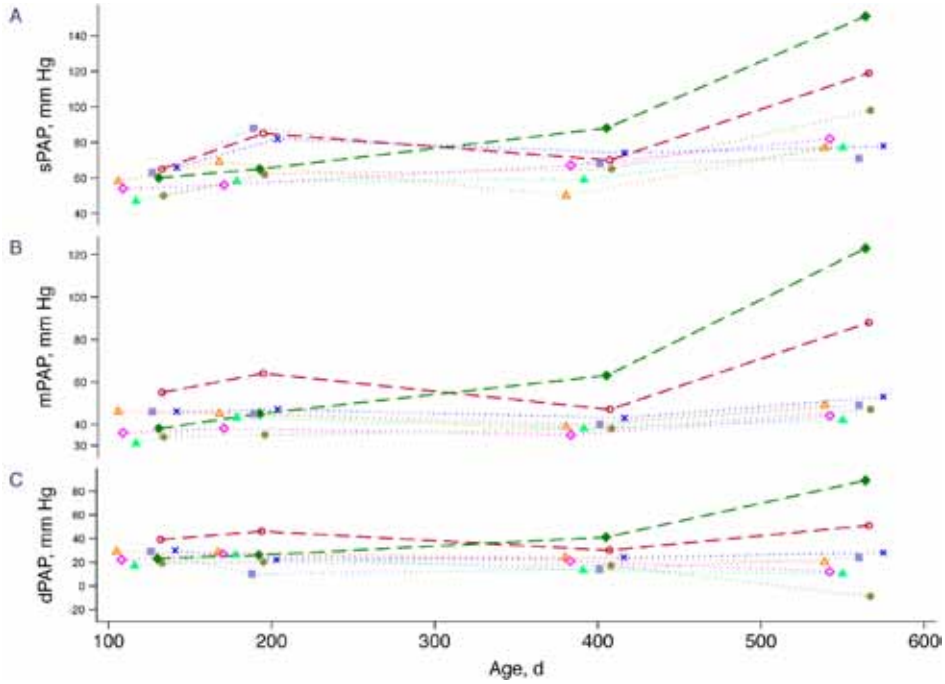

Figure 3. Illustrative line graph showing changes in A) systolic pulmonary arterial pressure (sPAP), B) mean pulmonary arterial pressure (mPAP), and C) diastolic pulmonary arterial pressures (dPAP) for male Angus calves tested at 4, 6, 13 (Group A), and 18 mo of age that did not receive a growth promoting implant (n = 8). Each calf is represented with a different marker and color. Dashed red and green lines represent 2 steers that had noticeably greater mPAP at 18 mo of age than the other 6 steers. Dotted lines link observations for the remaining 6 calves.

hypertension due to left-ventricular insufficiency is a contributing factor. We can also not rule out the possibility that, although body fat accumulation may predispose to greater mPAP and cardiac dysfunction in cattle, obesity before the development of CHF may confer survival advantages. In humans, this phenomenon has been termed the "obesity paradox" (Khalid et al., 2014).

There are several limitations to our study. First, only calves from 1 breeding program were followed into 1 feedlot, which means that we cannot extrapolate our results to other breeds or to ranches and confined feeding operations located at other altitudes. However, the Angus cattle studied represented the most common beef breed in the industry, and their management was reflective of production practices common to the Rocky Mountain region. Second, 30 calves were sold before entry into the feedlot. The mPAP of these calves was slightly greater than the group mean, which suggests that if any bias resulted from the removal of these calves from the study it would have been toward the null.

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