Temperature influences upon vascular dynamics in cattle measured by Doppler ultrasonography

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Abstract
1. Doppler-image ultrasonography was used to document vascular changes and blood flow rates of cattle (Bos taurus) under hot (32.7 °C) and cold (8.1 °C) conditions for 24 h.
2. Blood flow rates in the caudal artery increased from 27.8 (±2 h) to 43.4 mL/min at 23 h when cattle were maintained at 32.7 °C.
3. Cattle at 8.1 °C showed lower caudal artery flow rates after 23 h (29.3 at −2 h to 13.8 mL/min at 23 h), following a rate increase after the challenge.
4. The ability of Doppler-image ultrasound to detect vascular changes due to ambient temperature demonstrates its use as a non-invasive tool for assessing environmental stress on hemodynamics. Published by Elsevier Ltd.

1. Introduction
The use of Doppler-image ultrasound technology has been a mainstay in vascular assessment in human medicine for years. The primary utilization in animal agriculture and veterinary medicine has been to study the hemodynamics of horses (Hoffman et al., 2001; Mario et al., 1997; Raisis et al., 2000). Use in vascular studies of cattle has not been as prevalent; however, ultrasound imaging does offer a quick, efficient and non-invasive way of evaluating various changes in peripheral vessels of cattle (Raisis et al., 2000; King, 2006; Aiken et al., 2007). The objectives of this study were to develop a non-invasive technique for use in field studies and to evaluate its sensitivity in determining short-term changes peripheral vasculature of cattle during a moderate change in ambient temperature. Environmental changes are known to induce vascular changes in several species, including cattle (Folk, 1974; Hongo and Luck, 1953; Thauer, 1965). During heat stress, the peripheral vessels will dilate to facilitate the dissipation of body heat to prevent increases in core temperature, while during cold stress; the effect is vasoconstrictive to prevent heat loss to the peripheral tissues and a reduction in core body temperature. The initial dilation of the vessels is a local response, which allows for adjustments to blood flow without affecting systemic flow, especially when exposed to cold stress (Webb-Peploe and Shephard, 1968a–c).

Cattle grazing tall fescue containing the endophytic fungus Neotyphodium coenophialum often exhibit a variety of manifestations that are collectively referred to as fescue toxicosis (Cross, 2000; Oliver, 1997). A major component of fescue toxicosis is the vascular constriction of peripheral blood vessels, and the subsequent reduction in cutaneous blood flow. This reduction impairs the ability of the animal to dissipate heat and can result in hyperthermia, especially during heat stress (Oliver, 2005). Therefore, a field-adapted technique using Doppler ultrasonography to detect change in blood flow characteristics of cattle subjected to tall fescue toxicosis would be very valuable to producers, practitioners, and researchers. An experiment was conducted with cattle to determine if Doppler ultrasonography can be used to detect changes in blood flow characteristics over time when subjected to cold and heat challenges.

2. Materials and methods

2.1. Experimental animals
Six Angus crossbred steers (Bos taurus) (average body weight = 145.5 ± 17 kg) were halter broken and trained to stand. Three of the steers were housed in one of two environmental chambers located in the Brody Environmental Center at
the University of Missouri. The steers were maintained on a complete mixed ration formulated to meet 12.9% CP and 3.7% fat, and were fed at a rate of 2.0% of body weight for the duration of the study. All animals in this study were treated in compliance with the Animal Use Committee at the University of Missouri, Columbia.

2.2. Temperature regulation during thermal stress

The calves were acclimated to thermoneutral temperatures (18.8 °C for heat study and 16.5 °C for cold study) in the environmental chambers for 2 days prior to testing. They were then challenged by either raising the air temperature to 32.7 °C or reducing it to 8.1 °C, with temperature stabilization within 8 h (Fig. 1). The animals were challenged for 24 h at each air temperature and then returned to thermoneutrality. Ultrasound scans were repeated at −2 (i.e., 2 h prior to temperature change), 5, 23, 24 and 28 h (i.e., 4 h after returning to thermoneutrality at 18.8 °C for heat study and 16.5 °C for cold study).

2.3. Doppler ultrasound

Ultrasound scans of the caudal artery at the 4th coccygeal (Cd4) vertebrae were taken using an Aloka Prosound Ultrasound Unit (Aloka Inc., Wallingford, CT) with a UST-5542 (13 MHz) linear array transducer set to a 2-cm depth (Fig. 2). Three cross-sectional Doppler flow scans were taken to determine mean artery lumen area. Following the freezing of an individual scan, frames stored in the cine memory of the unit were searched to store the image exhibiting the maximum flow signal and this was assumed to be at peak systolic phase (Fig. 3). The flow signal was traced to estimate lumen area. Three Doppler spectra with a longitudinal transducer orientation also were collected (Fig. 4). Spectra were taken with a Doppler frequency of 6 MHz and a correction angle of 25°. Sample volume was set at 0.5 mm. Doppler gain was set at 40, which was the maximum setting before noise became apparent. Standardization of the techniques and verification of procedural efficacy were performed in two separate experiments as outlined in Aiken et al. (2007) and Aiken et al. (accepted for publication). Peak systolic velocity, end diastolic velocity, mean velocity, pulsatility index, flow rate per cardiac cycle (artery area x mean velocity x stroke time), and flow rate per minute (stroke volume x heart rate) were measured over three cardiac cycles within each scan and then averaged over the three scans. Pulsatility index was used as an indicator of vascular resistance (Petersen et al., 1997).
and the pulsatility index was calculated as described by Zwiebel (2000). Respiration was obtained by visual observations and counting of flank movement. Each of the studies was analyzed as repeated measures using PROC MIXED of SAS (Littell et al., 2006). Data were analyzed as a randomized complete block design that evaluated animal as a block effect to evaluate vascular changes relative to observations recorded during the thermoneutral period taken 2 h prior to temperature changes.

3. Results

3.1. Response to 32°C

Exposure to heat stress caused the animal’s respiration rate to increase from 82 (−2 h) to 120 breaths/min (23 and 24 h of the study) (P < 0.05), with no change in mean heart rate (82 beats/min) (Table 1). Rectal temperature averaged 39.0 ± 0.1°C across all five scanning periods and did not significantly change throughout the study (P > 0.10). Blood flow rates in the caudal artery did not increase initially (27.8 mL/min at −2 to 5 h), but increased from 26.3 (5 h) to 43.4 mL/min at 23 h of exposure (P < 0.10) in the heat-stressed cattle (Fig. 5). Flow rate declined toward the end of the study, but was not significantly different from the initial or the highest flow rates at 23 h. Flow rate per cardiac cycle was similar to the flow rate per minute (Table 1). Vessel diameter tended to increase (0.047–0.063 cm²) with the onset of the heat stress (Fig. 6), but was not statistically significant (P > 0.10). Change was also noted within the cardiac cycle of the heat-stressed animals. When air temperature increased, flow rate of the cardiac cycle showed no difference from the flow rate at the peak of the systolic portion of the cycle (Table 1). At the end of the diastolic cycle, there was a decline in the velocity from the thermoneutral level after 24 h (P < 0.05; Table 1). The pulsatility index tended to decreased due to vasodilation, but was not statistically significant (P > 0.10; Table 1) from the initial index values at thermoneutral.

3.2. Response to 3°C

Exposure to 3°C resulted in no change in mean heart rate (79 beats/min) across all five time periods (P > 0.10; Table 2). Respiration rate decreased from 62 (−2 h) to 36 breaths/min (23 h), and then increased to 53 breaths/min upon return to the original thermoneutral temperature (28 h) (P > 0.05; Table 2). Rectal temperature in the cattle housed in the cool chamber averaged 38.6 ± 0.1°C across the five periods and did not statistically change throughout the study (P > 0.10). Exposure to 3°C also reduced flow rate to the caudal artery after 23 h (P < 0.05) (29.3 at −2 h to 13.8 mL/min at 23 h). Blood flow rates of the cattle initially increased after the challenge. After 5 h at the lower ambient temperature, blood flow rate was 44.1 mL/min, increasing above the baseline of 29.3 mL/min (P < 0.05), but then declined to 13.8 mL/min at 23 h (Fig. 7). Similar patterns were observed in the blood flow per cardiac cycle (P < 0.05; Table 2) and in addition, the arterial area increased from 0.057 to 0.072 cm² at 5 h and then decreased (P < 0.01) to 0.043 cm² at 23 h (Fig. 8). The blood flow in the caudal artery of the cattle at 8°C showed no changes (P > 0.10) in peak systolic velocity or end diastolic velocity (Table 2). The pulsatility index was not statistically different with the decline in air temperature (P > 0.10; Table 2).

4. Discussion

4.1. Response to 32°C

The exposure of cattle to ambient temperatures marginally above the thermoneutral zone elicited physiological change. In an effort to dissipate heat, the animals increased the rate of respiration without an increase in heart rate. These reactions are consistent with results from several studies with cattle (Kibler et al., 1949; Kibler and Brody, 1949, 1950; Findlay, 1957). Normally, heart rate increases when cattle are exposed to upper air temperature extremes (Kibler et al., 1949; Kibler and Brody, 1949, 1950), but with this study the air temperature likely was neither high enough nor sustained long enough to elicit such a response. This was supported by the lack of change in rectal temperature. Had the cattle been exposed to for a longer duration to heat stress, a rise in rectal temperature might have been noted as shown in other studies (Kibler et al., 1949; Kibler and Brody,

![Fig. 5. Flow rates (mL/min) of cattle exposed to 32°C measured at −2 (2 h prior to exposure), 5, 23, 24 and 28 (4 h post-exposure) hours. P < 0.06; bars represent standard error of the least mean squares; coefficient of variation = 18.1%.

Table 1

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Cattle response to exposure of 32°C measured at −2 (2 h prior to exposure), 5, 23, 24 and 28 (4 h post-exposure) hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (h)</td>
<td>Heart rate (beats/min)</td>
</tr>
<tr>
<td>---------</td>
<td>------------------------</td>
</tr>
<tr>
<td>−2</td>
<td>87 ± 5</td>
</tr>
<tr>
<td>5</td>
<td>83 ± 5</td>
</tr>
<tr>
<td>23</td>
<td>83 ± 5</td>
</tr>
<tr>
<td>24</td>
<td>85 ± 5</td>
</tr>
<tr>
<td>28</td>
<td>83 ± 5</td>
</tr>
</tbody>
</table>

Means are separated by ± of standard error of the least means squares.
Alterations, such as the increase in vascular diameter and increase in blood flow rate to the caudal artery were expected in an effort to dissipate heat from the body. This has been shown in the extremities of several species, including humans, sheep, dogs, cats and monkeys (Folk, 1974; Hongo and Luck, 1953). Studies of the caudal artery in the tail of a monkey (Cercopithecus pygerythrus) showed increased vessel diameter and blood flow rate during heat stress (Hongo and Luck, 1953). In the present study, changes were noted within the cardiac cycle. The flow rate per cardiac cycle reacted in a mirror image to the flow rate per minute because heart rate was unchanged. Had the exposure air temperature been more severe, there might have been a change in these two components. Flow rates of the cardiac cycle showed no differences between the peak of the systolic portion of the cycle and the end of the diastolic cycle. However, there was a decline in the velocity from the thermoneutral level after 24 h. If the vessels were dilated during heat stress, the velocity of flow at the end of the diastolic phase would have decreased due to less resistance in the vessel and the change at the peak of the systolic phase would have been minimally affected. This was demonstrated as the animals returned to the thermoneutral temperature. The strength of the pulse, or pulsatility index, decreased due to vasodilation and reduced resistance to peripheral blood flow, as would be expected with heat stress, but was not statistically significant. The lack of statistical significance of pulsatility index may have been due to the high variability of these measurements and the small sample size in this experiment.

### 4.2. Response to 8°C

The cattle exposed to the cooler environment showed no change in heart rate across all five sample times. Cold exposure should demonstrate opposite effects from exposure to heat stress. Respiration rate decreased under the cool condition in an effort to reduce heat loss (Kibler and Brody, 1949, 1950) and returned to thermoneutral rate at the end of the study with increase in air temperature to the initial level. Cattle responded to the lower temperature with reduced flow rate to the caudal artery after 23 h of exposure, as would be expected with cold exposure (Folk, 1974).

Blood flow rate initially increased with the cold challenge. After 5 h in the cold, flow rate increased 175% over baseline readings; this is a normal response to cold exposure, where a temporary dilation of the peripheral vessels occurs before constriction of the vessel. This phenomenon has been described as the “rise and die away” in human extremities (Hongo and Luck, 1953). The effect has also been described in the extremities of cats, dogs, pigs and sheep (Folk, 1974; Schwinghamer and Adams, 1969). This effect in the present study was noted in the arterial area, with increased flow from −2 to 5 h and then a decrease at 23 h, showing the caudal artery was constricted. This effect also has been observed in dogs (Webb-Peploe and Shephard, 1968a–c). Blood flow per cardiac cycle reflected the change in blood flow per minute as found in the heat-stressed cattle. As in those cattle, the lack of influence upon the heart rate caused the changes to be the same. The blood flow in the caudal artery at 8°C exhibited no change in peak systolic velocity. With arterial constriction, we should have seen an increase in the end diastolic velocity due to increased

![Fig. 6. Cross-sectional arterial area (cm²) of cattle exposed to 32°C measured at -2 (2 h prior to exposure), 5, 23, 24 and 28 (4 h post-exposure) hours. P > 0.10; bars represent standard error of the least mean squares; coefficient of variation = 19.8%.

![Fig. 7. Flow rates (mL/min) of cattle exposed to 8°C measured at -2 (2 h prior to exposure), 5, 23, 24 and 28 (4 h post-exposure) hours. P < 0.05; bars represent standard error of the least mean squares; coefficient of variation = 23.1%.

### Table 2

Cattle response to exposure of 8°C measured at -2 (2 h prior to exposure), 5, 23, 24 and 28 (4 h post-exposure) hours

<table>
<thead>
<tr>
<th>Time (h)</th>
<th>2</th>
<th>5</th>
<th>23</th>
<th>24</th>
<th>28</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>81 ± 5</td>
<td>81 ± 5</td>
<td>77 ± 5</td>
<td>79 ± 5</td>
<td>76 ± 5</td>
</tr>
<tr>
<td>Respiration rate (breaths/min)</td>
<td>62 ± 5</td>
<td>82 ± 5</td>
<td>36 ± 5</td>
<td>34 ± 5</td>
<td>53 ± 5</td>
</tr>
<tr>
<td>Flow rate per cardiac cycle (cm/s)</td>
<td>0.34 ± 0.06</td>
<td>0.53 ± 0.07</td>
<td>0.20 ± 0.06</td>
<td>0.28 ± 0.06</td>
<td>0.40 ± 0.06</td>
</tr>
<tr>
<td>Peak systolic velocity (cm/s)</td>
<td>31.7 ± 3.8</td>
<td>29.3 ± 4.6</td>
<td>28.3 ± 3.8</td>
<td>24.8 ± 3.8</td>
<td>24.3 ± 3.8</td>
</tr>
<tr>
<td>End diastolic velocity (cm/s)</td>
<td>8.2 ± 3.5</td>
<td>6.4 ± 4.2</td>
<td>10.5 ± 3.5</td>
<td>7.5 ± 3.5</td>
<td>5.9 ± 3.5</td>
</tr>
<tr>
<td>Pulsatility index</td>
<td>7.7 ± 2.8</td>
<td>4.2 ± 3.4</td>
<td>8.5 ± 2.8</td>
<td>8.7 ± 2.8</td>
<td>4.2 ± 2.9</td>
</tr>
</tbody>
</table>

Means are separated by ± of standard error of the least means squares.
resistance to blood flow. In this instance, we noted a trend toward the highest values at 23 h, but this was not statistically significant. The pulsatility index also tended to increase with the decrease in temperature as would be expected with increased vasoconstriction, but was not significant statistically, most likely affected by the low numbers of animals and the numerous factors affecting the calculation of these values (Evans et al., 1989).

5. Conclusions

The primary goal of this study was to determine the effectiveness of Doppler-image ultrasonography to detect blood flow changes in cattle exposed to mild deviation in ambient temperature. With the limited number of cattle in each study, the ability of this technology to detect changes in the vascular system without changes in the core temperature is encouraging for detection of hemodynamic changes associated with increasing core body temperature. Evaluations of these changes in the field are difficult, but the use of Doppler-image ultrasound offers the potential to non-invasively look at vascular changes in cattle and to assess the complex vascular and hemodynamic changes that occur in cattle.

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References