

Levels of D- and L-lactate in Rumen Liquid, Blood, and Urine in Calves With and Without Evidence of Ruminal Drinking

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Objective

To find out whether ruminal drinking is associated with systemic lactic acidosis.

Materials and Methods

Calves (age: \pm 28 d upon admission) that were admitted to the II. Medical Animal Clinic between August 1997, and September 1998.

On the basis of pH in rumen liquid and base excess (BE) upon admission, calves were assigned to one of four groups:

Group A ("normal"): rumen pH \geq 6, BE \geq - 5 mmol/l (n = 21); group B ("ruminal drinkers"): rumen pH < 6, BE \geq - 5 mmol/l (n = 20); group C ("acidotic"): rumen pH \geq 6, BE < - 5 mmol/l (n = 20); group D ("ruminal drinkers and acidotic"): rumen pH < 6, BE < - 5 mmol/l (n = 20). Toward the end of the study, calves were recruited selectively to even up the groups. All groups included calves with and without diarrhea. Other parameters

determined on the day of admission: rumen liquid: D- and L-lactate; blood: D- and L-lactate, urea, creatinine, blood gases, anion gap (= [Na + K] - [Cl + HCO₃]); urine: D- and L-lactate, creatinine.

Results

Results are summarized in the table. Values given are mean \pm standard deviation (SD); all values are given in mmol/l, except those for D-lactate/creatinine and L-lactate/creatinine ratios in urine which are without dimension (mol/mol). These are calculated to correct for different degrees of urine concentration). Values with indexes that include identical letters are not statistically significantly different.

Discussion

Rumen lactate concentrations demonstrate an abnormal lactate load in the reticulorumen in the calves classified as ruminal drinkers on the basis of rumen pH

	A ("normal")	B ("ruminal drinkers")	C ("acidotic")	D ("r.d. and acidotic")
Rumen liquid				
D-lactate	2.5 \pm 2.7 ^a	16.7 \pm 15.9 ^b	3.7 \pm 3.7 ^a	21.6 \pm 15.5 ^b
L-lactate	1.9 \pm 4.3 ^a	24.0 \pm 16.2 ^b	3.5 \pm 5.1 ^a	24.2 \pm 13.9 ^b
Blood				
D-lactate	1.1 \pm 1.4 ^a	1.4 \pm 0.9 ^a	6.0 \pm 4.3 ^b	7.0 \pm 4.1 ^b
L-lactate	1.5 \pm 0.9 ^a	2.9 \pm 1.6 ^b	2.1 \pm 1.0 ^a	2.0 \pm 1.4 ^{ab}
HCO ₃	27.8 \pm 3.6 ^a	29.0 \pm 4.3 ^a	12.4 \pm 4.7 ^b	11.9 \pm 4.6 ^b
anion gap	7.1 \pm 3.0 ^a	8.2 \pm 4.2 ^a	19.2 \pm 5.9 ^b	20.4 \pm 4.5 ^b
urea	6.7 \pm 4.5 ^a	6.9 \pm 4.4 ^a	19.8 \pm 11.6 ^b	19.4 \pm 16.5 ^b
Urine				
D-lactate	4.6 \pm 4.8 ^a	5.8 \pm 4.2 ^a	20.4 \pm 22.9 ^b	22.4 \pm 26.3 ^b
D-lact/creat	.59 \pm 0.49 ^a	.60 \pm 0.61 ^a	3.34 \pm 4.28 ^b	3.72 \pm 5.12 ^b
L-lactate	.8 \pm 1.2 ^a	2.5 \pm 4.1 ^{ab}	2.7 \pm 5.9 ^{ab}	2.1 \pm 1.9 ^b
L-lact/creat	.04 \pm .05 ^a	.29 \pm .73 ^{ab}	.26 \pm .36 ^b	.34 \pm .59 ^b

alone. But this was not associated with a concomitant rise in blood lactate levels in group B.

Groups C and D apparently were more dehydrated than groups A and B (higher urea concentration).

L-lactate seems to be much more readily metabolized, while D-lactate is excreted in larger quantities via urine. Any systemic source of lactate (e.g. anaerobic glycolysis in poorly perfused organs) should produce only L-lactate, while non-systemic sources (rumen and/or intestines) should yield D- and L-lactate in approximately equal amounts.

Mild lactic acidosis was present in the two "acidotic" groups (C and D), which mainly was due to D-

lactate. Accumulation of lactate explains the increase in anion gap from a normal of around 15 mmol/l ([135 + 5] – [100 + 25]) in these two groups.

The reticulorumen does not seem to be the main source of D-lactate that was present in the blood, and particularly in the urine, of calves in groups C and D. An obvious alternative source would be the intestines. An entirely different explanation would be depletion of bicarbonate in groups C and D, with loss of the capacity to buffer a typical load of lactate.