

Comparison of the clinical pictures in experimentally induced metabolic acidosis, hyper-L-lactatemia and hyper-D-lactatemia.

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In calves with diarrhoea, systemic acidosis and hyper-L- and D-lactatemia constitute the most important metabolic consequences. Recent studies indicate, however, that clinical signs are determined more by the extent of D-lactate ! accumulation than by the metabolic acidosis. We summarize the results of different trials in which the single effects of metabolic acidosis, hyper-L-lactatemia, and hyper-D-lactatemia on the clinical condition were studied.

1st trial: induction of metabolic acidosis:

15 Holstein calves received 4.0 l of a 0.1M HCl solution i.v. over a period of 80 minutes (50 ml/min).

The procedure managed to provoke a relatively severe metabolic acidosis, that reached a peak ABE mean value of -22.4 mmol/l (range -17.0 to -33.1 mmol/l). Despite the acidemia during the entire observation period no calves showed any clinical repercussions.

2nd trial: induction of hyper-L-lactatemia:

5 Holstein calves received 400 ml of a 25% L-lactate solution: 100 ml as i.v. bolus (1 min.), and 300 ml via an i.v. infusion over 35 minutes.

The experimental procedure provoked a severe hyper-L-lactatemia that reached a peak mean value of 25.0 mmol/l at the end of the infusion (range 18.8 to 38.3 mmol/l). No clinical signs accompanied the hyper-L-lactatemia. Signs of metabolic alkalosis were observed.

3rd trial: induction of hyper-D-lactatemia:

5 Holstein calves received 100 ml of a 25% D-lactate solution via an i.v. bolus (1 minute).

A clear increase of the D-lactatemia was observed right from the first control after the administration. At this time the mean value was 16.4 mmol/l (range 13.8 to 18.7 mmol/l).

Afterwards the D-lactatemia tended slowly to recover its pre-trial value.

Between eight and 40 minutes after the injection all calves showed the following clinical signs: impairment of the palpebral reflex, somnolence, staggering, prolonged periods of motionless, standing with unphysiological postures, lowered and sometimes waving head, and drooping ears. Calves lay down often and for longer, and had to be helped to rise. No impairment of the sucking reflex was observed. After four hours all calves appeared clinically normal. No deviation of the acid-base balance was observed.

Conclusions

The results of the summarized studies show that with the exception of any impairment of the sucking reflex, all the signs once attributed to metabolic acidosis can be reproduced by inducing hyper-D-lactatemia without acidosis.