

6 Summary

The objective of the study is to evaluate the use of the intravenous infusion of 5M NH_4Cl to induce an experimental non-respiratory (metabolic) acidosis in calves and young camels. The effect was evaluated on the basis of the parameters of Henderson-Hasselbalch model and Stewart's strong ion model.

32 clinically healthy calves (age: 4-104 days) and 24 young camels (age: ≤ 3 -5 months) were infused with 5M NH_4Cl at a dose of 1.0 ml/kg. The 1:10 diluted final solution was infused into the jugular vein via a permanent catheter for a period of 2-2.5 hrs. Venous blood samples were collected before the initiation (zero time) and 2, 4, 6, 8, 24 and 48 hrs after the beginning of the infusion. In the same time urine samples were collected. Blood and urine samples were used for the determination of various acid-base parameters.

In response to the intravenous infusion of 5M NH_4Cl solution, respiratory rate (RR) increased significantly from the initial values of 28-60 breaths/min to 30-65 breaths/min (calves) and from 9-13 breaths/min to 13-20 breaths/min (young camels) after 2 hrs, which can be considered as a respiratory compensation. Heart rate decreased from the initial values of 80-160 beats/min to 78-110 beats/min (calves) and from 58-65 beats/min to 40-55 beats/min (young camels) after 4 hrs due to hyperkalaemia.

Acidaemia was observed after 2 hrs in both calves and young camels, which was characterised by a sharp decrease in venous blood pH from the initial values of 7.38-7.40 to 7.32-7.36 (calves) and from 7.42 to 7.32-7.34 (young camels) in response to the experimentally induced metabolic acidosis. The young animals were observed to be more acidotic than the older ones. In calves, venous blood P_{CO_2} decreased significantly from the initial values of 5.7-6.8 kPa to 5.2-5.6 kPa 8 hrs after the beginning of the infusion, which can probably consider as a compensatory respiratory alkalosis. In calves, blood- $[\text{HCO}_3^-]$ decreased significantly from the initial values of 26-32 mmol/l to 21-26 mmol/l by 2 hrs after the beginning of the infusion. Blood- $[\text{BE}]$ decreased significantly from the initial values of 2-6 mmol/l to -5-0 mmol/l after 2 hrs in response to the experimentally induced metabolic acidosis in calves. Venous blood P_{CO_2} , blood- $[\text{HCO}_3^-]$ and blood- $[\text{BE}]$ had critical influence on the venous blood pH in calves in accordance with the Henderson-Hasselbalch theory.

In both calves and young camels, serum- $[\text{SID}_3]$ decreased significantly from the initial values of 44-47 mmol/l to 38-44 mmol/l (calves) and from 43-44 mmol/l to 37-38 mmol/l (young camels) after 2 hrs and maintained at a lower level after 24 and 48 hrs. The decrease in serum- $[\text{SID}_3]$ observed could be due to hyponatraemia (143-146 mmol/l, young camels), hyperkalaemia (4.9-5.8 mmol/l, calves) and mainly due to hyperchloraemia (101-107 mmol/l,

calves and 114-115 mmol/l, young camels). Serum- $[A_{tot}]$ decreased significantly from the initial values of 10.5-14.0 mmol/l to 9-13 mmol/l (calves) and from 11.8-13.4 mmol/l to 10.5-11.8 mmol/l (young camels) after 4 hrs and maintained at a lower level after 24 and 48 hrs, which can be considered as a compensatory metabolic alkalosis. The decrease in serum- $[A_{tot}]$ could be related to hypophosphataemia in both calves and young camels. In young camels, hypoproteinaemia (48-51 mmol/l) and hypoalbuminaemia (27.5-30 mmol/l) were observed by 4 hrs after the beginning of the infusion and can be considered as the main factors of lowering serum- $[A_{tot}]$ in addition to hypophosphataemia occurred.

On the basis of the Stewart's theory, serum- $[SID_3]$ and serum- $[A_{tot}]$ had a marked influence on the venous blood pH in both calves and young camels. The significant decrease in blood pH was accompanied by a respiratory compensation characterised by hyperventilation and consequently caused a decrease in P_{CO_2} .

Urine pH decreased gradually with time from the initial values of 6.6-6.9 to 5.3-5.6 (calves) and from 7.6-8.0 to 6.0-6.7 (young camels) in response to the experimentally induced metabolic acidosis after 8 hrs and remained at a lower level after 24 and 48 hrs after the beginning of the infusion, suggesting the renal compensation to the acute metabolic acidosis. Urine osmolality increased significantly from the initial values of 100-300 mOsmol/kg to 400-700 mOsmol/kg (calves) and from 900-1100 mOsmol/kg to 1200-1300 mOsmol/kg (young camels) after 24 and 48 hrs. FE_{Na^+} was increased from the initial values of $< 0.1\%$ to 0.2-0.3% in response to the intravenous infusion only in the young camels. FE_{K^+} increased significantly from the initial values of 20-30% to 25-60% (calves) and from 10-15% to 20-40% (young camels) after 8 hrs. FE_{Cl^-} increased from the initial values of 0.06-4% to 2-4% (calves) and 1.5-1.6% to 1.6-2% (young camels) by 8 hrs after the beginning of the infusion. FE_{P_i} increased significantly in response to the experimentally induced metabolic acidosis only in the young camels from the initial values of 0.2-0.7% to 0.9-2.0% after 8 hrs. The significant increase in the urine osmolality and the $FE_{electrolyte}$ during the experimental period and after 24 hrs in both calves and young camels can be considered as the main signs of induction of the experimentally metabolic acidosis.

We conclude that the intravenous infusion of 5M NH_4Cl was successfully used to induce experimentally metabolic acidosis in both calves and young camels. The results show that young animals ($\leq 4^{th}$ w, calves and ≤ 3 m, young camels) were more sensitive to metabolic acidosis than the older ones. Clinically, a particular case must therefore be taken to prevent metabolic acidosis in young animals.