VASOPRESSIN RESPONSE TO WATER AND SALT LOAD IN NORMAL COWS.

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Introduction.

Arginine vasopressin (AVP) or antidiuretic hormone plays a primary role in the regulation of the body fluids. Little is known at present about the regulation of AVP secretion in cattle. Our interest in the regulation of the release of AVP in cattle arises from two basic questions.

First, it has been demonstrated recently that administration of AVP to adult ruminants stimulates reticular groove contraction[1]. This may be of interest, as suggested by others[1,2] for the oral administration of drugs that should bypass the ruminoreticulum. No data are available on the role of AVP in the consistent closure of the reticular groove in the young calf.

Secondly, pyloric stenosis in cattle results in a metabolic alkalosis. A dehydration occurs, accompanied by a hypochloraemia, a hypokalaemia, a metabolic alkalosis and a uraemia. An extreme dehydration can be found, with a production of diluted urine. No data are available of the plasma AVP concentrations in these cases.

To evaluate vasopressin values from patient cases and experimental animals, reference values were collected in 5 normal dairy cows during a water or a salt load.

Experimental design

Five adult dairy cows of the Dutch Friesian or Holstein Friesian breed were used.

	Body weight	Production	Stage
Cow A	673 kg	25 l/day	8 w post partum
Cow B	616 kg	12 l/day	6 m pregnant
Cow C	567 kg	32 l/day	3 w post partum
Cow D	733 kg	20 l/day	3 m pregnant
Cow E	633 kg	12 l/day	6 m pregnant

In all cows plasma Arginine Vasopressine, AVP, was measured after oral water load and, approximately 10 days afterwards after intravenous salt load.

Water load: food was withdrawn 12 hours previous to the experiment and water 6 hours previous to the experiment (to avoid influences by the drinking of water shortly before the experiment). Blood was sampled 20 minutes before, and at the beginning of the experiment. Water was then administered by nasogastric tube in the amount of 15 % of body weight. After that blood was sampled at 30, 60, 120, 180, 240 and 300 minutes.

Salt load: food was withdrawn 12 hours previous to the experiment. Blood was sampled 20 minutes before and at the beginning of the experiment. A solution of 20 % NaCl was then administered in the right-hand jugular vein by means of an infusion pump at the speed of 25 ml/min during 120 minutes (thus giving 3000 ml, containing 600 gram of NaCl). Blood was sampled during this infusion from the left jugular vein at 20, 40, 60, 80, 100 and 120 minutes.

All blood samples were tested for AVP, Na^+, K^+, Cl^-, HCO_3^- and PCV.

Results and discussion.

Water load resulted in all five cows in a distinct decrease of plasma Na concentrations, as is shown in figure 1. The relation between sodium and AVP during water load is shown in figure 2. Although in four cows low levels of AVP were observed during the experiment, a consistent decrease was not found. Pre-experimental AVP concentrations in cow C showed already levels above those found in the other experiments. This could be due to thirst. Because milk production of cow C was considerable (32 l/day), she must have been used to a considerable uptake of water. So the 6 hours thirst period in this experiment might have been too long for this cow. However, none of the determined blood parameters showed any sign of dehydration. Consequently the mechanism by which this supposed thirst could have induced an increased AVP level is not clear. Even if the results of cow C are left out, the water load experiments did not show any correlation between plasma sodium and AVP concentrations.

Plasma sodium concentrations after intravenous salt load increased to high values up to 171 mmol/l, as shown in figure 3. Salt load induced an increase of AVP concentrations, which ranged in most cases from about 10 to 20 pg/ml and in Cow B became as high as 102 pg/ml. Based on other experiments it has been suggested that an AVP concentration of about 60 pg/ml is needed for reticular groove contraction[2].

AVP concentration showed a positive correlation with plasma sodium (figure 4). The correlation coefficient was 0.48, the regression equation was:

$$AVP = -100.7 + 0.73\ Na$$

Mean pre-experimental AVP concentration was 4.0 pg/ml, with standard deviation 2.35 pg/ml.

It is concluded that AVP concentration during hypernatremia is related to plasma sodium levels, whereas during hyponatremia no further decrease in plasma AVP concentration occurs.

References

1. Mikhail, M., Untersuchungen über die Wirksamkeit von Vasopressin auf die Schlundrinnenkontraktion beim erwachsenen Rind und deren Nutzung bei der Behandlung an Durchfall erkrankter Patienten. Inaugural Dissertation, Tierärztliche Hochschule Hannover, 1986. - 2. Mikhail, M., Scholz, H., Untersuchungen zur Nutzung der Schlundrinnenkontraktion in der Behandlung innerer Erkrankungen des erwachsenen Rindes. 2. Mitteilung: Vasopressinkonzentrationen im Blutplasma. Tierärztliche Umschau 42: 378-382, 1987.

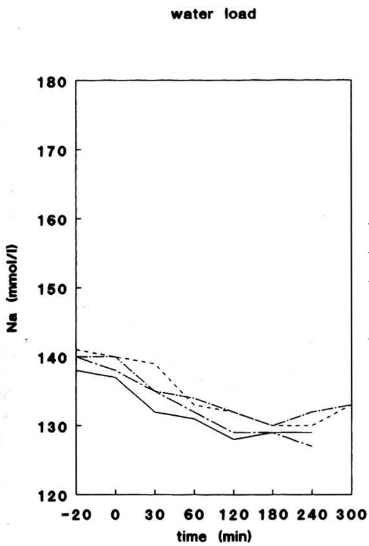

Figure 1: Plasma Na⁺ in five cows (A to E) after an oral water load of 15% of body weight.

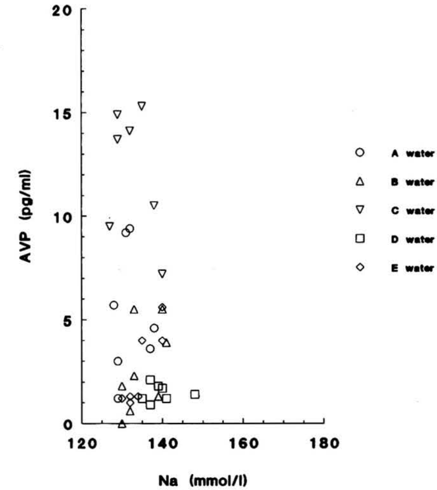

Figure 2: Plasma AVP and plasma Na⁺ in five cows (A to E) after oral water load. No obvious correlation between AVP and Na⁺ is observed.

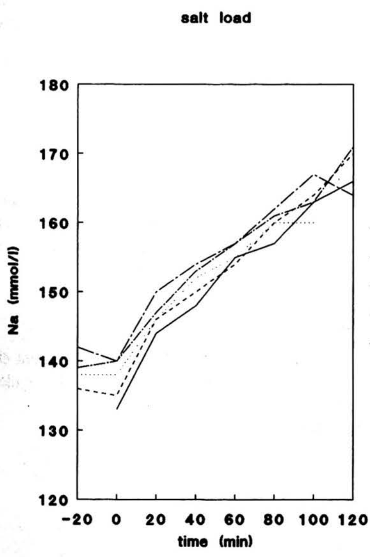

Figure 3: Plasma Na⁺ in five cows (A to E) during intravenous administration of 3000 ml 20% NaCl-solution in 120 minutes.

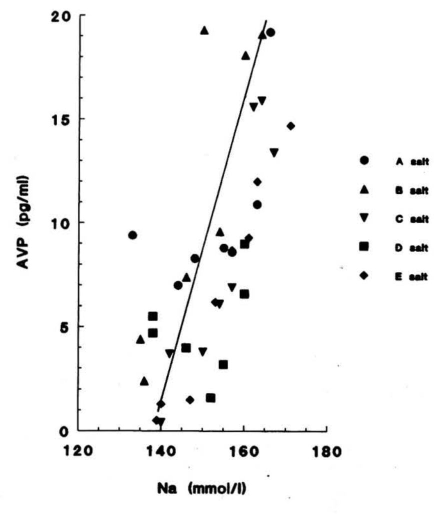

Figure 4: Plasma AVP and plasma Na⁺ in five cows (A to E) during intravenous administration of 3000 ml 20% NaCl-solution in 120 minutes. The regression equation is: AVP = -100.7 + 0.73 Na