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CHOLINESTERASE LEVELS IN BLOOD PLASMA AND ERYTHROCYTES FROM CALVES, NORMAL DELIVERING COWS AND COWS SUFFERING FROM PARTURIENT PARESIS

By

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FORSLUND, K., C. BJÖRKMAN and M. ABRAHAMSSON: *Cholinesterase levels in blood plasma and erythrocytes from calves, normal delivering cows and cows suffering from parturient paresis.* Acta vet. scand. 1983, 24, 185—199. — Plasma cholinesterase (pChE) levels and erythrocyte acetylcholinesterase (eAChE) levels were studied in 6 cows before, during and after parturition (Group I), their calves (Group II), 38 cows suffering from parturient paresis (Group III) and 14 newly delivered non-paretic cows (Group IV).

The mean of the pChE level in Group I was 1.5 μkat/l ± 0.20 before parturition and decreased significantly ($P \leq 0.05$) to 1.2 μkat/l ± 0.16 after parturition. The eAChE level was before parturition ≃ 140 μkat/l and decreased to ≃ 130 μkat/l 4—5 weeks after parturition.

At birth the pChE level was 12.8 μkat/l ± 5.9 in Group II. After 4 weeks the level had decreased to 2.3 μkat/l ± 0.3. In the bull calves the pChE level started to increase when they were 6 weeks old and reached a level of 5.7 μkat/l ± 0.6 before slaughter at 6 months of age. The heifers did not show this increase. They had a level of around 2 μkat/l throughout the investigation. The eAChE level at birth was 119 μkat/l and increased slowly to a level of 145 μkat/l at 6 months. No differences between the sexes were found.

The cows suffering from parturient paresis had a pChE level of 1.80 μkat/l ± 0.30 before treatment with calcium (Ca). The level decreased significantly ($P \leq 0.001$) after Ca-infusion to a level of 1.67 μkat/l ± 0.29. Group IV had a pChE level of 1.65 μkat/l ± 0.42 at parturition. Two to 4 months later the cows that had recovered from milk fever had a level of 1.61 μkat/l ± 0.31 and the control cows 1.66 μkat/l ± 0.48. No differences between the groups were found for the eAChE level.

The findings show that parturition influences the pChE level in cows and that sex influences the pChE level in calves between 6 weeks to at least 6 months of age. Furthermore the elevated pChE level found in the cows suffering from parturient paresis before Ca infusion may be a further sign of a disturbance in the cholinergic system with a special preference to the neuromuscular junctions.

acetylcholinesterase; calcium; calves; cows; magnesium; parturient paresis; phosphorus; plasma cholinesterase.

Parturient paresis in cows is in spite of 60 years of intensive research still a disease of uncertain etiology. A hypocalcemia in non-ruminants causes a tetany in contrast to the paralysis seen in the hypocalcemic ruminants. Furthermore, the hypocalcemia in cows has not been explained by failure in the Ca-regulating hormones and vitamins (*Mayer et al.* 1975, *Horst et al.* 1977, *Jönsson et al.* 1980, *Forslund et al.* 1980). *Kowalczyk & Mayer* (1972) reported that the calcium concentration of the muscle of paretic cows was not significantly less than that for parturient control cows, despite more severe hypocalcemia in the paretic cows. They did find an increase in sodium and a decrease in potassium in the muscles of the paretic cows compared to the control cows. It is known that ruminants have very little cholinesterase (ChE) in blood plasma compared to other species (*Augustinsson* 1959). They are also extremely sensitive to neuromuscular blocking agents as e.g. d-tubocurarine and succinylcholine (*Adams* 1977).

Changes in acetylcholinesterase (AChE) activities in cerebrospinal fluid of patients with senile dementia of Alzheimer type has been reported (*Soininen et al.* 1981). ChE activity in the blood plasma of patients with Alzheimer's disease does increase, but not the red cell acetylcholinesterase (eAChE) activity (*Smith et al.* 1982). A pathway from the nerve cell to the endothelial cells has also been demonstrated, that enables the neuron to communicate with the vasculature (*Kreutzberg & Tóth* 1974). Therefore it was of interest to study the level of ChE in blood from cows suffering from parturient paresis compared to non paretic cows and to follow the normal levels of plasma ChE (pChE) and eAChE before, during and after parturition in cows without parturient paresis.

A method for determination of AChE activity in erythrocytes and ChE in blood plasma from cattle is also described.

MATERIALS AND METHODS

Acetylthiocholine was used as substrate and 2,2′-dithiopyridine as a reagent on produced thiocholine. The reaction product 2-thiopyridone has an absorption maximum at 343 nm. Blood samples were obtained through puncture of the jugular vein in tubes with 10 IU/ml of heparin added.

Packed cell volume (PCV) was measured in each blood

sample. The rest of the sample was divided into 2 parts. One part was frozen as whole blood. The other was centrifuged and plasma was removed and frozen. The freezing procedure hemolysed the whole blood. A 0.10 mmol/l 2,2′-dithiopyridine (Sigma Chemical Co.) solution was prepared in 0.05 mol/l phosphate buffer pH 8.0. One ml of this solution was mixed with 50 µl of the sample. After 10 min 20 µl 0,075 mol/l acetylthiocholine iodide (Sigma Chemical Co.) was added and the increase in absorbance measured during 3 periods of 30 s each at 343 nm and 37 °C in a Bausch & Lomb Spectronic 2100 enzyme analyzer. The solutions were made fresh each day and the acetylthiocholine iodide solution was stored on ice. Blood plasma was analysed undiluted and whole blood was diluted 1:11 or 1:21 in 0.9 % NaCl. Triplicate measurements were made on the whole blood samples and duplicate measurements on the plasma samples. Within each test series one standard serum sample (Precinorm E from Boehringer and Mannheim) and a reagent blank sample were analysed. Cholinesterase activities (expressed as µkat/l) were calculated according to Lambert-Beer's law. The ChE activity in erythrocytes was calculated from the PCV and the ChE activities in whole blood and plasma as follows:

$$\alpha_{\text{erythrocytes}} = \frac{\alpha_{\text{whole blood}} - \left(1 - \frac{\text{PCV}}{100}\right) \times \alpha_{\text{plasma}}}{\frac{\text{PCV}}{100}}$$

where α is ChE activity.

The method used is a modification of *Elleman et al.* (1961). The use of 2,2′-dithiopyridine and a wave length of 343 nm made it possible to use visible light and the same reagent for measurements in whole blood and plasma without disturbances from hemoglobin. The effect of these disulphides on ChE activities has earlier been discussed by *Augustinsson & Eriksson* (1974). *Elleman et al.* (1961) and *Augustinsson et al.* (1978) describe methods for measuring AChE in erythrocytes in humans. They use selective inhibitors to inhibit the plasma butyrylcholinesterase (BuChE). The main part of ChE present in blood plasma from cows is AChE. The erythrocytes have exclusively AChE. These facts made it impossible to use inhibitors to separate pChE from eAChE.

Washing erythrocytes is very time consuming and the ChE

activity in erythrocytes was therefore calculated as described above. To check the reliability of this procedure a test with blood samples from 18 animals was done. In these samples the ChE activities in erythrocytes washed twice in 0.9 % NaCl were analysed and the results were compared with the calculated activities in the erythrocytes. There was no statistically significant difference ($P > 0.05$) between the ChE values from the calculated erythrocyte activities compared to the activities in the washed erythrocytes.

To measure the variability of the method samples were taken once a week from 2 cows during 20 weeks. The 2 samples were pooled and the PCV of the pooled sample was measured. The pooled sample was split into 3 parts and the parts were frozen as whole blood and blood plasma. The ChE activities in the blood plasma and whole blood were analysed the next day. This was repeated the 2 following weeks. This procedure was done because the ChE activity decreased after 4 weeks of storage (see below). For the ChE activities found in the 20 samples a two-factor analysis of variance was used with sample as a fixed factor and week treated as a random factor. There were a within-assay variation of 2.8 % for the ChE activities in plasma and 4.5 % in erythrocytes and a between-assay variation in plasma of 4.8 % and in erythrocytes of 8.5 %. This can be compared with the coefficient of variation of about 4 % reported by *Ellman et al.* (1961).

To determine to what extent the measured ChE activities were due to BuChE the following test was done: the enzyme activity was first measured on whole blood and plasma from cows as described above. Then to a new set of reaction mixtures prepared from the same sample 20 µl of 6 mmol/l BuChE inhibitor Astra 1397 (a gift from Astra Research Development Laboratory) was added and the ChE activity was measured. Significant amounts (13—20 %) of the ChE activity in plasma but not in erythrocytes were inhibited when acetylthiocholine iodide was used as substrate and Astra 1397 was added. The ratio of AChE — BuChE was not altered when the total amount of ChE increased.

Changes in ChE activity in blood stored 1—8 h in refrigerator and at room temperature were measured and no decrease in activity was found until after 5 h. Storage in —18°C was tested for 1—6 weeks and the ChE activity decreased after 4 weeks.

The plasma calcium (Ca) was determined fluorometrically as described by *Skerry* (1965) and inorganic phosphorus (P) according to *Fiske & Subbarow* (1925). The plasma magnesium (Mg) was determined by atomic absorption spectroscopy (*Hansen & Freier* 1967). For the statistical evaluation Student's t-tests for paired and unpaired data were used.

Animal experiments

Four groups of cattle were studied. The first group consisted of 6 cows: 5 Swedish Red and White Breed (SRB) and 1 cross Swedish Red and White × Swedish Friesian Breed (SLB). These cows were regarded as normal lactating cows. Their ages varied between 5 and 8 years. Blood samples were collected once a week 4 weeks before parturition until 16 weeks after parturition, depending on when the cows were available. The treatment and handling of the blood samples were the same as described in the methods section. Centrifugation and freezing of the samples were completed within 5 h after collection.

The second group consisted of the calves, 3 males and 3 females, from the cows in Group I. Blood samples were collected from their birth until 6—7 months of age, when they were slaughtered. The blood samples were collected at first once a week but later every second week.

Thirty-eight cows suffering from parturient paresis from different farms in the neighbourhood of Uppsala were included in the third group. They were of either SLB or SRB or mixed (SLB-SRB) breeds. Their ages varied between 5 and 13 years. Two blood samples were collected from each cow, the first in conjunction with the clinical examination and before Ca-solution was given as a treatment. The second blood sample was collected immediately after the Ca-infusion. The Ca-infusion contained about 8 g of Ca (Cadexil, Agrivet, Uppsala, Novocalc, Ferrosan and Kalciflex vet. Ferrosan, Malmö). From 14 of these cows it was possible to recollect blood samples at 2—4 months after the recovery from milk fever.

The fourth group consisted of 11 cows of the same breeds as above and from the same farms as mentioned above. Their ages varied between 4 and 8 years. They were newly delivered but not paretic. Only 1 blood sample was collected from these 0—2 days post partum. It was possible to follow 7 of these animals in Group IV with another blood samples 2—4 months later.

RESULTS

One of the cows (No. 6) in the first group suffered parturient paresis and was excluded from this group. The cow was treated twice with a Ca-solution before recovery. None of the other cows showed any clinical signs of milk fiver throughout the investigation. The blood Ca-level slightly decreased in all animals at parturition (Fig. 1).

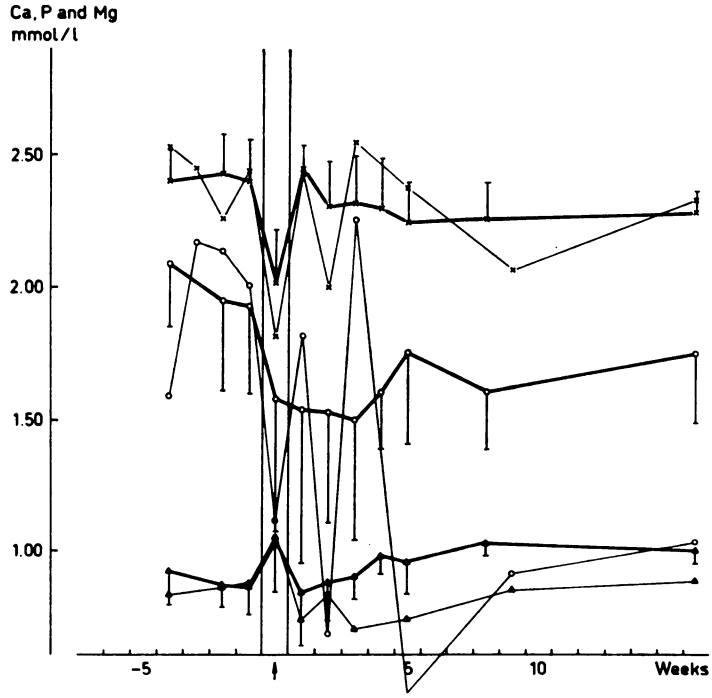
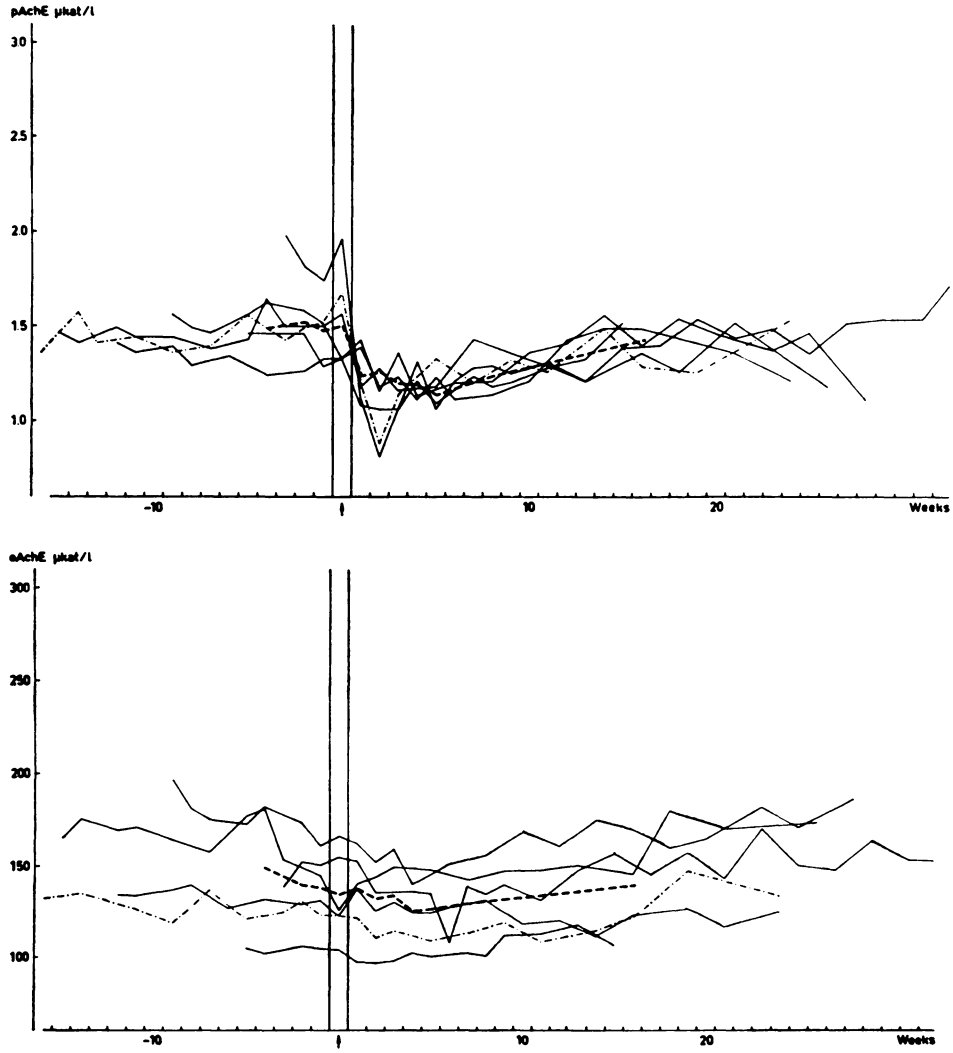


Figure 1. Mean blood Ca (×——×), P (o——o) and Mg (▲——▲) levels ± s for cows in Group I, before, during and after parturition (↑). The thin lines represent the cow suffering from parturient paresis.

Inorganic phosphate (P) also decreased at parturition and magnesium (Mg) increased (Fig. 1). The pChE decreased significantly ($P \leq 0.05$) 1 week after parturition (Fig. 2a). After 2 weeks the level of pChE started to slowly increase to the level seen before calving. The decrease of eAChE seen after parturition was not significant (Fig. 2a and b).

There were high levels of pChE in all calves at birth. The mean level gradually decreased to 2.3 μkat/l ($n = 6$) in week 6. The level of eAChE is on the contrary lower at birth and start



Figures 2a and b. The levels of pChE and eAChE in individual cows (——). The thick dotted (- - -) line represents the mean of the 5 cows in Group I before, during and after parturition (↑). The thin dotted line (- · - · -) represents the cow suffering from parturient paresis.

to increase after 5—6 weeks. From 2 of the calves samples from their earliest weeks are lacking. After the 6th week the blood level of pChE in the heifers and the bulls separated significantly (Fig. 3). For eAChE the influence of individual animal was

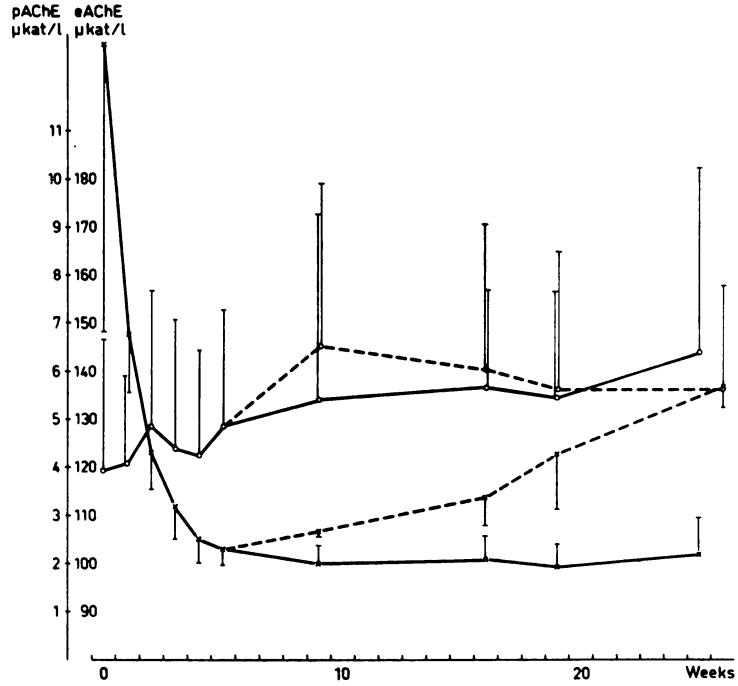


Figure 3. Mean levels of pChE (×——×) and eAChE (o——o) in Group II. Until the 6th week the lines represent 6 animals each, 3 females and 3 males. After 6 weeks the dotted lines (- - -) represent the bulls (n = 3) and the continuous lines represent the heifers (n = 3).

greater than the influence of sex (Fig. 3). Ca, P and Mg did not alter from normal values at any time during investigation.

The means, ranges and standard deviations for the pre-treatment levels of Ca, P and Mg in cows suffering from parturient paresis (Group III) and 14 newly delivered non-paretic cows (Group IV) are presented in Table 1. The pChE was significantly ($P \leqq 0.001$) higher in the blood samples collected before treatment compared with the level after Ca-infusion. This was not found for the eAChE level (Table 1).

The pChE level ($P \leqq 0.10$) and eAChE ($P > 0.05$) in the blood samples from the newly delivered non-paretic cows in Group IV were not significantly different from the paretic cows before treatment (Table 1).

The level of pChE in the blood samples collected before treatment in the paretic cows was statistically significantly higher ($P < 0.001$) than in the samples collected at 2—4 months after

Table 1. Means, standard deviations (s) and ranges for the blood substances studied in Groups III and IV.

Substance	Cows suffering from parturient paresis		Control cows	
	a n = 38	b n = 14	a n = 11	b n = 7
Ca mmol/l				
mean	1.25	2.36	2.07	2.35
s	0.35	0.12	0.24	0.17
range	0.70—1.95	2.18—2.55	1.62—2.43	2.15—2.62
P mmol/l				
mean	0.49	1.44	1.33	1.53
s	0.29	0.38	0.42	0.24
range	0.10—1.26	0.74—1.96	0.80—1.88	1.23—1.85
Mg mmol/l				
mean	0.96	0.92	0.93	0.96
s	0.25	0.13	0.14	0.09
range	0.11—1.62	0.69—1.09	0.79—1.17	0.91—1.15
	Ca-infusion			
	Before	After		
pChE μkat/l				
mean	1.80	1.67	1.61	1.65	1.66
s	0.30	0.29	0.31	0.42	0.48
range	1.01—2.44	0.90—2.32	1.14—2.12	1.32—2.63	1.06—2.50
eAChE μkat/l				
mean	125.0	123.3	128.2	124.5	121.8
s	16.2	16.5	16.3	17.1	12.2
range	104.0—170.8	98.4—156.7	111.1—158.9	97.3—152.5	104.3—134.5

a: At parturition.
b: 2—4 months after parturition.

recovery from milk fever. This was not found when comparing the sample collected after treatment to the sample collected 2—4 months later. No difference were found for eAChE.

When comparing the pChE and the eAChE levels in the blood samples from the non-paretic cows collected at calving with those collected 2—4 months later, there were no statistically significant differences ($P > 0.05$).

DISCUSSION

The main purpose of this investigation was to find out whether paretic cows had different levels of ChE in plasma and

erythorcytes compared to non-paretic newly delivered cows. To begin with the normal patterns of pChE and eAChE in cows around parturition had to be determined.

One of the 6 normal cows in Group I suffered parturient paresis and was excluded from the group. The clinical signs in combination with the lowered Ca level in plasma verified the diagnosis. Though the other cows had lowered Ca level (1 even lower than the paretic cow) none of them showed any clinical signs of paresis. The lowered Ca level around parturition is accepted as normal (*Hallgren et al.* 1959). Also the decrease in P seen in all animals at parturition is to be regarded as normal and has earlier been reported by several authors e.g. *Luthman & Persson* (1975). The slight increase in Mg is also to be accepted as normal (*Carlström* 1961).

Edqvist et al. (1978) described the level of 15-keto-13,14-dihydro-$PGF_{2\alpha}$ in cows from 60 days before to 40 days after parturition. They found the metabolite of $PGF_{2\alpha}$ increased at parturition and then slowly decreased during the following 20 days. *Borda et al.* (1982) suggested that endogenous $PGF_{2\alpha}$ facilitates the action of acetylcholine through an increase in the concentration of the cholinergic neurotransmitter, probably via an inhibition of the acetylcholinesterase activity.

The significant ($P \leqq 0.05$) lowering of pChE in all cows after parturition might be a consequense of the high prostaglandin $PGF_{2\alpha}$ release during and after parturition and this might reflect the influences of $PGF_{2\alpha}$ on the cholinergic system.

There was no significant decrease after parturition for eAChE, but there was a tendency for eAChE to decrease when the pChE level increased and vice versa.

High levels of pChE in newborn calves have earlier been found by *May et al.* (1978). *Chubb et al.* (1978) reported significantly higher levels of AChE in plasma from premature calves and lambs compared to full term animals. High levels of pChE in newborn lambs compared to sheep have also been described by *Bell & van Petten* (1976). They found that pChE decreased in 2 days to ⅔ of the level at birth. On the contrary *Zsigmond & Downs* (1971) reported only 50—70 % pChE in newborn humans compared to adults. However, the differences between pChE in ruminants and in humans should be considered.

The influence of sex is only seen in pChE, not in eAChE (Fig. 3). It is known that testosterone influences the non-specific

esterases in mice (*Andersson & Tegelström* 1979). However, on the contrary to the results found here, *Schmidt & Schmidt* (1978) found that female rats had higher levels of pChE than male rats.

The cows suffering from parturient paresis had as expected low Ca and P values and a slight increase in Mg levels at parturition (Table 1). The normal parturient cows had a slight decrease in P and Ca but as mentioned earlier this is to be regarded as normal (*Hallgren et al., Luthman & Persson*). The significant lowering ($P \leqq 0.001$) of pChE after treatment with Ca might indicate a complication in the cholinergic system in the paretic cows.

The individual levels of pChE and eAChE seen in normal cows (Fig. 2a and b) made it important to follow the cows suffering from parturient paresis and the normal delivering cows with a blood sample 2—4 months later. The Ca, P and Mg levels in these samples were as expected normal (Table 1). The decrease in pChE after 2—4 months was significant ($P \leqq 0.001$) in the cows that had been suffering from parturient paresis but not in normal delivering cows. This adds further weight to the theory that there can be some disturbances in the cholinergic system of the paretic cows.

Bowen et al. (1970) found that the cow has a lower margin of safety of neuromuscular transmission compared to non-ruminants. This sensitivity in neuromuscular transmission might be individual and this may explan why some cows are able to behave normally on Ca levels that are lower than in cows suffering from parturient paresis. However, the significant increase in pChE is not likely the reason for such a sensitivity but a further sign of a disturbance in the neuromuscular transmission. In humans suffering from Alzheimer's disease pChE activity is about 100 % higher than in similar age controls and eAChE activity tended to be lower in patients than controls (*Smith et al.* 1982). These patients are suffering from loss of cholinergic neurons or disturbed cholinergic metabolism in the brain and are by some authors said to be cured by phosphatidylcholine (lecithin), the usual dietary form of choline (*Corkin* 1981). Contrary to humans the pChE in blood from cows is mainly AChE. If the pChE is reflecting the activity in the neuromuscular junction is not known for sure to day. However, *Fambrough et al.* (1982) have concluded that there is a high level of homology between the acetylcholinesterase of erythrocytes and of the

neuromuscular junction. Furthermore, *Kreutzberg & Tóth* (1974) demonstrated a pathway from the nerve cell to the endothelial cell that enables the neuron to communicate with the vasculature. The results in this study add further weight to the assumption that cholinergic activity might be reflected by the pChE levels in blood plasma.

The onset of lactation demands very much of the dairy cow. Beside a huge loss in Ca and P to the milk there among other things is a great need for acetate, the predominate carbon precursor for fatty acid synthesis in the ruminant mammary gland (*Bauman & Davis* 1974). Phosphatidylcholine (lecithin) is thought to be an integral part of the assembly of milk droplets (*Dils* 1977). Lecithin is synthesized in the liver (*MacIntosh & Collier* 1976), but to some extent also in a few other tissues, e.g. the mammary gland in ruminants (*Kinsella & Infante* 1974). The immediate source of acetyl groups for ACh is acetyl-CoA (*MacIntosh & Collier* 1974), in ruminants synthesized from acetate. Both lecithin and acetate are the main precursors to ACh (*MacIntosh & Collier* 1976, *Dreyfus* 1975). The elevated pChE levels found in cows suffering from parturient paresis might indicate a disturbance in the cholinergic system.

Consideration should likely be taken of these factors and not only failure in Ca-regulating mechanisms when parturient paresis is discussed. The decreasing blood plasma Ca level might just be a catalysing factor for the paralysis when the function of neuromuscular junctions in the cholinergic system is disturbed for some other reason.

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REFERENCES

Adams, H. R.: Neuromuscular blocking agents. In: Veterinary Pharmacology and Therapeutics. Eds. Meyer Jones, L., N. Booth & L. McDonald. The Iowa State University Press, Ames 1977, p. 165—187.

Andersson, M. & H. Tegelström: Effects of testosterone propionate and polychlorinated biphenyls (PCB) on the activity of nonspecific esterases in the house mouse. Hereditas 1979, *91*, 97—103.

Augustinsson, K.-B.: Electrophoresis studies on blood plasma esterases. Acta chem. scand. 1959, *13*, 571—592.

Augustinsson, K.-B. & H. Eriksson: The effects of two disulphides on cholinesterase activity in the spectrophotometric assay. Biochem. J. 1974, *139*, 123—127.

Augustinsson, K.-B., H. Eriksson & Y. Faijersson: A new approach to determining cholinesterase activities in samples of whole blood. Clin. Chim. Acta 1978, *89*, 239—252.

Bauman, D. E. & C. L. Davis: Biosynthesis of milk fat. In: Lactation Eds. Larsson, Bruce & Smith. Acad. Press 1974, p. 31—75.

Bell, J. U. & G. R. Van Petten: Plasma esterase development in the perinatal lamb. Biol. Neonate 1976, *30*, 232—237.

Borda, E., N. Contreras-Ortiz, R. Gutnisky & M. F. Gimeno: In vitro effect of acetylcholine and bethanechol on the contractions of the human detrusor muscle. Influence of prostaglandins. Arch. int. Pharmacodyn. 1982, *259*, 31—39.

Bowen, J. M., D. M. Blackmon & J. E. Heavner: Neuromuscular transmission and hypocalcemic paresis in the cow. Amer. J. vet. Res. 1970, *31*, 831—839.

Carlström, G.: Phosphorus, magnesium and pH in bovine blood serum normally, after parturition, and in parturient paresis. Acta vet. scand. 1961, *2*, 350—356.

Chubb, I. W., R. S. Comline, M. Silver & A. D. Smith: Acetylcholinesterase in bovine and ovine plasma: Is the concentration an index of foetal maturity? Proceedings of the Australien Physiological and Pharmacological Society 1978, *9*, 130 p.

Corkin, S.: Acetylcholine, aging and Alzheimer's disease. Implications for treatment. Trends in Neurosciences, Cambridge, U.K. 1981, p. 287—290.

Dils, R. R.: Mammary Glands. In: Lipid Metabolism in Mammals. 2. Ed. Fr. Snyder. Plenum Press, New York and London 1977, p. 131—144.

Dreyfus, P.: Identification de l'acetate comme précurseur du radical acétyle de l'acétylcholine des jonctions neuro-musculaires du Rat. (Identification of acetate as a precursor to the acetyl radical in acethylcholinesterase of neuromuscular junctions in the rat). C. R. Acad. Sc. Paris 1975, *280*, 1893—1894.

Edqvist, L.-E., H. Kindahl & G. Stabenfeldt: Release of prostaglandin $F_{2\alpha}$ during the bovine peripartal period. Prostaglandins 1978, *16*, 111—119.

Ellman, G. L., K. D. Courtney, V. Andres, Jr. & R. M. Featherstone: A new and rapid colorimetric determination of acetylcholinesterase activity. Biochemical Pharmacology 1961, 7, 88—95.

Fambrough, D. M., A. G. Engel & T. L. Rosenberry: Acetylcholinesterase of human erythrocytes and neuromuscular junctions: Homologies revealed by monoclonal antibodies. Proc. Natl. Acad. Sci. 1982, *79*, 1078—1082.

Fiske, C. H. & Y. S. Subbarow: The colorimetric determination of phosphorus. J. biol. Chem. 1925, *66*, 375—400.

Forslund, K., K. Lundström & L.-E. Edqvist: Equal calcitonin response in paretic and non-paretic cows after intravenous calcium infusion. Acta vet. scand. 1980, *21*, 171—184.

Hallgren, W., G. Carlström & G. Jönsson: Studies on parturient paresis in dairy cows. Nord. Vet.-Med. 1959, *11*, 217—249.

Hansen, J. L. & E. F. Freier: The measurement of serum magnesium by atomic absorption spectrophotometry. Amer. J. Med. Technol. 1967, *33*, 158—166.

Horst, R. L., J. A. Eisman, N. A. Ingensen & H. F. De Luca: Adequate response of plasma 1.25-dihydroxyvitamin-D to parturition in paretic (milk fever) dairy cows. Science 1977, *196*, 662—663.

Jönsson, G., B. Pehrsson, K. Lundström, L.-E. Edqvist & J. W. Blum: Studies on the effect of the amount of calcium in the prepartal diet on blood levels of calcium, magnesium, inorganic phosphorus, parathyroid hormone and hydroxyproline in milk fever prone cows. Zbl. Vet.-Med. A 1980, *27*, 173—185.

Kinsella, J. E. & J. P. Infante: Acyl-CoA-acyl-sn-glycerol-3-phosphorylcholine acyltransferase of bovine mammary tissue. Lipids 1974, *9*, 748—751.

Kowalczyk, D. F. & G. P. Mayer: Cation concentration in skeletal muscle of paretic and non paretic cows. Amer. J. vet. Res. 1972, *33*, 751—757.

Kreutzberg, G. W. & L. Tóth: Dendritic secretion: A way for the neuron to communicate with the vasculature. Naturwissenschaften 1974, *61*, 37.

Luthman, J. & J. Persson: Lipid mobilization in parturient cows. Acta vet. scand. 1975, *16*, 63—75.

MacIntosh, F. C. & B. Collier: Neurochemistry of cholinergic terminals. In: Neuromuscular Junction. Ed. E. Zaimis, Springer-Verlag, Berlin, Heidelberg, New York 1976, p. 99—228.

May, I., P. Pop & G. Roth: Serum cholinsterase in cattle: variation with age and the influences of disease and trichorphon therapy. Lucrari Stiintifice Institutul Agronomic Timisoara. Seria Medicina Veterinara 1978, *15*, 95—98.

Mayer, G. P., J. W. Blum & L. J. Deftos: Diminished prepartal plasma calcitonin concentration in cows developing parturient hypocalcemia. Endocrinology 1975, *96*, 1478—1485.

Skerry, D. W.: Di-(2 hydro-phenyl-imino)-ethane: A new indicator for the EDTA titration of serum calcium. Clin. Chim. Acta 1965, *12*, 593—597.

Schmidt, E. & F. W. Schmidt: Sex differences of plasma cholinesterase in the rat. Enzyme 1978, *23*, 52—55.

Smith, R. C., B. T. Ho, L. Hsu, G. Vroulis, J. Claghorn & J. Schoolar: Cholinesterase enzymes in the blood of patients with Alzheimer's disease. Life Sci. 1982, *30*, 543—546.

Soininen, H., T. Halonen & P. J. Riekkenen: Acetylcholinesterase activities in cerebrospinal fluid of patients with senile dementia of Alzheimer type. Acta neurol. scand. 1981, *64*, 217—224.

Zsigmond, E. K. & J. R. Downs: Plasma cholinesterase activity in newborns and infants. Canad. Anaesth. Soc. J. 1971, *18*, 278—285.

SAMMANFATTNING

Cholinesterashalten i blodplasma och erytrocyter hos kalvar, normala peripartala kor och kor med puerperal pares.

Cholinesteraskoncentrationen i blodplasma (pChE) samt acetylcholinesteraskoncentrationen i röda blodkroppar (eAChE) bestämdes hos 6 kor, före, under och efter kalvning (grupp I), hos deras kalvar (grupp II), hos 38 kalvningsförlamningskor (grupp III) samt hos 14 nykalvade kor som ej hade kalvningsförlamning (grupp IV).

Grupp I hade ett medelvärde av pChE på 1,5 μkat/l ± 0,20 före kalvning. Efter kalvningen sjönk pChE nivån signifikant ($p \leq 0.05$) till 1,2 μkat/l ± 0,16. Medelvärdet av eAChE var före kalvning $\simeq$ 140 μkat/l och sjönk till $\simeq$ 130 μkat/l 4—5 veckor efter kalvning.

Grupp II hade vid födelsen ett medelvärde av 12,8 μkat/l ± 5,9 pChE. Efter 4 veckor hade pChE värdet sjunkit till 2,3 μkat/l ± 0,3. Vid 6 veckors ålder började pChE nivån hos tjurkalvarna att stiga till 5,7 μkat/l vid 6 månaders ålder, då de slaktades. Kvigkalvarna låg kvar på en nivå av omkring 2 μkat/l hela undersökningstiden. Inga könsskillnader förelåg för eAChE-värdet, som vid födelsen var 119 μkat/l för att sedan stiga till 145 μkat/l vid 6 månaders ålder.

Grupp III hade före Ca-behandling ett medelvärde av pChE på 1,80 μkat/l ± 0,30. Detta värde sjönk signifikant ($p \leq 0.001$) till 1,67 μkat/l ± 0,29 efter behandlingen. Medelvärdet av pChE hos grupp IV låg på 1,65 μkat/l ± 0,42 vid förlossningen. Två—4 månader efter kalvningen hade både grupp III och IV ett pChE värde på 1,61—1,66 μkat/l.

Inga skillnader i eAChE värdena konstaterades. Dessa höll sig omkring 125 μkat/l hela tiden.

De slutsatser man kan dra av denna undersökning är att förlossning, ålder och kön påverkar pChE nivåerna i blodet hos kor och kalvar. Vidare att de förhöjda värdena av pChE funna hos kor med kalvningsförlamning kan vara en indikation på en störning av den neuromuskulära transmissionen i det cholinerga nervsystemet.

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