Acta vet. scand. 1984, *25*, 467—479.

From the Department of Biochemistry, College of Veterinary Medicine, Helsinki, Finland, and the Department of Animal Hygiene and Clinical Nutrition, College of Veterinary Medicine, University of Agricultural Sciences, Uppsala, Sweden.

TREATMENT OF BOVINE KETOSIS WITH INVERT SUGAR, GLUCOCORTICOIDS, AND PROPYLENE GLYCOL

By

Kauko Kauppinen and *Yrjö Gröhn*

KAUPPINEN, KAUKO and YRJÖ GRÖHN: *Treatment of bovine ketosis with invert sugar, glucocorticoids and propylene glycol.* Acta vet. scand. 1984, *25*, 467—479. — Four different treatments of bovine ketosis, using 3 different pharmaceutical preparations, were monitored. The main antiketogenic and glucogenic ingredients of the preparations were as follows: invert sugar (Metabol), prednisolone and dexamethasone (Predasen), the former preparations combined (Metabol & Predasen) and propylene glycol and dexamethasone (Dexaprol). Blood samples were drained from the ketotic cows prior to the treatment. The animals were sampled again 2 and 7 days after the first sampling. The whole blood concentrations of acetoacetate (AA concn), β-hydroxybutyrate (BHB concn) and glucose (gluc concn) were determined. The measures of the antiketogenic and glucogenic efficacy of the various treatments applied were their ability to reduce the AA concn and BHB concn and to elevate the gluc concn.

Invert sugar (Metabol) alone had no antiketogenic efficacy, although in some cases it led to a subjective clinical improvement as reported by the owners of the animals. Invert sugar and glucocorticoids (Metabol & Predasen) dexamethasone and prednisolone (Predasen) and dexamethason plus propylene glycol (Dexaprol) were equally effective in bringing the AA, BHB and gluc concns to normal range. The mean gluc concn of the cows treated with Dexaprol was higher than that of the cows in the other treatment groups at 2 days ($P < 0.01$). No other differences between the 3 antiketogenic treatments could be shown.

acetoacetate; β-hydroxybutyrate; blood glucose; clinical trial; dairy cows; Finland.

The treatment of bovine ketosis seems to be as complicated as this metabolic disorder itself. Various therapies have been recommended. Glucose and glucocorticoids, singly or together, have long been used satisfactorily for the treatment of severe acute

cases of bovine ketosis. Glucogenic substances, such as glycerol, sodium propionate and propylene glycol, have been shown to be effective in alleviating ketotic situations. Glycerol and sodium propionate unfortunately have undesirable side-effects (*Pehrson* 1972). Propylene glycol has a broad safety margin. *Kronfeld* (1976), however, has suggested that propylene glycol either increases the milk yield or acts as an antiketogenic substance, but not both.

The investigations of *Goetsch et al.* (1956) and *Kouider et al.* (1978) support the use of invert sugar for the therapy of bovine ketosis, since the half-life of glucose and fructose combined is longer than when either is used singly.

Primary bovine ketosis is very difficult to induce. The efficacy of various treatments should therefore be investigated under field conditions. Especially the instability of acetoacetate (AA) in blood, however, has limited field studies. *Työppönen & Kauppinen* (1980) have developed a standardized method for the treatment of blood samples, with particular regard for the stability and automatic determination of AA in blood samples obtained under field conditions.

In this study the antiketogenic efficacy of the 4 treatments for bovine ketosis widely used in Finland were monitored. The main antiketogenic ingredients of these preparations were invert sugar, glucocorticoids and propylene glycol. The measures of the antiketogenic efficacy of the various preparations used were their ability to reduce the whole blood concentration of AA (AA concn) and β-hydroxybutyrate (BHB concn) and to elevate that of glucose (gluc concn).

MATERIALS AND METHODS

Animals

A total of 50 Ayrshire dairy cows were obtained from commercial farms in different parts of Finland. The cows, ranging in age from 2 to 12 years, were treated 11—64 days after parturition by one of 4 different treatments according to a predetermined allotment. Since 7 cows were misdiagnosed and 3 cows needed further treatment after 2 d 10 of the 50 cows were excluded from further study. Each cow was sampled 2 (2d) and 7 days (7d) after the treatment.

Clinical diagnosis and sampling

Primary bovine ketosis was diagnosed initially by routine clinical examination. The clinical symptoms ranged from slight inappetence to nervous disorders. AA and acetone were detected in the milk/or urine of each cow with Ketostix (Ames). A blood sample was obtained from each cow before treatment and the clinical diagnosis was confirmed by clinical-chemical parameters. A cow was regarded as ketotic if the AA concn was measured to be over 1.05 mmol/l (*Kauppinen* 1983). According to this criterion, 7 cows had been clinically misdiagnosed and were excluded from further study.

Analysis

Heparinized whole blood 0.5 ml was immediately precipitated with 2.0 ml of 0.6 mmol/l perchloric acid and samples were frozen as soon as possible to ensure the stability of AA and BHB according to *Työppönen & Kauppinen* (1980).

The AA and BHB concns were determined by a Gilford 3500 Computer Directed Analyzer (Gilford Instrument, Inc., Oberlin, Ohio, U.S.A.) according to the method of *Työppönen & Kauppinen* (1980). The gluc concn was determined by a standard Gilford method GOD-PAP (Trinder method) with Boehringer reagents (Boehringer Corp. Ltd.).

Preparations and treatments

T r e a t m e n t g r o u p 1. Dexaprol (D) is a combination product containing cobalt chloride 1.5 mg, dexamethasone 3 mg and propylene glycol ad 100 ml. Twelve cows were treated with 1000 ml of D. The dosage was 200 ml twice a day orally.

T r e a t m e n t g r o u p 2. Metabol (M) is composed of fructose 25 g, glucose 25 g, acetylmethionine 2 g, and thiamine chloride 50 mg in 100 ml water. The dosage was 400 ml administered i.v. within 5 min. Seven animals were treated.

T r e a t m e n t g r o u p 3. Predasen (P) is a combination of dexamethasone pivalate 2.5 mg resp. dexamethasone 2.06 mg and prednisolone 7.5 mg in 1 ml water. Eight ml of Predasen was administered i.m. to 12 cows.

Treatment group 4. Metabol and Predasen combined (M & P). Nine cows were treated with a dose of 400 ml of M i.v. as in group 2 and 8 ml of P i.m.

All the above preparations are the products of Orion Pharmaceutical Company, Espoo, Finland.

Statistical methods

Changes in the clinical-chemical parameters among treatment groups were analyzed using Friedman two-way analysis of variance (*Siegel* 1956). The differences between the means of the 4 treatment groups at various sampling times was evaluated pairwise by Student's t-test.

RESULTS

There only was a weak association between the severity of the clinical symptoms on the one hand and the AA, BHB and gluc concn on the other. The highest AA concn and BHB concn and the lowest gluc concn were not measured for the cows with nervous symptoms. Only two cases of ketosis with nervous signs, however, were found in this study.

The mean AA concn in the various treatment groups before and after the treatment is given in Fig. 1. There were no significant differences between the initial mean levels of the AA concn of the various groups. The individual values ranged from 1.06 to 4.30 mmol/l.

In the second set of blood samples the mean AA concn was statistically significantly ($P < 0.001$) lower than in the initial set in all groups except in group 2. The AA concns were as follows: Group 1: (mean ± s) 0.58 ± 0.36 mmol/l; range 0.08—1.08 mmol/l, Group 2: 1.87 ± 1.32 mmol/l; range: 0.51—4.07 mmol/l, Group 3: 0.70 ± 0.14 mmol/l; range 0.36 ± 0.93 mmol/l, Group 4: 0.66 ± 0.23 mmol/l; range 0.23—1.01 mmol/l. The mean AA concn in group 2 at 2d was significantly ($P < 0.01$) higher than in any of the other groups. The mean AA concn of the third set of samples was even lower ($P < 0.01$) than that of the second set in treatment group 3 (0.24 ± 0.16 mmol/l; range: 0.11—0.61 mmol/l) and group 4 (0.32 ± 0.48 mmol/l); range: 0.05—1.58 mmol/l), but not in group 1 (0.54 ± 0.59 mmol/l; range: 0.09—1.61 mmol/l). The mean AA concn was lower but not significantly so in the third set of samples than in the second ones in treat-

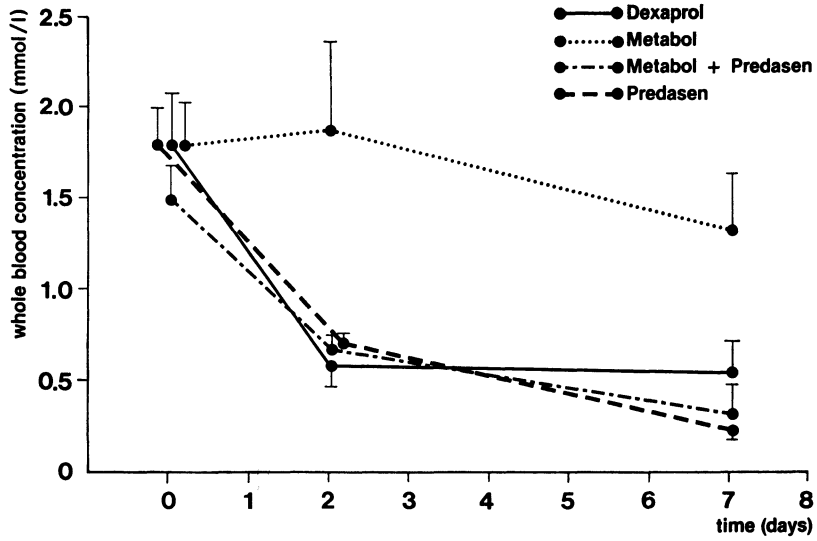

Figure 1. Acetoacetate whole blood concentration in 4 treatment groups before and after treatment (mean ± s.e.m.).

ment group 2 (1.33 ± 0.82 mmol/l; range: 0.25—2.67 mmol/l). The mean AA concn of group 2 was significantly ($P < 0.05$) higher than that in the other groups in the third set of samples.

What happened to the mean BHB concn in the various treatment groups is shown in Fig. 2. The pre-treatment mean BHB concn of the different treatment groups of clinically ketotic cows varied from 3.09 to 4.14 mmol/l. There were no significant differences in the mean intial values between the various treatment groups. In treatment group 1 the mean BHB concn decreased significantly ($P < 0.05$), to 1.58 ± 0.90 mmol/l (range: 0.25—2.83 mmol/l) in the first post-treatment blood samples and to 1.52 mmol/l (range: 0.46—4.01 mmol/l) in the second ones. In treatment group 2 the mean BHB concn decreased, but not significantly so, t 3.39 ± 1.54 mmol/ l(range: 1.51—5.15 mmol/l) and then to 2.91 ± 1.61 mmol/l (range: 1.08—5.41 mmol/l). The BHB concn decreased highly significantly ($P < 0.001$) in group 3. In the samples obtained at 2 d, the mean BHB concn was 2.41 ± 0.97 mmol/l (range: 1.14—4.62 mmol/) and at 7 d 1.14 ± 0.46 mmol/l (range: 0.49—1.99 mmol/l). In group 4, the mean BHB concn decreased to 2.03 ± 0.73 mmol/l (range: 1.14—3.20 mmol/l) at 2 d and then to 1.06 ± 0.78 mmol/l (range: 0.47—2.84 mmol/l) at 7 d ($P < 0.001$). The mean BHB concn in group 2 was higher

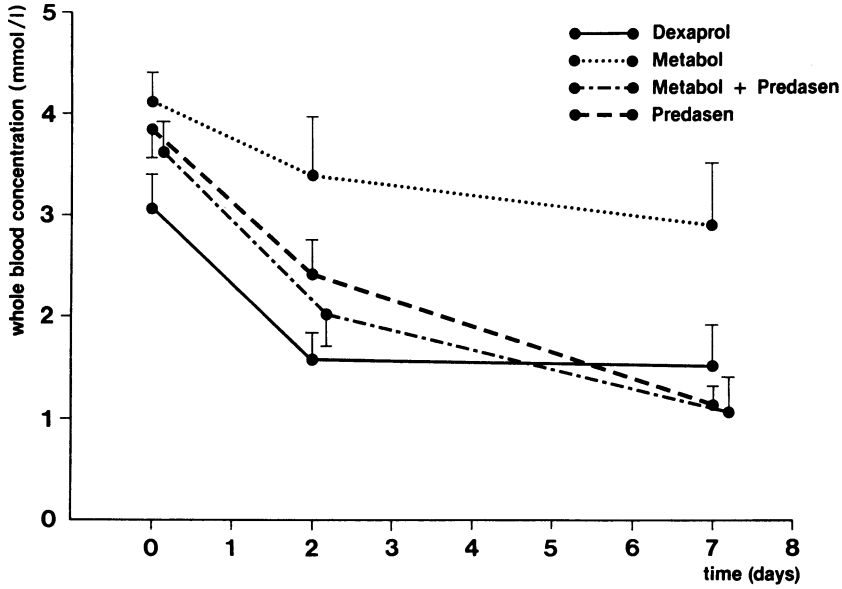

Figure 2. β-hydroxybutyrate whole blood concentration in 4 treatment groups before and after treatment (mean ± s.e.m.).

than in the other groups at the $P < 0.01$ level of significance at 2 d and 7 d. There were no significant differences in the BHB concn between the other groups.

Fig. 3 shows the mean gluc concn before and after the treatments in the various treatment groups. The initial values of the clinically ketotic cows varied from 1.2 to 2.7 mmol/l. The initial mean gluc concn in group 3 was lower than in the other groups ($P < 0.05$). The mean gluc concn in the samples of group 1 at 2 d was significantly ($P < 0.001$) higher than the corresponding initial (5.94 ± 2.21 mmol/l; range: 3.4—10.2 mmol/l) level. The mean was statistically significantly higher than that of the other groups at the same sampling time ($P < 0.01$). At 7 d the gluc concn was 3.04 ± 0.71 mmol/l (range: 1.8—4.4 mmol/l), which did not differ significantly from the corresponding means of the other groups. The gluc concn remained at the same level as in the initial sampling (2.26 ± 0.28 mmol/l; range: 2.0—2.7 mmol/l) in both the second sampling (2.41 ± 0.58 mmol/l; (range: 1.20—3.90 mmol/l) and in the third one (2.51 ± 0.58 mmol/l; range: 1.90—3.70 mmol/l) in group 2. In group 3 the mean gluc concn at 2 d was found to be 3.13 ± 0.60 mmol/l (range: 1.90—4.10 mmol/l). The increase from the initial level was highly significant

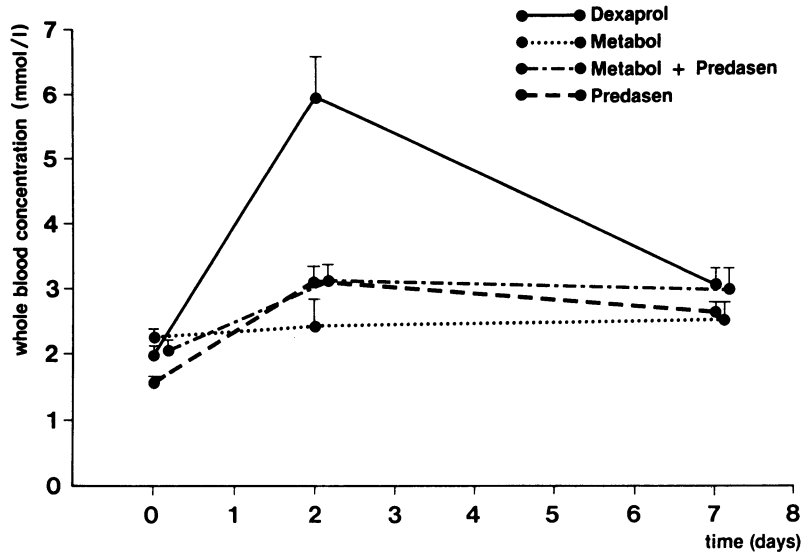

Figure 3. Glucose whole blood concentration in 4 treatment groups before and after treatment (mean ± s.e.m.).

($P < 0.001$). At 7 d the mean gluc concn was 2.65 ± 0.49 mmol/l (range: 1.50—3.30 mmol/l). In group 4 the mean gluc concn increased almost significantly at 2 d ($P < 0.05$) from the pre-treatment level to 3.12 ± 0.63 mmol/l (range: 2.10—4.10 mmol/l). The mean gluc concn in this group was at almost the same level (3.00 ± 0.89 mmol/l; range: 1.10—4.10 mmol/l) at 7 d. There were no significant differences in the mean gluc conc between groups 2, 3 and 4 at 2 d and 7 d.

DISCUSSION

The mean AA concn and BHB concn measured at 2 and 7 d in treatment group 2 were not statistically significantly lower than the pre-treatment means. The invert sugar of M did not give an antiketogenic response. This was to be expected on the basis of the literature on bovine ketosis. *Bergman & Roberts* (1967) have shown that the infusion of gluc reduced the blood concentration of ketone bodies only temporarily. The rapid infusion of invert sugar within 5 min did not seem to reduce the rate of hepatic ketogenesis. In addition to invert sugar M contained methionine and thiamine. Both of them have been used separately for the treatment of bovine ketosis. *McCarthy et al.* (1968) have shown

methionine to be of benefit in the treatment of ketosis. The single dose of 8 g of acetylmethionine in M did not reduce the AA concn or the BHB concn. Thiamine has been regarded as a stimulant of appetite and has been used in the treatment of bovine ketosis as well (*Kronfeld & Emery* 1970). The 200 mg of thiamine chloride in M did not reduce either the AA concn or the BHB concn.

Goetsch et al. (1956) have shown that there was less sugar loss in urine when fructose or invert sugar was employed than when glucose was used alone. They concluded that both fructose and invert sugar are more rapidly utilized by hepatic or extrahepatic tissues than glucose alone. According to the authors this suggests a possible advantage of fructose or invert sugar in the treatment of ketosis. In the present study the mean gluc concn of the cows treated with M at 2 and 7 d was not statistically significantly higher than the pre-treatment mean. This was predictable on the basis of the work of *Goetsch et al.* (1956). They showed that the total blood sugar values in healthy cows returned to normal 2 h following invert sugar treatment. *Bergman & Roberts* (1967) have shown that the gluc concn was at the pretreatment level 2 and 7 days after treatment with glucose infusion. It is not known how soon this level was reached, because only 2 post-treatment blood samples were taken. Nevertheless, the recovery of a cow from ketosis and the probability of relapse was studied at 2 and 7 days post-treatment in the present study. The intervals were considered logical for the evaluation of the results of the treatments. Methionine and thiamine seemed not to influence the gluc concn either. A single infusion of invert sugar, although combined with methionine and thiamine, is thus not a sufficient treatment of bovine ketosis. Invert sugar might be regarded as the drug of choice for correcting hypoglycemia of bovine ketosis, but the need of recurrent infusion is evident just as the case when glucose is administered (*Kronfeld & Emery* 1970). At 7 d the mean gluc concn in this group did not differ from that in the other ones.

It is of interest to notice that 3 of the 7 ketotic cows treated with M were regarded by owners as healthy at 7 d. They were eating normally. On the other hand 3 of the cows were really in bad form and AA concns higher than the pre-treatment levels were measured for them. They had completely lost their appetite and needed further treatment immediately. The individual response to this treatment seemed to be extremely variable. Four

out of 7 individual AA concns were lower than the pre-treatment levels as were 6 out of 7 BHB concns. AA concn within normal range was even measured for 1 cow treated with M at 7 d. Accordingly it was difficult to judge which factors are involved in inappetence of a cow during ketosis. *McClymont & Setchell* (1956) and *Reid* (1968) have proposed hypoglycemia, ketonemia or some other metabolic disorder as probable causes of inappetence. The AA and BHB concns of cows with inappetence were higher than the pre-treatment levels and gluc concn lower. Which of these factors caused the inappetence thus remains obscure. *Hikosaka et al.* (1979) have noticed that neither an increase nor a decrease of plasma glucose affected the short-term food intake in sheep. Some other factors e.g. hormonal regulation might be involved.

The infusion of invert sugar raising temporarily the blood sugar concentration did not seem to have an immediate influence on the appetite of the cows. None of those treated with M ate better during the 24 post-treatment hours than before the treatment. The return of appetite of 3 cows in this group might be due to methionine and/or thiamine. *Schultz* (1959), however, stated that some ketotic cows recover without any treatment and they start to eat gradually. This may be due to stimulated adrenocortical excretion.

The mean AA concn in groups 1, 3 and 4 at 2 d were highly significantly ($P < 0.001$) lower than before the treatment. So was the mean BHB concn in groups 3 and 4 ($P < 0.001$) and in group 1 ($P < 0.05$). The mean AA concn and BHB concn, however, were not at the normal level. *Baird & Heitzman* (1971) made the same finding following glucocorticoid treatment of bovine ketosis. The pre-treatment levels of the AA concn and BHB concn in each treatment group were the same. The only exception was the mean BHB concn of group 2, which was higher than that of group 1 ($P < 0.05$). But this does not affect the interpretation of the results of the treatment. The mean BHB concn of group 1 was lower than of group 3 at 2 d ($P < 0.05$). The mean AA and BHB concn of group 2 were higher ($P < 0.01$) than those of the other groups at 2 and 7 d. This shows that M alone is less antiketogenic than the other treatments. There were no significant differences between the other groups in the mean AA and BHB concn at 2 and 7 d. As far as the results of various treatments are concerned it may be of importance that the standard deviations of the AA

concn were reduced considerably by the treatment in groups 1, 3 and 4, but increased in group 2.

The mean AA concn and BHB concn of groups 3 and 4 were lower in the blood samples taken at 7 d than in those taken at 2 d. In group 1 the means were at the same level in both samples. This may be a sign of future relapses in this group.

In group 1 the mean gluc concn at 2 d was highly significantly ($P < 0.001$) higher than the pre-treatment mean and significantly ($P < 0.01$) higher than the means of the other groups. Glucocorticoid and propylene glycol of D seemed to elevate the gluc concn to a hyperglycemic level. In this respect D was superior to the other treatments. 20 mg of dexamethasone pivalate with 80 mg of prednisolone in single i.m. dose of P seemed not to have the same glucogenic effect as 30 mg of dexamethasone in propylene glycol given orally, according to the gluc concn at 2 d. According to the results of *Stöckl et al.* (1969), dexamethasone plus prednisolone elevated the blood sugar concentration above the normal level faster and remained at the higher level longer than dexamethasone alone. Since the gluc concn in the treatment group 1 was significantly higher than that in groups 3 and 4 at 2 d it seems probable that the hyperglycemic level reached with D was due to the treatment of 3 days duration with dexamethasone and propylene glycol. When administration was terminated at 3 d the gluc concn came down, as measured at 7 d. Propylene glycol seemed to be effective in the treatment of bovine ketosis, as *Johnson* (1954) and *Pehrson* (1972) have also shown. The treatments M & P and P also had a significant glucogenic effect at 2 d respectively, although it was not as extensive as with D.

The efficacy of glucocorticoid therapy in the treatment of bovine ketosis has once more been demonstrated in this study. The action mechanism of glucocorticoids in the treatment of bovine ketosis has been thoroughly investigated by many researchers, among others by *Baird & Heitzman* (1971). They have shown that the hepatic concentrations of all intermediates of the citric acid cycle increased following the administration of glucocorticoids. The mobilization of amino acids from muscle tissue is of great significance in this respect. Propylene glycol had a clear positive effect when used with dexamethasone. A certain tendency toward a relapse of ketosis, however, was seen when D was used. When the invert sugar was combined with the glucocorticoid treatment (M & P) the clinical recovery was faster than

among cows treated with glucocorticoids alone (P). In the former group cows began to eat earlier than in the latter. The individual response to each treatment seemed to be variable. A certain tendency towards a relapse of ketosis could be seen in the clinical-chemical parameters of some cows in each group. It was of significance that any restrictions on milking or alteration of feeding were not applied in the present study. There may be individual variations in the susceptibility to the relapse of bovine ketosis according to the level of milk yield of a cow. This might explain the wide individual variations in the values of the parameters analysed.

ACKNOWLEDGEMENTS

We wish to thank professor Paul Lindberg, Department of Biochemistry, and professor Timo Pekkanen, Department of Food Hygiene, College of Veterinary Medicine, Helsinki, Finland, and associate professor Juhani Hakkarainen, Department of Animal Hygiene and Clinical Nutrition, College of Veterinary Medicine, Uppsala, Sweden, for their help and advice while preparing this work; also the Orion Pharmaceutical company, for providing pharmaceutical preparations used in this study.

REFERENCES

Baird, G. D. & R. J. Heitzman: Mode of action of a glucocorticoid on bovine intermediary metabolism. Possible role in controlling hepatic ketogenesis. Biochim. biophys. Acta 1971, *252*, 184—198.

Bergman, E. N. & S. J. Roberts: Urinary glucose excretion and blood concentrations in ketotic cattle after treatment with glucose. Cornell Vet. 1967, *57*, 624—629.

Goetsch, D. G., M. S. Gravers, K. L. Underbjerg & M. J. Swenson: The utilization of intravenously administered glucose, invert sugar and fructose in cattle. Amer. J. vet. Res. 1956, *17*, 213—216.

Hikosaka, K., Y. Sasaki & T. Tsuda: Effects of glucose, insulin and FFA in food intake in sheep. Ann. Rech. Vet. 1979, *10*, 237—239.

Johnson, R. B.: The treatment of ketosis with glycerol and propylene glycol. Cornell Vet. 1954, *44*, 6—21.

Kauppinen, K.: Prevalence of bovine ketosis in relation to number and stage of lactation. Acta vet. scand. 1983, *24*, 349—361.

Kouider, S., F. E. Kolb, I. Müller & K. Pfüller: Untersuchungen über das Verhalten verschiedener Blutbestandteile (Glukose, Fruktose, Insulin, Laktat, Pyruvat, freie Fettsäuren, anorganisches Phosphat) und über die Halbwertzeit der Monosaccharide im Blutplasma nach i.v. Infusion von Glukose-, Fruktose-, Galaktose- und Invertzuckerlösung bei Wiederkäuern. [Studies into behaviour of several blood components (glucose, fructose, insulin, lactate, pyruvate, free fatty acids, and inorganic phosphate)

and into half-life of monosaccharides in blood plasma following intravenous infusion to ruminants of glucose, fructose, galactose, and invert sugar solutions. 2nd communication: Testing of cattle]. Arch. exper. Vet.-med. 1978, *32*, 699—714.

Kronfeld, D. S. & R. S. Emery: Acetonemia. *In* Bovine Medicine and Surgery and Heard Health Management. Ed. W. J. Gibbons, E. J. Catcott, and J. F. Smithcors. Amer. Vet. Publ., Wheaton, Ill. 1970, p. 350—376.

Kronfeld, D. S.: The potential importance of glucogenic, lipogenic and aminogenic nutrients in regard to the health and productivity of dairy cows. Fortschritte in der Tierphysiologie und Tierernährung 1976, *7*, 5—26.

McCarthy, R. D., G. A. Porter & L. C. Griel Jr.: Bovine ketosis and depressed fat test in milk: a problem of methionine metabolism and serum lipoprotein aberration. J. Dairy Sci. 1968, *51*, 459—462.

McClymont, G. L. & B. P. Setchell: Insulin induced hypoglycemic encephalopathy in the sheep and its implication as regards pathogenesis of the disease. Aust. vet. J. 1956, *32*, 97—109.

Pehrson, B.: The effect of orally administered glycogenic substance to dairy cows. III. Nord. Vet.-Med. 1972, *24*, 423—426.

Reid, R. L.: The physiopathology of undernourishment in pregnant sheep, with particular reference to pregnancy toxemia. Adv. Vet. Sci. 1968, *12*, 163—238.

Schultz, L. H.: Treatment of ketosis in dairy cattle with sodium propionate. Cornell Vet. 1952, *42*, 148—155.

Siegel, S.: Nonparametric Statistics. M. C. Graw-Hill International Book Company Tokyo, 1956.

Stöckl, W., K. Onderscheka & M. K. Zacherl: Zur Frage des Eintritts und der Dauer von Corticoidwirkungen. (Rapidity and duration of the effect of corticoid preparations). Wien. tierärztl. Mschr. 1969, *56*, 229—232.

Työppönen, J. & K. Kauppinen: The stability and automatic determination of ketone bodies in blood samples taken in field conditions. Acta vet. scand. 1980, *21*, 55—61.

SAMMANFATTNING

Behandling av ketos med invert socker, glukokortikoider och propylen glykol

Fyra olika behandlingsmetoder av ketos undersöktes. Tre farmaceutiska preparat användes i behandlingen. Preparatens huvudsaklika antiketogena och glukogena beståndsdelar var följande: invertsocker (Metabol), prednisolon och dexametason (Predasen), de föregående preparaten tillsammans (Metabol och Predasen) och propylen glykol och dexametason (Dexaprol). Före behandlingen togs blodprov från korna. På den andra och sjunde dagen efter den första provtagningen omtogs blodprov från djuren. Koncentrationen i blodet av acetoacetat (AA), β-hydroxybutyrat (BHB) och glukos (gluc) bestämdes.

Förmågan att sänka koncentrationen av AA och BHB i blodet och att höja gluc koncentrationen användes som mått på antiketogen och glukogen effekt hos de olika behandlingsmetoderna.

Invertsocker (Metabol) allena hade ingen antiketogen effekt, ehuru det gav en subjektiv klinisk förbättring enligt djurens ägare i några fall. Invertsocker och glukokortikoider (Metabol och Predasen), dexametason och prednisolon (Predasen) och dexametason plus propylen glykol (Dexaprol) var lika effektiva när det gällde att få blodkoncentrationen av AA, BHB, och gluc till normal nivå. Dexaprol ökade blodkoncentrationen av gluc mera än de andra behandlingsmetoderna ($P < 0.01$) 2 dagar efter behandlingen. Ingen annan skillnad mellan de 3 antiketogena behandlingarna kunde påvisas.

(Received February 9, 1984).

Reprints may requested from: Kauko Kauppinen, the Department of Biochemistry, College of Veterinary Medicine, Hämeentie 57, 00550 Helsinki 55, Finland.