Zeitschrift: Schweizer Archiv für Tierheilkunde SAT : die Fachzeitschrift für Tierärztinnen und Tierärzte = Archives Suisses de Médecine Vétérinaire ASMV : la revue professionnelle des vétérinaires

Herausgeber: Gesellschaft Schweizer Tierärztinnen und Tierärzte

Band: 138 (1996)

Heft: 7

Artikel: Post surgical convalescence of dairy cows with left abomasal displacement in relation to fatty liver

Autor: Rehage, J. / Mertens, M. / Stockhofe-Zurwieden, N.

DOI: https://doi.org/10.5169/seals-592315

[1] *Clinic for Diseases of Cattle,* [2] *Department of Pathology,* [3] *Department of Physiology, Veterinary School of Hannover*

Post surgical convalescence of dairy cows with left abomasal displacement in relation to fatty liver

J. Rehage[1], M. Mertens[1], N. Stockhofe-Zurwieden[2], M. Kaske[3], H. Scholz[1]

Summary

Blood parameters, feed intake and milk yield were determined in 53 cows with a left displacement of the abomasum (LDA) on the day of surgery (ds; laparotomy and omentopexy) and during the following four days (d1-d4). Using histological methods severe (group SF), moderate (group MF) or no/mild (group NF) fatty liver was found in 32%, 40% and 28% of the patients, respectively. Moderate and severe fatty liver were found almost exclusively in cows in the first three weeks post partum. Post surgery, feed intake and daily milk yield increased steadily in cows of the NF- and MF-group; in cows suffering from severe fatty liver feed intake remained low ($p < 0.05$). On ds, mean serum levels of nonesterified fatty acids (NEFA), beta-hydroxybutyrate (BHB), total bilirubin, aspartate aminotransferase (ASAT), gamma-glutamyl transpeptidase (GGT) and glutamic dehydrogenase (GLDH) in SF-cows were significantly ($p < 0.05$) higher and values of cholesterol significantly lower ($p < 0.05$) as compared to the NF- and MF-group; no significant differences were found between the groups in mean serum glucose concentrations. In the four day period following surgery, in all groups mean serum levels of ASAT, GGT, GLDH and cholesterol remained nearly unchanged, whereas total bilirubin, NEFA, BHB and glucose decreased significantly ($p < 0.05$). Apart from LDA, 55% of the patients were suffering from mastitis, endometritis or lameness. Within three weeks post surgery, 3 cows of the SF-group and 1 cow of the MF-group developed recumbency and liver coma, and were culled for that reason. In conclusion, post surgical convalescence of cows with LDA is clearly re-

Postoperative Rekonvaleszenz von Kühen mit linksseitiger Labmagenverlagerung und Leberverfettung

Es wurden 53 HF-Kühe mit linkseitiger Labmagenverlagerung (LMV) in der rechten Flanke laparotomiert und die Labmagenverlagerung durch Omentopexie dauerhaft beseitigt. Die Patienten wurden am Tag der Operation (ds) sowie über weitere 4 Tage (d1-d4) untersucht. 32% der untersuchten Kühe wiesen in Leberbioptaten histologisch eine hochgradige (Grp. H), 40% eine mittelgradige (Grp. M) sowie 28% keine oder geringgradige Leberverfettung (Grp. G) auf. 74% der untersuchten Kühe befanden sich in den ersten drei Wochen post partum; diese Kühe litten häufiger an schweren Leberverfettungen ($p < 0.05$) als Patienten in späteren Laktationsstadien. Innerhalb der 4 Tage post operationem wurden keine nennenswerte Veränderungen im Leberfettgehalt festgestellt. Während in Grp. H die tägliche Futteraufnahme postoperativ unverändert gering blieb, stieg diese in Grp. M und Grp. G stetig an ($p < 0.05$). In allen Gruppen war postoperativ im Durchschnitt ein vergleichbarer Anstieg der Milchleistung zu beobachten. An ds waren bei den Patienten der Grp. H im Vergleich zu denen in Grp. M und Grp. G die Serumkonzentrationen von NEFA und BHB am höchsten ($p < 0.01$). Die Serum-Glucosekonzentrationen waren bei den Patienten aller Gruppen vergleichbar hoch. Die Serumspiegel von Gesamtbilirubin (GB) sowie der Aspartat-Aminotransferase (ASAT), der Gamma-Glutamyltranspeptidase (GGT) und der Glutamatdehydrogenase (GLDH) waren im Mittel in Grp. H höher ($p < 0.05$), die von Cholesterin niedriger ($p < 0.05$) als in Grp. M und Grp. G.

lated to disturbances of energy metabolism and fatty liver. Therefore, successful treatment of cows suffering from LDA requires the effective treatment of excessive lipomobilization, ketosis and fatty liver along with the immediate surgical correction of LDA.

Key words: **left displacement of the abomasum – fatty liver – ketosis – fat cow syndrome**

Während die mittleren Serumspiegel von ASAT, GGT, GLDH und Cholesterin bis d4 unverändert blieben, sanken diejenigen von GB, NEFA, BHB und Glucose in allen Gruppen ab ($p < 0.01$). 55% aller Patienten mussten zusätzlich wegen Mastitis, Endometritis oder Lahmheit behandelt werden. Innerhalb von drei Wochen post operationem wurden 6 Patienten verwertet: 3 Kühe aus Grp. H sowie eine Kuh aus Grp. M aufgrund eines Leberkomas, 2 Kühe wegen Euterentzündung. Die Ergebnisse zeigen, dass bei Kühen mit LMV die postoperative Rekonvaleszenz massgeblich vom Grad der Leberverfettung und der Lipomobilisation sowie den zusätzlichen Erkrankungen beeinflusst wird. Eine erfolgreiche Behandlung der Labmagenverlagerung muss daher neben der unverzüglichen Operation auch die Behandlung des Lipomobilisationssyndroms einschliessen.

Schlüsselwörter: **Linksseitige Labmagenverlagerung – Leberverfettung – Ketose – Lipomobilisationssyndrom**

Introduction

During early lactation in high-producing dairy cows, regular energy intake does not meet the energy requirement for milk production, resulting in body fat mobilization (Morrow, 1976; Stöber and Dirksen, 1982). Excessive lipomobilisation leads to enhanced production of ketone bodies and accumulation of fat - in particular triglycerids (TGL) - in the liver cells (Stöber and Dirksen, 1982; vandenTop et al., 1995); the milk yield decreases, and fertility as well as immunocompetence are affected, leading to a higher risk for infectious diseases such as endometritis, mastitis or lameness (Morrow et al., 1979; Gerloff et al., 1986; Herdt, 1991). The development of liver failure may even cause the loss of the animal in severe cases (Stöber and Dirksen, 1982; West, 1990; Muylle et al., 1992).

Another common disease of high-producing dairy cows in early lactation is the left displacement of the abomasum (LDA). The causes for LDA have been a subject of controversy (Breukink, 1990; Geishauser, 1995). However, a typical clinical sign is the loss of appetite and thereby decreased energy intake resulting in drastically enhanced lipomobilization and often severe fatty liver (Herdt et al., 1982; Holtenius and Niskanen, 1985). Thus, the immediate surgical correction of LDA is important in improving the general condition and thereby the feed intake of cows suffering from LDA. However, in our clinic we have observed that despite successful surgical correction of LDA the general condition of several cows remained poor during the post surgical period. It was the objective of this study to investigate whether post surgical convalescence of dairy cows with LDA is related to the extent of fatty liver.

Animals, material and methods

Fifty-three cows (Holstein-Frisian; mean age: 5.4±1.5 years; mean body weight: 554±64 kg) suffering from LDA admitted to our Clinic were randomly choosen for this investigation. According to the owner information clinical signs were present on average of 2.5±1.2 days before referral. On the day after admission to the clinic, surgical correction of LDA was performed on all cows after local anaesthesia via laparotomy in the right flank in a standing position. Reposition of the abomasum and omentopexy were carried out according to Dirksen (1967).

The body condition score (BCS) was estimated according to Edmonson et al. (1989). The animals were fed twice daily (7.00 and 15.00 h) with hay, concentrates, wheat bran and beets. Feed consumption and milk yield (kg/day) were determined on the day of surgery, on the second and on the fourth day post surgery. Energy intake was calculated as MJ NEL per day.

On the day of surgery (dS) and 4 days later (d4) blood samples were taken from the jugular vein prior to morning feeding. After 30 minutes the serum was separated via centrifugation (10 min, 3000 x g) and stored until analysis at -82 °C. Using commercial test kits total bilir-

ubin, aspartate aminotransferase [ASAT], gamma-glutamyltranspeptidase [GGT], glucose, cholesterol (Hoffmann-La Roche, Basel, Switzerland), glutamic dehydrogenase [GLDH] (Boehringer Mannheim, Mannheim, Germany), nonesterified fatty acids ([NEFA]; Wako Chemicals, Neuss, Germany) and beta-hydroxybutyrate ([BHB]; Sigma, Deisenhofen, Germany) were measured in serum using an automated analysing system (Cobas Mira®; Hoffmann La-Roche, Basel, Switzerland).

Liver samples were taken percutaneously in the 10th intercostal space using Tru-Cut®-biopsy needles (15 cm cannula; 14G; Travenol Laboratories, USA; Scholz et al., 1989) on dS and d4. One bioptate was formalin fixed for haematoxylin-eosin staining and a second one was frozen in liquid nitrogen for oil-red staining. Liver fat content was classified histologically into different degrees ranging from 0 (no fat droplets visible) to 5 (panlobular fatty infiltration) according to Mertens (1992). Thereafter, patients were assigned to three groups according to the degree of fatty liver on dS: no/mild (degree 0/1), moderate (degree 2/3) and severe (degree 4/5) fatty liver. Liver glycogen content was determined histologically via PAS-staining (Mertens 1992). The glycogen content was classified into different degrees, ranging from 0 (no glycogen) to 5 (high glycogen content). Additionally, liver bioptates were used for further histological and enzyme-histochemical investigations. Results are described in detail elsewhere (Mertens, 1992; Rehage et al., 1992; Mertens et al., 1996).

All cows were treated routinely on dS with 150 g glucose intravenously, and on the following days b. i. d. with 100 g sodium propionate orally.

Statistical analysis of results was performed on a level of significance of $p < 0.05$ using the statistic programm SAS (SAS user's guide 1985). Laboratory data were log-normal distributed. Ordinal data were subjected to Wilcoxon-Rank-Test, frequencies were tested by FISCHER's EXACT-test. A two-factorial analysis of variance (factor: time; grouping factor: liver fat content; GLM procedure for repeated measures) was used for continuous data. In cases where the ANOVA modell revealed a significant effect, a modified t-test (Bonferroni) was performed as a consecutive test. Results are given as means ± SEM.

Results

On the day of surgery (ds), 32% of the 53 investigated cows with LDA were suffering from severe fatty liver (fig. 1). A moderate fatty liver was found in 40%, and a mild fatty liver in 28% of the cases. Four days after surgical correction of LDA, the extent of fatty infiltration of the liver was almost unaffected (fig. 1). LDA developed in 74% of the patients within three weeks post partum (fig. 2). These cows showed on average higher degrees of fatty liver ($p < 0.01$) than cows in later lactational stages. Depending on the degree of fatty liver the liver glycogen content decreased ($p < 0.05$; table 2). In the 4 day period following surgery no significant changes in liver glycogen were found.

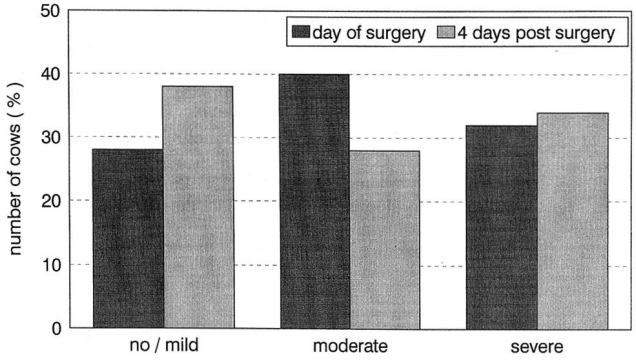

Figure 1: Prevalence of fatty liver in dairy cows with left displacement of the abomasum (N = 53) on the day of surgery and 4 days post surgery

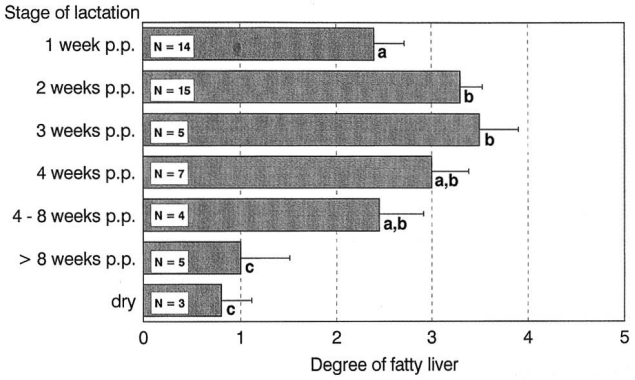

Figure 2: Degree of fatty liver related to the stage of lactation in dairy cows (N = 53) with left displacement of the abomasum (means with different superscripts are significantly different; $p < 0.05$)

Feed intake (table 1) was markedly reduced in all cows on the day of surgery. In cows with mild or moderate fatty liver, feed intake increased steadily during the following days ($p < 0.01$) while in cows with severe fatty liver feed intake remained low ($p < 0.05$). Daily milk yield increased steadily independent of the degree of fatty liver from ds to d4 (table 1).

Results of clinical biochemistry are shown in table 2. On ds the concentration of total bilirubin and the activities of ASAT, GGT and GLDH in serum were on average significantly higher ($p < 0.05$) in cows with severe fatty liver, than in cows with mild or moderate fatty liver. Whereas the mean concentration of bilirubin decreased until d4 ($p < 0.01$) the serum activities of the liver enzymes remained nearly unchanged.

On ds mean serum concentrations of NEFA and BHB were significantly higher ($p < 0.01$) in cows with severe fatty liver than in the other ones. No statistically significant differences were found for the mean values of glucose whereas cholesterol was lower ($p < 0.05$) in cows with moderate or severe fatty liver as compared to those with mild fatty liver. During the 4 days post surgery, chol-

esterol remained nearly unchanged in all groups, whereas the serum concentrations of NEFA, BHB and glucose decreased ($p < 0.01$).

Body condition score was significantly higher in cows with moderate or severe fatty liver ($p < 0.05$) as compared to cows with mild fatty liver (table 3). Apart from LDA, most of the animals (55%; table 3) were suffering from additional diseases, such as endometritis, mastitis or lameness; the prevalence of these additional diseases was similiar in all groups. Within three weeks post surgery, 6 of the studied cows (11%) were culled (table 3). Three cows with severe fatty liver and one cow with moderate fatty liver had to be euthanized because of recumbency and severe depression in their general condition. Mastitis was the reason for the slaughter of 2 other cows.

Table 1: Nutritional energy intake and milk yield of dairy cows suffering from left abomasal displacement (N = 53) on the day of surgery, 2 days and 4 days post surgery related to the degree of fatty liver (x ± SEM)

		Degree of fatty liver			
		no / mild N=15#	moderate N=21	severe N=17	statistics
Nutritional energy intake [MJ NEL / d]	ds	$22.9 \pm 2.0^{a,1}$	$14.8 \pm 1.7^{a,1}$	$8.7 \pm 1.5^{b,1}$	group: **
	d2	63.1 ± 5.3^{2}	49.0 ± 4.9^{2}	$38.9 \pm 3.2^{1,2}$	time: ***
	d4	$81.3 \pm 6.9^{a,3}$	$64.6 \pm 7.9^{a,b,3}$	$28.8 \pm 5.7^{b,2}$	
Milk yield [kg / d]	ds	7.6 ± 1.5^{1}	12.2 ± 1.1^{1}	7.8 ± 1.2^{1}	group: n.s.
	d2	10.1 ± 3.5^{2}	14.9 ± 1.2^{2}	12.5 ± 1.5^{2}	time: ***
	d4	14.9 ± 2.2^{3}	16.6 ± 1.4^{3}	13.8 ± 1.6^{2}	

ds = day of surgery; d2, d4 = 2, 4 days post surgery, respectively;
* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, n.s.: not significant
Means within one row (group differences) with different superscript letters are statistically significantly different ($p < 0.05$).
Means within one column (time differences) with different superscript ciphers are statistically significantly different ($p < 0.05$).
Results of milk yield from N = 12 (3 cows with mild fatty liver were dry).

Table 2: Results of clinical biochemistry of dairy cows suffering from left displacement of the abomasum (N = 53) on the day of surgery and 4 days post surgery related to the degree of fatty liver (x ± SEM)

		Degree of fatty liver			
		no / mild N=15	moderate N=21	severe N=17	statistics
Liver glycogen content (degree 0 - 5)	ds	$2.8 \pm 0.2^{a,1}$	$1.9 \pm 0.1^{b,1}$	$0.5 \pm 0.2^{c,1}$	group: **
	d4	$3.5 \pm 0.2^{a,2}$	$2.5 \pm 0.2^{b,2}$	$0.9 \pm 0.2^{c,1}$	time: *
Total bilirubin (µmol/l) [< 8.55]#	ds	$7.8 \pm 1.5^{a,1}$	$13.2 \pm 1.5^{b,1}$	$19.1 \pm 3.0^{c,1}$	group: ***
	d4	$5.1 \pm 0.6^{a,2}$	$7.8 \pm 0.9^{a,2}$	$14.4 \pm 2.3^{b,2}$	time: ***
ASAT (U/l) [< 50]#	ds	57.5 ± 5.1^{a}	72.4 ± 8.5^{b}	114.8 ± 11.5^{c}	group: ***
	d4	63.1 ± 6.6^{a}	79.4 ± 7.5^{b}	107.2 ± 12.0^{c}	time: n.s.
GGT (U/l) [< 20]#	ds	18.2 ± 10.6^{a}	15.3 ± 2.0^{a}	26.3 ± 5.4^{b}	group: *
	d4	17.8 ± 7.2^{a}	16.6 ± 1.6^{a}	26.3 ± 5.0^{b}	time: n.s.
GLDH (U/l) [< 8]#	ds	11.7 ± 1.6^{a}	12.9 ± 2.7^{a}	38.9 ± 14.1^{b}	group: *
	d4	14.1 ± 2.8	16.2 ± 2.1	27.5 ± 4.2	time: n.s.
NEFA (µmol/l) [< 600]#	ds	490 ± 93^{a}	$1000 \pm 113^{b,1}$	$1445 \pm 214^{c,1}$	group: ***
	d4	302 ± 46^{a}	$525 \pm 87^{a,b,2}$	759 ± 84^{2}	time: ***
BHB (mmol/l) [< 1.0]#	ds	$0.44 \pm 0.07^{a,1}$	$1.48 \pm 0.17^{b,1}$	$2.19 \pm 0.24^{c,1}$	group: ***
	d4	$0.36 \pm 0.06^{a,2}$	$0.60 \pm 0.15^{a,2}$	$1.32 \pm 0.24^{b,2}$	time: ***
Glucose (mmol/l) [2.4 - 3.8]#	ds	3.72 ± 0.26^{1}	3.47 ± 0.23^{1}	3.36 ± 0.30^{1}	group: n.s.
	d4	3.09 ± 0.17^{2}	2.63 ± 0.16^{2}	2.82 ± 0.40^{2}	time: **
Cholesterol (mmol/l) [2.0 - 4.0]#	ds	2.14 ± 0.22^{a}	1.51 ± 0.20^{b}	1.41 ± 0.14^{b}	group: *
	d4	2.04 ± 0.17^{a}	1.55 ± 0.16^{b}	1.35 ± 0.13^{b}	time: n.s.

ds = day of surgery; d4 = 4 days post surgery, respectively;
* = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$, n.s.: not significant
Means within one row (group differences) with different superscript letters are statistically significantly different ($p < 0.05$).
Means within one column (time differences) with different superscript ciphers are statistically significantly different ($p < 0.05$).
Reference values according to Stöber and Gründer (1990) and Scholz (1990).

Table 3: Body condition score, additional diseases and results of follow-up of dairy cows suffering from left displacement of the abomasum (N = 53) related to the degree of fatty liver

	Degree of fatty liver			
	no / mild	moderate	severe	total
	N = 15	N=21	N=17	N=53
body condition score	2.7 ± 0.1[a]	3.2 ± 0.2[b]	3.4 ± 0.1[b]	3.1 ± 0.1
additional diseases+				
endometritis	13%[a]	29%[a]	35%[a]	26%
mastitis	7%[a]	14%[a]	29%[a]	17%
lameness	33%[a]	14%[a]	18%[a]	21%
no additional diseases	47%[a]	57%[a]	29%[a]	45%
culled within 3 weeks post surgery	0%[a]	10%[a]	24%[a]	11%#

Values with different superscripts within one row are significantly different ($p < 0.05$).
4 cows with no feed consumption, bad general condition and recumbency; 2 cows with mastitis.
+ 5 cows suffered from two additional diseases.

Discussion

There commonly remains an imbalance between nutritional energy intake and energy requirement in the first 100 days of lactation in high-yielding dairy cows (Baird, 1980). The cows mobilize energy reserves, in particular body fat, to meet their energy demands, which results frequently in subclinical or clinical ketosis and an increased risk of fatty liver (Baird, 1980; Baird, 1982; Herdt, 1988). LDA develops predominantly in the first weeks of lactation (fig. 2; Dirksen, 1967), markedly aggravating this sensitive catabolic-metabolic condition. Due to the disturbance of digesta passage during LDA, feed intake and thereby energy intake declines (Dirksen, 1967). In our study, daily energy intake on the day of surgery (table 1) was on average about 20 MJ NEL, while energy requirement was about 65 MJ NEL (Meyer et al., 1993). Thus a severe energy deficit of 45 MJ NEL/d (equivalent to about 14 l milk) had been developed. Accordingly, most of the investigated cows with LDA showed high serum levels of NEFA (table 2) indicating a drastically enhanced lipomobilization.

Low feed intake results in a decreased availability of gluconeogenic precursors. Consequently, liver glycogen content decreases rapidly and the intrahepatic metabolism of NEFA shifts to increased production of ketone bodies and reesterification of NEFA to triglycerids (Baird, 1982; Lomax, 1992). On ds, about 70% of the LDA-patients were affected by moderate or severe fatty liver (fig. 1) and BHB serum levels of > 1 mmol/l, indicating clinical ketosis. Concurrently, in LDA-cows with severe fatty liver the liver glycogen content was significantly lower as compared to cows with moderate or mild fatty liver. Similiar observations have been made by Holtenius and Niskanen (1985) and Muylle et al. (1990). Until admission to the clinic, the animals had been ill for an average of 2.5 days. In high-yielding dairy cows two to three days of starvation are sufficient to double the liver fat content (Fürll et al., 1993). The retention of reesterified triglycerids within the hepatocytes has been attributed to reduced hepatic lipoprotein secretion (Rayssiguier et al., 1988). Cholesterol is a major constituent of lipoproteins. According to Holtenius (1989), the low cholesterol serum levels in LDA-cows with severe and moderate fatty liver (table 2) may provide evidence that lipoprotein secretion in the cows with fatty liver was reduced. The causes for reduced lipoprotein secretion are still unknown. Excessive lipomobilization and fatty liver are favoured by high levels of growth hormone and low levels of insulin which are found predominantly in the first weeks of lactation (Baumann and Currie, 1980; Bines and Hart, 1982). Therefore, the risk for severe lipomobilization and thereby for hepatosteatosis in LDA-patients decreases as the lactation progresses (fig. 2). In accordance with observations of Morrow (1976) and Stöber and Dirksen (1982) we found that the body condition of cows suffering from moderate or severe fatty liver was better (table 3) than BCS of cows with mild hepatosteatosis. This seems to be related to an increased fat mobilization of cows with a good body condition as compared to cows with lower BCS-values (Morrow, 1976; Stöber and Dirksen, 1982; Reid et al., 1986). On the other hand, Holtenius and Niskanen (1985) and Lotthammer (1992) suppose that severe fatty liver is not merely the consequence of excessive lipomobilization due to the low feed intake during LDA. The authors suggest that the same imbalances in feeding which favour the development of fatty liver in early lactation possibly may also cause abomasal displacement.

In cows suffering from ketosis, normally serum glucose concentrations are low (Baird, 1982). However, in LDA-patients, serum glucose concentrations were within the physiological range even in cows which developed ke-

tosis (table 2). From studies of Meirhaege et al. (1988b) and Holtenius and Traven (1990) it is known that the relatively high serum levels of glucose in LDA-patients are accompanied by high insulin concentrations. High glucose and high insulin levels may be explained by an insulin resistance in cows with LDA (Meirhaege et al., 1988b; Holtenius and Traven, 1990). In recent years it was assumed that the observed high insulin levels in LDA-patients are involved in the etiology of LDA (Breukink, 1990), since Meirhaege et al. (1988a) showed that high plasma insulin levels may provoke abomasal atony. Abomasal atony favours the accumulation of abomasal gases, a precondition for LDA. However, the causes of abomasal atony have not yet been clarified finally (Breukink, 1990; Schemann et al., 1994; Geishauser 1995). In our study mean serum glucose levels decreased immediately post surgery although energy intake increased, and serum NEFA and BHB levels decreased. The decrease in glucose may be due to the increased glucose drainage by the mamma for milk production (Frobish and Davis, 1977). However, further studies are necessary to differentiate between cause and effect in metabolic and hormonal changes that occur in cows suffering from abomasal displacement.

During the convalescence period following surgery marked differences in feed intake have been found depending on the degree of fatty liver. After surgical correction of LDA mean energy intake increased significantly in cows with no or moderate hepatosteatosis (table 1). Concurrently, milk yield increased and serum NEFA and BHB levels decreased by about 50% indicating an improvement in energy supply. In contrast, feed intake of cows with severe fatty liver remained low after surgery. Similarly, West (1989 and 1990) reported that feed intake in cows with severe liver damage is low. Low appetite can be interpreted as one of the first signs of an hepatic encephalopathy which may develop in cows with liver failure (Meier, 1992; Scholz et al., 1992). The low feed

Complications postopératives de vaches avec dislocation de la caillette vers la gauche et syndrôme de lipomobilisation

53 vaches de la race «Holstein-Frisian» atteintes de déplacement de la caillette vers la gauche ont été opérées par laparotomie au flanc droit et le déplacement de la caillette corrigé par omentopexie. Les vaches ont été examinées le jour de l'opération (dO) et pendant 4 jours après l'opération (d1-d4). 32% des vaches ont montré à l'examen histologique de biopsies de foie une dégénérescence graisseuse très grave (Grp. G). 40% ont montré une dégénérescense moyenne (Grp. M) et 28% aucune ou seulement une faible dégénérescence (Grp. F). 74% des vaches suivies se trouvaient dans le 3 premières semaines après le partus. Les vaches avaient subie une dégénérescence graisseuse du foie plus grave par rapport à celles qui se trouvaient à un niveau de lactation ultérieure ($p < 0.05$). Pendant les 4 premiers jours de la recherche nous n'avons pu noter aucun changement remarquable du degré de dégénérescence graisseuse du foie. Alors que la consommation journalière de fourrage durant la période postopératoire restait faible dans le Grp. G, elle augmentait continuellement dans le Grp. M et Grp. F ($p < 0.05$). Indépendant du degré de dégénérescence graisseuse la production laitière augmentait dans tous les groupes. La lipomobilisation et l'acétonémie, évaluées par la concentration sanguine moyenne en NEFA et en BHB ont été plus prononcées que dans le Grp. G que dans les Grp. M et Grp. F ($p < 0.05$). Les concentrations sanguines en glucose ont été

Riconvalescenza postoperatoria di mucche con depostazione sinistra dell'abomaso e con lipomatosi epatica

La laparotomia nel fianco destro è stata eseguita in 53 mucche HF con depostazione sinistra dell'abomaso. La depostazione dell'abomaso è stata eliminata definitivamente attraverso l'omentopexia. I pazienti sono rimasti sotto osservazione durante il giorno dell'operazione e in seguito per altri quattro giorni (d1-d4). Il 32% delle mucche fecero riscontrare all'analisi istologica nelle biopsie del fegato, una forte lipomatosi epatica (gruppo H), il 40% una lipomatosi epatica media (gruppo M) ed il 28% una lipomatosi leggera oppure nessuna lipomatosi (gruppo G). Il 74% delle mucche prese in considerazione si trovava nelle prime tre settimane dopo il parto: queste mucche soffrivano più frequentemente di una lipomatosi grave ($p < 0.05$) che quelle che erano in uno stadio più avanzato della lattazione. Nei 4 giorni dopo l'operazione non si notavano cambiamenti nel contenuto di grasso del fegato. Mentre che nel gruppo H l'ingestione giornaliera di foraggio rimaneva scarsa, per i gruppi M e G questa aumentava gradualmente ($p < 0.05$). In tutti i gruppi l'incremento della produzione lattifera era simile. Nel giorno dell'operazione i valori degli acidi grassi insaturi e del beta-idroxibutirato nel siero erano nel gruppo H i più alti ($p < 0.01$) se confrontati con i due altri gruppi M e G. I valori del glucosio nel siero erano invece simili per tutti e tre i gruppi. I valori della bilirubina totale, dell'aspartato-aminotransferasi (ASAT), della gammaglutamiltransferasi (GGT) e della glutamino

comparables dans les 3 groupes. Pendant que les niveaux sanguines en bilirubine GB, ASAT, GGT et GLDH étaient plus élevées dans le Grp. G ($p < 0.05$), la concentration en cholestérol était par contre dans le Grp. G plus basse ($p < 0.05$) par rapport aux Grp. M et Grp. F. Alors que les niveaux sanguines en ASAT, GGT, GLDH et cholestérol sont restés constants jusqu'au d4, les concentrations du sang en GB, NEFA, BHB et en glucose tombaient dans les 3 groupes ($p < 0.05$). 55% des vaches ont été traités à la suite de mammite, d'endométrite ou de boiterie. Dans l'intervalle de 3 semaines après l'opération, 6 vaches ont dû être abattus: 3 vaches du Grp. G et une du Grp. M avaient montré un coma hépatique ainsi que 2 vaches atteintes de mammites sévères. Nos résultats montrent que le degré de lipomobilisation et de dégénérescence graisseuse du foie ont une influence dans la reconvalescence postopératoire ches les vaches atteintes de déplacement de la caillette vers la gauche. En conséquence le succès du traitement à dépend une opération immédiate mais aussi à un traitement supplémentaire efficace du syndrôme de lipomobilisation excessive.

deidrogenasi (GLDH) erano mediamente maggiori nel gruppo H, mentre i valori della colesterina erano minori che nei gruppi M e G. I valori medi degli enzimi ASAT, GGT, GLDH e della colesterina rimanevano invariati fino al quarto giorno dopo l'operazione, mentre la bilirubina totale, gli acidi grassi insaturi, il beta-idroxibutirato ed il glucosio diminuivano in tutti i gruppi ($p < 0.01$). Il 55% dei pazienti doveva inoltre essere curato per mastite, endometrite o paralisi. Tre settimane dopo l'operazione 6 pazienti sono stati sacrificati: 3 pazienti del gruppo H ed un paziente del gruppo M a causa di un coma epatico, 2 mucche a causa di un'infiammazione della mammella.
I risultati mostrano che la riconvalescenza postoperativa nelle mucche con depostazione sinistra dell'abomaso, dipende in gran parte dalla lipomatosi epatica e dal fenomeno della lipomobilizzazione, e viene inoltre influenzata da malattie concomitanti. Una terapia effettiva della depostazione sinistra dell'abomaso deve includere perciò non solo l'operazione instantanea, ma anche la cura della sindrome della lipomobilizzazione.

intake was followed in four cows by ataxia, general depression and later by recumbency. Finally, these cows died in liver coma or had to be slaughtered for that reason. Therefore, low feed intake following surgical correction of LDA seems to be a valuable sign indicating severe liver damage. Of course, other diseases which can cause depression of appetite have to be taken into consideration. This is especially important as LDA is often accompanied by infectious diseases such as mastitis, endometritis or lameness (table 3; Dirksen, 1967; Breukink, 1990).

In cows with severe fatty liver, mean milk yield increased unexpectedly from 8 kg/d on ds to 14 kg/d on d4. Concurrently, serum NEFA and BHB levels on average decreased by 40% although feed intake remained low. We have no convincing explanation for this observation.

Clinical biochemistry may provide additional information concerning the degree of liver damage. However, the range of the laboratory data even in cows with comparable degree of fatty liver varies considerably. Thus, the diagnosis of liver damage needs additional careful assessment of clinical signs. In agreement with Gerloff et al. (1986), West (1989 and 1990) and Holtenius and Hjort (1990) we found in LDA-cows with severe fatty liver on ds significantly higher ($p < 0.05$) mean serum levels of total bilirubin, ASAT, GGT and GLDH as compared to cows with moderate or mild fatty liver (table 2). Although the degree of fatty liver remained nearly unchanged in the 4 day period following surgery, total bilirubin levels decreased significantly in all groups ($p < 0.01$). This decrease of bilirubin reflects probably the concurrent decrease in the NEFA serum levels (Qualmann et al., 1995) as hepatic bilirubin uptake increases in presence of low NEFA levels (Feverey and Heirwegh, 1980; Qualmann et al., 1995).

In conclusion, results of the study show that post surgical convalescence of cows with LDA is clearly related to disturbances in energy metabolism and fatty liver. Therefore, for successful treatment of cows suffering from LDA, the effective treatment of excessive lipomobilization, ketosis and fatty liver in addition to immediate surgical correction of LDA is required.

References

Baird G. D. (1980): Liver metabolism in the dairy cow: Problems involved in meeting the demands of high productivity. Proc. IV Int. Conf. on Production Diseases in Farm Animals, München, Germany, 87–93

Baird G. D. (1982): Primary ketosis in the high-producing dairy cow: clinical and subclinical disorders, treatment, prevention and outlook. J. Dairy Sci. *65*, 1–10

Baumann D. E., Currie E. (1980): Partitioning of nutrients during pregnancy and lactation: A reviev of mechanism involving homeostasis and homeorhesis. J. Dairy Sci. *63*, 1514–1529

Bines J. A., Hart I. C. (1982): Metabolic limits to milk production, especially roles of growth hormone and insulin. J. Dairy Sci. *65*, 1375–1389

Dirksen G. (1967): Gegenwärtiger Stand der Diagnostik, Therapie und Prophylaxe der Dislocatio abomasi sinistra des Rindes. Dtsch. Tierärztl. Wschr. *74*, 625–633

Edmonson A.J., Lean I.J., Weaver L.D., Farver T., Webster G. (1989): A body condition scoring chart for Holstein dairy cows. J. Dairy Sci. *72*, 68-78

Fevery J., Heirwegh K.P.M. (1980): Bilirubin Metabolism. Int. Rev. Physiol. *21*, 171-220

Frobish R.A., Davis C.L. (1977): Effects of abomasal infusions of glucose and propionate on milk yield and composition. J. Dairy Sci. *60*, 204-209

Fürrl M., Kirbach H., Knobloch B. (1993):Glucokorticosteroid-einfluss auf die fastenstimulierte Lipolyse und die Leberfunktion bei Milchkühen. Tierärztl. Praxis *21*, 399-403

Geishauser T. (1995): Abomasal displacement in the bovine - a review on character, occurence, aetiology and pathogenesis. J. Vet. Med. A *42*, 229-251

Gerloff B.J., Herdt T.H., Emery R.S. (1986): Relationship of hepatic lipidosis to health and performance in dairy cattle. J. Am. Vet. Med. Assoc. *188*, 845-850

Herdt T.H. (1988): Fatty liver in dairy cows. Vet. Clin. of North Am.: Food Animal Practice *4*, 269-287

Herdt T.H. (1991): Relationship of fat metabolism to health and performance in dairy cattle. Bov. Practitioner *26*, 92-95

Holtenius P. (1989): Plasma lipids in normal cows around partus and in cows with metabolic disorders with and without fatty liver. Acta Vet. Scand. *30*, 441-445

Holtenius P., Hjort M. (1990): Studies of the pathogenesis of fatty liver in cows. Bov. Practitioner *25*, 91 -94

Holtenius P., Niskanen R. (1985): Plasma lipids in normal cows around partus and in cows with metabolic disorders with and without fatty liver. Dtsch. tierärztl. Wschr. *92*, 398-400

Holtenius P., Traven M. (1990): Impaired glucose tolerance and heterogenity of insulin responses in cows with abomasal displacement. J. Vet. Med. A *37*, 445-451

Lomax M.A. (1992): Visceral and hepatic metabolism in relation to ruminant ketosis. Proc. VIII Int. Conf. on Production Diseases in Farm Animals, Berne, Switzerland, 184-197

Lotthammer K.H. (1992): Epidemiologische Untersuchungen über das Vorkommen von Labmagenverlagerungen (dislocatio abomasi) in Milchrinderbeständen. Tierärztl. Umschau *47*, 320-328

Meier C. (1992): Veränderungen der Aminosäuren- und Ammoniakkonzentrationen in Blutplasma und Liquor cerebrospinalis bei Leberfunktionsstörungen des Rindes und ihre mögliche Bedeutung für zentralnervöse Ausfallerscheinungen («Hepatische Enzephalopathie»).Vet.-Med. Diss, Hannover

Meirhaege H. van, Deprez P., van den Hende C., Muylle E. (1988a): The influence of insulin on abomasal emptying in cattle. J. Vet. Med. A *35*, 213-220

Meirhaege H. van, Deprez P., van den Hende C., Muylle E. (1988b): Plasma glucose clearance and insulin response in cows with abomasal displacement. J. Vet. Med. A *35*, 221-228

Mertens M. (1992): Leberbiopsie beim Rind: Histologische und enzymhistochemische Auswertung von Bioptaten aus Kühen mit Dislocatio abomasi sinistra. Vet.-Med. Diss, Hannover

Mertens M., Stockhofe-Zurwieden N., Rehage J., Scholz H. (1996): Histological and enzyme-histochemical investigations in liver bioptates obtained from dairy cows with left abomasal displacement and fatty liver (in preparation)

Meyer H., Bronsch K., Leibetseder J. (1993): Supplemente zu Vorlesungen und Übungen in der Tierernährung. 8. Aufl., Verlag Schaper, Alfeld, Hannover

Morrow D. (1976): Fat cow syndrome. J. Dairy Sci. *59*, 2-11

Morrow D., Hillman D., Dade A.W., Kitchen H. (1979): Clinical investigation of dairy herds with the fat cow syndrome. J. Am. Vet. Med. Assoc. *174*, 161-167

Muylle E., van den Hende C., Sustronck B., Deprez P. (1990): Biochemical profiles in cows with abomasal displacement estimated by blood and liver parameters. J. Vet. Med. A *37*, 259-263

Muylle E., de Keersmaeker F., Sustrock B., Deprez P. (1992): Vet lever syndrome en hepatisches coma bij vier hoogproduktieve melkkoieen met lebmaagenverplaatsing naar links. Vlaams Diergeneeskd. Tijdschr. *61*, 18-20

Qualmann K., Rehage J., Beyerbach M., Scholz H. (1995): Hyperbilrubinemia in dairy cows with lipomobilisation syndrome and its relation to high serum concentrations of nonesterified fatty acids. Proc. IXth Intern. Conf. on Prod. Diseases in Farm Animals, Berlin, 11. - 14. Sept. 1995, 50

Rayssiguier Y., Mazur A., Gueux E., Reid I.M., Roberts C.J. (1988): Plasma lipoproteins and fatty liver in dairy cows. Res. Vet. Sci. *45*, 389-393

Reid, I.M., Roberts, C.J. Treacher, R.J. Williams L.A. (1986): Effect of body condition on calving on tissue mobilization, development of fatty liver and blood chemistry of dairy cows. Anim. Prod. *43*, 7-15

Rehage J., Stockhofe-Zurwieden N., Mertens M., Meier C., Scholz H. (1992): Histological and enzyme-histochemical investigations of liver bioptates in cows suffering from left-sided displacement of the abomasum. Proc. VIII Int. Conf. on Prod. Diseases of Farm Animals, Berne, Switzerland, 205

S.A.S. User's Guide (1985): Basics and Statistics. Version 5. edition, SAS Institute Inc., Box 8000, Cary, NC

Schemann M., Reiche D., Geishauser T. (1994): Identification of motility disorders associated with abomasal displacement. Proc. Soc. Nutr. Physiol. *3*, 54

Scholz H. (1990): Stoffwechselkontrolle in der Milchkuhherde anhand von Blut- und Milchparametern. Prakt. Tierarzt Coll.Vet. *XXI*, 32-35

Scholz H., Zipori G., Höltershinken M. (1989): Erfahrungen mit einem neuen Instrument zur Leberbiopsie. Prakt. Tierarzt, Coll. Vet. *XIX*, 21-22

Scholz H., Rehage J., Meier C., Mertens M., Höltershinken M. (1992): Veränderungen von Aminosäurenindex und Ammoniak-konzentrationen in Blut und Liquor cerebrospinalis des Rindes mit Leberschäden. Proc. XVII World Buiatrics Congress, St.Paul, Minnesota, USA, 312-317

Stöber M., Dirksen G. (1982): Das Lipomobilisationssyndrom der Milchkuh. Prakt. Tierarzt, Coll. Vet. *XII*, 79-88

Stöber M., Gründer D. (1990): Kreislauf. in ROSENBERGER G. (Hrsg.), Die klinische Untersuchung des Rindes. 3. Aufl., Paul Parey, Berlin, Hamburg, 216-218

Top van den A.M., Wensing T., H. Geelen M.J., Wentink G. H., van't Klooster A. T., Beynen A. C. (1995): Time trends of plasma lipids and enzymes synthesizing hepatic triacylglycerol during postpartum development of fatty liver in dairy cows. J. Dairy Sci. *78*, 2208-2220

West, H.J. (1989): Liver function of dairy cows in late pregnancy and early lactation. Res. Vet. Sci. *46*, 231-237

West, H.J. (1990): Effect on liver function of acetonaemia and the fat cow syndrome in cattle. Res. Vet. Sci. *48*, 221-227

Korrespondenzadresse: J. Rehage, Klinik für Rinderkrankheiten, Tierärztliche Hochschule Hannover, Bischofsholer Damm 15, D-30173 Hannover