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EVOLUTION OF THE POSTNATAL METABOLISM IN THE HEALTHY OR DIARRHOEIC CALF

C. DEMIGNÉ and C. RÉMÉSY

Laboratoire des Maladies Métaboliques - Institut National de la Recherche Agronomique
Theix, 63110 Beaumont, France

Résumé

EVOLUTION DU METABOLISME POSTNATAL CHEZ LE VEAU SAIN OU DIARRHEIQUE
— L’évolution postnatale du métabolisme ainsi que celle des produits terminaux de la flore ont été étudiées chez des veaux demeurés sains ou ayant présenté des accès de diarrhée.

Chez les animaux sains, une flore digestive productrice d’acides gras volatils (AGV) semble s’installer rapidement. Chez les animaux diarrhéiques on enregistre une élévation du pH fécal avec une chute des AGV ; parallèlement, du lactate est présent dans les fèces et pourrait provenir en partie d’un accroissement de la glycolyse tissulaire.

Les concentrations en lactate restent très élevées durant les premières 24 heures, alors que la glycémie augmente rapidement. L’utilisation de surcharges en L-lactate chez des veaux âgés de 1 à 2 jours suggère que le turnover du lactate est très élevé durant la période postnatale. L’élévation de l’urémie semble être une des variations plasmatiques les plus précoces en cas de diarrhée.

La comparaison avec des situations de jeûne limité indique que le veau sain est capable de maintenir une glycémie relativement élevée et peut effectuer une lipomobilisation importante avec toutefois une cétogenèse très modérée.

L’étude de cas aigus de diarrhée souligne l’importance des hypoglycémies très sévères accompagnées généralement d’hyperlactatémies, sans lipomobilisation ni cétogenèse importantes. Ces perturbations métaboliques ont été discutées, en relation avec les perturbations provoquées par la diarrhée au niveau du métabolisme hydrominéral.

The young calf is frequently affected by digestive disorders, such as neonatal diarrhoea, that generally occur during the first three weeks after birth. The lack of immunological defences at birth necessitates the immediate ingestion of colostrum which provides a passive supply of γ-globulins which are particularly important for the survival of the calf (Dardillat, 1973). The establishment of the microbial flora in the alimentary tract occurs very early, but this flora changes during the first two weeks of life (Contrepois and Gouet, 1973). The origin of the diarrhoea is certainly complex: nutrition, bacteria, viruses or a combination of these factors may be involved. In addition the motility of the digestive tract may be affected, particularly that of the abomasum (Dardillat, 1975).

Some disturbances associated with calf diarrhoea have already been described: water-
electrolyte changes in the various compartments of the body (Fayet, 1968a,b), water and electrolytes transfers to and from the intestine (Bywater, 1973) and metabolic changes such as acidosis or hypoglycaemia (Tennant et al., 1968, 1972; Lewis et al., 1975). However, a comparison of changes with age in the overall metabolism of healthy and diarrhoeic calves after birth has never been made. The aim of this work was to compare the metabolic response of the young calf to starvation and to diarrhoea, and to study the changes with age in the end-products of the microbial flora metabolism in healthy and diarrhoeic calves.

Experimental

1. Animals

Comparison of the postnatal changes in the metabolism of the healthy or diarrhoeic calves.

Sucking Salers or crossbred Salers calves, from the experimental herd of Marcenat, were used in the experiment. The dams were fed either with 85% hay and 15% of concentrate (barley and ground-nut seed-cake) or with hay, with straw ad libitum. The calves were allowed to suck twice daily, with fresh water available all days long.

Blood sampling has been carried out: before the first meal (1 to 6 hours after birth), 4 to 6 hours after the first meal (which occured 3 to 6 hours after birth), at 2, 8 and 21 days (3 hours after the morning meal which occured at 7.00 h). For diarrhoeic calves, blood samples were also taken at an average age of 5 and 8 days. Blood was sampled from the jugular vein, centrifuged in heparinized tubes and the plasma was immediatly frozen. Results are means for 20 healthy calves (group 1) and 30 diarrhoeic calves (group 2). In the diarrhoeic group, only 13 calves were sampled at 8 days, and 21 at 21 days. Faeces were sampled after the morning meal.

Effect of starvation

This was studied on calves of an average age of 5 days: either sucking (fed as previously indicated) or bucket-fed Fresian × Holstein receiving daily 2 meals of a reconstituted milk (crude protein, 2.6%; fat, 2.3%; carbohydrate, ca 4.0%). These calves were starved for 24 hours. More detailed observations were made on the latter type of calf starved 33 hours.

Lactate infusions

Sodium (L)-lactate at pH 7.4 was infused at the rate of 0.1 mmole/min/kg during 35 minutes (volume infused: 500 ml) in the jugular vein of 5 bucket-fed calves (1 to 2 day-old), 6 hours after the morning feed. Blood samples were collected as indicated in the figure 5.

Diarrhoeic calves

Observations of the metabolic changes during acute diarrhoea were made on bucket-fed calves, particularly during the period preceeding the death.

2. Biochemical techniques

Volatile fatty acids (VFA) determinations were made by the previously described methods (Remésy and Demigné, 1974 & 1976). For the determination of D and L lactate in faeces, the frozen samples were centrifuged (2 min, 8000 g) and 1 volume of supernatant was treated by 19 volumes of 0.4 M HCl O₄. Lactic acid was determined by an enzymatic method using either the D (EC 1.1.1.28) or L (EC 1.1.1.27) lactate dehydrogenase with the simultaneous action of the glutamic pyruvic transaminase (Noll, 1974). The following plasma metabolites were determined by enzymatic methods: glucose (Bergmeyer et al., 1974), lactate (Gutmann & Wahlefeld, 1974) alanine (Williamson, 1974), 3-hydroxybutyrate (Williamson & Mellamby, 1974) and urea (Gutmann & Bergmeyer, 1974). The determination of plasma free fatty acids (FFA) and amino acids has been described (Remésy et al., 1978).

Results

Post-natal changes in the end-products of the microbial flora metabolism (Table 1).

In normal animals, the pH of the faeces (and thus of the caecum and colon contents) is quite low during the first days (pH 5.7). This corresponds to a type of fermentation with a higher molar percentage of butyrate than of propionate. As carbohydrates are the most likely substrate for acid fermentations, it may be inferred that carbohydrates of endogenous origin or lactose may have reached the caecum. During the following weeks, in healthy calves, the pH increased to about 6 and the percentage of propionate becomes higher than that of butyrate. In diarrhoeic calves,
there is generally an increase in the faecal pH, the origin of which is probably a decline in VFA (from about 55 to 23 mM), but possibly in combination with an increase of ammonia, and losses of Na⁺ or HCO₃⁻ into the small intestine.

Faecal lactate (L and D forms) was generally low in the healthy sucking animals. During diarrhoea, there were no striking changes of the end-products of digestive fermentations, except for a decrease in VFA concentration related to the decrease in faecal dry matter percentage. However, in acute diarrhoea there was an increase of lactate (mainly the L-form); at 21 days of age, previously diarrhoeic calves had higher VFA concentrations in the faeces, possibly because the absorptive capacities of the intestine were not fully restored.

Post-natal changes in plasma metabolites (Fig. 1 & 2)

Plasma glucose at birth is low, and increases rapidly after the first meal. At the beginning of diarrhoea, blood glucose concentration did not significantly decline; but under the same conditions in an other experiment, we observed a significantly lower plasma glucose in diarrhoeic than in healthy calves.

Plasma lactate at birth was always high (3-5 mM), and decreased rather slowly, reaching a normal value (about 1 mM) between 2 and 8 days after birth. During diarrhoea, plasma lactate was high, probably due to an overproduction of lactate. In calves suffering mild diarrhoea, hyperlactemia was sometimes observed, without marked hypoglycaemia.

The changes in plasma alanine are similar to those of lactate. Relatively high concentrations were found in 2-day-old calves, probably related to the increase of blood glucose and to the catabolism of amino acids. Plasma alanine was generally high during diarrhoea.

Plasma urea concentrations declined to about 3 weeks, at which time protein catabolism was high. The period of relatively high blood urea in the first week probably corresponded to a high secretion of corticoid (Hudson et al., 1976). At a young age, differences were observed between healthy and diarrhoeic calves in urea, which seems to be one of the most sensitive parameters. In acute diarrhoea, urea was very high due to an increased nitrogen catabolism and a decrease of urinary excretion. Full recovery seemed slow, since at

<table>
<thead>
<tr>
<th>AVERAGE AGE</th>
<th>GROUP</th>
<th>VFA MOLAR PERCENTAGE OF</th>
<th>pH</th>
<th>D-Lactate (mM)</th>
<th>L-Lactate (mM)</th>
<th>Propionate</th>
<th>Butyrate</th>
<th>Acetate</th>
<th>TOTAL VFA (mM)</th>
<th>PERCENTAGE DRY MATTER</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5 days</td>
<td>Healthy (10)</td>
<td>29.3 ± 2.3</td>
<td>5.69 ± 0.06</td>
<td>54.3 ± 7.3</td>
<td>0.4 ± 0.4</td>
<td>23 ± 3</td>
<td>2.1 ± 0.4</td>
<td>1.2 ± 0.4</td>
<td>54.0 ± 0.06</td>
<td>54.0 ± 0.06</td>
</tr>
<tr>
<td></td>
<td>Diarrhoeic (20)</td>
<td>27.2 ± 1.7</td>
<td>5.95 ± 0.05</td>
<td>54.0 ± 5.0</td>
<td>1.5 ± 0.3</td>
<td>27 ± 2</td>
<td>1.5 ± 0.3</td>
<td>1.8 ± 0.6</td>
<td>54.0 ± 0.06</td>
<td>54.0 ± 0.06</td>
</tr>
<tr>
<td>5.5, days</td>
<td>Healthy (9)</td>
<td>25.4 ± 1.2</td>
<td>5.81 ± 0.08</td>
<td>62.7 ± 13.7</td>
<td>2.0 ± 0.5</td>
<td>22 ± 3</td>
<td>2.0 ± 0.5</td>
<td>1.7 ± 0.9</td>
<td>62.7 ± 13.7</td>
<td>62.7 ± 13.7</td>
</tr>
<tr>
<td></td>
<td>Diarrhoeic (2)</td>
<td>25.4 ± 1.4</td>
<td>6.03 ± 0.09</td>
<td>66.7 ± 11.7</td>
<td>2.0 ± 0.2</td>
<td>22 ± 3</td>
<td>2.0 ± 0.2</td>
<td>1.7 ± 0.9</td>
<td>66.7 ± 11.7</td>
<td>66.7 ± 11.7</td>
</tr>
<tr>
<td>8 days</td>
<td>Healthy (8)</td>
<td>25.4 ± 1.4</td>
<td>6.03 ± 0.09</td>
<td>66.7 ± 11.7</td>
<td>2.0 ± 0.2</td>
<td>22 ± 3</td>
<td>2.0 ± 0.2</td>
<td>1.7 ± 0.9</td>
<td>66.7 ± 11.7</td>
<td>66.7 ± 11.7</td>
</tr>
<tr>
<td></td>
<td>Diarrhoeic (7)</td>
<td>23.4 ± 1.7</td>
<td>6.48 ± 0.11</td>
<td>110.1 ± 6.7</td>
<td>4.1 ± 0.5</td>
<td>16 ± 2</td>
<td>4.1 ± 0.5</td>
<td>1.7 ± 0.9</td>
<td>110.1 ± 6.7</td>
<td>110.1 ± 6.7</td>
</tr>
<tr>
<td>21 days</td>
<td>Healthy (7)</td>
<td>23.4 ± 1.7</td>
<td>6.48 ± 0.11</td>
<td>110.1 ± 6.7</td>
<td>4.1 ± 0.5</td>
<td>16 ± 2</td>
<td>4.1 ± 0.5</td>
<td>1.7 ± 0.9</td>
<td>110.1 ± 6.7</td>
<td>110.1 ± 6.7</td>
</tr>
</tbody>
</table>

Table 1: Changes in the characteristics of the digestive contents of a group of healthy calves and a group of diarrhoeic calves which showed diarrhoeic symptoms at the age of 5 to 10 days; thus, the sampling at 5-5 days corresponds, in average, to the onset of the diarrhoea. The values are means ± S.E.M. for the number of calves given in parentheses. The statistical significance of the differences between healthy and diarrhoeic calves is indicated by * (P < 0.05).
3 weeks the uremia of the previously ill calves was still higher than that of healthy calves.

FFA at birth were high and probably originated from the limited lipid stores. Except for the first sample, all sampling was performed after a meal, and FFA were low despite a high intake of lipids. The 3-hydroxybutyrate was always very low during the first 3 weeks of life.

Amino acids. Postnatal changes in glycine were rather similar to those of alanine, whereas the concentration of most of the other amino acids increased between 2 and 8 days. There was a characteristic change in glutamate and glutamine: just after birth, glutamine declined whilst glutamate increased; later, glutamine increased slightly, whilst glutamate declined to low values (about 0.06 mM). During diarrhoea, there was a decline in glutamate, threonine, valine and arginine and to a lesser extent in lysine, histidine and serine. For most of the amino acids, diarrhoea occurs during the period of maximal plasma concentrations probably corresponding to a high supply of amino acids.

**Effects of starvation (Fig. 3 & 4)**

In sucking calves, both the postprandial increase of blood glucose, and the decrease after 24 h of starvation were slight. Plasma lactate did not noticeably vary during the starvation period. The FFA decreased after the meal and then increased markedly after 24 hours of starvation. Ketogenesis as measured by 3-hydroxybutyrate in the plasma, although increasing during the starvation period, remained relatively low. Starvation resulted in a slight increase of blood urea concentration.

In bucket-fed calves, the same changes were observed, with however a smaller lipid mobilisation, as judged by FFA concentration, and slight increase in urea. With longer starvation (33 h), glycaemia did not decrease, while the lipid mobilisation and ketogenesis continued to increase.

**Load of lactate (Fig. 5)**

To explain the presence of high blood lactate just after birth, a lactate infusion was made. Theoretically, with impaired utilization, the perfusion should produce a large accumulation of lactate in the plasma, while an increased turnover of lactate should produce smaller lactate accumulations. In fact, high lactataemia (5 mM) after infusion very rapidly returned to a normal level 15 minutes after the end of the infusion, without striking modifications of plasma glucose or FFA. Consequently, lactate utilization seems very efficient and hyperlacta-

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**Fig. 1**: Postnatal changes in plasma metabolites of healthy and diarrhoeic sucking calves. The time at which ingestion of colostrum occurred is shown by arrows for both groups. For details see the experimental part.

**Fig. 2**: Postnatal changes in plasma amino acids of healthy and diarrhoeic sucking calves.
taemia could be due to a high rate of production of this metabolite.

**Metabolic response to acute diarrhoea (Fig. 6)**

Blood glucose always fell to extremely low values in agonal calves, with generally very high lactataemia. Plasma urea sometimes reached concentrations higher than 15 mM. However, FFA were unchanged or moderately increased without marked ketogenesis.

In fatal diarrhoea, we observed 6/7 calves with hyperlactataemia. All the calves were hypoglycaemic (except when recently treated), sometimes having extremely low values of glucose, and all were hyperuraemic.

**Discussion**

The pH of the faeces should permit a distinction between infectious and nutritional diarrhoea, the latter being characterized by very acid digestive contents and lactic and (or) butyric fermentation. In fact, the relatively high pH in the faeces at the outset of neonatal diarrhoea in the sucking calves does not suggest an impaired absorption of lactose followed by a fermentation of this carbohydrate in the caecum and colon. Moreover, in infectious diarrhoea, if lesions of the brush-border in the jejunum and the ileum are present, as claimed with some viral diarrhoea (Mebus, 1976), a deficient digestion of lactose may occur owing to the disappearance of lactase.

It appears that VFA concentrations in the faeces are higher in previously diarrhoeic calves: this fact could be related to a sequel of the diarrhoea, disturbing the digestion of some substrates such as lactose and proteins, and thus providing larger amounts of fermentable substrates to the caecal flora.

The role of VFA as a barrier against pathogenic bacteria has never been convincingly demonstrated. The VFA-producing flora which develops in the digestive tract immediately after birth does not appear to protect against acute diarrheal disease. The origin of the drop in pH just after the establishment of the flora is still not clear, it might be due to the presence of complex carbohydrates in the diet (colostrum oligosaccharides) during that period or to insufficient VFA absorption.

The lactate measured in the faeces of diarrhoeic calves is mainly in the L form. This fact suggests that a large percentage of this lactate could be of endogenous origin, for instance intestinal glycolysis increased by hypoxia, ionic disturbances or toxins. However, further investigations are necessary to confirm this hypothesis and faecal samples are probably not the most accurate reflect of the changes in the intestinal metabolism.

The lack of postnatal hypoglycemia in the calf that we observed (in disagreement with Daniels et al., 1975) may be due to glycolysis and to the initiation of the gluconeogenesis which becomes fully active just after birth (Warnes et al., 1977). Some authors (Prior and Christenson, 1977) even consider that the gluconeogenesis is already efficient during the end of the foetal period. The hyperlactataemia observed in the early postnatal period probably does not originate from an impaired utilization although a role of the development of the lactate dehydrogenase.

**Fig. 3:** Changes in plasma metabolites during 24 hours after the morning meal in sucking or bucket-fed calves. The ingestion of the meal is indicated by an arrow. Results are means for 4 calves.

**Fig. 4:** Changes in plasma metabolites during 33 hours after the morning meal in bucket-fed calves. Results are means for 4 calves (± S.E.M.)
Isoenzymes has been proposed (Hinks and Masters 1964); indeed, lactate is well-utilized in foetal tissues (Burd et al., 1975), and large loads of lactate are normalized in about 15 minutes in 24 hour-old calves. In fact, a relationship between the efficiency of γ-globulin absorption and blood lactate has been proposed (Hardy, 1969). A considerable glycolysis could be induced by catecholamines (Comline and Edwards, 1968), particularly in peripheral tissues, despite high levels of cortisol (Hudson et al., 1976).

The young calf can withstand starvation without major metabolic disturbances and calves maintained quite high blood glucose even with drastic starvation (Goodwin, 1957). Basal concentrations of FFA, and apparent lipid mobilization differed between bucket-fed and suckling calves, these latter calves apparently having higher capacities of lipomobilization.

One of the main metabolic modifications in acute diarrhoea is lactic acidosis, aggravated by a decrease of the alkaline reserve. The increase of the lactate is generally concomitant with hypoglycaemia (Lewis et al., 1975). It seems that death occurs when the blood glucose becomes lower than 1-2 mM, there may be only traces of circulating glucose in dying calves. The production of lactate corresponds to a partial utilization of glucose which produces limited amounts of energy. Energy necessary for further synthesis of glucose from lactate is higher than energy produced in aerobic glycolysis; thus, considerable glycolysis can lead to an energy deficiency. In addition, lactate utilization by the liver could be saturated; (i) by hepatic hypoxemia linked to hypovolaemic shock (Tennant et al., 1968); a more reduced redox state of the cytosol may impair lactate utilization, (ii) the fatty acids availability could limit the supply of ATP and acetyl CoA for activation of pyruvate carboxylase. In some species, an active nitrogen catabolism and a strong uremia can result in a decreased utilization of lactate in the liver (Rémésy et al., 1978).

The loads of lactate tolerated by healthy young calves do not mean that the liver can always adapt to a very high supply of lactate: in fact, in healthy animals, the lactate may be also utilized by peripheral tissues (red muscles, adipose tissue...), while the situation is probably very different in the diarrhoeic calves. Authors have stressed the role of the hypovolaemia in tissue hypoxia (Lewis et al., 1975), furthermore, the O₂ delivery to the tissues is probably disturbed in the course of metabolic

![Graph](image1)

**Fig. 5:** Changes in plasma metabolites in response to an infusion of L-lactate in bucket-fed calves. Results are means for 5 calves, (± S.E.M.) For experimental details, see the text.

![Graph](image2)

**Fig. 6:** Changes in plasma metabolites during acute diarrhoea in bucket-fed calves.

The normal values are shown for fed (F) or starved (S) calves. Each point is a value for calves sampled at different states before death: O, calves 1 and 2, 12 hours before death; □ calf 3, 6 hours before death (previously treated by infusion of a glucose-bicarbonate formula); ■ calf 4, 1 hour before death; ▼ ▼ calves 5 and 6, dying calves; ♦ calf 7, 1 hour before death (with hypolactatemia).
acidosis, either by the decrease of the P CO₂ or by the modification of blood 2,3 diphosphoglycerate (Even, 1977). Indeed, the metabolism in the muscles could be modified as a result of ionic disturbances induced by the diarrhoea: the efflux of K⁺ from the cell (Fisher and Mc Ewan, 1967) is similar to the phenomenon of depolarisation with modifications of the cellular Ca²⁺ (Larner, 1976). Glycogenolysis could be followed by a greater production of lactate. Glycolysis also could be stimulated via the intracellular penetration of glucose. This problem is still poorly understood in the diarrhoeic calf and could be linked to ionic disturbances (Czech, 1977) or to the presence of endotoxins; in other species, hyperinsulinism after endotoxin administration has been suggested (Blackard et al., 1976), as stimulated lactate production by white blood cells (Hinshaw et al., 1976) or impaired gluconeogenesis (Groves et al., 1974).

The factors responsible for the poor lipid mobilisation during diarrhoea, even when marked hypoglycaemia occurs, are still undetermined. Exhaustion of the fat stores is certainly not the only factor responsible. The finding of an almost complete lack of ketogenesis in diarrhoeic animals during hypoglycaemia is not clear, although the moderate FFA concentrations could partially explain this fact. The lack of ketogenesis is undoubtedly unfavourable for calf survival since ketone bodies would allow an efficient saving of glucose in peripheral tissues (Berger et al., 1976). The high availability of lactate in the liver can contribute to a diversion of acetyl-CoA to the tricarboxylic cycle rather than to ketogenesis (Krebs et al., 1974). However, little is known about the control of the acyltransferase system in the young calf.

In the intestine, control of glycolysis is still poorly characterized. The process appears to be relatively independent of intestinal oxygenation with lactate production depending on the absorption of glucose. Further investigations on this last point are necessary, since some oral treatments supply large amounts of glucose (Hamm and Hicks, 1975; Bywater, 1977) in order to increase intestinal Na⁺ transport. Galactose, poorly metabolised by the intestine, but actively absorbed, could be preferred.

The metabolism of the intestine could be a target for the bacterial enterotoxins. Findings of Moss et al., (1978) about the heat-labile E. coli enterotoxin suggest that the activation of adenylic cyclase is associated with hydrolysis of NAD, which could disturb the intracellular metabolism. There is some evidence that in the calf the E. coli enterotoxin is mainly of the heat-stable type and its biochemical effects are still poorly known (Dubourguier et al., 1978).

The decrease in concentration of some plasma amino acids during diarrhoea may be related to undernutrition and to increased utilization during this disease; however, in the comparison between healthy and diarrhoeic calves it must be considered that: (i) the sample were not systematically obtained in the acute stage of diarrhea during which striking increase of plasma amino acids may occur in investigations; (ii) healthy calves in a comparable state of undernutrition should constitute more valid controls. The fate of glutamate is interesting as its decline seems to begin very early, as for the increase of urea. In fact, the turnover of glutamate during the first 8 days of life is probably high: loads of glutamate are very readily utilized (results not shown). The origin of the plasma glutamate is still not clear; it is suggested that intestinal absorption of glutamate occurs without extensive metabolism, resulting in a strong supply since the milk proteins are rich in glutamate and glutamine. It is not surprising during diarrhoea that glutamate is the most rapidly decreased since it cannot originate from tissue proteolysis (alanine and glutamine being the main vehicle of the -NH₂ group).

However, for evaluation of the importance of glutamate turnover (as of the origin of hyperlactataemia), it would be necessary to consider the interorgan relations of these metabolites between the digestive tract, the liver and the peripheral tissues. At present, the difficulties of blood vessels catheterism in the neonatal calf has limited such investigations.

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Summary

Postnatal evolution of metabolism and of end products of microbial flora has been studied in healthy or diarrhoeic calves. In healthy animals, the establishment of a flora producing volatile fatty acids (VFA) seems to be rapid. In diarrhoeic animals, an increase in the faecal pH with a diminution of VFA is observed; simultaneously lactate is present in faeces and may partly be produced by an increase of the tissular glycolysis. There is high plasma lactate concentrations during 24 hours after birth, while glycaemia increases rapidly. The utilization of L-lactate load by 1-2 days old calves suggests that there is a high turnover of lactate during the post-natal period. In diarrhoea, the increase in the uraemia seems to be one of the earliest plasma variations. Comparison with starvation periods shows that the healthy calf can conserve a relatively high glycaemia and effect an important lipid mobilisation with a very moderate ketogenesis. Studies of acute cases underline the importance of very serious hypoglycaemia often associated with hyperlactataemia and without an important lipid mobilisation or a high ketogenesis. These metabolic perturbations are discussed in relation with the perturbations produced by the diarrhoea in hydromineral metabolism.

References


