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Original Article

ECG Changes in Acute Experimental Ruminal Lactic Acidosis in Sheep

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Abstract

For induction of ruminal acidosis, 10 clinically healthy three years old non pregnant female sheep were selected. Prior to the infusion of sucrose (0 hour), rumen and blood samples were obtained in order to determine baseline rumen and blood pH, respectively. Electrocardiogram (ECG) was also recorded. Acute ruminal acidosis was induced experimentally with sucrose at a dose of 18g kg^{-1} body weigh through rumen fistula. ECG was recorded and blood and rumen samples collected at 3, 6, 9, 12, 15, 18, 21, 24, 30, 36, and 48 hours after the infusion of sucrose. Results indicated that blood and rumen pH decreased significantly at 15, 18, 21, 24, 30, 36 and 48 hours and at 3, 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 hours, respectively. Acidosis produced a marked increasing in heart rate and a decrease in PR interval at 15 and 18 hour significantly with little apparent effect on the ST and PR segment. The P amplitude increased significantly at 6, 9, 12, 15, 18, 21, 24 and 30 hours. The T amplitude increased significantly at 9, 12, 15, 18, 21, 24, 30 and 36 hours. The RR interval decreased significantly at 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 hours. In conclusion acute ruminal acidosis caused significant changes in ECG of sheep though there was not any detectable arrhythmia in the ECG in acute ruminal acidosis.

Key words: Acute Experimental Ruminal Acidosis, ECG, Sheep

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Introduction

Acute ruminal acidosis is one of the most dramatic forms of ruminal microbial fermentative disorders and in some cases is lethal in less than 24 hours.[1] The condition has been named lactic acidosis, toxic indigestion, grain engorgement, grain overload and D-lactic acidosis. This problem is the result of excessive consumption of readily fermentable carbohydrates, which causes a rapid fermentation with production of lactic acid and a decrease in ruminal pH to physiologically inappropriate levels.[2,3] This occurs when animals consume an excess of concentrate feeds (e.g., if animals are suddenly exposed to the feeds without prior adaptation; if animals already on such feeds suddenly consume an excessive quantity because of accidental access; or if animals that have been off feed return to feed and are offered unrestricted access to concentrates). The problem is more common when animals are grouped than when they are separate; probably because the psychology of competition induces them to over consume. The severity of ruminal acidosis and disease signs vary considerably, depending on the amount and type of carbohydrate-rich feed consumed and the degree of prior ruminal microbial adaption to the carbohydrate substrate.[4,5] Acidosis alters the electrical activity of cardiac muscle, having marked effects on the most of the polarisable membranes.[6-8] However, the effect of acidosis on the action potential will not be the same in all regions of the heart, because of regional differences in the expression of the ion channels underlying the membrane currents.[9,10] Acidosis induced changes in any current that is not uniformly distributed throughout the heart will result in varied changes in action potential configuration. The aim of this study was investigation of the effect of acute experimental ruminal acidosis on ECG of sheep.

Materials and Methods

Ten clinically healthy three years old non pregnant female sheep (Lori-Bakhtiari), with average weight of 41.2 ± 2.25 kg were selected for this study. There was not any parasitic infectious in animals according to the examination in the Department of Veterinary Clinical Pathology. All of the animals, for two weeks, were kept under the condition for adaptation to the new environment. One week prior to induction of rumen acidosis, rumen of animals was fitted with a fistula by ruminostomy technique.[8] For maintenance of anaerobic environment in rumen, the fistula was closed with a plug. During one week, the animals were ensured to be healthy and free from any clinically detectable abnormality. Prior to the infusion of sucrose (0 h), samples from rumen content were obtained using a 60 mL syringe through the rumen fistula and its pH was measured to determine normal values (pH meter C G822 Schott Gerate, Germany). To measure the blood pH (Radiometer ABL 5, Copenhagen, Denmark), blood samples were collected via jugular vein by plastic syringe with 0.1 - 0.2 mL heparine (5 U mL L^{-1}), the syringe was covered with a tight fitting cap and delivered on ice to the laboratory immediately. The base apex technique (the left electrode was placed on the heart area, the right electrode applied to the jugular furrow and the earth electrode was attached on the right flank) was used to record ECG in lead I.[2] Acute ruminal acidosis was induced by intraluminal administration of sucrose at a dose of 18 g kg^{-1} body weight.[13] ECG was recorded (Figs 1. & 2) and samples of rumen fluid and venous blood collected at 3, 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 h after the infusion of sucrose for above-mentioned purposes. To assess differences between

the control (hour zero) and induction of acidosis (hours 3 to 48), one-way ANOVA for repeated measurements with Tukey separation of the means in case of significance was performed. Probabilities of *P<0.05* were considered to be statistically significant.

Results

Mean values (± SEM) for variables measured in the rumen fluid, venous blood and for clinical parameters in the 10 sheep are shown in Table 1. Mean values (± SEM) for ECG parameters are shown in Table 2. Mean ruminal pH fell rapidly over the first 3 hours and decreased significantly at 3, 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 hours after the infusion of sucrose (*P < 0.05*). Blood pH decreased at 15, 18, 21, 24, 30, 36 and 48 hours after the infusion of sucrose (*P < 0.05*). Heart rate increased significantly at 9, 12, 15, 18, 21, 24, 30 and 48 hours (*P < 0.05*). Acidosis produced a marked increasing in heart rate and a decrease in P-R interval at 15 and 18 hours significantly with little effect on the ST and PR segment (*P<0.05*). The P amplitude increased significantly at 6, 9, 12, 15, 18, 21, 24 and 30 hours (*P<0.05*). The T amplitude increased significantly at 9, 12, 15, 18, 21, 24, 30 and 36 hours (*P<0.05*). The RR interval decreased significantly at 6, 9, 12, 15, 18, 21, 24, 30, 36 and 48 hours (*P<0.05*). There was not any detectable arrhythmia in acute ruminal acidosis during the experiment.

Table 1. Measurement of acute rumen pH, blood parameters and heart rate (mean ± SE) in experimentally induced ruminal acidosis in the sheep

	Item		
Time (h)	**Rumen pH**	**Blood pH**	**Heart rate**
0	7 ± 0.13	7.40 ± 0.02	70 ± 5.88
3	6.31 ± 0.34	7.44 ± 0.03	100 ± 6.26
6	5.45 ± 0.2*	7.39 ± 0.04	90 ± 3.81
9	4.97 ± 0.21*	7.36 ± 0.03	91.8 ± 5.3*
12	4.91 ± 0.22*	7.33 ± 0.02	96 ± 2.04 *
15	4.43 ± 0.28*	7.29 ± 0.05	102.2 ± 5.12*
18	4.32 ± 0.28*	7.22 ± 0.07*	100 ± 5.16*
21	4.16 ± 0.27*	7.13 ± 0.05*	101 ± 4.19*
24	4.1 ± 0.18*	7.18 ± 0.06*	95 ± 3.2*
30	4.02 ± 0.06*	7.16 ± 0.08*	90 ± 4.3*
36	4.04 ± 0.24*	7.15 ± 0.08*	85 ± 4.41
48	4.24 ± 0.18*	7.23 ± 0.06*	90 ± 4.03*

* Values are significant at $P < 0.05$

Table 2. ECG parameters (mean ± SE) in experimentally induced ruminal acidosis in the sheep

	Item					
Time	**P-amplitude**	**PR segment**	**PR interval**	**RR interval**	**ST segment**	**T- amplitude**
0	0.25 ± 0.05	0.08 ± 0.001	0.15 ± 0.001	0.69 ± 0.0005	0.13 ± 0.001	0.65 ± 0.002
3	0.25 ± 0.05	0.08 ± 0.001	0.15 ± 0.001	0.69 ± 0.0009	0.13 ± 0.0009	0.55 ± 0.022
6	0.20± 0.02*	0.08 ± 0.00	0.14 ± 0.001	0.61 ± 0.004*	0.14 ± 0.001	0.76 ± 0.006
9	0.19 ± 0.00*	0.08 ± 0.00	0.14 ± 0.001	0.54 ± 0.003*	0.12 ± 0.001	0.96 ± 0.01*
12	0.18 ± 0.02*	0.08 ± 0.00	0.14 ±0.001	0.51 ± 0.001*	0.12 ± 0.001	1.16 ± 0.008*
15	0.16 ± 0.02*	0.08 ± 0.00	0.12 ± 0.00*	0.50 ± 0.001*	0.10 ± 0.0006	1.13 ± 0.006*
18	0.16 ± 0.02*	0.08 ± 0.00	0.12 ± 0.00*	0.48 ± 0.0006*	0.10 ± 0.0006	1.23 ± 0.003*
21	0.15 ± 0.00*	0.0 8 ± 0.00	0.13 ± 0.00	0.49 ± 0.001*	0.11 ± 0.001	1.25 ± 0.005*
24	0.15 ± 0.00*	0.0 8 ± 0.00	0.13 ± 0.00	0.50 ± 0.00*	0.12 ± 0.00	0.95 ± 0.005*
30	0.10 ± 0.00*	0.0 8 ± 0.00	0.13 ± 0.00	0.51 ± 0.001*	0.12± 0.00	0.90 ± 0.00*
36	0.22 ± 0.03	0.0 8 ± 0.00	0.13 ± 0.001	0.56 ± 0.002*	0.13 ± 0.001	0.85 ± 0.005*
48	0.25 ± 0.00	0.0 8 ± 0.00	0.13 ± 0.001	0.59 ± 0.01*	0.13 ± 0.001	0.75 ± 0.02

* Values are significant at $P < 0.05$

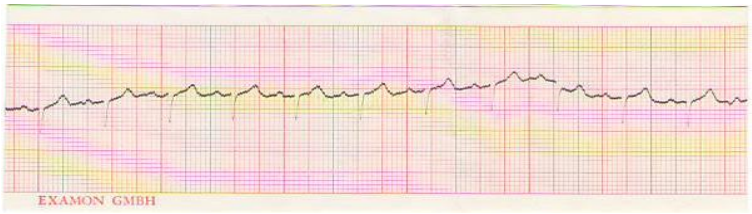

Fig 1. Normal heart rate before ruminal acidosis

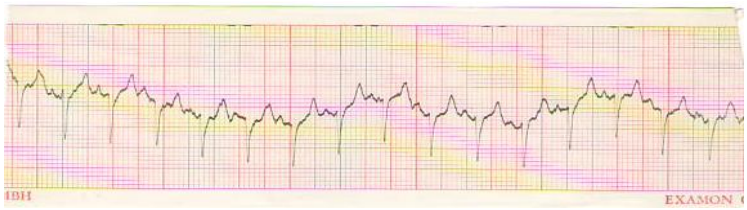

Fig 2. Sinus Tachycardia in ruminal acidosis

Discussion

The present finding will be compared largely with those experiment observed previously in sheep.[2-4] The reduction of ruminal pH to a minimum of 4.02 at 30 hours after the sucrose administration, caused most of the systemic and clinical changes observed in the present study. The ingestion of excessive quantities of highly fermentable feeds by a ruminant is followed within 2-6 h by a marked change in the microbial population in the rumen.[11,12] There is an increase in the number of *Streptococcus bovis*, which utilize the carbohydrate to produce large quantities of lactic acid. In the presence of a sufficient amount of carbohydrate the organism will continue to produce lactic acid which decreases the rumen pH.[2,3] Decreasing in ruminal pH occurred when lactic acidosis was induced in sheep by sucrose at a dose of 18 g kg^{-1} body weight.[13] This finding has much similarity to the earlier workers.[14,15] Lactic acid are Produced in the rumen which markedly increases ruminal osmolality, and water drawn in from the systemic circulation and accumulation of fluid within the rumen. In addition, gas production in the rumen results to ruminal distension and impedes venous return. These factors may be impaired hepatic perfusion and thus superimpose poorer lactate utilization and systemic acidosis occurs. Cao et al. (1987) recorded similar founding in acid-base disturbance in goat which given 18 g kg^{-1} body weight sucrose. Lal et al. (1993) reported a significant increase in P amplitude at 24 hours which remained elevated up to 96 hours, a significant increasing in amplitude of QRS complex at 24 hours onward, a significant decrease in PR interval from 48 to 72 hours, no significant changes in ST segment, a significant decrease in QT interval, a significant increase in T wave from 24 hours onward, in experimentally induced rumen lactic acidosis in goat. In the spite of difference between severity of our study and study of Lal et al. (1993), the ECG changes in our study were similar to this study.

The increase in the P wave amplitude was not to that extent where it can be attributed to right atrial hypertrophy. Further, increase in various cardiac potentials has been attributed to the increased absorption of lactic acid and/or volatile fatty acids from rumen.[8] Decrease in ST and QT intervals can be attributed to tachycardia. Shortening of QT interval has been inversely related to the total volatile fatty acid concentration. The increase in amplitude of T wave corroborated with metabolic acidosis. These data suggest that acute ruminal acidosis accelerates pacemaker activity in the heart, thus

increasing heart rate, but has no significant effect on the time course of the spread of the action potential through the ventricles. The decline of the PR interval could be due either to a rapider propagation of the action potential through the ventricles, or to rapider transmission of the action potential through the AV node. The above data suggest that the effect of acidosis on the ECG was due principally through its effects on the SA and AV nodes. Because Ito (depolarization produced by Na+ influx leads to activation of a transient outward current mainly carried by K+, this current is called Ito.) is known to be present in these nodes, play a role in their function, and to be altered by acidosis at depolarized membrane potentials. These data suggest, therefore, that the effects of ruminal acidosis on heart rate and on the PR interval are not due to changes in Ito. It has previously been suggested that an acidosis induced decrease in resting potential which might contribute to the TQ segment depression during ischemia.[6,16] However, such depression would only be expected during regional acidosis, whereas in the present study the entire heart was perfused with acid solution. The present data suggest either that regional differences in the response of cardiac cells to acidosis are too small to be detected by the ECG, or that electro tonic current spread in the intact heart minimizes such regional differences in the intact heart when all of the heart is made acid**.**[6,17]

In conclusion acute ruminal acidosis caused significant changes in ECG of sheep though there was not any detectable arrhythmia in the ECG in acute ruminal acidosis.

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