RESPIRATORY-METABOLIC ACIDOSIS

IN PERIPARTURIENT CALVES

By

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Submitted to the Faculty of the
Graduate College of the
Oklahoma State University
in partial fulfillment of
the requirements for
the Degree of
MASTER OF SCIENCE
December, 1988

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Thesis Approved:

Thesis Advisor

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PREFACE

This study was done in an attempt to explain the problems encountered by dystocia stressed calves. Calves born to an experimental group of heifers belonging to the Animal Science Department of Oklahoma State University provided the main body of the data along with dystocia cases presented to the Boren Veterinary Medical Teaching Hospital during the spring of 1987.

I would like to thank my major advisor, Dr. Lawrence E. Rice, for his idea for this research, his help and much needed advise.

Sincere thanks need to be extended to Dr. Gregor L. Morgan for his encouragement, time, and suppport during my residency. Also thanks to Dr. James E. Breazile for his enduring method of teaching physiology.

Thanks to David Cox, Mark Anderson, and David Gay of the south range without whose help and time this study could not have been accomplished.

And special thanks to Eva Harrall for her time in helping me to make this thesis presentable.

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CHAPTER I

INTRODUCTION

Dystocia remains the number one cause of perinatal calf mortality (10,11,20,29,30). Though a calf may survive the parturition process the effects of a dystocia can affect the ability of the calf to survive in the neonatal period (34).

The neonate is born in a state of respiratory-metabolic acidosis (20,24,25,26,27) and over the next 24 hours utilizes mechanisms to establish a normal pH of 7.4. Recognizing and understanding the normal processes in perinatal adaptation may aid in recognizing and treating abnormal cases.

In this study blood gas parameters and clinical observations will be evaluated for predicting viability of dystocia stressed calves. Evaluations should therefore include: 1) dystocia, 2) normal values for pH, HCO_3, pCO_2, and lactic acid at birth and 24 hours, and 3) calf viability scores.

The following null hypotheses will be tested: 1) no effect of dystocia on calf viability, 2) no effect of dystocia on acidosis, 3) no effect of acidemia on calf viability, 4) no effect of dystocia on lactic acid, and 5) no effect of lactic acid on acidemia.

CHAPTER II

REVIEW OF THE LITERATURE

Introduction

A normal fetus initiates the termination of pregnancy at the end of a normal gestation. Parturition is a time of major change for the fetus; the neonate is forced to adapt a fetal state of placental dependence to one of independence in his new environment. A viable calf is one that can make the necessary changes and adaptations. Dystocia places additional stress on the fetus at this critical time and can increase the chances of a less viable calf.

Parturition

Parturition marks the termination of pregnancy. It is a process that involves a highly coordinated sequence of maternal and fetal hormonal signals and events.

Initiation of parturition is from a fetal signal (1) which has been established to be fetal cortisol (2,3,4). The signal originates in the fetal brain with maturation of the hypothalamo-pituitary axis. ACTH from the fetal pituitary triggers an increase in cortisol production from the maturing fetal adrenal cortex (2,4,6). This rise in fetal cortisol begins six to seven days before parturition and a rapid rise occurs during the first stage of parturition (2,4,5).

The target of fetal cortisol is the placenta and its estrogen

production (3), altering placental steroidogenesis (4). Maternal estrogen concentrations are relatively high during the last quarter of gestation with a gradual increase over the last two weeks (2,3,5,6) and a rapid rise in the last four to five days of gestation (4). The concentration of maternal progesterone decreases gradually during the last 20 days of gestation (2,6) with a sudden fall one to two days before parturition (3,4,5). The increase in estrogen stimulates production and release of prostaglandin F (2,6), in contrast to the progesterone of pregnancy that acted to inhibit prostaglandin release from the endometrium (3,4). A sharp increase in prostaglandin F concentrations occurs the last 24-48 hours of gestation (5) incriminating prostaglandin F as the agent responsible for the lysis of the corpus luteum of pregnancy (3), a key event in parturition (2,3,4,5,6).

Once signals for initiation of parturition are set into action, delivery of the calf depends on coordinated rhythmic contractions of uterine smooth muscle, involuntary contractions of abdominal muscles, and on the relaxation and opening of the birth canal (3).

Increased myometrical electrical activity occurs with steady increases in uterine prostaglandin F concentrations (3,4). These initial contractions and vaginal distention stimulate the release of oxytocin (3,4). Positive feedback between oxytocin and prostaglandin F (3,4,6) produces high pulsate levels of oxytocin for the expulsion efforts during the final stages of labor (7). The effects of oxytocin occur under the influence of high estrogen and low progesterone concentrations with the end of gestation (2,3,4,6).

Dilation of the cervix and softening of the birth canal occurs with

high estrogen, prostaglandin F, oxytocin and relaxin levels (2,3,4,6,8). Though the exact mechanism remains unknown, administration of porcine relaxin to beef heifers during late gestation induces marked increases in cervical dilation and expansion of the pelvic area (8).

Clinically, parturition has been divided into three stages. Stage I involves the early myometrial contractions resulting in cervical dilation (3,9). This initial stage may take two to four hours (3,9). Stage II is characterized by abdominal contractions bringing the fetus into the dilated birth canal, rupture of the allantoic sac, and finally expulsion of the fetus (3,6,9). Delivery should be completed within two hours from the appearance of fetal membranes (3,9). Stage III involves the involution of the uterus and the expulsion of the fetal membranes (3,9). Fetal membranes should be expelled by 12 hours (3,9).

Dystocia

Dystocia is recognized when any stage in the parturition process is slow to develop or fails to progress normally (9,10). Causes of dystocia involve many factors: 1) relative fetal oversize (the size of the calf in relation to the pelvic area of the dam), 2) malpresentation, malposition, or malposture of the fetus, and 3) uterine inertia and incomplete cervical or vaginal dilation (10). Relative fetal oversize is the most important single cause (1,10), and in itself accounts for over 50 percent of all dystocias (3,10,11,12,13).

Classically, intervention and assistance in dystocia has been recommended when a cow has made no progress from Stage I or when the cow has made little or no progress in Stage II after two to three hours (3,9). However, recent studies indicate that Stage II may be shorter

than originally described, being as short as 30 minutes in cows and one hour in heifers (3). Also early intervention has had no detrimental effects on either the dam or calf (14,15). In fact a tendency towards decreased calf vigor has been seen with increases in length of Stage II (3) or with increases in calving difficulty (16). Positive correlations have been demonstrated between ease of calving and calf survival (17) and early obstetrical assistance may increase calf viability (18) and improve maternal postpartum return to estrus (15).

Perinatal Adaption

Parturition requires a large number of adaptive changes for the fetus as it leaves the "controlled" uterine environment and enters the external environment (19). The increase in fetal cortisol at near term is not only responsible for the initiation of parturition but is also involved in preparing the fetus for survival in its new environment (4). Cortisol is important for final lung maturation and surfactant production (2,3,4,6) along with the maturation and differentiation of other tissues (3,4). Neonatal cortisol levels within five minutes after birth are double those observed during labor (5).

The neonate's dependence on the placenta is terminated at birth, and it must immediately begin breathing to oxygenate blood and convert fetal to adult circulation (20). Episodic fetal respiratory movements (rapid, irregular breathing) become a continuous rhythmic breathing pattern in the neonate (24). At birth initial gasping movements (21), are followed by a period of panting before normal respiration (20,21). The hypoxia and hypercapnea (increased pCO_2) present at birth interact to stimulate respiration (20,21) and initial gasping and panting acts to

ensure a rapid expansion and aeration of the neonatal lungs. With the rise in plasma pO_2 and fall in pCO_2 as the lungs begin to function, rhythmic breathing and ventilation become established (20). Labor stimulated catecholamine release may also be important in pulmonary adaptation by increasing surfactant release and decreasing tracheal fluid production (22).

Fetal blood pH and blood gas parameters remain relatively stable during gestation (5,23) up until the last five minutes before actual delivery (20). With labor contractions there are concommitant transient periods of hypoxemia in the fetus (20). No changes in umbilical arterial or venous pO_2 are seen with uterine contractions unless accompanied by abdominal straining (24). These intermittent periods of hypoxia are overcome in the normal fetus by peripheral vasoconstriction and rebound increases in placental blood flow (20,24,25). These oxygen conserving mechanisms result in anaerobic glycolysis in those tissues with a minimal blood supply (25).

During asphyxiation the blood pO_2 falls rapidly and oxygen saturation is less than 10 percent within two minutes (20). Anaerobic breakdown of lactic acid provides the necessary energy for continued survival during hypoxia (19,20). The cardiac ventricles of most newborns contain relatively high glycogen concentrations for energy needed to maintain circulation. The brain of neonates contains virtually no glycogen reserves and therefore continued functioning is dependent on glucose from other stores, namely the liver (20). During asphyxiation, blood pH falls due in part to increasing pCO_2 but primarily to the production of lactic acid by the myocardium (20) and other tissues from anaerobic metabolism.

At birth there are major changes in the blood composition; pH and pO_2 fall, while pCO_2 and lactic acid rise (23,25,26,28). The neonate is born in a state of respiratory-metabolic acidosis (20,24,25,26,27). The labored breathing pattern of the newborn contributes to the rise in pCO_2 producing a respiratory acidosis. Accumulation of acid metabolites (primarily lactic acid) from anerobic metabolism during hypoxemic periods produces a metabolic acidosis (23,24,25). Studies in pigs and lambs have also shown that the fall in pH at birth was associated with an increase in blood lactic acid (23,28). This acidosis is a transient state in the normal calf which will employ normal mechanisms to compensate to establish a normal blood pH (25,26,27).

Calf Viability

A viable calf can be defined as one that is capable of an "independent" existence at or within a few hours of birth; one that has the ability to establish normal respiration, maintain body temperature, and gain mobility to obtain nurishment without assistance. The majority of perinatal losses occur between birth and 72 hours of age (10,20). Dystocia is the number one cause of perinatal calf mortality (10,11,20,29,30). Other causes include infectious diseases, trauma, chilling, starvation, congenital abnormalities, and predation (10,18,29). It is possible that some losses result from an inability in the calf to establish homeostasis and adapt in his new environment. In one study dystocia calves actually tended to have lower cortisol levels at birth than unassisted calves (31). Whether the cortisol levels were low as a result of dystocia or the dystocia a result of the low cortisol levels has not been determined.

During dystocia additional stresses are placed on the fetus. Combined with an increased incidence of anoxia during dystocia is the increased chance of trauma to the calf. After the initiation of Stage II of parturition the fetus with an intact placenta can survive for six to nine hours in utero (6,32). Prolonged hypoxia is an important cause of fetal demise during dystocia (1,20,33) and while not always immediately fatal can affect the ability of the calf to survive the neonatal period (34). At necropsy the cause of death in dystocia calves has been attributed to anoxia or asphyxia (32,34) with widespread hemorrhages a consistant finding (12,20,33,34).

Clinically, dystocia calves are less viable (10,30,34); they are slower to stand and nurse and are more susceptible to infectious diseases (10). These calves require extra nursing care over the first 12-24 hours of life.

The viability of newborn calves assessed by various means has been related to the severity of asphyxiation based on blood pH values (24,25,27,35). In one study the clinical state of a calf was shown to be related to the degree of metabolic acidosis present as determined from blood pH and bicarbonate values (25). As vital cell functions cannot take place in severe acidotic states (25) the neonate's ability to compensate and establish a normal pH is vital. Cell death can result from prolonged hypoxia and acidosis (25,27).

Calves that require substantial aid at birth show a more pronounced respiratory-metabolic acidosis than those born unassisted (25,27), and blood lactic acid concentrations increased with the degree of difficulty of calving (25,26). The pCO_2 values were shown to be similar between calves following dystocia and calves following normal parturition. This

may indicate that metabolic acidosis is more severe than is respiratory acidosis (26). Observations in lambs have also indicated that pathologic degrees of fetal hypoxia and acidemia may be associated with prolonged or vigorous ovine labor (33,36).

Acid Base Balance

Acid-base status constitutes an essential part of homeostasis (19). A stable pH in body fluids is essential (37) for life. Acid base balance is reflected in the elements of the Henderson-Hasselbalch equation:

$$pH = pK + \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

To maintain a constant acid-base balance in the internal environment a regulatory system of buffers is utilized (25,37,38,39). Buffers have the ability to resist changes in H+ ion concentrations by their ability to bind or release H+ in solution (37,39). Acids are defined as H+ donators while bases are H+ acceptors (37,39). The body's buffer systems function to keep the pH of extracellular fluid relatively constant at 7.4 even with the addition of acids or bases. Buffers are composed of a salt of a weak acid and a weak acid pair.

The major buffer system against acidosis in extracellular fluid is bicarbonate (25,37,38,39). Other buffers include phosphate, acetate, lactate, hemoglobin and plasma proteins (37,39).

The following equilibrium exists in the body and governs CO_2 transport and acid-base balance:

$$H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H+ + HCO_3^-$$

Carbonic anhydrase (CA) catalyzes the first reaction. Carbonic anhydrase of red blood cells and renal tubular cells and respiratory

elimination of CO_2 causes the equilibrium to be shifted far to the left in normal plasma (39).

A functioning respiratory system and renal system are essential in the maintenance of a stable acid-base balance. The lungs function rapidly (minutes) to remove CO_2 from the blood across alveolar membranes (37,39). This drives the equilibrium to the left removing H+ ions from the plasma and producing H_2O. The kidneys function more slowly (hours) to conserve HCO_3^- through reabsorption mechanisms in the renal tubules and to excrete H+ as titratable acids in the urine (37,39).

Acid base disturbances are divided into categories dependent on the cause for the disorder: 1) respiratory acidosis, 2) metabolic acidosis, 3) respiratory alkalosis, and 4) metabolic alkalosis (37,38,39). Blood gas values are used in determining these disturbances by interpreting pH, pCO_2, and HCO_3^- values. Respiratory disturbances refer to primary changes in blood pCO_2 causing pH changes, while metabolic disturbances refer to additions or losses of HCO_3^- causing pH changes (37,38,39).

Acidemia refers to a blood pH below 7.4. Respiratory acidosis results from an increase in blood pCO_2 due to decreases in ventilation-perfusion balance in the lungs (39). The renal tubules respond to compensate for the increased pCO_2 by enhanced reabsorption of HCO_3^-. Metabolic acidosis results from a decrease in HCO_3^- due to titration by acids or by loss. Anaerobic metabolism produces lactic acid (39), a significant source of H+, that titrates HCO_3^-. The body acts to compensate for the decreased HCO_3^- by decreasing pCO_2 levels with mechanisms that increase ventiliation to blow off CO_2, while the

kidneys assist by conserving HCO_3^- and excreting excess H+. These defense mechanisms function to restore pH to normal. While acidemia may no longer be present in fully compensated states, a state of acidosis exists with high pCO_2 or low HCO_3^-.

CHAPTER III

MATERIALS AND METHODS

Population

A group of 46 Hereford and Angus first-calf heifers were chosen for this study. Of these, 25 either calved before the study was initiated or calved unobserved and therefore no data could be obtained. Data collected from dystocia cases presented to the clinic also contributed along with an elective cesarean case. Data collected from a total of 26 calves was used in this project.

The group of heifers had been synchronized for breeding to give a shorter calving period. Several bulls had been used with pasture breeding. The heifers were maintained in the same pasture and were confined to a calving lot when impending parturition was noted. The heifers were fed in the late afternoon in an attempt to enhance parturition during daylight hours. All heifers were in good condition and health.

Sample Collection

Immediately upon delivery blood samples were taken from each calf. A venous blood sample was collected into a heparinized one cc glass syringe for blood gas analysis. Any air bubbles were expelled, the needle was capped with a rubber stopper, and the syringe was placed on ice (26). A vacutainer was used to collect the other blood samples. A

sodium heparin containing tube was filled and placed on ice until the plasma could be separated for lactic acid and glucose determination. An EDTA containing tube was filled for determining packed cell volume and total protein, and a clot tube was filled for serum immunoglubulin level determination.

At one hour and two hours post delivery blood samples were obtained for blood gas analysis, blood lactic acid, and blood glucose using the techniques described above.

At 24 hours of age blood samples were collected for blood gas analysis, blood lactic acid, blood glucose, packed cell volume, total protein, and serum immunoglobulin levels.

All samples were taken from the external jugular veins.

Sample Evaluation

Blood gas analysis was performed by a Corning 175 B.G. Analyzer determining pH, HCO_3^-, pCO_2, and pO_2. Samples were stored on ice for up to four hours before analysis was completed.

Blood lactic acid and glucose analysis was performed by a DuPont ACA II-60. Samples were stored on ice for up to four hours before the plasma was separated. Plasma samples were refrigerated over night if necessary until analysis was completed.

Method of Evaluation

Observation of Parturition

Heifers were evaluated at approximately eight months of gestation for signs of pending parturition such as increased udder development and vulvular edema (springing). At this time rectal pelvic measurements

were taken using a pelvimeter. Heifers were observed daily and when parturition was imminent (maximum springing and relaxation of the sacrosciatic ligament) the heifers were placed in a calving lot for closer monitoring. These heifers were watched through the day and checked every six hours through the night.

Observations were first recorded when a heifer began showing signs of discomfort and/or straining. Length of Stage II was determined by the time of first appearance of membranes to delivery of the calf.

Assistance was given if delivery was not completed by four hours or sooner if obvious distress was observed or normal progress ceased. Assistance included manual and mechanical extraction, and an episiotomy when indicated.

Parturitions were scored as: 1) eutocia, requiring no intervention, 2) token dystocia, relieved easily by manual traction, 3) dystocia, relieved by extreme manual traction or mechanical extraction, and 4) cesarian dystocia.

Observation of Calf Viability

Immediately upon delivery, the calf was evaluated using a Modified APGAR Scale for viability (47). Scores were interpreted as: viable = 7-8, depressed = 4-6, and nonviable = 0-3. Assistance was given to depressed calves to insure an open air passage and to stimulate breathing.

Calves were observed from the time of delivery until the calf nursed. The length of time that passed before the calf stood and nursed was recorded. If a calf did not stand and nurse by four hours post delivery assistance was given. Colostrum was given by a stomach tube if

the calf would not nurse and additional nursing care or treatment was given when indicated.

TABLE I

MODIFIED APGAR SCALE

	SCORE		
Parameter	1	2	3
Heart rate	Absent	<100/min.	>100/min.
Respiratory effort	Absent	Weak Irregular rhythm	Good Normal rhythm
Muscular tone and movement	Flaccid	Reduced	Vigorous movement
Suckle reflex	None	Reduced	Good

CHAPTER IV

ANALYSIS OF DATA

Analysis of the data was based on the following null hypotheses: 1) no effect of dystocia on calf viability, 2) no effect of dystocia on degree of acidosis at birth, 3) no effect of dystocia on blood lactic acid levels, 4) no effect of acidosis on calf viability, and 5) no effect of blood lactic acid on acidosis at birth. The null hypothesis assumes that there is no difference between treatment groups. The treatment groups consist of: 1) eutocia, 2) token dystocia, and 3) dystocia. Independent samples came from groups of unequal size.

Clinical observations and statistical analysis were used to test the effect of dystocia on calf viability and the effect of acidosis on calf viability. Statistical analysis of the collected clinical data was used to test the remaining null hypotheses. Analysis of variance was done on the mean values from the treatment groups to test the effect of dystocia on degree of acidemia (pH) and on blood lactic acid levels and to test the effect of dystocia on calf viability. Simple linear regression and least square estimation was used to test if a linear relationship existed for blood lactic acid and degree of acidemia (pH). A significance level of 5 percent was used for testing hypotheses.

TABLE II

DEGREE OF ACIDEMIA (pH) AT BIRTH

	Eutocia	Token Dystocia	Dystocia
	7.202	7.205	7.190
	7.227	7.239	6.933
	7.259	7.306	6.992
	7.195	7.202	7.155
	7.102	7.031	7.254
	7.173	7.031	7.254
	7.042	7.238	
	7.139	7.217	
	7.122		
$\bar{Y}i$	7.162	7.184	7.105

F value = 1.032

$p > .05$

TABLE III

LACTIC ACID (MMOL/L) AT BIRTH

	Eutocia	Token Dystocia	Dystocia
	5.5	5.9	11.1
	4.4	7.1	12.8
	10.3	7.0	15.0
	5.4	6.3	11.7
	12.1	6.8	6.1
	5.2	6.1	
	6.2	2.2	
	8.5	7.6	
	5.6		
$\overline{Y}i$	7.02	6.13	11.34

F value = 7.172

$p < .05$

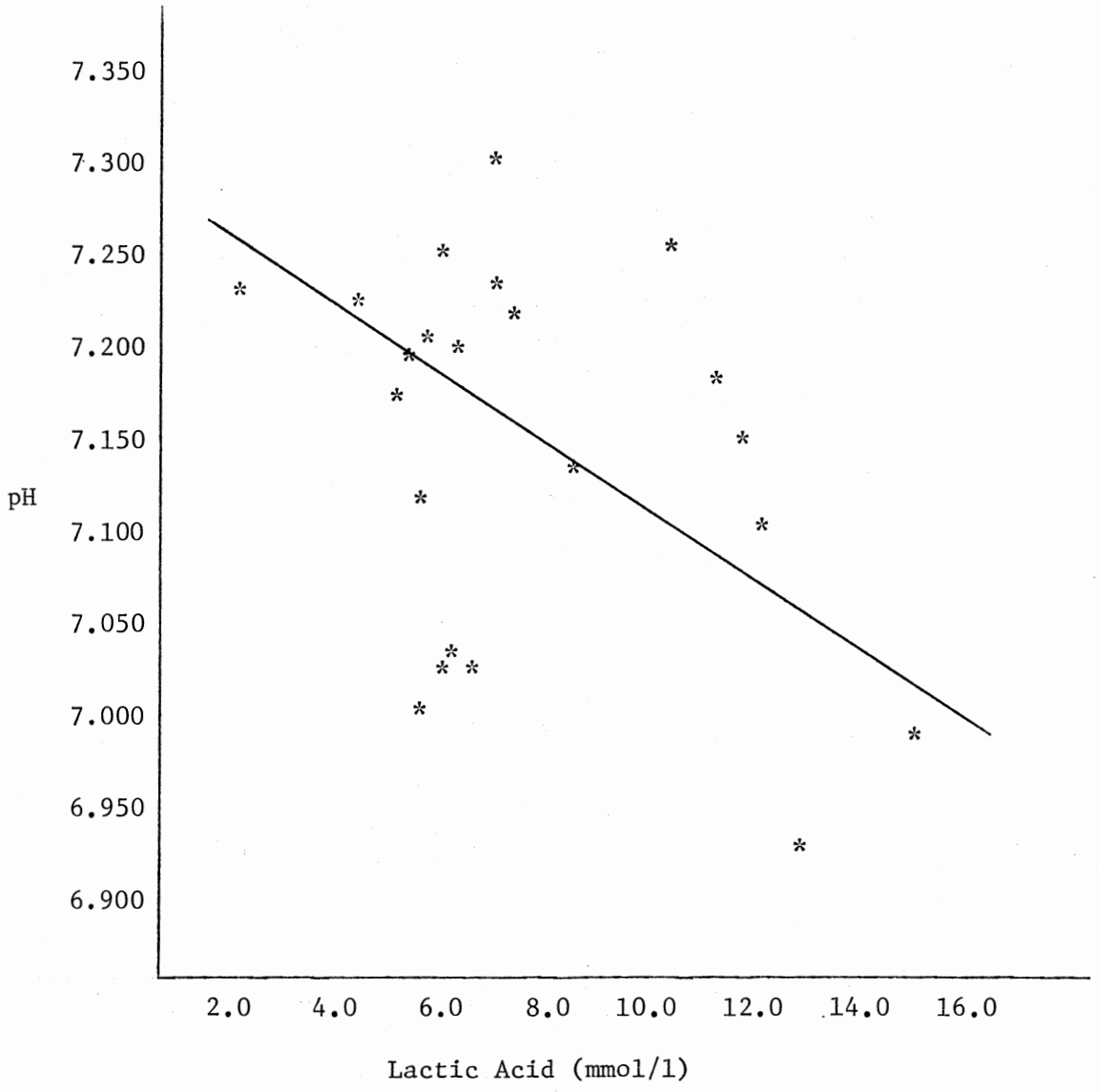

Figure 1. Plot of Lactic Acid and pH at Birth

TABLE IV

LACTIC ACID (MMOL/L) AND
DEGREE OF ACIDEMIA (pH)
AT BIRTH

Lactic Acid	pH
5.5	7.202
4.4	7.227
10.3	7.259
5.4	7.195
12.1	7.102
5.2	7.173
6.2	7.042
8.5	7.139
5.6	7.122
5.9	7.205
7.1	7.239
7.0	7.306
6.3	7.202
6.8	7.031
6.1	7.031
2.2	7.238
7.6	7.217
11.1	7.190
12.8	6.933
15.0	6.992
11.7	7.155
6.1	7.254
$\overline{X}$ 7.68	$\overline{Y}$ 7.157

$t = 2.27$

Correlation Coefficient = -.543

TABLE V

CALF VIABILITY (APGAR SCORE) AT BIRTH

	Eutocia	Token Dystocia	Dystocia
	8	8	7
	8	7	7
	8	7	7
	8	8	7
	8	8	7
	8	8	
	8	8	
	8	7	
$\overline{Y}i$	8.0	7.6	7.0

F value = 1.647

$p > .05$

CHAPTER V

RESULTS

Limitations and Error

Observation of parturition was done by herd managers and the principal investigator. Some heifers calved without observation making their calves unavailable for inclusion in this study as samples were to be collected within seconds of birth. The main obstacle encountered during the study was in allowing Stage II of labor to proceed for an extended length of time before intervening. Originally a limit of four hours was set before intervention was to be given but in actuality no heifers were in Stage II for longer than three hours before assistance delivered the calf. Those heifers that calved unassisted had lengths of Stage II of labor from one to two and one/half hours, while those that were given any assistance ranged from two to three hours in Stage II of labor.

The length of Stage II of labor for both the token dystocia and the dystocia treatment groups ranged from two to three hours. This made our effect of dystocia more related to the degree of trauma inflicted on the calf due to relative fetal oversize rather than an effect of a prolongation of Stage II of labor on the calf.

Only one heifer in the original study group required a C-section to deliver the calf due to malposture of the calf's head and a judgement of relative fetal oversize. As the sample collector was occupied as the

surgeon, no birth samples were taken. Data from two clinic C-section delivered calves was obtained during this study. Due to the small number of calves in the C-section catagory it was not included as a treatment group in the statistical analysis.

Acidosis and Lactic Acid

Previous studies found calves at birth to be in a state of respiratory-metabolic acidosis (23,24,26,28). In this study all calves were born in a state of acidemia having pH's less than 7.4 (range 6.849-7.306). While one study found dystocia calves to have significant deviations in pH from normal calves (26), this could not be confirmed with data from this trial. While the mean of the pH values for the dystocia treatment group (7.105) was lower than those for the other groups (7.162,7.184) this was not a statistical difference at the five percent level. Therefore, the null hypothesis that there was no effect of dystocia on acidosis cannot be rejected. Perhaps if more time for Stage II of labor had been allowed before a parturition was assisted the differences would have been more demonstratable by allowing a more sustained hypoxia to affect the calf.

Acidemia at birth has been described as a mixed respiratory-metabolic acidosis (23,25,26,28). Blood gas parameters (pH, pCO_2, HCO_3^-) were used to determine the acidotic state of the calves in the study. High pCO_2 levels (range 57.1-83.4) were found across the treatment groups, indicating a respiratory factor for the acidemia. Hypoxic periods during parturition generate high pCO_2 levels in the calf (20,25) presenting a respiratory acidosis to the neonate. A ventilation perfusion problem may be present at birth until

normal respiration is established. Fluid containing alveoli, circulatory shunts, and irregular breathing patterns in the newborn delay mechanisms that remove pCO_2 from the blood.

The determination of a metabolic factor responsible for the acidemia was less clear. Functioning as a buffer, HCO_3^- is consumed by lactic acid, the primary metabolite produced from anaeraobic metabolism during hypoxic periods of parturition. Statistical analysis of this trial's data found a negative correlation between lactic acid and pH values by least squares estimation. Higher lactic acid levels were related to lower pH levels at birth (see Figure 1). This evidence disputes the null hypothesis that there was no effect of lactic acid on acidemia. The lactic acid can account for a metabolic acidosis at birth.

Statistically the dystocia treatment group calves had significantly different lactic acid levels at birth (mean 11.3) from the eutocia group (mean 7.0) and the token dystocia group (mean 6.1). This coincides with other studies that found lactic acid levels in calves at birth to be related to the degree of calving difficulty (20,25,26). Since there was no difference in the length of Stage II of labor between the two groups given assistance, the higher lactic acid levels found in the dystocia group were likely produced by trauma and/or hypoxia due to relative fetal oversize in utero and the forced extraction. From this the null hypothesis that there was no effect of dystocia on lactic acid can be rejected. Although the dystocia calves had a statistical difference in lactic acid from the other groups, no statistical difference in pH was found between the treatment groups. Again it is a combination of respiratory acidosis and metabolic acidosis that produces the acidemia

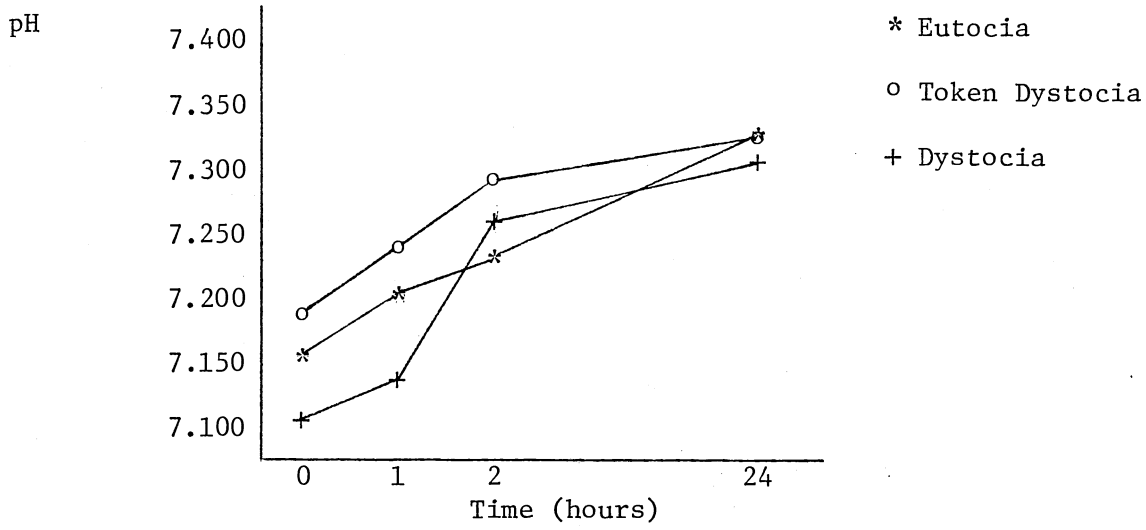

Figure 2. Mean Changes in pH After Parturition

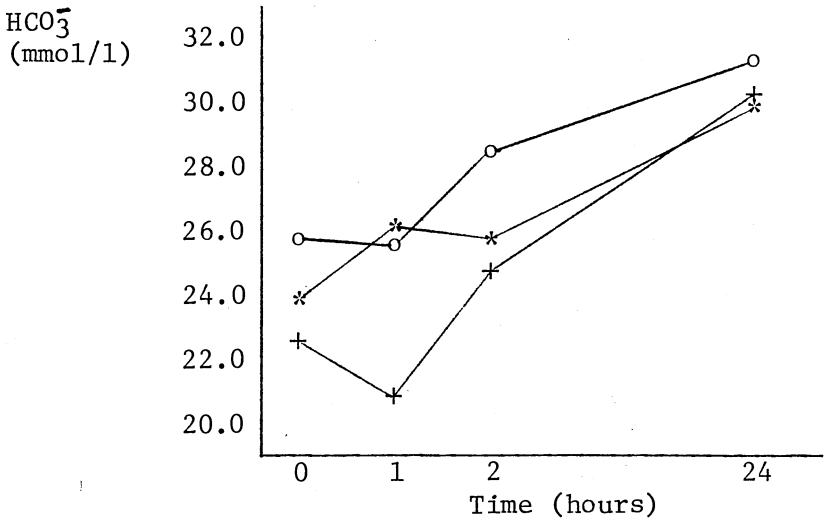

Figure 3. Mean Changes in HCO_3^- (mmol/1) After Parturition

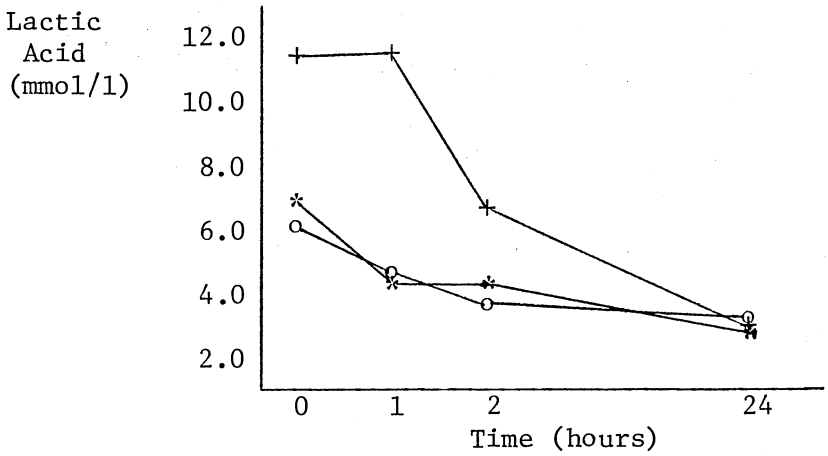

Figure 4. Mean Changes in Lactic Acid (mmol/1) After Parturition

seen at birth.

Over the 24 hours following birth blood pH and HCO_3^- increased while pCO_2 and lactic acid declined (see Figures 2,3,4). At 24 hours there was little difference in these values between treatment groups. While some calves had successfully compensated to a normal pH of 7.4, the majority of the calves in all treatment groups remained slightly acidemic (overall range 7.223-7.413). Using pCO_2 and HCO_3^- values, this was defined as a state of partially compensated respiratory acidosis seen with high pCO_2 levels (means 57.2, 59.5, 61.6) and high HCO_3^- values (means 30.0,31.4,30.2). By 24 hours lactic acid values (means 3.1, 3.5, 3.4) had declined significantly from birth levels indicating metabolic factors were probably no longer complicating the acidemia.

Calf Viability

Clinical observations were used to access calf viability at birth and the calves were scored using a Modified APGAR Scale (Table I). All but one of the calves in the eutocia treatment group scored the maximum 8 points. The exception was a calf that was born with fetal membranes covering its muzzle who was quite depressed and received a score of 4. Token dystocia group calves all received either 7 or 8 points for viability while the dystocia group calves all scored 7 points. The main distinction between calves that received 7 points versus 8 points was a reduction in muscle tone and movement at birth. All the dystocia treatment group calves appeared slightly duller with slower responses when compared to eutocia calves. Statistically no difference was found in the means of the APGAR scores between the treatment groups and

therefore the null hypothesis that there was no effect of dystocia on calf viability cannot be rejected. However, clinically the dystocia calves consistently appeared slightly depressed. This is similar to findings from other studies that relate calf viability with the degree of calving difficulty (3,10,30,34). Calves in the dystocia treatment group were slower to stand and nurse, but by 24 hours there were no noticable differences between dystocia calves and eutocia calves.

Inadequate evidence was found to reject the null hypothesis that there was no effect of acidosis on calf viability. This differs from other studies that found calf viability to be related to the degree of metabolic acidosis present at birth (25). No significant difference in the means of the pH values at birth could be demonstrated to coincide with the difference in viability seen between the treatment groups. While no difference in the degree of acidemia was found, these dystocia calves did have a significant increase in lactic acid levels from eutocia calves perhaps indicating a more severe metabolic acidosis did exist.

The calf with the lowest APGAR score (4 points) was clinically very depressed and dull. The calf's muzzle appeared blue and cyanotic when the fetal membranes were removed from its face. Assistance was given to help stimulate breathing and within minutes the calf was pink. This calf's pH was higher than the mean for eutocia group (7.195 versus 7.162) and its lactic acid was low (5.4 versus 7.0). The calf required assistance at 4 hours to nurse and at 24 hours could stand but had a noticeble head tilt to the right. The calf's blood parameters at 24 hours were comparable to the rest of the eutocia group. This calf was not included in the statistical analysis of calf viability due to the

complication from fetal membranes causing hypoxia.

Discussion

One extreme dystocia case that was presented to the clinic during this study seems to illustrate some conclusions. The cow presented with a history of trying to calve for at least 12 hours. The calf was in a caudal presentation and due to relative fetal oversize was delivered by C-section. At birth the calf was given a viability score of 6, lacking in muscular tone and movement and in respiratory effort. The calf was given assistance to stimulate breathing.

At birth this calf's pH was 6.849 with a HCO_3^- of 9.9 (mmol/l) and a pCO_2 of 57.1 (mmHg). Lactic acid was 30.7 (mmol/l) which was twice that of dystocia treatment group calves. Since no trauma was inflicted during delivery by C-section this high lactic acid reflects a build up from hypoxic periods during Stage II of labor. The high lactic acid and low HCO_3^- are reflected in the low pH for this calf while the pCO_2 was comparable to other dystocia calves. The calf suffered from a severe lactic acidosis that complicated the respiratory acidosis.

While this calf was severely stressed, its glucose at birth was only 26 mg/dl. By 2 hours it had dropped to 8 mg/dl. This may reflect an exhaustion of limited glucose reserves in the neonatal calf with extended periods of anoxia.

By two hours the lactic acid levels were slowly declining while the pH and HCO_3^- were slowly rising. The calf required assistance and was tubed at two hours in order to receive colostrum. Even with assistance the calf never stood up and died by 24 hours. This calf seemed to display an inability to overcome severe acidosis by normal mechanisms.

This study addresses some of the problems presented by the dystocia calf that may dictate treatment regimes. Depressed calves at birth should receive stimulation to help initiate breathing as well as assistance in obtaining colostrum and temperature control. As traumatized calves may suffer from a more severe metabolic acidosis due to increases in lactic acid, an infusion of a buffer is indicated (25). At this time $NaHCO_3$ is the most widely used buffer. Dextrose infusions could be helpful to those calves suffering from extended periods of stress, as they have a limited reserve of glycogen. The severely depressed dystocia calf must be given a poor prognosis, as even with aggressive treatment these calves may fail to survive (26).

A practice of early intervention followed by correct assistance may reduce calf losses in dystocias (18). In this trial, calves born unassisted were delivered within two hours of Stage II of labor. Further prolonging of Stage II of labor only increases the potential for periods of hypoxia in the calf.

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APPENDIXES

APPENDIX A

BLOOD pH, HCO_3^-, pCO_2, AND LACTIC ACID AT BIRTH

TABLE VI

BLOOD pH, HCO_3^-, pCO_2 AND LACTIC ACID AT BIRTH

	Eutocia	Token Dystocia	Dystocia
pH			
mean	7.162	7.184	7.105
range	7.042-7.259	7.031-7.306	6.933-7.254
HCO_3^- (mmol/l)			
mean	24.0	25.8	22.4
range	20.4-26.4	21.0-30.2	15.0-28.6
pCO_2(mmHg)			
mean	67.1	69.2	70.1
range	59.0-83.4	60.7-83.4	60.6-80.4
Lactic Acid (mmol/l)			
mean	7.0	6.1	11.3
range	4.4-12.1	2.2-7.6	6.1-15.0

APPENDIX B

BLOOD pH, HCO_3^-, pCO_2, AND LACTIC ACID AT 24 HOURS

TABLE VII

BLOOD pH, HCO_3^-, pCO_2 AND LACTIC ACID AT 24 HOURS

	Eutocia	Token Dystocia	Dystocia
pH			
mean	7.329	7.329	7.303
range	7.223-7.413	7.256-7.388	7.287-7.318
HCO_3^- (mmol/l)			
mean	30.0	31.4	30.2
range	24.4-33.4	27.8-35.5	27.9-33.3
pCO_2 (mmHg)			
mean	57.2	59.5	61.1
range	50.0-71.7	55.7-65.0	56.1-65.1
Lactic Acid (mmol/l)			
mean	3.1	3.5	3.4
range	1.6-4.5	1.8-6.6	2.9-3.3

APPENDIX C

REGRESSION LINE DETERMINATION FOR LACTIC ACID AND pH USING LEAST SQUARES EQUATION

REGRESSION LINE DETERMINATION FOR LACTIC ACID

AND pH USING LEAST SQUARES EQUATION

$$\hat{Y} = \hat{\beta}_o + \hat{\beta}_1 X$$

$$\hat{\beta}_1 = \frac{\Sigma XiYi - n\bar{X}\bar{Y}}{\Sigma Xi^2 - n\bar{X}^2}$$

$$\hat{\beta}_o = \bar{Y} - \hat{\beta}_1 \bar{X}$$

$$\hat{Y} = 7.284 + (-.0166)(X)$$

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