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Predictive measures of fetal distress in calves during delivery

by

Kristin Elizabeth Hard

A thesis submitted to the graduate faculty

in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

Major: Animal Physiology

Program of Study Committee:
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Ames, Iowa

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CHAPTER ONE

GENERAL INTRODUCTION

Thesis Organization

The following thesis is organized into 3 chapters and one appendix. Chapter 1 is a review of the physiological adaptations at birth that occur in the fetus at birth as well as the implications of dystocia. Chapter 2 is a summary of research conducted to characterize the predictive measures of fetal distress in calves during delivery. General conclusions about the research conducted are in Chapter 3. Appendix A contains the color chart used throughout the research.

Review of Literature

Physiological Adaptations at Birth and Implications of Dystocia

Introduction

The process of parturition that is initiated by the fetus involves a cascade of endocrine events that leads to myometrial contractions, dilation of the cervix, expulsion of the fetus, and finally the expulsion of the placenta (Senger, 1999). This transition from fetal to neonatal life is the most traumatic and dramatic change that occurs during the life of the animal. Factors that disrupt either the fetal or maternal system during birth may result in dystocia, or an abnormal birth. According to Berger (1996) there has been a decrease in the percentage of unassisted births and an increase in the percentage of births requiring considerable force or extreme difficulty. Estimates of the rate of calf mortality range from 7 to 25 percent with the majority of deaths occurring at or around birth or during the first week of life (Dennis, 1981; Rice and Wiltbank 1972; Laster and Gregory

1973; Berger, 1995). Therefore, there is a great need for information regarding calving intervention strategies as well as possible fetal monitoring protocols for use on dairies to ensure a lower calf mortality rate (Mee, 2004; Uysterpruyst et al., 2000; Bleul and Kähn, 2008).

Initiation of Parturition

The initiation of parturition has been most clearly defined in sheep. In the ewe, the signal for parturition originates in the fetus via the fetal hypothalamus. Several days prior to birth, the fetal adrenal gland secretes an increased amount of cortisol. This increase in fetal cortisol affects placental function. There is a resulting decrease in the secretion of progesterone and an increase in estrogen secretion from placental tissue. Increased placental estrogen induces an increased concentration of prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) in the maternal cotyledons and myometrial tissue. Elevated levels of $PGF_{2\alpha}$ induce a heightened sensitivity to oxytocin in the myometrium. The increased responsiveness to oxytocin causes distention and dilation of the cervix and induces uterine contractions which ultimately results in expulsion of the fetus (Liggins et al., 1973).

In cattle, the role of fetal cortisol in the initiation of parturition is less clear than in sheep. In the calf, plasma cortisol levels begin to progressively rise at about nine days before parturition. There is a sharp rise in the concentration of fetal cortisol at the onset of parturition; maximum levels in the fetus are reached within 3 hours of birth (Comline et al., 1974). However, during this same time period maternal cortisol concentrations remain relatively low (Fairclough et al., 1975). The increase in bovine fetal cortisol concentrations in the last days before parturition is similar to that seen in the lamb,

suggesting that the fetal adrenal is similarly involved in the control of bovine parturition (Fairclough et al., 1975; Hunter et al., 1977).

Progesterone concentrations in the cow slowly decline to approximately 4 ng/ml plasma one week before delivery, and then decline further to less than 1 ng/ml plasma in the final 24 h before delivery (Stabenfeldt et al., 1970). This decline in the concentration of progesterone concentration appears to be a necessary component in the initiation of parturition (Stabenfeldt et al., 1970). In the last 24-48 h prior to parturition, $PGF_{2\alpha}$ concentrations rise sharply to a maximum of 5.5-9.0 ng/ml, peaking at delivery in the cow (Fairclough et al., 1975). The increase in the concentration of prostaglandin causes luteolysis and therefore a sharp decline in plasma progesterone levels preceding delivery of the fetus (Edqvist et al., 1978; Fairclough et al., 1975).

The main source of estrogen in late pregnancy is the fetal placenta (Hoedemaker et al., 1990). The increase in fetal adrenal activity stimulates estrogen production in the placenta (Comline et al., 1974; Hunter et al., 1977). Fetal cortisol directly affects steroid synthesis in the trophoblast cells of the placenta (Hoedemaker et al., 1990).

In cattle, myometrial oxytocin receptor concentrations increase gradually, reaching the highest density near delivery. At the start of labor, decreases in receptor density occur. Significant increases in maternal oxytocin levels occur during labor and secreted into the maternal blood of the cow. This surge is thought to be the cause of myometrial contractions during parturition (Fuchs et al., 1992)

Intercaruncular endometrial oxytocin receptors also increase in concentration during pregnancy and reach maximum levels at the onset of parturition (Fuchs et al., 1992). There is an interaction of oxytocin with its endometrial receptors which leads to

the release of $PGF_{2\alpha}$. This oxytocin-induced release of $PGF_{2\alpha}$ enhances the contractile force of uterine contractions initiated by oxytocin alone. This interplay of $PGF_{2\alpha}$ and oxytocin induces uterine contractions and thus the expulsion of the calf (Fuchs et al 1992).

Stages of Parturition

Parturition or birth of the fetus is classically divided into three stages: stage one consists of increased periods of restlessness of the cow and positioning of the fetus in preparation for delivery, stage two involves the expulsion of the fetus, and stage three involves the expulsion of the fetal membranes.

The first stage of parturition is often characterized by the obvious discomfort of the cow. This discomfort is often shown by restlessness, rapid flicking of the tail, bellowing, kicking at the abdomen, and attempts by the cow to separate from the herd. During this time, the calf moves into delivery position and the pressure of the amniotic sac on the opening of the cervix initiates cervical dilation (Straiton, 1994). Regular contractions of the myometrium begin to occur usually at the rate of 12-24 contractions per hour. This stage of parturition usually lasts between 6-24 hours, but can be shorter if it is a multiparous cow (Peters and Ball, 1995).

The second stage of parturition involves the expulsion of the calf. This stage is characterized by the onset of regular contractions. These myometrial contractions force the calf backwards toward the pelvic cavity causing abdominal contractions. These contractions occur in a series of five or six, and each series last from 60 to 90 seconds (Schrag and Singer, 1992; Straiton 1994). The pressure of the fetus on the cervix stimulates the release of oxytocin which in turn leads to further contractions. As the

contractions progress the amniotic sac ruptures which will provide lubrication for the birth. During this stage of labor the frequency and duration of the contractions will cause the cow to lie down. The cow will strain from 0.5 hours to 4 hours until the explusion of the fetus is complete (Peters and Ball, 1995). The umbilical cord will break during the delivery process.

After the expulsion of the fetus and the abdominal contractions cease; the myometrium still contracts to expel the placenta. This expulsion of fetal membranes is considered to be the third and final stage of parturition. This stage can last up to 6 hours after birth (Peters and Ball, 1995). If the placenta is not expelled it is termed 'retained placenta' which is a postpartum disorder and has pathological implications (Peters and Ball, 1995; Straiton, 1994).

Changes from fetal to neonatal life

Respiration and Circulation

During fetal development, respiration and circulation is a function of the shared placenta. Fetal lungs are essentially secretory organs which play no role in respiratory gas exchange. Rapid removal of the fluid located in the lungs during or soon after birth is an essential event involved in the switch from placental to pulmonary gas exchange (Bland and Nielson, 1992). In fetal sheep, it has been found that lung luminal fluid contains almost no protein, a bicarbonate concentration of less than 3 mEq/L, and a Cl^- concentration 50% greater than that of fetal plasma (Adams et al., 1963). The volume of liquid in airspaces of fetal lambs increases from 4 to 6 ml/kg body weight at mid-gestation to more than 20ml/kg near term. This increase in lung liquid volume reflects the increase in pulmonary vascular and epithelial area due to proliferation of lung capillaries

and respiratory units (Bland and Nielson, 1992). The secretion of lung liquid begins to decrease 2 to 3 days prior to spontaneous birth. Epinephrine inhibits secretion of fetal lung liquid and also leads to an increase in the concentration of surfactant in the liquid (Brown et al., 1983). There is a sharp rise in plasma epinephrine (adrenaline) concentration detected during the last part of labor (Brown et al., 1983). The absorption of the fluid in the lungs observed during the latter part of labor is mediated by adrenaline circulating in the blood (Brown et al., 1983). Epinephrine stimulates Na^+ uptake by the respiratory tract epithelium which drives liquid from the lung lumen into the interstitium where it is absorbed into the blood stream or removed through lymphatics (Bland and Nielson, 1992).

During the fetal stage of life the ductus arteriosus and foramen ovale function to divert blood away from the lungs. Shortly after birth, circulation in the fetus is altered. When the first breath is taken by the newborn, there is a decrease in pulmonary vascular resistance and an increase in pulmonary blood flow (Dawes et al., 1953). The interruption of umbilical circulation is also followed by a fall in heart rate and a rise in systemic arterial pressure (Assali et al., 1962). This leads to a change in blood flow through the ductus arteriosus. Circulation is reversed and blood flows from the aorta to the pulmonary trunk (Dawes et al, 1953; Amoroso et al., 1957; Assali et al., 1962). Also, during fetal life there is a difference in heart pressure gradients when compared to the adult. Before birth, pressures in the chambers of the right side of the heart are consistently higher than those of the left chambers. The right side of the fetal heart is more muscular and represents the high pressure system (Assali and Morris, 1964). Therefore, it can be concluded that the right ventricle of the fetus performs more work than the left ventricle. After birth, both

the ductus arteriosus and foramen ovale close, the left ventricle performs more work than the right, and overall patterns of circulation occur as in the adult animal. Closure of the ductus arteriosus and foramen ovale occurs primarily due to the response to high P_{O2} in the blood and the change in pulmonary and systemic pressures (Walsh and Lind, 1978).

Blood Gasses and Acid-Base Balance

Fetal blood gas tensions differ from those in the neonate. Mean values for P_{O2}, P_{CO2}, and pH range between 38-40 mmHg, 43-45 mmHg, and 7.384-7.395, respectively (Comline et al., 1974). Blood gas tensions and pH remain stable in the fetus until a few minutes prior to birth. In the time immediately after birth there is a decrease in blood pH to approximately 7.25 and an increase in P_{CO2} to approximately 65 mm Hg. (Comline et al., 1974). Stable P_{O2} levels are reached within 15 min of birth and pH is restored within 1 hour in unstressed calves (Comline et al., 1974). Concentrations of electrolytes and lactate increase steadily in the first few hours after birth (Strawn et al., 1996). The base excess, or titrateable base in blood, tends to decrease more for stressed calves for the first 3 h after birth compared to unstressed calves (Strawn et al., 1996). Strawn et al. (1996) reported that arterial blood $[H^+]$ in eutocic calves is 5.0×10^{-8} and 6.7×10^{-8} in dystocic calves. However, there was no difference reported between dystocic and eutocic calves in regards to arterial P_{CO2}. Arterial P_{O2} was reported to be 32 mm Hg in dystocic calves and 41 mm Hg in normal calves. Most abnormal values in dystocic calves reached normal values by 16 h of age; however, concentrations of hemoglobin and oxygen tensions remained low in arterial blood into the second day of life (Strawn et al., 1996).

Renal Function

In most neonatal mammals, the renal system is immature at birth in respect to glomerular filtration rate, tubular function, and renal plasma flow (Dalton, 1968c). However, in the neonatal calf the renal system is considered to be far more efficient and renal function is comparable to adult renal function. The neonatal calf produces highly concentrated urine when dehydrated and readily excretes excess fluid (Dalton, 1968b). Also, the calf's urea clearance is similar to that of an adult human in contrast to a typical mammal neonate (Dalton, 1968b). Approximately half of the urea secreted in the glomeruluar filtrate is reabsorbed in the tubules (Dalton, 1968c). The ability of the calf to maintain fluid homeostasis is of importance because the primary source of nutrients is milk (Dalton, 1968a).

Thermoregulation

During fetal life the calf does not need to thermoregulate because heat is transferred to the fetus via the placenta and uterus. Fetal temperature regulation is dependent on maternal heat transfer until birth. After the calf is born it is directly exposed to the extrauterine environment and it utilizes thermogenic processes to counteract heat loss. The newborn calf often faces immediate thermal stress at birth (Asakura, 2004). After the first hour of life the calf's temperature will begin to stabilize at around 38.8°C. The postnatal drop and rise in temperature corresponds to the evaporation of amniotic fluid and drying of the calf's hair coat (Vermorel et al., 1983). Heat production in newborn animals is the result of the metabolic rate of body tissues, the metabolism of brown adipose tissue, shivering, and physical activity (Vermorel et al., 1983, Asakura, 2004).

There are two types of adipose tissue, brown adipose tissue and white adipose tissue. In white adipose tissue, the sources of fat deposited are triacylglycerols arising either from blood lipoproteins or in situ synthesis. The presence of white adipose tissue reserves are relevant to the newborn due to the role it plays in metabolism after birth. White adipose tissue reserves increase postnatal survival because the newborn can use these fat deposits as energy reserves to prevent accidental starvation (Medina et al, 1992). Brown adipose tissue is located in the perirenal, inguinal, and prescapular regions at about 2% of total body weight (Vermorel et al., 1983). Brown adipose tissue differs morphologically and metabolically from white adipose tissue. Brown adipose tissue contains many mitochondria, fat vacuoles, and abundant sympathetic innervation, and increased blood supply. In the mitochondria of brown adipose tissue, ATP synthesis is uncoupled from the oxidative process via an uncoupling protein. This protein provides a system for continuous heat production by channeling protons across the inner mitochondrial membrane which dissipates the membrane potential (Medina et al., 1992). When ATP synthesis is separated from the oxidative process then heat is produced that is necessary for non-shivering thermogenesis. During the first month of life, brown adipose tissue is rapidly converted to white adipose and it begins to react less and less (Vermorel et al., 1983).

Metabolism

In utero, the fetus is solely dependent on maternal sources of energy. The placenta permits transport of sugars, amino acids, vitamins and minerals to the fetus for substrates for fetal metabolism and growth. The placenta also serves as a storage organ for glycogen as well as iron (Riddle and Tyler, 2003).

At birth, the newborn calf must assume control of nutrient metabolism. The first several hours of life for the neonate are crucial for survivability. During these first few hours, physiological changes are tremendous and energy demands are high. Fructose is synthesized in the placenta and is present in large quantities in the blood of fetal ruminants (Alexander et al., 1970). Fructose is not metabolized by the fetus under normal conditions, but it may act as a reserve carbohydrate for utilization when glucose concentrations are low (Alexander et al., 1970). Fructose concentrations are high in lambs and in calves but diminish during the first 24 hours of life (Alexander et al., 1970; Kurz and Willett; 1991).

In contrast, glucose levels are low in the fetus and at birth, but increase to normal concentrations by 6 h of birth in the ruminant neonate (Kurz and Willett, 1991). Approximately 70% of the glucose transferred from mother to fetus is used for placental metabolism (Ktorza and Ferre, 1992). Some glucose is metabolized in the placenta to lactate. A significant amount of the lactate produced is transferred to the fetus, which leads to lactate concentrations 2 to 3 fold higher than that of the mother (Ktorza and Ferre, 1992). Glucose also contributes to energy storage in the form of hepatic and muscular glycogen. In sheep, 60% of glucose is oxidized immediately, whereas, 40% is taken up by the fetus and used for glycogen accretion. Accumulation of hepatic glycogen is particularly important in terms of energy homeostasis. Liver glycogen stores are utilized when placental transfer of glucose abruptly stops at parturition (Ktorza and Ferre, 1992).

Concentrations of free amino acids are 2- to 3-fold higher in fetal blood than in maternal blood. This suggests that amino acids are actively transported across the

placenta to the fetus. Amino acids are involved in oxidative metabolism in the fetus and contribute primarily to protein accretion (Ktorza and Ferre, 1992).

Non-esterified fatty acids and ketone bodies readily cross the placenta to the fetus. These substrates are a poor source of energy, but they are used for the synthesis of energy stores (Ktorza and Ferre, 1992).

Dystocia

Dystocia is defined as any abnormal or difficult delivery process, encompassing malpresentations, prolonged parturitions, and difficulties due to inappropriate assistance. In order to quantify dystocia, many scoring systems have been devised. The most commonly used system in the United States is the 5-point calving ease scale. A calving ease score of 1 indicates that no assistance was provided, but provides no assurance that assistance was not needed. Where as, a calving ease of 5 is a difficult parturition resulting in severe mechanical assistance. However, a calving ease score of 5 may be the result of an uncorrected malpresentation of the fetus, or inappropriate timing of assistance. Despite inconsistencies and the subjective nature of the scoring system, it serves as a quantitative asset (Mee, 2008b; Mee 2004).

Causes of Dystocia

Maternal factors

Dystocia caused by maternal factors account for 24% of all dystocial births. The most frequent abnormality associated with the dam is the failure of the cervix and vagina to dilate completely. Sloss and Johnson (1967) reported that there was a 53% maternal death loss in cases of improper dilation. Maternal death rate is most affected by maternal causes of dystocia (32%) compared to fetal causes (13%) (Sloss and Johnson, 1967).

There is also interplay between maternal pelvic size and calf size that affects dystocia. Feto-pelvic disproportion is by far the most common factor associated with dystocia in domestic dairy cattle (Mee, 2008b). Maternal pelvic size, although important, is difficult to assess. Accuracy of pelvic measurements is questionable; measurements can vary with the same animal with different technicians and tools. Because of these issues, the predictive power of these measurements prior to calving for pelvic size at calving is extremely poor (Gaines et al., 1993).

Age, parity and body weight of the dam are other factors associated with dystocia. Reported dystocia rates range up to 83% depending on breed and maternal age (Rice and Wiltbank, 1972; Laster et al., 1973; Philipsson et al., 1979). Primiparous cows have a much greater incidence of dystocia (Pollack and Freeman, 1976). Sieber et al. (1989) reported that over 50% of first parity births required assistance. Dystocia scores differ among parities. Lombard et al. (2008) reported that 18.9% of calves born to primiparous dams were classified as severe dystocias, but only 6.9% of calves born to multiparous dams were classified as severe dystocias. Dystocia in 2 year old cows is 36% higher than in 3-year olds and 45% higher than in 4- and 5-year old cows (Laster et al., 1973).

Some factors affecting dystocia are breed-related. Holsteins have the highest incidence of dystocia of any dairy breed averaging a 40% incidence rate. Holsteins have the highest ratio of calf birth weight to dam body weight; it averages 7.1%, but it is often over 10%. In contrast, Jersey dams rarely suffer from prolonged or difficult deliveries. The calf to dam weight ratio for the Jersey breed averages 5.6 to 6.3% and rarely exceeds 8% (Holland and Odde, 1992). Crossbreeding cattle results in a lower incidence of

dystocia. Holland and Odde (1992) reported 15.9% calving difficulty for Jersey x Holstein cattle.

Dystocic cows have altered hormonal profiles when compared to cows experiencing normal parturition. Progesterone concentrations in cows experiencing dystocia are higher than cows experiencing no dystocia (O'Brien and Stott, 1976). This increased progesterone concentration may have an inhibitory effect on the circulating estrogen. Cows experiencing dystocia have two to four times lower estrogen concentrations than that of normal cows between 23 days to 10 days prepartum (O'Brien and Stott, 1976).

Fetal factors

The most common fetal cause of dystocia is calf size (Rice and Wiltbank, 1972; Sloss and Johnson, 1967; Mee, 2008). McDermott et al. (1992) found birth weight as the most important factor in predicting dystocia. Odds of dystocia increase by 13% per kg increase in calf birth weight (Johansen and Berger, 2003). In Holstein cattle the threshold of calf birth weight lies between 42 and 45 kg; for any calf above this threshold, dystocia rates increase significantly (Menissier and Foulley, 1979). Also, birth weights above the average of 40.3 kg have an exponentially increasing risk of mortality (Johanson and Berger, 2003). Probabilities of mortality for birth weights of 29, 35, 40, 46, and 52 kg are 2.1, 2.5, 3.4, 5.1, and 9.6%, respectively (Johanson and Berger, 2003). However, since calf size is related to sex of the calf, this may be an effect of prolonged maternal exposure to testosterone (Bellows et al., 1993), but most researchers assume that male calves have more difficulty due to the fact that they are simply larger at birth (1-3 kg) than their female counterparts (Meijering, 1984; McDermott, 1992; Mee 2008).

Environmental factors

The incidence of dystocia is variable across seasons. The highest incidence rate occurs in late fall and winter periods (Lindhe, 1966). There can be several explanations for this phenomenon, although the most intriguing being the possibility of heat stress (Collier et al., 1982). Exposure to high environmental temperatures affects pregnancy and the fetus in several ways. These high temperatures during mid-gestation or during the third trimester restrict placental development and depress fetal development to term (Shelton, 1964; Bell et al., 1987; Collier et al., 1982).

Effects of Dystocia

Maternal

Dystocia can have dramatic effects on the cow. According to the National Animal Health Monitoring System, calving problems or dystocia is the leading cause of death in dairy cows (NAHMS, 1996). Cow deaths increase 4.1% for cows that experience extreme difficulty calving when compared to cows that calve normally (Dematawewa and Berger 1997). The incidence of postpartum disorders, postpartum production, reproductive performance, milk production, and future incidence of calving difficulty are all affected by dystocia. Dystocia is associated with a 2-fold increase in the incidence of milk fever, 3-fold increase in cystic ovaries, 2-fold increase in the risk for left-displaced abomasums, and a 2-to 3-fold increase in retained placenta and metritis. The increased incidence of postpartum disorders leads to increased culling rates (Laster et al., 1973; Rajala and Grohn, 1998) .

Dam parity also has a significant impact on economic and production losses associated with dystocia. The most recent published data (Dematawewa and Berger,

1997) regarding the effects of dystocia on the primiparous dam suggests these cows needing assistance during parturition experienced highly significant losses in 305-d adjusted milk, fat, and protein yields. The milk production of the primiparous dam steadily decreases as calving score increase to scores of 3, 4, and 5. At the score of 5, losses exceed 700 kg of milk per 305-day milk production, 24 kg milk fat loss, and protein losses exceed 20 kg. For second parity cows, adjusted milk, fat, and protein significantly decreased for calving scores of 4 and 5. For cows in the third lactation or more, losses were reported only in extreme difficulty or a score of 5 (Dematawewa and Berger, 1997). These losses reported by Dematawewa and Berger (1997) are greater than those previously reported in an earlier study by Djemali (1987).

Reproductive performance of the dam following a dystocic parturition is also adversely affected. The number of services per conception increases with increasing calving scores of 2, 3, 4, and 5. A dam that has extreme calving difficulty requires approximately 0.22 more services than those dams which have no difficulty (Dematawewa and Berger, 1997). According to Thompson et al. (1982) an increase in calving difficulty results in more days open as well as a longer interval to first breeding and more services per conception. Heifers that experience calving difficulty have calving intervals that are an average of 11 d longer than heifers needing no assistance (Pollak and Pelissier, 1980). According to Dobson et al. (2001) delayed uterine involution, delayed onset of luteal activity postpartum, and more abnormal progesterone profiles occur after a dystocic birth. The percentage of cows detected in estrus during the 45 day AI period is 14.4% lower in cows that required assistance at previous calving. The conception rate is also decreased for cows that experienced dystocia; 69.4% in dystocic cows compared to

85.3% for cows experiencing no dystocia (Laster et al., 1973). Cows that experience dystocia are more likely to experience the condition again in a subsequent calving (Mee et al., 2007).

Fetal

Calf losses during a stressful parturition are less well-documented than losses of the dam. It has been reported that 50% of stillbirths are a direct result of dystocia (Meyer et al., 2000; Lombard et al., 2007). In stillborn calves, approximately 90% of the losses were associated with a delay in receiving assistance or the amount of difficulty and time required to remove the calf (Laster and Gregory, 1973). This may suggest that applications of intervention strategies during calf deliveries are inadequate. In another study by Meyer et al. (2001) it was reported that a slight calving problem increased the odds of stillbirth by 2.91 in primiparous cows and 4.67 in multiparous cows. In more difficult calvings, primiparous cows were 6.67 times more likely to have a stillborn calf whereas multiparous cows were 11.36 times more likely to have a stillborn calf (Meyer et al., 2001). Calf mortality associated with a difficult birth or a calving ease score of 5 increases 2- to 3- fold over births with slight problems (Pollak and Pelissier, 1980). Wells et al. (1996) reported that as severity of dystocia increases there is a direct negative effect on dairy heifer survivability.

Fetal stress at birth

The majority of calf deaths are associated with fetal stress at birth. Prolonged hypoxia and significant acidosis are common problems in calves that experience dystocia, which can cause immediate death of the calf or reduce long-term survival (Breazile et al., 1988). Severe hypoxia increases the rate of respiratory movements due to the fetus

attempting to breathe in response to reduced oxygen (Kasari, 1994). Hypoxia also causes the discharge of meconium (Wensvoort, 1968). Expulsion of meconium from the intestinal lumen into the amniotic cavity is thought to be due to increased peristalsis and the relaxation of the anal sphincter resulting from vagal stimulation (Grignaffini et al., 2004). Due to the hypoxia-induced gasping, inspiration of meconium-contaminated aminotic fluid occurs resulting in meconium aspiration syndrome postnatally (Schoon and Kikovic, 1989).

Calves that experience hypoxic stress during birth are often weak and slow to stand and suckle (Odde, 1988; Dufty, 1977; Kasari, 1994). These impairments negatively affect ability to absorb colostrum leading to failure of passive transfer (Townsend, 1994). Consumption of colostrum in dystocic calves is reduced by 74% during the first 12 h after birth (Vermorel, 1989). However, it has been reported that calves that experienced dystocia that were tube-fed pooled colostrum have an equal amount of immunoglobulin absorptive capacity when compared to eutocic calves (Strawn et al., 1996; Donovan et al., 1986).

Dystocia and hypoxia depresses the calf's ability to utilize non-shivering thermogenesis. Therefore a difficult parturition can impair the cold tolerance of neonates during the early postnatal period (Carstens, 1994). Vermorel et al. (1989) reported a decrease in heat production and an increase in heat-loss in dystocic calves. This is in part due to the low concentrations of NEFA and delayed increase of limited lipid stores in the calf (Vermorel, 1989). Hypoxia also reduces muscular tonicity and inhibits shivering at birth which are important components of thermogenesis (Vermorel, 1989).

Economic

There are several economic costs associated with dystocia. Dematawewa and Berger (1997) estimated the total cost of dystocia in association parities to be $0.00, $50.45, $96.48, $159.82, and $397.61 for no assistance, slight assistance, needed assistance, considerable forced assistance, and extreme difficulty, respectively. Dematawewa and Berger (1997) also estimated total average cost of dystocia for primiparous cows was $28.01 compared to $11.10 for multiparous cows. In a previous study conducted by Djemali et al. (1987) economic loss in calves were valued at $70 for males and $150 for females.

Calving Management

Calving management practices can directly and indirectly affect calf survivability. These management factors include selection of a defined calving season, appropriate frequency of observation, appropriate timing of assistance, and use of appropriate calving facilities (Dargatz et al., 2004).

Dargatz et al. (2004) reported that only 39.6% of calvings occur in operations that use special calving facilities. The use of specialized calving facilities may provide for increased observation frequency and more timely assistance and provide protection that would decrease calf death losses. Due to the decreased ability for calves to thermoregulate the first few hours after birth, adequate shelter from the environment is also essential (Dargatz et al., 2004; Dennis, 1981). Cows should be moved to maternity unit within 1 to 2 days of calving depending upon available facilities (Mee, 2004). According to Mee (2004), some 10 to 20% of cows, particularly heifers, begin stage 2 of labor with no signs of stage 1. Monitoring cows near their calving date is very important.

Over 90% of United States operations observe heifers or cows on a regular basis during calving season. However, the frequency of observation was only every 6.7 hours for heifers and 9.6 hours for cows (Dargatz et al., 2004). The effects of infrequent observations and lack of allowed labor time prior to calving signifies that some heifers and cows are probably experiencing prolonged labor (Dargatz et al., 2004).

Obstetrical Assistance

Over 50% of all calf losses could be prevented by timely and correct obstetrical assistance (Bellows et al., 1987b). A large number of calf deaths are attributed to trauma, which suggests an inappropriate timing of assistance or excessive force during assistance (Dufty, 1973). Schuijt (1990) reported that 40% of stillborn calves born to heifers from veterinary-assisted deliveries had fractured ribs and 10% had vertebral fractures. An excessive amount (25%) of trauma has been reported in calves normally extracted. However, this can be explained by assuming that some powerful extractions are wrongly classified as normal. The moderate force of 2 men varies greatly; force of traction can be less than 160 kg, but may also range from 200-400 kg (Schuijt, 1990; Meijering and Postma, 1984). As little as 275 kg of force will fracture the long bones of neonatal calves (Ferguson, 1994).

When excessive force is applied during the delivery process, trauma inflicted can affect several body systems (Schuijt, 1990; Kelly and Rowan; Kasari, 1989; Straiton, 1994). Kasari (1989) reported that tracheal collapse can occur in calves older than one week as a consequence from an assisted delivery, but these collapses are often misdiagnosed. A higher incidence of meningeal hemorrhages within or around the cranial and spinal meninges has been reported in dystocic calves (Haughey, 1975; Dennis, 1981).

Rupture of the liver has also been reported as a direct result from the compression during delivery. Calf deaths in this case may not occur for 12 to 24 h (Johnson and Maclachlan, 1986; Dennis, 1981). Most traumatic injuries sustained during extraction are suspected to remain undiagnosed (Kelly and Rowan, 1993).

Another consequence of forced extraction of the fetus is the premature rupture of umbilical vessels. In cattle the umbilical cord is very short. During extraction when the hind legs are pulled from the vagina, the cord ruptures (Dufty, 1973). Kinmond et al. (1993) reported that immediate umbilical cord clamping deprives the human neonate of significant blood volume. There is a reduction in incidence of hemorrhages with a one minute delay in cord clamping (Kinmond et al., 1993). In foals, early rupture of umbilical vessels leads to a loss of approximately 1500 ml or 30% of potential blood volume (Mahaffey, 1961). Hammer (1998) reported that calves receiving premature assistance that ruptured the umbilical cord earlier during delivery had low P_{O2} and high P_{CO2} values postnatally. These effects were theorized to be due to poor pulmonary perfusion because of decreased neonatal blood volume. However, these effects were not seen by Riddle and Tyler (2003) who reported no effect of the timing of umbilical clamping on residual placental blood volume. Symptoms of early umbilical cord rupture include failure to thermoregulate, delayed time to stand, and inability to regulate respiration, all of which have been noted in calves experiencing dystocia (Mahaffey, 1961; Adams et al., 1990; Strawn et al., 1996).

Fetal monitoring at birth

In human medicine, extensive and continuous monitoring of the fetus is standard procedure whereas in cattle there is virtually no monitoring (Bleul and Kähn, 2008). Fetal

monitoring during calving is very limited to observations and subjective signs of stress (Mee, 2008b). According to Mee (2004) when the calf begins to show signs of reduced vigor such as lingual edema, buccal or lingual cyanosis, scleral hemorrhages or reduced responsiveness to stimulation, intervention should occur. There is currently a lack of any published data supporting these observations.

In humans, several monitoring protocols and technologies have been developed. Pulse oximetries as well as fetal electrocardiogram (ECG) are tools that can be used during labor after membrane rupture with scalp electrodes (Nielson, 2006; Grignaffini et al., 2004). High false positives often occur using ECG which has led to the widespread use of fetal pulse oximetry in humans. Fetal pulse oximetry safely and accurately indicates fetal oxygenation by measuring the percentage oxygen saturation of arterial hemoglobin (Grignaffini et al., 2004; Uystepruyst et al., 2000). This tool can aid in the early diagnosis of fetal hypoxia (Grignaffini et al., 2004). The use of scalp electrodes, however, is not a practical approach for monitoring calves during delivery. Tongue pulse oximetry has also been utilized in young children during surgery, and clinical trials have demonstrated that tongue oximetry is a reasonable alternative location for the probe site compared to more conventional peripheral sites (Coté et al., 1992).

In human infants, assessment protocols regarding the color of lips, mucous membranes, and nail beds have also been established. Cyanosis is a blue coloration of the skin and mucous membranes associated with a decreased amount of oxygen in the blood (Roberts, 1975; Dain, 2007; O'Donnell et al., 2007). When skin changes color rapidly, it is a result of vasodilatation, vasoconstriction, or the amount of hemoglobin present in the

blood (Roberts, 1975). There is however, substantial variation in the perception of newborn infant color among observers (O'Donnell et al., 2007).

Currently, the dairy industry's goal of maintaining healthy cattle is partially limited by a lack of useful methods for determining the vitality of the calf during parturition (Bluel and Kähn, 2008). Blood gas analysis in bovine fetuses can assess vitality, but only provide point-in-time information and techniques are not suitable for continuous fetal monitoring (Bleul and Kähn, 2008). Subjective, visual methods are used, but have not been tested in a research setting to determine their usefulness as accurate and repeatable predictors of fetal stress. In addition, pulse oximetry could provide a non-invasive, immediate, and portable technique to assess oxygenation and diagnose hypoxic calves (Uystepruyst et al., 2000).

Literature Cited

Adams, F.H., T. Fujiwara and G. Rowshan.1963. The nature and origin of the fluid in the fetal lamb lung. J. Pediatr. 63:159.

Adams, R. F.B. Garry, B. Aldrige, M.D. Holland, and K.G. Odde. 1990. Arterial blood gas values in newborn beef calves. Proc. Internat. Conf. Vet. Perinatol. P. 33.

Alexander D.P., H.G. Britton, and D.A. Nixon. 1970. The metabolism of fructose and glucose by the sheep foetus: Studies on the isolated perfused preparation with radioactively labeled sugars. Q. J. Exp. Physiol. 55:346-362.

Amoroso, E.C., G.S. Dawes, and J.C. Mott. 1957. Patency of the ductus arteriosus in the newborn calf and foal. Brit. Heart. J. 20:92-96.

Asakura, Hirobumi. 2004. Fetal and neonatal thermoregulation. J. Nippon Med. Sch. 71(6):360-370.

Assali, N.S., and J.A. Morris. 1964. Circulatory and metabolic adjustments of the fetus at birth. Mol. Neonat. 7:141-159.

Assali, N.S., N. Sehgal, and S. Marable. 1962. Pulmonary and ductus arteriosus circulation in the fetal lamb before and after birth. Am. J. Physiol. 202:536-540.

Bell, A.W., R.B. Wilkening, and G. Meschia. 1987. Some aspects of placental function in chronically heat-stressed ewes. J. Dev. Physiol. 9:17-26.

Bellows, R.A., R.B. Staigmiller, L.E. Orme, R.E. Short, and B.W. Knapp. 1993. Effects of sire and dam on late-pregnancy conceptus and hormone traits in beef cattle. J. Anim. Sci. 71:714-723.

Bellows, R.A., R.E. Short, R.B. Staigmiller, and W.L. Milmine. 1987a. Effects of induced parturition and early obstetrical assistance in beef cattle. J. Anim. Sci. 66:1073-1080.

Bellows, R.A., D.J. Patterson, P.J Burfening, and D.A. Phelps. 1987b. Occurrence of neonatal and postnatal mortality in range beef cattle. II. Factors influencing calf death. Theriol. 28:573-586.

Berger, P.J. 1995. Early calf livability in U.S. Holsteins. Mimeo. Iowa State University, Ames.

Berger, P.J. 1996. Summary of calving ease score data. Mimeo. Iowa State University, Ames.

Breazile, J.E., L.A. Vollmer, and L. E. Rice. 1988. Neonatal adaptation to stress of parturition and dystocia. Vet. Clin. North Am. Food Anim. Pract. 4:481-499.

Brown, M.J., R.E. Oliver, C.A. Ramsden, L.B. Strang, and D. V. Walters. 1983. Effects of adrenaline and of spontaneous labour on the secretion and absorption of lung liquid in the fetal lamb. J. Physiol. 344:137-152.

Bland, R D., and D.W. Nielson. 1992. Developmental changes in lung epithelial ion transport and liquid movement. Ann. Rev. Physiol. 54:373-394.

Bleul, U. and W. Kähn. 2008. Monitoring the bovine fetus during stage II of parturition using pulse oximetry. Theriol. 69:302-311.

Carstens, G.E. 1994. Cold thermoregulation in the newborn calf. Vet. Clin. N. Am.: Food Anim. Pract. 10:69-107.

Collier, R.J., S.G. Doelger, H.H. Head, W.W. Thatcher, and C.J. Wilcox. 1982. Effects of heat stress during pregnancy on maternal hormone concentrations, calf birth weight and postpartum milk yield of Holstein cows. J. Anim. Sci. 54:309-319.

Comline, R.S., L.W. Hall, R.B. Lavelle, P.W. Nathanielsz, and M. Silver. 1974. Parturition in the cow: Endocrine changes in animals with chronically implanted catheters in the foetal and maternal circulations. J. Endocr. 63:451-472.

Coté, C.J., A.L. Daniels, M. Connolly, S.K. Szyfelbein, and C.D. Wickens. 1992. Tongue oximetry in children with extensive thermal injury: comparison with peripheral oximetry. Can. J. Anaesth. 39(5):454-457.

Dain, S. 2007. Color changes in cyanosis and the significance of congenital dichromasy and lighting. Color Res. Appl. 32(6):428-432.

Dalton, R.G. 1968a. Renal function in neonatal calves: Diuresis. Br. Vet. J. 124:370-381.

Dalton, R.G. 1968b. Renal function in neonatal calves: Urea clearance. Br. Vet. J. 124:450-490.

Dalton, R. G. 1968c. Renal function in neonatal calves: Inulin, thiosulphate and para-aminohippuric acid clearance. Br. Vet. J. 124:498.

Dargatz, D.A., G.A. Dewell, and R.G. Mortimer. 2004 Calving and calving management of beef cows and heifers on cow-calf operations in the United States. Theriol. 61:997-1007.

Dawes, G.S., J.C. Mott, J.G. Widdicombe, and D.G. Wyatt. 1953. Changes in the lungs of the new-born lamb. J. Physiol. 121:141-162.

Dematawewa, C.M.B., and P.J. Berger. 1997. Effects of dystocia on yield, fertility, and cow losses and an economic evaluation of dystocia scores for Holsteins. J. Dairy Sci. 80:754-761.

Dennis, S.M. 1981. Low viability of calves at birth. *in:* The Veterinary Annual no. 21. G.S.G. Grunsell and F.W.G. Hill, eds. Scientechnica, Bristol. Pp. 63-73.

Djemali, M. A.E. Freeman, and J. Berger. 1987. Reporting of dystocia scores and effects of dystocia on production, days open, and days dry from dairy herd improvement data. J. Dairy Sci. 70:2127-2131.

Dobson, H, J. E. Tebble, R.F. Smith, and W.R. Ward. 2001. Is stess really all that important? Theriogenology. 55:65-73.

Donovan, G.A., L. Badinga, R.J. Collier, C.J. Wilcox, and R.K. Braun. 1986. Factors influencing passive transfer in dairy calves. J. Dairy Sci. 69:754-759.

Dufty, J.H. 1973. Clinical studies on bovine parturition - foetal aspects. Aust. Vet. J. 49:177-181.

Dufty, J.H., and V. Sloss. 1977. Anoxia in the bovine fetus. Aust. Vet. J. 53:262-267.

Edquivist L.E., H. Kindahl, and G. Stabenfeldt. 1978. Release of prostaglandin F2α during bovine peripartal period. Prostaglandins. 16:111-119.

Fairclough, R.J., J.T. Hunter, and R.A.S Welch. 1975. Peripheral plasma progesterone and utero-ovarian prostaglandin F concentrations in the cow around parturition. Prostaglandins. 9:901-914.

Ferguson, J.G. 1994. Femoral fractures in the newborn calf: Biomechanics and etiological considerations for practitioners. Can. Vet. J. 35:628-630.

Fuchs A.R., H. Helmer, O. Bwhrens, H-C. Liu, L. Antonian, S.M. Chang, and M.J. Fields. 1992. Oxytocin and bovine parturition: A steep rise in endometrial oxytocin receptors precedes onset of labor. Biol. Repro. 47:937-944.

Gaines, J.D., D. Peshel, R.G. Kaufman, D.M. Schaefer, G. Badtram, J. Kumi-Diaka, M.K. Clayton, and G. Milliken. 1993. Pelvic growth, calf birth weight and dystocia in Holstein x Hereford heifers. Theriogen. 40:33-41.

Grignaffini, A., E. Soncini, E. Ronzoni, E. Piazza, S. Anfuso, and E. Vadora. 2004. Meconium-stained aminotic fluid and fetal oxygen saturation measured by pulse oximetry during labour. Acta. Bio. Medica. Ateneo. Parmense. 1:45-52.

Hammer, C.J. 1998. Effect of obstetrical assistance on Jersey calves. Masters thesis. Ames.

Haughey, K.G. 1975. Meningeal haemorrhage and congestion associated with the perinatal mortality of beef calves. Aust. Vet. J. 51:22-27.

Hoedemaker, M., P.G. Weston, W.C. Wagner. 1990. Influence of cortisol and different steroidogenic pathways on estrogen synthesis by the bovine placenta. Am. J. Vet. Res. 51:1012-1015.

Holland, M.D., and K.G. Odde. 1992. Factors affecting calf birth weight: a review. Theriogenol. 38:769-798.

Hunter, J.T., R.J. Fairclaugh, A.J. Peterson, S.R. Welch. 1977. Foetal and maternal changes preceding normal bovine parturition. Acta Endocr. 84:653-662.

Johansen, J.M and P.J. Berger. 2003. Birthweight as a predictor of calving ease and perinatal mortality in Holstein cattle. J. Dairy Sci. 86:3745-3755.

Johnston, W.S., and G.K.Machlachlan. 1986. Incidence of rupture of the liver in neonatal lambs in the north of Scotland. Vet Rec. 118:610-611.

Kasari, T.R. 1989. Weakness in neonatal calves associated with dystocia. Agri-Practice 10:19-23.

Kelly, D.F., and T.G. Rowan. 1993. Neonatal mortality in a pair of identical twin calves: clinical and post mortem findings. Vet. Rec. 133:475-476.

Kinmond, S., T.C. Aitchison, B.M. Holland, J.G. Jones, T.L. Turner, and C.A.J. Wardrop. 1993. Umbilical cord clamping and preterm infants: a randomised trial. Brit. Med. J. 306:172-175.

Kruz M.M and L.B. Willett. 1991. Carbohydrate, enzyme, and hematology dynamics in neborn calves. J. Dairy Sci. 74:2109-2118.

Ktorza, A. and P. Ferre. 1992. Fuel metabolism in the fetus. In: Perinatal biochemistery. Pp. 149-177.

Laster D.B., H.A. Glimp, L.V. Cundiff, and K.E. Gregory. 1973. Factors affecting dystocia and the effects of dystocia on subsequent reproduction in beef cattle. J. Anim. Sci. 36:695-705.

Liggins, G.C., R.J. Fairclough, S.A. Grieves, J.Z. Kendall, and B.S. Knox. 1973. The mechanism of initiation of parturition in the ewe. Res. Prog. Horm. Res. 29:111-159.

Lindhe, B. 1966. Dead and difficult births in cattle and measures for their prevention. World Rev. Anim. Prod. 2:53-57.

Lombard, J.E., F.B. Garry, S.M. Tomlinson, and L.P Garber. 2007. Impacts of dystocia on health and survival of dairy calves. J.Dairy Sci. 90:1751-1760.

Mahaffey, L.W. 1961. Pulmonary syndrome in newborn foals. *in:* Somatic Stability in the Newly Born. G.E.W. Wolstenholme and M. O'Connor, *eds.* Little, Brown and Company, Boston.

Medina, J.M., C. Vicario, M.C. Juanes, and E. Fernandez. 1992. Biochemical adaptations to early extrauterine life. In: Perinatal Biochemistry. 233-242.

Mee, J.F. 2004. Managing the dairy cow at calving time. Vet. Clin. Food Anim. 20:521-546.

Mee, J.F. 2008a. Prevalence and risk factors for dystocia in dairy cattle: A review. The Veterinary Journal. 172:93-101.

Mee, J.F. 2008b. Newborn dairy calf management. Vet. Clin. Food Anim. 24:1-17.

Meijering, A. 1984. Dystocia and stillbirth in cattle - a review of causes, relations and implications. Livestock Prod. Sci. 11:143-177.

Meijering, A., and A. Postma. 1984. Morphologic aspects of dystocia in dairy and dual purpose heifers. Can. J. Anim. Sci. 64:551-562.

Menissier, F. and J.L. Foulley. 1979. Present situation of calving problems in the EEC. Incidence of calving difficulties and early calf mortality in beef breeds. In: Calving Problems and Early Viability of the Calf. EEC Seminar, May 1977, Frising, FRG. Cur. Top.Vet. Med. Ans. 4:30-85.

Meyer, C.L., J. Berger, and K.J. Koehlert. 2000. Interactions among factors affecting stillbirths in Holstein cattle in the United States. J. Dairy Sci. 83:2657-2663.

Meyer, C.L., J. Berger, K.J. Koehler, J.R. Thompson, and C.G. Sattler. 2001.Phenotypic trends in incidence of stillbirth for Holsteins in the United States. J. Dairy Sci. 84:515-523.

Neilson, J.P. 2006. Fetal electrocardiogram (ECG) for fetal monitoring during labour. Cochrane Database of Systematic Reviews. Issue 3. Art. No.: CD000116. DOI: 10.1002/14651858.CD000116.pub2.

National Animal Heath Monitoring System (NAHMS). 1996. Part I: Reference of 1996 dairy management practices. USDA:APHIS:VS. Fort Collins

O'Brien, T., and G.H. Stott. 1977. Prepartum serum hormone concentrations related to dystocia in Holstein heifers. J. Dairy Sci. 60:249-253.

Odde, K.G. 1988. Survival of the neonatal calf. Vet. Clin. North Am. Food Anim. Pract. 4:501-508.

O'Donnell, C.P.F., C.O.F. Kamlin, P.G. Davis, J.B. Carlin, and C.J. Morley. 2007. Clinical assessment of infant colour at delivery. Arch. Dis. Child Fetal Neonatal Ed. 92:F465-467.

Peters, A.R., and P.J.H. Ball. 1995. Parturition and lactation. In: Reproduction in Cattle. Pp. 127-144. Blackwell Science Ltd. Cambridge.

Philipson, J. 1976. Studies on calving difficulty, stillbirth and associations with Swedish cattle breeds. IV. Relationship between calving performance, precalving body measurements and size of pelvic opening in Friesian heifers. Acta. Agri. Scand. 26:221.

Pollack. E.J., and A.E. Freeman. 1976. Parameter estimation and sire evaluation for dystocia and calf size in Holsteins. J. Dairy Sci. 59:1817-1824.

Pollak, E.J., and Pellisier, C.L. 1980. Relationships among problems associated with calving. Nineteenth Annual California Dairy Cattle Day. March 26, 1980. Department of Animal Science and Cooperative Extension. University of California, Davis.

Rice, L.E., and J.N. Wiltbank. 1972. Factors affecting dystocia in beef cattle. J. Am. Vet. Med. Assoc. 161:1348-1358.

Rajala, P.J. and Y.T. Grohn. 1998. Effects of dystocia, retained placenta and metritis on milk yield in dairy cows. J. Dairy Sci. 81:3172-3181.

Riddle, A.L., and H.D. Tyler. 2003. Factors affecting residual placental blood volume following parturition in dairy cattle. Masters thesis. Ames.

Roberts, S.L. 1975. Skin assessment for color and temperature. Am. J. Nursing. 75(4):610-613.

Schoon, H.A., and D. Kikovic. 1987. Morphological demonstration and pathogenetical evaluation of amniotic fluid aspiration supporting the diagnosis of pulmonary asphyxia in newborn calfs and foals. Dtsch. Tierarztl. Wschr. 94:73-76.

Schrag, L., and H. Singer. 1992. The birth of the calf. In: The Calf Book. Pp. 20-28. Schober Verlags-Gmbh. Hengersberg.

Schuijt, G. 1990. Iatrogenic fractures of ribs and vertebrae during delivery in perinatally dying calves: 235 cases (1978-1988). JAVMA 197:1196-1202.

Senger, 1999. Placentation, the endocrinology of gestation and parturition. Pathways to pregnancy and parturition. Pp. 232-247.

Shelton, M. 1964. Relation of environmental temperature during gestation to birth weight and mortality of lambs. J. Anim. Sci. 23:360-364.

Sieber, M., A.E. Freeman, and D.H. Kelly. 1989. Effects of body measurements and weight on calf size and calving difficulty of Holsteins. J. Dairy Sci. 72:2402-2410.

Sloss, V., and D.E. Johnson. 1967. The causes and treatment of dystocia in beef cattle in western Victoria. 2. Causes, methods of correction and maternal death rates. Aust. Vet. J. 43:13-21.

Stabenfeldt G.H., B.I. Osburn, and L.L. Ewing. 1970. Peripheral plasma progersterone levels in the cow during pregnancy and parturition. Am. J. Phys. 218:571-575.

Straiton, E. 1994. Calving the Cow and Care of the Calf. Fourth Edition. Farming Press, Ipswich.

Strawn, K.K., H.D. Tyler, M.A. Faust, and B.J. Nonnecke. 1996. Effects of birth stress in calves. J. Dairy Sci. 79 (Suppl 1):130.

Thompson, J.R., E.J. Pollack, and C.L. Pelissier. 1982. Interrelationships of parturition problems, production of subsequent lactation, reproduction, and age at first calving. J. Dairy Sci. 66:1119-1127.

Townsend, H.G.G. 1994. Environmental factors and calving management practices that affect neonatal mortality in the beef calf. Vet. Clin. N. Am. 10:119-127.

Uystepruyst, CH., J. Coghe, F. Bureau, and P. Lekeux. 2000. Evaluation of accuracy of pulse oximetry in newborn calves. Vet. J. 159:71-76.

Vermorel, M., C. Dardillat, J. Vernet, Saido, and C. Demigne. 1983. Energy metabolism and thermoregulation in the newborn calf. Ann. Rech. Vet. 14:382-390.

Vermorel, M., J. Vernet, C. Dardillat, Saiso, C. Demigne, and M. Davicco. 1989. Energy metabolism and thermoregulation in the newborn calf; Effect of calving conditions. Can. J. Anim. Sci. 69:113-122.

Walsh, S.Z., and Lind. 1978. The fetal circulation and its alteration at birth. In: Perinatal Physiology. U. Stave (ed.) Plenum Medical Book Company. New York.

Wensvoort, P. 1968. Post mortal findings in lambs which died of asphyxia during parturition. Tijdschr. Diergeneesk. 93:1270-1278.

CHAPTER TWO

PREDICTIVE MEASURES OF FETAL DISTRESS IN CALVES DURING DELIVERY

K.E. Hard, H.D. Tyler, and J.D. Quigley III

INTRODUCTION

The transition from fetal to neonatal life is the most hazardous period in the life of dairy calves. The risk of disease and death peaks during the first two weeks of life. The greatest risk factor for both early mortality and morbidity is stress during the delivery process, most commonly a result of dystocia. Dystocia is defined as a prolonged or difficult birth. Lombard et al. (2007) reported that dystocia and subsequent health effects account for nearly 50% of all calf mortality. The greatest death losses occur as stillbirths. Stillbirths are defined as calves that die just prior to, during, or within 24 to 48 h of parturition (Meyer et al., 2001). The cost of stillbirths to the U.S. dairy industry has been estimated at $132 million per year (Thompson et al., 1981). Over 50% of all calf losses could be prevented by timely and correct obstetrical assistance (Bellows et al., 1987).

In stillborn calves, approximately 90% of the losses have been associated with a delay in receiving assistance or the amount of difficulty and time required to remove the calf (Laster and Gregory, 1973). Wells et al. (1996) reported that a dystocia requiring forced extraction, compared with an unassisted calving, was 4.22 times more likely to result in heifer calf death within the first 21 days of life.

In human medicine, extensive and continuous monitoring of the fetus is standard procedure whereas in cattle there is virtually no monitoring (Bleul and Kähn, 2008). Fetal monitoring during calving is very limited to observations and subjective signs of stress

(Mee, 2008b). According to Mee (2004) when the calf begins to show signs of reduced vigor such as lingual edema, buccal or lingual cyanosis, scleral hemorrhages or reduced responsiveness to stimulation, intervention should occur. There is currently a lack of any published data supporting these observations.

In humans, several monitoring protocols and technologies have been developed. Pulse oximetries as well as fetal electrocardiogram (ECG) are tools that can be used during labor after membrane rupture with scalp electrodes (Nielson, 2006; Grignaffini et al., 2004). High false positives often occur when using ECG which has led to the widespread use of fetal pulse oximetry in humans. Fetal pulse oximetry safely and accurately indicates fetal oxygenation by measuring the percentage oxygen saturation of arterial hemoglobin (Grignaffini et al., 2004; Uystepruyst et al., 2000). This tool can aid in the early diagnosis of fetal hypoxia (Grignaffini et al., 2004). The use of scalp electrodes, however, is not a practical approach for monitoring calves during delivery. Tongue pulse oximetry has also been utilized in young children during surgery, and clinical trials have demonstrated that tongue oximetry is a reasonable alternative location for the probe site compared to more conventional peripheral sites (Coté et al., 1992).

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Currently, the dairy industry's goal of maintaining healthy cattle is partially limited by a lack of useful methods for determining the vitality of the calf during parturition (Bluel and Kähn, 2008). Blood gas analysis in bovine fetuses can assess vitality, but only provide point-in-time information and techniques are not suitable for continuous fetal monitoring (Bleul and Kähn, 2008). Subjective, visual methods are used, but have not been tested in a research setting to determine their usefulness as accurate and repeatable predictors of fetal stress. In addition, pulse oximetry could provide a non-invasive, immediate, and portable technique to assess oxygenation and diagnose hypoxic calves (Uystepruyst et al., 2000).

Therefore the objective of this study was to assess the accuracy of pulse oximetry and tongue parameters when evaluating fetal stress during parturition.

MATERIALS AND METHODS

Fifty eight calves from three breeds of dairy cattle (Holsteins, Jerseys, and Jersey X Holsteins) were monitored beginning at the onset of parturition. Duration of calving was defined as the period from the first appearance of hooves to umbilical rupture. From the time the tongue was first visible, measurements for tongue color, length and reflex were taken approximately every two minutes until the time of umbilical cord rupture. Tongue color was determined using a color chart that included 17 distinct colors (Appendix A). Tongue length (mm) was measured from the middle point of the nose to the tip of the tongue. Tongue reflex was assessed with a sharp pinch on the tongue on a scale from 0 to 3 based on the following criteria:

0: No reflex: calf does not respond to hard pinch of the tongue

1: Poor reflex: calf responds poorly only to very hard pinch of the tongue

2: Fair reflex: calf responds to a moderate pinch of the tongue

3: Good reflex: calf immediately responds to a light pinch of the tongue

Measurements of fetal oxygen saturation and fetal heart rate were attempted intrapartum using pulse oximetry (Nellcor N-595, Boulder, CO). A modified lingual sensor was clamped to the tongue as soon as possible during the delivery process. The sensor consisted of light-emitting diodes and a photodiode as the receiver. Measurements were collected using a software program that collated data every second (Score Software 1.1 a).

Calving ease score was based on the following criteria:

1. cow had no problem during parturition
2. cow had slight problem during parturition requiring slight assistance
3. cow required assistance during parturition to extract the calf
4. considerable force required to extract calf
5. extremely difficult extraction requiring mechanical assistance

All abnormal occurrences were recorded, including abnormal presentation, meconium staining, or other problems. All decisions regarding assistance were made by the maternity barn personnel.

Arterial blood samples were taken to establish stress parameters (P_{O2}, P_{CO2}, pH) defined previously by Strawn et al., 1996. Samples were drawn anaerobically within 20 minutes of birth into sterile 3 ml syringes containing 50 units lyophilized lithium heparin (safePICO; Radiometer, Copenhagen). Samples were analyzed immediately by using a

blood gas/electrolyte analyzer (ABL77; Radiometer, Copenhagen) to determine P_{O2}, P_{CO2}, pH, $[Na^+]$, $[K^+]$, $[Cl^-]$, and $[Ca^{2+}]$.

Statistical analysis

Data were analyzed by analysis of variance using the Proc MIXED procedure of SAS (SAS Institute, 2003) with tongue color, length and responsiveness included as the dependent variables. Orthagonal contrasts were used to test differences between normal tongue color vs. color extreme, between normal vs. dark tongue colors, and between light vs. dark tongue colors. Pearson correlation coefficients were also calculated for all measured parameters using the Proc CORR procedure of SAS. Significance was declared at $P \leq 0.05$ unless otherwise noted and probability values between 0.05 and 0.15 were defined as tendency towards significance. Standard errors presented were for the differences among least squares means.

RESULTS

Three color categories were devised from the original individual 17 colors (Table 1). The categories were devised by using PROC SORT in SAS using tongue color and defined stress parameters of pH, P_{O2}, and P_{CO2}. Eleven of the observed calves were classified in color category 1, 44 calves were classified in the color category 2, and 3 calves were classified in color category 3 (Table 1). The calves were also classified by calving ease (Table 2). Twenty-nine of the calves were classified as calving ease of 1, 15 of which were Holstein, 6 were Jersey and 8 were Holstein x Jersey. Seventeen calves were classified as having a calving ease of 2, 10 of which were Holstein, 3 were Jersey, and 3 were Holstein x Jersey. Six calves were classified as a calving ease of 3, 5 of which were Holstein and 1 was Holstein x Jersey. Six calves were classified as a calving

ease of 4, 4 of which were Holstein, 1 was Jersey, and 1 was Holstein x Jersey. No calves were classified in calving ease score 5.

Color Categories

Both initial and final tongue colors were significantly different (P<.0001) across color categories (Figure 1 & 2). Surprisingly parameters reflecting oxygen delivery (P_{O2}, hematocrit, total hemoglobin, oxygen saturation, and total oxygen content) were not correlated with color categories (Table 3 and Figures 5, 6, and 7). Also, color categories were not significantly correlated with pH or P_{CO2} (Table 3). This suggests that dark tongue color is a poor predictor of hypoxia and acidosis.

Tongue length

Mean tongue length tended to be positively correlated with P_{CO2} and tended to be negatively correlated with pH (Table 3), indicating that calves with longer tongues tended to have a lower pH and a higher P_{CO2}. Lower pH and a higher P_{CO2} are the best parameters reflecting birth stress (Strawn et al., 1996). However tongue length was not correlated with oxygen delivery parameters (Table 4). This suggests that length is a good predictor of acidosis but not hypoxia. Final tongue length was significantly positively correlated with calving ease score, suggesting that calves with longer tongues were more likely to have a higher calving ease score. Final tongue length was also significantly negatively correlated to final tongue color (Table 3 & Figure 4). This suggests that as the tongue lengthened the tongue color was darker. Tongue length was the parameter that was most predictive for calving difficulty. This suggests that acidotic calves were more likely to receive assistance than calves experiencing hypoxia.

Tongue reflex

Final tongue reflex was significantly negatively correlated to tongue length (Table 4), suggesting that calves with longer tongues were less responsive to tactile stimulation. However, tongue reflex was not correlated to parameters associated with hypercapnia, acidosis, or hypoxia. This suggests that tongue reflex alone is a poor predictor of hypoxia or acidosis. Least squares means for tongue reflex are shown in Figure 3.

Calving ease

Factors most related to calving ease were pH, P_{CO2}, P_{O2}, and tongue length (Figures 8-12). This shows that calving assistance decisions provided by personnel were appropriate during the period that the study was conducted.

Pulse Oximetry

Several attempts were made to successfully monitor the fetus using transmittance pulse oximetry. . However, due to the ineffective lingual clamp and cow movement, data was obtained for one delivery only. In future attempts, a new lingual clamp must be developed to keep the diodes parallel and stable.

Table 1: Distribution of final tongue color categories

Color Categories	Holstein	Jersey	Holstein x Jersey	Total
1	4	5	2	11
2	29	5	10	44
3	2	0	1	3

Color Category 1: Final tongue color 1-3
Color Category 2: Final tongue color 4-9
Color Category 3: Final tongue color 10-17

Table 2: Calving ease distribution

Calving Ease Scores	Holstein	Jersey	Holstein x Jersey	Total
1	15	6	8	29
2	10	3	3	17
3	5	0	1	6
4	4	1	1	6
5	0	0	0	0

Table 3: Probability values for Pearson Correlation Coefficients for tongue color

	Color Category	Initial Tongue Color	Final Tongue color	Color Change
Initial Tongue Color	<.0001*		<.0001*	.5344
Final Tongue Color	<.0001*	<.0001*		.0239*
Color Change	.0922	.5344	.0239*	
Final Tongue Reflex	.5283	.4275	.2604	.7089
Initial Tongue Length	.0451*	.0145*	.0179*	.6273
Final Tongue Length	.0596	.0660	.0051*	.7575
Change in Length	.4272	.2979	.1600	.4202
Mean Tongue Length	.0291*	.0152*	.0025*	.8524
pH	.8818	.9461	.1527	.6399
PCO2	.3927	.2944	.2597	.3393
PO2	.9719	.0843	.3336	.9107
Calving Ease Score	.9295	.8491	.6720	.6747
[Hct]	.1078	.1505	.0610	.3933
$[K^+]$	.8346	.2326	.8782	.8546
$[Ca^{2+}]$	.4705	.0142*	.4743	.5206
$[Cl^-]$	.4601	.4527	.5760	.1956
ctHb	.1077	.1490	.0624	.3994
cHCO3	.2886	.1531	.9188	.1904
Base excess	.3769	.1287	.5166	.2175
ctCO2	.3890	.0669	.7587	.1174
Anion Gap	.5477	.8414	.5625	.9471
sO2	.5271	.2565	.6928	.5520
ct O2 Vol%	.8526	.1376	.1997	.8715

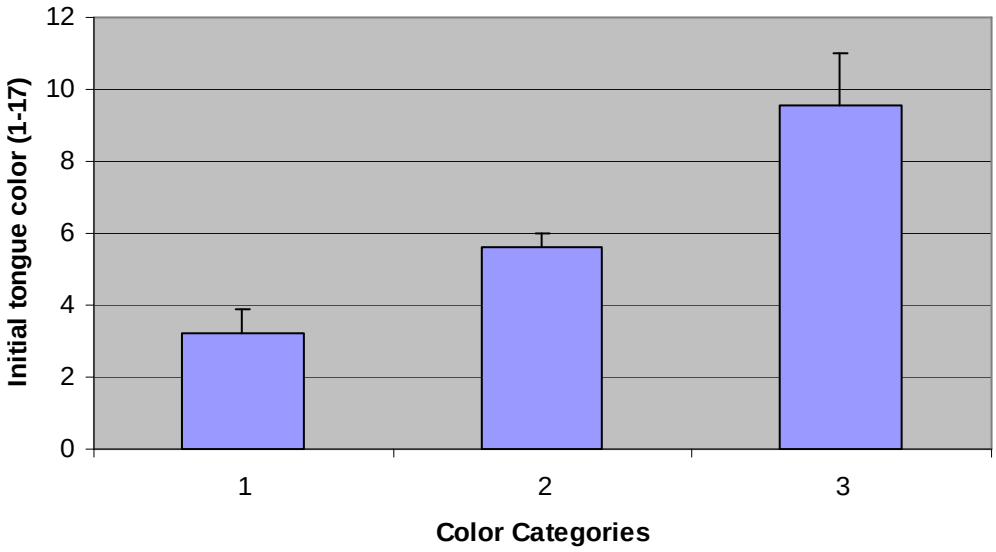
***** P< .05

Table 4: Probability values for Pearson Correlation Coefficients for tongue length

	Initial Tongue Length	Final Tongue Length	Mean Tongue Length
Final Tongue Reflex	.6500	.0256*	.0237*
pH	.2890	.2193	.1369
PCO2	.1300	.1164	.0534
PO2	.6230	.2180	.6583
Calving Ease Score	.2497	.0014*	.0139*
Hct	.8333	.7469	.7627
$[K^+]$	.9977	.7059	.5797
$[Ca^{2+}]$	.1615	.8804	.2670
$[Cl^-]$	.8381	.5520	.4455
ctHb	.8438	.7450	.7630
$[HCO3^-]$	.5506	.6355	.5914
Base excess	.6518	.8389	.7970
ctCO2	.3358	.4561	.3609
Anion Gap	.1285	.8615	.5262
sO2	.5604	.3640	.6075
ct O2 Vol%	.3550	.5117	.5274

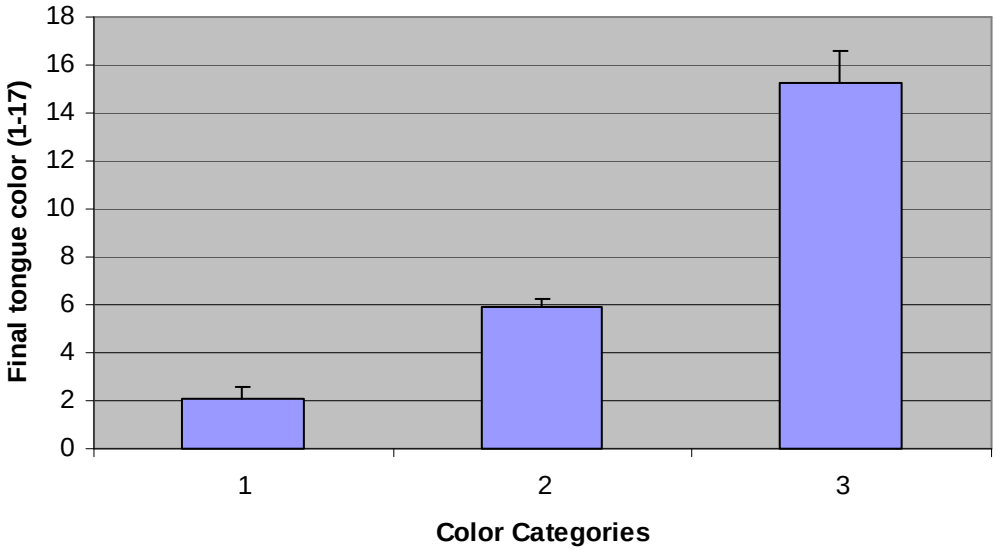
*P<.05

Figure 1: Mean initial tongue color of calves across color categories (least squares means ± standard error of the mean).



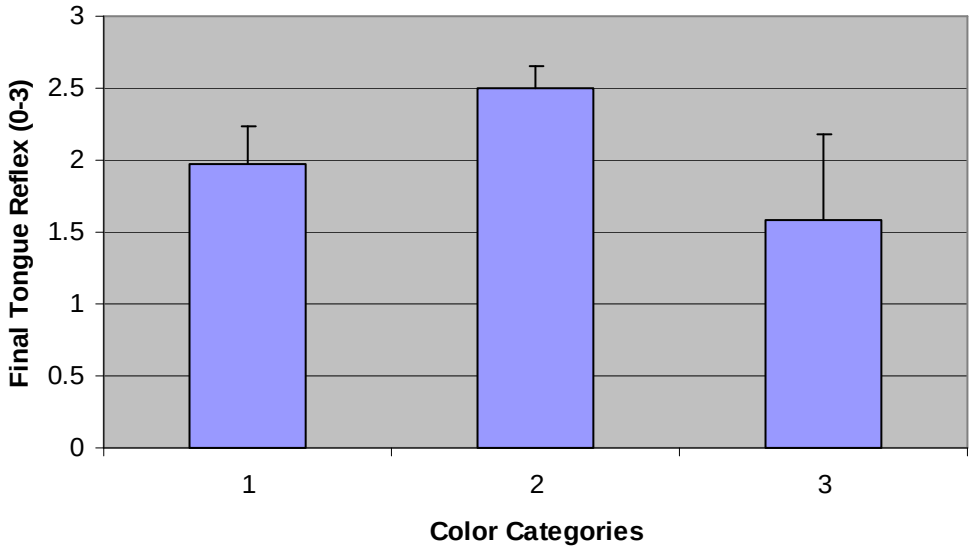
Color Category 1: Final tongue color 1-3: n=11
Color Category 2: Final tongue color 4-9: n=44
Color Category 3: Final tongue color 10-17: n=3

Figure 2: Mean final tongue color of calves across color categories (least squares means ± standard error of the mean).



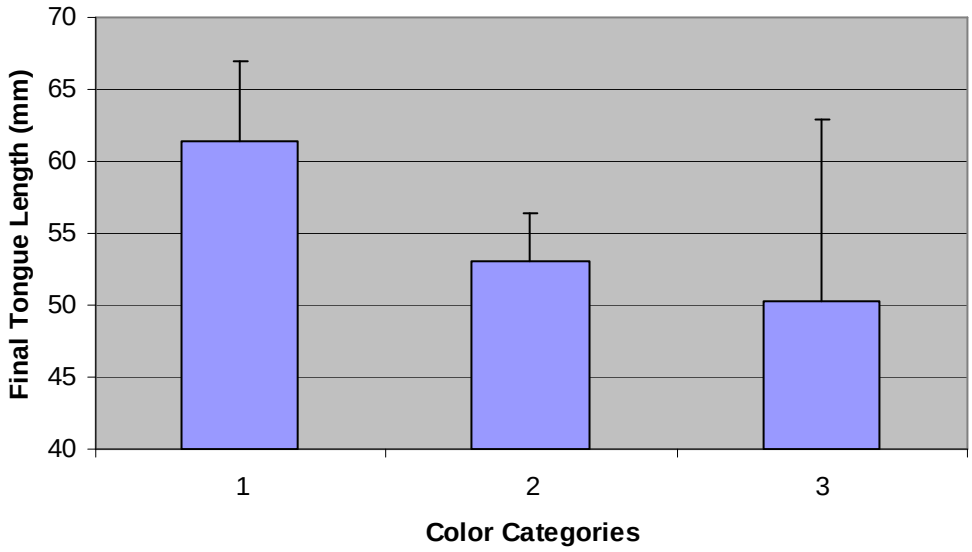
Color Category 1: Final tongue color 1-3: n=11
Color Category 2: Final tongue color 4-9: n=44
Color Category 3: Final tongue color 10-17: n=3

Figure 3: Mean final tongue reflex of calves across color categories (least squares means ± standard error of the mean). The higher the number, the greater the tongue reflex to tactile stimulation.



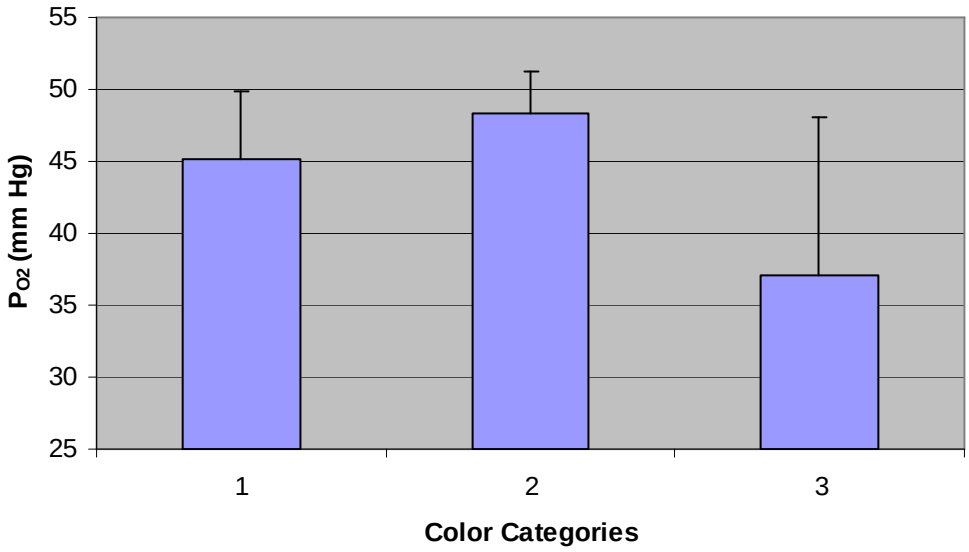
Color Category 1: Final tongue color 1-3: n=11
Color Category 2: Final tongue color 4-9: n=44
Color Category 3: Final tongue color 10-17: n=3

Figure 4: Mean final tongue length of calves across color categories (least squares means ± standard error of the mean).



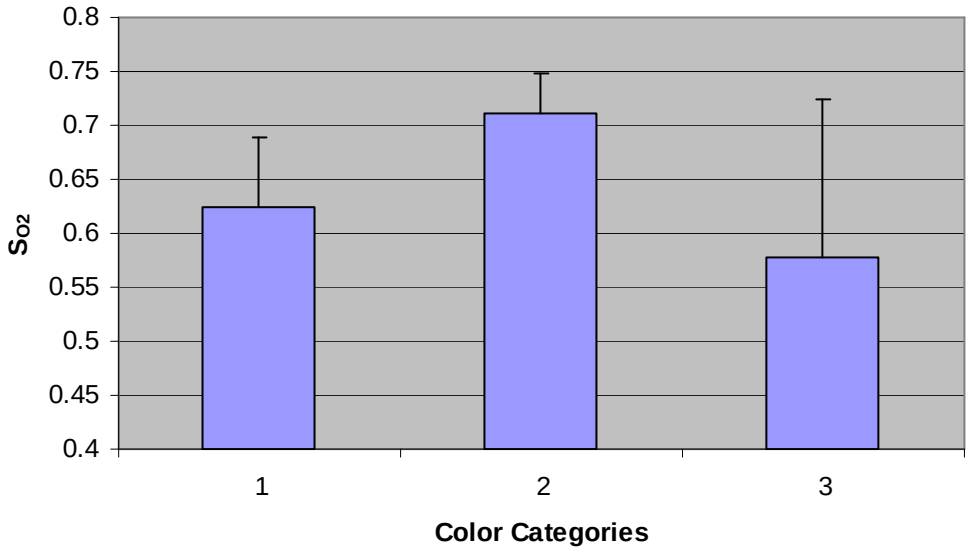
Color Category 1: Final tongue color 1-3: n=11
Color Category 2: Final tongue color 4-9: n=44
Color Category 3: Final tongue color 10-17: n=3

Figure 5: Mean partial pressure of oxygen in arterial blood calves across color categories (least squares means ± standard error of the mean).



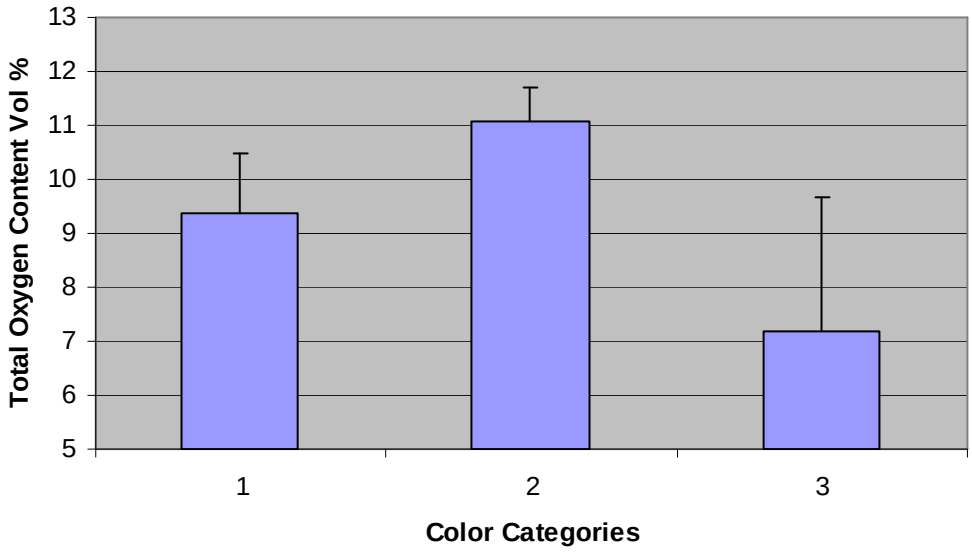
Color Category 1: Final tongue color 1-3: n=11
Color Category 2: Final tongue color 4-9: n=44
Color Category 3: Final tongue color 10-17: n=3

Figure 6: Mean oxygen saturation in arterial blood of calves across color categories (least squares means ± standard error of the mean).



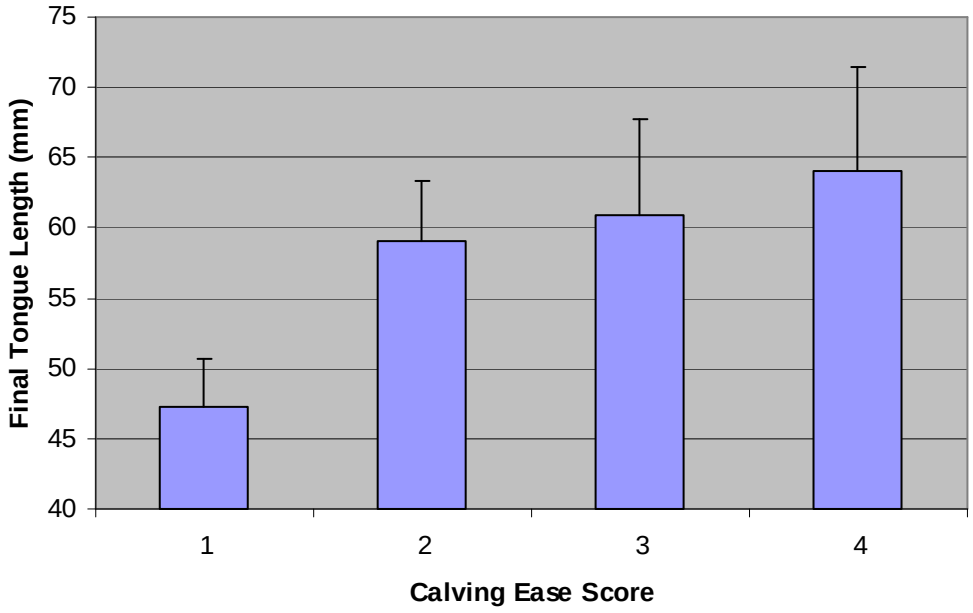
Color Category 1: Final tongue color 1-3: n=11
Color Category 2: Final tongue color 4-9: n=44
Color Category 3: Final tongue color 10-17: n=3

Figure 7: Mean total oxygen content in arterial blood of calves across color categories (least squares means ± standard error of the mean).



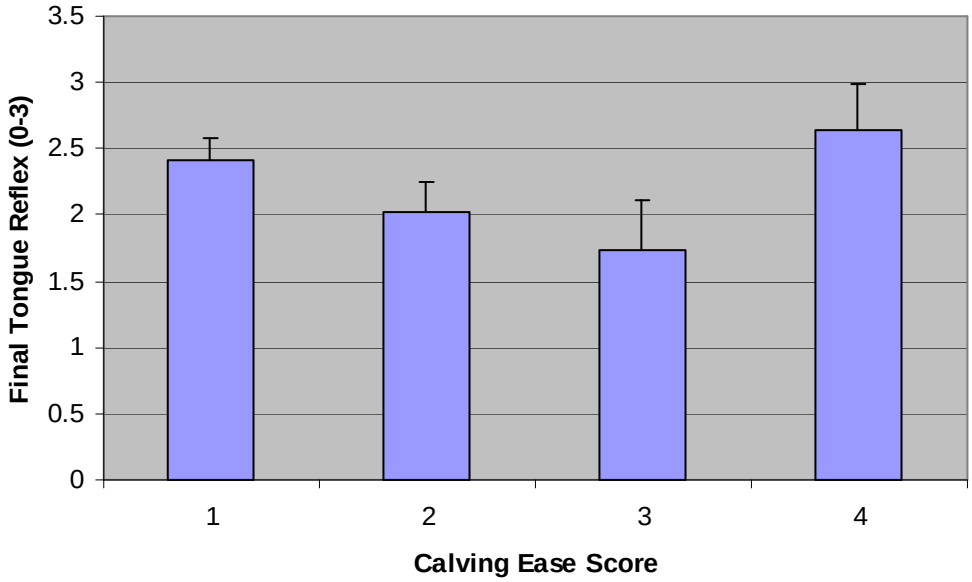
Color Category 1: Final tongue color 1-3: n=11
Color Category 2: Final tongue color 4-9: n=44
Color Category 3: Final tongue color 10-17: n=3

Figure 8: Mean final tongue length of calves across calving ease (least squares means ± standard error of the mean).



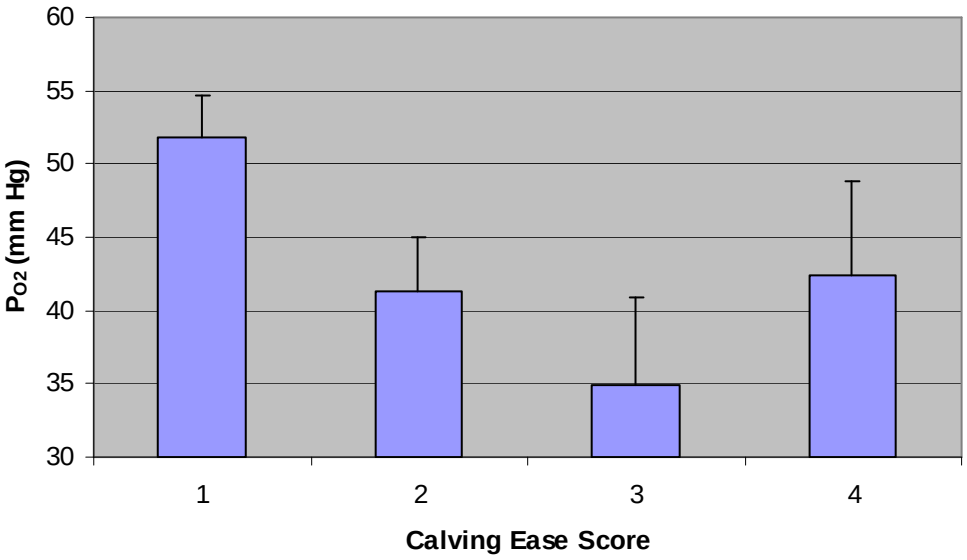
1: cow had no problem during parturition: n=29
2: cow had slight problem during parturition requiring slight assistance: n=17
3: cow required assistance during parturition to extract the calf: n=6
4: considerable force required to extract calf: n=6
5: extremely difficult extraction requiring mechanical assistance: n=0

Figure 9: Mean final tongue reflex of calves across calving ease (least squares means ± standard error of the mean). The higher the number, the greater the tongue reflex response to tactile stimulation.



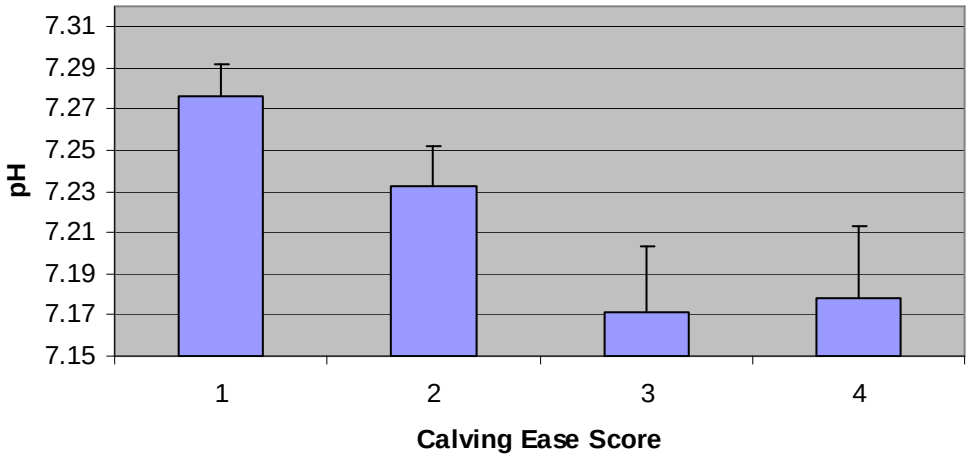
1: cow had no problem during parturition: n=29
2: cow had slight problem during parturition requiring slight assistance: n=17
3: cow required assistance during parturition to extract the calf: n=6
4: considerable force required to extract calf: n=6
5: extremely difficult extraction requiring mechanical assistance: n=0

Figure 10: Mean partial pressure of oxygen in arterial blood of calves across calving ease (least squares means ± standard error of the mean).



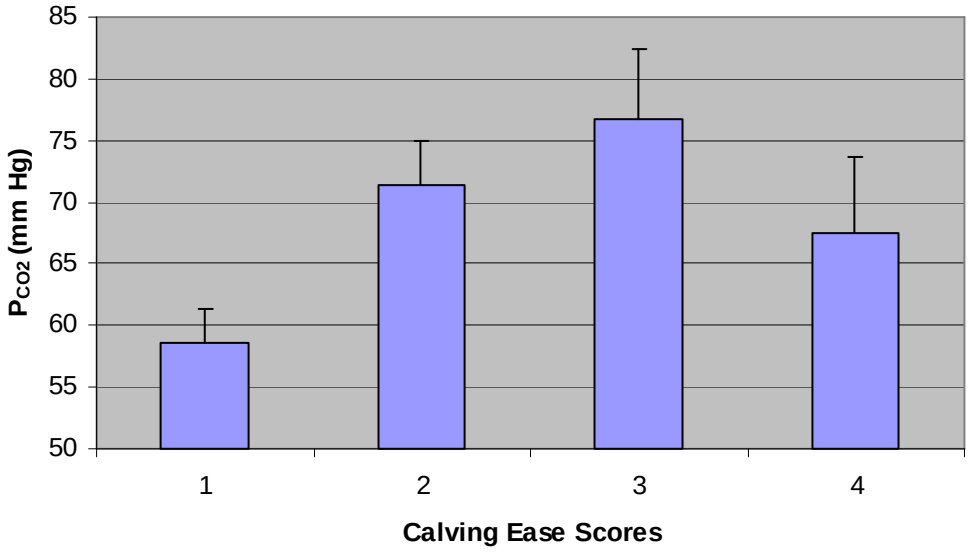
1: cow had no problem during parturition: n=29
2: cow had slight problem during parturition requiring slight assistance: n=17
3: cow required assistance during parturition to extract the calf: n=6
4: considerable force required to extract calf: n=6
5: extremely difficult extraction requiring mechanical assistance: n=0

Figure 11: Mean pH in arterial blood of calves across calving ease (least squares means ± standard error of the mean).



1: cow had no problem during parturition: n=29
2: cow had slight problem during parturition requiring slight assistance: n=17
3: cow required assistance during parturition to extract the calf: n=6
4: considerable force required to extract calf: n=6
5: extremely difficult extraction requiring mechanical assistance: n=0

Figure 12: Mean partial pressure in arterial blood of carbon dioxide of calves across calving ease (least squares means ± standard error of the mean).



1: cow had no problem during parturition: n=29
2: cow had slight problem during parturition requiring slight assistance: n=17
3: cow required assistance during parturition to extract the calf: n=6
4: considerable force required to extract calf: n=6
5: extremely difficult extraction requiring mechanical assistance: n=0

DISCUSSION

Data generated in this study would suggest that individual tongue parameters have limited value for predicting fetal distress in calves. In human infants color is predictive of oxygen delivery parameters, although variation in perception of color is high between individuals (O'Donnell et al., 2007; Dain, 2007; Roberts, 1975). To avoid these issues with variances in color perception, a color chart was developed with 17 discrete colors (Appendix A). Differences in tongue pigmentation between breeds were a confounding factor that may have impacted our ability to detect differences in oxygen status. Pulse oximetry techniques would overcome this limitation, however, the different morphology of the bovine tongue proved to be a difficult obstacle for successful application of this technique. The bovine tongue is thicker and more muscular than tongues of non-ruminants. Other researchers have utilized reflectance oximetry, which uses different diode attachment sites. Reflectance type oximetry has been used extensively and successfully in human intrapartum monitoring (Elchalal et al., 1995), but has had limited success as a technique for bovine intrapartum monitoring (Bleul and Kähn, 2008). Because this technique requires adherence of the diodes to the hard palate, we chose to use a transmission type oximeter with a modified lingual clamp.

Hypoxia also causes loss of muscle control. This issue has been most commonly apparent in meconium-stained calves (Schoon and Kikovic, 1987; Wensvoort, 1968). We used tongue length as an early measure of loss of muscle control. Although our data did not did not confirm a relationship between oxygen delivery parameters and tongue length, we did document a relationship between tongue length, hypercapnia, and acidosis. Since hypercapnia and acidosis are associated with birth stress (Strawn et al., 1996),

tongue length may be a viable predictor for fetal distress. Tongue length was most closely associated with calving ease score in this study.

Calving difficulty in this study was associated with decreased pH, increased P_{CO2}, decreased S_{O2}, and decreased total oxygen content. These data agree with previously reported results from this lab (Strawn, 1996). In addition our data showed that increasing tongue length is also associated with calving difficulty, suggesting that this may be a useful predictive calving management tool.

Due to the differences in tongue color between breeds, the data was analyzed excluding the Jersey calves (n=46). There was a significant negative correlation between initial tongue color and $P_{O2,}$ oxygen saturation of hemoglobin, and total oxygen content (P=.0249, P=0.0416, and P=0.0568). This shows that color can be correlated with oxygen parameters within the Holstein breed. However, there was no significance shown in final tongue color or mean tongue color.

Literature Cited

Bellows, R.A., D.J. Patterson, P.J Burfening, and D.A. Phelps. 1987. Occurrence of neonatal and postnatal mortality in range beef cattle. II. Factors influencing calf death. Theriol. 28:573-586.

Bleul, U. and W. Kähn. 2008. Monitoring the bovine fetus during stage II of parturition using pulse oximetry. Theriol. 69:302-311.

Coté, C.J., A.L. Daniels, M. Connolly, S.K. Szyfelbein, and C.D. Wickens. 1992. Tongue oximetry in children with extensive thermal injury: comparison with peripheral oximetry. Can. J. Anaesth. 39(5):454-457.

Dain, S. 2007. Color changes in cyanosis and the significance of congenital dichromasy and lighting. Color Res. Appl. 32(6):428-432.

Elchalal, U., A. Weissman, Y. Abramov, D. Abramov, and D. Wienstein. 1995. Intrapartum fetal pulse oximetry: present and future. Int. J. Gynecol. Obstet. 50:131-137.

Grignaffini, A., E. Soncini, E. Ronzoni, E. Piazza, S. Anfuso, and E. Vadora. 2004. Meconium-stained aminotic fluid and fetal oxygen saturation measured by pulse oximetry during labour. Acta. Bio. Medica. Ateneo. Parmense. 1:45-52.

Lombard, J.E., F.B. Garry, S.M. Tomlinson, and L.P Garber. 2007. Impacts of dystocia on health and survival of dairy calves. J.Dairy Sci. 90:1751-1760.

Mee, J.F. 2004. Managing the dairy cow at calving time. Vet. Clin. Food Anim. 20:521-546.

Mee, J.F. 2008. Newborn dairy calf management. Vet. Clin. Food Anim. 24:1-17.

Meyer, C.L., J. Berger, and K.J. Koehler. 2000. Interactions among factors affecting stillbirths in Holstein cattle in the United States. J. Dairy Sci. 83:2657-2663

Meyer, C.L., J. Berger, K.J. Koehler, J.R. Thompson, and C.G. Sattler. 2001. Phenotypic trends in incidence of stillbirth for Holsteins in the United States. J. Dairy Sci. 84:515-523.

Neilson, J.P. 2006. Fetal electrocardiogram (ECG) for fetal monitoring during labour. Cochrane Database of Systematic Reviews. Issue 3. Art. No.: CD000116. DOI: 10.1002/14651858.CD000116.pub2.

O'Donnell, C.P.F., C.O.F. Kamlin, P.G. Davis, J.B. Carlin, and C.J. Morley. 2007. Clinical assessment of infant colour at delivery. Arch. Dis. Child Fetal Neonatal Ed. 92:F465-467.

Roberts, S.L. 1975. Skin assessment for color and temperature. Am. J. Nursing. 75(4):610-613.

Schoon, H.A., and D. Kikovic. 1987. Morphological demonstration and pathogenetical evaluation of amniotic fluid aspiration supporting the diagnosis of pulmonary asphyxia in newborn calfs and foals. Dtsch. Tierarztl. Wschr. 94:73-76.

Strawn, K.K., H.D. Tyler, M.A. Faust, and B.J. Nonnecke. 1996. Effects of birth stress in calves. J. Dairy Sci. 79 (Suppl 1):130.

Thompson, J.R., E.J. Pollack, and C.L. Pelissier. 1982. Interrelationships of parturition problems, production of subsequent lactation, reproduction, and age at first calving. J. Dairy Sci. 66:1119-1127.

Uystepruyst, CH., J. Coghe, F. Bureau, and P. Lekeux. 2000. Evaluation of accuracy of pulse oximetry in newborn calves. Vet. J. 159:71-76.

Wells, S.J., D.A. Dargatz, and S.L. Ott. 1996. Factors associated with mortality to 21 days of life in dairy heifers in the United States. Prev. Vet. Med. 29:9-19.

Wensvoort, P. 1968. Post mortal findings in lambs which died of asphyxia during parturition. Tijdschr. Diergeneesk. 93:1270-1278.

CHAPTER THREE

GENERAL CONCLUSIONS

This research represents a continuing effort to understand and establish methods of assessing fetal stress. The transition from fetal life to the neonate has a major impact on calf survivability. The objective of the study was to assess the ability of several tongue parameters and transmittance tongue oximetry to detect fetal stress.

This study provided several starting points for future studies. Tongue parameters may be an effective method for monitoring fetal stress in cattle. Tongue length monitoring was indicative of acidosis in calves. This is due to the loss of muscle control and contractility that occurs during stress. Tongue reflex was shown to be negatively correlated with tongue length. Calves with longer tongues are less responsive to tactile stimulation. When these data were analyzed after the exclusion of Jersey calves (that have naturally darker tongues), there was a significant correlation between initial tongue color and oxygen parameters. Our data, while not conclusive, strongly suggest that tongue parameters can be useful as predictive measures of stress, but when all three stress indicators are present in the calf (long tongue, reduced reflex, and dark color) it is more definitively indicative of stress than the presence of a single indicator. There were six calves in the study that had long tongues (greater than 55 mm), dark color (color 3 or lower), and poor responsiveness (reflex equal to 1). Five out of the six calves or 83% were stressed ($pH \leq 7.25$, $P_{O2} < 50$, and $P_{CO2} > 60$). However, there were 12 total stressed calves six of which did not display two or more tongue parameters measured.

Due to the variability of transmittance pulse oximetry and success of reflectance oximetry reported by Bleul and Kähn (2008), reflectance oximetry could be used to better

correlate tongue parameters to stress. Ideally, a large number of Holstein calves would be monitored in a futures study. Reflectance oximetry during delivery and arterial blood gases at birth can be utilized to assess stress and then these data can be correlated with tongue paramters.

Our data can provide a basis for new more useful techniques in fetal monitoring. Calving management practices would benefit from an easy way to determine stress in the calf. Such techniques could potentially reduce calf morbidity and mortality and increase future production for these animals.

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Appendix: Color Chart