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To the Graduate Council:

I am submitting herewith a thesis written by Jacqueline Paradeis Drewry entitled "Effects of arterial pCO2 on IgG absorption efficiency in neonatal Holstein calves." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Animal Science.

J.D. Quigley III, Major Professor

We have read this thesis and recommend its acceptance:

F. Hopkins, M. Welborn, D. Geiser

Accepted for the Council:
Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

To the Graduate Council:

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Accepted for the Council:

Associate Vice Chancellor and
Dean of The Graduate School

EFFECTS OF ARTERIAL pCO_2 ON IgG ABSORPTION EFFICIENCY IN NEONATAL HOLSTEIN CALVES

A Thesis

Presented for the

Master of Science

Degree

The University of Tennessee, Knoxville

Jacqueline Paradeis Drewry

August 1997

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ABSTRACT

Absorption of colostral Ig within the first 24 h of life provides the neonate with passive immunity. Delayed first feeding of colostrum, low colostral Ig concentration or compromised metabolic state may reduce the apparent efficiency of Ig absorption (**AEA**) calculated as the mass of Ig in plasma (g) divided by Ig intake (g). Objectives of this study were to determine if elevated arterial pCO_2 ($PaCO_2$) in blood reduced AEA in calves and to determine if assisted ventilation of animals with elevated $PaCO_2$ increased AEA. Holstein calves (n = 48) from primiparous and multiparous dams were separated at birth and blood was collected at 1 h and analyzed for $PaCO_2$, arterial pO_2 (PaO_2), arterial pH (pHa), arterial actual bicarbonate (HCO_3^-), and base excess. Three treatments were assigned based on $PaCO_2$ at 1 h: non respiratory acidosis (**NA**; n = 19), respiratory acidosis-not ventilated (**ANV**; n = 17), and respiratory acidosis-ventilated (**AV**; n = 12). Assisted ventilation was administered via Ambu Bag™ with mask at 1.5 h. Calves received 12 assisted breaths per min for 5 min. Post ventilation blood gas analysis indicated this method reduced $PaCO_2$. Mean 1 h $PaCO_2$ for calves on **NA**, **ANV** and **AV** treatments were 45.52, 53.04, and 53.22 (SE = 0.71) mm of Hg, respectively. Blood was sampled by jugular venipuncture at 1, 13, 25, 37, and 49 h for analysis of plasma IgG by single radial immunodiffusion. Evans' blue dye (1.5 ml) was injected into the jugular vein for estimation of plasma volume and AEA. Treatment had no effect on AEA at 25 h, plasma IgG at 13, 25 or 37 h or plasma volume at 25 h ($P > 0.10$). Mean AEA, plasma IgG and plasma volume at 25 h of age were 26% (SE = 1.5), 11.73 g/L (SE = 0.50) and 3.422 L (SE = 0.001), respectively. Regression of AEA and plasma IgG on $PaCO_2$ at 1 h

indicated no effect of 1 h $PaCO_2$ on IgG absorption. Results from this study indicate that acquisition of passive immunity was not inhibited by elevated $PaCO_2$ at 1 h.

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1. INTRODUCTION

High preweaning calf mortality is responsible for significant economic losses in dairy calves. Reports from the National Animal Health Monitoring System (NAHMS, 1996) reported an 11% preweaning calf mortality in the U.S. during 1995. Scours and respiratory problems comprised the greatest percentage of losses, accounting for 60.7 and 24.5% of deaths, respectively. Many factors may contribute to early calfhood mortality. Research has indicated a large percentage of death occurs when calves fail to achieve adequate levels of serum immunoglobulin (Ig) concentrations (Gay et al., 1965; Boyd, 1972; Gay, 1983; Gay, 1984; Hussni et al., 1991; Besser and Gay, 1994). This is referred to as failure of passive transfer (FPT). Serum IgG is used as a reference molecule for indicating FPT, because of its long half life and prevalence in the circulation (Gay, 1984). A 24 h serum IgG_1 of 10 g/L has been suggested as a minimum IgG in calves to prevent FPT and provide adequate protection during the early neonatal period (Gay, 1983; Besser and Gay, 1994).

The transfer of Ig to the calf is influenced by a number of factors, including age at first feeding, volume and Ig concentration of colostrum fed, birth weight, method of feeding, feeding of maternal versus pooled colostrum, seasonal influences, stress, disease, use of colostral supplements, presence of the dam, and individual calf variation in efficiency of absorption (Kruse, 1970b; Selman et al., 1971; Gay, 1984; Petrie, 1984). The physiological status of the calf at birth and during the early postpartum period may also influence the transfer of colostral Ig. Factors controlling acid-base balance may impair colostral Ig absorption (Eigenmann et al., 1983; Boyd, 1989; Besser et al., 1990;

Guy et al., 1996).

Eigenmann et al. (1983) observed a 52% decrease in colostrum intake resulting in a 35% decrease in serum IgG concentration in calves considered to be severely acidotic (plasma pH < 7.15) when compared to calves with a plasma pH > 7.25. However, decreased serum IgG concentration may be the result of decreased intake, rather than the acid-base status in these calves. Mean plasma IgG concentration in calves suffering from disease during the first week of life was 19.8 g/L whereas healthy calves had a mean plasma IgG concentration of 26.0 g/L. However, Boyd (1989) did not report a negative correlation between post feeding serum IgG_1 concentration and prefeeding venous blood pH (pHv), although, there was a significant correlation between prefeeding venous pCO_2 ($PvCO_2$) and post feeding serum IgG_1 concentration. Calves were in a state of partially compensated respiratory acidosis. From his results, Boyd concluded that reduced absorption of IgG_1 from colostrum was associated with hypercapnia in apparently healthy newborn calves.

Besser et al. (1990) confirmed the work of Boyd (1989), reporting that decreased IgG_1 absorption from colostrum was associated with respiratory acidosis, rather than metabolic acidosis. Blood was analyzed for $PvCO_2$, pHv and venous bicarbonate concentration (HCO_3^-). Lower blood pHv at 2 and 4 h after birth was associated with decreased IgG_1 absorption in one study but not in a second. Both studies reported a significant inverse relationship between blood $PvCO_2$ and serum IgG_1 concentration absorbed from colostrum. Results indicated that despite adequate colostrum intake, postnatal respiratory acidosis in calves adversely affected serum IgG_1 concentration

absorbed from colostrum.

Boyd (1989) and Besser et al. (1990) analyzed serum IgG_1 concentrations 12 h after initial colostrum feeding to determine the effects of acid-base status on serum IgG_1 concentration. However, measuring serum IgG_1 concentration 12 h after feeding may not accurately indicate the true peak serum IgG_1 absorption. Tyler and Ramsey (1991a) observed an extended period of IgG absorption in newborn calves with altered PaO_2 when compared to calves with unaltered PaO_2. It is possible that altered $PaCO_2$ may exert a similar effect. Furthermore, previous studies failed to stress the relevance of arterial sampling on blood gas analysis. Arterial blood is the most accurate indicator of acid-base status, oxygenation and respiratory function and therefore the only accurate indicator of respiratory acidosis (Filley, 1971; Adams et al., 1991). Finally, since absorption of Ig is influenced by several factors (Gay, 1984; Petrie, 1984; Hussni et al., 1991), variation in these factors should be accounted for. Standardized research accounting for a number of these factors has not been conducted to determine the true effect of respiratory acidosis on the absorption of colostral Ig by calculating an AEA in calves under the same conditions and during the same season.

2. LITERATURE REVIEW

Acid-Base Balance during the Perinatal Period

Introduction

During the periparturient period the calf undergoes the most dramatic adjustments that occur in life. The ability of the neonate to successfully make the transition from dependance on the dam's circulation to initiation of spontaneous respiration and self sufficiency has a tremendous influence on the acid-base status of the calf. Acid-base balance and blood gas physiology are highly complex processes in which four variables are controlled; pCO_2, the bicarbonate ion (HCO_3^-), pH and pO_2. Furthermore, metabolism is essentially aerobic and respiratory function is closely coupled with metabolic demands and functions.

The three highly regulated variables within the acid-base system are pCO_2, pH, and HCO_3^- (Gardner, 1978). They are expressed in different units and have different roles with respect to time and mode of action. For example, pCO_2 is regulated by the lungs and is expressed as an intensity factor in CO_2 transport. The HCO_3^- is a quantity factor in acid-base regulation. It is expressed as a concentration in milliequivalents per liter (mEq/L) or mmol/L and is largely controlled by the kidneys. Finally, pH is the measure of the electrochemical potential of protons and indicates acidity (Filley, 1971; Kirk, 1983). It is the negative logarithm of the H^+ concentration. Both pCO_2 and HCO_3^- concentration have a role in the regulation of pH.

Carbon dioxide

Carbon dioxide is the least stable parameter in the acid-base system and changes more rapidly than other variables (Filley, 1971). It is the acid component of the CO_2 /HCO_3^- buffer system and is regulated by respiration (Gardner, 1978). It is produced in every tissue cell and diffuses through all body compartments (Filley, 1971). Considering the amount produced by normal sedentary humans is 15,000 mEq CO_2 per day and its potential toxic effect, the physiological significance of CO_2 regulation is paramount for homeostatic maintenance and acid-base regulation in humans as well as other species (Filley, 1971; Haskins, 1977a; Gardner, 1978; Guyton and Hall, 1996).

The pCO_2 is the partial pressure of CO_2 and is expressed in mm of Hg. The pCO_2 can also be defined as a kilopascal (kPa) where one kPa is equivalent to 7.5006 mm of Hg (Gardner, 1978). It is the tendency of CO_2 molecules to move from a region of high CO_2 concentration to a region of low CO_2 concentration (Filley, 1971; Guyton and Hall, 1996). Blood CO_2 is controlled primarily by the lungs' ability to eliminate CO_2. The CO_2 diffuses from the capillary venous blood into the alveoli of the lungs via a partial pressure gradient between these two areas. The CO_2 is then removed from the alveoli by ventilatory exchange of atmospheric air with alveolar gases. The $PaCO_2$ is established on the basis of the extent to which ventilation affects the alveolar partial pressure which in turn determines the amount of CO_2 diffusing from the capillaries (Kirk, 1983). The partial pressure of CO_2 is greater in blood, causing net diffusion toward the gas phase in the alveoli, and escape from the lungs (Guyton and Hall, 1996).

Normal $PaCO_2$ in adult awake mammals ranges from 35 to 45 mm of Hg (Kirk,

1983; Smith, 1996). Often an increase in the partial pressure of $PaCO_2$ lowers pHa resulting in a mild acidosis (Garry, 1993). Researchers (Walser and Maurer-Schweizer, 1979; Szenci, 1985b; Blood and Radostits, 1989; Besser et al., 1990; Garry, 1993) have determined that acid-base imbalance is common in bovine neonates. Metabolic, respiratory and mixed acidosis occur frequently, while significant alkalosis is rarely seen (Garry, 1993). Risk factors associated with postnatal acidosis include: 1) duration of observed second stage labor greater than 2 h; 2) dystocia requiring traction; and 3) weakness of a calf at birth (Szenci, 1985a; Besser et al., 1990; Kasari, 1994).

Adams et al. (1993) reported mean $PaCO_2$ in normal calves from single deliveries at 1 h after birth to be 50.4 ± 5.27 mm of Hg. This was not significantly different from twin calves with $PaCO_2$ of 47.56 ± 4.44 mm of Hg. Similar research by Eigenmann et al. (1984) observed mean $PvCO_2$ in healthy calves not exhibiting respiratory distress syndrome of 58 ± 2 mm of Hg. However, this experiment utilized venous samples which expresses elevated pCO_2 and decreased pH compared to arterial samples (Szenci, 1985b). Mean $PaCO_2$ in foals at birth was 60.7 ± 1.5 mm of Hg decreasing to 50.4 ± 2.0 mm of Hg at 15 min (Stewart et al., 1984).

Garry (1993) determined mean $PaCO_2$ of 50 mm of Hg and a range of 43 to 59 mm of Hg to be expected values for healthy newborn calves. With the onset of steady respiration, these levels dropped to adult levels (35 to 45 mm of Hg) within 24 to 48 h (Kirk, 1983; Haskins, 1977a). Chemosensitive respiratory reflexes of newborn infants respond to hypoxia and hypercapnia by increasing ventilation (Kirk, 1983). However, neonates have a diminished ventilatory response to CO_2. A similar response may be

expected in calves (Stewart et al., 1984). Some research indicates that calves born acidotic as the result of increased $PvCO_2$, are not able to compensate for this increase in $PvCO_2$ by as late as 48 h after birth (Boyd, 1989; Besser et al., 1990; Kasari, 1994). This is commonly referred to as respiratory acidosis since the respiratory component, CO_2, and not the metabolic component, HCO_3^-, is the factor altering pHv.

Published research using mammals often reports blood gas values obtained from venous samples. Moore (1969) and Szenci (1985b) reported that $PvCO_2$ levels ranging from 41 ± 5.9 to 67.4 ± 7.2 mm of Hg were representative of newborn calves. Values greater than 65 mm of Hg occurred in two calves whose births were delayed by dystocia (Moore, 1969). However, venous samples are less satisfactory than arterial samples for pCO_2 and should be restricted to evaluating metabolic conditions (Kirk, 1983; Kaneko, 1989; Smith, 1996). Effects of regional metabolism and circulation on venous blood alter the CO_2 tension and pH in an unpredictable manner such that the respiratory contribution cannot be accurately assessed (Donawick and Baue, 1968). Furthermore, application of a stasis during venous sampling will cause blood from a peripheral vein to become engorged. The results may be extremely misleading due to the stagnation of venous blood, causing $PvCO_2$ to appear higher and pHv to appear lower compared to arterial blood (Filley, 1971; Haskins, 1977b; Szenci, 1985b). To reduce perinatal mortality resulting from acid-base disturbances, it is necessary to understand the rapid changes the cardiopulmonary system undergoes during the successful transition to extrauterine life. Blood gas measurements on arterial blood are the only practical method of monitoring these changes (Adams et al., 1991). Arterial samples are the most accurate indicator of

respiratory function, oxygenation and metabolic acid-base balance and therefore the most accurate indicator of blood $PaCO_2$ (Filley, 1971; Adams et al., 1991).

Blood CO_2 is regulated by the respiratory system and controlled by central chemoreceptors located in the medulla oblongata (Gardner, 1978; Guyton and Hall, 1996). These chemoreceptors are highly sensitive to changes in both $PaCO_2$ and pHa (Gardner, 1978). Respiration rate doubles when $PaCO_2$ is increased by only a few mm of Hg. Likewise, inhalation of CO_2 enriched air or a slight drop in pHa near or in the cells of the receptors will cause immediate hyperventilation (Filley, 1971; Kirk, 1983). Medullary chemoreceptors are far more receptive to alterations in $PaCO_2$ than to hypoxia (Gardner, 1978; Tyler and Ramsey, 1991b). In calves subject to hypoxic conditions, receiving inspired air with 10.5% O_2, Tyler and Ramsey (1990; 1991b) reported similar $PaCO_2$ in both hypoxic and normoxic calves. In a similar study with three day old piglets, Torrance and Wittnich (1992) concluded that development of clinical hypercapnia only occurred with severe hypoxia. Animals exposed to acute hypoxia were able to maintain normal acid-base homeostasis.

The pCO_2 control mechanism is active in utero and the bovine fetus exhibits some degree of compensation for acute acidosis in the dam during the terminal stage of pregnancy. Szenci (1982) administered a single large dose of sucrose at 269 days of gestation to induce acute acidosis. The response of the dam was a moderately severe acidosis which lasted only one day. The acid-base status of Caesarean-derived calves from these cows did not differ significantly from the control group, indicating compensation from short-term maternal acidosis occurred in utero. In the same study,

calves born to dams showing a progressively aggravating and severe acidosis, lasting up to 7 d, were either born acidotic or developed acidosis within a few hours of birth.

Other researchers have examined the effect of dry cow diets on the acid-base status of the neonate. Tucker et al. (1992) indicated that the cation-anion balance of the diet consumed by dry cows did not affect the acid-base status or plasma mineral content of their calves. The objectives of their study were to evaluate the effectiveness of a low dietary cation-anion difference [$(Na^+ + K^+) - (Cl^- + S^-)$] in preventing milk fever and udder edema in dry cows consuming high-Ca diets and to determine effects of this diet on calves delivered by these cows. In a similar study, Guy et al. (1996) found no difference in $PvCO_2$ or pHv in calves born to cows fed anionic diets when compared to calves born to cows fed cationic diets.

HCO_3^- and its relationship with pCO_2

Most blood CO_2 is carried as a bicarbonate ion (HCO_3^-) and the remainder is carbonic acid (H_2CO_3) and CO_2 gas (Filley, 1971; Kirk, 1983). The HCO_3^- is often referred to as the metabolic or non-respiratory component in acid-base regulation (Haskins, 1977a). It is affected by the metabolic production of acid and base and activity of the kidneys (resorption and synthesis of HCO_3^-; Gardner, 1978). The metabolic component does not influence acid-base balance and pH in isolation because of its relationship with CO_2 (Haskins, 1977a). The HCO_3^- concentration can be misleading as a quantitative estimate of the metabolic component of acid-base regulation because a primary change in CO_2 directly causes a change in HCO_3^- concentration (Haskins, 1977a; Gardner, 1978).

The interrelationship between the three forms of carbon dioxide is expressed by the carbonic acid equilibrium equation:

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

(Filley, 1971; Gardner, 1978; Kirk, 1983). The law of mass action applies to this equilibrium at all times. For instance, high H^+ concentrations move the equation to the left, decreasing HCO_3^- concentration and increasing CO_2, resulting in acidosis. In healthy individuals this does not occur frequently due to the rigid control of the respiratory center (Gardner, 1978).

However, H^+ concentration may increase in response to the production of non-volatile acids. One example is hypoxia, common during parturition, when fetal membranes rupture and uterine contractions begin. Tissue hypoxia is also a frequent occurrence during periods of intense exercise (Gardner, 1978; Szenci, 1985a; Szenci, 1985b; Tyler and Ramsey, 1990; Garry, 1993; Kasari, 1994). Hypoxia causes lactic acid (the product of anaerobic glycolysis) to accumulate in the blood. Lactic acid is a strong acid with the ability to deplete plasma HCO_3^-, lowering pH and resulting in a mild to severe metabolic acidosis (Gardner, 1978; Kasari, 1994).

Elevated pCO_2 and decreased pH seen in respiratory acidosis will inevitably result in increased HCO_3^- concentration (Haskins, 1977a; Gardner, 1978). The increased HCO_3^- concentration would not be considered metabolic alkalosis because the primary component disorder would be of respiratory origin and not metabolic origin. Likewise, depressed pH, decreased HCO_3^- concentration, and depressed or normal pCO_2 would

indicate an acidosis of metabolic origin.

Researchers have attempted to eliminate the respiratory influence on HCO_3^- concentration by restoring in vitro samples to a $PaCO_2$ of 40 mm Hg under standard conditions of body temperature and complete hemoglobin oxygenation (Haskins, 1977a). This is referred to as standard HCO_3^- or T_{40} HCO_3^- and is commonly used for clinical evaluations (Donawick and Baue, 1968; Haskins, 1977a).

The normal HCO_3^- concentration in adult mammals is 24 ± 3 mEq/L (Kirk, 1983; Smith, 1996). Concentrations less than 21 mEq/L indicate metabolic acidosis and concentrations in excess of 27 mEq/L indicate metabolic alkalosis (Kirk, 1983). Decreased HCO_3^- concentration indicates consumption of HCO_3^- to neutralize acid released during metabolic acidosis (Torrance and Wittnich, 1992). Szenci (1985b) and Moore (1969) determined representative values in healthy newborn calves to be 24.2 ± 2.7 to 28.2 ± 4.4 mEq/L. Calves born with HCO_3^- concentrations as low as 15 mEq/L can develop a plasma HCO_3^- concentration of 27 mEq/L by 24 h without medical intervention (Garry, 1993). Similar research demonstrated increasing HCO_3^- concentration in newborn foals during the first 2 days of life (Stewart et al., 1984). This study observed a mean umbilical artery standard HCO_3^- of 24.0 ± 0.8 mEq/L at 1 h after birth increasing to 25.7 ± 0.6 mmol/L by two days.

Calves exposed to hypoxic conditions exhibit HCO_3^- concentrations as low as 13 to 18 mmol/L (Tyler and Ramsey, 1990). Hypoxic conditions were imposed by introducing a mixture of inspired gas via a face mask for 24 h. Decreased HCO_3^- concentration was the result of a shift in the ratio of HCO_3^- : H_2CO_3 since $PaCO_2$

remained stable. The HCO_3^- concentration in these calves returned to normal levels at 24 h, following the return of PaO_2 to normal levels.

Base Excess/deficit

Base excess/deficit (BE/BD) is another method of quantifying the metabolic contribution to the acid-base balance in clinical situations (Kirk, 1983). It is defined as the titratable acidity/alkalinity of a blood sample at 37°C, at complete hemoglobin saturation and a $PaCO_2$ of 40 mm Hg when titrated to a pHa of 7.4 (Haskins, 1977a; Kirk, 1983). Base excess/deficit provides a quantitative estimation of the surplus acid or base. The normal base excess/deficit is 0 ± 4 mEq/L (Haskins, 1977a). Positive values greater than 4 mEq/L indicate an excess of base (alkalosis); values less than -4 mEq/L indicate a deficit of base (acidosis; Donawick and Baue, 1968; Haskins, 1977a; Kirk, 1983).

Szenci (1985b) viewed calving information from approximately 140,000 calves born in 1978 and 1979. The study utilized venous samples to determine acid-base parameters. Base excess for calves born unassisted in anterior presentation did not decrease below -3 mmol/L. Mean pHv for this group of calves was 7.2 ± 0.052. From this information, Szenci concluded that respiratory-metabolic acidosis was physiological (Szenci, 1985b). In the same study, calves extracted with assistance in anterior or posterior presentation were born exhibiting respiratory-metabolic acidosis. Blood pHv and BE were between 7.2 and 7.0 and -3 to -10 mmol/L, respectively.

pO_2

The PaO_2 is an index of the functional efficiency of the lungs (Kirk, 1983). Unlike $PaCO_2$ and HCO_3^- concentration, PaO_2 does not directly influence pHa. However,

its indirect influence on acid-base balance has been demonstrated in neonatal piglets, calves, and foals (Sonea, 1985; Torrance and Wittnich, 1992; Kasari, 1994). The normal range is 70 to 100 mm of Hg in adult awake mammals (Kirk, 1983). Position during sampling can have a marked influence on PaO_2 concentrations. A mean decrease of 14 mm Hg in PaO_2 was observed when comparing samples taken in the upright position to samples taken in lateral recumbency (Wagner et al., 1990; Smith, 1996). Wagner et al. (1990) confirmed that, in the absence of sedative or anesthetic drugs, both lateral and dorsal recumbency in cattle significantly impair arterial oxygenation. Hypoventilation reduces PaO_2 while hyperventilation increases PaO_2.

Uterine contractions during parturition impact the availability of O_2 to the fetus. Blood flow is reduced with each contraction resulting in intermittent tissue hypoxia (Randall, 1978; Kasari, 1994). Hypoxia arises during normal parturition but it is especially frequent during prolonged second stage labor and dystocia (Garry, 1993; Schuijt and Taverne, 1994). Following disruption of umbilical blood flow, high lactate concentrations, resulting from the breakdown of glycogen, are present in the newborn calf (Randall, 1978; Kasari, 1994). During asphyxia, blood pHa falls, due, in part, to increased $PaCO_2$, but mainly to the production of lactic acid (Randall, 1978). Lactic acid requires neutralization by HCO_3^- and decreased HCO_3^- concentration results in metabolic acidosis (Torrance and Wittnich, 1992). The improvement of systemic circulation at birth causes lactic acid, previously accumulated in tissue supplied with a minimum of O_2, to be released from this tissue into the circulation (Walser and Maurer-Schweizer, 1979). This can further depress pH. During the following hours, normalization of acid-base

balance and respiration occur, thereby, correcting the metabolic acidosis (Kasari, 1994).

Torrance and Wittnich (1992) produced significantly depressed PaO_2 in 3 day old piglets by exposure to severe hypoxia. Hypoxemia resulted from providing piglets with a 95% N: 5% CO_2 inhaled mixture. Mean pHa of the severe hypoxic group was 7.14 ± 0.08 thirty minutes post ventilation, compared to a mean pHa of 7.33 ± 0.03 in normoxic piglets. Adams et al. (1993) observed a mean PaO_2 1 h post calving of 43.7 ± 18.6 mm of Hg with a corresponding mean pHa value of 7.30 ± 0.05. At 24 h, PaO_2 had increased to 68.54 ± 9.09 mm of Hg with a corresponding mean pHa of 7.4 ± 0.03. Likewise, PaO_2 values as low as 39 mm of Hg and pHa values of 7.30 for foals at birth are common (Sonea, 1985). The PaO_2 of foals stabilized at 75 mm of Hg within 4 h of birth (Sonea, 1985; Smith, 1996).

pH

Both pCO_2 and HCO_3^- are uniquely involved in the regulation of pH. The Henderson-Hasselbalch equation states that pH is the logarithm of the ratio of concentration of base to concentration of acid. The CO_2 is the acid fraction and HCO_3^- is the base fraction of pH regulation (Haksins, 1977a; Gardner, 1978). More precisely, pH is viewed as: pH = pK + log [base] /[acid]. This equation can be expressed as: pH = pK + log $[HCO_3^-]$ / $[CO_2]$. The CO_2 and HCO_3^- are in equilibrium with each other and the actions they exert are not independent. A rise in CO_2 will result in an increase in HCO_3-, but the increase in HCO_3- will be proportionally smaller due the logarithmic relationship of the two variables and therefore pH will decrease (Gardner, 1978).

When clinically evaluating acid-base values, pHa should be the first consideration

(Haskins, 1977a). Subsequent evaluation of the respiratory ($PaCO_2$) and the metabolic (HCO_3^- concentration and base excess/deficit) components assist in determining relative contributions of each parameter to pHa. Rarely does a natural compensatory mechanism overcompensate, and as a general rule, pHa will vary in the direction similar to the primary component disorder (Haskins, 1977a).

Normal pHa in adult mammals ranges between 7.35 and 7.40, whereas pHv and interstitial fluids are at the lower end of this range or approximately 7.35 (Haskins, 1977a; Kirk, 1983; Guyton and Hall, 1996). The difference between arterial and venous pH is the result of CO_2 released from the tissue forming H_2CO_3. A pHa range of 7.20 to 7.60 is not considered life threatening (Filley, 1971; Kirk, 1983), although others have reported that the tolerable range was 7.0 to 7.8 (Haskins, 1977a). However, pHa values below 7.00 and above 7.80 are considered to be extreme threats to the welfare of the animal and are not conducive to prolonged survival (Kirk, 1983).

A representative range of pHv in newborn calves is 7.22 ± 0.05 to 7.24 ± 0.08 (Moore, 1969; Szenci, 1985). Samples from these studies were taken immediately and at 1 h postpartum, respectively. However, method of calf delivery may affect blood pHv (Szenci, 1985a; Schuijt and Taverne, 1994). Calves not assisted at parturition had mean pHv at 10 min of 7.10 ± 0.20. Mean pHv for calves requiring forced extraction was 7.02 ± 0.14. Garry (1993) reported a mean pHa at birth in calves of 7.30 compensating without intervention by 24 h to 7.40. Foals also establish pHa near 7.40 after parturition (Stewart et al., 1984). Mean umbilical blood pHa for 10 foals at birth was 7.30 ± 0.02, increasing to 7.36 ± 0.01 by 2 h postpartum.

Mild metabolic and respiratory acidosis frequently occurs during the immediate postpartum period. Some researchers view this as physiological (Assali, 1963; Walser and Maurer-Schweizer, 1979; Szenci, 1985b; Garry, 1993; Kasari, 1994), while others consider it abnormal and a possible threat to the health and survival of the neonate (Boyd, 1989; Besser et al., 1990). Neonatal calves and foals have internal homeostatic mechanisms compensating, to some degree, for the alterations in blood pH, pCO_2, HCO_3^- concentration, and base excess/deficit at and immediately after birth (Stewart et al., 1984; Garry, 1993). Other research indicates the metabolic (HCO_3^- concentration or base excess/deficit) portion will resolve itself within the first 4 h of life while the respiratory portion (pCO_2) will prevail as an abnormality for 48 h or longer (Boyd, 1989; Besser et al., 1990). The prevalence of respiratory acidosis during the immediate postpartum period could inhibit the neonate's ability to adapt to the extra uterine environment. It is during this period that respiration and circulation are up regulated and absorption of colostral immunoglobulins occurs.

Immunoglobulins and Passive Immunity

Introduction

The transfer of maternal Ig across the placenta does not occur in domestic livestock; thus animals are born with low levels or no circulating antibodies. This condition is referred to as hypo- or agammaglobulinemia. Passively acquired immunity does not reproduce and provides protection in domestic farm animals from birth to the onset of endogenous Ig production (Brambell, 1958). Immunity developed in response to antigen exposure is referred to as active immunity and is characterized by the production of antibodies. Animals can continue to produce antibodies in decreasing quantities for long periods of time after exposure and throughout life, without further exposure to that antigen (Brambell, 1958). Examples of animals receiving Ig by passive immunity are the piglet and calf. Piglets acquire approximately 3 g of Ig within the first few hours after birth and the calf may acquire twenty times that amount (Porter, 1979) or more.

Species variations exist in the placentation and transfer of antibodies. The epitheliochorial placenta of pigs and horses, consisting of 6 tissue layers, and the syndesmochorial placenta of ruminants, consisting of 5 tissue layers, do not allow the fetal blood to intermingle with maternal blood (Brambell, 1958; Tizard, 1987; Knobil and Neill, 1994). Placenta is classified as hemochorial in humans and other primates (Tizard, 1987; Hafez, 1993). Primates allow iron and IgG to cross the trophoblast via a receptor-mediated transport mechanism providing the neonate with Ig in the blood at birth comparable to the level and antigen specificity of the mother (Brambell, 1958; Tizard, 1982; Knobil and Neill, 1994).

Because domestic farm animals are born hypogammaglobulinemic, they must ingest colostrum as their source of circulating Ig during the early postpartum period. Low levels of serum Ig have a significant relationship to high incidence of preweaned calf mortality and morbidity (Gay et al., 1965; Klaus et al., 1969; Kruse, 1970b; Selman et al., 1971; Boyd, 1972; Penhale et al., 1973; Williams et al., 1975; Besser and Gay, 1994; Gay, 1994). In a farm study, Boyd (1972) determined that calves with significantly lower post-colostral serum Ig, total protein and specific gravity scoured more frequently than calves with higher post-colostral serum Ig, total protein and specific gravity. Williams et al. (1975) reported an association between low levels of serum Ig and higher incidence of pneumonia in calves. Similar research reported a 17.4% mortality rate among calves that were markedly or totally deficient in serum Ig (Gay et al., 1965). This indicates the close association between mortality and morbidity and low serum Ig.

Immunoglobulins: classification and function

Immunoglobulins are divided into 5 classes: IgG, IgA, IgM, IgE and IgD. These large protein macromolecules range in size from 150,000 to 900,000 d (Bush and Staley, 1980; Butler, 1981; Tizard, 1987; Halliwell and Gorman, 1989). Properties and functions of different classes of Ig allow them to be divided into subclasses which vary among species. For example, four subclasses of IgG are recognized in humans: IgG_1, IgG_2, IgG_3 and IgG_4 and three subclasses of IgG are recognized in horses: IgGa, IgGb and IgGc (Halliwell and Gorman, 1989). Two IgG subclasses have been widely recognized and extensively analyzed in cattle: IgG_1 and IgG_2 (Butler, 1971; Porter, 1979; Halliwell and Gorman, 1989).

Cattle have four distinct classes of immunoglobulins: IgG, IgA, IgE, and IgM (Penhale and Christie, 1969; Butler,1971; Thatcher and Gershwin, 1989; Roy, 1990). The IgG fraction, consisting of IgG_1 and IgG_2, represents the largest portion of serum and colostral Ig or approximately 90% of the total Ig (Oyeniyi and Hunter, 1978; Fallon, 1990; Roy, 1990; Besser and Gay, 1994). Serum IgG concentrations in adult cattle, representing the 90% of total Ig, are comprised of approximately 11.0 g/L and 7.9 g/L for IgG_1 and IgG_2, respectively (Penhale and Christie, 1969; Fallon, 1990). The remaining 10% in serum is represented by portions of IgA, IgE, and IgM with IgM contributing a greater portion in most species including cattle (Tizard, 1987; Fallon, 1990; Roy, 1990). Humans are the exception having higher IgA concentrations compared to IgM (Tizard, 1987).

The IgM class appears to prevent systemic invasion and induce rapid intravascular detoxification of endotoxins such as those produced by *E. coli* (Thatcher and Gershwin, 1989; Fallon, 1990). The molecular weight of IgM molecules is 900,000 to 1,000,000 d and the molecule is comprised of 5 identical subunits (Stott and Menefee, 1978; Bush and Staley, 1980; Tizard, 1987). There are no known subclasses for IgM, although two types exist; a large polymer that associates with plasma and the monomeric form located on the lymphocyte cell surface (Halliwell and Gorman, 1989). Immunoglobulin M is the major Ig produced in a primary immune response (Tizard, 1987). Mean serum IgM in adult cattle is approximately 2.6 g/L (Fallon, 1990; Penhale and Christie, 1969). Colostral IgM concentrations are significantly lower than IgG concentrations and are approximately 4.2 g/L compared to 50.5 g/L of IgG (Penhale and Christie, 1969; Fallon, 1990; Besser and

Gay, 1994; Quigley et al., 1994).

Immunoglobulin A serves to selectively protect body surfaces from antigen adherence and is found in the highest concentrations in external secretions of nonruminants (Butler et al., 1972; Porter, 1979; Tizard, 1987; Thatcher and Gershwin, 1989; Fallon, 1990). It is the principal Ig in the exocrine glands, gut, and respiratory tract and usually occurs as a dimer (Butler, 1971; Butler et al., 1972; Butler, 1981). The molecular weight of IgA is 160,000 to 170,000 d and the mean serum concentration in the adult bovine is 5.01 g/L (Stott and Menefee, 1978; Halliwell and Gorman, 1989; Fallon, 1990). Penhale and Christie (1969) evaluated differences in serum IgA concentrations attributable to breed and reported a mean IgA range of 2.2 to 3.4 g/L in 5 breeds of cows.

The IgA class of Ig predominates in porcine and equine milk and exerts a local protective effect within the gastrointestinal tract (Bush and Staley, 1980). The surface protection provided by IgA benefits the intestinal lining by guarding against bacterial pathogens. Immunoglobulin A also has the ability to recognize coliform bacteria and other organisms and to activate lysis of these organisms (Bush and Staley, 1980; Fallon, 1990; Saif, 1990). Mammals can be classified into 3 groups regarding IgA concentration in lacteal secretions: 1) IgA predominates both in colostrum and throughout lactation (humans); 2) the predominant Ig of colostrum is IgG, but, during the first few days of lactation, there is a sharp drop in IgG so that IgA becomes predominant (dogs, pigs, and horses); 3) IgA is a minor Ig both in colostrum and throughout lactation (cows, goats and sheep; Vaerman, 1971; Porter, 1979). In human colostrum, IgA accounts for approximately 95% of the 10 to 20 g/L of Ig (Larson et al., 1980). Fallon (1990)

determined a mean IgA concentration in bovine colostrum of 3.9 g/L decreasing to a mean concentration of 0.14 g/L in milk. Kruse (1983) reported a similar decline in IgA from sow colostrum with a mean concentration of 24 g/L decreasing to 3.6 g/L by 3 days postpartum. Other researchers reported colostral IgA concentrations ranging from 0.8 to 1.9 g/L in 5 dairy breeds (Butler et al., 1972; Muller and Ellinger, 1981; Quigley et al., 1994) .

Little is known about the dynamics of bovine IgE. Hammer et al. (1971) first described the presence of IgE, and although much is known about its role in parasitic and allergic disease in other species, the role of IgE in cattle is an enigma. IgE has a molecular weight of 196,000 d and it is unique among Ig in that it is destroyed by heating to 56^{0}C for 30 minutes (Tizard, 1987). The source of IgE in colostrum is unknown and endogenous production in calves begins after 4 wk of age (Thatcher and Gershwin, 1989).

The IgG class has the lowest molecular weight of Ig in cattle. It is approximately 150,000 to 163,000 d (Stott and Menefee, 1978; Bush and Staley, 1980; Halliwell and Gorman, 1989). Immunoglobulin G is the Ig found in highest concentration in blood, serum and colostrum of domestic farm animals (Penhale and Christie, 1969; Butler, 1971; Butler et al., 1972; Porter, 1979; Devery-Pocius and Larson, 1983; Halliwell and Gorman, 1989; Saif, 1990; Besser and Gay, 1994). It is important for general protection against pathogenic organisms (Butler, 1971; Fallon, 1990). As the major class of Ig found in plasma of all species, IgG represents 65 to 90% of the total Ig concentration (Penhale and Christie, 1969; Oyeniyi and Hunter, 1978; Halliwell and Gorman, 1989; Saif, 1990). Its relatively small size suggests it can more easily escape from the

circulation than other Ig (Husband et al., 1972; Tizard, 1987). Mean serum IgG concentration in cattle ranges from 7 to 20 g/L, showing significant seasonal and breed variation (Penhale and Christie, 1969; Kruse, 1970a; Butler, 1971; Fallon, 1990). Serum IgG concentrations in cattle are relatively high compared to those observed in humans, horses and dogs which are as low as 2.5 g/L (Butler, 1971; Tizard, 1987).

The major function of IgG is to promote the removal of microorganisms and to neutralize toxins via antibody-mediated response mechanisms, also referred to as a secondary response (Tizard, 1987; Halliwell and Gorman, 1989). Immunoglobulin G has the ability to bind to toxins and either prevent the toxin from binding to its receptor or to initiate recognition by phagocytic cells such as macrophages or neutrophils (Halliwell and Gorman, 1989).

Ig concentration in colostrum

The transfer of IgG from serum to colostrum begins several weeks prior to calving and continues until parturition (Butler et al., 1972; Larson et al., 1980; Andrews, 1990; Besser and Gay, 1994). The selective, receptor-mediated transfer of IgG_1 from the blood of the dam across the mammary gland secretory epithelium, causes colostral Ig concentration to exceed the blood plasma Ig concentration by 10 to 15 fold (Penhale and Christie, 1969; Butler et al., 1972; Larson et al., 1980; Besser and Gay, 1994). Immunoglobulin G_1 is secreted into colostrum via a membrane bound receptor on the basal side of the secretory epithelium, allowing micropinocytosis to occur (Besser and Gay, 1994). The shorter half-life of IgG_1 compared to IgG_2 supports the concept of selective transport of IgG_1 into secretions because IgG_1 levels are maintained by both

local synthesis and transport from serum (Butler et al., 1972).

Maximum transfer of Ig occurs 1 to 3 d before calving (Fallon, 1990). Multiparous cows have been observed to transfer > 3 kg of Ig to the mammary gland, with IgG contributing 85 to 90% of total Ig (Oyeniyi and Hunter, 1978; Larson et al., 1980). Tizard (1987) reported all IgG, most IgM and approximately half of the IgA in bovine colostrum are derived from the maternal serum, but only 30% of IgG and 10% of IgA in milk are so derived. Oyeniyi and Hunter (1978) also stated that the majority of Ig in colostrum are derived directly from plasma by active transport across the alveolar epithelium. However, Larson et al. (1980) and Butler et al. (1972) have reported that IgA and IgM are synthesized locally in the udder by plasmocytes.

Several factors have been associated with the ability of cows to produce colostrum with sufficient Ig to provide adequate passive transfer to the newborn. Beef breeds generally have higher colostral Ig concentrations than dairy cows (Petrie, 1984; Besser and Gay, 1994). Kruse (1970a) and Oyeniyi and Hunter (1978) determined significant effects of breed and lactation number on colostral Ig concentration in dairy cows. Kruse (1970a) reported that Red Danish cattle achieved significantly higher colostral Ig concentrations compared to Jersey cattle. Besser and Gay (1994) also reported higher colostral IgG_1 concentrations in Jersey cows compared to Holstein cows. Quigley et al. (1994, 1995) reported mean colostral Ig concentration in Jersey cows of 65.8 and 84.6 g/L in samples taken from the same herd over a two year period. These concentrations are higher than concentrations of 29.8 and 35.4 g/L reported in Holstein cows at first milking (Butler et al., 1972; Oyeniyi and Hunter, 1978).

Likewise, animals in their first lactation have lower colostral Ig concentrations than animals in second or later lactations (Kruse, 1970a; Oyeniyi and Hunter, 1978). In contrast, Quigley et al. (1994) and Pritchett et al. (1991) reported lower colostral IgG concentrations in second lactation cows compared to cows in first lactation and older cows. No explanation was given for the discrepancy in varying colostral IgG concentrations in second lactation cows. Logan (1977) reported a decrease in colostral Ig volume in beef cattle subjected to protein or energy deficiencies during late pregnancy. However, there was no correlation between colostral Ig concentration and treatment. Second or later milkings of colostrum contain much lower Ig concentrations than first-milking colostrum, because Ig transport into the mammary gland is essentially complete by the time of parturition (Oyeniyi and Hunter, 1978; Besser and Gay, 1994).

Absorption of colostral Ig

Absorption may be defined as movement of substances from the lumen of the intestine to the blood (Bush and Staley, 1980). The absorption of macromolecules may be delineated into two phases: 1) uptake and internalization within intestinal epithelium; and 2) transport or subsequent expulsion of macromolecules into a compartment (blood; Lecce and Broughton, 1973). Antibodies are proteins with biological activity that is dependant on their surface configuration. The biological activity of absorbed Ig is identical to a colostral Ig of the same identity and therefore, these molecules must be absorbed intact (Brambell, 1958). Colostral Ig are transported through the enterocytes by micropinocytosis and the quantity transported increases from the cranial to the caudal part of the small intestine (Kruse, 1985; Staley and Bush, 1985; Jochims et al., 1994).

Uptake of macromolecules into cells appears to be nonselective; however, not all substances are transferred to the blood (Bush and Staley, 1980; Kruse, 1983). Bush and Staley (1980) observed the uptake of ferritin-Ig by the apical tubular complex of the jejunal cell in calves without transport beyond the apical end of the cell. In the ileal cell, ferritin solutions were transported into the base of the cell but not observed to move out of the cell. In contrast, Lecce and Broughton (1973) reported that in piglets, lambs and calves, all macromolecules are taken up by the gut cell and transported to the blood.

Similar patterns of transport selectivity have been observed in other species (Lecce and Broughton, 1973; Bush and Staley, 1980). Lecce and Broughton (1973) observed different molecules in blood compared to molecules absorbed from the intestines of guinea pigs, hamsters and rabbits. From this evidence, researchers concluded that the intestinal epithelium exerts a degree of selectivity in transfer of proteins to the blood. Loss of absorbed Ig from blood has also been attributed to their entrance into interstitial fluid (Husband et al., 1972; Roy, 1990). Taking into account Ig distribution into the intravascular and extrasvacular pools, Penhale et al. (1973) and Kruse (1970b) postulated that 99% of the IgG, 59% of the IgM, and 48% of the IgA were absorbed from the intestines.

Evidence suggests that a structural change in the absorptive cells of the intestine is initiated by exposure to colostrum. Uniform columnar epithelial cells without vacuoles or inclusions have been reported in newborn unfed pigs (Staley et al., 1969). One hour after pigs were exposed to colostrum, approximately 20% of the cells on the upper two-thirds of the villus contained dense vacuoles. By 3 h after pigs had been given colostrum,

50% of these cells contained colostrum. At 6 h post feeding, 75% of cells on the villus had vacuoles containing colostrum. Similar research in newborn calves showed an increase in enterocytic vacuolation from the cranial to the caudal part of the small intestine (Jochims et al., 1994). Stott et al. (1979b) stated that contact of intestinal epithelial cells with ingested colostrum immediately excites pinocytotic activity, with rapid uptake of available macromolecules and other ingested substances into cells, until a finite amount of pinocytotic activity had occurred.

Kruse (1970b) reported that serum Ig concentration during the first 24 h was a function of mass of Ig fed to calves, age at first colostrum feeding and birth weight of the calf. Mass of Ig consumed and age of first feeding were the two predominant factors. Stott et al. (1979c) determined these two factors were linearly related to Ig concentration. A negative linear relationship existed with age where increased age decreased concentration and a positive linear relationship existed with quantity where increased quantity (up to 2 L) increased concentration. Contrasting the work of Kruse (1970b), Stott et al. (1979c) did not observe an influence of calf body weight on maximum serum Ig concentrations.

Controversy remains regarding the influence of time of first colostrum feeding on maximum serum Ig concentrations. Räjala and Castrén (1995) reported a decrease in total serum Ig concentration at 24 h associated with delayed feeding. Their results indicated that with each 30 min delay in the intake of colostrum, serum Ig was decreased by 2 g/L. However, Michanek et al. (1989) reported no difference in transmission of IgG from the first feeding in calves fed at 1, 8, 16, and 24 h. Although, in the second, third

and fourth feedings, calves that received their first colostrum at one hour of age transmitted significantly more marker molecules (dextran, ovalbumin, and albumin) than did calves in other groups. Todd and Whyte (1995) observed no significant differences in mean serum Ig concentrations between calves fed at different times after birth (2, 4, 6, and 8 h).

Constituents of colostrum, such as trypsin inhibitor, may exert a protective effect on colostral components, protecting these components from proteolytic degradation. Quigley et al. (1995) reported that trypsin inhibitor in colostrum was positively related to concentration of IgG in colostrum and further stated that the effect of trypsin inhibitor was probably related to its ability to provide protection to immune components of colostrum from proteolytic degradation. High concentrations of trypsin inhibitor may exert a protective effect on colostral Ig by protecting them from degradation and enhancing absorption when feeding is delayed. Other researchers confirm the effect of trypsin inhibitor by stating that it has a positive influence on the acquisition of passive immunity in newborn piglets (Oyeniyi and Hunter, 1978; Kruse, 1983; Besser and Gay, 1994).

Closure

Lecce and Morgan (1962) defined closure as 'the cessation of the uptake or internalization of macromolecules via pinocytosis into intestinal epithelium'. Other researchers (Jochims et al., 1994) state that closure may be defined as 'a multifactorial event comprising the replacement of the fetal enterocytes, which are able to absorb macromolecules, by a more mature population, a retrograde cessation of transfer and

increasing intracellular proteolytic activity by lysosomes'. Lecce and Broughton (1973) classified mammals into three groups with respect to time of closure: 1) short duration, less than 36 h of age; 2) medium duration, approximately 6 d of age; and 3) long duration, approximately 17 to 40 d of age.

The normal period during which absorption of macromolecules may occur in calves and pigs through the intestinal wall is 24 to 36 h (Deutsch and Smith, 1957). Age at first feeding may affect the amount of time in which the intestines can absorb macromolecules. Stott et al. (1979a) determined that age at first colostrum feeding influenced mean closure time. As feeding of colostrum was delayed, estimated time for closure was also delayed. In calves fed at birth (0 h), closure occurred at approximately 21 h for IgG and 23 h for IgM and IgA (Stott et al., 1979a). However, when feeding was delayed until 24 h after birth, closure occurred at 33, 31, and 32 h for IgG, IgM and IgA, respectively (Stott et al., 1979a). The actual length of time the calf absorbed Ig was reduced from approximately 21 to 9 h. In a similar study using piglets and lambs, Lecce and Morgan (1962) determined that the ability of piglets and lambs to absorb large molecules was not dependant upon the age of the animal, but on whether the animal was fed and amount of feed it consumed. Their work reported that nursing piglets lost the ability to absorb a high molecular weight, nonprotein test molecule (polyvinylpyrrolidone) when approximately 24 to 36 h old. When nursing was mimicked by feeding 300 - 400 ml of bovine colostrum, closure occurred at 24 h. In the same study, starved piglets were able to absorb the test molecule when 86 h old, suggesting that feeding colostrum may shorten the period from birth to closure.

The effect of early colostrum feeding on closure may be associated with the amount of Ig presented to the intestines. Michanek et al. (1989) suggested that early ingestion of colostrum will trigger intestinal closure only if the amount of Ig is large. When calves were fed colostrum at 0 h, closure did not begin until after a second feeding was presented to the animal. On the other hand, closure appeared to begin after the first feeding in calves that received their first feeding of 1 or 2 L of colostrum at 8, 16, and 24 h. Stott and Fellah (1983) reported a linear relationship between the concentration of Ig in colostrum and the rate at which Ig were absorbed. The higher the concentration of Ig fed, the more rapidly the calf absorbed Ig immediately after being fed. A possible explanation for the conflicting results of Michanek et al. (1989) is that calves in this study were fed colostrum at 3% of body weight. Depending on colostral Ig concentration, the amount fed may have supplied calves with considerably less Ig than recommended. Other researchers (Kruse, 1970c; Gay, 1983; Stott and Fellah, 1983; Besser et al., 1991) have suggested feeding neonates approximately 100 g of Ig from colostrum in the first feeding to prevent FPT.

Blood gas parameters associated with respiratory functions may also influence mean closure time. Tyler and Ramsey (1991a) determined that when colostrum was fed at 0 and 12 h, time of closure was significantly delayed beyond 20 h in calves receiving a gas mixture of 21% O_2 when compared to calves receiving a gas mixture of 10.5% O_2. Calves in the 21% O_2 group exhibited a mean closure time of 40.5 h. Tyler and Ramsey postulated that postnatal acidosis may extend the period of time during which colostral IgG may be absorbed from the intestine, and not decrease the amount of IgG that is

absorbed. Besser et al. (1990) also reported a significant decrease in colostral Ig absorption associated with respiratory acidosis. However, the sampling protocol tested for IgG_1 concentrations 12 h after colostrum feeding and it is likely that closure had not yet occurred. Previous research states a mean closure time of 24 to 36 h for calves and pigs (Deutsch and Smith, 1957; Lecce and Morgan, 1962; Stott et al., 1979a). Analysis of IgG_1 prior to 24 to 36 h may not represent peak serum concentrations.

Apparent efficiency of absorption

Apparent efficiency of Ig absorption (AEA) is defined as the ratio of mass of circulating Ig to quantity of Ig consumed in colostrum (Husband et al., 1972). Mass of Ig in plasma is the product of peak concentration of Ig in plasma and plasma volume. The quantity consumed is the product of Ig concentration of colostrum and amount of colostrum consumed. Much research conducted in the area of colostral absorption considers Ig concentration at a point in the neonate's life instead of calculating an actual AEA. Furthermore, researchers calculating AEA often estimate plasma volume at 7% of body weight when determining amount of Ig in plasma (McEwan et al., 1970). Either consideration of Ig concentration without regard to mass of Ig consumed or the estimation of plasma volume introduce error into determination of newborn's immunological status and ability to absorb Ig. Therefore, calculating AEA is a more accurate indicator of the neonate's absorptive ability.

Several factors may influence the AEA of newborn calves. Besser et al. (1985) found a negative correlation between the AEA and the mass of IgG_1 and IgM fed. Both IgG_1 and IgM appeared to be absorbed with greater efficiency when smaller amounts

were consumed. An increase in the percent of IgM absorbed as the amount of IgM ingested decreased was also observed by Stott and Menefee (1978). However, Stott and Menefee (1978) showed that the AEA of both IgG and IgA did not change as intake varied. Earlier work (Brandon and Lascelles, 1971) suggested no significant differences in the relative AEA for IgG_1, IgG_2, IgM and IgA. If differences exist, possible explanations accounting for the decreased AEA could include the presence of a shared macromolecular transport mechanism or a regulation of serum Ig concentration not exceeding a threshold level (Besser et al., 1985).

Physiological challenges to which the neonate is exposed to may also contribute to decreased AEA. Recent research on the effect of acid-base balance and blood gas parameters on absorption of colostral Ig suggests decreased absorption of colostral Ig associated with acidosis (Eigenmann et al., 1983; Szenci, 1985b; Boyd, 1989; Besser et al., 1990; Guy et al., 1996). Researchers suggest that metabolic acidosis resolves itself within the first 4 h of life and the respiratory portion prevails (Boyd, 1989; Besser et al., 1990). Because respiratory acidosis may be prevalent throughout the colostral absorptive period, attention has been placed on the importance of understanding the effect of respiratory acidosis on colostral Ig absorption. However, many researchers analyzed single blood samples for determination of plasma or serum Ig concentrations as indicators of the animal's ability to absorb colostral Ig and did not calculate AEA. Calculating the AEA is a more accurate indicator of the animal's actual physiological ability to absorb Ig molecules and therefore a more accurate indicator of the effect of respiratory acidosis on colostral Ig absorption. Furthermore, researchers have utilized venous blood for

determination of blood gas analysis. Venous blood is not an accurate indication of respiratory function or blood gas values.

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3. MATERIALS AND METHODS

Animal assignments and experimental design.

Forty-eight Holstein calves (25 heifers and 23 bulls) from the University of Tennessee Agricultural Experiment Station were assigned at birth to one of three treatment groups based on $PaCO_2$ at 1 h after birth in a completely randomized design. Treatments were: 1) non-acidotic (respiratory; **NA**), calves born with $PaCO_2 < 50$ mm of Hg; 2) acidotic - non ventilated (**ANV**), calves born with $PaCO_2 \geq 50$ mm of Hg with no intervention; and 3) acidotic - ventilated (**AV**), calves born with $PaCO_2 \geq 50$ mm of Hg and ventilated with a self-inflating resuscitation bag (Ambu™). Artificial respiration was given to calves in the **AV** group via Ambu™, with a breath occurring every 5 sec for a period of 5 min.

Pregnant cows were housed on a drylot and were observed for indications of parturition. Dry cow diet consisted of a mixed grass hay and commercial dry cow concentrate. Estimated dietary cation:anion difference $[(Na^+ + K^+) - (Cl^- + S^-)]$ for dry cow diets was approximately +65 mEq/100 g DM. Cows were closely monitored at the onset of parturition for signs of difficult birth and assistance was provided if needed. Births were scored on a scale of 1 to 4: 1) normal birth, no assistance required (n = 22); 2) requiring assistance - easy pull (n = 15); 3) requiring assistance - difficult pull (n = 11); and 4) cesarean section (n = 0). Newborn calves remained with the dam for a short period of time for drying and stimulation, however, calves were not allowed to nurse. Within 30 min of birth, navels of calves were dipped with a 7% iodine solution and

calves were given rotavirus/coronavirus vaccine orally (SmithKline Beecham, West Chester, PA). Calves were then moved to the calf facility where they were weighed, rectal temperatures were recorded, calves were identified, and placed in individual stalls (2.5 X 1.5 m) bedded with shavings. Stalls were cleaned regularly and completely replaced between every 3 to 4 calves.

Feeding Management and Colostrum Analysis

First and second milking colostrum was collected by staff at the University of Tennessee Agricultural Experiment Station and pooled. Pooled colostrum was stored at -20°C, gradually thawed in warm water and heated to approximately 40°C before feeding. During periods of intense calving, when colostrum demand was high, pooled colostrum was stored at 4°C and warmed to 40°C prior to feeding. Calves received 2 L colostrum at 2 h ± 5 min after birth from a nipple bottle. Due to a shortage of colostrum, one animal received 1.8 L at 2 h. There was no difference in IgG intake between the three treatment groups ($P > 0.4$). Ten calves were fed < 2 L of colostrum, with 2 calves receiving 1.3 L. Calves not drinking the entire 2 L were tubed with an esophageal tube. Calves were also fed pooled colostrum 12, 24, and 36 h after the first feeding. Due to limited colostrum availability, colostrum pools fed at 12, 24 and 36 h differed. Amount of intake and method of feeding were accurately recorded at each feeding time.

A 10-ml sample of all pooled colostrum was stored at -20°C prior to analysis for IgG by single radial immunodiffusion (VMRD, Inc., Pullman, WA) by method of Fahey and McKelvey (1965). Samples were thawed in a 37°C water bath and diluted 5:1 with deionized water. A 3 μL sample was placed into a well containing bovine IgG antisera in

buffered agarose. After 22 h of incubation at room temperature, precipitation rings were measured using an ocular micrometer on a stereomicroscope (American Optical, Buffalo, NY).

Arterial Blood Sampling and Analysis

Arterial blood was collected from the brachial artery at 1, 13, and 25 h for blood gas analysis. Animals were placed in lateral recumbency and blood was drawn into 3 ml heparinized (1000 IU/ml) syringes by method of Adams et al. (1991). Immediately after blood was drawn, excess air was expelled and the syringes were sealed to exclude entry of O_2 or loss of gases. Samples were placed on ice and analyzed for pHa, $PaCO_2$, PaO_2, bicarbonate concentration (HCO_3^-), base excess (BE), and total CO_2, (TCO_2) within 30 min of sampling. Samples were analyzed using a Blood Gas Manager 1312 (Instrumental Laboratory, Lexington, MA). Correction equations were used to calculate measured parameters for pHa, $PaCO_2$ and PaO_2 at body temperatures other than 37^0 C.

Venous Blood Sampling and Analysis

At 1, 13, and 25 h, venous blood (5 ml) was collected by jugular venipuncture into evacuated containers without anticoagulant. Heparinized micro-hematocrit capillary tubes (n = 2; Fisher Scientific Corp., Pittsburg, PA) were filled, sealed with rubber and centrifuged immediately for hematocrit determination. From the remaining sample, 2 ml of blood were placed into a 15 ml centrifuge tube containing 4 ml of perchloric acid (8%) for analysis of lactate. At 1, 13, and 25 h a second venous sample (10 ml) was collected by jugular venipuncture into evacuated containers containing ethylenediaminotetra-acetate (EDTA) for analysis of plasma cortisol and plasma IgG. At 37 and 49 h, calves

were sampled by jugular venipuncture into evacuated containers containing EDTA for analysis of plasma IgG. Upon collection, evacuated containers and centrifuge tubes were inverted and placed on ice until centrifugation. Samples were centrifuged (3000 x *g*) and supernatant liquid was pipetted into 5 ml culture tubes and stored at -20°C for later analysis.

Concentration of lactate (L+) was determined using a spectrophotometer (Sequoia-Turner Corp. Mountain View, CA) at 340 nm measuring the formation of pyruvate from lactate with excess nicotinamide adenine dinucleotide (NAD; Sigma Diagnostics, St. Louis, MO). Total plasma cortisol was measured by solid-phase radioimmunoassay (Coat-A-Count, Diagnostic Products Corp. Los Angeles, CA). Plasma IgG was measured by single radial immunodiffusion, as described for colostrum, but without dilution.

Plasma volume was measured at 25 h using Evans' Blue dye as described by Dalton and Fisher (1961). Initial samples of venous blood were collected into evacuated containers containing EDTA (as described for plasma cortisol and IgG determination). Calves were injected by jugular venipuncture with a preweighed amount of dye. The dye was allowed to equilibrate for 10 min; then a second sample was collected into an evacuated container containing EDTA. Samples were placed on ice until centrifugation. Plasma volume was determined by reading absorbance of standard and unknown with a spectrophotometer (Sequoia-Turner Corp. Mountain View, CA) at 620 nm.

Apparent Efficiency of Absorption (AEA)

Apparent efficiency of absorption (AEA) was determined at 25 h as calculated by the method of Husband et al. (1973) using the formula:

$$AEA = \frac{\text{plasma IgG (g/L) X plasma volume (L)}}{\text{Colostral IgG (g/L) X Colostrum intake (L)}}$$

Statistical Analysis

Blood gas parameters, plasma lactate, and total plasma cortisol at 1 h were analyzed as a completely randomized experimental design using the model: $Y_{ij} = \mu + T_i + e_{ij}$ where μ = overall mean; Y_{ij} = blood gas, lactate or cortisol at 1 h; T_i = effect of i^{th} treatment; e = random error associated with the i^{th} treatment and the j^{th} calf (i = 1,2,3; j = 1,2,..16).

Plasma IgG concentrations at 1, 13, 25, 37, and 49 h and AEA at 25 h were analyzed as a completely randomized experimental design with IgG intake as a covariate using the model : $Y_{ij} = \mu + T_i + \beta (X_{ij} - \bar{X}) + e_{ij}$ where μ = overall mean; Y_{ij} = plasma IgG (13, 25, 37, and 49 h) or AEA at 25 h; T_i = effect of i^{th} treatment; $\beta (X_{ij} - \bar{X})$ = adjustment for the effect of the covariate; e_{ij} = random error associated with the i^{th} treatment and the j^{th} calf (i = 1,2,3; j = 1,2,..16). General Linear Model procedures were used in the analysis of variance and to generate least squares means and standard errors for blood gas parameters, plasma IgG, and AEA.

Regression analysis was used to determine relationships among blood gas parameters, plasma IgG concentrations and AEA. All possible combinations of variables were analyzed to determine variables most highly associated with plasma IgG at 25 h and

AEA. Models of best fit were determined and 1 h $PaCO_2$ was added to that model to determine its effect on plasma IgG and AEA. Declaration of significance was $P < 0.05$ unless otherwise noted.

4. RESULTS AND DISCUSSION

Introduction

No calves died during the study and general health and appearance of the animals was good. However, due to complications with the Blood Gas Manager 1312 (BGM 1312; Instrumental Laboratories, Lexington, MA), one sample was analyzed on an IRMA Blood Gas Analyzer (Diametrics Medical, Inc., St. Paul, MN). The sample analyzed for $PaCO_2$ on the IRMA Blood Gas Analyzer was considerably lower than samples analyzed on the BGM 1312. Furthermore, simultaneous analysis of identical samples indicated variation of 4 to 7 mm of Hg between the two machines. This sample was discarded and all blood gas values are representative of samples on 47 calves and least squares means are reported.

Blood Gases

Mean $PaCO_2$ for all calves at 1 h was 50.80 mm of Hg (SE = 0.67) and decreased to 45.74 (SE = 0.44) and 45.44 (SE = 0.46) at 13 and 25 h, respectively (Table 1). These values are within the normal expected range for neonatal calves at birth (Moore, 1969; Garry, 1993; Guy et al., 1996). The decrease in $PaCO_2$ over time indicated that calves compensated for increased $PaCO_2$ within the first 12 to 24 h after birth without intervention as reported by Walser and Maurer-Schweizer (1979) and Garry (1993). The range in $PaCO_2$ at 1 h was 41.2 to 59.8 mm of Hg and by 25 h had decreased to 38.4 to 51.4 mm of Hg, indicating that the acid-base balance of calves stabilized as calves approached homeostasis.

Mean arterial pH at 1 h of 7.303 (SE = 0.008) indicated that calves were born

Table 1. Descriptive statistics of pHa, $PaCO_2$, PaO_2, HCO_3^-, and base excess.

Parameter[1,2]	n	Min	Max	Mean	SE
$PaCO_2$, mm of Hg					
1 h	47	41.20	59.80	50.80	0.67
13 h	47	38.50	52.30	45.74	0.44
25 h	46	38.40	51.40	45.44	0.46
pHa					
1 h	47	7.113	7.400	7.303	0.008
13 h	47	7.300	7.440	7.386	0.004
25 h	46	7.346	7.500	7.425	0.005
PaO_2, mm of Hg					
1 h	47	31.0	112.0	69.1	2.8
13 h	47	43.0	123.0	80.6	2.7
25 h	46	39.0	118.0	72.2	2.8
HCO_3^-, mmol/L					
1 h	47	13.60	29.60	24.72	0.45
13 h	47	20.70	31.50	27.30	0.32
25 h	46	23.20	33.40	29.66	0.35
Base Excess (BE), mEq/L					
1 h	47	-13.90	4.40	-0.98	0.52
13 h	47	-3.90	7.40	2.85	0.33
25 h	46	-1.40	9.60	5.74	0.36

[1]All blood gas values were taken via arterial sample and analyzed within 30 min of sampling.
[2]All blood gas values were corrected to body temperature.

slightly acidotic (Table 1). Acidosis of calves in this study supports other research indicating that calves exhibit a partially compensated acidosis at birth (Walser and Maurer-Schweizer, 1979; Szenci, 1985b; Boyd, 1989; Besser et al., 1990; Kasari, 1994). At 13 h, mean pHa had increased to 7.386 (SE = 0.004) and by 25 h mean pHa was slightly over optimal (7.4) at 7.425 (SE = 0.005). The pHa at 1 h ranged from 7.113 to 7.400 (Table 1). Four calves had pHa levels below 7.25 at birth, which is considered moderately to severely acidotic (Boyd, 1989). Three of these four calves achieved pHa levels > 7.40 by 25 h. The remaining animal achieved a pHa of 7.373 by 25 h.

Mean PaO_2 at 1 h was 69.1 (SE = 2.8) mm of Hg (Table 1) and was below the ideal for adult mammals (90 to 100 mm of Hg; Kirk, 1983). The PaO_2 increased to 80.6 (SE = 2.7) at 13 h and decreased to 72.2 (SE = 2.8) at 25 h. This observed variation was probably due to the initiation of spontaneous respiration and adjustments in fetal lungs during the early postpartum period (Mortolla, 1987). An increase in PaO_2 is consistent with findings of Adams et al. (1993) and Stewart et al. (1984) in calves and foals. Stewart et al. (1984) reported a gradual increase in PaO_2 in foals during the first week of life. These foals did not reach adult concentrations (86.9 mm of Hg) until approximately 1 wk of age. Range in PaO_2 for all three sampling periods in the present study was large; 31 to 112 mm of Hg at 1 h after birth, 43 to 123 mm of Hg at 13 h, and 39 to 118 mm of Hg at 25 h. Large variations may be due to the time animals were in lateral recumbency and struggling during the sampling period (Wagner et al., 1990; Smith, 1996). Wagner et al. (1990) reported a significant decrease in PaO_2 in animals placed in either left or right lateral recumbency for 5 min. However, pHa, $PaCO_2$, HCO_3^- concentration, and base

excess were unaffected. Excited animals were difficult to sample and consequently, were in lateral recumbency for greater than 5 min.

Mean HCO_3^- concentration at 1 h was 24.72 mmol/L (SE = 0.45) increasing to 27.30 mmol/L (SE = 0.32) by 13 h (Table 1). Mean HCO_3^- concentration at 1 h appeared normal for mammals (24 ± 3 mmol/L; Kirk, 1983; Smith, 1996) and indicated calves were not in a state of metabolic acidosis. At 25 h, mean HCO_3^- concentration had reached 29.66 (SE = 0.35) mmol/L and was slightly higher than expected levels observed in adult mammals. Increased HCO_3^- concentration suggests either a partial metabolic alkalosis or compensation for an acidosis of respiratory origin (Haskins, 1977a; Blood and Radostits, 1989).

Mean base excess (BE) at 1 h was -0.98 mmol/L (SE = 0.52) increasing to 5.74 (SE = 0.36) by 25 h. Base excess greater than +4 expresses metabolic alkalosis while base excess less than -4 expresses a metabolic acidosis in adult mammals (Haskins, 1977a; Smith, 1996). Considering these values, BE at 1 h would be normal. Base excess at 25 h, however, is consistent with increasing HCO_3^- concentrations suggesting a partially compensated metabolic alkalosis (Table 1). However, some researchers (Walser and Maurer-Schweizer, 1979; Garry, 1993; Kasari, 1994) have reported values similar to our study in newborn calves for both HCO_3^- concentration and base excess.

Body Weight, Immunoglobulin Concentration, Plasma Volume, and Selected Metabolites

Body weights (BW) of calves ranged from 22.3 to 50.0 kg (Table 2). The 22.3 kg calf was twin to a 33.6 kg calf and was 8 kg lighter than the second lightest calf. Mean BW was 39.78 kg (SE = 0.69). Calving ease was scored on a scale of 1 to 4 with a mean score of 1.79 (SE = 0.12; Table 2). A score of 1 indicated a normal, unassisted birth, 2 indicated an easy pull, and 3 indicated a difficult pull. No calves were scored 4, indicating no Cesarean sections were performed. Eleven of 48 births were considered difficult pulls and all were births by primiparous dams. Parity of dam ranged from 1 to 5 with a mean parity of 2.08 (SE = 0.21; Table 2).

Mean plasma IgG concentration at 13 h was 8.31 (SE = 0.50) g/L and ranged from 3.66 to 16.51 g/L (Table 2). Mean plasma IgG concentration increased to 11.73 g/L (SE = 0.66) at 25 h and ranged from 3.82 to 22.00 g/L for 48 calves (Table 2). The increase in plasma IgG concentration from 13 to 25 h indicated macromolecules were absorbed after the second feeding at 13 h. This mean plasma IgG concentration at 25 h indicated that calves achieved adequate transfer of colostral Ig after the first 2 feedings. Failure of passive transfer is implicated with plasma IgG concentrations < 10 g/L (Gay, 1983). Seventeen calves (35%) had plasma IgG concentrations < 10 g/L at 25 h indicating FPT. However, no significant morbidity was observed during the 48 h experimental period. Health status of calves was not followed beyond 48 h and calves with lower plasma IgG concentrations may have been at a higher risk for developing disease during the preweaning period compared to calves with higher plasma IgG concentrations. Mean

Table 2. Descriptive statistics of calving data and venous blood parameters.

	N	Min	Max	Mean	SE
BW, kg	48	22.30	50.00	39.78	0.70
Calving ease[a]	48	1.00	3.00	1.79	0.12
Parity	48	1.00	5.00	2.08	0.21
Plasma IgG (g/L)					
1 h	48	1.19	2.67	1.40	0.03
13 h	48	3.66	16.51	8.31	0.50
25 h	48	3.82	22.00	11.73	0.66
37 h	48	3.61	22.82	12.90	0.74
49 h	48	3.31	22.80	11.75	0.72
Hematocrit, %					
1 h	47	27.5	54.0	36.9	0.8
13 h	47	21.0	50.5	32.9	0.8
25 h	48	22.5	49.0	32.1	0.8
Plasma IgG (g)[b]	43	12.22	96.80	41.55	2.90
Plasma volume, L[b]	43	2.277	5.752	3.422	0.001
Plasma volume, % BW[b]	43	5.70	13.80	8.49	0.33
Colostral IgG, g/L[c]	65	12.10	65.95	42.37	1.93
IgG intake, g[c]	48	65.7	247.4	165.0	8.2
AEA, %[d]	43	9.8	47.6	26.0	1.5
Lactate, mmol/L					
1 h	48	1.42	12.32	4.93	0.39
13 h	48	0.88	7.52	3.55	0.19
25 h	48	1.03	7.49	2.67	0.16
Cortisol, ng/ml					
1 h	48	104.2	232.2	151.9	4.6
13 h	48	38.5	143.8	77.7	3.6
25 h	48	21.8	126.6	65.5	3.1

[a] Scored as 1 = normal birth; 2 = easy pull; 3 = difficult pull; 4 = Cesarean section.
[b] Values calculated at 25 h.
[c] Colostrum for 1st and 2nd feeding included.
[d] Calculated as mass of IgG in plasma / mass of IgG consumed.

plasma IgG concentrations at 25 h were similar to data from Stott et al. (1979c) reporting maximum serum IgG concentrations of 14.9 g/L in calves fed 2 L of colostrum at birth. Besser et al. (1985) also observed a similar range in serum IgG of 9.1 to 22.2 g/L in calves fed within 3 h of birth. Other work at this location (Abel and Quigley, 1993) reported mean serum IgG_1 concentrations of 18.7 g/L (SE = 1.92) at 24 h. These values are higher compared to values observed in the present study. However, Abel and Quigley (1993) fed calves a mixture of fresh and frozen colostrum. Feeding fresh colostrum has been demonstrated to produce higher serum Ig concentrations compared to feeding frozen colostrum (Petrie, 1984).

Mean plasma IgG concentration at 37 h increased to 12.90 (SE = 0.74) g/L and ranged from 3.61 to 22.82 g/L (Table 2) indicating IgG was absorbed between 25 and 37 h. The increase in plasma IgG during this time is consistent with work by Tyler and Ramsey (1991a) who observed an increase in plasma IgG levels from 24 to 42 h. However, the increase was marginal indicating that the intestine had lost some of its permeability during the first 24 to 36 h of life or movement of plasma IgG between intra and extravascular pools (Deutsch and Smith, 1957; Kruse, 1970b). At 49 h, mean plasma IgG decreased to 11.75 (SE = 0.72) g/L with a range from 3.31 to 22.80 g/L (Table 2), indicating IgG was not absorbed after colostrum feeding at 37 h. This is consistent with research by Abel and Quigley (1993) who reported a decrease in mean serum IgG_1 concentrations from 18.7 to 14.2 g/L at 24 and 48 h, respectively, in calves fed maternal colostrum.

Mean hematocrit at 1 h was 36.9% (SE = 0.8) ranging from 27.5 to 54.0%

(Table 2). Mean hematocrit decreased to 32.9% (SE = 0.8) at 13 h and 32.1% (SE = 0.8) at 25 h. Decreased hematocrit in the early postpartum period is associated with the expansion of plasma volume caused by the ingestion of colostrum (McEwan et al., 1968).

Mean plasma volume (PV) for 43 calves at 25 h was 3.422 L (SE = 0.001) with a range from 2.277 to 5.752 L (Table 2). Five calves were excluded from PV, plasma IgG (g), and AEA calculations due to sampling errors associated with the PV technique (Dalton and Fisher, 1961). Expressed as a percent of BW, PV was 8.49 (SE = 0.33), which is higher than values of 6.5% of BW reported in 1 d old calves by Möllerberg et al. (1975) or 6.3% of BW reported in calves 12 h after feeding (Husband et al., 1973). Values from the present study agreed with those from McEwan et al. (1968) ranging from 5.6 to 12,9% of BW. Other researchers have used PV estimates of 5 and 7% of BW when calculating AEA (Kruse, 1970b; McEwan et al., 1970; Besser et al. 1985; Cruywagen, 1990; Morin et al., 1997). Mean blood volume was estimated by the method of Cronin (1954) and Dalton and Fisher (1961) using the formula:

$$\text{Blood volume} = \frac{\text{Plasma Volume X 100}}{\text{100 - (Hematocrit X 0.96)}}$$

Estimated blood volume was 12.4% of BW. This is higher than estimates by Möllerberg et al. (1975) of 9.3% of BW in 1 d old calves. Roy (1990) also indicated blood volume ranges in young calves of 7.4 to 10.6% of BW.

Grams of IgG in plasma at 25 h were calculated as the product of plasma IgG concentration (g/L) at 25 h and plasma volume (L) at 25 h. Plasma IgG (g) at 25 h ranged from 12.22 to 96.80 g with a mean of 41.55 g (SE = 2.90; Table 2). Seventeen calves had

plasma IgG concentrations < 10 g/L at 25 h indicating FPT. Assuming a 26% efficiency of absorption and a mean PV of 3.422 L, the grams of IgG required in colostrum to achieve a 25 h plasma IgG concentration of ≥ 10 g/L can be predicted (McEwan et al., 1970):

$$\text{plasma IgG (g/L)} = \frac{\text{IgG intake} * \text{apparent efficiency of absorption}}{\text{Plasma volume}}$$

Where: plasma IgG (g/L) = 10
IgG intake = X
Apparent efficiency of absorption = 26%
Plasma volume = 3.4 L

Solving for X determines that 130 g of IgG must be ingested from maternal colostrum before closure to prevent FPT. Ten of the seventeen calves with 25 h plasma IgG concentrations < 10 g/L had ingested < 130 g of IgG from colostrum during the first 2 feedings.

Mean IgG concentration in colostrum was 42.37 g/L (SE = 1.93;Table 2) and would be considered moderate according to Fleenor and Stott (1980). Concentration of IgG in colostrum ranged from 12.10 to 65.95 g/L. Due to periods of intense calving which created a shortage of colostrum pools, some calves were fed from different colostrum pools on the first and second feedings. This made it necessary to utilize > 48 samples. Pooled colostrum was derived from the first or second milking after calving. Most colostrum was from multiparous cows and should have had an adequate IgG concentration (Kruse, 1970a; Foley and Otterby, 1978; Pritchett et al., 1991). Grams of IgG intake were calculated as the product of IgG concentration in colostrum (g/L) and the amount of colostrum consumed by calves. Mean intake of IgG for 48 calves was 165.0 g

(SE = 8.2) and ranged from 65.7 to 247.4 g (Table 2) indicating a large variation in colostral IgG concentration and intake.

Mean apparent efficiency of IgG absorption (AEA) for 43 calves was 26.0% (SE = 1.5) as calculated by Husband et al. (1973) by dividing the mass of circulating IgG at 25 h (g) by the quantity of IgG intake from the first 2 colostrum feedings (Table 2). A minimum AEA of 9.8% and a maximum AEA of 47.6% were determined using the same calculation. Besser et al. (1985) observed an AEA range for IgG_1 of 22.8% to 49.5% in calves fed 2.84 L of colostrum by 3 h of age. Similar research by Kruse (1970b) and McEwan et al., (1970) reported mean absorption efficiencies of 20 and 25% in calves, respectively.

Mean lactate at 1 h was 4.93 mmol/L (SE = 0.39) and decreased at 25 h to 2.67 mmol/L (SE =0.19; Table 2). Lactate concentrations were higher than the range recommended for bovines of 0.56 to 2.22 mmol/L (Kaneko, 1989). Mean lactate concentrations in this study were also slightly higher than values observed in other research. Kasari (1994) reported that lactate concentrations reaching 4.4 mmol/L were often present in newborn calves and were indicative of lactic acidemia. Randall (1978) also stated that high lactic acid concentrations caused by low glycogen stores resulting in anaerobic glycolysis during parturition are common in the newborn. As respiration reaches homeostasis and glucose is provided from colostrum, anaerobic glycolysis ceases and lactate levels decrease. Tyler and Ramsey (1991b) observed lactate concentrations > 6 mmol/L in hypoxic calves. The authors concluded that these elevated lactate concentrations were due to anaerobic metabolism exceeding the capacity of the neonate

to utilize lactate as an energy source. Likewise, increased lactate concentrations in the present study may represent lactate production in excess of the calf's ability to utilize it. High lactate values contribute to a progressive metabolic acidosis (Tyler and Ramsey, 1991b). Lactic acidosis in calves < 8 days of age is common (Naylor, 1987). Lactate concentrations in this study suggest an acidosis that is of metabolic origin.

Venous total cortisol concentrations at 1 h on 48 calves ranged from 104.2 to 232.2 ng/ml with a mean concentration of 151.9 ng/ml (SE = 4.6; Table 2). Cortisol decreased over time and mean concentrations at 13 and 25 h were 77.7 and 65.5 ng/ml, respectively. The 1 h mean was similar to results of Johnston and Oxender (1979) who reported a mean cortisol concentration at 0 h after birth > of 140 ng/ml. However, cortisol concentrations in the present study were higher at 13 and 25 h compared to mean values of approximately 50 and 43 ng/ml at 12 and 24 h, respectively (Johnston and Oxender, 1979). Plasma cortisol levels are elevated in the calf at birth as a consequence of the initiation of parturition (Massip, 1980) or possibly due to stress associated with obtaining arterial samples. Elevated cortisol levels in calves with acidosis is thought to be a reaction of the adrenal gland by secreting steroids in response to stress (Massip, 1980).

Treatment Interactions with Blood Gas Parameters

Lease squares means are reported due to missing values in calculations for AEA and loss of one blood gas analysis due to the use of a different blood gas analyzer. Treatments were assigned based on $PaCO_2$ at 1 h, and $PaCO_2$ at 1 h was lower for the **NA** group compared to **ANV** and **AV** groups ($P < 0.001$; Table 3). The PaO_2 also tended to

Table 3. Least squares means for 1 h blood gases, cortisol, lactate, and IgG.

Item	Treatment[1] NA	ANV	AV	SE	Contrasts[2] 1	2
$PaCO_2$, mm of Hg[3]	45.52	53.04	53.22	0.71	***	NS[4]
PaO_2, mm of Hg[3]	75.11	66.94	63.25	4.91	*	NS
pHa[3]	7.309	7.293	7.308	0.010	NS	NS
HCO_3^-, mmol/L[3]	22.91	25.37	26.50	0.71	***	NS
Base Excess[3], mEq/L	-2.23	-0.75	0.58	0.96	**	NS
Cortisol, ng/ml[3]	154.6	149.5	147.4	8.3	NS	NS
Lactate, mmol/L[3]	5.673	4.318	4.057	0.610	**	NS
IgG Intake, (g)[5]	174.5	166.6	145.6	14.9	NS	NS
Plasma IgG, g/L						
13 h	7.92	8.71	8.62	0.82	NS	NS
25 h	10.67	12.56	13.21	0.92	NS	NS
37 h	11.74	13.69	13.56	0.90	NS	NS
AEA, %[5]	24.1	26.1	30.3	03.0	NS	NS

[1]Treatments were: NA = non respiratory acidosis, ANV = acidotic-not ventilated, and AV = acidotic-ventilated.

[2]Contrasts: 1 = NA vs (AV + ANV), 2 = AV vs ANV.

[3]Measured at 1 h.

[4]$P > 0.10$.

[5]Sum of first two feedings.

* $P < 0.09$.
** $P < 0.05$.
*** $P < 0.001$.

be different by treatment ($P < 0.09$; Table 3). However, pHa was unaffected by treatment (Table 3), indicating factors other than $PaCO_2$ and PaO_2 at 1 h were contributing to the depression observed in pHa.

Treatment had a significant effect on 1 h HCO_3^- concentration ($P < 0.001$; Table 3). Both **ANV** and **AV** had significantly higher HCO_3^- concentrations compared to **NA** ($P < 0.001$). Increased $PaCO_2$ increased HCO_3^- concentrations through carbonic acid (H_2CO_3) and the equilibrium $CO_2 + H_2O$ and HCO_3 (Filley, 1971; Haskins, 1977a; Kaneko, 1989): $CO_2 + H_2O \rightleftarrows H_2CO_3 \rightleftarrows H^+ + HCO_3^-$. An increase in 1 h $PaCO_2$ drives the equation to the right and calves with increased $PaCO_2$ exhibited higher HCO_3^- concentrations compared to calves with a lower $PaCO_2$. Expected mean HCO_3^- concentration for calves at 1 h is 23.52 mmol/L (Kirk, 1983; Kaneko, 1989; Smith, 1996). Means for all three groups were near expected concentrations in calves at 1 h. Lower HCO_3^- concentrations and most negative base excess (BE) observed in the **NA** group suggests the metabolic portion of the acid-base system contributed more to the acid-base balance of these calves.

Mean BE at 1 h differed by treatment ($P < 0.05$). This indicated calves in the **NA** group would require the addition of more strong base to neutralize pHa compared to calves in the **ANV** and **AV** group. Base excess is an indication of metabolic contributions to the acid-base system, and in the present study is consistent with decreased HCO_3^- concentrations observed in the **NA** group, suggesting pHa in calves of the **NA** treatment group was influenced by metabolic contributors.

Plasma cortisol levels at 1 h were not affected by treatment (Table 3). However, plasma lactate concentrations at 1 h were significantly higher for the **NA** group when compared to **ANV** and **AV** groups and were 5.673, 4.318, and 4.057 mmol/L, respectively (Table 3). Calves in this study were well above recommended ranges for 'normal' (0.56 to 2.22 mmol/L) lactate concentrations. The combination of increased lactate concentration and decreased HCO_3^- concentration and BE of the **NA** group, indicate tissue metabolism had not reached balance by 25 h after birth. These factors suggest that calves in the **NA** group were in a state of acid-base compromise similar to calves in both **ANV** and **AV** groups. All three groups appeared to by physiologically challenged at the time of birth.

Treatment Interactions with IgG Absorption

Plasma IgG (g/L) at 13, 25, and 36 h was unaffected by treatment (Table 3; Figures 1, 2). Apparent efficiency of absorption was also unaffected by treatment (Table 3; Figures 3, 4). The lack of response in treatment on both plasma IgG and AEA in this study contradict research regarding the association of Ig absorption and respiratory acidosis (Boyd, 1989; Besser et al., 1990). Variation may result from differences in sampling techniques (arterial vs venous blood) and sampling time. The amount of CO_2 and O_2 in blood drawn from the jugular vein is a reflection of arterial concentrations less utilization by the head region. This makes venous samples a poor indicator of respiratory function or tissue CO_2 concentration. Furthermore, the fetal lungs are making the transition from fluid-filled compartments in utero where they are responsible for only 10% of the cardiac output, to accommodating the majority of cardiac output (Kasari,

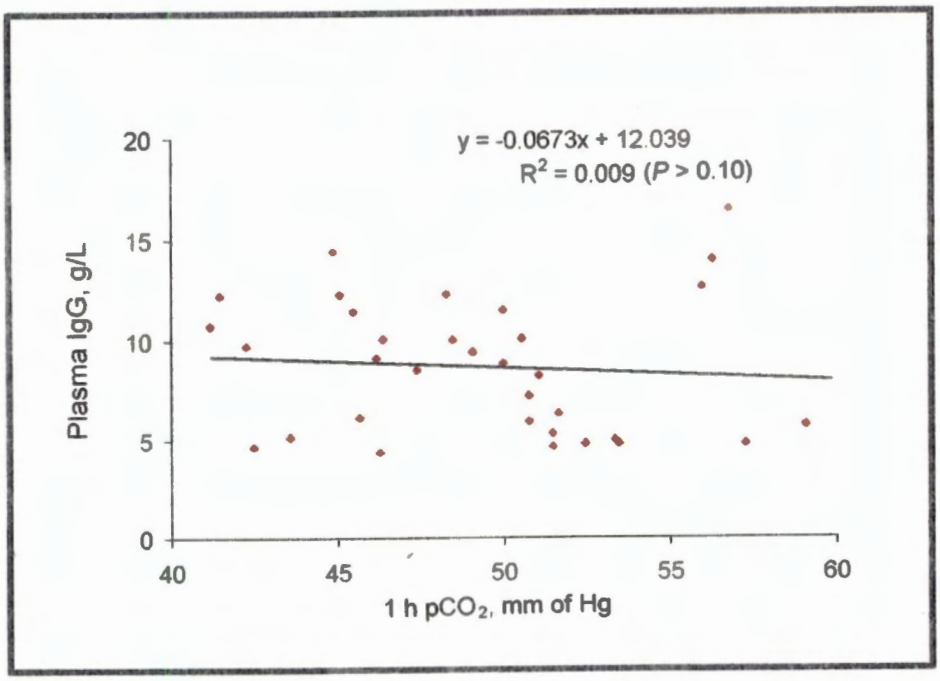

Figure 1: Regression analysis of 1 h $PaCO_2$ and 13 h plasma IgG.

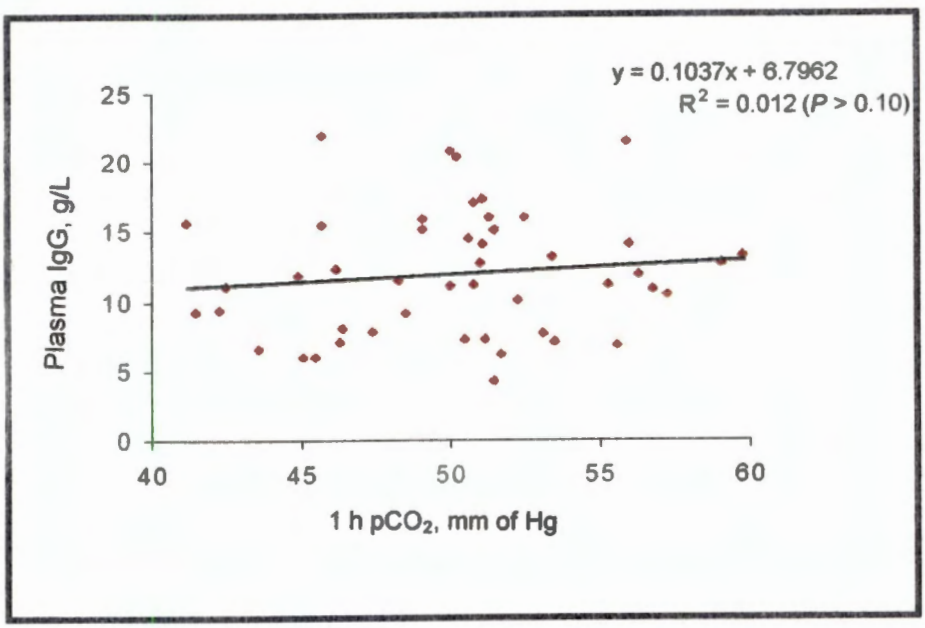

Figure 2: Regression analysis of 1 h $PaCO_2$ and 25 h plasma IgG.

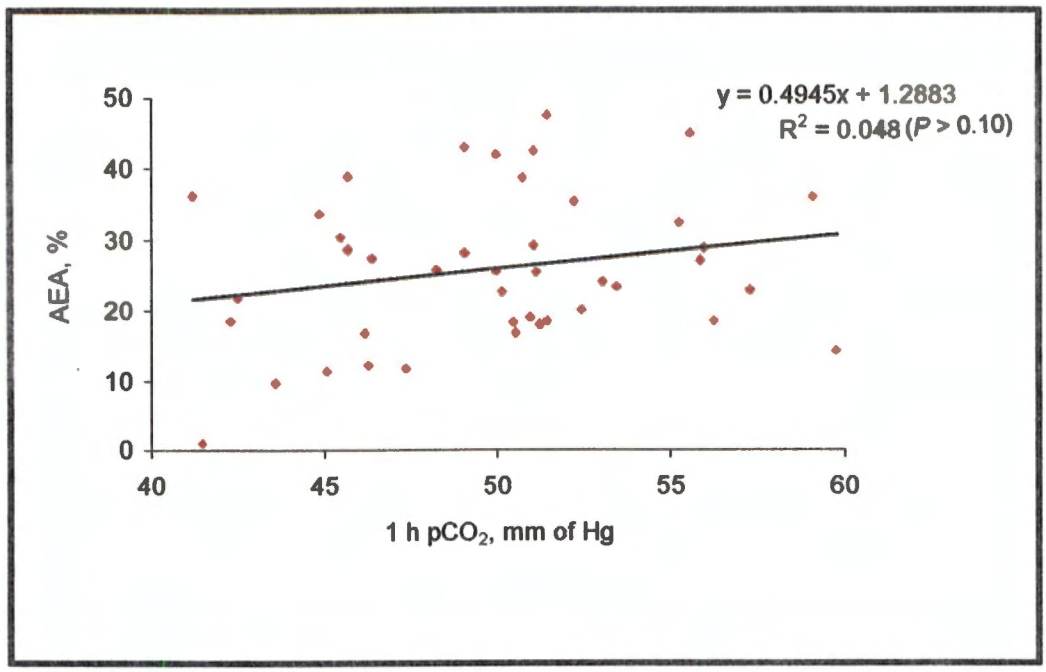

Figure 3: Regression analysis of 1 h $PaCO_2$ on apparent efficiency of IgG absorption at 25 h (AEA).

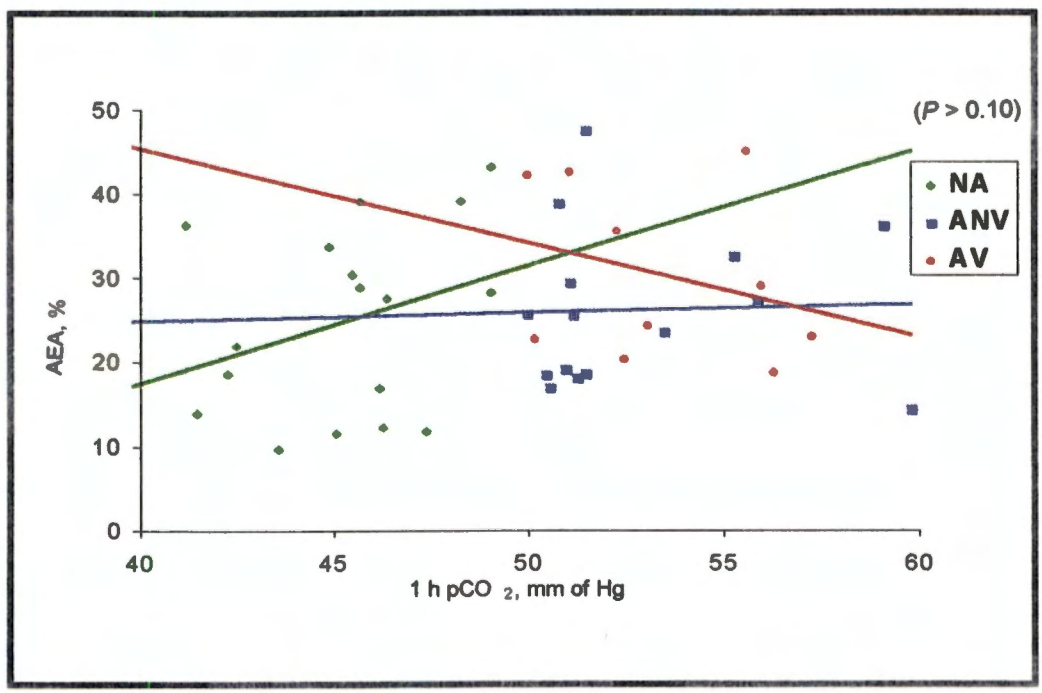

Figure 4: Regression analysis of 1 h $PaCO_2$ on apparent efficiency of IgG absorption at 25 h (AEA) for non-acidotic (NA), acidotic-non ventilated (ANV), and acidotic-ventilated (AV).

1994). Neonates have a decreased ability to expire CO_2 compared to adults and therefore, arterial blood going to tissue would already have elevated $PaCO_2$. Tissue metabolism would further increase the CO_2 in venous blood and the relationship between arterial and venous blood CO_2 levels may not be linear. This may explain the contradicting results observed in this study compared to studies reporting a significant negative relationship between $PvCO_2$ at birth and post feeding serum IgG concentrations.

Artificial respiration had no effect on any parameters measured in this study (Table 3). Post ventilation blood gas analysis indicated that artificial respiration was successful at lowering $PaCO_2$. However, this effect appeared to be temporary and did not influence the absorption of Ig or concentrations of selected metabolites.

Independent variable combinations were analyzed using regression procedures (SAS, 1996) to determine variables that contributed significantly to differences in plasma IgG concentrations at 25 h. The best one variable model describing plasma IgG at 25 h was IgG intake (Table 4). The best two variable model explaining plasma IgG (g/L) concentration included IgG intake and AEA (Table 4). Both IgG intake and AEA are expressed in the equation: plasma IgG (g/L) = IgG intake (g) x AEA / PV (L). Therefore, both were highly associated with IgG concentration in plasma at 25 h. This two variable model had an r^2 of 0.7077 and both variables were highly significant ($P < 0.0001$; Table 4). Inclusion of $PaCO_2$ at 1 h in this model was not significant and the resulting 3 variable model had only a marginally increased r^2 of 0.7150 (Table 4).

The best three variable model explaining variation in plasma IgG included IgG intake, AEA, and BW (Table 4). The r^2 for this model was 0.7451 and was not increased

Table 4. Regression models for plasma IgG at 25 h (g/L).

Model/variable	Coefficient	*P*	R^2
Plasma IgG, g/L			
IgG Intake		0.0001	0.4015
Intercept	3.430	0.0387	
IgG Intake	0.051	0.0001	
IgG Intake * AEA		0.0001	0.7077
Intercept	-5.730	0.0048	
IgG Intake	0.065	0.0001	
AEA	0.271	0.0001	
IgG Intake * AEA * $PaCO_2$, 1 h		0.0001	0.7150
Intercept	-9.750	0.0375	
IgG Intake	0.065	0.0001	
AEA	0.265	0.0001	
$PaCO_2$, 1 h	0.083	0.3323	
IgG Intake * AEA * BW		0.0001	0.7451
Intercept	2.880	0.4835	
IgG Intake	0.064	0.0001	
AEA	0.265	0.0001	
BW	-0.207	0.0236	
IgG Intake * AEA * BW * $PaCO_2$, 1 h		0.0001	0.7465
Intercept	0.590	0.9277	
IgG Intake	0.064	0.0001	
AEA	0.262	0.0001	
BW	-0.197	0.0385	
$PaCO_2$, 1 h	0.038	0.6484	

by the addition of $PaCO_2$ at 1 h to the model. Regression coefficients for IgG intake and AEA were positive indicating increasing plasma IgG concentration with increasing IgG intake and AEA. The negative coefficient for BW indicated declining plasma IgG concentration with increasing BW, which probably was a function of increasing PV. Furthermore, 1 h $PaCO_2$ was not significant when included in this model (Table 4). This three variable model is consistent with work by Kruse (1970b) who determined that serum Ig concentration during the first 24 h is a function of mass of Ig fed, age at first colostrum feeding and calf birth weight. Age at first colostrum feeding is representative of AEA. Rajala and Castrén (1995) reported a 30-min delay in first colostrum feeding decreased total Ig concentrations in serum by 2 g/L. The association between BW and PV is well established (Cronin, 1954; Kruse, 1970b; Möllerberg et al., 1975). Therefore, plasma IgG concentration may again be explained by the equation: plasma IgG (g/L) = IgG intake (g) x AEA / PV (L).

The lack of effect of 1 h $PaCO_2$ on plasma IgG concentrations at 25 h is likely due to compensatory mechanisms enabling neonates to reduce $PaCO_2$ prior to the onset of intestinal closure (Garry, 1993; Stott et al., 1979a). Walser and Maurer-Schweizer (1979) stated normal delivery is often followed by a pre-pathological acidosis. However, recovery to expected values for acid-base balance is completed by 24 h, or prior to the onset of intestinal closure. Calves in this study had not reached peak plasma IgG concentrations by 25 h as indicated by a slight increase in mean plasma IgG concentration at 37 h (Table 3). This study confirms work suggesting calves and piglets are capable of absorbing Ig 24 h after birth (Deutsch and Smith, 1957; Lecce and Morgan, 1962; Tyler

and Ramsey, 1991a).

Data differ from work by Besser et al. (1990) and Boyd (1989) in the relationship of 1 h $PvCO_2$ to plasma IgG concentration. Both studies determined a significant negative correlation between pre-feeding $PvCO_2$ and 12 h post-feeding serum IgG concentration. Age at first colostrum feeding in Besser and co-workers study was 1 h and work by Boyd ranged from 4 to 9 h. Colostrum was fed at the initial feeding only. Therefore, the age at serum Ig determination ranged from 13 to 21 h. The conflicting results may be due to a difference in sampling time, technique and sample storage. Our data indicated closure had not occurred by 21 h. Furthermore, venous samples artificially increase pCO_2 and are not true indicators of respiratory function (Donawick and Baue, 1968; Kirk, 1983; Szenci, 1985a; Kaneko, 1989; Smith, 1996). Besser et al. (1990) reported that several calves had a $PvCO_2 >$ 90 mm of Hg within 4 h after birth. These values are considerably higher than results from the present study. Furthermore, work done by Besser et al. (1990) stored blood gas samples on ice for up to 24 h prior to analysis. Prolonged storage of blood (> 6 h) prior to analysis will overestimate $PvCO_2$ by 2 to 3 mm of Hg and underestimate pHv by 0.026 (Haskins, 1977b; Kirk, 1983). Szenci and Besser (1990) also confirmed that changes in blood gas values on samples stored on ice for up to 24 h prior to analysis were not significant except for $PvCO_2$, which increased significantly.

Strawn (1996) examined relationships among blood gas parameters, acid-base balance and absorption of colostral IgG in stressed and normal calves. Calves were blocked on the basis of calving ease scores. Stressed calves were assigned based on

degree of birthing difficulty and dystocia. Dystocia and prolonged second stage labor have been implicated as risk factors associated with postnatal acidosis (Besser et al., 1990; Garry, 1993; Szenci, 1985a). Strawn (1996) observed no difference in the $PaCO_2$ of stressed and normal calves throughout the first 36 h of life. Furthermore, throughout the sampling period (36 h), no significant differences were observed in plasma IgG_1.

Similar results were observed in the relationship between 1 h $PaCO_2$ and AEA at 25 h. No differences were observed among treatments (Table 5; Figures 3,4). Using regression analysis (SAS, 1996), models were analyzed to determine variables explaining AEA. The single variable explaining the most variation in AEA was IgG intake ($P < 0.05$; Table 5). However, the best two variable model removed IgG intake. It explained 15.5% of the variation in AEA and included 1 h $PaCO_2$ and 1 h PaO_2 (Table 5). Both $PaCO_2$ and PaO_2 had a positive relationship with AEA. Neither 1 h $PaCO_2$ nor 1 h PaO_2 were significant when considered independently. Combining these variables not only created a significant model ($P < 0.05$) but also indicated that 1 h PaO_2 was significant and 1 h $PaCO_2$ tended to be significant ($P < 0.10$). Correlation analysis indicated the relationship between $PaCO_2$ and PaO_2 was significant ($P < 0.01$) and this relationship appeared to affect AEA.

Regression analysis of IgG intake independently on AEA was significant ($P < 0.05$; Table 5) while independently regressing 1 h PaO_2 on AEA only tended to be significant ($P < 0.09$; Table 5). Furthermore, the r^2 was 0.1090 and 0.0671 for IgG intake and 1 h PaO_2 models, respectively. This indicated IgG intake had a greater influence on AEA compared to 1 h PaO_2.

Table 5. Regression models for AEA at 25 h.

Model/variable	Coefficient	*P*	R^2
AEA, %			
IgG Intake, g		0.0328	0.1090
Intercept	35.920	0.0001	
IgG Intake	-0.058	0.0328	
$PaCO_2$, 1 h		0.2670	0.0307
Intercept	7.612	0.6506	
$PaCO_2$, 1 h	0.374	0.2670	
PaO_2, 1 h		0.0977	0.0671
Intercept	17.000	0.0048	
PaO_2, 1 h	0.135	0.0977	
$PaCO_2$, 1 h * PaO_2, 1 h		0.0377	0.1548
Intercept	-21.530	0.2866	
$PaCO_2$, 1 h	0.684	0.0512	
PaO_2, 1 h	0.199	0.0216	
IgG Intake * PaO_2, 1 h		0.0484	0.1439
Intercept	27.710	0.0012	
IgG Intake	-0.050	0.0690	
PaO_2, 1 h	0.100	0.2150	
IgG Intake * $PaCO_2$, 1 h * PaO_2, 1 h		0.0169	0.2277
Intercept	-9.670	0.7442	
IgG Intake	-0.042	0.1192	
$PaCO_2$, 1 h	0.631	0.0498	
PaO_2, 1 h	0.164	0.0570	
IgG Intake * $PaCO_2$, 1 h * PaO_2, 1 h * Lactate, 1 h		0.0404	0.2316
Intercept	-14.44	0.5259	
IgG Intake	-0.039	0.1519	
$PaCO_2$, 1 h	0.660	0.0636	
PaO_2, 1 h	0.160	0.0676	
Lactate, 1 h	0.675	0.2749	

Increasing the model to three variables, with IgG intake entering the model again, appeared to be the model of best fit. In addition to increasing the r^2 to 0.2277, the three variable model indicated 1 h $PaCO_2$ was significant ($P < 0.05$) and 1 h PaO_2 tended to be significant ($P < 0.10$). However, including the blood gas parameters in the model appeared to mask the importance of IgG intake, which was no longer significant ($P > 0.10$). Furthermore, both 1 h $PaCO_2$ and 1 h PaO_2 were positively correlated to AEA while colostral IgG was negatively correlated to AEA.

Although little current literature examines the relationship between $PaCO_2$ and AEA, most have reported a negative relationship between $PvCO_2$ and IgG concentration (Boyd, 1989; Besser et al., 1990). Differing results may be attributed to time and method of sampling. The $PaCO_2$ declines rapidly during the first hour of life (Strawn, 1996). Samples at 1 h in the present study may have lower $PaCO_2$ compared to samples taken immediately after birth. The present study also utilized arterial sampling which is the only true method of assessing respiration and blood gas values (Donawick and Baue, 1968). Other researchers (Boyd, 1989; Besser et al., 1990) utilized venous samples which increase pCO_2 values and decrease pH (Filley, 1971; Szenci, 1985a). Furthermore, $PaCO_2$ and pHa observed in the present study are well within physiological norms and should not be regarded as pathological (Assali et al., 1963; Garry, 1993; Smith, 1996). Calves with increased $PaCO_2$ were able to absorb colostral IgG with similar AEA, indicating homeostatic compensation for elevated $PaCO_2$ at 1 h (Figures 1, 2, 3, and 4).

The negative correlation between AEA and colostral IgG (Figure 5) intake

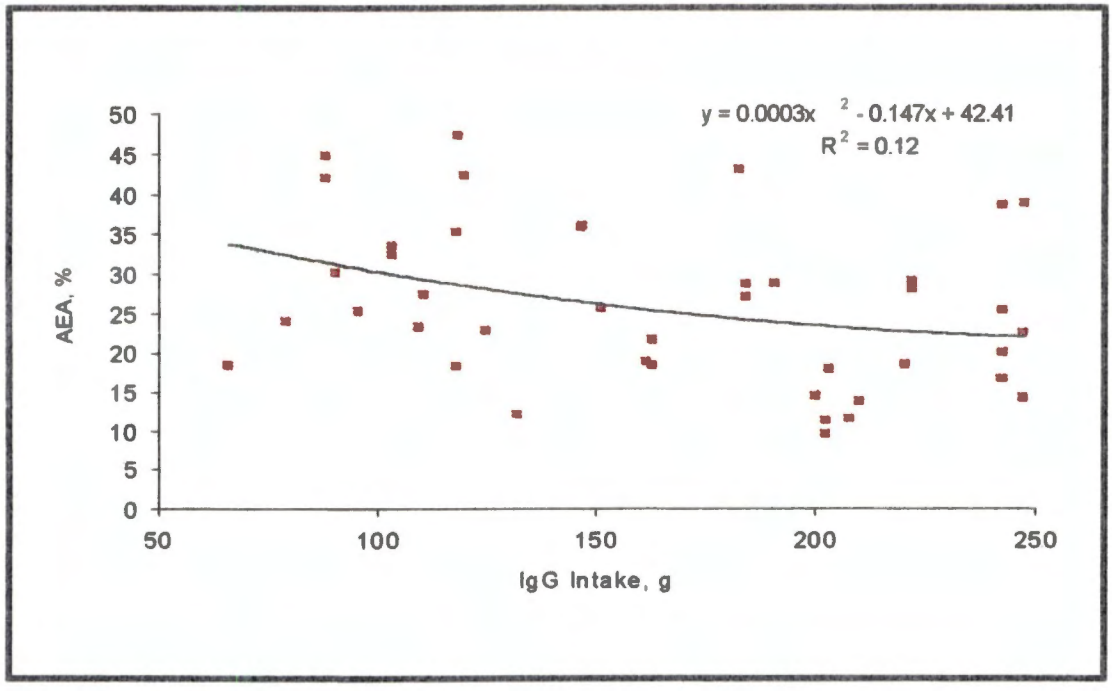

Figure 5: Regression analysis of IgG intake and AEA.

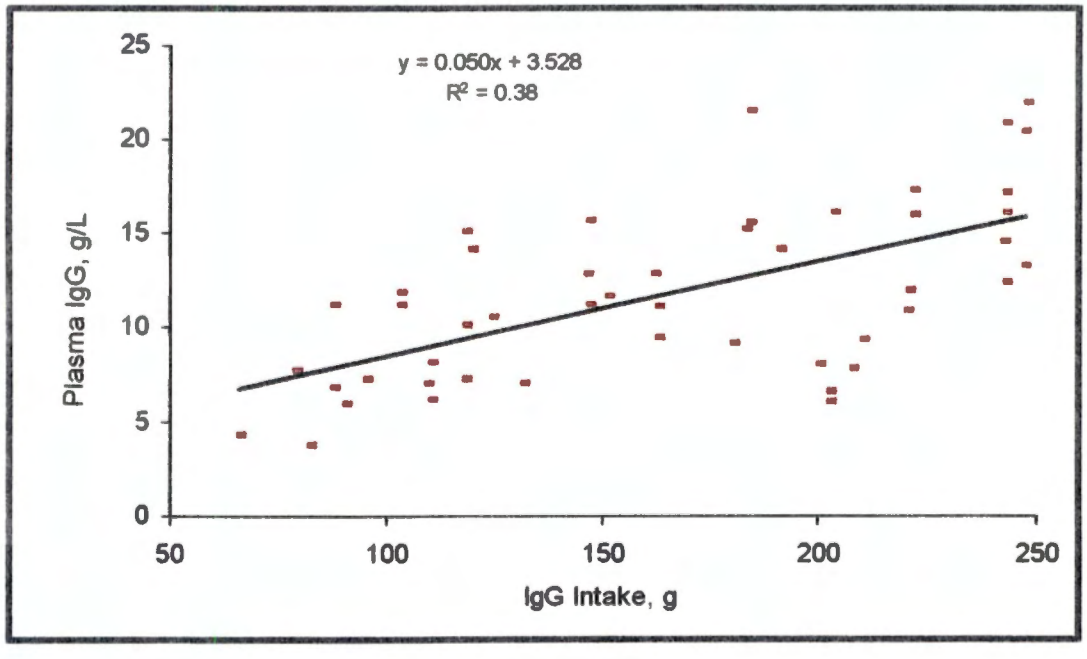

Figure 6: Regression analysis of IgG intake and plasma IgG.

suggests a possible curvilinear relationship between mass of IgG consumed and plasma IgG. However, regression analysis (SAS, 1996) indicated the relationship was best fit by a linear function and not by a polynomial function (Figure 6). Besser et al. (1985) observed a negative correlation between AEA and colostral IgG mass in two experiments where colostrum IgG concentration varied from 11 to 151 g/L. In the present study, only 2 calves included in AEA analysis were fed different volumes of colostrum within the first 24 h. Therefore, difference in IgG intake is the result of the variation in colostrum IgG concentrations and the negative correlation observed in AEA and IgG intake is similar to observations found by Besser et al. (1985). Other researchers (Stott and Menefee, 1978) observed no change in absorption efficiencies of IgG with varying colostrum intakes of similar concentrations. On the other hand, Kruse (1970b) reported a positive linear relationship between mass of IgG given and the absorption coefficient for IgG.

5. SUMMARY AND CONCLUSIONS

In summary, effects observed in the present study indicate that a myriad of homeostatic mechanisms are active during the neonatal period. Possible mechanisms involved in the decrease of colostral Ig observed by other researchers may be associated with factors other than $PaCO_2$ or $PvCO_2$. Acidosis generally depresses cardiac output and contractility in the denervated heart (Haskins, 1977a; Blood and Radostits, 1989). Furthermore, decreased blood flow to the intestine may impair absorptive ability (Haskins, 1977a; Blood and Radostits, 1989; Guyton and Hall, 1996). However, in a non pathological condition, acidosis activates the sympathetic nervous system resulting in increased cardiac contractility, increased heart rate and increased cardiac output (Blood and Radostits, 1989). Decreased content of O_2 in blood flowing to the intestine may impair absorptive ability, cellular metabolic function and respiration (Staley and Bush, 1985). Finally, an increase in lactate concentrations and inhibition of glycolysis and protein synthesis may also contribute to malabsorption of colostral Ig (Staley and Bush, 1985; Guyton and Hall, 1996).

Isolation of individual effects on a single parameter such as IgG absorption or AEA is often difficult to surmise. Significant negative correlations between elevated $PaCO_2$ and IgG absorption and AEA were not observed in this study. Various blood gas and metabolic parameters differed among three treatment groups. Calves in this study were born acidotic, as indicated by depressed pHa at 1 h. However, neither plasma IgG at 25 h nor AEA were compromised by depressed pHa. Furthermore, blood gas and acid-base values for neonates should not be compared with adult levels. Further research is

required utilizing pHa to determine mechanisms that may possibly impair absorption of colostral Ig reported in other research.

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Vita

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