Effect of high arterial carbon dioxide ten on efficiency of immunoglobulin G absor in calves

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Objectives—To determine whether high $Paco_2$ reduced apparent efficiency of IgG absorption (AEA) in calves and whether assisted ventilation of calves with high $Paco_2$ increased AEA.

Animals—48 Holstein calves.

Procedures—Arterial and venous blood samples were collected 1, 13, and 25 hours after birth; an additional venous sample was collected at 37 hours after birth. Arterial samples were analyzed for $Paco_2$, Pao_2, pH, and bicarbonate and base excess concentrations; venous samples were analyzed for plasma IgG concentrations. On the basis of 1-hour $Paco_2$, calves were assigned to nonrespiratory acidosis ($Paco_2$ < 50 mm Hg; n = 19) or respiratory acidosis ($Paco_2$ ≥ 50 mm Hg; 29) groups. Calves in the respiratory acidosis group were assigned randomly to receive no further treatment (n = 17) or to be given 5 minutes of assisted ventilation (12). All calves received between 1.8 and 2 L of colostrum 2, 14, 26, and 38 hours after birth. Plasma volume and AEA were determined 25 hours after birth.

Results—1-hour $Paco_2$ had no effect on AEA or on plasma IgG concentrations determined 13, 25, or 37 hours after birth. Artificial ventilation had no effect on plasma IgG concentration or AEA.

Conclusions and Clinical Relevance—Lack of effect of 1-hour $Paco_2$ on AEA and IgG concentration indicated that calves compensated for moderate acid-base imbalances associated with birth. Calves born with high $Paco_2$ achieved adequate plasma IgG concentrations if fed an adequate amount of high-quality colostrum early in life. The effect of artificial ventilation on $Paco_2$ was temporary and did not increase AEA. (*Am J Vet Res* 1999;60:609–614)

Calves are born hypogammaglobulinemic and require acquisition of passive immunity through absorption of colostral immunoglobulins. Research has indicated a large percentage of calves die if they fail to achieve adequate serum immunoglobulin concentrations.[1-4] Transfer of immunoglobulins to calves is influenced by a number of factors, including age at first feeding, volume and immunoglobulin concentration of colostrum fed, birth weight, method of feeding, seasonal influences, stress, disease, use of colostral supplements, presence of the dam, and individual variation in absorption efficiency.[3,5-7] Aci during the early postpartum period detrimental effect on the absorp immunoglobulins.[8-10,a] Furthermore, cated that metabolic, respiratory, an develop frequently at birth in calve prolonged or difficult labor and dyst

A 52% decrease in colostrum in 35% decrease in serum IgG concent acidotic calves (venous blood pH < with that in calves with venous bl however, decreased serum IgG conce calves may have been the result of rather than acid-base status. Boyd [8] cant correlation between prefeedi ($Pvco_2$) and postfeeding serum Ig Calves of that study were in a state pensated respiratory acidosis. The that reduced absorption of IgG_1 fro associated with hypercapnia in a newborn calves. Similar work by Bes cated that decreased IgG_1 absorptio was associated with respiratory aci metabolic acidosis. Boyd[8] and Besse blood for $Pvco_2$, venous pH (pHv), bonate (HCO_3^-) concentration. Resul indicated a significant inverse rela prefeeding $Pvco_2$ and 12-hour postfe concentration. Despite adequate int postnatal respiratory acidosis in calve ed concentrations of IgG_1 absorbed f

Previous studies failed to stress using arterial blood samples for bloo the importance in standardizing time lection to analyses. Effects of regiona circulation on venous blood alters Pv unpredictable manner, so that the res tion to an acidotic state cannot be d Analysis of arterial blood provides t indicator of acid-base status, oxygena tory function and, therefore, is the m cator of respiratory acidosis.[13] Furthe samples for > 6 hours will cause $Pvco_2$ and underestimation of pHv objectives of the study reported here whether high $Paco_2$ 1 hour after birt ent efficiency of IgG absorption(AE whether assisted ventilation in c 1-hour $Paco_2$ increased AEA.

Materials and Methods

Animals and treatment groups—F calves (25 heifers and 23 bulls) were ass

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following groups on the basis of $PaCO_2$ measured 1 hour after birth: **nonrespiratory acidosis (NA)**, $PaCO_2$ < 50 mm Hg (n = 19); and respiratory acidosis, $PaCO_2$ ≥ 50 mm Hg (29). Calves in the respiratory acidosis group were randomly assigned to receive no further treatment (**acidosis/not ventilated [ANV]**; n = 17) or 5 minutes of assisted ventilation (**acidosis/ventilated [AV]**; 12). Beginning immediately after assignment of a calf to the AV group, and using a self-inflating resuscitation bag,[b] a breath was stimulated every 5 seconds for 5 minutes. Arterial blood gas analysis after ventilation indicated that treatment was effective at reducing $PaCO_2$.

Calf management and sample collection—Pregnant cows were housed on a drylot and fed a diet of mixed-grass hay and commercial nonlactating cow concentrate. Estimated dietary cation-to-anion difference was approximately +65 mEq/100 g of concentrate. At birth, calving ease scores were assigned on the basis of a scale of 1 to 4. A score of 1 indicated a normal (unassisted) birth, 2 indicated an easy assisted birth, 3 indicated a difficult assisted birth, and 4 indicated a cesarean section. Calves were separated from their dams at birth and were not allowed to suckle.

Arterial blood samples were collected 1, 13, and 25 hours after birth. Arterial blood was collected from the brachial artery into heparinized (1,000 IU/ml) syringes.[14,c] Analyses were performed within 30 minutes of sample collection to determine **arterial pH (pHa)**, $PaCO_2$, PaO_2, and HCO_3^- and **base excess (BE)** concentrations. Correction equations were used to calculate pHa, $PaCO_2$ and PaO_2 at rectal temperatures other than 37 C. Venous blood samples were collected 1, 13, 25, and 37 hours after birth for analysis of plasma IgG and lactate concentrations. Plasma volume was measured at 25 hours, using the Evans' blue dye technique.[16]

Feeding management—Calves were fed between 1.8 and 2.0 L of independent pools of colostrum from a nipple bottle 2 hours (± 5 minutes) after birth. If calves did not drink the entire amount, remaining colostrum was fed via an esophageal tube. Calves also were fed pooled colostrum 14, 26, and 38 hours after birth. Colostral IgG concentration was measured by use of single radial immunodiffusion[d] and the method of Fahey and McKelvey.[17]

Apparent efficiency of IgG absorption was calculated at 25 hours by the method of Husband et al,[18] using the formula:

$$\text{AEA} = [\text{plasma IgG (g/L)} \times \text{plasma volume (L)}] / [\text{colostral IgG (g/L)} \times \text{colostral intake (L)}]$$

Data analysis—Blood gas tensions ($PaCO_2$ and PaO_2) and plasma lactate concentration 1 hour after birth were analyzed, using the model:

$$Y_{ij} = \mu + T_i + e_{ij}$$

where Y_{ij} = $PaCO_2$, PaO_2, or lactate concentration; μ = overall mean; T_i = effect of ith treatment group; e = random error associated with the ith treatment group and the jth calf (I = 1, 2, 3; j = 1, 2, ...n).

Plasma IgG concentrations 13 and 25 hours after birth and AEA at 25 hours were analyzed, with IgG intake as a covariate, using the model:

$$Y_{ij} = \mu + T_i + (X_{ij} - \bar{X}) + e_{ij}$$

where Y_{ij} = plasma IgG concentrations or AEA; μ = overall mean; T_i = effect of ith treatment group; $(X_{ij} - \bar{X})$ = adjustment for the effect of the covariate; e_{ij} = random error associated with the ith treatment group and the jth calf (I = 1, 2, 3; j = 1, 2, ...n).

General linear model procedures were used in ANOVA and to generate least squares means and SEM for blood gas tensions, plasma IgG concentration, and AEA. Regression analysis was used to determine relationships among blood gas tensions, plasma IgG concentrations, and AEA.

Results

Body weight of calves ranged from 22.3 to 50.0 kg with a mean ± SEM of 39.8 ± 0.70 kg (**Table 1**). Mean calving ease score was 1.79 ± 0.12, and ranged from 1 to 3, indicating that cesarean sections were not performed. Eleven of 48 births were considered difficult and assisted; all dams in this group were primiparous. Parity of dams ranged from 1 to 5, with a mean parity of 2.08 ± 0.21.

Mean plasma IgG concentration for all calves 13 hours after birth was 8.31 ± 0.50 g/L, and ranged from 3.66 to 16.51 g/L (Table 1). Mean IgG concentrations increased to 11.73 ± 0.66 g/L at 25 hours and 12.90 ± 0.74 g/L at 37 hours. Plasma IgG concentrations 13, 25, and 37 hours after birth were unaffected by treatment group (**Table 2; Fig 1 and 2**) and increased in all 3 groups throughout the first 37 hours of life.

Mean AEA determined 25 hours after birth for 43/48 calves was 26.0 ± 1.5% (Table 1). Apparent efficiencies of IgG absorption were unaffected by treatment group (Table 2; **Fig 3**) and were similar to values reported by others.[5,19]

Mean IgG concentration in colostrum was 42.37 ± 1.93 g/L; colostrum was considered of moderate quality.[20] Concentrations of IgG in colostrum ranged from 12.10 to 65.95 g/L and did not differ among treatment groups. Grams of IgG intake were calculated as the product of IgG concentration in colostrum (g/L) and the amount of colostrum (L) consumed by calves. Intake of IgG from the first 2 feedings determined for 48 calves ranged from 65.7 to 247.4 g and did not differ among treatment groups (Tables 1 and 2). Mean IgG intake for all calves was 165.0 ± 8.2 g.

Table 1—Descriptive statistics for data collected from 48 Holstein calves beginning 1 hour after birth. Calves did not suckle from dams but received between 1.8 and 2 L of colostrum 2, 14, 26, and 38 hours after birth

Variable	Mean ± SEM	Range
Body weight (kg)	39.80 ± 0.700	22.30–50.00
Ease of birth*	1.79 ± 0.12	1.00–3.00
Parity of dam	2.08 ± 0.21	1.00–5.00
Colostral IgG concentration (g/L)†	42.37 ± 1.93	12.10–65.95
IgG intake at 25 h (g)‡	165.00 ± 8.20	65.70–247.40
Plasma IgG concentration (g/L)		
1 h after birth	1.40 ± 0.03	1.19–2.67
13 h after birth	8.31 ± 0.50	3.66–16.51
25 h after birth	11.73 ± 0.66	3.82–22.00
37 h after birth	12.90 ± 0.74	3.61–22.82
Total IgG in plasma at 25 h (g)§	41.55 ± 2.90	12.22–96.80
AEA at 25 h (%)‖	26.00 ± 1.50	9.80–47.60
Plasma lactate concentration (mmol/L)		
1 h after birth	4.93 ± 0.39	1.42–12.32
13 h after birth	3.55 ± 0.19	0.66–7.52
25 h after birth	2.67 ± 0.16	1.03–7.49

*1 = unassisted birth. 2 = easy assisted birth. 3 = difficult assisted birth. 4 = cesarean section. †Determined from values for colostrum used at 2 and 14 h. ‡Product of colostral volume fed 2 and 14 h after birth and colostral IgG concentration. §Product of plasma volume (determined by use of Evans' blue dye technique) and plasma IgG concentration. ‖Apparent efficiency of IgG absorption (AEA) determined for 43 calves using the equation: (total IgG in plasma/ IgG intake) × 100.

Table 2—Least squares means of variables measured by analyses of arterial blood samples 1 hour after birth, and of plasma IgG and lactate concentrations, and apparent efficiencies of IgG absorption (AEA) for 48 Holstein calves assigned to 2 groups on the basis of 1-hour $Paco_2$. Calves in the nonrespiratory acidosis (NA) group ($Paco_2$ < 50 mm Hg) received no treatment. Calves with $Paco_2$ ≥ 50 mm Hg were assigned randomly to respiratory acidosis/not ventilated (ANV) or respiratory acidosis/ventilated (AV) groups; the AV group received assisted ventilation for 5 minutes 1 hour after birth. Calves did not suckle from dams but received between 1.8 and 2 L of colostrum 2, 14, 26, and 38 hours after birth

	Treatment groups (n)			Comparisons*
Variable	NA (19)	ANV (17)	AV (12)	NA vs (AV + ANV)
$Paco_2$ (mm Hg)	45.52	53.04	53.22	$P < 0.001$
Pao_2 (mm Hg)	76.11	66.94	63.25	NS
pHa	7.309	7.283	7.308	NS
Bicarbonate(mmol/L)	22.91	25.37	26.50	$P < 0.001$
Base excess (mEq/L)	−2.23	−0.75	0.58	$P < 0.05$
1-h plasma lactate (mmol/L)	5.673	4.318	4.057	$P < 0.05$
IgG intake (g)†	174.5	166.6	145.6	NS
Plasma IgG (g/L)				
13 h after birth	7.92	8.71	8.62	NS
25 h after birth	10.87	12.56	13.21	NS
37 h after birth	11.74	13.89	13.56	NS
AEA at 25 h (%)‡	24.1	26.1	30.3	NS

*Significant differences not detected between ANV and AV groups. †Amount of IgG in volume of colostrum fed at 2 and 14 h. ‡(Total IgG in plasma/ IgG intake) × 100.
NS = not significant ($P \geq 0.05$).

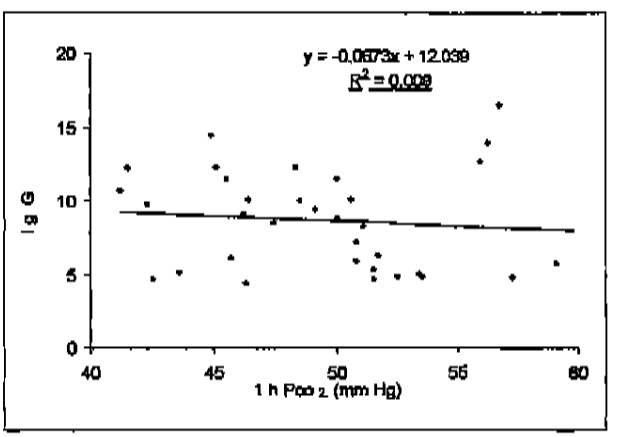

Figure 1—Scatterplot of 1-hour $Paco_2$ versus 13-hour plasma IgG concentrations (g/L) for 48 Holstein calves that did not suckle from dams but received between 1.8 and 2 L of colostrum 2 hours after birth. The line determined by the depicted regression equation is superimposed.

Mean plasma lactate concentration for all calves 1 hour after birth was 4.93 ± 0.39 mmol/L and decreased to 2.67 ± 0.16 mmol/L at 25 hours. However, 1-hour plasma lactate concentration was significantly ($P < 0.05$) higher for calves of the NA group (5.673 mmol/L), compared with concentrations for calves of the ANV and AV groups (4.318 and 4.057 mmol/L, respectively; Table 2). Mean lactate concentration in calves of all treatment groups decreased during the first 25 hours of life, although concentrations at all times measured remained above the recommended range (0.56 to 2.22 mmol/L).

Mean $Paco_2$ for all calves 1 hour after birth was 50.80 ± 0.67 mm Hg and decreased to 45.74 ± 0.44 and 45.44 ± 0.46 mm Hg at 13 and 25 hours, respectively

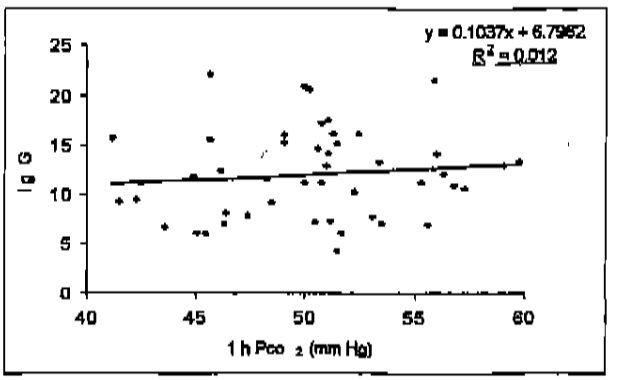

Figure 2—Scatterplot of 1-hour $Paco_2$ versus 25-hour plasma IgG concentrations (g/L) for 48 Holstein calves that did not suckle from dams but received between 1.8 and 2 L of colostrum 2 and 14 hours after birth. The line determined by the depicted regression equation is superimposed.

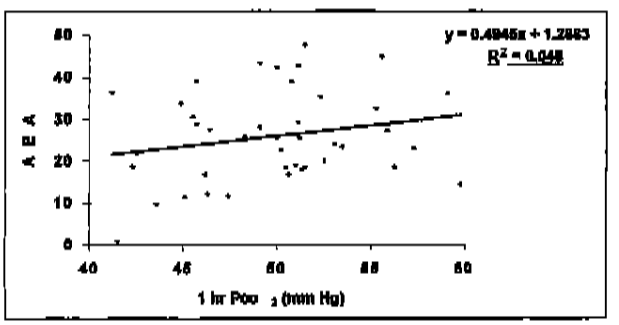

Figure 3—Scatterplot of 1-hour $Paco_2$ versus apparent efficiency of IgG absorption (AEA; [total IgG in plasma 25 hours after birth/IgG intake from colostrum fed] X 100) for 48 Holstein calves that did not nurse from dams but received between 1.8 and 2 L of colostrum 2 and 14 hours after birth. The line determined by the depicted regression equation is superimposed.

(Table 3). Calves were assigned to groups on the basis of 1-hour $Paco_2$, and, thus, 1-hour $Paco_2$ among groups were significantly ($P < 0.001$; Table 2) different. Calves of the ANV and AV groups had higher 1-hour $Paco_2$, compared with calves of the NA group. One-hour $Paco_2$ for 47 of 48 calves ranged from 41.20 to 59.80 mm Hg and were within the reference range established for samples collected from the brachial artery of neonatal calves.[11,21,Ae]

Mean pHa for all calves 1 hour after birth was 7.303 and ranged from 7.113 to 7.400 (Table 3). By 13 hours, mean pHa had increased to 7.386 ± 0.004 and by 25 hours was 7.425 ± 0.005, slightly greater than the optimal pH of 7.4; pHa at any time measured did not differ among groups (Table 2). Mean pHa for all treatment groups indicated that calves were born slightly acidotic and had partially compensated by 25 hours of age.[8,9,23,24] Similar pHa values determined at or shortly after birth have been reported.[11,21] A steady compensation in pHa also was observed in those studies, with mean pHa of 7.4 measured 24 hours after birth.

Mean Pao_2 for all calves 1 hour after birth was 69.1 ± 2.8 mm Hg and increased to 80.6 ± 2.7 mm Hg at 13 hours (Table 3). A wide range of Pao_2 values was observed at all 3 sample collection times. At 25 hours, mean Pao_2 decreased to 72.2 ± 2.8 mm Hg, with a range of 39.0 to 118.0 mm Hg. Calves of the NA group

Table 3—Descriptive statistics for variables measured in arterial blood samples collected from 48 Holstein calves

Variable*	Mean ± SEM	Range
$Paco_2$ (mm Hg)		
1 h after birth (n = 47)	50.80 ± 0.67	41.20–59.80
13 h after birth (47)	45.74 ± 0.44	38.50–53.30
25 h after birth (46)	45.44 ± 0.46	38.40–51.40
pHa		
1 h after birth (47)	7.303 ± 0.008	7.113–7.400
13 h after birth (47)	7.386 ± 0.004	7.300–7.440
25 h after birth (46)	7.426 ± 0.005	7.346–7.500
Pao_2 (mm Hg)		
1 h after birth (47)	69.1 ± 2.8	31.0–112.0
13 h after birth (47)	80.6 ± 2.7	43.0–123.0
25 h after birth (46)	72.2 ± 2.8	39.0–118.0
Bicarbonate (mmol/L)		
1 h after birth (47)	24.72 ± 0.45	13.60–29.60
13 h after birth (47)	27.30 ± 0.32	20.70–31.50
25 h after birth (46)	29.66 ± 0.35	23.20–33.40
Base excess (mEq/L)		
1 h after birth (47)	−0.98 ± 0.52	−13.90–4.40
13 h after birth (47)	2.85 ± 0.33	−3.80–7.40
25 h after birth (46)	5.74 ± 0.36	−1.40–9.60

*Arterial blood samples were analyzed within 30 min of collection. Blood gas values corrected for rectal temperature.

tended to have higher 1-hour Pao_2 than did calves of the ANV and AV groups, but the difference was not significant (Table 2).

Mean HCO_3^- concentration 1 hour after birth was 24.72 ± 0.45 mmol/L, and increased to 27.30 ± 0.32 mmol/L by 13 hours. At 25 hours, mean HCO_3 concentration had increased to 29.66 ± 0.35 mmol/L, ranging from 23.20 to 33.40 mmol/L. One-hour HCO_3^- concentrations differed significantly ($P < 0.001$) among groups (Table 2); calves of the ANV and AV groups had significantly higher HCO_3^- concentrations, compared with calves of the NA group.

Base excess concentration for all calves 1 hour after birth ranged from -13.90 to 4.40 mEq/L with a mean of −0.98 ± 0.52 mEq/L. Base excess concentration at 1 hour differed significantly among groups (Table 2); calves of the NA group had significantly ($P < 0.05$) lower BE concentration, compared with calves of the ANV and AV groups. Mean BE concentration for all calves increased to 2.85 ± 0.33 mmol/L at 13 hours and 5.74 ± 0.36 mmol/L at 25 hours.

Discussion

Mean plasma IgG concentration of 11.73 g/L 25 hours after birth indicated that calves in our study achieved adequate transfer of colostral IgG after the first 2 feedings. Failure of passive transfer is indicated by plasma IgG concentration < 10 g/L.[24] However, plasma IgG concentrations at 13 and 25 hours, and AEA determined at 25 hours, were unaffected by treatment. Regression analysis of plasma IgG concentrations at 13 and 25 hours and AEA at 25 hours on $Paco_2$ also indicated that a relationship did not exist between these variables (Fig 1–3). Our results indicated that calves with high $Paco_2$ 1 hour after birth were able to compensate for increased co_2 and absorb adequate amounts of colostral IgG and are in agreement with results of other studies.[11,24,c] Partial compensation of acidosis also was indicated by a decrease of mean $Paco_2$ to 45.74 and 45.44 mm Hg at 13 and 25 hours, respectively. Lack of effect of 1-hour $Paco_2$ on plasma IgG concentrations at 25 hours is likely attributable to compensatory mechanisms enabling neonates to reduce $Paco_2$ prior to the onset of closure of intestinal absorptive capacity.[11,c] Walser and Maurer-Schweizer[23] stated that unassisted deliveries often are followed by prepathologic acidosis in neonates. However, recovery to expected values for acid-base balance is completed by 24 hours, allowing for adequate absorption of colostral immunoglobulins.

Lack of a significant effect of treatment on plasma IgG concentrations and AEA determined 25 hours after birth among groups in this study contradicts results of other studies regarding association of immunoglobulin absorption and respiratory acidosis.[9,8] Discrepancies may be attributable to differences in sample collection techniques (arterial vs venous blood, sample collection site), experimental design, sample collection time, sample handling, and degree and type of acidosis. Values for $Paco_2$ in neonatal calves of this study are in agreement with values obtained from brachial artery blood samples in other studies.[11,13,c] Compensation of high $Paco_2$ observed in those studies occurred within 24 hours without medical intervention.[11,c]

Differences between $Pvco_2$ and $Paco_2$ are an important consideration in interpreting data from these types of studies. Neonatal lungs have decreased ability to expire co_2, compared with adult lungs and, therefore, arterial blood would contain high concentrations of co_2. Tissue metabolism would contribute further to increased co_2 concentration in venous blood, thereby making analysis of venous blood a poor indicator of respiratory function or tissue co_2 concentration. Furthermore, in our study, blood gas analysis was performed within 30 minutes of sample collection, compared with storage times as long as 24 hours in other studies. Prolonged storage of blood (> 6 hours) will lead to overestimation of $Pvco_2$ by 2 to 3 mm Hg.[14,15] Our ability to rapidly determine blood gas tensions could have contributed further to observed differences in results. Finally, calves of our study appeared clinically normal. Differences in results among our study and others may have been a function of differences in calves' metabolic state, degree of acidosis, or both.

Experimental design dictated that calves were assigned to groups on the basis of 1-hour $Paco_2$ which, therefore, differed significantly among groups. Significant differences in 1-hour Pao_2 were not detected among groups, but Pao2 was related inversely to $Paco_2$. However, pHa at any time measured did not differ among groups, indicating that factors other than $Paco_2$ and Pao_2 at 1 hour were contributing to the decrease in pHa that occurred during the study. Furthermore, mean pHa (7.303) for all calves suggested that, as a group, calves were born slightly acidotic, and was in agreement with results of other studies.[11,c] Data from studies utilizing venous samples indicated a mean pHv of 7.22 in calves born of a normal delivery, compared with a mean pHv of 7.06 in calves considered at high risk for acidosis.[9] In our study, mean calving ease score of 1.79 ± 0.12 indicated that few calves were predisposed to risk factors associated with severe

acidosis at birth. Although calves were slightly acidotic 1 hour after birth, they appeared clinically normal. Therefore, metabolic contributions of HCO_3^- and BE concentrations to pHa should be considered as possible influencing factors.

One-hour HCO_3^- and BE concentrations were significantly lower in the NA group, compared with the ANV and AV groups. Higher HCO_3^- concentrations observed in calves of the ANV and AV groups may have resulted from increased $PaCO_2$ through the equilibrium of carbonic acid. Increased HCO_3^- concentrations in these calves suggest either a partial metabolic alkalosis or compensation for acidosis of respiratory origin. Significantly lower HCO_3^- and BE concentrations in calves of the NA group suggest that the metabolic portion of the acid-base system contributed more to the acid-base balance in these calves, whereas the respiratory portion, or $PaCO_2$, contributed more to acid-base balance in calves of the ANV and AV groups.

Plasma lactate concentrations at 1 hour were significantly higher in the NA group, compared with the ANV and AV groups. Lactate concentrations in calves of this study were above recommended ranges for mature cattle (0.56 to 2.22 mmol/L). However, Kasari[15] reported that lactate concentrations of 4.4 mmol/L in newborn calves indicated lactic acidemia. Randall[25] also reported that high lactic acid concentrations caused by low glycogen stores resulting in anaerobic glycolysis during parturition are common in neonates. As respiration reaches homeostasis and glucose is provided by colostrum, anaerobic glycolysis ceases, and lactate concentrations decrease. Tyler and Ramsey [26] observed lactate concentrations > 6 mmol/L in hypoxic calves. Those authors concluded that high lactate concentrations were attributable to anaerobic metabolism exceeding the capacity of neonates to utilize lactate as an energy source. Similarly, high lactate concentrations in our calves represent lactate production in excess of the ability of calves to utilize lactate. High concentrations contribute to progressive metabolic acidosis,[26] which is common in calves < 8 days old.[27] Values for lactate concentrations for calves of the NA group suggested acidosis of metabolic origin.

The combination of increased lactate concentration and decreased HCO_3^- and BE concentrations in calves of the NA group, compared with calves in the other groups, indicated that tissue metabolism had not reached a balance by 1 hour after birth, and calves of the NA group were in a state of acid-base compromise similar to calves of the ANV and AV groups. All 3 groups appeared to be physiologically but not clinically challenged at the time of birth. However, lack of effect of treatment on plasma IgG concentrations and AEA among groups indicated that calves were able to compensate for acid-base disturbances during the first 24 hours after birth and achieved adequate immunity through absorption of colostral IgG.

Assisted ventilation had no effect on any of the variables measured in this study. Blood gas analysis after ventilation indicated that this intervention was successful at decreasing $PaCO_2$ and increasing pHa. However, the effect appeared temporary and did not influence absorption of immunoglobulins or plasma concentrations of selected metabolites. Correction of high $PaCO_2$ did not require clinical intervention. However, further research is warranted to evaluate the duration and degree of compensation required by calves to correct high $PaCO_2$ associated with birth.

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[b]PMR2 Adult Manual Resuscitator, Puritan Bennett Corp. Kansas City, Mo.

[c]Blood Gas Manager 1312, Instrumental Laboratory, Lexington, Mass.

[d]VMRD Inc, Pullman, Wash.

[e]Straun CM. *Effects of birth stress in calves*. MS thesis, Department of Animal Science, Iowa State University, Ames, Ia 1996.

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