

## Effects of dystocia on blood gas parameters, acid-base balance and serum lactate concentration in heavy draft newborn foals

Yuki KIMURA<sup>1,2</sup>, Takahiro AOKI<sup>1,2,3\*</sup>, Akiko CHIBA<sup>1</sup> and Yasuo NAMBO<sup>1,2,3</sup>

<sup>1</sup>Department of Applied Veterinary Medicine, Obihiro University of Agriculture and Veterinary Medicine, Hokkaido 080-8555, Japan

<sup>2</sup>United Graduate School of Veterinary Sciences, Gifu University, Gifu 501-1193, Japan

<sup>3</sup>Research Center for Global Agromedicine, Obihiro University of Agriculture and Veterinary Medicine, Hokkaido 080-8555, Japan

---

*Dystocia is often lethal for neonatal foals; however, its clinicopathological features remain largely unknown. We investigated the effect of dystocia on the foal blood profile. Venous blood samples were collected from 35 foals (5 Percheron and 30 crossbreeds between Percheron, Belgian, and Breton heavy draft horses) at 0 hr, 1 hr, 12 hr and 1 day after birth. Dystocia was defined as prolonged labor >30 min with strong fetal traction with or without fetal displacement. The dystocia group (n=13) showed lower mean values for pH (P<0.01), bicarbonate (P<0.01), total carbon dioxide (P<0.05), and base excess (P<0.01) and higher mean values for anion gap (P<0.05) and lactate (P<0.01) immediately after birth than the normal group (n=22). Remarkably high pCO<sub>2</sub> values (>90 mmHg) were observed in three foals in the dystocia group but in none of the foals in the normal birth group immediately after birth. These results suggest that dystocia results in lactic acidosis and may be related to respiratory distress.*

**Key words:** dystocia, heavy draft horse, lactic acidosis, neonatal foal, pH

---

**J. Equine Sci.**  
**Vol. 28, No. 1**  
**pp. 27–30, 2017**

In equine reproduction, dystocia can occur due to factors such as fetal displacement, inadequate size between a dam and a fetus, and uterine torsion [4, 17] and may result in death of the foal if no assistance is provided [7]. Dystocia is more prevalent in heavy draft horses than in light breed horses [4, 17]. Dystocia in horses [5] and prolonged labor in humans [10] and cattle [14] are associated with neonatal acidosis. Prolonged equine labor is associated with fetal asphyxia, hypoxia, and a higher risk of foal mortality; thus, early recognition and assistance are important in cases of dystocia [1, 4]. Stage II of labor (from rupture of the chorio-allantois to completion of delivery) takes less than 2 hr in humans [8] and 2–3 hr in cattle [13]. However, it characteristically takes only 20–30 min in mares [4]. Therefore, some researchers defined dystocia as labor >30 min and reported

that dystocia according to this definition was associated with a higher risk of stillbirth and neonatal morbidity and mortality [12, 15]. On the other hand, sick foals have higher blood lactate (Lac) concentrations than healthy foals [3, 16], and hyperlactatemia is associated with severity of illness and death [3]. Reference values for blood gas parameters, indexes for the acid-base balance in the whole blood and the serum lactate concentration in the newborn foal born by normal birth have not been undetermined. Furthermore, the negative impact of dystocia on the blood profile has remained unknown, particularly in heavy draft horses, which have a high incidence of dystocia. The purpose of this study was to reveal the blood profile of the foal born by normal delivery and to examine the effect of dystocia on blood gas parameters, indexes for the acid-base balance in the whole blood and the serum lactate concentration in heavy draft newborn foals.

Thirty-five foals born between January 2013 and May 2015 at 3 stud farms (Tokachi, Hokkaido, Japan) were included. Their dams were 5 Percherons and 24 crossbreeds between Percheron, Belgian, and Breton heavy draft horses. Six mares gave birth twice during the experimental period. The foals included 5 Percherons and 30 crossbreeds

---

Received: May 6, 2016

Accepted: February 13, 2017

\*Corresponding author. e-mail: aokit@obihiro.ac.jp

©2017 Japanese Society of Equine Science

This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial No Derivatives (by-nc-nd) License. (CC-BY-NC-ND 4.0: <https://creativecommons.org/licenses/by-nc-nd/4.0/>)

**Table 1.** Background information of the normal birth and dystocia groups

	Normal birth (n=22)	Dystocia (n=13)
Duration of stage II of labor (min, mean $\pm$ SD)	21.2 $\pm$ 5.7	45.7 $\pm$ 16.4
Gestation length (days, mean $\pm$ SD)	336.2 $\pm$ 5.3	340.4 $\pm$ 6.9
Age of mare (years, mean $\pm$ SD)	10.4 $\pm$ 4.5	8.5 $\pm$ 4.7
Parity of mare (mean $\pm$ SD)	5.1 $\pm$ 3.7	3.2 $\pm$ 2.9
Breed of dam (n)		
Percheron	5	2
Crossbreed	17	11
Breed of foal (n)		
Percheron	3	2
Crossbreed	19	11
Foal's gender (n)		
Colt	12	5
Filly	10	8
Year foaled (n)		
2013	7	6
2014	10	3
2015	5	4
Month foaled (n)		
January	1	1
February	1	2
March	8	4
April	6	3
May	6	3
Time foaled (n)		
Night (18:00–5:59)	18	9
Daytime (6:00–17:59)	4	4
Farm (n)		
A	2	2
B	10	5
C	10	6

between Percheron, Belgian, and Breton heavy draft horses. Prepartum dams showing signs of foaling were monitored. Foaling events (such as onset of labor pains, rupture of the chorioallantois, appearance of the fetal sac, rupture of the amnion, and delivery of foals) and the duration of stage II were recorded. Cases were excluded from the study if foaling occurred in the absence of witnesses or in the case of abortion, premature birth, or cesarean section. In this study, dystocia was defined as prolonged labor with strong fetal traction with or without fetal displacement. If stage II was >30 min and labor did not progress, traction was applied to the fetus. In the normal group (n=22), stage II labor was <30 min, with spontaneous or assisted delivery with mild traction by 1 or 2 persons. In the dystocia group (n=13), stage II labor was >30 min [15], with strong traction by more than 3 persons or mechanical tools with or without correcting fetal displacement; in this group, 1 foal died 1 day after birth. Background information of both groups is shown in Table 1. Blood was collected from the jugular vein of foals using plain 5 ml vacuum tubes (Venoject II VP-P050K,

Terumo Corporation, Tokyo, Japan) and 5 ml vacuum tubes containing heparin (Venoject II VP-H050K, Terumo Corporation) using 38 mm 21G needles (MN-2138MS, Terumo Corporation). Sampling times were immediately (0 hr), 1 hr (before suckling colostrum), 12 hr, and 1 day (24–48 hr) after birth. The 0 hr sample was collected within 5 min after birth. Blood samples were stored on ice and analyzed within 2 hr. Heparinized whole blood samples were used for blood gas measurements. In each sample, pH, partial pressure of carbon dioxide (pCO<sub>2</sub>), bicarbonate (HCO<sub>3</sub><sup>-</sup>), total carbon dioxide (TCO<sub>2</sub>), base excess (BE), and anion gap (AG) were analyzed using a portable clinical analyzer (i-STAT 300F, Fuso Pharmaceutical Industries, Ltd., Osaka, Japan) and dedicated cartridges (i-STAT cartridge EC8+, Fuso Pharmaceutical Industries, Ltd.). Samples in plain tubes were incubated (37°C, 90 min) and then centrifuged (12 min, 1,000  $\times$  g). Serum was separated and stored at -30°C until assayed. Lac was measured using an automated clinical analyzer (TBA120FR, Toshiba Medical Systems Corporation, Tochigi, Japan). The sequence of postnatal blood data

**Table 2.** Results for blood pH, partial pressure of carbon dioxide (pCO<sub>2</sub>), bicarbonate (HCO<sub>3</sub><sup>-</sup>), total carbon dioxide (TCO<sub>2</sub>), base excess (BE), anion gap (AG), and serum lactate (Lac) concentrations in newborn heavy draft foals within 1 day after birth

	Time after birth				Repeated measures ANOVA		
	0 hr	1 hr	12 hr	1 day	Group	Time	Interaction
pH							
Normal birth	7.25 ± 0.05 **	7.37 ± 0.03 †	7.38 ± 0.05	7.40 ± 0.04	*	**	**
Dystocia	7.18 ± 0.07	7.33 ± 0.08	7.39 ± 0.05	7.40 ± 0.04			
pCO <sub>2</sub> (mmHg)							
Normal birth	70.7 ± 6.9	55.9 ± 5.9	50.8 ± 5.3	51.1 ± 3.1	NS	*	NS
Dystocia	76.9 ± 14.8	57.7 ± 9.7	50.8 ± 5.3	51.4 ± 5.6			
HCO <sub>3</sub> <sup>-</sup> (mmol/l)							
Normal birth	30.8 ± 1.9 **	32.4 ± 2.9 †	29.8 ± 3.2	31.4 ± 2.7	NS	**	**
Dystocia	28.4 ± 2.9	30.4 ± 3.3	30.7 ± 1.7	32.0 ± 2.3			
TCO <sub>2</sub> (mmol/l)							
Normal birth	32.9 ± 2.0 *	34.1 ± 3.0 †	31.7 ± 2.5	32.9 ± 2.7	NS	**	**
Dystocia	30.6 ± 3.4	32.0 ± 3.5	32.2 ± 1.8	33.5 ± 2.4			
Base excess (mmol/l)							
Normal birth	3.6 ± 2.4 **	7.3 ± 3.0 *	5.1 ± 2.8	6.6 ± 3.2	NS	**	**
Dystocia	0.1 ± 2.7	4.6 ± 4.0	5.6 ± 1.9	7.4 ± 2.4			
Anion gap (mmol/l)							
Normal birth	12.7 ± 2.0 *	10.5 ± 3.1	10.8 ± 1.3 *	9.7 ± 2.1	*	**	NS
Dystocia	14.2 ± 2.1	12.2 ± 2.6	12.1 ± 2.1	9.3 ± 1.6			
Lactate (mg/dl)							
Normal birth	75.8 ± 15.7 **	47.0 ± 9.0 †	50.0 ± 16.2 †	37.7 ± 13.6	**	**	**
Dystocia	103.6 ± 20.9 **	67.8 ± 34.4 †	60.5 ± 18.2 †	41.9 ± 10.7			

Normal birth group (n=22). Dystocia group (n=13). Data are shown as the mean ± standard deviation. Statistical significance is denoted by asterisks (\**P*<0.05; \*\**P*<0.01). Statistical tendency is denoted by daggers (†*P*<0.1).

was analyzed by repeated measures analysis of variance (ANOVA). Significant differences between the two groups for each sampling period were determined using Student's or Welch's *t*-test. Results with a *P*-value <0.05 were considered significant, and those with *P*<0.1 were considered to have a tendency. The statistical analyses were conducted using Statcel3 (OMS Ltd., Saitama, Japan).

Results for blood parameters are shown in Table 2. ANOVA revealed statistical main effects by group or interaction in pH, HCO<sub>3</sub><sup>-</sup>, TCO<sub>2</sub>, BE, AG, and Lac. The *t*-tests revealed that the dystocia group showed significantly lower pH (0 hr), HCO<sub>3</sub><sup>-</sup> (0 hr), BE (0 hr, 1 hr), and TCO<sub>2</sub> (0 hr) and higher AG (0 hr, 12 hr) and Lac (0 hr) than the normal group. Although there was no significant difference in mean values of pCO<sub>2</sub> between the both groups, remarkably high pCO<sub>2</sub> values, with the values being more than 90 mmHg, were observed in three foals in the dystocia group but in none of the foals in the normal birth group immediately after birth.

In the dystocia group, the low pH, HCO<sub>3</sub><sup>-</sup> and TCO<sub>2</sub>, and high BE indicated metabolic acidosis, and the high Lac and AG confirmed that it was lactic acidosis [2], thereby indicating that dystocia caused metabolic (lactic) acidosis in the foals immediately after birth. In both human and equine dystocia [6, 18], compression of the fetus and premature

placental separation interferes with fetal and placental perfusion and results in hypoxia. This condition cause anaerobic metabolism and an increase in accumulated Lac [9]. In this study, foals in the dystocia group were likely exposed to these conditions. It has been reported that hyperlactatemia is present in sick foals and increases morbidity [3, 16], which, as indicated by this study, may be caused by dystocia. Strategies for preventing foals from overproducing Lac or developing acidosis are important to maintain their health. In metabolic acidosis with hyperlactatemia following hypoperfusion and hypoxemia, the goal of therapy is correction of the pathogenesis, and fluid therapy (e.g., acetated fluids) is recommended [11]. This treatment in the early stage might be useful for lactic acidosis. Remarkably high pCO<sub>2</sub> values (more than 90 mmHg) were observed in three foals in the dystocia group immediately after birth. Reduced placental perfusion might interfere with placental gas exchange. Decreased pulmonary perfusion results in atelectasis [5], which may increase carbon dioxide. In this study, lactic acidosis, and possibly respiratory distress in foals of the dystocia group, may have been caused by this decreased fetoplacental perfusion and hypoxia. Statistical differences between groups had disappeared before 1 day. The reason for this is unclear; it might have resulted from the fact that most of the cases of dystocia examined in this study were

moderate cases and the fact that the mortality rate was very low in the dystocia group (only one case). Investigation of more severe cases of dystocia will be necessary in a future study.

This study revealed some blood parameters of neonatal foals born by normal delivery and the negative impact of dystocia on blood gas parameters, indexes for acid-base balance in the whole blood and the serum lactate concentration in heavy draft newborn foals. We hope that these fundamental data will contribute to the development of equine neonatology in the future.

## References

1. Byron, C.R., Embertson, R.M., Bernard, W.V., Hance, S.R., Bramlage, L.R., and Hopper, S.A. 2003. Dystocia in a referral hospital setting: approach and results. *Equine Vet. J.* **35**: 82–85. [[Medline](#)] [[CrossRef](#)]
2. Carlson, G.P. 2009 Clinical chemistry tests. pp. 375–397. *In: Large Animal Internal Medicine*, 4th ed. (Smith, B.P. ed.), Mosby, St. Louis.
3. Castagnetti, C., Pirrone, A., Mariella, J., and Mari, G. 2010. Venous blood lactate evaluation in equine neonatal intensive care. *Theriogenology* **73**: 343–357. [[Medline](#)] [[CrossRef](#)]
4. Frazer, G. 2011. Dystocia management. pp. 2479–2496. *In: Equine Reproduction*, Vol. 2, 2nd ed. (McKinnon, A.O., Squires, E.L., Vaala, W.E., and Varner, D.D. eds.), Wiley-Blackwell, Oxford.
5. Galvin, N., and Collins, D. 2004. Perinatal asphyxia syndrome in the foal: review and a case report. *Ir. Vet. J.* **57**: 707–714. [[Medline](#)] [[CrossRef](#)]
6. Gherman, R.B., Chauhan, S., Ouzounian, J.G., Lerner, H., Gonik, B., and Goodwin, T.M. 2006. Shoulder dystocia: the unpreventable obstetric emergency with empiric management guidelines. *Am. J. Obstet. Gynecol.* **195**: 657–672. [[Medline](#)] [[CrossRef](#)]
7. Ginther, O.J., and Williams, D. 1996. On-the-farm incidence and nature of equine dystocias. *J. Equine Vet. Sci.* **16**: 159–164. [[CrossRef](#)]
8. Janni, W., Schiessl, B., Peschers, U., Huber, S., Strobl, B., Hantschmann, P., Uhlmann, N., Dimpfl, T., Rammel, G., and Kainer, F. 2002. The prognostic impact of a prolonged second stage of labor on maternal and fetal outcome. *Acta Obstet. Gynecol. Scand.* **81**: 214–221. [[Medline](#)] [[Cross-Ref](#)]
9. Lagutchik, M.S., Ogilvie, G.K., Wingfield, W.E., and Hackett, T.B. 1996. Lactate kinetics in veterinary critical care: a review. *J. Vet. Emerg. Crit. Care* **6**: 81–95. [[Cross-Ref](#)]
10. Leung, T.Y., Stuart, O., Sahota, D.S., Suen, S.S.H., Lau, T.K., and Lao, T.T. 2011. Head-to-body delivery interval and risk of fetal acidosis and hypoxic ischaemic encephalopathy in shoulder dystocia: a retrospective review. *BJOG* **118**: 474–479. [[Medline](#)] [[CrossRef](#)]
11. Magdesian, K.G. 2009. Critical care and fluid therapy for horses. pp. 1487–1505. *In: Large Animal Internal Medicine*, 4th ed. (Smith, B.P. ed.), Mosby, St. Louis.
12. McCue, P.M., and Ferris, R.A. 2012. Parturition, dystocia and foal survival: a retrospective study of 1047 births. *Equine Vet. J. Suppl.* **44**: 22–25. [[Medline](#)] [[CrossRef](#)]
13. Mee, J.F. 2008. Prevalence and risk factors for dystocia in dairy cattle: a review. *Vet. J.* **176**: 93–101. [[Medline](#)] [[CrossRef](#)]
14. Murray, C.F., and Leslie, K.E. 2013. Newborn calf vitality: risk factors, characteristics, assessment, resulting outcomes and strategies for improvement. *Vet. J.* **198**: 322–328. [[Medline](#)] [[CrossRef](#)]
15. Norton, J.L., Dallap, B.L., Johnston, J.K., Palmer, J.E., Sertich, P.L., Boston, R., and Wilkins, P.A. 2007. Retrospective study of dystocia in mares at a referral hospital. *Equine Vet. J.* **39**: 37–41. [[Medline](#)] [[CrossRef](#)]
16. Pirrone, A., Mariella, J., Gentilini, F., and Castagnetti, C. 2012. Amniotic fluid and blood lactate concentrations in mares and foals in the early postpartum period. *Theriogenology* **78**: 1182–1189. [[Medline](#)] [[CrossRef](#)]
17. Vandeplasseche, M. 1993. Dystocia. pp. 578–587. *In: Equine Reproduction* (McKinnon, A.O., and Voss, J.L. eds.), Lea & Febiger, Philadelphia.
18. Wilkins, P.A. 2011. Perinatal asphyxia syndrome. pp. 147–153. *In: Equine Reproduction*, Vol. 1, 2nd ed. (McKinnon, A.O., Squires, E.L., Vaala, W.E., and Varner, D.D. eds.), Wiley-Blackwell, Oxford.