



## Comparison of Apgar score, serum lactate, and blood gas analysis in neonates born by elective cesarean section or cesarean section after dystocia

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**ABSTRACT:** *In some situations, the neonatal mortality rate in dogs can be high, and perinatal and postnatal veterinary care is essential to improve survival. This study to compared the Apgar score, serum lactate levels, and blood gas analysis results in 30 neonates born by elective cesarean section (GCE, n = 18) or cesarean section due to dystocia (GD, n = 12). Neonates were assessed at five time points: T0, at birth; T1, 3 h after birth; T2, 6 h after birth; T3, 12 h after birth; and T4, 24 h after birth. At T0, in the GCE group, 55.6% of the animals presented with adequate vitality, while 38.8% showed moderate changes in the Apgar score. In the GD group, 83.3% of the neonates had severe loss of vitality. At T1, none of the GCE neonates and 8.3% of the GD neonates presented with vitality deficits. Hyperlactatemia was observed in 83.3% of the GD neonates, and mixed acidosis (metabolic and respiratory) was observed at T0 in most neonates in both groups (GCE, 55.6%; GD, 72.7%). Compared to GCE neonates, the acid-base disorders were more severe and their recovery slower in GD neonates. At T0, GD neonates had higher hyperlactatemia and a lower Apgar score (worse vitality) than those of GCE neonates.*

**Key words:** neonates, Apgar score, lactate, cesarean section, dog.

## Comparação do escore Apgar, lactato sérico e hemogasometria em neonatos nascidos por cesariana eletiva e cesariana após distocia

**RESUMO:** *A taxa de mortalidade neonatal em cães pode ser elevada em algumas situações e a assistência veterinária peri e pós-natal mostra-se essencial para melhorar a sobrevivência dos filhotes nesse período. O objetivo deste estudo foi comparar o escore Apgar, lactato sérico e hemogasometria em neonatos nascidos por cesariana eletiva (GCE, n=18) e por cesariana após distocia (GD, n=12). Os neonatos foram avaliados em cinco momentos: (T0) ao nascimento; (T1) três horas; (T2) seis horas; (T3) 12 horas; e (T4) 24 horas. Ao nascimento, no GCE, 55,6% dos animais apresentaram boa vitalidade, de acordo com escore Apgar, e 38,8% demonstraram moderada vitalidade. No GD, 83,3% dos neonatos apresentaram baixa vitalidade. Ao T1 (três horas após o nascimento), nenhum (0%) dos neonatos do GCE e 8,3% dos neonatos do GD evidenciavam perda de vitalidade. Observou-se hiperlactatemia em 83,3% nos neonatos do GD e acidose mista (metabólica e respiratória) ao nascimento na maioria dos neonatos de ambos os grupos (55,6% no GCE e 72,7% no GD). Conclui-se que o principal distúrbio ácido-base observado ao nascimento foi acidose mista (metabólica e respiratória) em ambos os grupos. Os distúrbios ácido-base nos neonatos do GD foram mais graves e sua recuperação mais lenta quando comparados com neonatos do GCE. Ao nascimento, neonatos no GD apresentaram maior hiperlactatemia e menor escore Apgar (pior vitalidade) em relação aos nascidos no GCE.*

**Palavras-chave:** neonatos, escore Apgar, lactato, cesariana, cão.

## INTRODUCTION

Dogs can reach a neonatal mortality rate of 30% in the first week of life, often because of failures in the evaluation or clinical management of neonates (SILVA et al., 2008). Neonatal monitoring and provision of specialized care immediately after birth are instrumental in reducing this high mortality rate (DOMINGOS et al., 2008; VERONESI et al., 2009). At birth, the transition from the intrauterine

environment, which is liquid, closed, and has a stable temperature and adequate nutrition and gas exchange, to the extrauterine environment, which is dry, open, and has periods of fasting and variable temperature, generates circulatory, respiratory, metabolic, and neurological changes in neonates. This requires rapid and profound physiological adaptation to ensure survival (DOMINGOS et al., 2008).

The Apgar score, developed in 1952 to evaluate human neonates, is based on parameters that

monitor the main vital functions and determine the necessary medical interventions. It was introduced in Veterinary Medicine and has been modified and adapted for several animal species (HOGAN et al., 2007). The modified Apgar score for canine neonates estimates their vitality by evaluating the heart rate, respiratory effort, reflex irritability, muscle tone, and mucosal coloration (VERONESI et al., 2009).

Lung maturity is essential for proper gas exchange in the period of adaptation to extrauterine life. From birth to the time of spontaneous pulmonary breathing, neonates undergo a transient period of asphyxia and acidosis caused by increased carbon dioxide pressure ( $PCO_2$ ). Acid–base balance changes are usually normalized within 48 h after birth (LAWLER, 2008).

In addition to the Apgar score and blood gas analysis, the lactate analysis is useful to assess the severity and prognosis of neonatal disorders in addition to being a good marker of blood perfusion and respiratory changes (LOURENÇO & MACHADO, 2013). During labor, fetal lactate rises because of anaerobic metabolism resulting from transient hypoxia (NORDSTRÖM et al., 2001). Chou et al. (1998) showed that high lactate levels associated with hypoxia at delivery are a valid indicator of the neonatal prognosis.

This study evaluated and compared the Apgar score, serum lactate levels, and blood gas analysis results in neonates born by elective cesarean section (ECS) or cesarean section after dystocia (CSD).

## MATERIALS AND METHODS

Thirty neonates from the clinical/surgical routine at the UFMG Veterinary Hospital were enrolled. Of these, 18 were born by ECS and 12 by CSD. We included thirteen pregnant bitches without clinical or laboratory abnormalities and one to three puppies per pregnant bitch. The bitches undergoing ECS ( $n = 6$ ) were monitored in the third trimester of pregnancy by ultrasonography and radiography to determine the number of fetuses and by electrochemiluminescence to determine serum progesterone doses. The timing of ECS was determined by complete fetal organogenesis, the presence of peristaltic bowel movements, and a progesterone dose  $<2$  ng/mL (FORSBERG, 2010). In the CSD group ( $n = 7$ ), the bitches underwent clinical obstetric examination, ultrasound to confirm dystocia, and radiography to determine the number of fetuses. In addition to the ultrasound diagnosis, the clinical criteria used to determine dystocia were the

presence of green, red, or brown vaginal discharge without fetal expulsion for 2–4 h; fluid expulsion due to ruptured membranes for 2–3 h; weakened parturient without contractions for more than 2–4 h; parturient with strong and regular contractions for more than 20–30 min without fetal expulsion; and other evidence of dystocia, such as the fetus visible in the birth canal. Once dystocia was confirmed, bitches with no more than 24 h of labor and the presence of at least one live fetus in utero were included in the study. Since the study was based on clinical routine and the total number of animals per litter varied, the selection criterion for neonates was birth order: first, intermediate, and last born.

The anesthetic protocol for the cesarean section in both groups was preanesthetic medication with tramadol hydrochloride (Cloridrato de Tramadol, União Química Farmacêutica Nacional S/A, Jabaquara, SP, Brazil); induction with propofol 1% (Fresofol 1%, Fresenius Kabi Brasil Ltda, Campinas, SP, Brazil); maintenance with isoflurane (Isothane 100 mL, Baxter Hospitalar Ltda, Santo Amaro, SP, Brazil) in a semi-closed circular system, 100% oxygen, with a minimum alveolar concentration of 1.4; and epidural anesthesia with lidocaine 2% without vasoconstrictors (Xylestesin 2%, Cristália, São Paulo, SP, Brazil).

Immediately after birth, the neonates were assessed for the heart rate, respiratory effort, reflex irritability, muscle tone, and mucosal coloration (VERONESI et al., 2009). All Apgar parameters were scored from 0 to 2 (Table 1). According to the sum of the scores, the neonates were classified in the following categories: good vitality, 7–10; moderate vitality, 4–6; and low vitality, 0–3. The evaluations were performed by the same professional at five pre-established time points: T0, at birth; T1, 3 h after birth; T2, 6 h after birth; T3, 12 h after birth; and T4, 24 h after birth.

The blood gas analysis results and lactate dose were measured using 0.1 mL of whole blood collected from the neonates via a jugular venipuncture. Three drops of blood were placed in the blood gas analysis cartridge to evaluate the pH,  $PCO_2$ ,  $HCO_3^-$  concentration, and base excess/deficit (BE), and one drop of blood was placed in the lactate analyzer (Accutrent Plus, Roche Diagnostics Systems, Portugal). Blood gas was processed in the portable blood gas analyzer I-Stat (Abbott, São Paulo, Brazil). Metabolic acidosis was determined by the Henderson–Hasselbalch method using the reference values established by DIBARTOLA (2012) and HOPPER, et al. (2014) as follows: pH, 7.35–7.45;

Table 1 - Apgar score evaluation parameters modified for canine neonates and their respective values (VERONESI, 2009).

Score	2	1	0
Heart rate	> 220 bpm	220–180 bpm	< 180 bpm
Breathing effort	> 15 mpm and	15–6 mpm and	< 6 mpm and
Vocalization	Loud cry	Weak cry	No cry
Reflex irritability	Rapid limb withdrawal	Weak limb withdrawal	No limb withdrawal
Muscle tonus	Active movements	Some flexions	Flaccidity
Mucosal coloration	Pink	Pale	Cyanotic

Scores: 7–10, good vitality; 4–6, moderate vitality; 0–3, low vitality.

PCO<sub>2</sub>, 35–45 mmHg; HCO<sub>3</sub><sup>-</sup>, 18–26 mmol/L; and BE, 4 to -4 mmol/L.

The neonates that breathed spontaneously (10–18 mpm) and had a heart rate above 120–150 bpm and rosy mucous membranes were considered to be stable. Neonates with low (80–100 bpm) or absent heart rates were resuscitated with external cardiac massage, oxygen therapy, and tactile chest respiratory stimulation. All neonates were monitored in an incubator (Fanem, São Paulo, Brazil) with their mothers during the first 3 h postoperatively under controlled temperature (29.5 °C–32.5 °C), humidity (55%–65%), and available oxygen support.

A descriptive statistical analysis of the clinical and blood gas variables of the neonates was performed. Nonparametric tests were performed for non-normally distributed variables. The nonparametric Mann–Whitney U test measured differences in variables between the two groups. The Friedman nonparametric test measured differences among evaluation times (T0–T4). The chi-square test was used to test frequency differences in qualitative variables between the groups. The Pearson correlation was used for the variables measured in the neonates to identify those that were highly correlated and those that were redundant in the analysis. Thus, these variables could be correlated with the vitality of neonates. A P-value < 0.05 was considered to be statistically significant.

## RESULTS AND DISCUSSION

### Apgar score

The Apgar score at T0 differed between groups (P < 0.001). In the ECS and CSD groups, 94.4% and 83.3% of the neonates had moderate to good vitality (ECS: 55.6% good vitality and 38.8% moderate vitality), respectively, showing a significant

difference (Table 2). These data corroborate those reported by GROPPETTI et al. (2010), who reported a low Apgar score in all neonates born by CSD. This difference between groups at T0 can be explained by the time the neonates remained in the uterus and vaginal canal in the CSD group, under the influence of contractions and with little oxygenation, which, associated with the effects of anesthetics, could aggravate fetal distress and lead to more pronounced depression and hypoxia, compromising neonate vitality soon after birth (VASSALO et al., 2015). Unlike the present study, SILVA et al. (2009) and BATISTA et al. (2014) reported low vitality in neonates born by ECS and considered anesthesia as a relevant factor affecting neonatal vigor after birth. However, in the present study, since both groups were subjected to the same anesthetic protocol, the reduced neonate vitality observed with CSD reflect worsening the initial conditions of hypoxia and depression at birth associated with dystocia, suggesting no influence of anesthesia on vitality at birth.

The Apgar score improved in both groups at T1. All ECS neonates and 91.7% of the CSD neonates recovered from the cesarean section and presented with good vitality (Table 2). Thus, the Apgar score at T0 differed from that at T1, T2, T3, or T4 in both groups. VASSALO et al. (2015) reported similar recovery in neonatal vitality 1 h after ECS.

### Lactate levels and Apgar score

The Apgar score showed a negative correlation with serum lactate levels at T0 (r = -0.57; P < 0.001), indicating low neonatal vitality at birth. This condition was also observed by CASTAGNETTI et al. (2017). Table 3 summarizes blood lactate levels in ECS and CSD neonates at each assessment time (T0–T4). Lactate levels were within the normal range for

Table 2 - Apgar scores of neonates at different time points after birth by elective cesarean section and cesarian section after dystocia.

		T0	T1	T2	T3	T4
N (%)		18 (100%)	18 (100%)	18 (100%)	18 (100%)	18 (100%)
Elective cesarean section (n = 18)	Low vitality (0–3 pts)	1 (5.6%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
	Moderate vitality (4–6 pts)	7 (38.8%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
	Good vitality (> 7 pts)	10 (55.6%)	18 (100%)	18 (100%)	18 (100%)	18 (100%)
N (%)		12 (100%)	12 (100%)	12 (100%)	12 (100%)	12 (100%)
Cesarean section after dystocia (n = 12)	Low vitality (0–3 pts)	10 (83.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
	Moderate vitality (4–6 pts)	2 (16.7%)	1 (8.3%)	0 (0%)	0 (0%)	0 (0%)
	Good vitality (> 7 pts)	0 (0%)	11 (91.7%)	12 (100%)	12 (100%)	12 (100%)
P-value		<0.001*	0.219	0.061	0.061	0.519

\* A P-value <0.001 denotes statistical significance. Data are expressed as median (Q1; Q3).

canine neonates (1.07–6.59 mmol/L; MCMICHAEL et al., 2005) in both groups at all assessment times (T0–T4) except at T0 in the CSD group (Table 3). In the CSD group, elevated lactate at T0 suggested progressive recovery of the neonates, and this parameter remained within the normal range until T4 (Table 3). These results contrast with those obtained by CASTAGNETTI et al. (2017), who reported elevated serum lactate ( $7.0 \pm 3.4$  mmol/L) in ECS neonates at birth compared to the other time points (2 h:  $3.1 \pm 2.3$  mmol/L; 12 h:  $2.9 \pm 2.9$  mmol/L; and 24 h:  $2.1 \pm 1.1$  mmol/L;  $P < 0.0001$ ). The neonates progressively recovered, and 24 h after birth, lactate levels were within the normal range, as in the present study. VASSALO et al. (2015) reported high lactate levels at birth ( $7.1 \pm 0.4$  mmol/L) in CSD neonates, which corroborated the results of this study. This condition is associated with the fetal length of stay in the uterus and vaginal canal after placental separation under the influence of uterine contractions and with occluded placental vessels, which compromise fetal oxygenation (GROPETTI et al., 2010; VASSALO et al., 2015).

Regarding the comparison among different types of delivery (cesarean section due to eutocia, ECS, and CSD), GROPETTI et al. (2010) reported higher serum lactate levels in CSD ( $8.3 \pm 5.9$  mmol/L) compared to ECS ( $3.5 \pm 1.2$  mmol/L), corroborating our study. Others studies evaluated neonates at the time of birth by ECS and reported a lactate level of 4.76 Mmol/L, close to that found in the present study in ECS neonates (median = 4.5 mmol/L [Q1: 3, 9; Q3: 5, 9]). CSD neonates had higher lactate levels and worse Apgar scores at T0 (Table 2). This negative correlation between Apgar score and high

lactate levels was also described by VASSALO et al. (2015), and CASTAGNETTI et al. (2017).

#### *pH and Apgar score*

Blood pH showed a strong positive correlation with the Apgar score ( $r = 0.83$ ;  $P < 0.001$ ), which corroborates VASSALO et al. (2015). Thus, the higher the pH, reflecting lower acidemia and values closer to the physiological normal, the higher the Apgar score, reflecting neonatal vitality.

#### *PCO<sub>2</sub> and Apgar score*

The Apgar score showed a strong negative correlation with PCO<sub>2</sub> ( $r = -0.84$ ;  $P < 0.001$ ), suggesting that increased PCO<sub>2</sub> is associated with low neonatal vitality at birth and; therefore, with lower Apgar scores. Thus, changes in the acid–base balance could significantly influence the Apgar score.

#### *Lactate levels, pH, and PCO<sub>2</sub>*

Lactate levels showed a moderate negative correlation with serum pH ( $r = -0.63$ ;  $P < 0.001$ ), suggesting that lactate elevation favors acidemia, as reported by VASSALO et al. (2015). In addition, lactate levels showed a moderate positive correlation with PCO<sub>2</sub> ( $r = 0.61$ ;  $P < 0.001$ ), suggesting that increased PCO<sub>2</sub> may result in hyperlactatemia.

#### *Acid–base disorders*

In both groups, the most frequent acid–base disorders observed at T0 were a mixed acid–base disorder (55.6% in ECS and 72.7% in CSD) and respiratory acidosis (44.4% in ECS and 27.3% in CSD; Figure 1). These disorders can be justified by the transient period of asphyxia that all neonates

Table 3 - Serum lactate levels at different time points after birth by elective cesarean section and cesarean section after dystocia.

		-----Lactate Mmol/L-----					
	p	N	T0	T3	T6	T12	T24
Elective cesarean section	0.126	18	4.5 (3.9;6.0) Aa	4.3 (3.8; 4.8) Aa	4.3 (3.6; 4.6) Aa	3.7(3.1; 4.5) Aa	4.1(2.9; 5.1) Aa
Cesarean section after dystocia	< 0.001	12	9.9 (7.2;11.4) Ab	4.6(3; 6.4) Aba	4.2 (3.3; 5.1) Ba	3.8 (2.7; 5.2) Ba	3.5 (2.4; 4.8) Ba
P-value			<0.001	0.582	0.916	0.539	0.340

A P-value <0.05 denotes statistical significance. Data are expressed as median (Q1; Q3).

Values followed by different capital letters in the same row show a significant difference ( $P < 0.05$ ) between evaluation times according to the Friedman test. Different lowercase letters in each column indicate a significant difference between groups by the Mann–Whitney U test.

undergo at birth, originating from hypercarbia, hypoxia, increased anaerobic metabolism, lactic acid production, and acidosis (respiratory and/or metabolic; CRISSIUMA et al., 2010; VASSALO et al., 2015). Mixed acidosis, regardless of the type of delivery, is as a frequent acid–base neonatal disorder at birth (LAWLER, 2008; CRISSIUMA et al., 2010; VASSALO et al., 2015). It was observed in the ECS group at all times except T4 and in the CSD group at T0 and T1 (Figure 1). Both groups had a significantly lower number of neonates with mixed acidosis at T1. After birth, the neonates were kept in an incubator under controlled temperature, humidity, and oxygen source and were stimulated to suckle. Thus, favorable conditions were created for maintaining hydration and increasing the body temperature by decreasing lactic acidosis and optimizing the metabolic parameters of mixed acidosis. However, increased neonatal respiratory acidosis was recorded in both groups at T1. This change may reflect adaptation of the neonate to extrauterine life (CRISSIUMA et al., 2010; VASSALO et al., 2015).

Neonatal metabolic acidosis was observed at T1 and T3 in the ECS group (Figure 1). Neonates are susceptible to minor temperature changes and may dehydrate easily if not adequately fed, possibly resulting in metabolic acidosis at these assessment times.

A moderate mixed acid–base disorder, characterized by respiratory acidosis and metabolic alkalosis, also occurred in both groups at T1, T2, T3, and T4 (Figure 1). Metabolic alkalosis was mainly observed in the CSD group at T2, T3, and T4 and in the ECS group at T4.

Metabolic alkalosis may result from the compensatory response to respiratory acidosis presented by neonates since birth. Bicarbonate

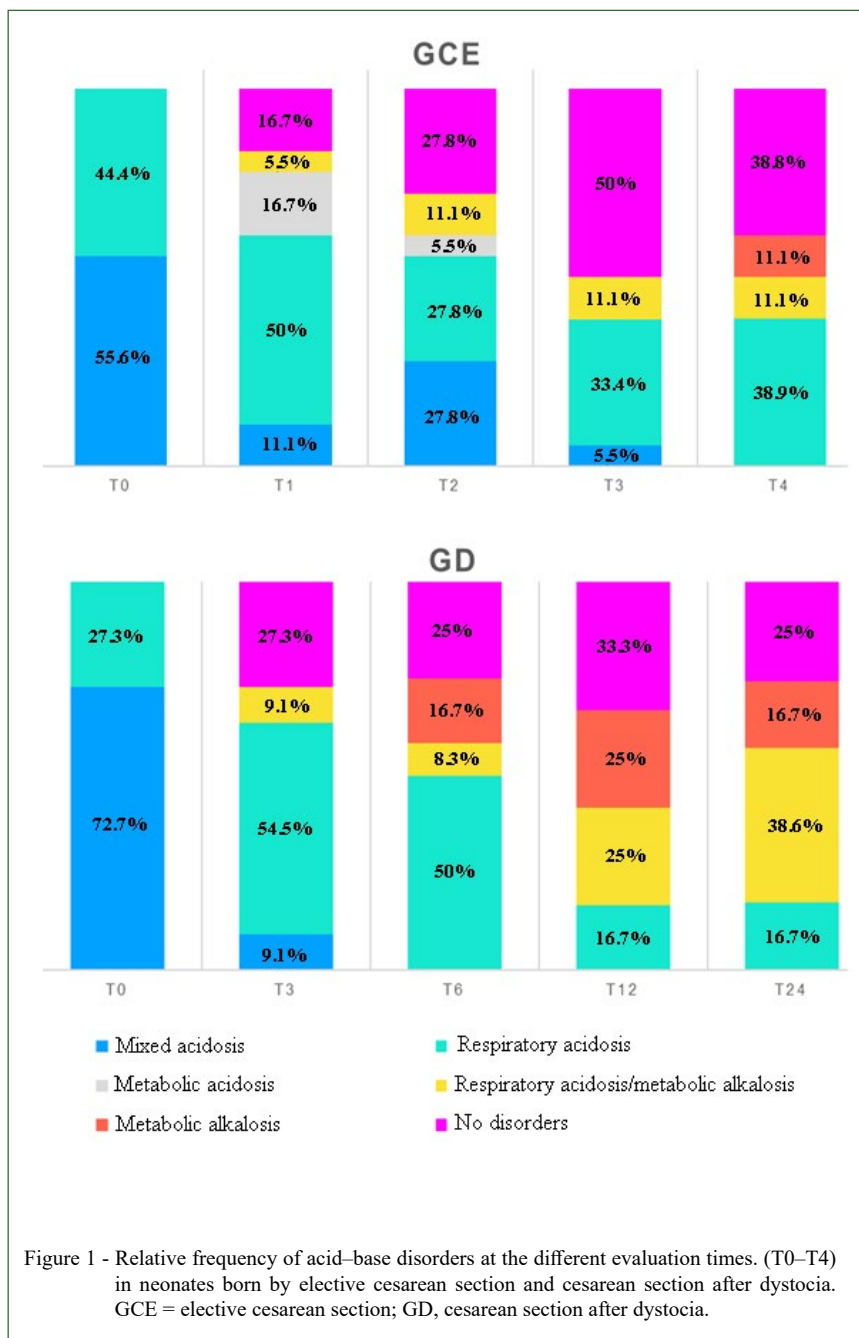
concentrations are expected to change as a secondary response (DIBARTOLA, 2012). The CSD group had a higher number of animals with the mixed disorder (respiratory acidosis and metabolic alkalosis) and metabolic alkalosis, which may be due to increased  $PCO_2$  compared to the ECS group, suggesting a slower recovery of the general condition in the CSD group.

Changes in the acid–base balance in the transient period of asphyxia at all assessment times reflect circulatory, metabolic, respiratory, and neurological adaptations resulting from abrupt neonatal changes between the intra- and extrauterine environments (DOMINGOS et al., 2008). At T4, 38.8% of the ECS neonates and 25% of the CSD neonates no longer presented with acid–base imbalance. LAWLER (2008) highlighted that these changes are corrected within 48 h after birth.

ECS neonates recovered faster compared to CSD neonates, which can be explained by the longer time CSD neonates took to be born, possibly causing more pronounced depression at birth (GROPETTI et al., 2010; VASSALO et al., 2015). ECS neonates did not develop dystocia, which helped maintain their vitality with higher oxygen levels and lower hypoxia and acidosis (VASSALO et al., 2015).

## CONCLUSION

In the present study, dystocia caused lower vitality at birth, with canine neonates showing a low Apgar score and hyperlactatemia but quick recovery. The main acid–base disorder at birth was mixed acidosis (metabolic and respiratory) in both ECS and CSD groups. Acid–base disorders were more severe in CSD neonates, with slower recovery compared to ECS neonates.



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## DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflict of interest. The founding sponsors had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, and in the decision to publish the results.

## AUTHORS'S CONTRIBUTIONS

All authors contributed equally to the manuscript.

## BIOETHICS AND BIOSSECURITY COMMITTEE APPROVAL

This study was approved by the Animal Ethics Committee (AEC/UFGM) under protocol no. 461/2015.

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