



Metabolic status in canine neonates – importance for survival

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INTRODUCTION

The vast majority of mortality in puppies occurs during the first 3 weeks after birth with hypoxia, hypothermia and hypoglycemia as the main non-infectious causes of neonatal death (1). Early diagnostics of pathology thanks to reliable methods of examination would allow to adapt nursing and treatment of puppies at risk.

The aim of this study was to evaluate metabolic status in canine neonates at birth and at 24 hours of life, and to determine their impact on neonatal mortality.



Photo 1. Weight measurement of the newborn puppy.



Photo 2. Assesment of the color of mucosa fo APGAR scoring.



Photo 3. Aspiration of blood by a disposable strip for home monitoring device for diabetics.



Photo 4. Urine collection by stimulation of urogenital area with a cotton gauze to measure urine density.

MATERIALS AND METHODS

- Within one breeding kennel, 347 puppies from small (141), medium (72) and large breeds (134) were included.
- Within the first 8 hours after birth, the first clinical examination was performed including evaluation of the APGAR score (Table 1) (2), weight, and urinary density (refractometry), blood glucose, β-hydroxybutyrate (β-ketone) (FreeStyle Optium, Abbott, Illinois, USA) and lactate (Lactate Pro, Arkray, Kyoto, Japan) measurements (Photo 1-4). Birth weight was categorized in quartiles depending on breed size. The second clinical examination, including the same health parameters, except APGAR score and weight, was performed 24 hours later.
- The mortality between birth and 21 days of age was recorded. The effects of parameters, measured at birth (<8h) and at 24h,
 on mortality with litter as a random term were tested with generalized linear mixed models (SAS, Cary, N.C., USA).



Table 1. APGAR scoring system for newborn puppies adapted from Veronesi et al. 2009 (bpm=beats per minute, rr=respiratory rate).

RESULTS - Metabolic status

Birth weight differed depending on breed size and APGAR score tended to (Fig. 1-2). Glucose, lactate and β -ketone concentrations, and urinary density differed depending on age (p<0.01 for all 4 tests) and breed size (Fig. 3-6).



Fig. 1. Proportion of puppies with different APGAR score at birth depending on breed size (p=0.09, n=347).

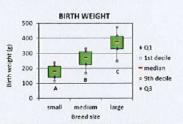


Fig. 2. Box and whisker plot of birth weight in small, medium and large breed sized dogs (p<0.001, n=347).*

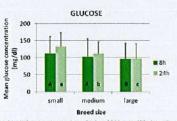


Fig. 3. Mean glucose concentration at birth and 24 hours of life depending on breed size (n=335, p=0.01; n= 322, p<0.001, respectively).*

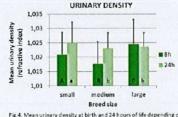
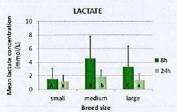


Fig. 4. Mean urinary density at birth and 24 hours of life depending on breed size (n=261, p<0.001; n=325, p=0.03, respectively).*



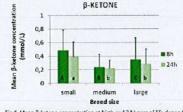


Fig. 5. Mean lactate concentration at birth and 24 hours of life depending on breed size (n=273, p<0.001; n=309, p<0.001, respectively).*

on breed size (n=241, p<0.001; n=308, p<0.001, respectively).*

RESULTS - Mortality

Among 347 included puppies, 70 died between birth and 21 days of age (20.2%). The risk of mortality was influenced by birth weight (p=0.007) and glucose concentration at 24h (p<0.001). Significantly more puppies with low birth weight (the lightest 25%) died until Day 21 compared with puppies with greater birth weights (Fig. 7).



Fig. 7. Proportion of puppies dying between birth and 21 days of age depending on birth weight (n=347, p<0.001).

The higher the glucose concentration was, the lower was the mortality in puppies (Fig.8). Among puppies for which glucose concentration decreased between birth and 24 hours of life, 13.4% (18/134) died before Day21 vs. 2.7% (5/183) of puppies for which glucose has increased (p=0.003).

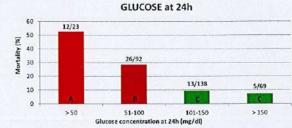


Fig.8. Proportion of puppies dying between 1 and 21 days of age depending on glucose concentration at 24 hours of life (n=322, p<0.001)

Neither blood lactate and β -ketone concentrations nor urinary density have any influence on mortality. The effect of the litter (random term) was significant for all models tested (p<0.001).

DISCUSSION & CONCLUSIONS

Among all parameters tested at birth and 24 hours of life, only birth weight and glucose concentration at Day1 indicated puppies at risk of death until 21 days of age. This study demonstrates that energy supply within the first days of life, especially in puppies with low birth weight, is crucial for survival in canine neonates. Therefore, glucose monitoring and eventual energy booster administration could help to decrease the neonatal mortality rates in breeding kennels.

^{*} Mean values with different upper case letters were significantly different (p < 0.05) at 8h and with different lower case letters were significantly different at 24h of age.

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Introduction and aim. The vast majority of mortality in puppies occurs before weaning. Reliable early predictors of mortality would allow to adapt nursing and treatment of puppies at risk. The aim of this study was to evaluate metabolic status in canine neonates at birth and at 24 hours of life, and to determine their impact on neonatal mortality.

Material and methods. Within one breeding kennel, 347 puppies from small breeds (S; n=141), medium breeds (M; n=72) and large breeds (L; n=134) were included. Within the first 8 hours after birth, the first clinical examination was performed including evaluation of the Apgar score (AS) (0-10 points) (1), weighing, and urinary density (refractometry), blood glucose, β-ketone (FreeStyle Optium, Abbott, Illinois, USA) and lactate (Lactate Pro, Arkray, Kyoto, Japan) measurements. Birth weight was categorized in quartiles depending on breed size. The second clinical examination, including the same health parameters, except AS and weighing, was performed 24 hours later. The mortality between birth and 21 days of age was recorded. The effects of parameters measured at birth, at Day1 and their difference on mortality with litter as a random term were tested with generalized linear mixed models (SAS, Cary, N.C., USA). Data are presented as mean \pm SD.

Results. At birth, average AS was 9.0 ±1.5, glucose 104 ±49 mg/dl, lactate 2.9 ±2.9 mmol/L, βketone 0.4 \pm 0.3 mmol/L, urinary density 1.022 \pm 0.009 and birth weight 177 \pm 45g, 260 \pm 66g and 369 ±87g for S, M and L breeds, respectively. At 24 hours of life, average glucose concentration was 114 ±43 mg/dl, lactate 1.4 ±1.0 mmol/L, β-ketone 0.3 ±0.2 mmol/L and urinary density 1.024 ±0.006. Among the 347 puppies included to the study, 70 puppies died between birth and 21 days of age (20.2%). The risk of mortality was influenced by birth weight (p=0.007) and glucose concentration at Day1 (p<0.001). 36.2% (25/69) of puppies with low birth weight (the lowest 25% among the breed size) died until Day 21 compared with 15.3% (39/254) of puppies with greater birth weights. Glucose concentration at Dayl in puppies surviving until Day21 was 120 ±40 mg/dl vs. 85 ±49 mg/dl in puppies dying before the end of the experiment. 52.3% (12/23) of puppies with glucose concentration at Day1 below 50 mg/dl died before Day21 vs. 28.3% (26/92) when glucose was between 51-100 mg/dl and 8.7% (18/207) when glucose was above 100 mg/dl. Among puppies for which glucose concentration decreased between birth and 24 hours of life, 13.4% (18/134) died before Day 21 vs. 2.7% (5/183) of puppies, for which glucose has increased (p=0.003). Neither blood lactate nor β-ketone concentrations have any influence on mortality. The effect of the litter (random term) was significant for all models tested.

Conclusions. Among all parameters tested at birth and 24 hours of life only birth weight and glucose concentration measured at Day1 indicated puppies at risk of death between birth and 21 days after birth. This study demonstrates that energy supply within the first days of life, especially in puppies with low birth weight, is crucial for survival in canine neonates. Therefore, glucose monitoring and eventual energy booster administration could help to decrease the neonatal mortality rates in breeding kennels.

References. 1) Veronesi et al., Theriogenology 2009; 72:401-407.













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