ABSTRACTS

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Prenatal and neonatal adaptations with a focus on the respiratory system

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The neonatal period demands extreme adaptive processes for an organism to endure extra-uterine life, in a continuous maturation progress commenced in utero. Among all modifications, pulmonary development is the most threatening, for lungs have to perform adequate gas exchange, which was previously of placental responsibility. Physiological or induced fetal pulmonary changes are of utmost importance to reduce neonatal mortality and morbidity. Furthermore, the precise diagnosis of lung failure during the initial moments of life determines the correct therapy institution. The Neonatal Respiratory Distress Syndrome is defined as the insufficiency of surfactant production or pulmonary structural immaturity, specially related to preterm newborns. In Human Medicine, the most successful manner to avoid such syndrome is the antenatal corticotherapy, in an attempt to induce surfactant production in loco.

During the final stage of the canine pregnancy, fetal lung progressively evolves to a sacular stage of development, characterized by an increase in gas exchange area and initial production of surfactant (1). Therefore, it may be inferred that artificial induction of surfactant synthesis could be achieved from the 55th day of pregnancy onwards. However, maternal prenatal betamethasone treatment of bitches at 55 days of gestation leads to structural changes of neonatal lung parenchyma and consequently an improvement in preterm neonatal respiratory condition, but not an increase in pulmonary surfactant production (2). Hence, the administration of maternal betamethasone in the prenatal stage enhances lung oxygen absorption by increasing gas exchange area, thus favoring the compensatory mechanisms of acid-base imbalance in preterm newborns. Parturition represents an important challenge to neonatal adaptation, for uterine and abdominal contractions during labor provoke intermittent hypoxia.

Consequently, the onset of pulmonary respiration has to effectively overdue low oxygen saturation and lung amniotic fluid content. In a radiographic survey, Silva et al. (3) verified that 17% of eutocic delivered puppies show moderate to severe lung alterations, especially represented by diffuse or restricted opacification of pulmonary parenchyma consistent with fluid collection. Immediately after birth, puppies present venous mixed acidosis (low blood pH and high dioxide carbon saturation), as well as low, but satisfactory, Apgar score of vitality (4). At birth, 75% of canine neonates show altered respiratory pattern and 30% present cyanosis (3). Thus, the physiological hypoxia during birth, coupled with the initial effortfulness to brim pulmonary alveoli with oxygen, give rise to an anaerobiosis condition. The major sign that corroborates birth as a stressful event is that puppies are born with high cortisol concentration. However, it is believed that hypoxia and stress during parturition trigger neonatal respiratory movement and effort. Indeed, as a neonatal adaptation follow-up, the Apgar analysis indicates a tachypnea response after one hour of life, which leads to a shift in blood acid-base status to metabolic acidosis. A one hour period is sufficient for canine neonates to achieve an ideal Apgar score; notwithstanding, a hemogasometric imbalance still persists.

In regards to the thermoregulatory adaptation, canine neonates present hypothermia earlier at 5 minutes of life, with a progressively decrease in body temperature after one hour (4). Thermal homeostasis is one of the critical points in canine neonatology that deserves special medical attention, as low body temperature delays overall adaptive mechanisms, such as gasometric and metabolic processes. Another important factor that may retard neonatal adaptation is the obstetrical condition or the form of parturition. In this respect, dystocia promotes long lasting bradychardia effect, slows down Apgar score progression, and aggravates metabolic acidosis and stress (4). Moreover, the parenteral administration of ocitocin to atonic/hypotonic labor has a neonatal hyperglycemiceffect, as well as a serious deleterious effect on the metabolic component of blood acidosis (4). The latest data reinforces the need to accurately intervene during canine parturition and offer adequate medical treatment to puppies derived from pathological labor. Neonates have to be considered a special patient that requires specific approach. On the other hand, canine neonatology is a currently developing area of Veterinary Medicine that demands scientific knowledge in order to assure a systematic manner to assist newborns.


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