

**Popular Article**

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**Normal Parturition In Dogs- A Review**

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**Introduction**

Understanding the neuroendocrine cascade of parturition assists the clinician in managing the dam and understanding the etiology of dystocia. The accepted neuroendocrine model of canine parturition is initiated by the fetus. The fetal hypothalamic-pituitary-adrenal axis is activated by fetal stress and leads to the secretion of fetal glucocorticoids. This increased glucocorticoid concentration stimulates maternal estrogen production, contributes to the synthesis and release of prostaglandins, and increases oxytocin receptors on the myometrium. Prostaglandins are luteolytic, contribute to the decline in circulating progesterone, remove the inhibition of myometrial contractility, and mediate the effects of oxytocin on the uterus. Maternal oxytocin is initially released from the hypothalamus in response to afferent stimulation of pressure receptors within the cervix and vagina. Relaxin hormone, produced by the ovary and placenta, assists fetal passage by allowing the interpubic ligament to elongate and the pubic bones to separate. The prolactin (lactation hormone) level, which increases gradually during gestation starting 21 to 28 days after ovulation, rises suddenly with the decline in the progesterone level.