Malfunction in Either the Inhibitory System or Stimulatory System, or Both, Changes the Timing of Birth or Mode of Delivery: A Hypothesis IV

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Abstract

Objective: Spontaneous preterm birth is the leading global cause of neonatal death, and countless efforts have failed to establish a single effective treatment for preterm labor. Labor dystocia is the major cause of primary cesarean delivery; however, its biological mechanisms during labor are poorly understood. When post-term pregnancy truly exists the cause is usually unknown. The objective of the study tries to identify the possible cause of these obstetric complications Design: Research Study Setting: Portiuncula University Hospital, Ireland Population: N/A Methods: This study investigated the current evidence-based literature that may support the hypothesis. Main Outcome Measures: N/A Results: Obstetric literature is replete with overwhelming evidence that malfunction of any exponential uterine wall tension (EUWT) component (stretch-dependent inhibitory system) terminates the pregnancy. Most of the current therapeutic preterm strategies are focused on preventing premature EUWT failure e.g. the cervical stitch. Progesterone treatment for preterm labor may be working by enhancing the stretch-dependent inhibitory system by modulating uterine wall plasticity and EUWT. Post-term pregnancy is due to the failure of the stimulatory system to transform the cervix into the lower uterine segment and to lose its resistance that terminating the pregnancy at full term. A combined malfunction of the two systems causes labor dystocia e.g rupture of fetal membranes with a long cervix. Conclusion: There is growing evidence to support the hypothesis that malfunction of the inhibitory system causes preterm labor, malfunction of the stimulatory system causes post-term pregnancy and labor dystocia is due to a combined malfunction of the two systems.

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