The Relationship Between AD/HD and Perinatal (Oxytocin) Pitocin Induction: Risk for Developmental Adversity?

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This August marks my 30th year as a psychotherapist serving children and families. About 15 years ago, I began tracking a clinical pattern that seemed to be more than coincidence. This pattern surfaced during my routine interviews of mothers when reviewing their children's developmental histories. In particular, children who sought treatment for AD/HD bore an all too common perinatal history; that of having been medically induced with a universally used obstetric drug, Pitocin. In addition, many of these children reported similar histories of a longer than average maternal gestation, an extended maternal labor, and a heavy birth weight. As this pattern among AD/HD children grew more typical, the need for scientific exploration of this trend could no longer be denied, and the project morphed into my doctoral dissertation. This study marks the first known attempt to identify a signature etiological mechanism in AD/HD.

To date, the precise etiology of Attention Deficit Hyperactivity Disorder (AD/HD) remains a mystery. The steadily rising incidence of AD/HD and the coincidental increase of (Oxytocin) Pitocin use in labor/delivery underscored the rationale to investigate the relationship between these two factors. Specifically, it was proposed that perinatal exposure to obstetric Pitocin risks AD/HD onset in children. The specific mechanism of this impact was proposed as being neurobiological in nature.

To test this theory, twenty-one variables were analyzed as potential AD/HD predictors: select obstetric complications, a confirmed AD/HD diagnosis and history of a first degree AD/HD relative. The medical labor and delivery records of a heterogeneous sample of 172 AD/HD and non-AD/HD children (ages 2-18) from throughout the U.S. and Canada were gathered from various agencies and independently reviewed for select obstetric markers and presence/non-presence of AD/HD.

A PLUM ordinal regression, chi-square, independent samples t-test and step-wise multiple regression comparatively analyzed and determined the predictive relationship(s) of obstetric factors to subsequent AD/HD onset. In support of this hypothesis, results revealed Pitocin induction/augmentation ($p<.001$) was *the chief AD/HD predictor* **(67.1%)**, while Pitocin exposure time ($p<.001$) and labor length ($p<.002$) also emerged as AD/HD predictors. Notable predictive trends included maternal epidurals, AROM (assisted rupture of membranes), post-birth oxygen supplementation, nuchal cord events, maternal gestation and newborn weight (LGA). Familial AD/HD (i.e. first degree relative) predisposition played no predictive role.

These findings suggest a Pitocin-linked, interactive constellation of factors initiates a neuro-developmental cascade that disrupts cognitive executive functioning, kindling AD/HD. These overlapping mechanisms: restricted fetal neural oxygen flow, insult to soft neural tissue, via prolonged, Pitocin-induced hypertonic uterine contractions and initial whetting of the neural appetite to a chemical stimulant, likely foster the perfect neuro-developmental storm. Specifically, it is highly suspected that this pressured uterine force may impose neural convolutions or architectural imprints on the immature fetal brain, altering cortical topography,

and adversely affecting long-term neural development. Other contributing dynamics may include the down-regulation of fetal (hormonal) oxytocin via obstetric Pitocin infusion, which could trigger a switch in inhibitory neurotransmitter (GABA) signaling in the fetal brain, risking hypoxia and neuronal cell death. Immuno-suppression of brain tissue and brain inflammation may also play a role.

As concerns of genesis peak around other neuro-developmental disorders (i.e. Autism spectrum disorders and Asperger's Syndrome), these results provide for a fresh etiological conceptualization of these issues. Earlier detection of AD/HD via perinatal biomarkers and intervention in high-risk perinatal circumstances is implied. In viewing AD/HD as a disorder likened to neural insult, clinical protocols may effectively employ multi-modal treatment approaches of cognitive behavioral skills, appropriate pharmacotherapy and specific strategies aimed at strengthening and maximizing cognitive executive functioning in AD/HD.

It is concluded that Pitocin induced labor carries iatrogenic fetal risks. Although Pitocin use during labor may not be the sole litmus test for AD/HD onset, it likely plays a key role in its outcome. These findings beg the revision of obstetric practices toward more intelligent management of obstetric Pitocin to ensure neural toxinsulation for the unborn fetus. Clearly the risks of prolonged fetal exposure to obstetric Pitocin should be carefully explored, as should the precise neurobiological mechanism(s) and pathophysiology inherently involved. Replication of this research via an expanded sample is urgently warranted to retest this hypothesis.

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