Hypopituitarism in pregnancy does not increase the risk of PPH despite disruption to oxytocin axis

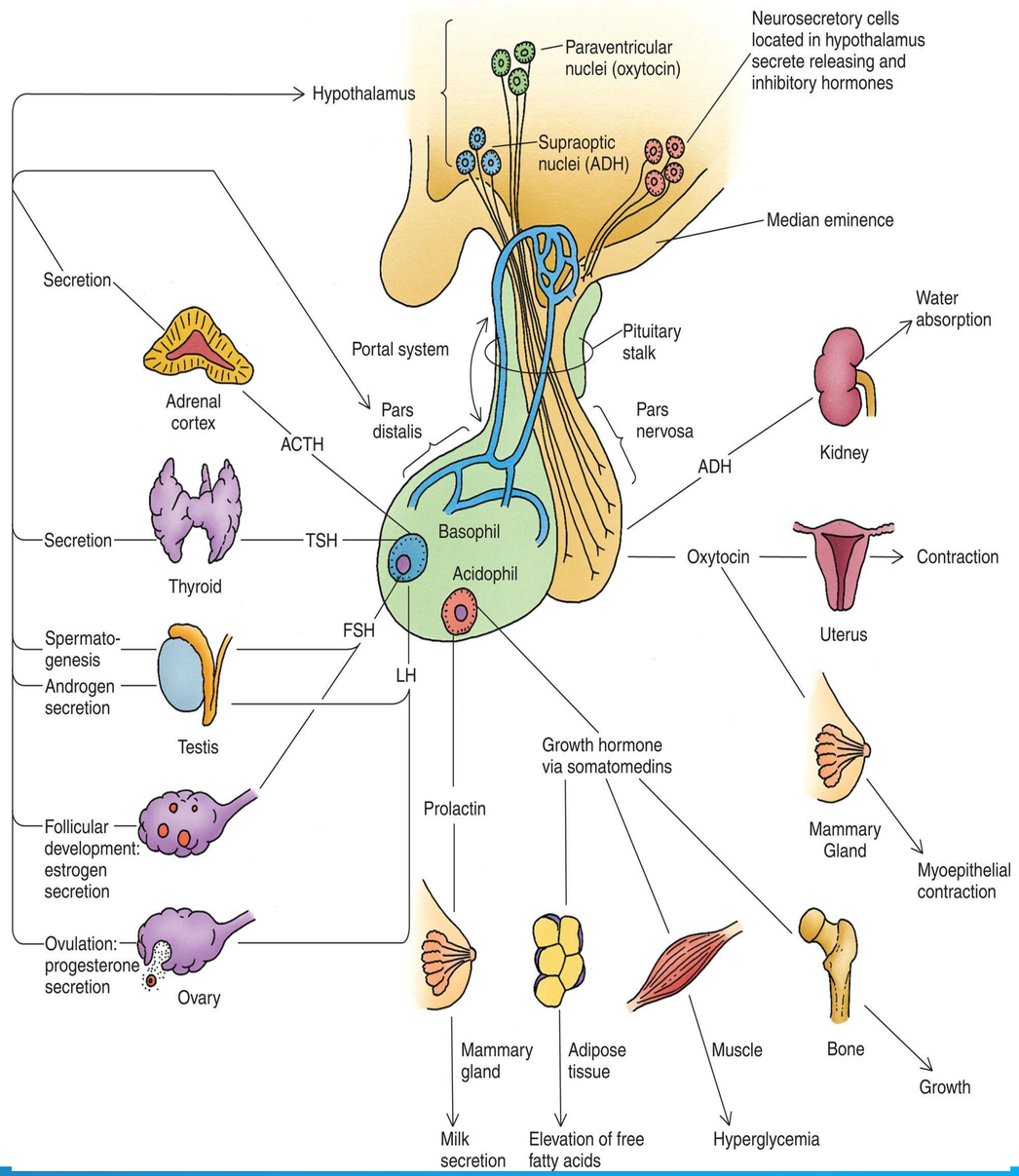
Alexander Meshel MD, Fariha Hanif BS, MPH; Edward Sutanto BS,
Andrew Warburton MD, Daniel Katz MD

May 3rd, 2026

Mount Sinai

Background and Hypothesis

- Hypopituitarism disrupts multiple pituitary axes governing blood pressure, electrolyte balance, and basal oxytocin production
- The hypothalamic-pituitary axis drives oxytocin release, which maintains uterine tone after delivery
- Theoretical mechanism: hypopituitarism → ↓ basal oxytocin → impaired uterine contraction → ↑ PPH risk
- Limited data exist on whether this physiologic disruption translates to clinically significant hemorrhagic outcomes in pregnancy
- Hypothesis: Pregnancies complicated by hypopituitarism will demonstrate higher rates of PPH, uterine atony, and related morbidity compared to unaffected pregnancies



Study Design and Methods

- Retrospective comparative analysis using the TriNetX Global Collaborative Network
- Compared pregnancies with hypopituitarism vs. without
- Primary outcome: postpartum hemorrhage
- Secondary outcomes: uterine atony, shock, sepsis, ICU admission
- Laboratory metrics collected: sodium, prolactin, estrogen, testosterone, TSH, ACTH
- Baseline matching to 1,577 patients per group
- Kaplan-Meier survival analysis used for time-to-event outcomes

Results

- 1,577 patients per group after baseline matching
- PPH: no significant difference between groups (HR CI 0.67–1.93, p = 0.714)
- Uterine atony: significantly increased in hypopituitarism group (HR 1.41, 95% CI 1.03–1.94, p = 0.034)
- Shock, sepsis, ICU admission: no significant differences
- Significant Lab values: ↓ sodium ($p < 0.001$), ↑ TSH ($p = 0.006$)
- Not significant lab values: prolactin, ACTH, estrogen, testosterone

Conclusion and Discussion

- Hypopituitarism did not significantly increase rates of PPH, shock, sepsis, or ICU admission despite theoretical disruption to the oxytocin axis
- Uterine atony was significantly more common (HR 1.41), but did not translate to clinically significant hemorrhagic outcomes
- Pituitary dysfunction alone may not be sufficient to confer clinically meaningful hemorrhagic risk as compensatory mechanisms may exist
- Significant hyponatremia and elevated TSH confirm expected endocrine disruption, validating the cohort
- Limitations: retrospective design, reliance on ICD coding, inability to confirm oxytocin levels or treatment details
- Future directions: prospective studies examining oxytocin supplementation protocols, dose requirements, and optimal peripartum management in hypopituitary patients