ALTERED APPETITE AND THIRST REGULATION IN A PATIENT WITH PANHYPOPITUITARISM WHO WAS MISDIAGNOSED WITH PRADER-WILLI SYNDROME.  A L Sloan, S Panja, K M Colleran.  Department of Internal Medicine, Division of Endocrinology, University of New Mexico, Albuquerque, New Mexico

**Background:** The hypothalamus is essential in the regulation of both thirst and appetite. Damage to the ventromedial hypothalamus leads to obesity in animals. Regarding thirst, the normal thirst response is sufficiently powerful to compensate for even the total absence of desmopressin. Thirst osmoreceptors reside in the area of the anterior hypothalamus that borders the anteroventral tip of the third ventricle. Resection of craniopharyngiomas is a difficult procedure during which the hypothalamus is often damaged.

**Case:** An 18 year-old Navajo-American morbidly obese male, with panhypopituitarism secondary to resection of craniopharyngioma ten years prior, presented with severe hypernatremia (serum sodium 180 mmol/L). The patient had a history of non-compliance with desmopressin injections associated with multiple admissions for hypernatremia with life-threatening sodium levels. During these episodes, he denied thirst. Regarding his obesity, the patient was normal weight as a child. After his craniopharyngioma resection, his body mass index increased at each yearly physical examination, rapidly exceeding 40. He was erroneously diagnosed with Prader-Willi Syndrome, and his obesity was attributed to this misdiagnosis. He reported experiencing a strong appetite and eating large quantities of food. Weight loss attempts were unsuccessful. **Conclusion:** This patient likely sustained damage to his hypothalamus, altering appetite regulation and thirst mechanism. Considering the possibility of incidental hypothalamic damage is essential to appropriate diagnosis and treatment of patients with panhypopituitarism status post neurosurgical procedures. In this patient, hypernatremia was caused primarily by panhypopituitarism but was exacerbated by damage to the hypothalamic thirst center, causing life-threatening serum sodium levels without thirst. Understanding that his thirst mechanism was damaged would have changed patient management by prompting closer monitoring of serum sodium and desmopressin administration. Similarly, his hyperphagia was secondary to altered hypothalamic appetite regulation. Understanding this association would have spared the patient the misdiagnosis of Prader-Willi Syndrome and would have prompted a different approach to weight loss. Mechanisms for hypothalamic-induced obesity and altered thirst will be discussed.