We propose a tapering protocol in the setting of potentially untreated Cushing’s Syndrome and suggest use as a bridge therapy to surgical intervention rather than destination therapy.

Pediatric Endocrinology
PEDIATRIC GROWTH AND ADRENAL DISORDERS
Utilizing Pituitary Volume (PV) and the Growth Hormone Stimulation Test (GHST) to Jointly Define the Etiology of Short Stature (SS): An Improved Diagnostic Criteria for Growth Hormone Treatment
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SAT-109
Background: We have previously shown that short children have significantly reduced PVs. In this study, we further define the etiology of SS in a larger cohort of siblings (SBs). Objective: To further investigate the efficacy of PV as an indicator of poor growth. Patients and Methods: Methods: The database of a pediatrics center was queried for SBs aged 6–18 yrs who underwent a GHST and subsequent MRI between 2013–2019. Their MRI results were calculated using the ellipsoid formula (LxWxH/2). Previous ROC curve analysis has defined 215.02mm3 as significantly smaller PVs than normal controls (NCs). Cutoff analysis was utilized to generate cutoff values. The diagnostic criteria for SS was identified in 96.1% of the SBs. 3 of the 4 pre-PB SBs, who did not meet the PV cutoff had PVs within 10% of the cutoff. Conclusion: We have shown that PV is not inferior to the GHST in the diagnosis of SS. Combining the GHST and PV defines the etiology of SS in 96.1% of patients. Jointly, the GHST and PV should be considered the new gold standard for identifying children who qualify for GH therapy. This criteria will significantly diminish the number of patients diagnosed with ISS.

Pediatric Endocrinology
PEDIATRIC GROWTH AND ADRENAL DISORDERS
Pituitary Volume as a Diagnostic Measure for Determining the Etiology of Short Stature in Children: Receiver Operating Characteristic Curve Analysis and Cutoff Values
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SAT-086
Background: It is speculated that pituitary volume (PV) is a marker of chronic growth hormone (GH) secretion. In previous studies, we determined that children with GH deficiency (GHD) and idiopathic short stature (ISS) had significantly smaller PVs than normal controls (NCs). Cutoff values for small PVs are needed to improve the clinical utility of PV in determining children who qualify for GH therapy.

Patients and Methods: The SS group was selected from the database of a pediatric endocrinology center, which was queried for siblings (SBs) aged 6–18 yrs who underwent a GH stimulation test and MRI between 2013–2019. All 77 SBs had SS, defined as 2 SDs below mean height for age, subnormal growth velocity for at least 6 months, or predicted height at least 2 inches discrepant from midparental height. The NC group was selected from the database of a neuroradiology center; these NCs consisted of 170 randomly selected subjects aged 6–18 yrs. Patients with MRI abnormalities were excluded. PVs were calculated using the ellipsoid formula (LxWxH/2). ROC curve analysis was utilized to generate cutoff values. The diagnosis of short stature was the dependent variable and PV was the independent variable. The PV with the highest Youden index was selected as the definitive cutoff for a small PV.

Results: The mean (MN) and median (MD) age of SBs was 13.0 ± 1.4 and 13.2 yrs, respectively, and the MN and MD age of the NCs was 12.6 ± 3.4 and 13.2 yrs, respectively. The MN and MD age of prepubertal SBs (n=29) and NCs (n=58) were GHD. 81.8% of all SBs had small PVs, while 84.4% were GHD. When combined, GHST and PV identify the etiology for SS in 96.1% of subjects. Discussion: The GHST recognized the etiology for SS in 84.4% of the SBs, while PV identified 81.8%. Using both criteria together, the etiology for SS was identified in 96.1% of the SBs. 3 of the 4 pre-PB SBs, who did not meet the PV cutoff had PVs within 10% of the cutoff.
were 9.3 ±1.2 and 9.7, and 8.6 ±1.4 and 8.6 yrs, respectively. The MN and MD age of pubertal SBs (n=48) and NCs (n=112) were 13.0 ±1.4 and 12.7, and 14.7 ±1.9 and 14.6 yrs, respectively. The difference in MN age between SBs and NCs was significant (p<0.05). For prepubertal subjects, sensitivity was 86.21% and specificity was 68.97%. The distance to corner was 0.3396, and the highest Youden index was 0.5517, corresponding to a PV of 215.02 mm3. The Area Under the Curve (AUC) was 0.8395 with a standard error of 0.0426 (p<0.001). Forpubertal subjects, sensitivity was 81.25% and specificity was 79.46%. The distance to corner was 0.2781, and the highest Youden index was 0.6071, corresponding to a PV of 315.0 mm3. The AUC was 0.8460 with a standard error of 0.0337 (p<0.001).

Conclusion: To our knowledge, we present the first study on the sensitivity and specificity of PV in determining the etiology of SS. Our data suggest that prepubertal patients with a PV<215.02 mm3 and pubertal patients with a PV<315.00 mm3 have small pituitary glands. Statistically calculated cutoffs are necessary to accurately diagnose pituitary hypoplasia and should be utilized to determine the etiology of SS. Future studies should include children with Tanner staging and height SDs to generate more accurate PV cutoffs.

Reproductive Endocrinology
FEMALE REPRODUCTION: BASIC MECHANISMS
Effect of Poor Circumstance in Utero on Adiponectin Gene Expressions Through Epigenetic Changes in Offspring
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MON-013
The links between obesity and metabolic syndrome in parents and their offspring and the role of genes and a shared environment are not completely understood. Adipocytokines play important roles in glucose and lipid metabolism. We have already developed the model mice for transgenerational effect of obesity and metabolic syndrome and demonstrated that exposure to a high fat diet in utero might cause a metabolic syndrome-like phenomenon through epigenetic modifications of adipocytokine, adiponectin and leptin gene expressions in offspring of the model mice. In this study, we examined whether poor circumstance in utero affected the adiponectin gene expression and epigenetic changes of this gene using samples from umbilical cord in patients with hypertensive disorder of pregnancy (HDP) and fetal growth restriction (FGR) or gestational diabetes mellitus (GDM) and heavy for date fetus (HFD) compared with normal pregnant women without HDP, GDM and abnormal fetal growth. We observed that the poor circumstance under HDP with HGR or GDM with HFD caused significantly lower adiponectin gene expression and higher methylation level of histone H3 at lysine 9 of the promoter of adiponectin gene compared with normal control. Thus, poor circumstance in utero affected adiponectin gene expressions through epigenetic modifications, which might result in the increased risk for metabolic syndrome of offspring.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORTS II
Novel Use of Abaloparatide to Augment Spinal Fusion in Patient Undergoing Cervicothoracic Revision Surgery
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MON-365
Objective To present a case of using Abaloparatide (PTHrP 1–34 analogue) to promote spinal fusion in a patient with history of cervical instability s/p multiple cervical operations with non-union. Case Presentation 66 year-old female with a history of multiple sclerosis, obesity and hypothyroidism underwent neurosurgical evaluation of neck pain. She was found to have cervical spinal stenosis causing neck pain, radiculopathy, motor deficits and ataxia. Initially underwent anterior cervical discectomy and fusion which temporarily alleviated symptoms before suffering nonunion. Subsequently underwent two additional surgeries which also eventually failed. She presented to our facility for revision corpectomy and spinal fusion. Given her history of non-union, endocrinology was consulted for evaluation of metabolic bone disease. No known personal or family history of metabolic bones disease. No history of chronic steroid use. Initial endocrine evaluation excluded common pathologies. A decision was made to pursue anabolic osteoporosis therapy to attempt to augment the spinal fusion process. Patient started on Abaloparatide 80mcg daily 2 weeks post procedure with planned 12-week therapy course. Cervical CT at 3 and 6 months showed post-surgical cervicothoracic fusion with no signs of non-union. Discussion Abaloparatide is a 34 amino acid synthetic analogue of parathyroid hormone related peptide (PTHrP) which works by selectively activating PTH1 receptor found on osteoblasts. Currently anabolic therapies are only FDA approved for treatment of osteoporosis but there is reported off label use in cases of spinal fusions, arthroplasty and fracture healing. Studies have shown that presence of PTH and PTHrP are necessary for fracture healing. Animal studies have also shown that intermittent PTH promotes spinal fusion. This case represents a novel use for Abaloparatide to augment spinal fusion in a human clinical model. Conclusion Further studies are warranted to better understand mechanism of action, drug timing and duration for optimal treatment of anabolic therapies in bone fractures and healing. The use of anabolic therapies like Abaloparatide can be considered in patients undergoing spinal fusion surgery at high risk for non-union or undergoing revision for failed fusion. References O’Loughlin PF, Cunningham ME, Bukata SV et al. Parathyroid Hormone Augments spinal fusion, fusion mass, and fusion mass quality in a rabbit spinal fusion model. Spine 2009 January; 34: 121–130.