# Abnormalities and Variations in Growth: When a Child Is "off the Chart"

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## Learning Objectives – Disorders of Growth and Stature

- Perform growth measurements and interpret growth charts to be able to identify children with short or tall stature.
- Differentiate among the common origins of short and tall stature and plan an appropriate diagnostic evaluation for a slowly or rapidly growing child.
- Describe when treatment is indicated for children with short and tall stature.
- Develop an osteopathic structural examination and manipulative treatment plan based on specific disorders of growth and stature.

LR Braun, R Marino. **Disorders of Growth and Stature.** *Pediatr Rev. 2017;38(7):293-304.*



## Practice Gap – Disorders of Growth and Stature

- It is often challenging to identify children with abnormal growth patterns and distinguish normal growth variants from pathologic variants.

LR Braun, R Marino. **Disorders of Growth and Stature.** *Pediatr Rev. 2017;38(7):293-304.*



## Disclosures…

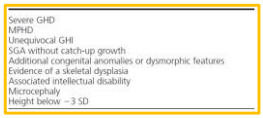
I have nothing to disclose…



## Introduction

## Factors That Increase the Likelihood for a Monogenic Cause of Short Stature

- There is no high-quality evidence to define the optimal approach to the genetic evaluation of short stature.
- Genetic testing in order to identify the various monogenic disorders that present with short stature should be tailored to each patient and in coordination with a pediatric endocrinologist or geneticist.




- GHD – Growth hormone deficiency
- GHI – Growth hormone insensitivity
- MPHD – Multiple pituitary hormone deficiency
- SGA – Small for gestational age

A Dauber, RG Rosenfeld, JL Hirschhorn. **Genetic Evaluation of Short Stature.** *J Clin Endocrinol Metab 2014;99(9):3080-3092.*



## Diagnoses for "High Risk" Children Separated by Gender (N = 555)

| Condition | Boys # | % | Girls # | % |
|---|---|---|---|---|
| **Familial short stature** | 133 | 37 | 74 | 37 |
| **Constitutional growth delay** | 102 | 28 | 47 | 24 |
| **BOTH** | 63 | 18 | 31 | 16 |
| **OTHER** | 29 | 8 | 24 | 12 |
| **Idiopathic short stature** | 16 | 5 | 11 | 6 |
| **GH deficiency** | **12** | **3** | **4** | **2** |
| **Turner syndrome** | NA | -- | **6** | **3** |
| **Hypothyroidism** | **2** | **< 1** | **1** | **< 1** |

- Most children growing **< 5 cm/yr** (a commonly used threshold rate) will not have an endocrine disorder.
- Many children (48% in this study) with GHD and others with Turner syndrome may currently *be unrecognized and untreated.*

R Lindsay, M Feldkamp, D Harris, et al. **Utah Growth Study: Growth standards and the prevalence of growth hormone deficiency.** *J Pediatr 1994;125(1):29-35.*



# Obesity



## Body Mass Index and Obesity and Overweight

- Currently, about one-third of children and adolescents in the United States are overweight or obese, and 5% are severely obese.
- Body mass index (BMI), based on weight and height, and adjusted for age and gender, is widely used to diagnose overweight and obesity.
- **A BMI at the 95th percentile or greater indicates obesity and at or above the 85th percentile designates a child as overweight.**
- BMI is an imprecise measure of adiposity; skinfold and abdominal girth are better measures.
- Extremely fit individuals, such as professional athletes, can have a high BMI, but little body fat.



## Childhood Obesity Prevalence and Risk Factors

- Overweight and obesity are more prevalent in black, Native American, and Hispanic children as compared to non-Hispanic white children, and in children from low-income families.
- Parental obesity significantly increases the risk of childhood obesity.
- Obese children and adolescents are at high risk of becoming obese adults and have increased lifetime rates of cardiovascular disease and metabolic syndrome.
- There is good evidence that a substantial component of adolescent obesity is established before age 5 years; prevention and early treatment of childhood overweight and obesity, therefore, has the potential to make a significant impact on lifelong health.



## Childhood Obesity Management

- The most evidence-based strategies include school-based programs with an emphasis on **increased physical activity, nutritional education, good-quality school food, and family-based interventions focused on increasing activity and decreasing screen time.**
- The most successful primary care–based programs for weight loss include motivational interviewing and setting goals for small, manageable changes.
- *Decreasing sugar-sweetened beverage consumption and limiting screen time* have been associated with success, although the effects are modest at best.

## Management of Overweight and Obesity

- Management of overweight and obese children should focus on education and behavior change for the whole family.
- The home environment should support healthy eating and increased physical activity.
- This includes having less unhealthy food brought into the home, greater availability of more nutritious foods, and less opportunity for sedentary behaviors.
- This environment can be bolstered by community and school-level interventions.



## Childhood Obesity Comorbid Conditions

- Children with obesity are likely to remain obese into adulthood, and the risk of adult obesity increases with age; adolescents have a 90% chance that their obesity will persist into adulthood.
- **Obesity is associated with comorbidities in childhood, including:**
  - Depression
  - Hepatic steatosis
  - Hyperlipidemia
  - Hypertension
  - Obstructive sleep apnea
  - Orthopedic conditions and joint pain
  - Type 2 diabetes mellitus



## Childhood Obesity Screen for Comorbidities

- Children with obesity should be screened for *hypertension, hyperlipidemia, and fatty liver disease.*
- Beginning at age 10 years or the onset of puberty (whichever occurs earlier), they should also be screened for type 2 diabetes mellitus if they have at least 1 of the following risk factors:
  - Family history of type 2 diabetes mellitus in a first- or second-degree relative
  - High-risk race/ethnicity (Native American, African-American, Latino, Asian American, or Pacific Islander)
  - Signs of insulin resistance on physical examination
  - Maternal history of diabetes or gestational diabetes during the child's gestation

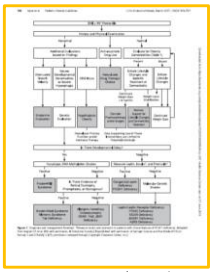


## When to Chase the Zebra…

1. *Abnormal history and physical examination*

**Additional findings from evaluation:**

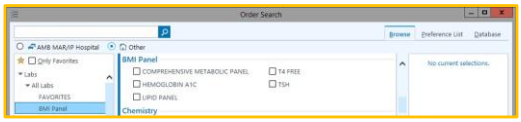
1. **Attenuated growth velocity**
2. **Neurodevelopmental abnormalities or severe hyperphagia**
3. **CNS injury**



DM Styne, SA Arslanian, EL Connor, et al. **Pediatric obesity—assessment, treatment, and prevention: an Endocrine Society clinical practice guideline.** *J Clin Endocrinol Metab. 2017;102(3):709-757.*



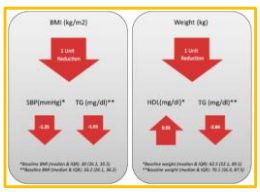
## Laboratory Diagnosis




- CMP
- Hemoglobin A1c
- Lipid Panel
- TSH
- Free T4
- Vitamin D level…



## The Proof in the Pudding…




- Decreased sugar-sweetened beverage consumption
- Higher level of activity
- Improved sleep
- Decreased screen time
- Increased family involvement
- **Improve disordered family function**
- Increased school involvement
- Increased breastfeeding

DM Styne, SA Arslanian, EL Connor, et al. **Pediatric obesity—assessment, treatment, and prevention: an Endocrine Society clinical practice guideline.** *J Clin Endocrinol Metab. 2017;102(3):709-757.*

# Failure to Thrive



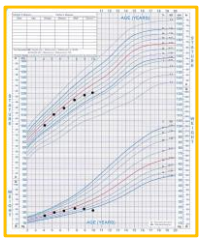
## Failure to Thrive

- Failure to thrive is a physical sign of a nutritional state that is inadequate to support normal growth and development.
- Often, the underlying causes of this growth failure are complex, involving organic, functional, and psychosocial components.
- The patient's history and physical examination are crucial and should alert the clinician to the possibility of a metabolic or genetic condition.
- Children with failure to thrive or poor weight gain fall into 1 of 3 categories: *insufficient caloric intake, insufficient absorption of the calories they consume, or increased caloric needs due to increased metabolic demand.*



## Diabetes Mellitus Type 1 and Celiac Disease

- Patients who have type 1 diabetes are at increased risk for the development of various autoimmune disorders.
- Celiac disease occurs in 5% to 6% of all children who have type 1 diabetes and is more common in those who develop type 1 diabetes before the age of 10 years.
- Adrenal insufficiency because of autoimmune adrenal failure is another possibility, but it is much less common than celiac disease (< 1% of children who have diabetes).





| Suspect an inborn error of metabolism when any one of the following (or combination) is present |
|---|
| History of acute, severe, and potentially life-threatening symptoms and signs (recurrent ketoacidosis, hypoglycemia) |
| Recurrent attacks of vomiting, lethargy, dehydration |
| Liver dysfunction |
| Developmental delay, hypotonia, seizures, stroke, ataxia |
| Cardiomyopathy, myopathy |
| Hearing loss or visual impairment |
| Organomegaly |
| Mild dysmorphic or coarse facial features |
| Pancytopenia |

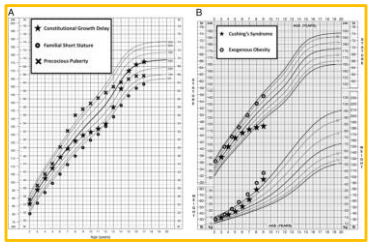


| Laboratory and Diagnostic Workup | | |
|---|---|---|
| **Baseline screening tests for FTT to rule out IEM** | **If there are abnormal neurologic findings** | **If there is a suspicion for IEM** |
| • **CMP**<br>• **CBC**<br>• **Urinalysis** | • Magnetic resonance imaging<br>• Magnetic resonance spectroscopy | • Plasma amino acids<br>• Plasma acylcarnitines<br>• Ammonia<br>• Blood lactate, pyruvate<br>• Creatine kinase<br>• Urine organic acids |



# Normal Growth Variations

## Typical Linear Growth Patterns for Normal and Abnormal Variations




LR Braun, R Marino. **Disorders of Growth and Stature.** *Pediatr Rev. 2017;38(7):293-304.*



## Some Growth Charts

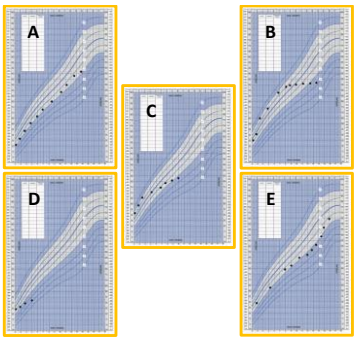
**A. Familial short stature**

B. "Underlying medical condition…"

C. "Underlying medical condition…"

D. "Underlying medical condition…"

**E. Constitutional growth delay**






## Peak Growth Velocity

- Prior to puberty and peak pubertal growth velocities, normal childhood growth rates are approximately **5 cm/yr (4 – 7).**
- The first evidence of true puberty in boys is testicular volume of at least 4 mL (testicular length > 2.5 cm).
- Pediatricians should keep in mind that **boys** do not reach their peak pubertal growth velocities (8 to 12 cm/yr) until they reach Sexual Maturity Rating stage *3 to 4*; **girls** typically reach peak growth velocities *between stages 2 and 3*.



## Mid-parental Height and Familial Short Stature

- Target height (adjusted mid-parental height) can be calculated as follows:

**For girls: [mother's height + father's height - 5 inches (13 cm)]/2**

**For boys: [mother's height + father's height + 5 inches (13 cm)]/2**

- The final height of most individuals is within 2 inches (1 standard deviation) on either side of this calculated target height.
- Familial short stature is associated with a bone age concordant with chronologic age and normal pubertal onset and progression.
- Final adult height is consistent with target height.



## Upper Body/Lower Body Segment Ratio

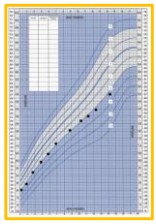
- The only abnormal feature of growth presented on the patient in this vignette is the increase in the upper body segment/lower body segment (U/L) ratio over the past year.
- The U/L ratio is an assessment of body proportions that compares the lower body segment (a measure from the top of the pubic symphysis to the floor in a standing patient) and the upper body segment (determined from subtracting the lower body segment from the standing height).
- The U/L ratio declines from birth and reaches its lowest point during early puberty.
- Prior to and during puberty, the legs grow faster than the trunk, which accounts for the trend of a decreasing U/L ratio.



## Upper Body/Lower Body Segment Ratio

- After pubertal growth, there is often a very slight increase in U/L ratio, as the legs stop growing prior to the trunk.
- The average U/L ratio is 1.7 for an infant, 1.3 at 3 years of age, and 1.0 for an adult.
- Several growth disorders and disease processes may be associated with abnormal patterns in the U/L ratio.
- **A decreased U/L ratio for age** may be associated with *skeletal dysplasias involving the spine* and also with disorders involving delayed or incomplete puberty such as Klinefelter, Marfan, and Kallmann syndromes.
- **Increased U/L ratios** may be seen in Turner syndrome, skeletal dysplasias involving the long bones (such as achondroplasia), or in patients with *precocious puberty.*

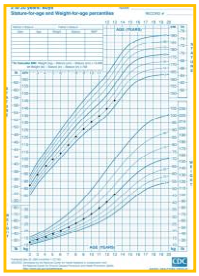
## Constitutional Growth Delay



- Children who have constitutional delay commonly have a **strong family history** of such delay, and in some cases, genes associated with hypothalamic hypogonadism have been identified in affected families.
- However, in most recent studies, no genes to explain constitutional delay have been found, although the pattern of growth is clearly heritable and as common in females as in males.
- Therefore, a boy whose mother experienced late menarche might be expected to have delayed puberty.



## Constitutional Growth Delay Diagnosis



- A **bone age** would provide the most useful information because this boy's bone age would be expected to be delayed to coincide with his current level of pubertal progression.
- Based on his growth curve, his height age (the age at which his current height is at the 50th percentile) is 10.25 years.
- A bone age between 9.75 and 10.75 years would strongly suggest that the child has constitutional delay.



## Constitutional Delay Treatment

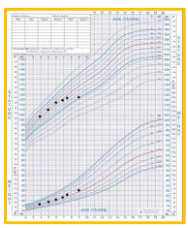
- **Treatment of constitutional delay typically involves only reassurance and observation.**
- However, *in select cases*, a 3- to 4-month course of testosterone cypionate or oxandrolone can be used to initiate central puberty (using the positive feedback of androgen on LH/FSH secretion in normal early puberty).
- Once a presumed diagnosis of constitutional delay is made, boys should be re-evaluated every 4 to 6 months to ensure that progression toward puberty ensues.



## Abnormal Growth Variations



## Endocrine Disease as Cause of Abnormal Growth Velocities

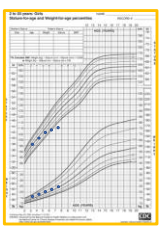


- A hallmark of short stature due to endocrine disease is **central adiposity and somewhat decreased muscle mass,** which is found in *growth hormone deficiency, hypothyroidism, and Cushing syndrome*.
- **Craniopharyngioma** may present with endocrine deficiency disorders such as growth hormone and thyroid-stimulating hormone deficiency, leading to hypothyroidism.



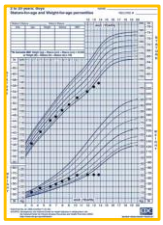
## Hypothyroidism as Cause of Abnormal Growth Velocities

- Growth charts
  - An endocrine cause of poor growth is likely when **linear growth arrests but weight gain is either normal or increasing.**
    - Falloff or "resetting" the linear growth is expected to occur by 2 years of age
    - Falloff after 2 years of age is not a normal growth pattern.
  - In most cases where poor growth is related to **undernutrition**, *falloff in weight gain will precede falloff in linear growth.*



## Celiac Disease or Renal Insufficiency as Cause of Abnormal Growth Velocities

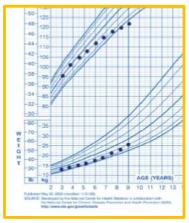
- *Celiac disease and renal insufficiency usually lead to weight loss when associated with slowing growth*, but additional symptoms would be expected.
- Growth curve in a child who has celiac disease documenting slowing of linear growth and weight loss.





## Cushing Syndrome as Cause of Abnormal Growth Velocities

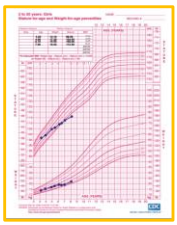
- The most common cause of Cushing syndrome in children is exogenous steroid exposure, including that from inhaled and topical steroids.
- The most common signs and symptoms of Cushing syndrome include **weight gain with linear growth failure**, round face, facial plethora, violaceous abdominal striae, hypertension, easy bruising, and proximal muscle weakness.
- Endogenous Cushing syndrome is very rare in children and can be due to an adrenocorticotropic-secreting pituitary adenoma or adrenal cortical overactivity.
- Laboratory investigation may show hypokalemia.





## Growth Hormone Deficiency

- Congenital growth hormone deficiency presents with linear growth deceleration after the first 6 to 12 months of life when growth hormone becomes important for linear growth.
- **Weight is not affected as much as length, so weight for length increases.**
- There may also be evidence of altered body composition with increased fat mass and decreased lean body mass.
- Other pituitary hormone deficiencies may be associated.





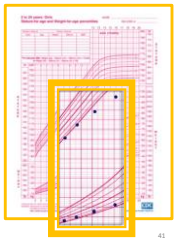
## Acquired Growth Hormone Deficiency

- Levels of **insulin-like growth hormone factor-1** and insulin-like growth factor-binding protein 3 are low and **growth hormone levels** after stimulation with 2 provocative agents remain less than 10 ng/mL.
- **Bone age is delayed.**
- Magnetic resonance imaging of the brain and sella turcica and testing for other pituitary hormone deficiencies should be undertaken in those found to have growth hormone deficiency.



## Turner Syndrome as a Cause of Abnormal Growth Velocities

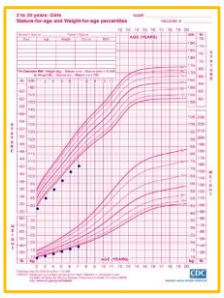
- The test most likely to reveal the diagnosis is a **karyotype**.
- Turner syndrome results from a missing or structurally abnormal X chromosome.
- About half of girls with Turner syndrome have a 45,X karyotype.
- The other half have a karyotype with a portion of an X chromosome missing or rearranged, or a karyotype that contains mosaicism, for example 45,X/46,XX.
- Features of Turner syndrome include small size at birth, history of recurrent otitis media, difficulty with math, **short stature with declining growth velocity**, epicanthal folds, *high-arched palate*, and multiple nevi.





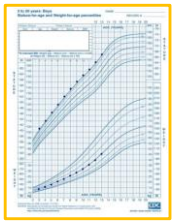
## Turner Syndrome

- Other common features include ptosis; low-set, prominent ears; *neck webbing; low posterior hairline; broad chest with wide-spaced nipples;* congenital heart disease (e.g., coarctation of the aorta); renal anomalies (e.g., horseshoe kidney); skeletal anomalies (scoliosis, shortened 4th metacarpals, high upper-to-lower segment ratio); and *primary ovarian failure.*
- **Short stature is the most common manifestation of Turner syndrome and may be the only presenting feature.**
- **Turner syndrome should be considered in all girls with short stature.**



## Klinefelter Syndrome as a Cause of Abnormal Growth Velocities

- Occurs in about 1 of 600 male newborns and is diagnosed by a karyotype showing 47,XXY.
- A patient's history of language delay, **tall stature for his family, low upper-to-lower segment ratio (relatively long legs),** and abnormal puberty are consistent with Klinefelter syndrome.
- In early adolescence, a normal upper-to-lower segment ratio is around 1.
- (The lower segment is measured from the pubic symphysis to the floor. The upper segment is calculated by subtracting the lower segment from total height).
- Individuals with Klinefelter syndrome have disproportionately long legs and are at higher risk for language delay and learning problems.





## Klinefelter Syndrome – Comorbidities in Boys with a 47,XXY Karyotype (N = 153)

| Comorbidity | Number (%) |
|---|---|
| Undescended testes | 5 (3) |
| Inguinal hernia | 8 (5) |
| Epilepsy | 12 (8) |
| Febrile seizures | 5 (3) |
| Learning disabilities | 102 (67) |
| Attention deficit disorder | 41 (27) |
| Psychosocial/behavioral problems | 51 (33) |
| Scoliosis | 12 (8) |
| Headaches | 13 (9) |
| Pervasive developmental disorder | 4 (3) |

- Klinefelter syndrome is a common condition that remains underdiagnosed, particularly prior to adulthood.
- Early detection could prevent morbidity and mortality, but the classic phenotype of small testes and tall stature may not be apparent until adolescence, and there is minimal guidance regarding whom to screen.

L Nahata, I Rosoklija, RN Yu, LE Cohen. **Klinefelter Syndrome: Are We Missing Opportunities for Early Detection?** *Clin Pediatr (Phila) 2013;52(10):936-941.*



## Marfan Syndrome

- Echocardiography would aid in the diagnosis of Marfan syndrome, a connective tissue disorder caused by mutations in *FBN1*, which encodes the fibrillin-1 protein.
- Marfan syndrome can affect the aortic root, causing enlargement and even aortic dissection.
- Features of Marfan syndrome include a tall, **thin body habitus and long arms (arm span is greater than height), legs, and fingers (arachnodactyly),** as well as other skeletal abnormalities (e.g., *scoliosis, pectus abnormality, crowded teeth*), hyperflexibility, striae, and eye abnormalities (e.g., myopia, dislocated lens).
- Marfan syndrome occurs in about 1 of 5,000 people.



## Tall Stature Differential Diagnosis

- Other etiologies of tall stature include precocious puberty, familial tall stature, and other genetic syndromes (e.g., Sotos syndrome).
- Precocious puberty causes early growth acceleration and tall stature during childhood, but final adult height may be compromised because of early cessation of growth.
- **Familial tall stature** is tall stature that is consistent with the genetic potential of having tall parents.
- It is the most common etiology of tall stature.
- These children are otherwise healthy and grow with normal height velocity along a height percentile as expected for their parents' heights.



## Osteopathic Considerations



## Osteopathic Manipulative Medicine – Pathophysiology: Viscerosomatic Reflexes

| Sympathetic Viscerosomatic Reflexes | | | |
|---|---|---|---|
| **Visceral Organ** | **Spinal Cord Level** | **Visceral Organ** | **Spinal Cord Level** |
| **Head and Neck** | T1-T4 | **Kidneys** | T10-T11 |
| **Heart** | T1-T5 (left) | **Upper Ureters** | T10-T11 |
| **Respiratory System** | T2-T7 | **Lower Ureters** | T12-L1 |
| **Esophagus** | T2-T8 | **Bladder** | T11-L2 |
| **Upper GI Tract** | T5-T9 | **Gonads** | T10-T11 |
| **Middle GI Tract** | T10-T11 | **Uterus/Cervix** | T10-L2 |
| **Lower GI Tract** | T12-L2 | **Erectile Tissue** | T11-L2 |
| **Appendix/Cecum** | T10-T12 | **Prostate** | T12-L2 |
| **Arms** | T2-T8 | **Legs** | T11-L2 |

## Osteopathic Manipulative Medicine – Pathophysiology: Viscerosomatic Reflexes

| Genitourinary Chapman Points | | |
|---|---|---|
| **Organ** | **Anterior** | **Posterior** |
| **Adrenals** | 1 inch lateral and 2 inches superior to the umbilicus (on respective side) | T11-T12 (on respective side) |
| **Kidneys** | 1 inch lateral and 1 inch superior to the umbilicus (on respective side) | T12-L1 (on respective side) |
| **Bladder** | Peri-umbilical area | L2 TP Bilaterally |
| **Ovaries/ Urethra** | Superior pubic ramus, 2 cm lateral to the symphysis | L2 TP bilaterally |
| **Prostate** | The outer femur (posterior IT band) | Lateral sacral base bilaterally |
| **Uterus** | Inferior pubic rami | L5 TP bilaterally, medial to PSIS |



## Osteopathic Manipulative Medicine – Pathophysiology: Viscerosomatic Reflexes

| Gastrointestinal Chapman Points | | |
|---|---|---|
| **Organ** | **Anterior Points** | **Posterior Points** |
| **Esophagus** | • Bilateral 2nd intercostal space, most medial aspect | • Bilateral face of the transverse process of T2, midway between the tips of the spinous and transverse processes |
| **Stomach hyperacidity** | • Left 5th intercostal space from mid-axillary line to sternum | • Left 5th intertransverse space, midway between the tips of the spinous and transverse processes |
| **Stomach peristalsis** | • Left 6th intercostal space from mid-axillary line to sternum | • Left 6th intertransverse space, midway between the tips of the spinous and transverse processes |
| **Pylorus** | • Midline on the sternum | • Face of the right 10th rib at the costotransverse junction |



## Osteopathic Manipulative Medicine – Pathophysiology: Viscerosomatic Reflexes

| Gastrointestinal Chapman Points | | |
|---|---|---|
| **Organ** | **Anterior Points** | **Posterior Points** |
| **Liver** | • Right 5th intercostal space, most medial aspect | • Right 5th intertransverse space, midway between the tips of the spinous and transverse processes |
| **Gallbladder** | • Right 6th intercostal space, most medial aspect | • Right 6th intertransverse space, midway between the tips of the spinous and transverse processes |
| **Spleen** | • Left 7th intercostal space, most medial aspect | • Left 7th intertransverse space, midway between the tips of the spinous and transverse processes |
| **Pancreas** | • Right 7th intercostal space, most medial aspect | • Right 7th intertransverse space, midway between the tips of the spinous and transverse processes |



## Osteopathic Manipulative Medicine – Pathophysiology: Viscerosomatic Reflexes

| Gastrointestinal Chapman Points | | |
|---|---|---|
| **Organ** | **Anterior Points** | **Posterior Points** |
| **Small intestine** | • Bilateral 8th, 9th, and 10th intercostal spaces, most medial aspect of the intercostal space | • Bilateral 8th, 9th and 10th intertransverse spaces, midway between the tips of the spinous and transverse processes |
| **Colon** | • Bilaterally along the anterior margin of the iliotibial band | • Bilaterally in a triangular area between the tips of the transverse processes of L2-L4 and the iliac crest |
| **Appendix** | • Tip of the right twelfth rib | • Between the right T11 and T12 transverse processes, lateral aspect of this intertransverse space |
| **Rectum** | • Bilateral lesser trochanter of femur | • Bilaterally over the sacrum adjacent to the inferior aspect of the sacroiliac joint |



## Summary Example – The Patient with Inflammatory Bowel Disease

**Physiology and Associated Somatic Dysfunctions**

**Parasympathetics**

- Increased tone = increased peristalsis
- Vagus nerve (CN X) exits the jugular foramen (composed of occiput and temporal bones).
  - **Somatic dysfunctions of occipitoatlantal joint (OA), atlantoaxial joint (AA), C2**
  - **Compression of occipitomastoid sutures**
- Pelvic splanchnics — S2–S4
  - **Sacroiliac dysfunctions**

**Sympathetics**

- Increased tone = decreased peristalsis
- Somatic dysfunction of **T5 – L2**
- **Abdominal fascial restrictions**
- Prevertebral ganglion — fascial restriction
  - **Superior mesenteric ganglion — fascial restriction**
  - **Inferior mesenteric ganglion — fascial restriction**



## Summary Example – The Patient with Inflammatory Bowel Disease

**Motor**

- Not applicable

**Other somatic dysfunctions**

- Rib dysfunctions
- Respiratory diaphragm somatic dysfunction
- Pelvic diaphragm somatic dysfunction
- Innominate dysfunctions

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## Summary Example – The Patient with Inflammatory Bowel Disease

**2-Minute Treatment**

- *Thoracic* — MFR
- *Lumbar* — MFR

**5-Minute Treatment**

- *Head* — OA release
- *Thoracic* — MET or HVLA
- *Lumbar* — MET or HVLA

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## Summary Example – The Patient with Inflammatory Bowel Disease

**Extended Treatment**

- *Head* — V spread technique
- *Cervical* — AA, C2, FPR, and/or HVLA
- Rib raising
- *Abdomen* — collateral ganglia release
- *Abdomen/other/viscerosomatic* — mesenteric release
- *Innominate* — MET
- *Innominate* — pelvic floor release DIR
- *Sacrum* — MET
- *Sacrum* — presacral fascia release technique

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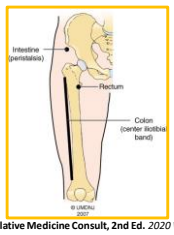
## Summary Example – The Patient with Inflammatory Bowel Disease

**Extended Treatment**

- *Abdomen/other/viscerosomatic* — Chapman's reflexes

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## Summary



## PREP Pearls – Obesity

- Documenting height, weight, and body mass index frequently will allow for early detection and intervention for children who are moving in the direction of overweight status.
- The American Academy of Pediatrics recommends the routine use of body mass index (BMI) to screen for obesity in all children beginning at 2 years of age.
- Normal weight children may have an elevated BMI and may require screening for diabetes mellitus. This will be missed if BMI is not routinely assessed.
- Prevention of obesity is important, as most lifestyle interventions later on in adolescence are less successful.



## PREP Pearls – Obesity

- A body mass index (BMI) at or above the 95th percentile indicates obesity and at or above the 85th percentile designates a child as overweight.
- Management of overweight and obese children should focus on education and behavior change for the whole family.
- Most successful interventions for obesity management have some degree of goal-setting and self-monitoring.
- Nearly all obesity is related to excess caloric intake and sedentary lifestyles; genetic syndromes and endocrinologic disorders account for a very small percentage of cases.
- Obesity in children is associated with an increased risk of hypertension, hyperlipidemia, type 2 diabetes mellitus, obstructive sleep apnea, hepatic steatosis, orthopedic conditions, and depression.

## PREP Pearls – Failure to Thrive

- Failure to thrive is a physical sign of undernutrition that is inadequate to support normal growth and development and that is usually multifactorial.
- An underlying metabolic cause of FTT is suggested by a history or physical examination finding of severe, life-threatening disease; recurrent vomiting and dehydration; developmental delay or regression; hypotonia, stroke, or seizure; organomegaly, particularly hepatomegaly; cardiomyopathy; visual or hearing deficit; dysmorphic features; or pancytopenia.
- Hospitalization for evaluation and treatment of failure to thrive is reserved for severely affected children or cases in which social factors place the child at significant risk.
- Evaluation and treatment of failure to thrive is aimed at improving nutrient quantity and quality, optimizing feeding practices, and overcoming food aversions; a multidisciplinary approach to feeding is preferred.



## PREP Pearls – Upper Body/Lower Body Segment Ratio

- The upper body segment/lower body segment (U/L) ratio decreases from birth and reaches its lowest point during early puberty.
- The average growth and weight gain for a child 3 years of age to the start of puberty is 4 to 7 cm/year and 2.5 kg/year, respectively.
- Measurement of the U/L ratio can aid in the evaluation of children with growth disorders.
- The upper-to-lower body segment ratio (U/L ratio) can be used to assess body proportions.
- A high U/L ratio indicates a relatively longer trunk and shorter legs, and it can be seen in skeletal dysplasias and precocious puberty.
- A low U/L ratio indicates a relatively shorter trunk and longer legs, and it can be seen in Klinefelter syndrome and delayed puberty.



## PREP Pearls – Constitutional Growth Delay

- Puberty is considered delayed in boys when there is a lack of testicular growth = 4 mL in volume (or 2.5 cm in length) by age 14 years. In girls, puberty is delayed when there is a lack of breast development by age 13 years.
- Constitutional delay of growth and puberty should be suspected in otherwise healthy children growing at a normal prepubertal growth velocity, and with a family history of delayed puberty.
- Constitutional delay is a diagnosis of exclusion and is considered a normal variant.



## PREP Pearls – Constitutional Growth Delay

- Treatment of constitutional delay consists of reassurance regarding final adult height with the option of a short course of sex steroid therapy for those who have significant distress regarding their short stature and delayed puberty.
- Constitutional delay of puberty in boys with associated psychosocial distress is an indication for a short course of testosterone to accelerate pubertal development and promote earlier initiation of the pubertal growth spurt.



## PREP Pearls – Idiopathic Short Stature

- Children with idiopathic short stature (ISS) have height at or below the 1.2 percentile, with a predicted height (based on bone age) of less than 5’3” for men and 4’11” for women, and no clearly identifiable diagnosis predisposing them to have poor growth.
- Referral to a pediatric endocrinologist is appropriate for evaluation and management of children with ISS, because this is an US Food and Drug Administration-approved indication for GH treatment.
- Children with ISS, despite a normal growth velocity, respond to GH treatment with a mean height gain of 3 inches beyond predicted final height.
- Growth hormone is approved by the US Food and Drug Administration for children with idiopathic short stature whose height is more than 2.25 standard deviations below the mean (< 1.2%) and who are unlikely to catch up in height.



## PREP Pearls – Short Stature

- An endocrine cause of poor growth is likely when linear growth slows down or arrests while weight gain is either normal or increasing.
- In children with normal growth velocity and normal final height prediction, reassurance is appropriate and no further evaluation is needed.
- Children with growth hormone deficiency (GHD) typically have short stature, abnormal linear growth velocity, and delayed osseous maturation.

## PREP Pearls – Chronic Disease and Growth Velocity

- An endocrine cause of poor growth is likely when linear growth velocity declines, but weight gain is either normal or increasing.
- In the majority of cases in which poor growth is related to under nutrition, falloff in weight gain will precede falloff in linear growth.
- Linear growth impairment is common in chronic kidney disease and is the result of multiple, complex causes including inadequate nutrition, metabolic acidosis, fluid and electrolyte imbalance, metabolic bone disease, severe anemia, and growth hormone insensitivity.
- Dialysis and renal transplantation for children with chronic kidney disease improves growth, but will not make up for the deficits that already occurred.



## PREP Pearls – Craniopharyngioma

- After age 3 years and outside of the peripubertal period, crossing height percentiles is never normal and should be evaluated.
- When height velocity is decreased with relative preservation of weight, an endocrine or genetic disorder should be suspected.



## PREP Pearls – Growth Hormone Deficiency

- In those with congenital growth hormone deficiency, linear growth becomes abnormal at about the age of 6 to 12 months.
- Patients with acquired growth hormone deficiency should undergo investigation for a brain neoplasm and other pituitary hormone deficiencies.
- The weight-for-length or body mass index curve is important to consider in narrowing the differential diagnosis of abnormal growth.



## PREP Pearls – Turner Syndrome

- The most common clinical feature seen in Turner syndrome is poor linear growth (short stature).
- Turner syndrome should be considered in all girls with short stature.
- Turner syndrome can present with normal physical examination findings other than poor growth or delayed puberty. Girls with unexplained short stature should have karyotyping performed.
- Primary ovarian failure is common in Turner syndrome and manifests as delayed puberty or abnormal pubertal progression.



## PREP Pearls – Cushing Syndrome

- An endocrine cause of poor growth is likely when linear growth slows down or arrests but weight gain is either normal or increasing.
- Cushing syndrome in children is characterized by linear growth failure, weight gain, pubertal changes from excess androgen production, as well as signs and symptoms similar to adults (easy bruising, facial plethora, myopathy, striae).
- Other signs and symptoms of Cushing syndrome include round face, facial plethora, violaceous abdominal striae, hypertension, easy bruising, and proximal muscle weakness.
- The most common cause of Cushing syndrome in children is exogenous steroid exposure. Endogenous Cushing syndrome is very rare in children.



## PREP Pearls – Tall Stature

- Familial tall stature is the most likely diagnosis when there is proportional growth (height, weight, and head circumference) consistent with family history and with no other concerning findings.
- Syndromic causes of tall stature are commonly associated with abnormal upper-to-lower body segment ratios, developmental delay, and characteristic facial findings.

## PREP Pearls – Klinefelter Syndrome

- Klinefelter syndrome, or 47,XXY syndrome, is the most frequent chromosomal disorder in males with gynecomastia and small testes; the diagnosis is often missed until adulthood.
- Other features include tall stature with disproportionately long legs and language delay.
- The best diagnostic test for a sex chromosomal disorder such as Turner syndrome or Klinefelter syndrome is a karyotype.
- Testosterone replacement therapy, as well as identification and management of the comorbid disorders, remain important outcome determinants for children with Klinefelter syndrome.



## Questions…



## PREP 2016 Question 61

- A 14-year-old adolescent was recently evaluated in the emergency department for vomiting, which has now resolved.
- As part of his evaluation, he had a comprehensive metabolic panel, which was normal except for an alanine aminotransferase of 54 U/L and aspartate aminotransferase of 70 U/L.
- Abdominal ultrasonography obtained at that time showed fatty deposition in the liver.
- On physical examination today, you note a body mass index greater than 95th percentile for age and acanthosis nigricans.
- His parents ask how his condition can be most effectively treated.



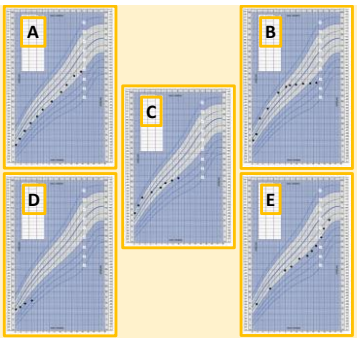
## 61., 2

- Of the following, the MOST effective initial treatment approach for this adolescent is
  - A. bariatric surgery
  - B. diet supplementation with vitamins E and C
  - C. family-based behavioral treatment
  - D. metformin
  - E. ursodeoxycholic acid



## PREP 2010 Question 58

- Of the following growth curves, the one MOST likely to be associated with familial short stature in a boy who had a birthweight of 3.3 kg is
  - A. Chart A
  - B. Chart B
  - C. Chart C
  - D. Chart D
  - E. Chart E



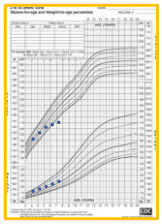



## PREP 2014 Question 42

- A 7-year-old at her well child check is noted to be growing poorly.
- Her height has gone from the 25th percentile to well below the 3rd percentile, while her weight is still at the 25th percentile.
- The mother reports her daughter is a picky eater but is otherwise well.
- The girl has no signs or symptoms of constipation, diarrhea, or malabsorption.

## 42, 2

- The family is not concerned, because the mother is 5 feet 1 inch (155 cm) tall and the father is 5 feet 6 inches (168 cm) tall.
- Both parents had delayed puberty with growth spurts in their late teenage years.





## 42, 3

- The MOST likely diagnosis is
  - A. Constitutional delay of growth
  - B. Crohn's disease
  - C. Familial short stature
  - D. Hypothyroidism
  - E. Inadequate caloric intake



## Resources

Pediatric Review Education Program
American Academy of Pediatrics
True Learn COMLEX Level 2 CE