REVIEW ARTICLE

PUBERTY: AN APPROACH TO DIAGNOSIS AND MANAGEMENT WITH AN OSTEOPATHIC COMPONENT

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KEYWORDS:

Diagnosis

Management

Osteopathic approach

Osteopathic manipulative treatment

Puberty

ABSTRACT:

Puberty is generally known as the emotional and physical maturation of a child to adulthood. This allows for sexual maturation and the means to reproduce. Children will undergo a pubertal growth spurt, as well as changes to the reproductive organs. While puberty is mostly associated with changes in reproduction and endocrine systems, it is multifaceted and affects the musculoskeletal, behavioral and vascular systems.

Puberty occurs due to activation of the hypothalamic-pituitary-gonadal (HPG) axis and a progressive increase in the amount of gonadotropin-releasing hormone (GnRH) released. The average age of puberty is 13 years old in girls and 14 years old in boys. Associated pubertal diseases are usually split into two categories, based on whether the physical indicators appear earlier or later than expected. When these indicators occur at two standard deviations (SD) early, it is known as precocious puberty, and when they are 2–2.5 SDs late, it is known as delayed puberty.

Because of the inseparability of physical and mental health, osteopathic medicine offers a practical approach for treatment of pubertal conditions using osteopathic manipulative treatment (OMT). Osteopathic medicine takes a holistic view of the person in which somatic, visceral and psychological dysfunction are united. Thus, physicians who incorporate OMT into their practice will be able to aid in promoting proper development during puberty as well as addressing accompanying somatic dysfunctions.

In this paper we will discuss the physiology of puberty, pubertal disorders, the epidemiology of puberty, current management protocols, osteopathic considerations in puberty and OMT's role in treatment.

INTRODUCTION

Puberty is generally known as the emotional and physical maturation of a child to adulthood, allowing for sexual maturation and the means to reproduce.[1,2] Children also undergo a pubertal growth spurt, as well as changes to the reproductive organs. In females, this manifests as growth of the ovaries, uterus and breasts, along with pelvis and hip shape change.[2,3] While puberty is mostly associated with changes in reproduction and endocrine, it also causes general changes in the body, affecting multiple bodily systems like the musculoskeletal, behavioral and vascular systems.[4,5]

Girls tend to start puberty around 8–13 years old.[5] Pubertal onset is marked by breast development (thelarche) along with the growth of pubic hair and is complete with menarche, which occurs about 2–2.5 years later. The menstrual cycles initially are irregular but over time become more regular.[2–4] Males usually enter puberty around the age of 9–14 years old.[5] The onset of puberty is marked by an increase in testicular volume to 4 mL, as well as development of body odor and pubic hair.[2] Males have growth of the penis, as well as growth of pubic and facial hair; their voice becomes deeper; and they also start to develop sperm in the testes.[2] The Tanner scale—developed in 1969–1970—is used to identify the development of pubic hair, breast development in females and genital growth in males.[2]

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 Print ISSN: 1877-573X
DOI:10.33181/13049

NORMAL PHYSIOLOGY

Puberty occurs due to activation of the hypothalamic-pituitary-gonadal (HPG) axis and a progressive increase in the amount of gonadotropin-releasing hormone (GnRH) released.[2,5] In utero and in early childhood, the HPG axis is active, but then it remains latent until puberty. This short activation is known as a "mini-puberty."[3,4] Once reactivated, the hypothalamus will secrete GnRH in pulses. GnRH activates the anterior pituitary, causing the secretion of

hormones—such as the follicle-stimulating hormone (FSH) and the luteinizing hormone (LH), among others—also in pulses.[1–3]

The other hormones released from the anterior pituitary include prolactin, the growth hormone (GH), the thyroid-stimulating hormone (TSH) and the adrenocorticotropic hormone (ACTH).[6] Prolactin functions to stimulate development of breast tissue and milk production.[7] GH affects the rate of growth of the body. TSH activates the thyroid gland follicular cells, resulting in the release of thyroid hormones T4 and T3. ACTH stimulates the release of androgens and glucocorticoids from the adrenal cortex. Thyroid hormones and glucocorticoids have widespread effects throughout the body.[6]

Initially, the release of FSH and LH is nocturnal, then increased for release throughout the day.[3,4] These 2 hormones then eventually cause the production of sex hormones and stimulate the gonads to mature.[1–3] FSH causes maturation of the germ cells, while LH causes production of estrogen and testosterone in females and males, respectively. Production of sex hormones allows for the secondary sexual characteristics to develop.[3] Pubic hair starts to grow due to androgens in both males and females and may start before, during or after pubertal onset.[8,9]

In males, testosterone levels increase to 10–30 nmol/L.[3] Increase in hormones will cause a growth spurt that tends to occur at Tanner stages 3–4 and testicular enlargement to greater than 4 mL at Tanner stage 2 in males. Females undergo a growth spurt at Tanner stage 2 and will begin to develop breasts at Tanner stage 2 as well.[3,8] While pubertal triggers remain unknown, it is known that the environment, genetics and neuroendocrinology play some role.[1,3] A child's skeletal maturation and onset of puberty seem to be related, because when the child achieves pubertal bone age, they usually then enter puberty.[4]

Other visceral changes that occur due to puberty affect various bodily systems. One major effect of puberty is a growth spurt that happens for 2–3 years.[5] While there is a difference between the sexes in how the growth spurt manifests, they both have changes in hormones and serum enzymes. Sex hormones indirectly and directly trigger the growth spurt by stimulating the production of GH.[5] Estrogen is needed for the fusion of the epiphyseal plates while androgens allow for wider bones.[4] Prompt bone growth results from high osteoblastic activity. This can be indicated by seeing an increase in the total concentration of alkaline phosphatase.[5]

Girls' bodies tend to grow nonuniformly, making certain parts look larger or out of proportion. The feet, head and hands are usually the first parts to grow, and the hips will also widen.[2] This starts at thelarche Tanner stage 2 and pubic hair Tanner stage 2, and it ends at the third Tanner stage for thelarche and pubic hair.[5] Boys' upper bodies, mainly the chest and shoulders, will broaden. Their muscle mass will also increase.[2] This starts at male genital Tanner stages 3 and 4, which is also when spermarche starts.[5] During male genital Tanner stage 4, boys start growing facial hair and undergo a deepening of their voice, due to a lengthening and thickening of the vocal cords, along with an increase in the size of the larynx. During this change, the voice can occasionally break.[2,5]

Along with a growth spurt, there is also a change in body composition leading to an increased body mass index. This occurs as androgens promote fat stores and muscular development, while estrogens stimulate lower body fat distribution and lipogenesis.[4]

Another common change in both boys and girls due to androgens occurs in the skin. Sebaceous glands are stimulated by increased oils in the skin, meaning more sebum is produced. The sebum mixes with extra skin cells leading to clogged pores, allowing bacteria, which can cause acne to grow. Once puberty is complete and the hormones level out, the acne will resolve, but acne can also be treated using antibiotics or other medications.[2]

The male apocrine sweat glands will increase the production of sweat in response to an increase in hormones. When bacteria come into contact with the sweat, an odor is created. The increased sweating leads to an increase in body odor in males. While this is normal, it can be reduced by basic daily hygiene care.[2]

Androgens can also lead to changes in behavioral health. During puberty, children's bodies undergo physical changes that can lead to issues regarding the body, especially those leading to bulimia nervosa and anorexia nervosa.[2] Males also have an increased likelihood of risky behavior due to the increase in testosterone during puberty.[2,5] However, there is some degree of normal risk-taking behavior in pubertal adolescents. There are many other factors that play a role in an individual's behavior, like family history and interaction, interactions with friends and other groups, and their view of their physical changes. There is also an increased risk of depression during puberty.[5]

During puberty, there are changes to the blood and serum. The concentration of hemoglobin will increase to about 13.5 g/DL, the mass of red blood cells will increase along with the growth spurt, and the total and bone-specific concentrations of alkaline phosphatase will both increase. Other changes include an increased blood pressure and a small increase in triglycerides, along with a decreased concentration of aldosterone, renin, thyroid hormones, phosphate and calcium.[5]

DISEASES/PATHOLOGY

Pubertal diseases are usually divided into two categories based on whether physical indicators appear earlier or later than expected. When these indicators occur at 2 standard deviations (SD) early, it is known as precocious puberty, and when they are 2–2.5 SDs late, delayed puberty.[1,3] While these symptoms are caused by pubertal diseases, they can also be caused by other pathologies that must be ruled out before making a proper diagnosis of pubertal diseases. Most of the time, the causes of both precocious and delayed puberty are not serious, as these are considered as extreme variants of the normal timing of puberty.[5]

PRECOCIOUS PUBERTY

In precocious puberty, girls have earlier development of breasts, while boys have earlier testicular enlargement due to an earlier activation of the HPG axis.[5,8] The established threshold

for precocious puberty in males is 9 years old.[5] There is a discrepancy in the female threshold, as puberty is considered precocious in Black girls when puberty occurs at 6 years old but 7 years old in other races.[8,9] Obesity in early childhood, a low birth weight, possible exposure to chemicals that disrupt the endocrine system and a family history of early maternal menarche, among many other triggers, are associated with precocious puberty in females.[8]

Activation of the HPG axis is usually caused by different triggers in boys and girls. While there is usually an idiopathic cause in girls, boys tend to have a pathological cause, often an intracranial pathology.[3] Gonadotropin-independent precocious puberty—a type of precocious puberty—occurs when there is an exogenous origin of androgens or excess secretion of androgens from the adrenal glands and the gonads.[3,4] A variant of precocious puberty is when there is no associated pathology. When signs of early puberty occur alongside neurological symptoms, there usually is an intracranial pathology, such as a cyst or tumor.[2,3] This is why it is precocious in Black girls when puberty occurs at six years old but seven years old in other races.[8,9] Obesity in early childhood, a low birth weight, possible exposure to chemicals that disrupt the endocrine system and a family history of early maternal menarche, among many other triggers, are associated with precocious puberty in females.[8]

In terms of mental health, it has been noted that both males and females who go through puberty earlier tend to be more popular. However, there is a difference between sexes in the negative effects seen. Boys tend to be more likely to partake in not only aggressive and antisocial behaviors but also sexual acts starting at a younger age. Girls tend to have increased rates of mental disorders like anxiety, depression, eating disorders and self-image issues.[5]

DELAYED PUBERTY

When girls lack breast development or boys lack testicular enlargement (genital Tanner stage 2) by the average age of 13 years and 14 years old, respectively, it is clinically known as delayed puberty.[1,5] This usually is caused by pathology that delays the activation of the HPG axis.[3] The most common cause of delayed puberty in both males and females is constitutional delay of growth and puberty (CDGP). According to a study from a referral center, 30% of girls and 65% of boys diagnosed with delayed puberty had CDGP.[1]

While the cause of CDGP remains relatively unknown, there seems to be a genetic influence.[1,3] A multitude of other factors also can cause delayed puberty, like chronic disorders (systemic, endocrine or metabolic) and malnutrition.[5] Two other types of delayed puberty are due to hypergonadotropic hypogonadism and hypogonadotropic hypogonadism.[1,3] Hypergonadotropic hypogonadism occurs due to gonad damage or failure, meaning there is no negative feedback from the gonads, leading to elevated levels of FSH and LH.[1,3]

Hypogonadotropic hypogonadism results when there is either a delay or an absence of the central HPG axis activation.[1,3] This can be due to multiple causes, including hypothalamic-pituitary deficiency from pituitary damage or a pathology.[3] Hypogonadotropic hypogonadism can be either transient or permanent, but both cause low levels of both FSH and LH. If transient, the delayed puberty is due to a pathology that causes a delayed maturation of the HPG axis. If permanent, either a pituitary or hypothalamic disorder caused the delayed puberty.[1]

ISOLATED PUBERTAL CHANGES

Pubertal variants include isolated pubertal changes.[3] Isolated precocious thelarche is when breast tissue development occurs without any other signs of puberty, like pubic hair development and increased bone age. This usually occurs before the child is 2 years old. While there is a prominent FSH peak in luteinizing hormone–releasing hormone (LHRH) tests, LH levels are within the normal range. In most cases, no further evaluation or treatment is necessary. This situation may also occur in children with severe primary hypothyroidism. A referral to a pediatric endocrinologist may be necessary if there is any advancing breast growth.[3,8,10]

Isolated precocious menarche is when there is vaginal bleeding in the absence of the development of pubic hair or thelarche, and no genital trauma in 5–8-year-old females. This is very rare, and in these cases, any vaginal lesion should be ruled out along with an ultrasound examination to ensure normal female anatomy, including a normal uterus. This can also occur in children with severe primary hypothyroidism. Sexual abuse is also a consideration, especially in the absence of an organic cause, and cannot be ruled out without proper investigation.[3,8]

Isolated precocious pubarche is the development of only pubic hair. There can also be axillary hair, acne and body odor. Normal levels of serum cortisol precursors should be measured after corticotropin stimulation.[8] Isolated adrenarche, or adrenal maturation, occurs in children between 6 and 8 years old because of the adrenal zona reticularis maturation. This presents differently in males and females. Males present with secondary hair but no testicular enlargement before the age of 9 years old. Females also present with secondary hair but there is no thelarche before the age of 8 years old. This is usually a benign finding, but clinicians should look for any adrenal gland steroidogenesis defect or an adrenal mass and refer the patient to a pediatric endocrinologist if suspected.[3,10]

If an infant has the unusual development of fine, straight genital hair over the scrotum or along the labia and a normal physical exam with no other signs of puberty, a pediatric endocrinologist referral is not necessary, and all that is needed is reassurance to the parents. This could become concerning if the child also starts to develop breasts or signs of progression of true puberty, in which case hormone testing is indicated, as it could be secondary to a disease process.[10]

EPIDEMIOLOGY

The average age of puberty in most children is 13 years old in girls and 14 years old in boys. About 50%–80% of all pubertal timing diseases have a strong genetic influence. Of all the children diagnosed with CDGP, 50%–75% of them have family

who have also been diagnosed with a type of delayed puberty. A study from a referral center found that 30% of girls and 65% of boys diagnosed with delayed puberty had CDGP.[1] Precocious puberty is more common in girls, and 90% of the time, it is idiopathic. When precocious puberty occurs in boys, only 50% of the cases are due to idiopathic reasons. Delayed puberty is more common in boys.[5] Girls had a 25% frequency of being diagnosed with hypergonadotropic hypogonadism, 20% had permanent hypogonadotropic hypogonadism and 20% had functional or transient hypogonadotropic hypogonadism. Boys, on the other hand, had a 5%–10% frequency of having hypergonadotropic hypogonadism; 10% had permanent hypogonadotropic hypogonadism; and 20% had functional or transient hypogonadotropic hypogonadism.[1]

DISCUSSION OF CURRENT PRACTICES AND MANAGEMENT

Puberty is evaluated by using inspection and the Sexual Maturity Rating staging or Tanner staging.[5,8] There are three scales used: female breasts (thelarche), male genitals, and pubic hair for both sexes. In girls, the thelarche Tanner stage 2 defines gonadal development, and male genital Tanner stage 2 defines testicular enlargement. At stage 3, pubertal development continues and is complete at Tanner stage 5, which identifies the adult stage.[5] The onset of puberty in girls is marked by thelarche, and in boys it is marked by testicular enlargement to > 4 mL.[8,9] When evaluating a patient with any pubertal disease, it is imperative that a complete family history is obtained and any underlying disease is ruled out.[1,8]

One of the main aspects of pubertal disease management is a thorough physical exam, along with an in-depth history, including the child's nutritional intake. The child's growth should also be reviewed using growth charts and pubertal staging via the Tanner stages.[1,3,8] The development of pubic hair should also be noted, as the presence of only pubic hair without thelarche in females can indicate an adrenal disorder. Mental disorders can play a role in pubertal development as well. It has already been noted that increased levels of mental disorders have been seen in girls with precocious puberty.[5,8]

If a problem is suspected regarding pubertal development, a boy's testicular volume should be measured. Changes in the volume can indicate their root cause. Central precocious puberty has an increase in testicular volume similar to that of normal puberty, but the volume remains the same—or prepubertal—in cases of peripheral precocious puberty secondary to testicular disorders.[8,9] The timing of pubertal progression can indicate whether delayed puberty is due to CDGP or due to permanent hypogonadism. In CDGP, the progression is normal, while it is either slow or absent in permanent hypogonadism. Many children with delayed puberty present with slow growth and a short stature for their age.[1]

In practice, diagnostic tests are ordered if an accurate assay can be obtained along with a consultation from a pediatric endocrinologist.[4] A multichannel platform assay is generally used. To diagnose a pubertal disorder, GnRH-stimulated values are usually used, due to the cyclic and diurnal differences of hormonal concentrations. Pubertal onset is usually marked by a peak of LH> 4.0 U/L when testing with either a GnRH agonist or GnRH.[4]

Hypothalamic-pituitary function is usually tested by measuring the levels of basal gonadotropins (FSH and LH) via sandwich immunoassay.[3] Delayed puberty—along with hypergonadotropic hypogonadism—is indicated by increased baseline levels of FSH and LH. If the hypothalamic-pituitary axis is damaged, baseline levels of FSH and LH are lower than normal. If a patient has precocious puberty and a pubertal response is observed with either a gonadotropin-releasing hormone agonist (GnRHa) or an LHRH test, that is suggestive of gonadotropin-dependent precocious puberty. If it were gonadotropin-independent precocious puberty, there would be no response when completing the GnRHa or LHRH tests.[3]

Gonadal function is assessed by measuring the levels of testosterone and estradiol. Regarding testosterone, it is mostly bound to proteins in circulation. The free testosterone is what can be measured using equilibrium dialysis and ultrafiltration.[3] Using testosterone levels for diagnoses must include multiple measurements because there is overlap between the Tanner stages and reference levels.[3]

If testosterone and gonadotropin levels are increased and the boy presents with precocious puberty signs, there is most likely a central pathology. If the testosterone levels are increased but the gonadotropin levels are low, this indicates there is an exogenous source of testosterone, ie, not from the gonads.[3] If the levels of LH, FSH and estradiol are higher than expected, this indicates gonadotropin-dependent precocious puberty. Gonadotropin-independent precocious puberty levels of LH and FSH are decreased while estradiol levels are increased. Hypogonadotropic hypogonadism usually presents with low levels of estradiol and gonadotropins. If the levels of estrogens and gonadotropins are initially low but slowly increase, CDGP is suggested.[3]

Nonbiological screening can include karyotyping to rule out Turner syndrome and an ultrasound of the abdomen and pelvis to rule out any malformation or masses of structures like the ovaries, Leydig cells and adrenal glands. Skeletal maturity can be monitored via nondominant hand and wrist x-rays. Intracranial pathologies can be ruled out with cranial magnetic resonance imaging (MRI).[1,3,8]

In at least 50% of precocious puberty cases in children of 6–8 years old, there is no need for treatment, as there is either no serious underlying pathology or the symptoms tend to decrease or regress on their own. The main cause in many of these cases is obesity.[5,8]

Studies have shown the use of GnRH antagonists to be helpful in treating precocious puberty, as they create a constant stimulation of the gonadotropins from the pituitary gland. This creates a desensitization and decreases the amount of FSH and LH released. Retrospective studies have shown that stopping GnRH antagonists around 11 years old has been associated with the most ideal height. Adverse effects of this treatment can include menopausal symptoms in girls, sterile abscesses at injection sites, and headaches. Studies have shown that obesity is not a common side effect of GnRH antagonists.[8]

If a central lesion (gonadotropin-dependent puberty) is causing the precocious puberty, there is no effect on pubertal development if the lesion is treated. Peripheral precocious puberty, or gonadotropin-independent puberty, can cause the pulsatile secretion of GnRH and eventually lead to central precocious puberty. On the other hand, if precocious puberty is caused by a gonadal tumor, the recommended course of treatment is surgery. The effects of chemotherapy and radiation have not been extensively studied.[8] There are a handful of studies that indicate the use of aromatase inhibitors as being successful in a few cases, mostly in instances where McCune-Albright syndrome is the underlying cause of precocious puberty.[8]

Delayed puberty and CDGP, on the other hand, can be treated with pharmacological therapies, such as low-dose sex steroids.[1,5] These steroids allow for an increase in growth and the development of secondary sexual characteristics. Studies conducted around the use of low-dose sex steroids revealed no prominent side effects. While most of these studies have been only conducted on males, it is generally understood that females have the same response to the same therapies also without any remarkable side effects, as long as the females are given correct amounts of estrogen.[1] CDGP can also be monitored without any intervention. When delayed puberty is associated with an underlying disease, that disease is treated, which will also treat the symptoms of delayed puberty.[1]

In children with hypogonadotropic hypogonadism, low doses of sex steroids are once again used. The only difference between this and CDGP is that the doses are progressively increased for about 3 years until the levels reach those of an adult. Exogenous gonadotropins or pulsatile GnRH must be given to allow fertility induction in both males and females. If after 1 year puberty still has not been reached, it is recommended that a brain MRI is completed to rule out intracranial pathologies.[1]

Further study is required to see if the use of anabolic steroids, aromatase inhibitors or growth hormones is also useful in treating delayed puberty, as the potential side effects seem to outweigh the benefits.[1,8] Trials showed an increased adult height along with delayed bone maturation in boys taking aromatase inhibitors. Another study found deformities in the vertebral bodies and hindrance of trabecular bone development. Growth hormone was found to have minimal effect in patients with CDGP regarding height. More data on efficacy and safety are needed before anabolic steroids and growth hormones can be used as treatments.[1,8]

A referral to a pediatric endocrinologist is also a possible option when treating pubertal disorders. Many cases are considered benign and can be followed by the pediatrician, while others, like premature adrenarche and thelarche, can be referred to a pediatric endocrinologist if necessary.[10]

OSTEOPATHIC APPROACH

Osteopathic medicine was founded in 1874 by Andrew Taylor Still, MD, DO. Central to his philosophy and creation of osteopathic medicine, osteopathic manipulative treatment (OMT) aims to provide patients with the ability to restore and maintain their natural, self-healing state. The 4 major tenets of the osteopathic medical philosophy are briefly explained here[11,12]:

1. *The body is completely united; moreover, the person is a fully integrated being of body, mind and spirit*. Because of this, any alterations in any part of the system, including an individual's mental and spiritual health, affect the function of the body as a whole.
2. *The body is capable of self-regulation, self-healing and health maintenance*. Health is the natural state of the body, and the body possesses self-regulatory mechanisms that it uses to heal itself from injury. OMT's function is to restore the body's self-healing and self-regulatory ability.
3. *Structure and function are reciprocally interrelated*. The structure of a body part governs its function, and thus abnormal structure can lead to abnormal function, which can inhibit its capacity for self-healing. In the same way, function governs structure.
4. *Rational treatment is based on an understanding of these three aforementioned principles*. These basic osteopathic tenets permeate all aspects of health maintenance and disease prevention and treatment. The osteopathic physician examines, diagnoses and treats patients according to these principles.

Given the significant physical, psychosocial, emotional and physiological changes that occur during puberty, adopting an osteopathic approach by incorporating the framework of these tenets could help optimize patient evaluation and management. Along with these tenets, there are 5 models of osteopathic care that osteopathic physicians use to facilitate diagnosis and treatment by applying an understanding of the various anatomical, psychological and physiological substrates of disease: neurologic, respiratory-circulatory, biomechanical, metabolic-nutritional and biopsychosocial.

The neurologic model addresses facilitated spinal cord segments, viscero-somatic and somato-visceral reflex phenomena, Chapman points, and abnormal parasympathetic effects from cranial or sacral nerve entrapment syndromes.[13,14] At every level of the central nervous system along the spine, the neurophysiology of somatic dysfunction inseparably links viscera, soma and psyche through complex viscero-somatic, somato-visceral, somato-psychological and psychosomatic feedback interrelationships. It is hypothesized that one component of these complex relationships cannot become problematic without impacting the others, and thus, treatment of any one aspect of somatic dysfunction is not complete without consideration of the others.[15–17]

The respiratory-circulatory model addresses respiratory and fluid mechanics in the body, such as congestive changes, lymphatic flow, venous return and edema formation.[13,14]

The biomechanical model addresses factors that alter posture, motion and gait. The goal of treatment is the restoration of free motion within the musculoskeletal system.[13,14]

The metabolic-nutritional model addresses metabolism, dietary deficiencies and excesses, food allergies, and effects of toxins.[13,14]

Lastly, there is the biopsychosocial model, which addresses the psychological and social components of a patient's health, such as stress, which is a well-known contributor to illness.[13,14]

OSTEOPATHIC MANIPULATIVE TREATMENT PROTOCOL IN TREATING PUBERTY

There are currently no studies done on the effectiveness of an OMT protocol for treating puberty and its many visceral, somatic and psychological changes. Thus, we will provide a suggested OMT sequence for assessing and treating somatic dysfunctions during this important phase of maturation. This sequence will be based on the areas of interest under each of the 5 osteopathic models. In puberty, because not much is known through research about the specific somatic dysfunctions that typically occur or their impact on the maturation process, it is imperative that physicians be guided by their clinical evaluation and the osteopathic structural exam to address the most relevant findings.

First, the physician will inspect the area of interest, after discussing and obtaining consent from the patient and, when appropriate, the parent or guardian. This inspection can be done as part of a symptom-focused assessment or, ideally, as part of a more comprehensive osteopathic structural exam. Second, the physician will palpate areas of interest to examine for somatic dysfunction. By identifying key areas of change during puberty, evaluating any patient complaints and applying one's knowledge of different viscero-somatic reflexes, one can identify relevant somatic dysfunctions and try to achieve desired clinical effects through the use of OMT.

It should be noted that the order of treatment should be modified as deemed fit for any individual patient. Also keep in mind that there is overlap in the effects of OMT under each of the 5 models, so techniques described under one model can often be used for their effects under another model, thus giving the clinician great flexibility in how to apply OMT. The contraindications for OMT are relatively few and straightforward. Do not use any of the following techniques if:

1. the patient and/or guardian refuses,
2. there is no somatic dysfunction,
3. there is significant or medically undiagnosed regional pathology, or
4. the somatic dysfunction suggests an underlying pathology that should be further evaluated before any OMT is rendered.

Puberty, as discussed, is a progressive nonlinear process from prepubescence to full sexual maturity through the interaction of biological, physical and psychological changes. Somatic dysfunctions can occur during puberty and will be considered under each of these five models. Because somatic dysfunction is a prerequisite for manual intervention, all 5 of these models can be applied when formulating a treatment plan using OMT. It is important to note that these five models serve as a convenient and pragmatic way to organize our clinical understanding and reasoning of how somatic dysfunction may relate to pubertal conditions, not as an absolute division among the categories. Thus, there will be some overlap at times in our discussion.

Puberty and the neurologic model

One example of the neurologic model concept affecting the autonomic nervous system (ANS) is tension headaches, which occur more frequently with puberty, especially in females.[18] These headaches tend to be felt as a "hurting or aching" sensation occurring in a frontal and/or occipital distribution.[18] Therefore, one may expect there to be a parasympathetically involved somatic dysfunction displayed in the cranium, as well as sympathetically involved somatic dysfunctions in the T1–T4 region.[19] Generally speaking, conditions in the head and neck can be associated with viscero-somatic responses in the T1–T4 region.[19] Further discussion of OMT in ANS dysfunctions will also be included in other models.

The parasympathetic nervous system exerts all its neurological control through the vagus nerve and the sacral plexus.[19] Thus, one can treat both areas to normalize parasympathetic influences. These techniques have parasympathetic effects:

- Influence on the vagus nerve, which controls the parasympathetics to the head, neck, heart, lungs, and upper and middle gastrointestinal system, by suboccipital release
- Repair of cranial and facial bone dysfunctions due to cranial treatments
- Myofascial release to the C1–C2 region to influence the vagus nerve
- Sacral rock and muscle energy of the sacrum, during which the sacral plexus provides the parasympathetic influences on the lower gastrointestinal system and lower genitourinary system viscera[19]

Suboccipital release is thought to exert its effects on the autonomic nervous system by influencing the vagus nerve. In fact, studies show suboccipital release has the capacity to modulate pain-induced autonomic control and regulation,[20] as well as affecting heart rate variability acutely.[21]

Puberty and the respiratory-circulatory model

One key area of change during puberty is the lungs, where studies suggest that pubertal growth leads to an increase in both FEV1 and FVC.[22] Studies also show that respiratory muscle endurance is significantly lower in prepubertal children when compared to children near the end of puberty.[23] Hence, the anatomical and physiological changes of the lungs suggest that there is a chance for dysfunction to arise during this maturation process. Through the viscera-somatic reflex arc, dysfunction of the respiratory system can manifest as somatic dysfunction in the T2–T4 region.[19]

Growth of the chest wall and lungs can also result in somatic dysfunction of the thoracic spine and ribcage, which in turn can affect respiratory function and lymphatic return, potentially leading to chest wall pain or back pain. While not necessarily indicative of a viscero-somatic reflex, the parallel growth and maturation of the musculoskeletal system and the internal organs

To treat this region, after diagnosing the dysfunctions, one could use:

- Rib raising bilaterally to normalize the sympathetic input to the area.
- Muscle energy to treat exhalation or inhalation dysfunctions of the ribs. This may remove the influence of rib dysfunctions on the ability of the lungs to properly expand, contract and function.
- Direct balanced ligamentous tension (BLT) to work on particular thoracic or rib dysfunctions.
- Doming of the diaphragm to relax the diaphragm, allowing the lungs more room to move and oxygenate the blood more effectively.
- Counterstrain, facilitated positional release (FPR) and other soft tissue techniques. These are techniques that most primary care providers would typically feel comfortable doing and most patients would be comfortable receiving.
- Thoracic and rib high-velocity, low-amplitude (HVLA) techniques, if deemed necessary.

Rib raising is a simple and commonly used technique that has been shown to be helpful in the regulation of the sympathetic nervous system. A recent study suggests that sympathetic nervous system activity may decrease immediately after rib raising, but as expected, this technique does not alter the hypothalamic-pituitary-adrenal axis and parasympathetic activity. Moreover, this was confirmed through the usage of salivary alpha-amylase as a biomarker.[25]

Another area of key change is the cardiovascular system. Physical symptoms that reflect ANS dysfunction can occur at the onset of puberty, compromising the homeostatic regulation of basic bodily functions. With respect to cardiovascular function, puberty is associated with an increased incidence of syncope, a transient loss of consciousness and postural tone—or presyncope—and a near-fainting experience, particularly in females, though both sexes are affected.[18] Through both sympathetic and parasympathetic innervation in the cardiovascular system, one can deduce that somatic dysfunctions may be present because of a viscero-somatic reflex. These reflex changes, as well as those from the respiratory system, would manifest at the T1–T5 region for the sympathetics, and the occiput, C1 and C2 regions for the parasympathetics.[19]

Given the overlap of the respiratory and cardiovascular systems, in addition to the treatments for the respiratory dysfunctions already described above, one could use:

- Rib raising bilaterally to normalize the sympathetic inputs to this region
- Muscle energy to treat exhalation or inhalation dysfunctions of the ribs
- Direct BLT to treat particular thoracic or rib dysfunctions
- Counterstrain, FPR and other soft tissue techniques
- Thoracic and rib HVLA, if deemed necessary

Puberty and the biomechanical model

Skeletal growth is one of the most striking characteristics of puberty and one of the first changes noticed in an adolescent. Moreover, linear-growth peak height velocity is attained at age 14 years old in boys and 12 years old in girls.[26,27] With the acceleration of spinal growth, practitioners should be aware of issues with scoliosis and posture in general. While it could be idiopathic, scoliosis has many occupational and environmental causes. Because many individuals at this age are students who may have heavy backpacks and are also sitting a lot—sometimes with poor posture—the risk of scoliosis increases. This can lead to back pain, as well as visceral restrictions, if severe enough.[28,29] Unsurprisingly, musculoskeletal pains and headaches are the most reported symptoms among those with advanced pubertal status.[30] It should be noted that headaches are more prominent among girls; however, musculoskeletal complaints are predominant in boys.[30]

Moreover, some studies show a weak, and others a strong, positive association between puberty and back pain, which remains after controlling for age and sex. These studies also show that results were consistent across the studies and that there was a linear increase of back pain, according to the stage of puberty.[31] In addition, puberty is a period often associated with more intense physical activity, such as competitive team sports, and thus the risk of overuse or traumatic injuries increases.

Because OMT is particularly effective at treating musculoskeletal pain, this modality can serve an important role during this period of physical maturation.[32] OMT offers a wide array of treatment options to reduce pain from head to toe and a full-body osteopathic structural exam (OSE) to diagnose a wide array of somatic dysfunctions. By diagnosing and correcting somatic dysfunctions, OMT can help optimize body mechanics and play an important role in promoting peak physical performance, as well as helping to both treat and potentially prevent injuries.

Studies show muscle energy and counterstrain techniques can play a significant role in lower back pain (LBP) from injuries.[33] They can lead to a reduction in pain and disability, and even an increase in lumbar flexion range of motion (ROM) immediately after one treatment session.[33] Moreover, these techniques can lead to a reduction of pain and disability.[33] An increase in lumbar ROM was observed in acute LBP patients following 2 treatment sessions.[33]

The variety of musculoskeletal pains can range anywhere from long bones to the erector spinae muscles, and thus it would be impossible to create a formulaic OMT protocol for this. However, if the patient is complaining of body aches, the physician should check the areas for somatic dysfunction and treat accordingly with OMT. In general, attention should be given to the head and neck, spine, and major joints (eg, shoulders, elbows, wrists, hips, knees, ankles) to optimize biomechanics.

Muscle energy, counterstrain and FPR can generally be applied to treat somatic dysfunction and are safe and well tolerated. Thus, physicians who find themselves treating unfamiliar regions can often employ these widely applicable techniques in addition to other techniques that they deem appropriate.

Puberty and the metabolic-nutritional model

Nutrition is one of the most important factors affecting pubertal development. Consuming an adequate and balanced healthy diet leading into and during puberty is necessary for both proper growth and normal pubertal development. However, some evidence suggests that obesity can accelerate the onset of puberty in girls and may delay the onset of puberty in boys.[34]

Consequently, severe primary or secondary malnutrition can delay the onset and progression of puberty.[34] This is why anorexia nervosa and bulimia during adolescence impose a nutritional risk on pubertal development. Moreover, many environmental endocrine disruptors can significantly impair the normal course of puberty as well.[34] OMT's role in this model is a little less clear but may still have some benefit if used strategically. For example, although previously covered under the respiratory-circulatory model, normalizing somatic dysfunctions of the thoracic spine and ribcage can optimize lymphatic flow. This in turn could potentially enhance clearance of toxins from body tissues that can affect metabolic function and maturation of organs and other body tissues. In the case of anorexia nervosa and bulimia, OMT can potentially ease some of the psycho-emotional stress that often accompanies these conditions if incorporated into the medical management plan.

With respect to viscero-somatic reflexes, the stomach and small intestine contain input from T6 to T10, and the large intestine from T11 to L1.[19] The parasympathetics to the upper and middle gastrointestinal tract are controlled by the vagus nerve, while the lower gastrointestinal tract is controlled by the sacral plexus.[19] If the sympathetic and parasympathetic innervations of the gastrointestinal tract are both normalized, then it would be reasonable to conclude that digestion of food will be more optimally regulated.

The following is a sample sequence one could use after diagnosing for somatic dysfunctions:

1. Rib raising bilaterally to normalize the sympathetic inputs to this region
2. Lumbar myofascial release to treat the lumbar region
3. Direct BLT to treat particular thoracic, lumbar or rib dysfunctions
4. Mesenteric lift to relieve the intestines and their mesentery of restrictions
5. Hepatic and splenic pumps to support organ function and support fluid circulation
6. Counterstrain, FPR and other soft tissue techniques
7. Thoracic and lumbar HVLA, if deemed necessary

One basis for the role of OMT in gastrointestinal conditions is that it has been used successfully for post–abdominal surgery patients to prevent ileus. A study found a significantly shorter time to first postoperative flatus in the OMT-provided group compared to the non–OMT-provided group by an average of 1.5 days.[35] Other studies indicated beneficial trends of bowel function in the OMT groups, and none of these studies reported adverse or negative findings regarding bowel movement after OMT.[36]

Puberty and the biopsychosocial model

Puberty is a formative transition, marked by great biological, psychological and social challenges.[36] During this stage, youth experience dramatic physical transformations, as previously noted. Moreover, this period is defined by brain remodeling and alterations in hormonal systems involved in sexual maturation and stress reactivity. Apart from these physical and biological changes, youth undergo psychological changes, reflected in self-perception and self-regulation, shifts in the dynamics of interpersonal relationships, and contextual changes like school transitions.[37]

Most of these changes—physical and psychological—are characterized by changes to the body due to 3 endocrine events: adrenarche, gonadarche and activation of the growth axis. The gonadal steroid hormones estrogen and testosterone, as well as their adrenal hormone counterparts, influence physical appearance, in addition to the brain and behavior.[38] Thus, the adrenal glands, gonads, thyroid and hypothalamus-pituitary-adrenal (HPA) axis all have an effect on behavioral changes during puberty, which makes the biopsychosocial model a useful way to understand and address this important phase of a child's development.

Each of the aforementioned viscera have corresponding spinal areas for viscero-somatic reflexes. Hence, assessing and treating the T8–L1 (adrenal sympathetic input), T10–T11 (gonadal sympathetic input), and C1–C2 (pituitary, adrenal and gonadal parasympathetic input) regions for somatic dysfunctions would promote the normal functioning of the involved viscera with respect to their neurological inputs and outputs.[19] When these viscera are functioning normally, hormonal regulation and the overall mental maturation process can occur more optimally.

Since these endocrine system levels overlap with the levels of the upper and middle gastrointestinal system, the physician would not need to repeat the following treatment sequence if it has already been used to address the same regions for their gastrointestinal effects. For convenience, the techniques used to address the endocrine organs are:

- Rib raising bilaterally to normalize the sympathetic inputs to this region
- Muscle energy to treat exhalation or inhalation dysfunctions of the ribs in this region
- Direct BLT to treat involved segmental dysfunctions
- Counterstrain, FPR and other soft tissue techniques
- Thoracic HVLA, if deemed necessary

Another important consideration is the psychological impact of pain. Comorbid mental health conditions, such as anxiety, depression and fear avoidance, are often associated with chronic pain. One study conducted shows that OMT was effective at

reducing pain, anxiety and psychiatric disorders that are comorbid with pain.[39] Other studies show that OMT produced a statisticallysignificant decrease in self-perceived fatigue. Thus, osteopathic manipulative treatment represents a potential modality to reduce self-perceived distress.[40] During puberty, in instances when anxiety and distress are present, OMT can be a potentially useful treatment option.

OMT EFFECTS ON PUBERTY STUDY DESIGN

A possible study could be done on the impact of OMT during puberty using a variety of clinical and validated measures. The participants would be randomly assigned into 2 groups in a prospective cohort study, using the selection criteria of adolescents with no genetic conditions or significant underlying musculoskeletal conditions. For this study, the participants would need to see their pediatrician 4 times a year from ages 8 to 15, which are average start and end points of puberty.[38]

The first group would have visits with their pediatrician every 3 months (4 times a year) from ages 8 to 15 to have a basic physical to track growth (height and weight); mental health (using a modified yet standardized depression and anxiety screening, such as the Patient Health Questionnaire-9); physical fitness (using a standardized fitness testing protocol depending on the age of the child); and overall wellness (HERO scale[41]). The second group would go through the same measurements but in each of their visits, the physician would spend a half-hour performing the OMT for somatic dysfunctions found on the OSE and deemed most relevant for any presenting complaint. The patient's metrics would be obtained at each time point. Outcome measures would look for trends and whether each timepoint falls within a "healthy" or "normal" range (these ranges are standardized by the tools themselves). The comparison of these results would give an interesting perspective regarding management of puberty-related conditions with OMT.

In addition, we could also have a qualitative component to our study design, in which we monitor and assess the subject for irritability, tension, anxiety, difficulty concentrating, diminished interest, feeling overwhelmed and sleep disturbances that they felt throughout the study period at those same time points. This could possibly show if OMT can relieve these stressors during puberty as well.

CONCLUSION

Puberty is a progressive nonlinear process starting from prepubescent to full sexual maturity through the interaction of biological, physical and psychological changes. It begins in children around the ages of 8–14 in males and females. Activation of the HPA axis, along with an increased release of GnRH, FHS and LH, activates and creates physical and behavioral changes. Pubertal diseases arise when there is either an early or a late activation of the HPA axis, resulting in precocious or delayed puberty, respectively. Precocious puberty is common in girls, while delayed puberty is common in boys. The Tanner stages are widely used as a tool to help assess a child's pubertal stage. Because most cases of pubertal diseases are benign, these cases are generally followed by the pediatrician. The four tenets of osteopathic medicine and the five models of osteopathic care can be applied when formulating a treatment plan using OMT. There are currently no studies done on the effectiveness of OMT for treating puberty-related conditions and its many visceral, somatic and psychological changes. However, the utility of OMT for a wide variety of musculoskeletal and organ-related conditions spanning all age groups suggests that OMT may play a role in the care of patients during puberty. We propose that OMT may have important effects on the somato-visceral and somato-psychological pathways and should be considered as an additional tool for use in puberty to address its many physiological, psychological and physical changes.

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