Chapter 4
Psychology and Neurobiology of Puberty

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Abstract Puberty is derived from the Latin word pubertas, which means adulthood. It represents the transition from childhood to adulthood and is characterized by physical maturation and attainment of fertility. Adolescence and puberty are not exchangeable terms. Puberty usually marks the beginning of adolescence and is a 3–4 year process where hormonal and physical changes cause an individual to reach sexual maturity. In females, the signal for maturity is the first menstrual period. Adolescence represents that time frame where social and psychological growth transform a dependent child into a functionally independent young adult; it encompasses puberty and can take up to a decade for full maturation to occur. The psychology of puberty is closely intertwined with the physiologic changes that take place as a female undergoes maturation. Physiologic changes are initiated by hormonal surges from the brain to the gonads (ovaries) which secondarily produce hormones that stimulate peripheral entities such as the breast, hair, and sexual organs. The attainment of the milestones of development is a process in which females undergo thelarche (breast development), pubarche (sexual hair development), menarche (onset of menstrual cycle), and the growth spurt. The neurobiology of puberty is a complex topic on which there is an abundance of information. The goal of this chapter is to provide basic information concerning the physiology; also the mental and behavioral characteristics surrounding this phase of life will equally be examined.

Keywords Developmental psychology • Hormonal changes • Growth curve • Bone development health • Adolescent sexuality • Thelarche • Adrenarche • Menarche • Psychosocial interactions • Early puberty • Mid-puberty • Late puberty • Adolescent

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Introduction

Puberty is derived from the Latin word pubertas, which means adulthood. It represents the transition from childhood to adulthood and is characterized by physical maturation and attainment of fertility. Adolescence and puberty are not exchangeable terms. Puberty usually marks the beginning of adolescence and is a 3–4 year process where hormonal and physical changes of the body cause an individual to reach sexual maturity. In females, the signal for maturity is the first menstrual period. Adolescence represents that time frame where social and psychological growth transform a dependent child into a functionally independent young adult; it encompasses puberty and can take up to a decade for full maturation to occur.

Overview

The psychology of puberty is closely intertwined with the physiologic changes that take place as a female undergoes maturation. Physiologic changes are initiated by hormonal surges from the brain to the gonads (ovaries) which secondarily produce hormones that stimulate peripheral entities such as the breast, hair, and sexual organs. The attainment of the milestones of development is a process in which females undergo thelarche (breast development), pubarche (sexual hair development), menarche (onset of menstrual cycle), and the growth spurt. The neurobiology of puberty is a complex topic on which there is an abundance of information. The goal of this chapter is to provide basic information concerning the physiology; also the mental and behavioral characteristics surrounding this phase of life will equally be examined.

Hypothalamo-Pituitary-Ovarian System

The organs at play in the female reproductive system include the brain (hypothalamus, pituitary gland), ovaries, fallopian tube, and uterus which together form the hypothalamo-pituitary-gonadal axis (Fig. 4.1). All organs in this axis communicate and do not act independently. The hypothalamo-pituitary-ovarian (HPO) system is well developed at the time of birth and is in fact intact by 12 weeks of gestation [1, 2]. Negative feedback of hypothalamic-pituitary gonadotropin is first acquired in fetal life by midgestation [1–3]. When development is normal, the feedback mechanism ensures that the process of puberty is beautifully orchestrated at the appropriate time.

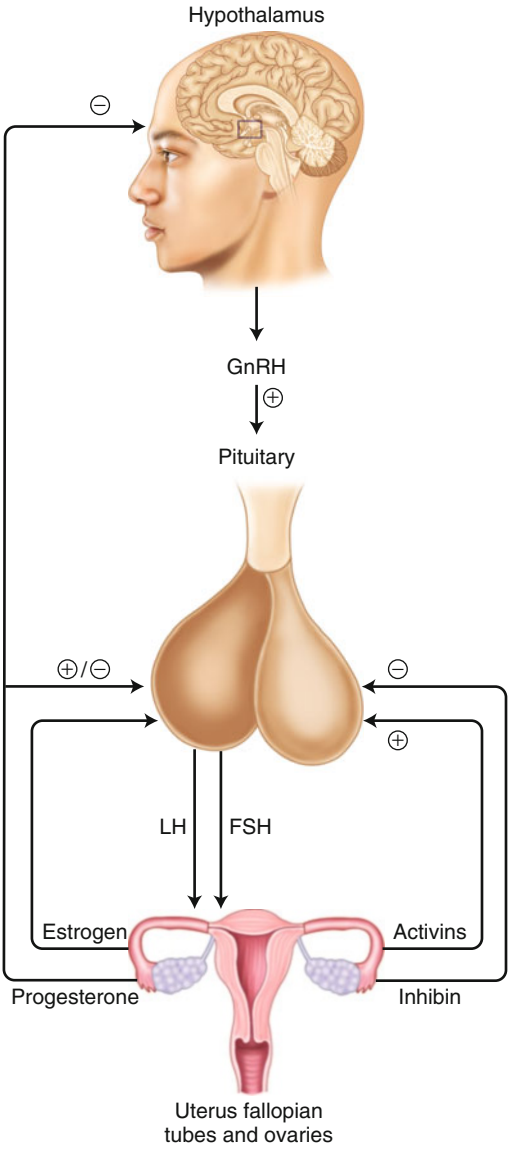

Fig. 4.1 Hypothalamic-hypophyseal-gonadal axis

Gonadotropin Releasing Hormone

The hypothalamus is an essential brain structure in the process of puberty and it produces the neurohormone, gonadotropin releasing hormone (GnRH), a 92 amino acid precursor protein encoded on the short arm of chromosome 8 with a half life of 2–4 min in vivo [4]. GnRH is responsible for the release of the gonadotropins,

follicle stimulating hormone (FSH), and luteinizing hormone (LH) from the anterior pituitary gland. The arcuate nucleus which lies within the tuberal hypothalamus contains the greatest number of GnRH-producing neurons [5, 6].

Studies have found that initially there is episodic release of GnRH in the neonatal and fetal periods and in response, FSH and LH are elevated. It is important to note that in females, FSH levels are usually greater than LH levels at this stage [2, 6]. At around 6 months of age, this process becomes downregulated and eventually dormant during childhood where GnRH secretion is minimal and intermittent, and subsequently, mean LH and FSH levels are low [7]. From the quiescent state of childhood, pulsatile release of GnRH begins with the onset of puberty and initially is expressed only during sleep. Upon establishment of functional menstrual cycles, the pulsatility prevails throughout the 24 h period [2, 8–10]. With the onset of puberty, GnRH levels increase, the levels of LH and FSH rise and once the normal menstrual cycle is subsequently established, LH secretion levels predominate that of FSH. This preferential inhibition of FSH during the reproductive years results from increasing levels of both estradiol and inhibin [7, 11].

Two main hypotheses (theories) exist regarding the mechanism of the onset of puberty. The first is the hypothalamic maturation theory which hypothesizes that the childhood downregulation of GnRH is due to central inhibition. The nature of this central inhibition is still unclear, however, gamma-aminobutyric acid (GABA), the main inhibitory neurotransmitter of the nervous system has been implicated in this process. It is thought that a critical factor in the onset of puberty is the disinhibition of GnRH-producing neurons from GABA [2, 11–13]. The second is the gonadostat theory which hypothesizes that the regulating system for gonadotropins is very sensitive in childhood which keeps the concentration of sex steroids low. During sexual maturation, this gonadostat becomes desensitized to steroid feedback and the shift in sensitivity permits gonadotropin secretion [2, 13]. Both theories agree that the onset of puberty is largely due to the upregulation of GnRH which occurs as a result of multiple complex series of events between hypothalamic oscillators, target cells, neuropeptides, neurotransmitters, and neurosteroids [14–17]. The overall process is controlled by multiple genetic and environmental factors. Several gene mutations have been identified in delayed or absent puberty and are estimated to account for approximately 30 % of individuals with disorders of puberty [18].

The suprachiasmatic nucleus of the hypothalamus (SCN) commonly known as the central circadian pacemaker is known to communicate with the arcuate nucleus, which is often described as the GnRH pulse generator; these hypothalamic oscillators are crucial in the upregulation of GnRH [19]. Target cells within certain reproductive organs (pituitary, ovaries, oviducts, uterus) have been described as possessing autonomous clocks, which contribute to the timing of events in reproductive physiology [20]. In addition, neuropeptides, neurotransmitters, and neurosteroids all underlie the onset of pubertal processes and can either act as stimulants, inhibitants or in some cases, both, depending on the timing.

This paragraph briefly mentions some of the key players that have been identified in the process of pubertal onset and is in no way inclusive of all entities involved in this poorly understood intricate process. The most potent identified activator of GnRH neurons is Kisspeptin, a neuropeptide encoded by the gene Kiss1, and whose

secretion is regulated by Neurokinin B [5, 17, 18, 21]. Studies undertaken reveal that kisspeptin is involved in virtually all aspects of reproduction from the initiation of puberty to the daily control of reproduction. Inactivation of the kisspeptin receptor causes hypogonadotrophic hypogonadism. Neuropeptide Y (NPY) is also a neuropeptide that acts as a neurotransmitter in the brain. It has been implicated as the hypothalamic brake in the absence of estrogen (before puberty) working with GABA, but once puberty starts it has been noted to stimulate pulsatile release of GnRH and in the pituitary potentiates the response to GnRH [13, 22]. An example of a peripheral player is Leptin, a protein hormone mainly produced in adipose tissue that has receptors in the hypothalamus; some noted on cell bodies on NPY neurons [23]. Serum leptin levels are proportional to fat stores in the body, and are thought to play a central function in the metabolic control of puberty. Circulating levels of Leptin have been noted to rise during the pubertal transition in females. Anorexia is associated with amenorrhea and females with this condition have lower levels of circulating Leptin and gonadotropins. In addition, individuals deficient in Leptin fail to initiate puberty which further solidifies the importance of Leptin in the process of puberty [23–26]. Adrenarche, a process which occurs prior to puberty may play a role in the maturation of the hypothalamic-hypophyseal-gonadal axis. It causes the elevation of the neurosteroid dehydroepiandrosterone (DHEA) and dehydroepiandrosterone sulfate (DHEA-S) which has been implicated in the upregulation of GnRH [27].

Gonadotropins, Follicle Stimulating Hormone, and Luteinizing Hormone

Pulsatile release of GnRH causes activation of the GnRH receptors in gonadotrope cells of the anterior pituitary which leads to the secretion of Gonadotropins, FSH, and luteinizing hormone (LH). They belong to the family of glycoprotein hormones (in conjunction with thyroid stimulating hormone [TSH] and human chorionic gonadotropin [HCG]). These specific protein hormones are structurally similar to gonadotropins as they consist of two peptide chains: a common alpha chain and a unique beta chain which confers biologic activity. Nonetheless, the gene for universal alpha subunit of LH, FSH, TSH, and HCG is located on chromosome 6, while the beta subunit gene specifically for LH and FSH is located on chromosome 19 and 11 respectively [28–30]. The gonadotropins are also referred to as sex hormones as their target organs are the gonads (ovaries or testes), where together they act synergistically and regulate many aspects of gonadal function in both males and females such as normal growth, sexual development, and reproductive function.

The female ovaries have a dual function: production of germ cells and steroidogenesis, but the ability to function requires the interplay of hormonal stimulation, in addition to the ability to respond or sense hormones. LH provides the androgen substrate for estrogen synthesis; it targets the theca cells of the ovary where androgens are produced via the process of steroidogenesis. These androgens are converted into estrogen by adjacent granulosa cells via the enzyme aromatase (CYP19),

a member of the cytochrome P450 superfamily. Receptors for LH exist on the theca cells at all stages of the cycle but only appear on the granulosa cell after the follicle matures under the influence of FSH and estradiol usually in the middle to late follicular phase. LH acts synergistically with FSH to help follicular maturation, and later plays a role in promoting follicular degradation. In addition, LH also assists with follicular rupture via prostaglandin synthesis to bring about ovulation. After release of the mature ovum from the follicle, it stimulates progesterone synthesis by the corpus luteum [31–33].

FSH primarily stimulates the growth of ovarian follicles and targets the granulosa cells of the ovary which results in estradiol production. It enables maturation of the follicle by activating enzymes essential to estrogen production, including aromatase, and ultimately stimulating LH receptors on granulosa cells. The ovarian steroids and other protein complexes produced by the ovaries in response to LH and FSH such as activin, inhibin, insulin-like growth factor, and progesterone exert feedback on the hypothalamus and on the pituitary generating the cyclic pattern of gonadotropin release characteristic of the female reproductive system [34, 35].

The menstrual cycle which will be described in detail in a later section is an attestation to the feedback mechanism. For ovulation to occur normally, which is essential in normal reproduction, there must be proper stimulation of gonadotropin release. The amplitude and frequency of the GnRH pulse varies throughout the menstrual cycle and specifically, there are two distinct modes of GnRH secretion, namely the pulsatile and surge mode. Pulsatile GnRH release drives tonic gonadotropin and regulates folliculogenesis; this tonic mode accounts for the low but pulsatile release of LH. The surge mode generates the preovulatory LH surge which triggers ovulation. In the female, pulsatile secretion is negatively regulated by estrogen and/or progesterone, and surge release is positively regulated by estrogen [6, 36].

Menstrual Cycle

Unlike males, the reproductive capacity of a female is intermittent and each female is born with a finite number of ova in the ovary. Much of the germ cell maturation occurs during intrauterine life. Primordial germ cells migrate from the entoderm of the yolk sac to the genital ridge in the second month of gestation and begin to multiply at a rapid rate. Within the genital ridge, which in females later becomes the ovary, these germ cells (now called oogonia) undergo mitosis and achieve their peak number of 6–7 million oocytes by 5–6 months of gestation. Through the process of apoptosis, the neonate eventually has 1–2 million oocytes at birth. At the time of puberty only 0.3–0.5 million oocytes remain [2, 32, 33]. Each ovary contains oocytes that are at different stages of development (Fig. 4.2). The menstrual cycle refers to the cyclic release of a single mature oocyte driven by alterations in hormonal levels and the feedback mechanism. On average the human menstrual cycle is approximately 28 days; however, a range between 21 and 37 days is considered normal [37].

In this section, we will describe the three main phases of the menstrual cycle.

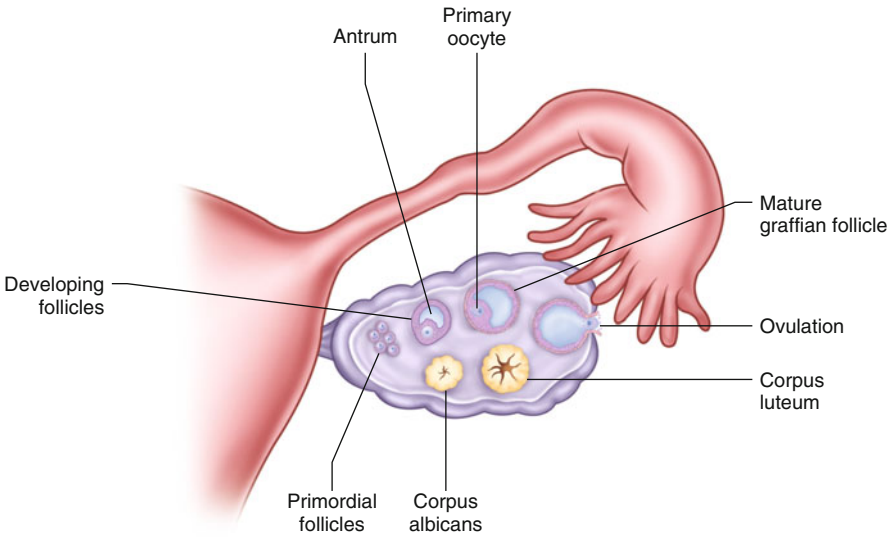

Fig. 4.2 Schematic representation of events occurring in the ovary during a complete follicular cycle

Phase 1: Follicular Phase

The follicular phase refers to the initial half of the cycle where recruitment and growth of the follicle take place; in a 28-day cycle, it corresponds to Day 0–13. Day 0 is assigned to the first day of menses when there is a low level of circulating estrogen and progesterone from the previous cycle, in response to the low levels of these hormones, the negative feedback of FSH (and to a lesser extent LH) to the anterior pituitary is removed. This stage also corresponds to the pulsatile mode of GnRH secretion where GnRH pulses can again cause an increase in LH and FSH levels. Initially, FSH release is preferentially higher due to the lower levels of estradiol which exerts a negative feedback effect on LH in the pituitary as mentioned above [11]. The increasing amount of FSH levels activates the FSH receptors on the granulosa cells and causes recruitment of follicles in the ovary which are propelled from primordial follicles to the preantral stage. At the preantral follicle stage, the follicle is a two cell (granulosa and theca cell) two gonadotropin (FSH and LH) system. Subsequently, the thriving granulosa cells cause estradiol levels to rise.

The dominant follicle also called the graffian follicle is selected between days 5–7 and this is the time that the dominant follicle develops receptors for LH. The physiologic mechanism by which the dominant follicle is selected has not been fully elucidated; however, this follicle has been shown to express a high concentration of FSH receptors [38]. As mentioned earlier, initially FSH release is greater

than LH, by mid- to late-follicular phase, an inhibitory signal develops for FSH and its level starts to decline. At this time, LH pulsatile release increases in frequency. Due to the declining level of FSH, non-selected follicles undergo atresia and the dominant follicle is spared as a result of its high density of FSH receptors and continues to secrete estradiol, and by this stage the dominant follicle also possesses LH receptors which respond to the increasing levels of LH [8–10].

Phase 2: Ovulation

As estradiol levels increases, a positive feedback loop is created for LH which leads to the ovulatory phase [11]. *Estrogen reaches a critical level at around 200 pg and is maintained for a sufficient duration of time which results in an LH surge*. The surge mode of GnRH secretion generates the preovulatory LH surge which triggers ovulation which occurs *about 36–44 h after the LH surge*, which is the process in which the oocyte is expulsed from the follicle. Though complex, the ovulatory phase is relatively short and corresponds to Days 13–14.

Phase 3: Luteal Phase

The luteal phase corresponds to Days 14–28; after ovulation, the evacuated graafian follicle becomes the corpus luteum, a temporary endocrine structure which secretes the hormone progesterone which is considered the "pregnancy hormone" as it is geared to support and maintain pregnancy. In the luteal phase, elevated circulating estradiol and progesterone suppress LH and FSH levels through negative feedback effect [39]. The lifespan of the corpus luteum is about 14 days. A sustained corpus luteum aids with implantation and retention of the pregnancy until B-HCG, produced from the fertilized egg, intervenes and contributes to maintaining the pregnancy. If the released egg is not fertilized, the corpus luteum regresses to become the corpus albicans. Progesterone is then no longer secreted and ongoing support of the endometrium is withdrawn, triggering a menstrual cycle. The levels of estradiol and progesterone plummet, and due to these low ovarian steriods, the feedback inhibition at the level of the hypothalamic-pituitary axis is removed. FSH levels start to rise and a new cycle is initiated (Fig. 4.3).

The ovaries produce other protein and hormones such as inhibin, activin, follastatin, and insulin growth factor (IGF) which play a critical role in the menstrual cycle and feedback mechanism [40]. Activin and inhibin are two closely related protein complexes that have opposite effects. Activin enhances FSH biosynthesis and secretion while inhibin downregulates FSH synthesis and inhibits FSH secretion [41]. There are two types of inhibin; A and B. Both possess a common alpha subunit but have different beta subunit. Inhibin *B* reaches a peak in the early- to mid-follicular phase, and a second peak at ovulation. Inhibin *A* reaches its peak in

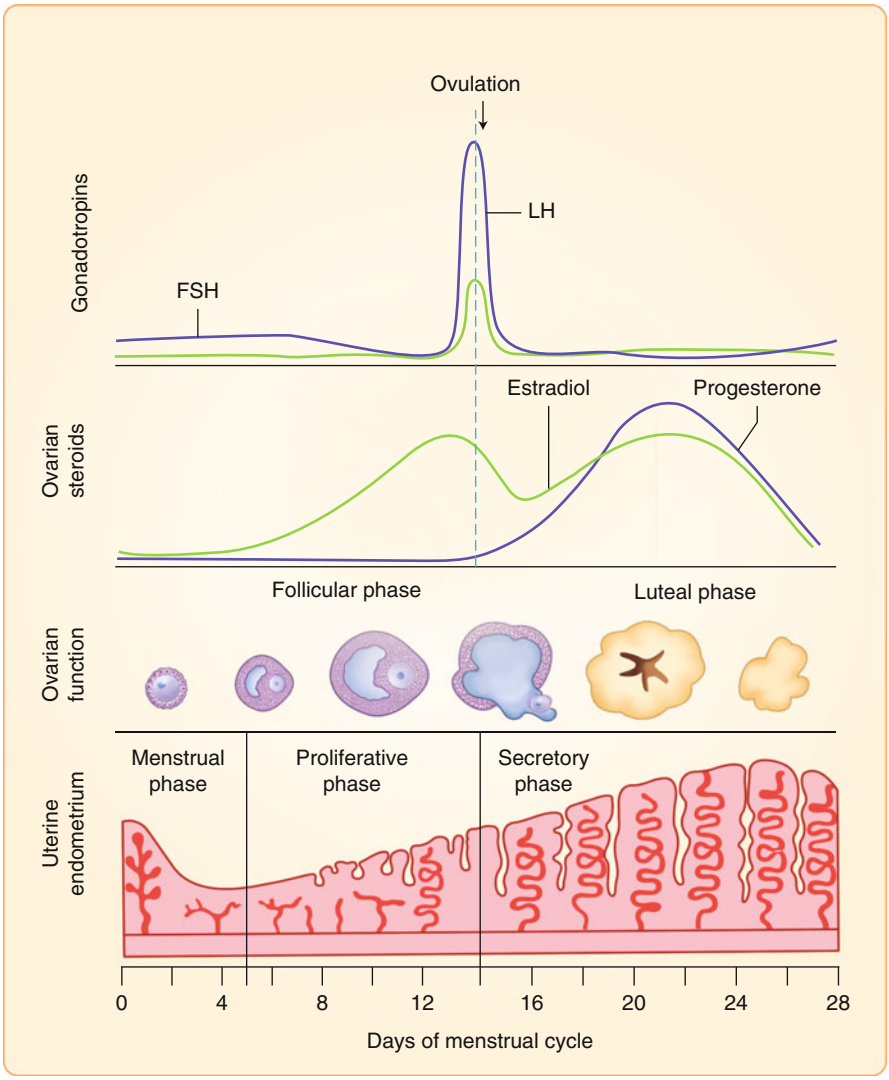

Fig. 4.3 Hormone levels during normal menstrual phase

the mid-luteal phase. Inhibin secretion is diminished by GnRH, and enhanced by insulin-like growth factor-1 (IGF-1). Follistatin is secreted by the ovaries and binds activin to decrease its activity thereby inhibiting FSH secretion [40]. IGF I and II promote steroidogenesis in theca and granulosa cells [34, 40, 42–44]. The insulin-like growth factors are peptides that are structurally similar to insulin and mediate growth hormone action. They induce the expression of cellular genes responsible for cellular proliferation and differentiation. IGF I has been demonstrated to stimulate DNA synthesis, steroidogenesis, aromatase activity, LH receptor synthesis, and

inhibin secretion. IGF II stimulates granulosa mitosis. All these hormones work synergistically to orchestrate the process of the menstrual cycle.

Endometrium During the Menstrual Cycle/Uterine Cycle [38]

The endometrium also responds to hormonal fluctuations during the menstrual cycle and has its own distinctive response. The epithelium lining the glands, stroma, and vasculature of the functional layer of the endometrium undergoes well-defined morphological changes. Similar to the ovarian cycle, there are also three main phases of the uterine cycle.

Menstruation Phase

When levels of hormonal steroid secretion decline as a result of corpus luteum regression, the menstrual cycle moves to the menstrual phase (menstruation), which signifies a failure to conceive. The spiral arteries of the endometrium constrict reduce the blood flow to the endometrium. Macrophages and leukocytes then invade the stroma and phagocytize the ischemic tissue. Eventually, the uterine lining sloughs off and is expelled through the vagina. This is the first day of menses, also described as a period. The normal duration of a period is less than 7 days and normal blood loss defined as less than 80 cc.

Proliferative Phase

This corresponds to the ovarian follicular phase, gradually rising estradiol levels lead to stimulation of the endometrium, allowing it to grow to a thick, blood vessel-rich, glandular tissue layer in preparation for pregnancy.

Secretory Phase

The last phase named the secretory phase (coincides with the ovarian luteal phase); once ovulation occurs, in addition to estrogen, the ovary also produces progesterone. This changes the proliferative pattern of the endometrium to a secretory pattern and is the time for continued increased vascularity in preparation for implantation.

Stages of Puberty

Puberty occurs as a result of hormonal influences and consists of a series of events that have been studied and described. While these events are mostly predictable, one must keep in mind that there is normal variance within ethnicities and environments.

Adrenarche is a process that typically occurs before the onset of puberty and refers to the activation of the adrenal cortex leading to the production of adrenal androgens [39]. Normal adrenarche may occur around the age of 6 years, when the zona reticularis of the adrenals respond to ACTH from the pituitary with the end product of DHEA-S and other androgens. The adrenals produce DHEA, DHEA-S, Androstenedione (all precursors of testosterone), testosterone, and androstenediol. Though it appears to be unrelated to the HPO axis, as mentioned earlier DHEA-S has been implicated in the upregulation of GnRH [27]. It has been hypothesized that adrenarche contributes to the triggering mechanisms of puberty and can perhaps be viewed as the initial stage of puberty. With the activation of the HPO axis, the ovaries also start to produce the androgens testosterone, androstenedione, and DHEA. DHEA-S is unique to the adrenals and can be used as a marker of adrenal androgen secretion.

Ultimately, androgens in the female (from the adrenals, ovaries, and peripheral conversion) contribute to the growth of sebaceous glands and promote hair growth in the axillae, pubis, and extremities. Abnormal production can lead to hirsutism and virilization. Estradiol, the main product of the follicle predominates during female development and promotes cellular differentiation and growth of primary sex organs: fat deposition, growth of breasts, oviducts, uterus, vagina, and external genitalia. Estradiol is also the principal hormone driving the pubertal growth spurt of females by increasing osteoblastic activity, epiphyseal maturation, and closure. Estrogen stimulation of the vagina mucosa also causes elongation of the vagina and a normal physiologic discharge which is a thin clear-white and non-foul smelling body fluid. All these increases in different hormonal levels manifest into the physical signs of puberty.

The earliest noticeable sign of puberty in the majority of females is breast development (therlache). Under the influence of estrogen and other steroids, the internal structure of the breasts starts to develop. Growth of underarm/pubic hair (Pubarche) follows, a process driven by androgens. Peak height velocity (growth spurt) occurs about 0.5 years prior to menarche and this process occurs earlier in females as compared to males. The first menstrual period (menarche) marks the end of puberty. Thus, the most common sequence of events during puberty is as follows: Thelarche > Pubarche > Growth spurt > Menarche.

Dr James M Tanner, a British pediatrician performed a study to describe the progression of puberty in males and females based on measurements and observation of secondary sexual characteristic development in males and females. In June of 1969, Drs. Marshall and Tanner published the Tanner Sexual Maturity Rating system (SMR) (also referred to as Tanner staging), a scale that ranges from prepubertal (stage I) to adult (stage V) and is wildly utilized in assessing human sexual

Table 4.1 Breast development

- Stage I (B1)—prepubertal; flat appearance with only the papilla (nipple) raised
- Stage II (B2)—the breast bud is present so that the areola protrudes
- Stage III (B3)—the breast tissue extends past the areola causing the elevation of the breast along with the areola. The contour of the areola is the same as the rest of the breast
- Stage IV (B4)—areola forms a separate contour from the rest of the breast creating what is referred to as the "mound on the mound" appearance
- Stage V (B5)—adult; the areola flattens down assuming the contour of the rest of the breast

Data from: American College of Obstetrics and Gynecology. Tool Kit for Teen Care: Tools for Adolescent Assessment. 2nd edition. 2010

Table 4.2 Pubic hair development

- Stage I (P1)—prepubertal, lanugo may be present in genital area but it is fine and downy
- Stage II (P2)—sparse growth of pubic hair in the midline, mainly at the base of the penis or along the labia majora
- Stage III (P3)—more hair grows so that it is visible from several feet, along with coarsening and increased pigmentation in some people
- Stage IV (P4)—hair now makes a triangle over the pubis
- Stage V (P5)—adult; hair is outside of triangle, extending up the abdomen and down the thighs

Data from: American College of Obstetrics and Gynecology. Tool Kit for Teen Care: Tools for Adolescent Assessment. 2nd edition. 2010

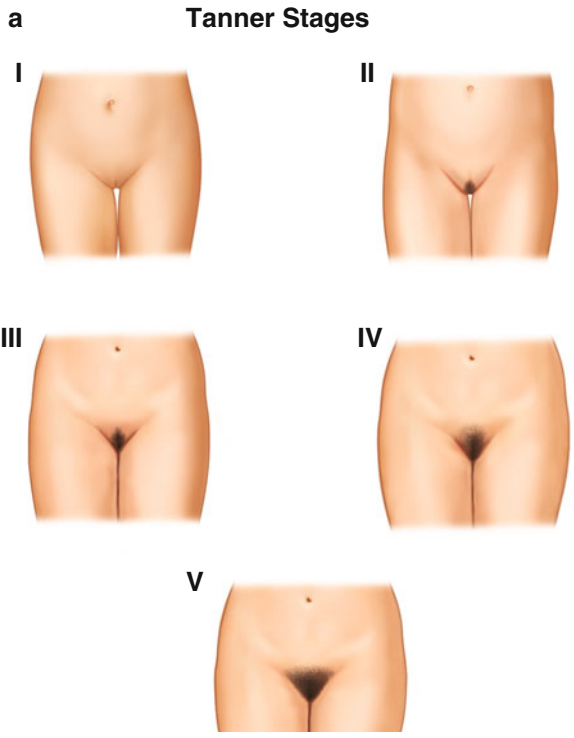

Fig. 4.4 Tanner stages of pubic hair (**a**) and human breast (**b**) development. (Created from data in Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child 1969 Jun;44(235):291–303)

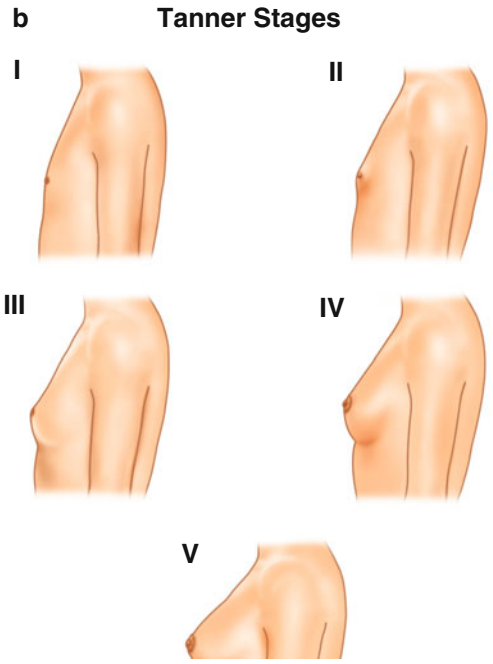

Fig. 4.4 (continued)

development. In the female, it describes the stages of puberty as based on breast size (B) and shape, and pubic hair (P) development and distribution (Tables 4.1 and 4.2, and Fig. 4.4a, b).

Nonetheless, despite achieving menstruation, the initial 3 years after menarche can be anovulatory. This is due to the delayed maturation of the positive feedback by estradiol [45]. A regular menstrual pattern is usually achieved by the third year post menarche. Furthermore, it is important to understand that ovulation is necessary for fertility, but may or may not accompany the earliest menses.

Age of pubertal onset and menarche is influenced by several factors and varies by geographical area and culture. In the United States, there is controversy regarding earlier sexual maturation as compared to data from the nineteenth century [46–52].

Dr. Henry P. Bowditch, an American physician, is often referred to as the pioneer of the study of menarche in the United States due to his publication in 1877 in which he reported the mean age of menarche as 14.75 [53]. Studies since then have indicated an earlier age of menarche. The first pubertal timing data on a representative US population was conducted by the CDC in a study called the National Health Examination Survey (NHES III) from the time period of 1966–1970. Results described the secondary sexual characteristics of American youth, ages 12–17 finding a mean age of menarche at 12.77 years. It was also noted in this study that African American girls were consistently more advanced than Caucasian girls for each chronologic age [54].

Table 4.3 Mean age of pubertal development summary of finding from various articles using data set from NHANES III

	African American	Mexican American	Caucasian
Thelarche (years)	9.5	9.8	10.3
Pubarche (years)	9.4	10.4	10.6
Menarche (years)	12.06	12.25	12.55

Data from: Sun et al. Is sexual maturity occurring earlier among U.S. children? Journal of Adolescent Health 2005 Nov 37 (5): 345–355; Wu T, Mendola P, Buck GM. Ethnic differences in the presence of secondary sex characteristics and menarche among US girls: the Third National Health and Nutrition Examination Survey, 1988–1994. Pediatrics. 2002 Oct;110(4):752–7; Sun SS, Schubert CM, Chumlea WC, Roche AF, Kulin HE, Lee PA, Himes JH, Ryan AS. National estimates of the timing of sexual maturation and racial differences among US children. Pediatrics. 2002 Nov;110(5):911–9

Table 4.4 Mean age of pubertal development summary of finding from PROS Study

	African American	Mexican American	Caucasian
Thelarche (years)	8.87 SD 1.93	ND[a]	9.96 SD 1.82
Pubarche (years)	8.78 SD 2.00	ND[a]	10.51 SD 1.67
Menarche (years)	12.16 SD 1.21	ND[a]	12.88 SD 1.20

Data from: Herman-Giddens ME, Slora EJ, Wasserman RC, Bourdony CJ, Bhapkar MV, Koch GG, Hasemeier CM. Secondary sexual characteristics and menses in young girls seen in office practice: a study from the Pediatric Research in Office Settings network. Pediatrics. 1997 Apr;99(4):505–12

[a]No data available

More recently, new data has been emerging studying development in different ethnic groups. Most recent National data comes from National Health and Nutrition Examination Survey (NHANES III), a survey carried out in the United States from the time period of 1988–1994. Mean age of menarche in this group was 12.43 years (Table 4.3) [48, 55, 56], with results revealing that African American girls and Hispanic girls tend to reach onset of puberty earlier as compared to their Caucasian peers [51, 52].

The Pediatric Research in Office Setting (PROS) network study [57] is the largest study to date and was a cross-sectional study of 17,077 girls ages 3–12 who presented to their pediatricians office from July 1992 to September 1993 and needed a complete physical exam. Tanner staging was utilized by the pediatricians in the collection of data. The data suggested that the girls were developing pubertal characteristics from an early age, even earlier than previously reported (Table 4.4).

Results from above-mentioned studies and others lead to the question as to whether American girls are reaching puberty at an earlier age. A review of sentinel articles performed by Slyper et al. looking at mean ages of secondary sexual characteristics using data available between 1948 and 2003 concluded that American girls are presenting to their physicians' offices with evidence of breast budding at a younger age. However, there has been little change in the timing of menarche with a mean age that is steady at approximately 12 years of age [46]. In an attempt to reach a consensus, an expert panel was asked to evaluate the weight of the evidence

available from all studies to date regarding puberty in American girls [49]. The entire panel did not reach the same conclusion; however, majority of the experts concluded that there was sufficient data to suggest a secular trend towards an earlier age of onset in breast development. In addition, the panel recommended the development of methods to discriminate fat versus breast tissue. Regarding menarche, majority of the experts based their conclusion mainly on results of the NHES III and NHANES III studies, and concluded that the data is sufficient to suggest a secular trend toward an earlier age of menarche given the decrease of approximately 2.5–4 months in the age of menarche between these two national studies. The minority did not believe that this was a significant difference to draw such a conclusion, thus it appears that the controversy regarding changes in menarche remains. Environmental factors such as obesity resulting in rapid body growth have also been implicated as a potential reason for reaching menarche earlier, but the evidence is inconclusive [46, 48, 56].

Psychosocial Development

Paralleling the biological process of puberty, the psychology of puberty is also quite complex. It is imperative that young women are provided with information regarding the changes their body is experiencing. There are additional implications as there can be a sense of confusion and a lack of understanding why the body is changing. In some cultures, it is considered taboo to discuss sexuality and yet the society in which our young women grow up in is becoming increasingly surrounded by sexuality. Furthermore, increase in sex hormones influences sexual behavior as neuropeptides involved in upregulation of GnRH are known to influence sexual behavior by acting mainly in the hypothalamic nuclei [58]. At the end of puberty, the young female becomes capable of participating in sexual reproduction, but not psychologically mature enough to be fully independent. The stage of adolescence which continues after puberty is divided into three periods: early (ages 11–13), middle (ages 14–16), and late (ages 17–20) [59]. The initial concerns among adolescents typically center around whether they are normal and fit in with their peers. Body image becomes the main focus following this, and certainly early or delayed puberty may result in distress or low self-esteem through alienation from peers [59, 60]. After this initial awareness, the stage of experimentation and authoritative defiance occurs as teens attempt to gain independence. This population is especially vulnerable for engaging in risky behaviors. Finally, towards the end of adolescence, the emerging young adult starts to think about the future and true independence. Some go through these stages smoothly while others do so with lots of turmoil. Throughout these stages friends, peers, school, and the family unit all play a critical role [61]. Healthcare providers who care for this population are also in a position to help with this transition. Imbalances can lead a young person down the wrong path. Therefore, it is important to be in a supportive environment where the adolescent develops a healthy identity and a sense of self.

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