# The Adolescent Female Athlete: Current Concepts and Conundrums

Donald E. Greydanus, MD, FAAP, FSAM, FIAP (H)<sup>a,b,\*</sup>, Hatim Omar, MD<sup>c,d</sup>, Helen D. Pratt, PhD<sup>a,e</sup>

### **KEYWORDS**

• Adolescent females • Athletes • Physiology

The adolescent female athlete has become a common part of the sports environment at all levels from childhood play to professional adult sports. <sup>1–16</sup> This article reviews basic sports physiology and then considers specific conditions, including iron deficiency anemia (IDA), stress urinary incontinence (SUI), breast issues (ie, pain, asymmetry, galactorrhea, injury), the female athlete triad (ie, menstrual dysfunction, abnormal eating patterns, and osteopenia or osteoporosis), and injuries. Various clinical conundrums are reviewed, including working with an athlete whose intense exercise patterns can lead to menstrual dysfunction and compromise of bone health.

### PHYSIOLOGY The Role of Gender

Children of both genders have basically the same physical condition with respect to such parameters as weight, height, injury risks, motor skills, percent body fat, endurance, strength, and hemoglobin levels. 1,8,9,11,12,14,15 Once the activation of the hypothalamic-pituitary-gonadal axis called puberty occurs, these specific parameters are altered;

<sup>&</sup>lt;sup>a</sup> Department of Pediatrics & Human Development, Michigan State University College of Human Medicine, 1000 Oakland Drive, Kalamazoo, MI 49008-1284, USA

<sup>&</sup>lt;sup>b</sup> Pediatrics Program, Kalamazoo Center for Medical Studies, 1000 Oakland Drive, Kalamazoo, MI 49008, USA

<sup>&</sup>lt;sup>c</sup> Division of Adolescent Medicine, Department of Pediatrics, KY Clinic, Room J422, University of Kentucky, Lexington, KY 40536-0284, USA

<sup>&</sup>lt;sup>d</sup> Department of Obstetrics & Gynecology, KY Clinic, Room J422, University of Kentucky, Lexington, KY 40536-0284, USA

<sup>&</sup>lt;sup>e</sup> Behavioral-Developmental Pediatrics, MSU/Kalamazoo Center for Medical Studies, 1000 Oakland Drive, Kalamazoo, MI 49008, USA

<sup>\*</sup> Corresponding author. Pediatrics Program, MSU/Kalamazoo Center for Medical Studies, 1000 Oakland Drive, Kalamazoo, MI 49008. E-mail address: Greydanus@kcms.msu.edu

changes in ability in sports competition are observable. Despite these differences, exercise training results are different for specific athletes depending on intensity of training and genetic traits rather than on gender alone. In the past, the female athlete was limited by inadequate equipment and limited training. As these factors have been corrected, the results in female athlete achievements have exponentially increased as well.<sup>1,4</sup>

### Effect of Puberty

The phenomenal process of puberty affects the female in various ways (**Table 1**).

For example, the body fat percentage is particularly affected in the female, and the final result is an average body percentage in adult females of 23% to 27% versus a range of 13% to 15% in adult males. <sup>1,4</sup> As a result of intense and prolonged training, the elite female athlete can lower these levels to 12% to 16% (in distance runners) and 8% to 10% (in sprinters) versus 4% to 8% in highly trained male gymnasts. <sup>1,4,14,15</sup>

The thermoregulatory capacity is similar between genders; having fewer sweat glands in the female is offset by producing less heat because of reduced body mass, reduced muscle bulk, and large body surface area. There is a higher risk for heatstroke in athletes of both genders if they are late maturing, are obese, or are exercising in hot climates. Females tend to have better balancing and flexibility abilities, which initiate in childhood and peak at 14 or 15 years of age; in contrast, males improve in flexibility from midadolescence until the end of puberty.<sup>1,4,7</sup>

After puberty, females develop less strength than males, as noted in **Table 2**. Although the proportion of muscle fiber type is similar, the muscle fiber size is less in the female. The females develop a small increase in muscle strength after menarche (onset of menstruation), whereas the male continues to increase muscle strength throughout the process of puberty. Trained female athletes can achieve about 70% body strength compared with males of similar training, whereas the upper body strength is 30% to 50% that of males. At 7,11-14

In the adolescent male, maximal speed peak occurs before peak height velocity (PHV) whereas power and strength peaks occur after PHV; a similar pattern is not observed in adolescent females, who typically have the most weight gain rate 12 to 14 months after maximum growth velocity in Tanner stage (sexual maturity rating [SMR]) 2 or 3.<sup>7,8,14–16</sup> In the female, there is a small muscle mass increase in contrast to a large increase in body fat. Research notes a heightened response to training (strength and endurance) 12 to 24 months after PHV at an SMR of 4 or 5. Intense weight training in females results in only a small increase in observable muscle and perhaps some measurable strength increase; intensive exercise may lead to less adipose tissue and more muscle definition. The progression of puberty allows males to grow into their chosen sport because they get closer to their physical optimum and thus, potentially reach their optimum sports performance. However, the process of puberty in females impedes their best sports performance by lowering their physical optimum.<sup>1,4</sup>

# Table 1 Changes in females induced by puberty

Size of heart, volume of cardiac stroke and size of left ventricle are smaller

Lung volume and aerobic capacity are less

Hemoglobin levels are reduced

Female is smaller and has shoulders that are more narrow and reduced articular surface

Female has greater flexibility and increased balance

Table 2 Strength comparison between equal or similar size females and males		
Ages birth to 10 years (before puberty): same strength		
Ages 11–12 years: female strength is 90% of males of same age		
Ages 13–14 years: female strength is 85%		
Ages 15–16 years: female strength is 75%		

Normal weight and height gains from childhood to puberty allow improved performance in various sports (including basketball, volleyball and swimming), depending on genetic potential and training. Females have shorter extremities and a lower center of gravity, which may have a potential competitive advantage in sports that emphasize balancing abilities, such as gymnastics. However, research tends to note that the center of gravity is influenced by the athlete's specific weight as well as height and not by gender itself.<sup>1,4</sup>

A delay in puberty can be an advantage for the athletically gifted female, who may be attracted to sports that place a priority on a thin or lean physique, such as synchronized swimming, gymnastics, dance, and figure skating. Society has not encouraged the female adolescent athlete to become involved in American football; she does not face the risks for concussion, brain damage, and spinal injury now faced by males in modern football teams at all levels because of the emphasis on winning.<sup>1</sup>

However, the pressure to win may result in parents and society failing to notice and prevent the sexual advances of a predatory coach or trainer, who may promise to take the female adolescent athlete to high levels of victory.<sup>1</sup>

### **IDA**

IDA (**Table 3**) is the most common cause of anemia in adolescents and research reports a prevalence of up to 24%, with frank anemia noted in 10% of 14- to 18-year-olds. <sup>17,18</sup> Iron deficiency (ID) and IDA are seen more commonly in females versus males and a similar prevalence is reported in female athletes versus nonathletes, except for long-distance runners, who have a higher prevalence of IDA. <sup>3,19–21</sup> In contrast to males, the adolescent female has 6% reduced red blood cells, reduced

Table 3 Laboratory parameters of IDA in adolescent females		
Hematocrit	<35%; 12-year-old females <36%; 12–18-year-old females	
Hemoglobin	<11.5 g/dL; 12-year-old females <12.0 g/dL; 12–18-year-old females	
Serum ferritin	<10 mg/L (normal, 15–200 mg/L)	
Mean corpuscular volume (MCV)	<76 fl in 12-year-old <78 fl in mid- and older adolescents	
Serum iron	<40 mg/dL (normal, 50–140 mg/dL)	
Serum transferrin saturation (Fe/TIBC ratio)	<16% (normal, 35%–40%)	
TIBC	350-500 mg/dL (normal, 250-380 mg/dL)	
FEP	> or = 150–200 mg/dL RBCs (normal, 54 $\pm$ 20)	

Abbreviations: Fe, iron; FEP, free erythrocyte protoporphyrin; TIBC, total iron-binding capacity. Reprinted from Greydanus DE, Patel DR. The female athlete: before and beyond puberty. Pediatr Clin North Am 2002;49:572; with permission. (up to 19%) lower hemoglobin levels, lower iron stores, and increased iron levels.<sup>17</sup> Those individuals dedicated to optimum performance in their sports are concerned about IDA, because even mild IDA may result in lowered sports performance.

Various issues can lead to IDA, including limited oral intake of dietary iron, menstruation, iron loss in sweat and urine resulting from exercise, gastrointestinal bleeding, and intravascular hemolysis, which can be precipitated by exercise. <sup>17,22</sup> Routine screening for IDA is not recommended for adolescent athletes unless they are at risk because of such issues as heavy menstruation, history of anemia, being a long-distance runner, or having a vegetarian diet. <sup>17,19,23</sup>

There are 3 stages of developing IDA: first there is a lowering of iron stores as well as serum ferritin, followed by a lowering of serum iron along with an increase in total iron-binding capacity; the third step is development of microcytic hypochromic anemia. The IDA that occurs is typically mild and basically asymptomatic. Most research concludes that there is no overt impairment of sports performance in those with non-anemic ID (ie, those with normal hemoglobin as well as hematocrit levels and low levels of serum ferritin). However, athletes with low to normal hemoglobin and low ferritin levels may report enhanced sports performance with iron supplementation; more research is needed in this area. Athletes who develop sports anemia or pseudoanemia do not require iron supplementation because this is a normal reaction to intense and prolonged exercise. The plasma volume may expand up to 20%, although iron supplementation is not needed as long as the red blood cell mass remains normal. When the intense exercise is stopped, plasma volume returns to normal pre-exercise levels.

IDA management involves educating the young people about proper nutrition, including foods with iron (such as fortified cereals and breads), fish, meat, and eggs. If iron supplementation is required because of overt ID, elemental iron is prescribed at a dose of 3 to 6 mg/kg/d. This strategy results in an increase in hemoglobin and hematocrit levels typically in 1 to 3 weeks, whereas normal ferritin levels may not develop for several months, indicating the development of normal body iron content.<sup>1,4</sup>

### SUI

SUI, the involuntary loss of urine during exercise, is reported in approximately onequarter of nulliparous female athletes with a mean age of 20 years. <sup>1,4</sup> SUI is particularly noted in activities referred to as impact sports such as gymnastics, basketball, jumping, and running (ie, track and field events); less commonly implicated sports include skiing, tennis, skating, and jogging. <sup>23,25–27</sup> Risk factors are noted in **Table 4** and the basic cause is usually linked to an increase in intra-abdominal pressure caused by exercise that leads to urethral sphincteric unit alterations. A medical history of the

Table 4 Risk factors for exercise-induced SUI
↑ age
Female gender
Hypoestrogenic amenorrhea
Involvement in high-impact sports activity
Heavy exertion
Parity that is increased
Possibly obesity

female athlete may reveal the presence of SUI, with further identification of known risk factors (see **Table 4**). A general medical examination may be performed, including a pelvic examination to assess for anatomic pelvic floor integrity and dysfunction of the posterior urethrovesical angle.<sup>1</sup>

Because SUI is normally a self-limiting, benign condition in the adolescent female athlete, basic education about the nature of this condition is usually all that is needed. The athlete can be given information about the need to take enough fluid for the exercise event but not so much as to induce SUI; sanitary napkins placed before exercise are also helpful. If the female feels that more treatment options are needed, a variety of approaches are available, including behavior management, Kegal exercises, pharmacologic management, biofeedback counseling, vaginal cones, and electrical stimulation. Imipramine and pseudoephedrine hydrochloride have been used to prevent or reduce exercise-induced SUI. Phenylpropolamine was withdrawn from the market in the United States because of an increase in reported cerebrovascular accidents in women less than age 50 years. Use of anticholinergic medications is not recommended because they may induce abnormal sweating and overt heat disorders.

# BREAST ISSUES Effect of Exercise

Exercise does not alter the breast size by changing muscle tissue, because there is only a small amount of muscle in the areolar area and none in the rest of the breast structure. However, exercise can alter the appearance of breast size by changes in the underlying pectoralis muscle. In addition, intense exercise can reduce mammary adipose tissue, with a resultant smaller breast. 1,4,28,29 The size of the breast is also affected by factors that enlarge or reduce breast size as noted with various dietary regimens. 2,9,30 Exercise provides a protective effect with regard to breast cancer in the adult years. 28,31–34

### Pain

Exercise-induced breast pain may be a hidden concern of the adolescent female athlete, especially in the individual with large breasts, and may prevent some of these athletes from participating in sports. <sup>28,30</sup> Breast soreness or tenderness induced by exercise was noted in 31% of female athletes in 1 report, and 52% of the women with breast discomfort also noted exercise-induced injury to breast tissue. <sup>1,4,35,36</sup> Research reveals that breast motion in exercise can be considerable, particularly in gymnastics, soccer, volleyball, basketball, running, and other sports. <sup>35–37</sup> Excessive breast movement can result in overt strain of the fascial attachments of the underlying pectoralis muscle in addition to intense shoulder pain. <sup>1,35,37</sup> Breast discomfort can also be increased from menstrual cycle-induced breast fluid retention, as noted by some individuals during the premenstrual phase of menses and with overt premenstrual syndrome (PMS). <sup>37,38</sup> Excessive perspiration can cause local excoriation, abscess development, and even intrabreast-fold cellulitis. If the female adolescent presents with breast pain, various underlying causes should be considered, including those already mentioned in addition to overt breast masses, such as a fibroadenoma. <sup>1,35,38,39</sup>

Breast discomfort and pain in female athletes may be reduced or prevented by having the individual wear a properly fitted sports brassiere that allows maximum support to the mammary tissue and minimizes exercise-induced breast movement. <sup>39–46</sup> A sports brassiere should consist of breathable (ie, minimizing sweating) material that is nonabrasive and is manufactured to have proper cups (soft, firm), few seams, and hooks that are few in number and padded in quality. Selected women

may also benefit from brassieres with shoulder straps that are properly padded. A properly fitted sports brassiere lifts and carefully separates the mammary glands in a way that reduces or minimizes overt breast motion. As noted with sports equipment in general, sports bras should be changed often (eg, every 6 months). Guidelines for sports brassieres have been published by researchers and manufacturers.<sup>28,36,45,46</sup>

### **Asymmetry**

Asymmetry of breasts is a common situation in growing adolescent females that resolves over time in most, although visible asymmetry remains in 1 in 4 adult women.<sup>35,39</sup> Examination should look for other causes, including a breast mass, although asymmetry is usually a normal variant in growth.<sup>1,39</sup> Injury to the breast may occur without proper protection, and padded brassiere and foam inserts should be used as needed; female swimmers with breast asymmetry can wear a swimming suit with breast supports. The athlete may find help at places that work with or specialize in patients who have had a mastectomy.

### Galactorrhea

Females who present with nipple discharge not associated with pregnancy (galactorrhea) need a medical evaluation to look for a variety of causes, such as mental health concerns (eg, anxiety, depression), effect of medications (eg, phenothiazines, oral contraceptives), hypothyroidism, pituitary neoplasms, and hypothalamic injury (eg, infection, surgery). Most cases are idiopathic and management depends on the underlying cause. For example, hypothyroidism can be corrected, neoplasm removed, implicated medication withdrawn, and counseling provided to stop selfmanipulation if appropriate.

## MENSTRUATION AND SPORTS Menstrual Physiology

Under the influence of an activated hypothalamic-pituitary-ovarian-uterine axis, the adolescent female begins to have menstrual periods that are controlled by changes in pubertal hormones (ie, estrogen and progesterone), resulting in 3 menstrual phases: follicular, ovulatory, and luteal. 1,39,47 Estrogen is produced by the ovaries and its increase leads to the follicular menstrual phase, in which there is endometrial growth characterized by endometrial gland growth (number and length) within a compact, proliferative stroma. When ovulation occurs, at some point after menarche, estrogen and progesterone are produced by the corpus luteum, which induces a secretory endometrium because of progesterone effects. The last part of the normal menstrual cycle is the luteal phase, with development of an endometrium with an edematous stroma containing dilated, tortuous glands. If conception does not occur, the corpus luteum becomes atretic, with a resultant precipitous decrease in the pubertal hormones and eventual menstruation.

It may take 1 to several years to proceed from menarche to menstrual periods that are regular, and this complex phenomenon is subject to a wide variety of factors, many of which can be found in the sports-minded female. An adult female (or mature adolescent female) has a menstrual cycle that occurs every 28 days ( $\pm 7$  days); the median blood loss per cycle is 30 mL, with an upper normal limit of 60 to 90 mL of blood.  $^{1,39,47}$ 

Irregular (infrequent) menstruation that occur at intervals of more than 45 days is called oligomenorrhea. The absence of menstrual cycles (amenorrhea) can be identified as primary or secondary amenorrhea, in which primary amenorrhea refers to absence of menstrual cycles by age 14 years with no pubertal development (SMR

or Tanner stage of 1) or absence of menses by age 16 years without respect to SMR rating. The absence of menses after menarche has occurred for a total of 3 previous periods or for 6 months without any periods after menarche is called secondary amenorrhea. Normal young females may have no menstrual periods for 3 to 6 months during years 1 and 2 after menarche. However, if an adolescent female presents with oligomenorrhea, primary amenorrhea, or secondary amenorrhea, the clinician should launch an investigation into potential causes (**Table 5**). 1,39,47

### Menstrual Cycles and Athletic Performance

Conflicting research results have been published regarding the effect of menstruation on female athletic performance. <sup>16,47–49</sup> One investigation of 86 female soccer players noted athletes reporting more injuries when having premenstrual symptoms than at any other menstrual phase, and anecdotal reports exist of exercise leading to increased menstrual bleeding or dysmenorrheal. <sup>1,49</sup> However, research tends to note fewer menstrual symptoms (ie, bleeding, pain, premenstrual symptoms) with exercise and no overt menstrual cycle-related differences in lactate levels, exertion efforts, or overall sports performance. <sup>1</sup>

### Amenorrhea

Menstrual dysfunction is well known in female athletes and includes oligomenorrhea, amenorrhea (primary or secondary), and luteal phase dysfunction; this includes 10% to 15% of female athletes and two-thirds of elite athletes. <sup>2,5,7,16,30,50–67</sup> A delay of menarche can be seen at a level of 5 months for each year of intense training before the onset of puberty; if the athlete lowers her level of exercise training, menarche or return of menstrual cycles usually results. <sup>1</sup> Secondary amenorrhea is commonly seen in females engaging in such sports as distance running, ballet, gymnastics,

Table 5 Causes of amenorrhea in adolescents		
Primary amenorrhea	Physiologic delay Pseudoamenorrhea Imperforate hymen Transverse septum Rare: agenesis of vagina, cervix, uterus Mayer-Rokitansky-Kuster-Hauser syndrome Turner syndrome Chronic illness Hypothalmic-induced Such as weight loss, eating disorders, exercise, stress, others Pituitary disorders Polycystic ovary syndrome (hyperandrogenemia syndromes) Thyroid disorders Others	
Secondary amenorrhea	Pregnancy Hypothalamic-induced Such as weight loss, eating disorders, exercise, stress Polycystic ovary syndrome (hyperandrogenemia syndromes) Thyroid disorders Pituitary disorders (pituitary adenoma) Chronic illness Others	

Reprinted from Greydanus DE, Patel DR. The female athlete: before and beyond puberty. Pediatr Clin North Am 2002;49:563; with permission.

and cycling. Menstrual dysfunction is reported in 12% of swimmers as well as cyclists, up to 20% in females reporting vigorous exercise, 44% of ballet dancers, 50% of female triathletes, and 51% of endurance runners. 1,16

Multiple issues underpin athletic amenorrhea, including genetics, percent body fat, intensity of exercise, age, weight, nutritional deficits, and stress. He type of sport chosen can influence menstrual dysfunction as well. For example, dance and gymnastics support or encourage a female athlete with a thin body habitus. However, specific weight alone does not lead to absence of menstruation because those with the same weight can be amenorrheic or have a normal menstrual pattern. Specific body fat is not the sole factor and earlier research suggesting that menstruation does not occur in females with body fat less than 17% has not been verified by research. The precise role of leptin in this complex process of menstruation is not clear.

A wide variety of causes must be considered when a clinician evaluates an adolescent female with amenorrhea or other menstrual dysfunction. 1,39,47 Often, amenorrhea in such athletes is classified as hypothalamic amenorrhea, with gonadotropin-releasing hormone and luteinizing hormone pulsivity abnormality. One theory suggests that menstrual dysfunction in athletes results from an energy drain because of the intense exercise level associated with a caloric intake that is not sufficient to maintain normal menstruation. 16,71,72 Such an energy drain can be compounded by other issues, such as having a previous history for menstrual problems, positive family history for menstrual dysfunction, and chronic illness. Thus, any athlete with abnormal menses (ie, oligomenorrhea or amenorrhea) should receive a comprehensive evaluation. 1,39,47,52,54,66 The evaluation should investigate such positive findings as congenital anomalies, short stature, galactorrhea, virilization, hypoestrogenemia, and other endocrine findings or disorders (see **Table 5**). 1,39,47 Some suggested laboratory testing is listed in **Table 6**.

The specific causes of amenorrhea in an adolescent female determine precise management plans; if the menstrual dysfunction is related to her intense exercise patterns, advice should be given to decrease exercise intensity and increase nutritional intake along with providing calcium supplements (see later discussion). <sup>16,73–79</sup> Improvement in the amenorrhea or oligomenorrhea will occur if this advice is followed and if there are no other underlying causes.

Table 6 Laboratory testing for amenorrhea in adolescents
Pregnancy test
Thyroid hormone levels
Bone age
Antiovarian antibodies
Chromosome evaluation
Head CT/MRI
LH and FSH: ↓ in ovarian failure/dysgenesis; normal or ↑ in others
Pelvic/abdominal MRI
Pelvic ultrasound to define anatomy
Prolactin levels
Renal ultrasound/IVP
Vaginal smear to evaluate for epithelial cell estrogenization virilization/hirsutism: DHEAS, LH/FSH ratio (normal <2.5:1), testosterone (total and free)

Abbreviations: CT, computed tomography; DHEAS, dehydroepiandrosterone sulfate; FSH, follicle-stimulating hormone; IVP, intravenous pyelogram; MRI, magnetic resonance imaging.

However, the experienced clinician quickly learns that many if not most committed athletes do not want to reduce their exercise intensity for fear of decreasing their sports performance. They often continue in this sports pattern even when informed that their menstrual problems may be related to a pattern of chronic hypoestrogenemia that may lead to reduced bone mineral density (BMD), osteopenia, and eventual osteoporosis (see later discussion).<sup>1,4</sup> The research in this area is complex and more is needed. However, studies in recent decades note that females with chronic amenor-rhea and low BMD may never acquire healthy BMD, even if the menstrual pattern eventually becomes normal.<sup>1,80</sup> Some female dancers and other athletes who have delayed menstruation progress into a state of low BMD and have an increased risk for stress fractures that can limit their sports performance.<sup>1,4</sup>

Experts generally recommend daily supplementation with calcium (1200–1500 mg) and vitamin D (400–800 IU) for the adolescent athlete with menstrual abnormalities or overt eating pattern dysfunction. The literature remains conflicted about the use of estrogen supplementation (ie, conjugated estrogen or oral contraceptives) for those with exercise-induced low BMD in attempts to prevent lowering of the BMD. If the athlete has low BMD, estrogen supplementation (oral contraceptive or conjugated estrogen) may help in some cases to preserve some bone loss. 1,4,39,47

One well-known approach is to avoid prescribing such hormonal treatments for amenorrheic athletes who are within 3 years of menarche, and clinicians should emphasize the need to lower the intensity of exercise workouts and the need for improved nutritional intake along with calcium supplementation. <sup>81</sup> Oral contraceptives are suggested if the athlete is 3 years after menarche, is more than 16 years of age, and is amenorrheic; earlier hormonal intervention is acceptable with a history of stress fracture. <sup>81</sup>

Clinical judgment is needed because there is no clear research-supported consensus on managing these young people and there is no proven benefit to providing such hormonal intervention to improve or preserve BMD with or without weight gain. <sup>1,4</sup> Use of combined oral contraceptives (COCs) does not correct the underlying physiologic dysfunction of this abnormal menstrual pattern and the amenorrhea or oligomenorrhea typically resumes once the COC is withdrawn. Side effects of the oral contraceptive can be distressing to some females; these include breast congestion, headache, and nausea. <sup>1,47</sup> Even with the past few decades of research and observation, it is not clear what the acute and long-term implications are for the female adolescent athlete with chronic amenorrhea and potential estrogen deficiency. <sup>1,80</sup>

Adolescents who are thin and inactive tend to have the lowest BMD. Also complicating this picture is that intense exercise with weight bearing may neutralize the low BMD effect of having a thin body habitus because of enhanced bone accretion. Thus, athletes in some sports (ie, tennis players, ice skaters, runners, gymnasts) who are amenorrheic may still have normal or even increased bone density because of exercise-induced high mechanical forces. Some research has observed an enhanced bone density effect in some athletes if they are taking oral contraceptives; however, osteoporosis may not be prevented if the pill has less than 50  $\mu$ g of ethinyl estradiol. COCs more than 50  $\mu$ g are not recommended for adolescent females because of the increased risks for adverse effects. Complicating this picture is the observation that the female acquires an increased risk for osteoporosis if she never acquires normal BMD. More research is needed in this arena.

### Oral Contraceptives and Athletic Performance

There is no evidence from research studies that athletic performance is reduced for females taking oral contraceptives. <sup>51,84,85</sup> COCs can provide a positive influence because of their beneficial effect on improving dysfunctional uterine bleeding (DUB),

anemia related to DUB, dysmenorrhea, PMS, absence of pregnancy, possibly reduced injury risk in those with dysmenorrheal or PMS, possibly reduced bone mineral loss (see earlier discussion), and possibly less risk for stress fractures. 1,39,47,86-90

Manipulation of COCs can be used to the athlete's advantage by allowing few menstrual cycles when she stays on active hormone pills for longer than usual. 1,4,39,47 For example, she can remain on a 21-day pack or not take the inactive pill that is part of the 28-day pack to prolong the interval between menstrual periods and thus avoid menstruation during an important sports event. Monophasic pills are less confusing to the female athlete than triphasic pills and provide more consistent hormone blood levels. However, concerns about potential adverse effects of COCs may prevent some athletes from going on or staying on these pills. 1,39,47 Also, pharmaceutical companies have now produced continuous pills to allow reduced menses. For example, current extended cycle hormonal contraceptive pills in the US market include Seasonale, Quasense, and Seasonique, which allow a menstrual period once every 3 months. Lybrel is an extended cycle hormonal formulation that is taken every day and prevents any menstrual period.

### **EXERCISE AND PREGNANCY**

Sexually active women who do not take contraception or use contraceptives ineffectively are at risk for pregnancy. The pregnancy certainly affects their overall sports performance. However, if a woman becomes pregnant, she may wish to continue to exercise to some degree. 1,91–95 Concern has been expressed that excessive exercise induces diversion of blood away from the fetus to the mother's exercising muscles, with possible fetal hypoxia as a result, or that fetal hyperthermia may occur because of increased core temperature in the exercising pregnant woman. The fetus is well insulated from these potential adverse effects of exercise and studies have not found these theories to be valid; thus, some physical activity is acceptable to clinicians and guidelines in this regard have been published to offer advice for the pregnant woman. Sensible exercise is recommended and includes swimming as perhaps the best form of exercise during pregnancy, but other forms of exercise are recommended, such as walking and cycling.

**Table 7** lists specific contraindications to exercise in the pregnant woman and **Table 8** provides reasons for reduced sports performance during pregnancy. Exercise should be based on acceptable guidelines and common sense for each specific woman based on her prepregnancy exercise pattern. Workouts and physical exertion that were not included in exercise patterns before the pregnancy should not be attempted during pregnancy. Excessive exercise is limited to 15 minutes, although strenuous anaerobic activity is typically never recommended. In addition to avoidance

Table 7 Contraindications to exercise in pregnancy	
Hypertension induced by pregnancy	
Preterm membrane rupture	
Second or third trimester bleeding that is persistent	
Intrauterine growth retardation	
Incompetent cervix or cerclage	
History of preterm labor or presence of preterm labor in a current pregnancy	

Table 8 Pregnancy-induced factors that may negatively affect sports performance		
Abdominal growth		
Adipose tissue		
Altered center of gravity		
Breast ducts		
Cardiac output		
Fluid retention		
Ligamentous laxity (caused by increased estrogen and relaxin levels)		
Maternal blood volume		
Overall expenditure of energy		

or limitation of excessive exercise, physical exertion should be avoided in very hot weather and cease if the mother has a fever (ie, more than 38°C). Pregnancy increases breast congestion and nipple prominence; thus, a well-fitted, supportive brassiere is necessary. Physical exertion using the upper body is permitted but it should not subject the upper torso to overt or excessive mechanical stress.

The pregnant athlete who is exercising should always be well hydrated and should cease exertion under specific conditions, such as the occurrence of dizziness, shortness of breath, vaginal bleeding, severe headache, tachycardia (pulse more than 180 beats per minute), muscle weakness, or pain in the chest, hips, back, or elsewhere. Jumping may lead to relaxation and pelvic ligament stretching and is thus not recommended. Also avoided is exercising in the supine position, even if this was practiced before the pregnancy developed. Several sporting activities should be avoided during pregnancy, including horseback riding, weight lifting, and scuba diving and other water sports.

Engaging in competitive sporting activities is not recommended, especially when the risk of injury is substantial and the activity is classified as a contact sport. After delivery, exercise may resume in 4 to 6 weeks after a vaginal delivery and 6 to 8 weeks after a cesarean section. <sup>95</sup> Breastfeeding is not a contraindication for exercise in the mother. <sup>96</sup> Diabetes mellitus in the pregnant woman is not a contraindication to sensible exercise if approved by her clinician and if the metabolic state is stable and closely monitored. <sup>97</sup>

### **FEMALE ATHLETE TRIAD**

The female athlete who becomes seriously engaged in her sports, especially sports that emphasize specific weight or body size, is at risk for what has been called the female athlete triad: amenorrhea, disordered eating, and osteoporosis (osteopenia). 5.7.52–54.58.60.66–68 Although not accepted by all researchers, the term is useful because it reminds clinicians of 3 important issues that may complicate the lives of adolescent athletes. It reminds clinicians that some sports may be more precarious for adolescent females than others, specifically those that focus on various weight categories, lean appearance, prepubertal appearance, or a lean body (**Table 9**). These athletes may struggle to attain an ideal sports body because their sport seems to demand goals that are difficult to achieve unless the athlete develops abnormal eating patterns that induce or promote nutritional deficiencies and abnormal exercise patterns. The result is a condition that may contain various components of this triad. 52–54,56

Table 9 Sports that increase risks for the features of the female athlete triad		
Emphasis on various weight categories	Judo Rowing Taekwondo Weight lifting Wrestling	
Emphasis on a prepubertal appearance	Ballet Figure skating Gymnastics	
Emphasis on a lean body	Cross-country skiing Long-distance running Swimming	
Emphasis on lean appearance	Dance Diving Figure skating Gymnastics Synchronized swimming	

### Disordered Eating

Dysfunctional eating schedules are noted in 15% to 75% of adolescent female athletes and are characterized by a combination of self-induced vomiting, fasting, skipping meals, and consumption of diet pills, laxatives, and/or diuretics. 1,73,75–77,98 Critical windows of time occur in the lives of these athletes when they become more vulnerable to developing such abnormal eating patters, such as the period of PHV, transition to high school or college (university), giving up sports competition, death (eg, member of the family, coach, trainer, friends), and postpartum depression. Sports that insist on intense exercise patterns and/or low body weights (ie, < what is physiologically normal) are sports with athletes at increased risk for the development of poor eating habits; these sports include but are not limited to swimming, diving, track, distance running, and gymnastics (see **Table 9**). 1,54

### Anorexia Nervosa

The increased risk for eating dysfunction is found in dedicated adolescent athletes, although most do not develop overt bulimia nervosa or anorexia nervosa. However, 5% to 20% of ballet dancers may develop anorexia nervosa; the incidence is associated with the intensity of exercise as well as level of competition. Hypothalamic amenorrhea occurs in these athletes with anorexia nervosa who develop disordered eating of a severe degree that leads to low bone density at a critical time of bone development, when they need to gain bone density not lose it. 1,98

The presence of anorexia nervosa in athletes and nonathletes may lead to osteopenia because of starvation, deficiency of estrogen, lowered intake of calcium, low BMD, increased glucocorticoid levels, and other factors. Research notes that osteopenia develops early and often in the female with anorexia nervosa and depletes normal bone health so much that she never develops normal BMD even if management eventually allows a return to normal weight. Her risk for development of overt osteoporosis (see earlier discussion) as an adult has also been noted by research studies.

Prevention of reduced BMD and disordered eating patterns is the best management strategy; however, if this is not possible, intense management should be directed to allow return to a normal weight as soon as possible during the adolescent years while

an increase in BMD is still possible. As noted earlier, prescribing conjugated estrogen formulations (such as the COC) have been used to increase BMD in those with anorexia nervosa and severe malnutrition. There is no overt proof that use of estrogen combinations restores normal bone density in adolescent athletes with anorexia nervosa. BMD may optimize in females with anorexia nervosa who resume normal menstruation.<sup>1,4</sup> Current research implies that BMD is not protected by such estrogen supplementation without weight gain and this becomes a major conundrum of sports participation for female adolescents and their caring clinicians.<sup>1</sup>

### Osteoporosis and Adolescence

There are various factors critical to osteoporosis acquisition, such as genetics (70%), estrogen status, calcium intake, exercise patterns, and body weight (**Table 10**). <sup>1,76,98–112</sup> Studies reveal that 50% to 63% of peak bone mass (ie, BMD amount gained during growth) develops during childhood, whereas up to 50% accumulates during the adolescent years. <sup>1</sup> During growth it is critical that sufficient amounts of calcium are acquired into BMD to maximize bone mineralization and gains are greatest during early pubertal periods if sufficient estrogen and calcium are available. Classic studies on females in late adolescence (ie, ages 17 to 20 years) reveal that no significant BMD increase was found after age 17 years. <sup>113</sup> In general, BMD usually decreases in the middle of the fourth decade, with significant lowering when menopause starts in the fifth or sixth decade of life. In addition, weight resistance training can lead to a 4.5% increase in BMD for 1 year in females who are postmenopausal in contrast to those not exercising or training. <sup>99</sup>

Adolescent females with delayed menarche and thin body habitus tend to have the lowest BMD and as noted, suboptimal BMD is found in adolescents with menstrual dysfunction (ie, amenorrhea or oligomenorrhea) complicated by anorexia nervosa or other features of hypothalamic amenorrhea, such as found in athletes with intense exercise patterns and weight loss.<sup>1,4</sup> Estrogen facilitates calcium movement into bone, and thus those with chronic, hypoestrogenic amenorrhea (including postmenopausal states) develop significant risk for osteoporosis and stress fractures.<sup>1</sup> Thus, dancers, runners, and other female athletes as well as those with eating disorders have increased incidence of stress fractures during their careers, even as adolescents.

As noted with eating disorders, the best management for osteopenia (and osteoporosis) is prevention, by preventing the various factors noted in **Table 10** from taking

### Table 10

### Risk factors for osteoporosis

Limited calcium intake in childhood/adolescence

Positive family history (first-degree relatives) for osteoporosis

Low levels of physical (weight-bearing) activity

History of amenorrhea/irregular menses

Thin habitus (anorexia nervosa, others)

Alcoholism (toxic to bone-building cells and possibly induces decreased calcium absorption)

Cigarette smoking (decreases estrogen effectiveness)

Medications (glucocorticoids, phenytoin, others)

Various chronic diseases (primary hyperparathyroidism, Cushing syndrome, Addison disease, leukemia, celiac disease, Crohn disease, others)

Others<sup>1,101</sup>

Reprinted from Greydanus DE, Patel DR. The female athlete: before and beyond puberty. Pediatr Clin North Am 2002;49:565; with permission.

root in the adolescent athlete. Always advise the athlete to eschew low intake of calcium because this is a significant factor in later development of osteoporosis. The adolescent female accumulates up to 240 mg of calcium in the bone mass each day, whereas the male adds 400 mg per day; increased calcium amounts are added during periods of rapid growth. All children, adolescents, and their parents (guardians) should be taught that a major issue in later osteoporosis development is to ensure sufficient calcium intake during childhood and adolescence. Adolescents have a daily calcium requirement of 1200 to 1500 mg, and another 400 mg per day is added for pregnant or breastfeeding women. There are many foods rich in calcium, such as canned sardines, salmon (with bones), tofu, skim milk, and yogurt (plain, nonfat). Calcium absorption can be enhanced with vitamin D, phosphorus, and citric acid, whereas inhibition of calcium absorption is noted with phytates, oxalates, and iron.

Other factors in osteoporosis development include physical inactivity, drug abuse (particularly nicotine addiction), and estrogen deficiency. Individuals at increased risk for low BMD or osteopenia should be cautioned about use of depomedroxyprogesterone acetate (Depo-Provera) because research has revealed it leads to bone loss.<sup>1,114–118</sup> As noted earlier, weight-resistant exercising may be beneficial in the prevention of stress fractures.

### **INJURIES**

Injuries in female and male adolescent athletes are generally the same for the same sports played, with the exception of an increase in anterior cruciate ligament (ACL) (noncontact) and patellofemoral disorders (PFD) in females. 9,10,119-126 The specific sport and intensity of play and not the gender of the athlete determine the prevalence and nature of injuries in most situations. Reduction of injuries in both genders is based on such basic and time-honored principles as appropriate sports equipment, proper training, and expert attention to exercise- or sports-related injuries before, during, and after the specific sport season. The athlete with low BMD, such as an amenorrheic female, is at increased risk for stress fractures with exercise, especially if the exertion is intense. Approximately half of all sports injuries in both genders are associated with overuse injuries characterized by microtrauma-induced damage to musculotendinous units. Lumbar spine and knee injures are common in female and male athletes involved in gymnastics, basketball, and volleyball.

The reasons for an increased prevalence of PFD in female athletes are not clear and remain unproven. <sup>119–126</sup> Cause is linked to such factors as the controversial concept of the female having an increased Q angle linked to a pelvis that is wider and a femoral notch that is narrower versus the male athlete; other cited factors include increased flexibility, muscles that are not so developed (eg, the vastus medialis obliquus), greater genu valgum, and increased external tibial tortion seen in the female. <sup>13</sup> Some research identifies neuromuscular reflex patterns specific to females as contributing to PFD and ACL injuries. <sup>1,16</sup>

Estrogen and progestin receptors are found in knee synovial tissues and this may be involved as well in injury tendencies of female athletes, although more research is needed. Some research has suggested that there are increased ACL injuries during the ovulatory phase of the menstrual period versus other menstrual phases. Studies note that foot problems in the female athlete are related to females often wearing sports shoes that were designed for the anatomy of males; these injuries include corns, bunions, calluses, and metatarsalgia. 1,16

# BREAST INJURIES Nipple Injury

The nipple is often the breast part most injured in sports because it is the most prominent breast part. Jogging or other breast motion during exercise can lead to abrasive nipple injury (acute or chronic) as a result of constant or frequent nipple rubbing. It is sometimes called jogger's nipple in male and female runners, associated with shirts that are tight-fitting as well as brassieres or other irritating clothes in contact with the nipples. 127–130 Nipple abrasion and trauma worsens if the nipple(s) stay in contact with any irritating or abrasive clothing or object while the sports-related motion continues. In 1 report there was a 20:1 male/female ratio of such trauma to nipples in marathon runners. 129 Complicating this injury is the presence of cold air and/or direct stimulation, which can induce nipple musculature to produce a prominent nipple. Painful, raw, and even bleeding nipples in bicycle riders exposed to cold air or wind have led to the term bicyclist's nipple. 130

To prevent this exercise-induced nipple injury, various steps of prevention are recommended, such as always using properly fitted sports brassieres and other measures as presented in **Table 11**. Always use good hygiene, reduce or prevent nipple trauma, and provide antibiotic management if secondary infection occurs. The clinician should also remember that a painful, bleeding nipple may also be suggestive of other diagnoses, such as nipple intraductal papilloma or carcinoma. 1,39,47

### Other Breast Injury

Direct trauma to breast tissue may also occur during sports play and although not common may result in breast contusion, abrasion, hematoma, or laceration. 1,39,47 The mechanism of such damage to mammary gland tissue may be from falls, seatbelt injuries, kicks, injuries from elbows, or abrasion injury from brassiere parts (ie, clips, straps, hooks, metal underwire). 1 Contusion of the breast is typically mild and caused by superficial capillary rupture, resulting in edema and ecchymosis that normally resolves within 21 days. A breast hematoma occurs because of a forceful hit, leading to deep blood vessel bleeding that may resolve into fat necrosis that has secondary induration, scarring, and even calcification for many years; this calcification may be misdiagnosed later as breast carcinoma. Breast trauma can also lead to mastitis or breast abscess. **Table 12** outlines basic management principles for such breast injuries. A laceration of the breast is typically closed surgically and then the athlete is followed for potential development of a painful breast abscess (see **Table 12**).

Thrombophlebitis of the superficial breast veins is called Mondor disease and may be a rare complication of breast trauma; however, history of breast injury is often not

# Table 11 Prevention of exercise-induced nipple injury

Before and during the physical activity, use a plastic bandage or petroleum jelly

Females should always wear a properly fitted sports brassiere

Try to avoid exercising in cold weather

Wind-breaking clothes can be placed over the chest area

Remember that nipple prominence and injury risk are increased in pregnancy

Data from Greydanus DE, Patel DR. The female athlete: before and beyond puberty. Pediatr Clin North Am 2002;49:560.

Table 12 Management of trauma-related breast injuries			
Contusion	Application of cold every 15–20 minutes for several hours Appropriate analgesia Firm support		
Abrasion	Direct pressure to control bleeding Suturing may be necessary		
Laceration	Close with steri-strips or sutures Use good hygiene principles Apply a firm postclosure dressing She should wear a supportive brassiere (including at night) Pain and swelling can be reduced with a cold pack Provide a tetanus toxoid if warranted Antibiotics may be needed, depending on the situation		
Hematoma	Most resolve without treatment Surgical aspiration may be necessary		

Reprinted from Greydanus DE, Patel DR. The female athlete: before and beyond puberty. Pediatr Clin North Am 2002;49:560; with permission.

identified.<sup>1,39,47</sup> There is usually spontaneous and full resolution. Females with augmentation surgery with silicone breast implants may develop implant rupture, bleeding, and breast deformity if subjected to direct breast trauma.

### **SUMMARY**

After centuries of being excluded from sports play, the female athlete has become a common part of competitive and noncompetitive sports activity in the twenty-first century around the world. 1,131 Male and female prepubertal athletes have basically the same physical potential in such parameters as weight, height, endurance, motor skills, strength (see **Table 2**), body fat percentages, hemoglobin levels, and injury risks. Puberty changes these dynamics in the female. Important issues for the female athlete include IDA, SUI, breast issues (eg, pain, asymmetry, injury), female athlete triad (menstrual dysfunction, abnormal eating patterns, osteopenia or osteoporosis), effects of oral contraception and menstruation on sports performance, and exercise during pregnancy. 1,131–137 Pediatricians can encourage the safe and rewarding sports play of adolescent females and help them prevent or manage such sports-related phenomena so they can stay in the game and prepare for a lifetime of exercise for their enhanced health and enjoyment. 1,3,4,137

### **REFERENCES**

- 1. Greydanus DE, Patel DR. The female athlete: before and beyond puberty. Pediatr Clin North Am 2002;49:553–80.
- 2. Joy EA, Van Hala S, Cooper L. Health-related concerns of the female athlete: a lifespan approach. Am Fam Physician 2009;79(6):79–84.
- 3. Greydanus DE, Tsitsika AK. Special considerations for the female athlete. In: Patel DR, Greydanus DE, Baker RJ, editors. Pediatric practice: sports medicine. New York: McGraw-Hill Medical Publishers; 2009. p. 86–101. Chapter 9.
- 4. Greydanus DE, Patel DR. Medical aspects of the female athlete at puberty. International Sportmed J 2004;5(1):1–25.

- 5. Griffin LY. The female athlete. In: DeLee JC, Drez D Jr, Miller MD, editors. DeLee & Drez's orthopaedic sports medicine. Principles and practice. Philadelphia: Elsevier/Saunders; 2003. p. 505–20. Chapter 13.
- Piya-Anant M. Common gynecologic problems in female athletes. Siriraj Med J 2008;60(6):366–7.
- 7. Nattiv A, Arendt EA, Hecht SS. The female athlete. In: Garrett WE, Kirkendall DT, Squire DL, editors. Principles and practice of primary care sport medicine. Philadelphia: Lippincott Williams and Wilkins; 2001. p. 93–113. Chapter 8.
- 8. Beunen G, Malina RM. Growth and physical performance relative to the timing of the adolescent spurt. Exerc Sport Sci Rev 1988;16:503.
- 9. Greydanus DE, Patel DR, Luckstead EF. Office orthopedics and sports medicine symposium. Adolesc Med 1998;9:425–626.
- Patel DR, Nelson TL. Sport injuries in adolescents. Pediatr Clin North Am 2000; 84:983–1007.
- 11. Wilmore JH. The application of science to sport: physiologic profiles of male and female athletes. Can J Appl Sport Sci 1979;4:103–15.
- 12. Komi PV, editor. Strength and power in sport. Oxford (UK): Blackwell Scientific; 1992. p. 404.
- 13. Ireland ML. Special concerns of the female athlete. In: Fu F, Stone R, editors. Sports injuries: mechanisms, prevention and treatment. 2nd edition. Baltimore (MD): Williams and Wilkins; 2000. p. 156–87.
- 14. Malina RM. Effects of physical activities on growth in stature and adolescent growth spurt. Med Sci Sports Exerc 1994;26:759.
- 15. Malina RM. Physical growth and biological maturation of young athletes. Exerc Sport Sci Rev 1994;22:389.
- 16. Yurko-Griffin L, Harris SS. Female athletes. In: Sullivan A, Anderson S, editors. Care of the young athlete. Rosemont (IL): American Academy of Orthopedic Surgery and American Academy of Pediatrics; 2000. p. 137–48. Chapter 15.
- 17. Kulkarni R, Gera R, Scott-Emuakpor AB. Adolescent hematology. In: Greydanus DE, Patel DR, Pratt HD, editors. Essential adolescent medicine. New York: McGraw-Hill Medical Publishers; 2006. p. 371–90. Chapter 17.
- 18. Patel DR. Hematologic conditions. In: Patel DR, Greydanus DE, Baker RJ, editors. Pediatric practice: sports medicine. New York: McGraw-Hill Medical Publishers; 2009. p. 167–80. Chapter16.
- 19. Harris SS. Exercise-related anemia. In: Drinkwater BA, editor. Women in sport. Oxford (UK): Blackwell Scientific; 2000. p. 311–20. Chapter 21.
- 20. Risser WL, Risser JM. Iron deficiency in adolescent and young adults. Phys Sportsmed 1990;18:87–101.
- 21. Balaban E, Cox J, Snell P, et al. The frequency of anemia and iron deficiency in the runner. Med Sci Sports Exerc 1989;21:643–8.
- 22. Greydanus DE, Torres AD, Wan JH. Genitourinary and renal disorders. In: Greydanus DE, Patel DR, Pratt HD, editors. Essential adolescent medicine. New York: McGraw-Hill Medical Publishers; 2006. p. 329–70. Chapter 16.
- 23. Nattiv A. Track and field. In: Drinkwater BA, editor. Women in sport. Oxford (UK): Blackwell Scientific; 2000. p. 470–85. Chapter 32.
- 24. Gera T, Sachdev HP, Nestel P. Effect of iron supplementation on physical performance in children and adolescents: systemic review of randomized controls. Indian Pediatr 2007;44(1):15–24.
- 25. Bo K. Urinary incontinence, pelvic floor dysfunction, exercise, and sports. Sports Med 2004;34(7):451-64.

- 26. Fine PM. Urinary symptoms: incontinence. In: Hillard PJA, editor. The 5-minute obstetrics and gynecology consult. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2008. p. 46–7.
- 27. NIH Consensus Development Panel. Urinary incontinence in adults. JAMA 1989; 261:2685–90.
- 28. Hindle WH. The breast and exercise. In: Hale W, editor. Caring for the exercising woman. New York: Elsevier Science Publishers; 1991. p. 83–92. Chapter 8.
- 29. Kaul P, Beach RK. Breast disorders. In: Greydanus DE, Patel DR, Pratt HD, editors. Essential adolescent medicine. New York: McGraw-Hill Medical Publishers; 2006. p. 569–90. Chapter 27.
- 30. Shangold MM. Gynecologic concerns in the woman athlete. Clin Sports Med 1984:3:869–79.
- 31. Friedenreich CM, Rohan TE. A review of physical activity and beast cancer. Epidemiology 1995;6:311–7.
- 32. Thune I, Brenn T, Lund E, et al. Physical activity and the risk of breast cancer. N Engl J Med 1997;336:1269–75.
- 33. Hoffman-Goetz L, Husted J. Exercise and breast cancer: review and critical analysis of the literature. Can J Appl Physiol 1994;19:237–52.
- 34. Frisch RE, Wyshank G, Albright NL, et al. Lower prevalence of breast cancer and cancers of the reproductive system among former college athletes compared to nonathletes. Br J Cancer 1995;52:885–91.
- 35. Greydanus DE, Patel DR, Baxter TL. The breast and sports: issues for the clinician. Adolesc Med 1998;9:533–50.
- 36. Haycock CE. How I manage breast problems in athletes. Phys Sportsmed 1987; 15:89–95.
- 37. Gehlsen S, Stoner LJ. The female breast in sports and exercise. Med Sport Sci 1987;24:13–22.
- 38. Greydanus DE, Tsitsika AD, Gains MJ. The gynecology system and the adolescent. In: Greydanus DE, Feinberg AN, Patel DR, et al, editors. Pediatric physical diagnosis. New York: McGraw-Hill Medical Publishers; 2008. p. 701–50. Chapter 21.
- 39. Greydanus DE, Matytsina L. Breast disorders in children and adolescents. Prim Care Clin Office Pract 2006;33:455–502.
- 40. Gehlsen G, Albohm M. Evaluation of sports bras. Phys Sportsmed 1980;8: 88–97.
- 41. Lorentzen D, Lawson L. Selected sports bras: a biomechanical analysis for breast motion while jogging. Phys Sportsmed 1987;15:128.
- 42. Berger-Dumound J. Sports bras: everything you need to know from A to D. Women's Sports and Fitness 1986;8:31–49.
- 43. Cummins C. Sports bra round-up. Women's Sports Fitness 1989;4:66.
- 44. Lee J. Sport support. Women's Sports and Fitness 1995;17:72-3.
- 45. American Society for Testing and Materials (ASTM). Standard classification of brassieres. 1982 Yearbook Standard F753-82. Philadelphia: ASTM; 1982.
- 46. Sports bras. Women's Sports and Fitness 1995;17:72.
- 47. Greydanus DE, Omar HA, Tsitsika AK, et al. Menstrual disorders in adolescent females: current concepts. Dis Mon 2009;55(2):39–114.
- 48. Pfeifer S, Patrizio P. The female athlete: some gynecological considerations. Sports Med Athlet Rev 2002;10(1):2–9.
- 49. Moller-Nielson J, Hammar M. Women's soccer injuries in relation to the menstrual cycle and oral contraceptive use. Med Sci Sports Exerc 1989;21:152–60.
- 50. Redman LM, Loucks AB. Menstrual disorders in athletes. Sports Med 2005; 35(9):747–55.

- 51. Ireland ML, Ott SM. Special concerns of the female athlete. Clin Sports Med 2004:23:623–36.
- 52. Beals KA, Meyer NL. Female athlete triad update. Clin Sports Med 2007;26: 69–89.
- 53. Nattive A, Loucks AB, Manore MM, et al. American College of Sports Medicine Position Stand. The female athlete triad. Med Sci Sports Exerc 2007;39: 1867–82.
- 54. Brunet M II. Female athlete triad. Clin Sports Med 2005;24:623-36.
- 55. Constantini NW, Gubnov G, Lebrun CM. The menstrual cycle and sports performance. Clin Sports Med 2005;24:51–82.
- 56. American Academy of Pediatrics. Medical concerns in the female athlete. Pediatrics 2000;106:610–3.
- 57. Greenfield TP, Blythe M. Menstrual disorders in adolescents. In: Greydanus DE, Patel DR, Pratt HD, editors. Essential adolescent medicine. New York: McGraw-Hill Medical Publishers; 2006. p. 591–612. Chapter 28.
- Gutterman DD, Hoffmann RG, Moraski L. Prevalence of the female athlete triad in high school athletes and sedentary students. Clin J Sport Med 2009;19(5): 421–8.
- 59. Callahan LR. The evolution of the female athlete: progress and problems. Pediatr Ann 2000:29:149–53.
- 60. Nichols JF, Rauh MJ, Lwson MJ, et al. Prevalence of female athlete triad among high school athletes. Pediatrics 2008;160(2):137–42.
- 61. Cobb KL, Bachrach LK, Greendale G, et al. Disordered eating, menstrual irregularity, and bone mineral density in female runners. Med Sci Sports Exerc 2003;35: 711–9.
- 62. Marshall LA. Amenorrhoea. In: Drinkwater BA, editor. Women in sport. Oxford (UK): Blackwell Scientific; 2000. p. 377–90. Chapter 26.
- 63. The Practice Committee of the American Society for Reproductive Medicine. Current evaluation of amenorrhea. Fertil Steril 2006;86(Suppl 4):S148–55.
- 64. Loucks AB, Vaitukaitis J, Cameron JL. The reproductive system and exercise in women. Med Sci Sports Exerc 1992;24(Suppl):S288–93.
- 65. Marshall LA. Clinical evaluation of amenorrhea in active and athletic women. Clin Sports Med 1994;13:371–89.
- 66. Hobart JA, Smucker DR. The female athlete triad. Am Fam Physician 2000;61: 357–64.
- 67. Yeager KK, Agostini R, Nattive A, et al. The female athlete triad: disordered eating, amenorrhea, osteoporosis. Med Sci Sports Exerc 1993;25:775–7.
- 68. DeSouza MJ, Williams NI, Alleyne J, et al. Correction of misinterpretations and misrepresentations of the female athlete triad. Br J Sports Med 2007;41(1): 58–9.
- 69. Laughlin GA, Yen SS. Hypoleptinemia in women athletes: absence of a diurnal rhythm with amenorrhea. J Clin Endocrinol Metab 1997;82:318–21.
- 70. Thong FS, McLean C, Graham TE. Plasma leptin in female athletics: relationship with body fat, reproductive, nutritional and endocrine factors. J Appl Physiol 2000;88:2037–44.
- 71. Loucks AB, Strachenfeld NS, DiPetro L. The female athlete triad: do female athletes need to take special care to avoid low energy availability? Med Sci Sports Exerc 2006;38:1694–700.
- 72. DeSouza MJ, Lee DK, Van Heest JL, et al. Severity of energy-related menstrual disturbances increases in proportion to indices of energy conservation in exercising women. Fertil Steril 2007;88(4):971–5.

- 73. Sundgot-Borgen J. Eating disorders. In: Drinkwater BA, editor. Women in sport. Oxford (UK): Blackwell Scientific; 2000. p. 364–76. Chapter 25.
- 74. American Psychiatric Association. Practice guideline for the treatment of patients with eating disorders, 3rd edition. Am J Psychiatry 2006;163(1 Suppl):4–54.
- 75. Sanborn CF, Horea M, Siemers BJ, et al. Disordered eating and the female athlete triad. Clin Sports Med 2000;19:1–11.
- 76. Mitan LAP. Diet, eating disorders: anorexia nervosa. In: Hillard PJA, editor. The 5-minute obstetrics and gynecology consult. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2008. p. 268–9.
- 77. Currie A, Morse ED. Eating disorders in athletes: managing the risks. Clin Sports Med 2005;24:871–83.
- 78. National Institutes of Health. Optimal calcium intake. NIH Consensus Statement 1994;12:1–31.
- 79. Teegarden D, Weaver CM. Calcium supplementation increases bone density in adolescent girls. Nutr Rev 1994;52:171.
- 80. Hergenroder AC, Smith EO, Shypailo R, et al. Bone mineral changes in young women with hypothalamic amenorrhea treated with oral contraceptives, medroxyprogesterone, or placebo over 12 months. Am J Obstet Gynecol 1997; 176(5):1017–25.
- 81. American Academy of Pediatrics. Amenorrhea in adolescent athletes. Pediatrics 1989;84:394–5.
- 82. Polatti F, Perotti F, Filippa N, et al. Bone mass and long-term monophasic oral contraceptive treatment in young women. Contraception 1995;51:221–4.
- 83. Rosen CJ, editor. Primer on the metabolic bone diseases and disorders of mineral metabolism. 6th edition. Hoboken (NJ): Wiley and Sons Publishers. American Society for Bone and Mineral Research; 2006.
- 84. Frankovich RJ, Lebrun CM. Muscle cycle, contraception and performance. Clin Sports Med 2000:19:1–6.
- 85. Labrun CM. Effects of the menstrual cycle and oral contraceptives on sports performance. In: Drinkwater BA, editor. Women in sport. Oxford (UK): Blackwell Scientific; 2000. p. 37–61. Chapter 3.
- 86. Greydanus DE, Patel DR, Rimsza ME. Contraception in the adolescent: an update. Pediatrics 2001;107:562–73.
- 87. Greydanus DE, Rimsza ME, Matytsina L. Contraception for college students. Pediatr Clin North Am 2005;52:135–61.
- 88. Kamboj MK. Metabolic bone disease in adolescents: recognition, evaluation, treatment, and prevention. Adolesc Med 2007;18:24–46.
- 89. Moller-Nielson J, Hammar M. Sports injuries and oral contraceptive use: is there a relationship? Sports Med 1991;12:152–60.
- 90. Paupoo A, Glass MLS. Osteoporosis and osteopenia. In: Hillard PJA, editor. The 5-minute obstetrics and gynecology consult. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2008. p. 130–1.
- 91. Morris SN. Exercise during pregnancy: a critical appraisal of the literature. J Reprod Med 2005;50:81–8.
- 92. Weiss Kelly AK. Practical exercise advice during pregnancy: guidelines for active and inactive women. Phys Sportsmed 2005;33(6):1–10.
- 93. Mottola MF, Wolfe LA. The pregnant athlete. In: Drinkwater BA, editor. Women in sport. Oxford (UK): Blackwell Science; 2000. p. 194–207. Chapter 14.
- 94. DeHoop TA. Exercise in normal pregnancy. In: Hillard PJA, editor. The 5-minute obstetrics and gynecology consult. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2008. p. 342–3.

- 95. American College of Obstetrics and Gynecology (ACOG) Committee on Obstetric Practice. Exercise and the postpartum period. ACOG Committee Opinion No. 267. Obstet Gynecol 2002;99(1):171–3.
- 96. Prentice A. Should lactating women exercise? Nutr Rev 1994;52:358-60.
- 97. Campaigne BN. Diabetes and sport. England. In: Drinkwater BA, editor. Women in sport. Oxford (UK): Blackwell Scientific; 2000. p. 265–79. Chapter 18.
- 98. Golden NH. Eating disorders: anorexia nervosa and bulimia nervosa in the adolescent. In: Greydanus DE, Patel DR, Pratt HD, editors. Essential adolescent medicine. New York: McGraw-Hill Medical Publishers; 2006. p. 635–50. Chapter 30.
- 99. Nelson ME, Fiatarone MA, Morganti CM, et al. Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures. JAMA 1994;272:1909.
- Gibson JH, Mitchell A, Harries MG, et al. Nutritional and exercise-related determinants of bone density in elite female runners. Osteoporos Int 2004;15:611–8.
- 101. Rutherford OM. Spine and total body bone mineral density in amenorrheic endurance athletes. J Appl Phys 1993;74:2904–8.
- 102. Nelson ME, Fisher EC, Catsos D, et al. Diet and bone status in amenorrheic runners. Am J Clin Nutr 1986;43:910–6.
- 103. Fruth SJ, Worrell TW. Factors associated with menstrual irregularities and decreased bone mineral density in female athletes. J Orthop Sports Phys Ther 1995;22:26–38.
- 104. Linnell ST, Stager JM, Blue PW, et al. Bone mineral content and menstrual regularity in female runners. Med Sci Sports Exerc 1984;16:343–8.
- 105. Snyder AC, Wenderoth MP, Johnston CC, et al. Bone mineral content of elite lightweight amenorrheic women. Hum Biol 1986;58:863–9.
- 106. Rosenthal DI, Mayo-Smith W, Hayes CW, et al. Age and bone mass in premenopausal women. J Bone Miner Res 1989;4:533–8.
- 107. Wolman RL, Clark P, McNally E, et al. Menstrual state and exercise as determinants of spinal trabecular bone density in female athletes. Br Med J 1990;301:516–8.
- 108. Harber VJ, Webber CE, Sutton JD, et al. The effect of amenorrhea on calcaneal bone density and total bone turnover in runners. Int J Sports Med 1991;12:505–8.
- Warren MP, Brooks-Gunn J, Fox RP, et al. Lack of bone accretion and amenorrhea: evidence for a relative osteopenia in weight bearing bones. J Clin Endocrinol Metab 1991;72:847–53.
- 110. Wolman RL, Clark P, McNally E, et al. Dietary calcium as a statistical determinant of spinal trabecular bone density in amenorrheic and estrogen-replete athletes. Bone Miner 1992;17:415–23.
- 111. Lane JM, Riley EH, Wirganowicz PC. Osteoporosis: diagnosis and treatment. J Bone Joint Surg Am 1996;78:618.
- 112. Lloyd T, Meyers C, Buchanan JR, et al. Collegiate women athletes with irregular menses during adolescence have decreased bone density. Obstet Gynecol 1998;72:639–42.
- 113. Theintz G, Buchs B, Rizzoli R, et al. Longitudinal monitoring of bone mass accumulation in healthy adolescents: evidence from a marked reduction after 16 years of age at the levels of lumbar spine and femoral neck in female subjects. J Clin Endocrinol Metab 1992;75:1060–5.
- 114. American College of Obstetricians and Gynecologists. Use of hormonal contraception in women with coexisting medical conditions. ACOG Practice Bulletin, Number 73. Obstet Gynecol 2006;107:1453–72.
- 115. Emery-Cohen A, Kaunitz AM. Contraception: hormonal: injection. In: Hillard PJA, editor. The 5-minute obstetrics and gynecology consult. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2008. p. 250–1.

- Schrager SB. DMPA's effect on bone mineral density: a particular concern for adolescents. J Fam Pract 2009;58(5):E1–8.
- 117. Cromer BA, Scholes D, Berenson A, et al. Depot medroxyprogesterone acetate and bone mineral density in adolescents. The black box warning: a position paper of the Society for Adolescent Medicine. J Adolesc Health 2006;39: 296–301.
- 118. Gibson J. Osteoporosis. In: Drinkwater BA, editor. Women in sport. Oxford (UK): Blackwell Scientific; 2000. p. 391–406. Chapter 27.
- 119. Cline S. Acute injuries of the knee. In: Patel DR, Greydanus DE, Baker RJ, editors. Pediatric practice: sports medicine. New York: McGraw-Hill Medical Publishers; 2009. p. 313–29. Chapter. 26.
- 120. Patel DR, Luckstead Sr EF, Greydanus DE. Sports injuries. In: Greydanus DE, Patel DR, Pratt HD, editors. Essential adolescent medicine. New York: McGraw-Hill Medical Publishers; 2006. p. 677–92. Chapter 33.
- 121. Loud K, Micheli L. Common athletic injuries in adolescent girls. Curr Opin Pediatr 2001;13:317–27.
- 122. Knowles SB. Is there an injury epidemic in girls sports? Br J Sports Med 2010; 44:38–45.
- 123. Hagglund M, Walden M, Atroshi I. Preventing knee injuries in adolescent female football players—design of a cluster randomized controlled trial. BMC Musculoskelet Disord 2009;10:75–82.
- 124. Sands WA, Shaltz BB, Newman AP. Women's gymnastics injuries: a five year study. Am J Sports Med 1993;21:271–6.
- 125. Liu SH, Al-Shaikh R, Panossian V, et al. Primary immunolocalization of estrogen and progesterone target cells in the human anterior cruciate ligament. J Orthop Res 1996;14:526–33.
- 126. Wojtys EM, Huston LG, Lindenfeld TN, et al. Association between the menstrual cycle and anterior cruciate ligament injuries in female athletes. Am J Sports Med 1998;26:614–9.
- 127. Otis CL. Women and sports: breast and nipple injuries. Sports Med Dig 1988; 10:7.
- 128. Rubin CJ. Sports injuries in the female athlete. N J Med 1991;88:643-5.
- 129. Neguin ND. More on jogger's ailments. N Engl J Med 1978;298:405-6.
- 130. Powell B. Bicyclist's nipples. JAMA 1983;249:2457.
- 131. Torstveit MK, Sungot-Borgen J. The female athlete triad: are elite athletes at increased risk? Med Sci Sports Exerc 2005;37:184–93.
- 132. Patel DR, Greydanus DE. The adolescent athlete. In: Hofmann AD, Greydanus DE, editors. Adolescent medicine. 3rd edition. Stamford (CT): Appleton and Lange; 1997. p. 612–4. Chapter 28.
- 133. Fulkerson J. Diagnosis and treatment of patients with patellofemoral pain. Am J Sports Med 2002;30:447–56.
- 134. Eliakim AB. Exercise training, menstrual irregularities, and bone development in children and adolescents. J Pediatr Adolesc Gynecol 2003;16(4):201–6.
- 135. Madd LM, Fornetti W, Pivarnik JM. Bone mineral density in college female athletes. J Athl Train 2007;43(3):403–8.
- 136. Barrack M, Rauh MJ, Barkai H-S, et al. Dietary restraint and low bone mass in female adolescent endurance runners. Am J Clin Nutr 2008;87(1):36–43.
- 137. Omar H, Greydanus DE, Patel DR, et al. Pediatric and adolescent sexuality and gynecology. New York: Nova Biomedical Books; 2010. 450 pages.