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Chapter 12

THE FEMALE ATHLETE

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The female athlete has today become an accepted part of sports in elementary school, junior high, senior high, and college. This chapter covers various aspects of the prepubertal and pubertal female athlete. An historical perspective will be presented and selected comments provided on psychological and physiologic aspects of the female athlete. Concepts of adolescent gynecology are reviewed, including breast concerns and menstrual dilemmas in relation to sports activity. Exercise during pregnancy is also considered. Iron deficiency anemia and stress urinary incontinence

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and selected comments on injuries in the female athlete are discussed. The pediatrician is encouraged to actively care for the girl and adolescent female who wishes to engage in the joy of sports activities.

INTRODUCTION

As the twentieth century progressed, the female athlete became an accepted part of sports in elementary school, junior high, senior high, and college. This discussion covers various aspects of the prepubertal and pubertal female athlete. After an historical perspective, selected comments are provided on psychological and physiologic aspects of the female athlete. Concepts of adolescent gynecology are reviewed, including breast concerns and menstrual dilemmas in relation to sports activity. Exercise during pregnancy is also considered. After reflecting on iron deficiency anemia and stress urinary incontinence, selected comments are provided on injuries in the female athlete. The pediatrician is encouraged to actively care for the girl and adolescent female who wishes to engage in the joy of sports activities.

HISTORY

The twentieth century witnessed a considerable growth in society's acceptance of the female athlete. The Ancient Greek Olympics were only for men and women were banned in 1896 from the first modern Olympic Games (1,2). Over the course of the twentieth century, outstanding female athletes gradually educated the public about sport being an activity in which both sexes can succeed (see table 1). In the 1900 Olympic Summer Games, a few women were allowed to play golf and lawn tennis; in 1912, women were allowed in the diving and swimming events (3). This encouraged a growing number of female participants in subsequent Olympic Games: 0.0% to 1896, 1.7% in 1900, 0.9% in 1904 and 35.1% in 1996 (2). World War II had a tremendous impact on the entire world, and the sports world was not excluded. Men went off to war and women stayed home, handling their jobs. The concept that

women can accomplish all types of physical tasks was conveyed in the WWII depiction of Rosie the Riveter, whereas the success of the All American Girls' Baseball League during WWII illustrated the athletic talent of females.

Table 1. Examples of pioneering female athletes in the 20th century

1900:	Charlotte Cooper: Tennis champion. Olympic games
1920's:	Two female stars: Helen Willis: Female tennis champion Gertrude Ederle: Swam English Channel two hours faster than anyone, even the men
1932:	Mildred "Babe" Didrikson-starred in the 1932 Olympic Games (Track and Field: 2 golds and one silver)
1960s:	Women's professional tennis: Billy Jean King
1972:	Olga Korbut: 3 gold and 1 silver in gymnasts: 1972 Olympic Games
1984:	Joan Benoit-wins first woman's marathon
1996:	Mia Hamm and United States Woman's soccer team wins the gold medal in the Olympic Games

The last **half** of the twentieth century escalated this trend. Title IX of the Educational Amendment Act of 1972 by the United States Congress mandated equal athletic facilities for both women and men attending any college or university receiving federal financial assistance (4). Guidelines of Title IX were passed into law in 1975 and this created an equal opportunity situation for females in sports (1). This approach eventually filtered down to the high school level as well, allowing improved training, coaching, and equipment for girls in various sports activities (5). The involvement of women and girls in sports play increased 700% in the 1980s, whereas the number of girls' high school teams increased from 15,000 to 70,000 (6). In the decade of the 90s, the number of women in intercollegiate athletics increased nearly 50%, whereas 1 in 3 high school girls participated in sports in 1998 versus 1 in 27 in 1972 (5,7).

The media has become an advocate for female athletics and encourages millions of people to watch women in sports. For example,

television coverage from the 1996 Centennial Olympic Games in Atlanta, Georgia to the 2008 Beijing Olympic Games broadcast throughout much of the world showed that women's sports were just as popular, if not more so, as the men's sports. This media influence has become considerable, especially as women have approached men in various records (8). Women now compete in sports that were previously limited to men (such as ice hockey or rugby). Thus, tremendous progress has occurred for women and girls in sports over the past century and more can be anticipated for the twenty-first century (9).

PSYCHOLOGY

Involvement in sports activity can be a positive and enriching experience in the physical and moral development of children and adolescents (10-12). Sports participation by girls can be a wonderful way to encourage regular exercise throughout life by setting the stage in childhood and adolescence for a physically active life. A lifetime of exercise leads to improved cardiovascular health, less obesity, and even reduced risk for breast cancer (13). High school females who are active in sports are more likely to graduate, have fewer pregnancies as adolescents, and demonstrate greater self-esteem than those who are not active in sports (3).

Nevertheless, there are still many problems that the female athlete must face. As noted with male athletes, the pressure from parents, coaches, and peers for the athlete to excel can be tremendous and overwhelming (11,12, 14, 15). Those identified with the ability to become "superstars" may be driven to the point of being abused if parents live their lives through their children or seek to have the child win a future scholarship or if coaches place their job security and personal self-esteem on the success of their student athletes. Many athletes must learn to play with pain for the sake of "winning at all cost" (16). All athletes can become involved with sports doping in efforts to win or become more attractive to peers (17). Gender still determines much about sports participation (16). The relationship between violence in our society and violence in sports remains to be determined (18).

Many high school female athletes drop out from team competition, because of burnout from overtraining and the lack of time for other activities (6). The high school female athlete is more apt to drop out than the male athlete, because sports participation by girls is not seen by peers to be as important as it is with boys (16). Often, only those athletes who play competitively on "team" sports are physically active on a regular basis; the rest sit and watch the games, deriving no physiologic benefit from the competition. Regardless of whether or not the female athlete receives playing time, societal pressure to be "thin" remains, leading to various nutritional consequences (see chapter on eating disorders) (19,20).

A serious, yet understudied and often hidden problem in the female athlete is the sexual harassment and abuse she may face from predator or pedophilic coaches, trainers, fellow male peer athletes, and others (19,21-26). It is an issue only formally recognized since the mid-1980s, and information is quite limited (21). Society places coaches, especially if winning, in a superior position over their athletes and allows the development of a sports milieu in which the line of abuse becomes ambiguous and difficult to identify (27). Male athletes are often more accepting than female athletes of actions that others consider abusive. Parents, peers, and school officials may be hard to convince that abuse has occurred and may subject the female athlete to considerable disbelief and ridicule. She may have already experienced abuse and then be quite vulnerable to further abuse, especially if she has a distant relationship with her own father (24). She may not know it is wrong or how to stop the abuse once it has started. Athletes with disordered eating patterns (see later) are often more vulnerable to such abuse as well.

Sporting organizations must have formal policies to screen coaches, trainers, and others for sexual abuse. It is important that parents, athletes, school officials, and others involved in sports acknowledge that this issue occurs and must be removed from athletics as much as possible. School officials must monitor their sports programs for such behavior and develop guidelines to prevent and manage sexual abuse and harassment in their sports programs at all levels. Our athletes, both female and male, must be taught how to resist such dangers, when they occur (28).

PHYSIOLOGY

Children

Prepubertal girls and boys are in comparable physical condition and essentially equal in all sports. Before puberty occurs, girls are similar to boys in terms of height, weight, percent body fat, motor skills, strength, endurance, hemoglobin, thermoregulation, and injury risks (1,6). Girls have the same or slightly less aerobic and anaerobic capacity as boys (6). Earlier studies suggesting that girls are weaker than boys before puberty reflected cultural issues encouraging boys to exercise and play sports from an early age while discouraging girls from doing so. Some research suggests that girls exhibit more joint flexibility than boys in preadolescence and that this continues on into adulthood (15).

Children (both girls and boys) tend to have shorter extremities in relation to total body length than adults. They tend to have a smaller muscle mass with limited strength and minimal response to strength exercises, such as weight training (29). Girls have less aerobic/anaerobic capacity, less percent body fat, less strength, and lower hemoglobin levels than adults (1).

The usual improvement of sports performance in children between the ages of 6 and 12 years is largely due to normal physiologic central nervous system maturation (30). Children can be introduced to various sports as their eye-to-hand sport-related coordination develops sufficiently to permit successful, safe play. Recommended ages for the introduction of specific sports to girls and boys include 6 for tennis, 8 for basketball, and 10 for ice hockey (3,4,6). Girls 8 years and younger may swim faster than boys in some events and overall winning times show no difference (4). In general, girls and boys can play sports together (coed) competitively until about the age of 10 or 11 years if they are of similar physical size.

Adolescents

After puberty has taken place, girls develop an increase in percent body fat in comparison to boys (see chapters 2 and 14). The body fat levels

average 13% to 15% for adult males versus 23% to 27% for adult females. Trained athletes of both sexes can reduce their fat levels considerably (31). For example, elite sprint and distance females may be at 8% to 10% and 12% to 16%, respectively; elite male gymnasts may be at the 4% to 8% fat level. Relative to boys, girls have smaller heart size, cardiac stroke volume, left ventricular mass, lung volume, aerobic capacity, and hemoglobin levels (1,32). Compared with the male athlete, the female athlete is generally smaller, has less overall articular surface, narrower shoulders, a relatively greater varus at the hips, and a greater valgus at the knees. Thermoregulatory capacity in females is comparable to that in males. Although females have fewer sweat glands compared with males, they compensate by having a relatively smaller body mass, relatively large body surface area, and less overall muscle bulk to generate heat (15). Obese, late maturing girls and boys are both at higher risk for heatstroke in hot weather exercise.

Strength in girls is less compared to boys and muscle fiber size is smaller, although the proportion of muscle fiber type is similar (1). There is only a minimal increase in muscle strength after menarche, whereas adolescent boys show gain in muscle strength throughout puberty (33-35). The upper body strength in females, even with training, will remain at 30% to 50% of similarly trained male peers while lower body strength is about 70% that of males (see table 2) (36-38).

Table 2. Comparison of strength between females and males of similar size

Ages birth to 10 years (before puberty): same strength
Ages 11-12: female is 90% as strong as males
Ages 13-14: 85% as strong
Ages 15-16: 75% as strong

In adolescent boys, the maximal speed peak precedes their peak height velocity, whereas strength and power speed peaks after peak height velocity; no such patterns are identified in adolescent girls (33,39,40). Pubertal girls may note the most rapid rate of weight gain 12 to 14 months after their maximum growth velocity (average age 11.5 ± 1 year if sexual maturity rating [SMR] is a 2 to 3); this is due to a smaller

increase in muscle mass with a greater increase in body fat. In girls, there is an increased response to strength and endurance training usually 1 to 2 years after peak height velocity and at SMR 4 to 5. Weight training in adolescent girls will lead to an increase in strength but usually only a slight increase in visible muscle mass. Vigorous training may lead to heightened muscle visibility because of the loss of subcutaneous fat and better muscle definition.

Much of the past stereotype suggesting poor athletic performance of girls and women when compared to boys and men is related to culturally induced lack of strength training and more limited training techniques with inadequate equipment. Also, it should be noted that there are variable responses to exercise noted among all athletes, even among individuals of the same sex at different weights and lifestyles; genetic influence seems a likely explanation for much of this variability.

Adolescent girls are more flexible and better at balance tasks than boys; this increase in flexibility begins in childhood and peaks at 14 or 15 years of age. In boys there is a decrease in overall flexibility from ages 7 or 8 years until midadolescence; it then increases through the rest of puberty (33,34,39,40). As the female adolescent grows in height and weight, she becomes better able to deal with such sports as basketball, volleyball, downhill skiing, swimming, others.

The female athlete may be at an advantage in sports requiring balance (such as gymnastics), because of her shorter extremities and, according to some experts, lower center of gravity; however, studies now note the center of gravity is determined mainly by height and weight and not sex per se (6,41). Late maturing girls are more likely to participate and excel in certain activities emphasizing thin or lean appearance (such as gymnastics, dance, figure skating, and synchronized swimming). Puberty may bring boys closer to a physical optimum for best sports performance while taking some girls away. Girls tend to grow out of their sport in puberty, whereas boys grow into their sport in puberty (6).

THE BREAST AND SPORTS

Exercise

Exercise does not affect breast size by changes in muscle, since the only muscle tissue present is a small amount in the areolar area; however, there may be the appearance of increased breast size due to exercise if there is enlargement of the underlying pectoralis muscle (see chapter 6). Intense exercise from before and after puberty may alter breast size by reducing the fat content of breast tissue (38,42-44). Weight gain or loss due to various factors affects breast size as well. Exercise not only does not lead to breast cancer but, in fact, reduces its risk (42,45-48).

Injuries- Nipple

The nipple, as the most prominent breast part, is a common site of injury in female athletes (see chapter 6). Tight-fitting bras, shirts, or other coarse clothing may lead to an abrasive injury (whether acute or chronic), which is particularly noted with jogging or other motions causing strong, repetitive rubbing of the nipple (6,36,49). The propensity of joggers (females and males) to this injury has led to the term jogger's nipple (42,49-52). The longer the nipple is in contact with an object in this manner, the more likely it is that nipple irritation and subsequent injury will occur. A persistent, painful process may lead to bicyclist's nipple in bicycle athletes riding in a cold wind; this may lead to the development of a bleeding, raw, painful nipple (53). Exposure to the cold or direct stimulation may affect muscle fibers in the nipple, resulting in a prominent nipple. A 20: 1 ratio of males to females with nipple injury in marathon runners has been noted in one study (54).

The differential diagnosis of a painful, bleeding nipple, besides exercise-induced injury, includes an intraductal papilloma and carcinoma (42,49,55). If an athlete has nipple injury from exercise, proper hygiene and treatment of any secondary infection is recommended. Prevention measures are listed in table 3. A well-fitting sports bra may help prevent these injuries.

Table 3. Prevention of exercise-induced nipple injury [61,165]

Use a plastic bandage (i.e., a Band-Aid) or petroleum jelly (Vaseline ^R) before and during the exercise.
Use a properly fitted sports bra.
Avoiding exposure to cold temperatures
Use wind breaking material over the chest
Special attention during pregnancy when nipples are more prominent

Breast parenchyma

Although uncommon, injuries from breast trauma in female athletes may be seen (4,6,36,56) (see chapter 6). Direct trauma (i.e., from falls, elbows, kicks, seat belt injuries, sports equipment) may result in contusions, lacerations, hematomas, or lacerations of breast tissue (42). Contusions are typically mild and reflect only superficial capillary rupture; it may take up to three weeks for normal resolution of typical edema and ecchymosis, which may develop. An abrasion may result from sliding in baseball or from falls in skiing, rodeo events, or biking accidents (mountain biking, road racing). The female athlete's bra may induce an abrasion from bra clips, underwires (metal), hooks, clips, or straps (57).

Lacerations should be carefully closed; a secondary painful abscess may develop. Infrequently, a strong blow to the breast can cause a hematoma. Fat necrosis may eventually result with secondary induration, scarring, and calcification, which can persist for years as residual tissue. Fat necrosis may be confused with breast carcinoma. Table 4 reviews basis management principles for these various injuries.

Trauma may uncommonly lead to thrombophlebitis of superficial breast veins or Mondor's disease (42,55,58). The athlete may or may not be able to identify a specific traumatic event that led to this disorder. Fortunately, spontaneous resolution is the typical course. Also, experts have suggested female athletes involved in contact sports not undergo breast augmentation surgery; direct trauma to the breast area may lead to rupture of silicone-filled implants with resultant bleeding and even breast deformity (50,59).

Table 4. Management of trauma-related breast injuries [61]

Contusion

Application of cold every 15-20 minutes-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 post-closure dressing
She should wear a supportive bra (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

Breast pain

Exercise-induced breast discomfort or pain from breast motion is a frequent, although not always reported, concern in female athletes, particularly in those with larger breasts (i.e., C and D cup) (4,42,44,60). Increased fluid retention seen in some women in the premenstrual phase or with premenstrual tension syndrome may worsen this problem (60-62). A survey of female athletes noted that 31% reported exercise-induced discomfort (soreness, tenderness), whereas 52% of this group with discomfort noted specific exercise-induced breast injury (50). Considerable displacement of the breasts in various directions may occur in female athletes involved in basketball, volleyball, running,

gymnastics, and many other sports (50,60,63,64). This pain from excessive breast motion may prevent some athletes from playing sports and can lead to strain of breast fascial attachments of the pectoralis muscle (49,57).

Prevention of such breast and even shoulder discomfort and pain is by wearing a properly fitted sports brassiere, which provides appropriate support to the breasts and minimizes breast motion (4,6,42, 60,61,63,65-69). A careful matching of the correct bra with each athlete is important, and a change approximately every six months may be necessary. The material should be nonabrasive and have soft and firm cups, few seams, and a limited number of hooks, which should be padded. The sports bra should lift and separate the breasts, limiting their motion as much as possible. Padding of the bra and shoulder straps may be necessary depending on breast size and type of sports activity. Guidelines for sports bras are available (42,50,61,69). These bras should be made of a "breathable" material to deal with sweating, especially during exercise; excessive sweating can lead to excoriations, abscess formation, and cellulitis in the intramammary folds. It is important to keep in mind that there are various causes for breast pain, including breast masses, which also must be considered (see chapter 6) (55,58).

Breast asymmetry

Female athletes with asymmetric breast development may also develop injury without proper protection (15). Asymmetric breast development is a common variant in many adolescents that typically resolves spontaneously by mid-to-late adolescence, although about 25% of adult women have permanent visible breast asymmetry (55) (see chapter 6). It may also reflect a variety of underlying disorders and a careful search for underlying factors is necessary (55,58). Those with asymmetry can be helped with a padded bra, and swimmers may use a bathing suit with breast supports. Large differences in breast size may be improved with foam inserts, which can be found in businesses servicing mastectomy patients.

Galactorrhea

Galactorrhea refers to discharge from the nipple not due to pregnancy (see chapter 6). Although often idiopathic in adolescents, a careful search is necessary for a variety of causes, including hypothyroidism, medications (i.e., oral contraceptives, phenothiazines), pituitary tumors, hypothalamic injury (infection, surgery), depression, anxiety, and others (36,55). Underlying disorders (e.g., pituitary tumors or thyroid dysfunction) must be treated, self-manipulation corrected, and medications that induce galactorrhea stopped.

MENSTRUAL DILEMMAS

Menstrual physiology

Normal menstrual cycles imply a responsive hypothalamic-pituitary-ovarianuterine axis, which controls three menstrual cycle phases: follicular, ovulatory, and luteal (see chapter 7). Increases and decreases in estrogen and progesterone levels control menstrual physiology (55,62). During the follicular phase, increasing estrogen levels from the ovaries lead to endometrial growth characterized by a compact stroma as well as increased number and length of endometrial glands. Estrogen and progesterone are produced by the corpus luteum after ovulation, and the endometrium changes from a proliferative to a secretory state under the effects of progesterone, which reduces the estrogen influence. During the luteal phase, a secretory state, an edematous stroma with dilated/tortuous endometrial glands is characteristic. The corpus luteum becomes atretic. If conception does not occur, the resultant drop in estrogen and progesterone levels leads to endometrial desquamation and menstrual flow.

It can take up to several years (1 to 5) from menarche (onset of menstrual periods) for the adolescent female to have a regular cycle of menstrual periods, and menstrual rhythmicity is subject to a number of influences. A number of menstrual problems may develop, including amenorrhea (absence of menses) and oligomenorrhea (infrequent, irregular bleeding at more than 45-day intervals). In a mature adult

female, the mean interval of menses is 28 days \pm 7 days and the duration of menses is 4 days \pm 2 to 3 days. The median blood loss is about 30 mL per month (with the upper limit of normal defined as 60-80 mL per month).

Amenorrhea can be identified as primary or secondary (see chapter 7). Primary amenorrhea refers to lack of any menses by age 14 at sexually maturity rating (SMR) 1 or lack of menses by age 16 regardless of SMR or pubertal stage. Secondary amenorrhea is defined as absence of menses after menarche for a total of three previous cycle intervals or a total of secondary amenorrhea for 6 months. In the first two years after menarche, it is normal to have amenorrhea for 3 to 6 months. The evaluation of amenorrhea requires a careful history, physical examination, and selective laboratory testing, depending on the specific situation (see chapter 7). Some causes are listed in table 5 (55,58,62).

FEMALE ATHLETE TRIAD

This term was introduced as a serious phenomenon in 1993 by Yeager et al and has evolved into a description of the interrelated spectrums of 1) energy availability (from optimal energy availability to low energy availability with or without an eating disorder), 2) menstrual dysfunction (from eumenorrhea to functional hypothalamic amenorrhea) and 3) bone mineral density (from optimal bone health to osteoporosis) which may occur in female athletes (70,71). A number of sports place major emphasis on either a lean appearance (such as synchronized swimming, figure skating, gymnastics, dance, and diving), a lean body for optimum performance (such as swimming, long-distance running, and cross-country skiing), or various weight classes (such as judo, weight lifting, rowing, wrestling and tae kwon do) (72,73). Some sports (such as gymnastics, ballet, and figure skating) even emphasize a prepubertal appearance. The result of this pressure for the "ideal" body (often found in nonathletic adolescents as well) along with the intense exercise required of many sports can lead to all or parts of this triad.

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
Hypothalamic-induced (such as weight loss, eating disorders, exercise, stress, others)
Pituitary disorders
Polycystic ovary syndrome (hypcrandrogenemia syndromes)
Thyroid disorders
Others
Secondary Amenon-hea
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

Disordered eating

Inappropriate eating patterns are described in 15% to 75% of adolescent athletes (3,7,19,20,74-77). These patterns vary and include fasting, skipping meals, and self-induced emesis and the use of diet pills, laxatives, or diuretics (74,76). A number of vulnerable times may occur in the athlete's-life during which she is more susceptible to nutritionally

poor eating habits. These include the growth spurt, traumatic events (i.e., loss of a loved one, including the coach), entering college, leaving formal athletic competition, and even postpartum depression (73). Females who participate in sports emphasizing low body weights and self-driven need for excessive exercise are especially vulnerable to develop disordered eating patterns; these sports include gymnastics, distance running, swimming, track, and diving (72).

Studies note an incidence of 5% to 20% of anorexia nervosa in ballet dancers, depending on the level of competition (10,19,20,78); however, most athletes with disordered eating patterns do not develop patterns sufficient to meet the diagnostic criteria for anorexia nervosa or bulimia nervosa (see chapter 14). Anorexia nervosa can induce hypothalamic amenorrhea in adolescent females. Osteopenia can occur frequently and early in the course of an adolescent with anorexia nervosa due to estrogen deficiency, elevated glucocorticoid levels, generalized malnutrition, calcium intake deficiency, and reduced bone mass (79).

It appears that she will not develop a normal bone mineral density (BMD), even if her weight is normalized; she may be at risk for osteoporosis later in life. Use of conjugated estrogen or oral contraceptives may increase BMD somewhat in girls with anorexia nervosa and severe malnutrition. The use of estrogen and progesterone in patients with anorexia nervosa yields conflicted results. The best improvement in bone density is in those with anorexia nervosa in whom menstruation was restored (80). Nevertheless, estrogen replacement therapy without an accompanying weight gain is unlikely to protect the bone mass.

Osteoporosis and adolescence

Peak bone mass (PBM) is the total amount of bone mass/density obtained during growth. Approximately 50% to 63% of the PBM is achieved during childhood and 37% to 50% during adolescence. Bone mineralization increases throughout childhood, rises at puberty, and plateaus after sexual maturity. Gains in bone mineralization in young girls tend to be most rapid during early puberty (usually 11-14 years of age, but this can vary depending on age of menarche). Estrogen is critical

for optimal adolescent bone mass acquisition in men as well as women. In sureties of Swiss women (17-20 years), no significant bone mass gains were found after age 17 years (81). Although studies note some continued gains in bone mineral composition occurring into the third decade, bone density cannot be significantly increased beyond the level obtained at the end of growth. Bone mass often starts to decline in adults after age 35 years and its loss markedly increases at menopause.

An adequate intake of calcium during childhood and adolescence is critical to developing maximum bone mass in adulthood. Bone loss cannot be replaced in premenopausal and postmenopausal women with the possible exception of some involved in weight resistance training. One study noted that weight resistance training of postmenopausal women for 1 year increased bone density 4.5% versus a loss of 3.8% in matched controls (82).

Multiple factors are involved in the development of osteoporosis, and some of these risk factors for this complex process are listed in table 6. Genetics seems responsible for up to 70% of the ultimate BMD, whereas the rest seems under the influence of other interrelated factors, such as body weight, calcium intake, estrogen status, exercise, and others. Thin habitus is a factor, especially for those who are thin and also have delayed menarche; they have the lowest BMD in comparison to other adolescents (83).

Table 6. 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's syndrome, Addison's disease, leukemia, celiac disease, Crohn's disease, others)
Others ⁴²

Excessive exercise with weight loss can lead to hypothalamic amenorrhea. Bone mineralization correlates best with body weight. Those with a history of amenorrhea/ irregular menses (oligomenorrhea) due to anorexia nervosa or other factors with hypothalamic amenorrhea have reduced BMD. Because estrogen facilitates calcium into bones, those with chronic estrogen deficiency (as noted in amenorrheic or postmenopausal females) are more susceptible to osteoporosis and stress fractures (72). Increased stress fractures are noted in some athletes (as runners and dancers) **and** those with overt eating disorders.

A major cause of osteoporosis is suboptimal calcium intake in childhood and adolescence. At peak growth velocity, the adolescent girl adds up to 240 mg of calcium per day to her skeleton and the boy adds up to 400 mg. Adolescents who are growing rapidly absorb calcium more rapidly than those who are not growing rapidly. Calcium absorption is promoted by vitamin D, citric acid, and phosphorus while it is inhibited by iron, phytates, and oxalates. The National Research Council's RDA for calcium is listed as 1,200 mg for girls and boys between ages 11 and 24 (84). A 1994 National Institutes of Health consensus statement recommended a higher intake-1,200 to 1,500 mg/day of calcium, especially for girls (85). Pregnant or lactating females need an additional 400 mg of calcium per day. Unfortunately, the average intake of calcium in American adolescents is often below these standards. Improving calcium intake is an important preventive measure to deter the future development of osteoporosis (84,86) (see table 7). Preventing drug use (especially cigarette smoking), physical inactivity, and causes of estrogen deficiency would also be helpful, as adequate calcium intake cannot compensate for these osteoporosis-inducing factors.

Table 7. Calcium-rich foods

300 mg of calcium in 8 oz of skim milk
400 mg per cup of yogurt (plain, non-fat)
250 mg in one cup of tofu (raw, firm)
200 mg per 3 oz of salmon (with bones)
220 mg per 2 oz of canned sardines
180 mg per cup of leafy green vegetables (collard greens)

Adolescents on depo-medroxyprogesterone acetate (DMPA) often develop amenorrhea. Studies in adults and adolescents on DMPA are not clear if there is a risk for subsequent osteoporosis (87). One study reported a 6-month bone loss of 12% in 18-to 21-year-old females on DMPA compared to nonusers (88) and another reported 4.4% lower bone mineralization in teens (mean age of 14-20 years) compared to matched controls in the first study year (89). Further research is needed to understand the long term effect of DMPA on bone mineral density (87). DMPA is not recommended as a contraceptive for adolescents who have or who are at risk for low bone mineral density (87).

Peak bone mass can be measured with various techniques: ultrasonography, quantitative CT, dual-energy X-ray absorptiometry, single-energy X-ray absorptiometry, and others (90-92). The best treatment of osteoporosis is prevention. In young women, this includes maximizing PBM while reducing losses premenstrually. Adequate calcium intake, avoidance of smoking, and weight training are recommended (82).

Amenorrhea

Amenorrhea (primary or secondary), oligomenorrhea, and luteal phase deficiency are well-known phenomena in adolescent athletes (44,93-98). Secondary amenorrhea is noted in 10% to 15% of female athletes and up to 66% in elite athletes (6). As noted with disordered eating patterns, secondary amenorrhea is common in such sports as distance running, ballet, gymnastics, cycling, and others. Menstrual difficulties are described in 5% to 20% of vigorously exercising females (94), 12% of swimmers and cyclists (83), 44% of ballet dancers (99), 50% of female triathletes (100) and 51% of endurance runners (101). There may be a five month delay of menarche for each year of intense prepubertal training (78), whereas decreased training leads to initiation or resumption of menses (97,102). The etiology of amenorrhea in female athletes is multifactorial, complex, and not fully understood (94).

There are many influences on the athlete's menstrual cycle, including age, weight, psychologic stress, nutritional inadequacies, genetic predisposition, percent body fat, amount of exercise, and others

(see chapter 7). The role of leptin in this process is unclear (103-105). As already noted, the athlete who is thin and prepubertal may have an advantage in certain sports such as gymnastics or dance. Body weight alone is not the only factor leading to amenorrhea; weights of amenorrheic runners can be the same as or even more than eumenorrheic athletes (106-108). Body fat is not the only factor, and previous theories that menses could not take place below 17% body fat have not been substantiated (31,107,109). Exercise is also not the only factor and various etiologies should be evaluated when the adolescent athlete presents with delayed menarche and secondary amenorrhea (6).

The amenorrhea noted in athletes is often classified as a hypothalamic amenorrhea and identified with abnormal GnRH and LH pulsivity (see chapter 7) (110,111). A popular theory has been the combination of insufficient caloric intake to keep up with the tremendous needs of the athlete who exercises at a high level to be successful in her chosen sport (6). Their disordered eating patterns may lead to an "energy drain" and eventually hypothalamic amenorrhea. Compounding this may be a history of previous menstrual irregularity, family history of menstrual disorders, chronic illness, and other factors.

A careful assessment is recommended for the adolescent athlete presenting with delayed menarche, secondary amenorrhea, or oligomenorhea (see chapter 7) (15,62,95). A thorough evaluation is recommended if she meets the definition of primary or secondary amenorrhea. A complete history and physical examination are necessary, including a rectal/pelvic examination to establish normal anatomy. Look for hypogonadism, associated congenital anomalies, virilization, short stature, galactorrhea, other endocrinopathies and other features suggestive of chronic illness (6,55,72,112). A hypogonadotropic state may be assessed with a vaginal maturation index (VMJ), serum estradiol level, body weight, and bone mineral densitometry. Laboratory testing may include those listed in table 8. A progesterone challenge can be done to assess the potential of the endometrium to shed when given a progestin (55).

Table 8. Laboratory investigations for amenorrhea in adolescents

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

Management of the athlete who presents with amenorrhea complicated by her intense exercise pattern can be difficult. If she will reduce her exercise intensity and increase her caloric intake, she will have an initiation or resumption of her menstruation if there are no other compounding factors; however, this may be difficult for the committed athlete. The concern remains that delayed menarche, prolonged secondary amenorrhea, and even oligomenorrhea may represent a prolonged hypoestrogenemia, which may contribute to decreased bone density, osteopenia, and eventually osteoporosis (72, 113-123). Females with prolonged amenorrhea and low bone density may never have normal bone density even after the menses are normalized (97). Delayed menses may lead to decreased BMD and then to an increase in stress fractures (as in dancers) (19,97).

Decreased training with an increase in calcium is often recommended for this athlete (124)]. The female athlete with disordered eating or menstrual dysfunction should have a daily intake of 1,200 to 1,500 mg calcium (or a 500 mg calcium tablet 3 times per day) and 400 to 800 IU vitamin D daily (6). Some research suggests that hormonal treatment will preserve some bone loss and that this treatment is beneficial in some cases (94,125,126). Thus, if the bone mineral density

is shown to be low in these athletes, estrogen supplementation with the oral contraceptive (OCP) or conjugated estrogen may be considered (6). Recommendations from the American Academy of Pediatrics for the amenorrheal adolescent who is within three years of menarche are to decrease exercise intensity and improve nutrition intake (including adequate calcium intake) but not to provide hormonal therapy (127). Estrogen supplementation is recommended for amenorrheal adolescents if they are three years postmenarche and older than 16 years of age; if she has had a stress fracture, then a younger age is permitted for initiation of hormonal therapy (127). If hormonal therapy is suggested, the oral contraceptive is often recommended (6,98, 127).

Those with the lowest bone density are those at a low weight who are often sedentary. Weight bearing enhances bone accretion, and gymnasts who are amenorrheal can have normal or increased bone density; vigorous, weight-bearing physical activity ("high mechanical forces") may offset the thin status effect on BMD. This seems to be especially true if the athlete is on oral contraceptives with 50 mg of ethinyl estradiol; pills under 50 mg ethinyl estradiol may not protect against osteoporosis (128). If the normal BMD peak is never reached, there is an increased osteoporosis risk as an adult, especially when postmenopausal (90).

Nevertheless, the immediate and long-term implications of prolonged amenorrhea and possible estrogen deficiency in the otherwise healthy adolescent female athlete are not known at this time (71,98). The use of oral contraceptives (OCPs) for amenorrheal athletes to preserve bone loss is controversial (6). The results of estrogen supplementation are conflicting and sufficient improvement of BMD is not ensured, whether the patient gains or does not gain weight (1,66,129). Not all these adolescent athletes develop low bone density. Some adolescents with a high level of sports achievement can show increased BMD versus controls, even though they have no menses; this has been reported in gymnasts, ice skaters, runners, and tennis players (130). Also, providing the OCP does not solve the underlying issue, and the menstrual dilemma resumes with cessation of the oral contraceptive pill. Also, the OCP has side effects that may be troubling to the female adolescent athlete; these include weight gain, nausea, breast congestion/tenderness, mood lability, and others (see chapter 8) (87). In summary, more research is needed to

understand the underlying mechanisms and the best management for the adolescent athlete with prolonged amenorrhea or oligomenorrhea (98).

Influence of menstrual cycles on athletic performance

There is no clear evidence that menstrual cycles have significant effects on sports performance in athletes (9,131). Completed studies have yielded conflicting data, with some suggesting a negative impact during certain menstrual phases, and others, a positive impact (4,6,9). Regular exercise may lead to less menstrual flow, less dysmenorrhea, and less premenstrual tension syndrome (6). Anecdotal reports may be found of negative effects on severe dysmenorrhea or menstrual bleeding; however, no significant differences in performance, exertion, or lactate levels are noted (96). One study of 86 soccer players reported that more injuries occurred in female athletes with premenstrual symptoms, and no difference in injuries throughout the menstrual cycle were noted if premenstrual symptoms were not present (132).

Influence of oral contraceptives on athletic performance

There is also no clear evidence that oral contraceptives exert a negative impact on sports performance (9,133,134). Studies on runners, for example, show no reduced performance on OCPs (134). The use of OCPs to control menstrual cycles, delay periods during sports events, reduce dysmenorrhea (with or without concomitant use of non-steroidal anti-inflammatory drugs), lessen premenstrual tension syndrome, reduce iron loss by controlled menses, and reduce pregnancy risk may exert a positive effect on the overall sports performance (87, 135, 136). There may be fewer musculoskeletal injuries in female athletes on OCPs if they have dysmenorrhea or premenstrual tension (135,136). The onset of a specific menstrual period can be delayed by 10 days or more by OCP use, if desired (137). Another benefit may be the prevention of bone mineral density loss and prevention of stress fractures (6,138). As stated previously, however, side effects or even the perception of side effects of

OCPs may be bothersome to the female athlete-as weight gain, breast congestion, nausea and others (see chapter 6) (87, 139).

EXERCISE IN PREGNANCY

The literature notes that exercise in general is not harmful to the pregnant female or her fetus (10,36,140-142). Certainly, her sports performance is impaired, due to pregnancy-induced weight gain, breast congestion (secondary to an increase in fat tissue, breast ducts, and fluid retention), abdominal growth, hormonal changes, altered center of gravity, and overall increased energy expenditure. Gains are also noted in maternal blood volume, cardiac output, estrogen, and relaxin levels (with resultant ligamentous laxity). The concern has been raised that blood will be shunted to exercising muscles, possibly reducing placental blood flow and causing fetal hypoxia; there is no clear literature to support this theory. There is also no support for the proposed concept that exercise will raise core temperature and harm the fetus, who is actually well-insulated and not subject to damage in this manner because the fetal core temperature is carefully maintained in a normal range.

The American College of Obstetrics and Gynecology (ACOG) has established guidelines for the pregnant adult who wishes to exercise during her pregnancy (143,144). Contraindications are listed in table 9. The pregnant woman should develop an individualized program with her clinician keeping in mind her usual level of prepregnancy activity. Good bra support will help with pregnancy-induced breast congestion and nipple prominence. She should avoid excessive high-intensity exercise, avoid exercising in extremely hot environments, and not exercise at all if febrile. ACOG guidelines provide warning signs to terminate exercise while pregnant and are listed in table 10.

Table 9. Absolute contraindications to aerobic exercise during pregnancy

Hemodynamically significant heart disease
Restrictive lung disease
Incompetent cervix/cerclage
Multiple gestation at risk for premature labor
Persistent second- or third-trimester bleeding
Placenta previa after 26 weeks of gestation
Premature labor during the current pregnancy
Ruptured membranes
Preeclampsia/pregnancy-induced hypertension

Relative contraindications

Severe anemia
Unevaluated maternal cardiac arrhythmia
Chronic bronchitis
Poorly controlled type 1 diabetes
Extreme morbid obesity
Extreme underweight (BMI less than 12)
History of extremely sedentary lifestyle
Intrauterine growth retardation in current pregnancy
Poorly controlled hypertension
Orthopedic limitations
Poorly controlled seizure disorder
Poorly controlled hyperthyroidism
Heavy smoker

Table 10. Warning signs to terminate exercise while pregnant: ACOG recommendations, 2002

Vaginal bleeding
Dyspnea prior to exertion
Dizziness
Headache
Chest pain
Muscle weakness

Calf pain or swelling (used 10 rule out thrombophlebitis)
Preterm labor
Decreased fetal movement
Amniotic fluid leakage

Prescription exercise plans usually limit the mother to 15 minutes of strenuous exercise and recommend the avoidance of such activities as jumping, since relaxin stretches pelvic ligaments. Pregnancy is not a time to start a new sports activity or even a new type of exercise program. It is recommended that exercise in the supine position be avoided until after the first trimester. Also, the pregnant female should not engage in such activities as weight lifting, scuba diving or other water sports, horseback riding, or strenuous anaerobic exercises. It is advisable that she not be participating in competitive events, especially if involved in a contact sport or if the risk of injury is high (141). Swimming may be the best exercise for her, but walking and biking are also safe.

Exercising with the upper body is fine; however, any activity that subjects the trunk to mechanical stress is to be avoided (145). Exercise for the pregnant female with diabetes is tolerated, if her metabolic status is carefully monitored (145). A common sense approach with the recommended guidelines in mind will allow the pregnant females to exercise, if she wishes, and not increase her risk of adverse pregnancy outcome (141,143,144). Postpartum exercising may be initiated in about 4 to 6 weeks after a vaginal delivery and 6 to 8 weeks after a cesarean section (36). Lactation is not a contraindication to exercise (146).

IRON DEFICIENCY

Iron deficiency is the most common cause of anemia in adolescents. Surveys in children and adolescents reflect an iron deficiency prevalence up to 24% and overt anemia in 10% of high school students (147,148). It is a common disorder in the athlete, especially in females (6, 149, 150). The female has 6% fewer red blood cells and 8% to 19% lower hemoglobin levels, in contrast to the male; she tends to have lower iron stores, increased iron levels, and increased iron deficiency anemia than

the male (32). Iron deficiency is probably not more common in athletes in contrast to the general population, except for long distance runners (104,151).

Causes of iron deficiency include low dietary intake, menstruation, loss of iron in urine and sweat during exercise, gastrointestinal bleeding, intravascular hemolysis (which can be precipitated by exercise), and other factors (147,152). Pseudoanemia or sports anemia is a physiologic response to vigorous **and** extended exercise in which plasma volume expands up to 20%; if the red blood cell **mass** is normal, no treatment is **needed**, as this expansion will **be** corrected once the exercise is decreased (104).

Iron deficiency develops in three stages. First, iron stores decrease, and a low serum ferritin level is noted. Then, the serum iron drops along with a parallel rise in total iron-binding capacity. Finally, overt microcytic, hypochromic anemia occurs. Most youth with iron deficiency, however, have mild asymptomatic degrees of anemia. Table 11 lists laboratory characteristics of iron deficiency in female adolescents (147). Even mild iron deficiency anemia can decrease sports performance.

Table 11. Laboratory parameters of iron deficiency anemia in adolescent females

Hematocrit	<35%; 12 year old females
Hemoglobin	<36%; 12-18 year old females <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	<16% (normal, 35-40%)
[iron/total iron binding capacity (Fe/TIBC) ratio]	
TIBC (total iron binding capacity)	350-500 mg/dl (normal, 250- 380 mg/dl)
FEP (free erythrocyte protoporphyrin)	≥150-200 mg/dl RBCs (normal, 54 +/-20)

Although there is no evidence that nonanemic iron deficiency (with normal hemoglobin/hematocrit and low serum ferritin levels) impairs sports performance, athletes with low ferritin levels and low-to-normal hemoglobin ranges may note improved performance with iron supplementation (153). Routine screening of these athletes is not recommended, except for those at high risk for iron deficiency anemia. High risk factors include having a history of anemia, endurance running, some nutritional diets (as vegetarianism), and others (104). If anemia is found, a careful search for underlying causes is important (147,149).

Management of iron deficiency involves education of the adolescent about eating iron-rich foods: meats, fish, fortified cereals, and breads. Iron is also available in eggs and various green vegetables (such as spinach), although the formation of phytate or phosphate salts may limit its absorption. Iron may be given orally in various forms; ferrous sulfate is the least expensive and most readily available. Although recommended doses vary, an adolescent who needs replacement doses can be prescribed 3 to 6 mg/kg/d of elemental iron in two or three divided doses (up to 60 to 80 mg three times a day); one 300 mg tablet of hydrated ferrous sulfate contains 60 mg of elemental iron. A rise in hematocrit and hemoglobin levels often occurs in 1 to 3 weeks, whereas repletion of body iron stores (such as normal ferritin levels) may take up to several months. Management of side effects to iron therapy, measurement of relevant blood studies, and use of parenteral iron therapy are well-described in the literature (147).

STRESS URINARY INCONTINENCE

The involuntary loss of urine during exercise, or stress urinary incontinence, is underreported in female athletes (1,104,154). It can occur during various physical activities but is especially noted in exercise that involves chronic, repetitive motion and involves high impact landings, jumping, and running (154). Thus, it is encountered in female athletes involved with gymnastics, track and field, basketball, and other "impact" sports; it is noted with less frequency in tennis players, skiers, skaters, and those who jog (155). Stress urinary incontinence has been identified in 28% of nulliparous female athletes with a mean age of 20 years (154).

Some exercise can lead to sufficient increase in intraabdominal pressure, which causes changes in the urethral sphincteric unit, resulting in urinary incontinence. Table 12 lists risk factors for this phenomenon according to a 1989 NIH consensus panel (156). It is worthwhile to ask female athletes about this possibility, especially if they are in a high-risk situation. An evaluation includes a careful history and examination. Pelvic examination can assess the integrity of pelvic floor anatomy, and further investigation, if warranted, can look for an anatomic defects at the posterior urethrovesical angle (1).

Table 12 Risk factors for exercise-induced stress urinary incontinence ¹¹⁵

Increasing age
Gender (female)
Increased parity
Heavy physical activity
High-impact sports
Hypoestrogenic amenorrhea
Obesity

Management includes education of female athletes about this relatively common and usually benign situation. This athlete should avoid excessive fluid intake before exercise, although dehydration must

be avoided as well. Sanitary napkins worn during the sporting event are helpful, as may be training in strengthening the pelvic floor muscles (the levator ani muscle). In general, further treatment is not necessary for the healthy athlete who notes intermittent incontinence during vigorous exercise. If further management becomes necessary, behavioral therapy involving Kegel exercises, with or without biofeedback, may be tried for several weeks. Vaginal cones and electrical stimulation have also been suggested as well for selected situations.

Pharmacologic therapy includes imipramine and alpha-mimetics (such as pseudoephedrine hydrochloride). Phenylpropanolamine hydrochloride has been used in the past, but it has now been banned by the US Food and Drug Administration due to increased association with cerebrovascular accidents in women younger than 50 years of age. The use of anticholinergic agents is discouraged because of potential impairment of the sweating mechanism: this complication may lead to exercise-induced heat disorders. The use of the oral contraceptives for amenorrhea or oligomenorrhea secondary to the vigorous exercising may also be beneficial (see chapters 7 and 8).

INJURIES

Injuries to female adolescent athletes tend to be the same as those noted with male athletes, except for an increase in noncontact anterior cruciate ligament (ACL) injuries and patellofemoral (PF) disorders (4,6,7,9,36,157-160). In general, injuries and their consequences are related to the specific sport, the training of an individual athlete, and availability of proper equipment as well as comprehensive management of injuries. Females athletes, like all athletes, need appropriate conditioning and training, both before the season begins and during the sports season (4,161). Common injuries in gymnastics involve the knee and lumbar spine, whereas extremity injuries are most common in basketball and volleyball (35, 162). As noted with males, up to 50% of injuries are overuse injuries (due to microtrauma) (35). Stress fractures occur with increased frequency in amenorrheal athletes and others with low bone mineral density.

More research is needed to identify the multiple factors behind the increase in noncontact ACL injuries and PF disorders in girls. The stated relationship of patellofemoral disorders with an increased Q angle has been controversial (4,157). Investigators have looked at a number of issues in this regard, such as girls having a wider pelvis, less developed muscles, increased flexibility, less developed vastus medialis obliquus (VMO), narrower femoral notch, greater degree of genu valgum, and greater external tibial torsion (36).

Some studies suggest that female athletes have unique neuromuscular reflex patterns, which lead to increased ACL injuries and increased PF tracking difficulties (6). Further study of the role of estrogen and progesterone receptors in knee synovial linings may lead to more understanding of the higher rate of ACL injuries in females (163). One study has identified increased ACL injury in the ovulatory phase of menstruation versus other menstrual phases (164). Foot problems (such as bunions, calluses, corns, and metatarsalgia) may be particularly related to the female athlete shoe being based on male foot anatomy (1,4,6). More research is clearly needed in these various areas.

SUMMARY

Female athletes have made significant progress in sport participation over past several decades. Although there are some physiological differences between the genders, overall female athletes compare well in athletic participation and performance compared with male athletes. Exercise and sports-related conditions and injuries of breasts are an important aspect of management of female athletes. The female athlete triad of disordered eating behaviors, osteopenia/ osteoporosis, and menstrual irregularities/ amenorrhea remains a concern in special population of female athletes. This is an important area where pediatricians and primary care physicians can make a difference by early recognition and prevention. Overall, the musculoskeletal injury patterns in female athletes are similar to those seen in male athletes, with few exceptions such as injuries of the anterior cruciate ligament and patella-femoral disorders.

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REFERENCES

- [1] Nattiv A, Arendt EA, Hecht SS. The female athlete. In: Garrott WE, Kirkendall DT, Squire DL, ed. *Principles and practice of primary care sport medicine*, Philadelphia, PA: Lippincott Williams Wilkins 2001:93-113.
- [2] Pfister G. *Women and the Olympic games: 1900-1997*. In: Drinkwater BA, ed. *Women in sport*. Oxford: Blackwell, 2000:3-19.
- [3] Agostini R, ed. *Medical and orthopedic issues of active athletic women*. Philadelphia, PA: Hanley Belfus, 1994:1-5.
- [4] Beim G, Stone DA. Issues in the female athlete. [*Orthop Clin North Am*] 1994;26:443-51.
- [5] Lopiano DA. Modern history of women in sports: twenty-five years of Title IX. [*Clin Sports Med*] 2000;19:1-9.
- [6] Yurko-Griffin L, Harris SS. Female athletes. In: Sullivan A, Anderson SJ, eds. *Care of the young athlete*. Chicago, IL: [*Am Acad Orthopedic Surg, Am Acad Pediatrics*], 2000: 137-48.
- [7] Callahan LR. Title evaluation of the female athlete: progress and problems. [*Pediatr Ann*] 2000;29:153-9.
- [8] Kane MJ. Media coverage of post IX female athlete: a feminist analysis of sport gender and power. [*Duke J Gender Law Policy*] 1996;3:95-127.
- [9] Frankovich RJ, Lebrun CM. Menstrual cycle, contraception and performance. [*Clin Sports Med*] 2000;19:1-6.

- [10] Greydanus DE, Tsitsika A. Special considerations for the female athlete. In: Patel DR, Greydanus DE, Baker RJ, eds. *Pediatric practice: Sports medicine*. New York: McGraw Hill 2008.
- [11] Patel DR, Greydanus DE, Pratt HD. Youth sports: more than sprains and strains. [*Contemp Pediatr*] 2001;18:45-74.
- [12] Pratt HD, Patel DR, Greydaous DE. Sports and the neurodevelopment of the child and adolescent. In: DeLee JC, Drez DD, Miller MD, eds. *Orthopedics sports medicine*. Philadelphia, PA: WB Saunders, 2003:58-70.
- [13] Freedson PS, Matthews CE, Nasca PC. Physical activity and risk factors for breast cancer. In: Drinkwater BA, ed. *Women in sport* Oxford: Blackwell, 2000:250-64.
- [14] Greydanus DE, Pratt HD. Psychosocial considerations for the adolescent athlete: lessons learned from the United States experience. [*Asian J Paediatr Pract*] 2000;3: 19-29.
- [15] Luckstead EF, Greydanus DE. *Medical care of the adolescent athlete*. Los Angeles, CA: [Pract Manage Corp], 1993.
- [16] Hasbrook CA. Gender: its relevance to sports medicine. In: Garrett Jr WE, Kirkendall DT, Squire DL, eds. *Principles and practice of primary care sports medicine*. Philadelphia, PA: Lippincott Williams Wilkins. 2001:215-20.
- [17] Greydanus DE, Patel DR. Sports doping in the adolescent athlete. [*Asian J Paediatr Pract*] 2000;4:9-14.
- [18] Pratt HD, Greydanus DE. Adolescent violence: concepts for a new millennium. [*Adolesc Med*] 2000; 11: 103-26.
- [19] Sundgot-Borgen J. Eating disorders in female athletes. [*Sports Med*] 1994;17:188.
- [20] Sundgot-Borgen J. Eating disorders. In: Drinkwater BA. ed. *Women in sport*. Oxford: Blackwell, 2000:364-76.
- [21] Brackenridge. CH. Ethical problems in women's sport. coaching focus. [*Nat Coach Foundation*] 1987;6:5-7.
- [22] Brackenridge CH. He owned me basically: women's experiences of sexual abuse in sport. [*Int Rev Sociol Sport*] 1997;32:15-130.
- [23] Brackenridge C. Harassment, sexual abuse and safety of the female athlete. [*Clin Sport Med*] 2000;19:1-9.
- [24] Brackenridge C. Sexual harassment and abuse. In: Drinkwater BA, ed. *Women in sport*. Oxford; Blackwell, 2000:342-50.
- [25] Caron SL, Halteman WA, Stacy C. Athletes and rape: Is there a connection? [*Percept Mot Skills*] 1997;85: 1379-93.
- [26] Crosset T, Benedict J, McDonald M. Male student-athletes reported for sexual assault: a survey of campus police departments and judicial affairs offices. [*J Sport Social Issues*] 1995;5: 126-40.
- [27] Ogilvie BC, Tofler IR, Conroy DE, et al. Comprehending role conflicts in the coaching of children, adolescents and young adults. [*Child Adolesc Psychiatr Clin North Am*] 1998;7:879-90.

- [28] Parrot A, Cummings N, Marchell TC, et al. A rape awareness and prevention model for male athletes. [J Am Coll Health] 1994;42: 179-84.
- [29] Small L, Micheli L. Strength development in children. [J Pediatr Orthoped] 1986;6: 143-6.
- [30] Jewett A, Barren KR, Seefeldt V. Physical education for the elementary school child. [Phys Sportsmed] 1984;12:99-124.
- [31] Frisch RE, McArthur JW. Menstrual cycles: fatness as a determinant of minimum weight for height necessary for their maintenance or onset [Science] 1974;185:949-50.
- [32] Bar-Or O. Physiologic responses to the exercise of the healthy child athlete: pediatric sports medicine for the practitioner: From physiologic principles to clinical applications. New York: Springer Verlag. 1983:1-65.
- [33] Beuman G, Malina RM. Growth and physical performance relative to the timing of the adolescent sport. [Exerc Sport Sci Rev] 1988;16:503.
- [34] Greydanus DE, Patel DR, Luckstead EF, eds. Office orthopedics and sports medicine symposium. [Adolesc Med] 1998;9:425-626.
- [35] Patel DR, Baker RJ. Musculoskeletal injuries in sports. [Prim Care]; 2006;33(2);545.
- [36] Ireland ML. Special concerns of the female athlete. In: Fu F, Stone R, eds. Sports injuries: mechanisms, prevention and treatment. 2nd edition. Baltimore, MD: Williams Wilkins, 2000:156-87.
- [37] Komi PV, ed. Strength and power in sport. Oxford: Blackwell, 1992:404.
- [38] Wilmore JH. The application of science to sport: physiologic profiles of male and female athletes. [Can J Appl Sport Sci] 1979;4: 103-15.
- [39] Malina RM. Effects of physical activities on growth in stature and adolescent growth spurt. [Med Sci Sport Exerc] 1994;26:759.
- [40] Malina RM. Physical growth and biological maturation of young athletes. [Exerc Sport Sci Rev] 1994;22:389.
- [41] Atwater AE. Biomechanics of the female athlete. In: Puhl J, Brown CH, Voy RO, eds. Sports science perspectives for women. Champaign, IL: Human Kinetics, 1985:1-12.
- [42] Hindle WH. The breast and exercise. In: Hale W, ed. Caring for the exercising woman. New York: Elsevier, 1991 :83-92.
- [43] Mayhew JL, Gross PM. Body composition changes in young women with high resistance weight training. [Res Q] 1974;45:433-40.
- [44] Shangold MM. Gynecologic concerns in the woman athlete. [Clin Sports Med] 1984;3:869-79.
- [45] Friedenreich CM, Rohan TE. A review of physical activity and breast cancer. [Epidemiology] 1995;6:311-7.
- [46] 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] 1985;52:885-91.

- [47] Hoffman-Goetz L, Husted J. Exercise and breast cancer: review and critical analysis of the Literature. [Can J Appl Physiol] 1994;19:237-52.
- [48] Thune I, Brenn T, Lund E, et al. Physical activity and the risk of breast cancer. [N Engl J Med] 1997;336: 1269-75.
- [49] Greydanus DE, Patel DR, Baxter TL. The breast and sports: issues for the clinician. [Adolesc Med] 1998;9:533-50.
- [50] Haycock CE. How I manage breast problems in athletes. [Phys Sportsmed] 1987;15:89-95.
- [51] Otis CL. Women and sports: breast and nipple injuries. [Sports Med Dig] 1988;10:7-9.
- [52] Rubin CJ. Sports injuries in the female athlete. (NJ Med] 1991;88:643-5.
- [53] Powell B. Bicyclist's nipples. [JAMA] 1983;249:2457.
- [54] Nequin ND. More on jogger's ailments [N Engl J Med] 1978;298:405-6.
- [55] Greydanus DE. Breast and gynecologic disorders In: Hofmann AD, Greydanus DE. eds. Adolescent medicine, 3rd ed. Norwalk, CT: Appleton Lange. 1997:520-34,
- [56] Haycock CE, Gillette JV. Susceptibility of women athletes to injury: myths vs reality. [JAMA]1976:236; 163.
- [57] Wells CL: Women, sport and performance: a physiologic perspective. Champaign, IL: [Human Kinetic]. 1991:1-367.
- [58] Greydanus DE, Parks DS, Farrell EG. Breast disorders in children and adolescents. [Pediatr Clin North Am] 1989;36:601-38.
- [59] Dellon AL. Blunt chest trauma: evaluation of the augmented breast. [J Trauma] 1980;20:982.
- [60] Gehlsen S, Stoner LJ. The female breast in sports and exercise. [Med Sport Science] 1987; 24:13-22.
- [61] American Society for Testing and Materials {ASTM}: Standard classification of brassieres. Philadelphia, PA: Am Soc Testing Materials, [Yearbook Standard] F753-82, 1982.
- [62] Greydanus DE. Gynecologic problems and sexually transmitted diseases. In: Greydanus DE. ed. Caring for your adolescent-ages 12 to 18. Elk Grove Village, IL: [Am Acad Pediatr], 1997:267-76.
- [63] Gehlsen G, Albohm M. Evaluation of sports bras. [Phys Sportsmed] 1980;8:88-97.
- [64] Lorentzen D, Lawson L. Selected sports bras: a biomechanical analysis of breast motion while jogging. [Phys Sportsmed] 1987;15:128.
- [65] Berger-Dir mound J. Sports bras: everything you need to know from A to D. [Women's Sports Fitness] 1986;8:31-49.
- [66] Cummins C. Sports bra round-up. [Women's Sports Fitness] 1989;4:66.
- [67] Lee J. Sport support [Women's Sports Fitness] 1995; 17:72-3.
- [68] Lorentzen D, Lawson L. Best health-support bets for active breasts: researchers rate 8 sports bras. [Self Health Watch] 1987;5:1.
- [69] Sports bras, Women's Sports Fitness 1995;17:72.

- [70] 70.. American Academy of Pediatrics. Medical concerns in the female athlete. [Pediatrics] 2000; 06:610-3.
- [71] Yeager KK, Agostini R, Nattiv A. et al. The female athlete triad: disordered eating, amenorrhea, osteoporosis. [Med Sci Sports Exerc] 1993;25:775-7.
- [72] Nattiv A, Loucks AB, Manore MM, Sanborn CF, Sundgot-Borgen J, Warren M. American College of Sports Medicine Position Stand: The female athlete triad. <http://www.acsm-msse.org>
- [73] Sanborn CF, Horea M. Siemers BJ, et al. Disordered eating and the female athlete triad. [Clin Sports Med] 2000;19:1-11.
- [74] Dummer GM. Rosen LW, Hensner WW, et al. Pathogenic weight control behaviors of young competitive swimmers. [Phys Sportsmed] 1987;5:22-7.
- [75] Nattiv A, Lynch L. The female athlete triad. [Phys Sportsmed] 1994;22:60-8.
- [76] Rosen LW, Hough DO. Pathogenic weight-control behaviors of female college gymnasts. [Phys Sportsmed] 1988; 16:141-6.
- [77] Wichmann S, Martin DR. Eating disorders in athletes. [Phys Sportsmed] 1993;21: 126-35.
- [78] Frisch RE, Wyshak G, Vincent L. Delayed menarche and amenorrhea in ballet dancers. [N Engl J Med] 1980;303:17.
- [79] Katzman DK, Zipursky RB. Adolescents with anorexia nervosa: the impact on the disorder on bones and brains. [Ann N Y Acad Sci] 1997;81:127-37.
- [80] Klibanski A, Biller BMK, Schonfield DA, et al. The effects of estrogen administration on trabecular bone loss in young women with anorexia nervosa. [J Clin Endocrinol Metabol] 1995;80:898-904.
- [81] Theintz G, Buchs B, Rizzoli R, et al. Longitudinal monitoring of bone mass accumulation in healthy adolescents: evidence for 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.
- [82] Nelson MB, Fiatarone MA, Morganti CM, et al. Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures. [JAMA] 1994;272:1909.
- [83] Sanborn CF, Martin BJ, Wagner WW. Is athletic amenorrhea specific to runners? [Am J Obstet Gynecol] 1982;143:859.
- [84] Hofmann AD. Adolescent nutrition. In: Hofmann AD, Greydanus DE, eds. Adolescent medicine, 3rd ed. Stamford, CT: Appleton Lodge, 1997:644-6.
- [85] National Institutes of Health. Optimal calcium intake. [NIH Consensus Statement] 1994;12:a-31.
- [86] Teegarden D, Weaver DM. Calcium supplementation increases bone density in adolescent girls. [Nutr Rev] 1994;52: 171.
- [87] Greydanus DE, Patel DR, Rimsza ME. Contraception in the adolescent: an update. [Pediatrics] 2001; 107:562-73.
- [88] Scholes D, LaCroix AZ, Ichikawa L, et al. Depo-medroxyprogesterone acetate exposure and bone mineral density in young women. [J Bone Min Res] 1996; 11(Suppl 1):5448.

- [89] Cromer BA, Blair JM, Mahan JD, et al. A prospective comparison of bone density in adolescent girls receiving depot medroxyprogesterone acetate (Depo-Provera), levonorgestrel (Norplant), or oral contraceptives. [J Pediatr] 1996;129:671-6.
- [90] Eastell R. Treatment of postmenopausal osteoporosis. [N Engl J Med] 1998;338:736-46.
- [91] Johnston CC, Slemenda CW, Melton LJ. Clinical use of bone densitometry. [N Engl J Med] 1991;324:1105-9.
- [92] Notelovitz M. Osteoporosis: screening, prevention, and management. [Fertil Steril] 1993;59:707-25.
- [93] Bullen BA, Skrinar GS, Beitzens IZ, et al. Induction of menstrual disorders by strenuous exercise in untrained women. [N Engl J Med] 1985;312: 1349.
- [94] Chen EC, Brzyski RG. Exercise and reproductive dysfunction. [Fertil Steril] 1998;71:1-6.
- [95] Constantini NW. Clinical consequences of athletic amenorrhea. Sports Med 1994;17:213-23.
- [96] De Souza MJ, Metzger DA. Reproductive dysfunction in amenorrheic athletes and anorexic patients: a review. Med Sci Sports Exerc 1991;23:995-1007.
- [97] Drinkwater BL, Breunner B, Chesnut CH. Menstrual history as a determinant of current bone density in young athletes. [JAMA] 1990;263:545-8.
- [98] Marshall LA. Amenorrhoea. In: Drinkwater BA, ed. Women in sport. Oxford: Blackwell, 2000:377-90.
- [99] Calabrese LH, Kirendall DT, Floyd M et al: Menstrual abnormalities, nutritional patterns and body composition in female classic ballet dancers. [Phys Sportsmed] 1983; 11:86-98.
- [100] Rutherford OM. Spine and total body bone mineral density in amenorrheic endurance athletes. [J Appl Phys] 1993;74:2904-8.
- [101] Dale E, Gerlach D, Martin D, et al. Physical fitness profiles and reproductive physiology of the female distance runner. [Phys Sportsmed] 1979;7:83-98.
- [102] Prior JC, Vigna YM, Schechter MI, et al. Spinal bone loss and ovulatory disturbances. [N Engl J Med] 1990;323:1221-7.
- [103] Laughlin GA, Yen SSC. Hypoleptinemia in women athletes: absence of a diurnal rhythm with amenorrhea. [J Clin Endocrinol Metab] 1997;82:318-21.
- [104] Nattiv A. Track and field. In: Drinkwater BA, editor. Women in sport. Oxford: Blackwell, 2000:470-85.
- [105] Thong FSL, 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.
- [106] Feicht CB, Johnson JS, Martin BJ, et al. Secondary amenorrhea in athletes. [Lancet] 1978;2: 1145-46.
- [107] Fruth SJ, Worrell TW. Factors associated with menstrual irregularities and decreased bone mineral density in female athletes. [JOSPT] 1995;22:26-38.

- [108] Nelson ME, Fisher EC, Catsos D, et al. Diet and bone status in amenorrheic runners. [Am J Clin Nutr] 1986;43:91 Q.6.
- [109] Warren MP. The effects of exercise on pubertal progression and reproductive function in girls. [J Clin Endocrinol Metabol] 1980;51:1150.
- [110] Loucks AB, Vaitukaitis J, Cameron JL. The reproductive system and exercise in women. [Med Sci Sports Exerc] 1992;24(Suppl):S288-93.
- [111] Marshall LA. Clinical evaluation of amenorrhea in active and athletic women. [Clin Sport Med] 1994;13:371-89.
- [112] Hobart JA, Smucker DR. The female athlete triad. [Am Fam Phys] 2000;61 :357-64.
- [113] Dugowson CE, Drinkwater BL, Clark JM. Nontraumatic femur fracture in an oligomenorrheic athlete. [Med Sci Sports Exerc] 1991;23:1323-5.
- [114] 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.
- [115] Lane JM, Riley EH, Wirganowicz PC. Osteoporosis: diagnosis and treatment [J Bone Joint Surg Am] 1996;78A:618.
- [116] Linnell ST, Stager TM, Blue PW, et al. Bone mineral content and menstrual regularity in female runners. [Med Sci Sports Exec] 1984;16:343-8.
- [117] Lloyd T, Meyers C, Buchanan IR, et al. Collegiate women athletes with irregular menses during adolescence have decreased bone density. [Obstet Gynecol] 1998;72:639-42.
- [118] Rosenthal DT, Mayo-Smith W, Hayes CW, et al. Age and bone mass in premenopausal women [J Bone Minec Res] 1989;4:533-8.
- [119] Snyder AC, Wenderoth MP, Johnston CC, et al. Bone mineral content of elite lightweight amenorrheic women. [Hum Biol] 1986;58:863-9.
- [120] Warren MP, Brooks-Gunn J, Hamilton LH, et al. Scoliosis and fractures in young ballet dancers: relation to delayed menarche and secondary amenorrhea. N Engl J Med 1986;314: 1348-53.
- [121] 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 Metabol] 1991 ;72:847-53.
- [122] Wolman RL, Clark P, McNally E, et al. Menstrual state and exercise as determinants of spinal trabecular bone density in female athletes. [BMJ]1990;301:516-8.
- [123] 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] J992; 17: 415-23.
- [124] Dueck A, Matt KS, Manore M, et al. Treatment of athletic amenorrhea with a diet and training intervention program. [Int J Sport Nutr] 1996;6:24-40
- [125] Gibson J. Osteoporosis. In: Drinkwater BA, ed. Women in sport. Oxford: Blackwell, 2000:391-406.

- [126] Haenggi W, Casez J-P, Birkhaeuser MH, et al. Bone mineral density in young women with long-standing amenorrhoea: limited effect of hormone replacement therapy with ethinyl oestradiol and desorgestrel. [*Osteoporos Int*] 1994;4:99-103.
- [127] American Academy of Pediatrics. Amenorrhea in adolescent athletes. [*Pediatrics*] 1989;84:394-5.
- [128] 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.
- [129] Hergenroder AC, Smith BO, Shypailo R, et al. Bone mineral changes in young women with hypothalamic amenorrhea treated with oral contraceptives, medroxy progesterone, or placebo over 12 months. [*Am J Obstet Gynecol*] 1997;176:1017-25.
- [130] Etherington J. Bone mineral density study of adult elite athletes. *J Bone Miner Res* 1996;11:1333.
- [131] Posthuma BW, Bass JJ, Bull SB, et al. Deleterious changes in functional ability in women with premenstrual syndrome. [*Am J Obstet Gynecol*] 1987; 156:275-8.
- [132] Molter-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.
- [133] Bryner RW, Toffle RC, Ullrich IH, et al. Effect of low dose oral contraceptive on exercise performance. [*Br J Sports Med*] 1996;30:36-40.
- [134] Labrun CM. Effects of the menstrual cycle and oral contraceptives on sports performance. In: Drinkwater BA, ed. *Women in sport* Oxford: Blackwell. 2000:37-61.
- [135] Burkman Jr RT. Noncontraceptive effects of hormonal contraceptives: bone mass, sexually transmitted disease and pelvic inflammatory disease, cardiovascular disease, menstrual function and future fertility. *Am J Obstet Gynecol* 1994;170:1569-75.
- [136] Moller-Nielson J, Hammar M. Sports Injuries and oral contraceptive use: is there a relationship? *Sports Med* 1991;12:152,60.
- [137] Sulak PJ, Cressman BE, Waldrop E, et al. Extending the duration of active oral contraceptives pills to manage hormone withdrawal symptoms. [*Obstet Gynecol*] 1997;89: 179-83.
- [138] Hartard M, Bottermann P, Bartenstien P, et al. Effects on bone mineral density of low-dosed oral contraceptives compared to management combined with physical activity. [*Contraception*] 1997; 55:87-90.
- [139] Reubinoff B. Fear of oral contraceptive side effects. [*Fertil Steril*] 1995;63:5]6,21.
- [140] Hall DC, Kaufmann DA. Effects of aerobic and strength conditioning on pregnancy outcomes. [*Am J Obstet Gynecol*] 1987;157:1199-203.
- [141] Mottola MF, Wolfe LA. The pregnant athlete. In: Drinkwater BA, ed. *Women in sport*. Oxford: Blackwell, 2000:194-207.

- [142] White J. Exercising for two: what's safe for the active pregnant woman. [Phys Sportsmed] 1992;20: 179-86.
- [143] Gavard JA, Artal R. Effects of exercise on pregnancy outcome. [Clin Obstet Gynecol] 2008;51:20:467-80.
- [144] American College of Obstetrics and Gynecology. Guidelines for exercise during pregnancy and postpartum. [Am Coll Obstet] Gynecol Tech Bull, 2002.
- [145] Campaigne BN. Diabetes and sport In: Drinkwater BA, ed. Women in sport. Oxford: Blackwell, 2000:265-79.
- [146] Prentice A. Should lactating women exercise? [Nutr Rev] 1994;52:358-60.
- [147] Greydanus DE, Mattano Jr LA, Wigginton JM. Hematology and oncology: iron deficiency anemia. In: Hofmann AD, Greydanus DE, eds. Adolescent medicine, 3rd ed. Stamford, CT: Appleton Lange, 1997:348-51.
- [148] Looker A, Dallman P, Calld M, et al. Prevalence of iron deficiency in the United States. [JAMA] 1997;27:973-6.
- [149] Harris SS. Exercise-related anemia. In: Drinkwater BA, ed. Women in sport. Oxford: Blackwell. 2000:311-20.
- [150] Risser WL, Risser JM. Iron deficiency in adolescent and young adults. [Phys Sportsmed] 1990;18:87-101.
- [151] 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.
- [152] Nickerson HJ, Holubets M, Weller BR. Causes of iron deficiency in adolescent athletes. [J Pediatr] 1989;114:657-9.
- [153] Garza D, Shrier I, Kohl H 3rd, et al. The clinical value of serum ferritin tests in endurance athletes. [Clin J Sport Med] 1997;7:46-53.
- [154] Nygaard I, Delancey J, Arnsdorf L, et al. Exercise and incontinence. [Obstet Gynecol] 1990; 75:848-51.
- [155] Bourcier AP, Juras JC. Nonsurgical therapy for stress incontinence. [Urol Clin North Am] 1995; 22:613-27.
- [156] NIH Consensus Development Panel. Urinary incontinence in adults. [JAMA] 1989;261 :2685-90.
- [157] Arendt EA. Orthopaedic issues for active athletic women. [Clin Sports Med] 1994; 13:483-503.
- [158] Arendt E, Griffin L. Musculoskeletal injuries In: Drinkwater BA, editor. Women in sport. Oxford: Blackwell. 2000:208-40.
- [159] Beachy B, Akau CK, Martinson M, et al. High school sports injuries: a longitudinal study at Punahou School 1988-1996. Am J Sports Med 1997;75:675-81.
- [160] DeLoes M. Epidemiology of sports injuries in the Swiss organization Youth and Sports 1987-1989. [Int J Sports Med] 1995;16:134-8.
- [161] DuRant RH, Linder CW, Sandes Jr. ME, et al. Adolescent females' readiness to participate in sports. [J Adolesc Health Care] 1988;9:310.
- [162] Sands WA, Shultz BB, Newman AP. Women's gymnastics injuries: a five year study. [Am J Sports Med] 1993;21:271-6.

-
- [163] Liu SH. Al-Shaikh R, Panossian Y, et al. Primary immunolocalization of estrogen and progesterone target cells in the human anterior cruciate ligament. [J Orthop Res] 1996;14:526-33.
- [164] 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.