AMERICAN ACADEMY OF PEDIATRICS

Committee on Sports Medicine and Fitness

Medical Concerns in the Female Athlete

ABSTRACT. Female children and adolescents who participate regularly in sports may develop certain medical conditions, including disordered eating, menstrual dysfunction, and decreased bone mineral density. The pediatrician can play an important role in monitoring the health of young female athletes. This revised policy statement provides updated and expanded information for pediatricians on these health concerns as well as recommendations for evaluation, treatment, and ongoing assessments of female athletes.

ABBREVIATIONS. BMD, bone mineral density; LH, luteinizing hormone.

Exercise is good for female children and adolescents. Special medical concerns should be considered, however, when caring for young female athletes. Athletes can develop abnormal eating patterns (termed disordered eating), which can be associated with menstrual dysfunction (amenorrhea or oligomenorrhea) and subsequent decreased bone mineral density (BMD), or osteoporosis. These 3 conditions—disordered eating, amenorrhea, and osteoporosis—often occur together and have been termed the female athlete triad.[1] Although these conditions may also be seen in the nonathlete, this statement will concentrate on the physically active and athletic female.

DISORDERED EATING

Some physically active females, particularly adolescents, may develop an energy deficit when the energy (calories) they expend exceeds their energy (calorie) intake.[2] This deficit may be unintentional, resulting from inadequate replenishment of the caloric (energy) demands of training, or may be intentional—a conscious attempt to lose weight or body fat in the interest of improved appearance or athletic performance. These athletes often restrict food intake but may develop other disordered eating behaviors, such as binge eating and/or purging by vomiting or use of laxatives, diuretics, and diet pills. Compulsive exercise, defined as excessive exercise in addition to a normal training regimen, is another form of "purging," or energy expenditure often overlooked in athletes. The spectrum of disordered eating behaviors ranges from mild—slight restriction of food intake or occasional binge eating and purging—to severe—significant restriction of food intake, as in anorexia nervosa, or regular binge eating and purging, as in bulimia nervosa.[3] Disordered eating may result in adverse health consequences, with the risk of morbidity and mortality increasing as the severity of the behavior increases.

Disordered eating behavior has been reported in young female athletes and dancers.[4–6] One study of young elite swimmers revealed that 60.5% of average-weight girls and 17.9% of underweight girls were trying to lose weight. Most of the girls were trying to lose weight by decreasing their food intake[7]; however, 12.7% were vomiting, 2.5% were using laxatives, and 1.5% were using diuretics.[7] Disordered eating can be seen in athletes participating in all sports. Sports that may place athletes at higher risk for the development of these behaviors include those in which leanness is emphasized (eg, gymnastics, ballet dancing, diving, and figure skating) or perceived to optimize performance (eg, long-distance running and cross-country skiing) and those that use weight classification (eg, martial arts and rowing).[3] A variety of factors may contribute to the development of disordered eating patterns in the young athlete, including pressure to optimize performance or meet inappropriate weight or body fat goals, social factors (eg, idealization of thinness in Western cultures), psychological factors (eg, poor coping skills, unhealthy family dynamics, and low self-esteem), and personality traits (eg, perfectionism, compulsiveness, and high achievement expectations).[3]

Disordered eating behaviors may impair athletic performance and increase risk of injury. Decreased energy (caloric) intake and fluid and electrolyte imbalance can result in decreased endurance, strength, reaction time, speed, and ability to concentrate. Because the body initially adapts to these changes, a decrease in performance may not be seen for some time, and athletes may falsely believe disordered eating practices are harmless. To the contrary, food restriction and purging can result not only in menstrual dysfunction and potentially irreversible bone loss but also in psychological and other medical complications, including depression, fluid and electrolyte imbalance, and changes in the cardiovascular, endocrine, gastrointestinal, and thermoregulatory systems.[8,9] Some of these complications are potentially fatal.

MENSTRUAL DYSFUNCTION

Menstrual dysfunction in athletes may include primary amenorrhea, secondary amenorrhea, oligomenorrhea, and luteal phase deficiency.[10–12]

An adolescent is considered to have delayed pu-

The recommendations in this statement do not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

PEDIATRICS (ISSN 0031 4005).

berty when breast development has not begun by 13.3 years of age.[13] Because sports involvement or poor nutrition may be associated with a delay in development, evaluation might be postponed until 14 years of age, as determined by clinical judgment. Primary amenorrhea is defined as the absence of menses by age 16 years. If menses have not occurred within 4.5 years after the onset of breast development, evaluation should be considered. Secondary amenorrhea is typically defined as the absence of at least 3 to 6 consecutive menstrual cycles in a female who has begun menstruating. Oligomenorrhea refers to menstrual periods that occur at intervals longer than every 35 days.[11,12,14] Although adolescents may have irregular periods or amenorrhea for 3 to 6 months in the first several years after menarche, the cessation of menses for longer than 3 months after regular cycles have begun or persistent oligomenorrhea is considered abnormal.[15]

Menstrual dysfunction is more common in athletes than in the general population. Athletes and dancers who begin training before menarche occurs may experience a later menarche and have an increased incidence of menstrual dysfunction when compared with girls who begin training after menarche occurs.[16–19] The prevalence of secondary amenorrhea in adult athletes ranges from 3.4% to 66% (depending on the sport studied and the criteria used to define amenorrhea),[10–12] compared with 2% to 5% of women in the general population.[12] The prevalence of secondary amenorrhea in the young athlete is unknown.

Luteinizing hormone (LH) pulsatility and, therefore, normal menstrual function are dependent on energy availability (dietary energy intake minus energy expenditure from exercise).[20] Low energy availability causes a hypometabolic state characterized by a variety of substrate and hormonal alterations, including hypoglycemia, hypoinsulinemia, hypothyroidemia, hypercortisolemia, and the suppression of the 24-hour mean and amplitude of the diurnal rhythm of leptin.[2,20–23] Amenorrheic and regularly menstruating athletes display reduced LH pulse frequencies[24] and similarly low 24-hour mean leptin levels.[25] However, amenorrheic athletes are distinctive in having a more extreme suppression and disorganization of LH pulsatility[24] and a complete suppression of the amplitude of the diurnal rhythm of leptin.[25] It is not known whether a particular threshold of energy availability is required to maintain normal reproductive function or whether the macronutrient composition of the diet is important.

Menstrual dysfunction may lead to decreased BMD. Other long-term consequences of a chronically estrogen-depleted state in young women are unknown at this time.

DECREASED BONE MINERAL DENSITY

Hypoestrogenism associated with amenorrhea may predispose to osteoporosis.[26,27] Osteoporosis is defined as premature bone loss and/or inadequate bone formation resulting in low bone mass and microarchitectural deterioration.[1] Adequate levels of estrogen slow bone resorption and improve or maintain bone mass.[28,29] The prevalence of osteoporosis in adult and adolescent women is unknown.[1] Studies of adult female athletes have shown that premature osteoporosis may occur as a result of amenorrhea and oligomenorrhea and may be partially irreversible despite resumption of menses, estrogen replacement, or calcium supplementation.[27,30,31] Amenorrheic adolescents, both athletes and nonathletes, have been found to have lower BMD than eumenorrheic adolescents.[28,32–34] This may be attributable to decreased bone accretion as well as increased bone loss.[35] An overall increase in BMD is demonstrated throughout adolescence. However, the amenorrheic teenager remains osteopenic in comparison with regularly menstruating teenagers.[28,32–35]

High-intensity exercise in some sports for many years may actually increase BMD in specific skeletal sites despite amenorrhea. Elite adolescent ice skaters and gymnasts have been found to have increased BMD in the lower skeleton, compared with controls, despite menstrual dysfunction.[29,36,37]

Girls who begin menarche at a later age and have a lower weight during adolescence have been found to have the lowest BMD when compared with their peers.[34,35] An increased incidence of stress fracture in dancers has been associated with older age at menarche.[38] Weight gain and the resumption of menses result in increased BMD.[33,35] Estrogen replacement therapy may decrease bone loss and potentially increase BMD in the adolescent with secondary amenorrhea.[28,35,39]

CLINICAL EVALUATION

The physical examination that precedes participation in sports is an ideal opportunity to screen for problems of disordered eating, menstrual dysfunction, and decreased BMD.[40] Signs of disordered eating may be recognized by parents, coaches, athletic trainers, teammates, or school nurses and brought to the physician's attention. If an athlete shows signs of disordered eating behavior, further evaluation by the physician, a nutritionist, and a mental health professional may be necessary.[3]

The diagnosis of primary amenorrhea or secondary amenorrhea in an athlete first requires a full evaluation to rule out pregnancy and underlying pathologic conditions. Pathologic conditions that may cause menstrual dysfunction include pituitary tumors (especially prolactinomas), thyroid dysfunction, polycystic ovary disease, premature ovarian failure, and other chronic illnesses.[10]

Evaluation of amenorrhea includes a complete physical examination and pelvic examination when indicated. A pregnancy test is usually indicated. Laboratory studies may include measurement of thyroid-stimulating hormone, prolactin, and follicle-stimulating hormone (FSH). If the athlete shows signs of androgen excess (eg, hirsutism, acne) or if the pelvic examination reveals polycystic ovaries, a determination of levels of LH, testosterone, dehydroepiandrosterone sulfate (DHEA-S), and 17-hydroxyprogesterone may need to be done. A progesterone challenge may help determine if the patient is hy-

poestrogenemic.[10,15] The possible use of anabolic steroids should also be considered.

In the athlete who has been amenorrheic, a study to evaluate BMD may be helpful.[29]

TREATMENT

The female athlete who has restrictive eating patterns because she is unaware of her energy needs may require only nutritional counseling. The female athlete who purposefully engages in disordered eating behaviors is often best treated using a multidisciplinary team approach: with a physician who monitors her medical status and ability to participate safely in sports, a nutritionist who provides appropriate nutritional guidance, and a mental health professional who addresses any psychological issues.[3]

Increased dietary energy intake or decreased energy expenditure (exercise) usually results in the development or resumption of menses and ovulation in adolescent girls and women with exercise-associated amenorrhea.[29,31] Daily requirements for calories, carbohydrates, and protein are greater in athletes than in more sedentary women and girls,[41] and diet should be changed accordingly. The recommended daily dietary allowance of calcium for adolescents is 1200 mg. Amenorrheic athletes should be encouraged to increase their calcium intake to at least 1500 mg daily. If intake of dietary sources of calcium is inadequate, calcium supplements may be recommended. If estrogen levels are deficient, the efficacy of calcium supplementation in improving bone mass may be impaired.[10,29,42]

In adolescents and young women with hypothalamic amenorrhea, estrogen-progesterone supplementation may help maintain bone density, protect the endometrium, and promote regular menses at predictable times.[10,28,35] The criteria for initiating estrogen replacement therapy and the optimal dosing schedule have not been determined. The minimum daily estrogen dose that has been shown to prevent bone loss in postmenopausal women is 0.625 mg of conjugated estrogens[43]; this dose, however, has not been shown to increase BMD in young women with hypothalamic amenorrhea.[28] Supplementation with a low-dose oral contraceptive (<50 μg of estrogen per day) is a readily available source of estrogen and may be associated with an increase in total body BMD.[39]

RECOMMENDATIONS

1. Exercise and sports participation should be promoted in girls and adolescents for health benefits and enjoyment.
2. Dietary practices; exercise intensity, duration, and frequency; and menstrual history need to be reviewed during evaluations that precede participation in sports or other medical encounters in which related problems may present.
3. Amenorrhea should not be considered a normal response to exercise. Exercise-associated amenorrhea or amenorrhea attributable to decreased energy availability should be considered a diagnosis of exclusion. A complete medical evaluation is required for any adolescent with primary or secondary amenorrhea or persistent oligomenorrhea.
4. Disordered eating should be considered in adolescents with amenorrhea. Treatment often requires a team of health care professionals, including a physician, nutritionist, and mental health professional, all experienced in the treatment of eating disorders, in addition to cooperation by coaches, parents, and teammates.
5. Education and counseling should be provided to athletes, parents, and coaches regarding disordered eating, menstrual dysfunction, decreased bone mineralization, and adequate energy (calorie) and nutrient intake to meet energy expenditure and maintain normal growth and development.
6. When athletes and coaches want to know what weight and amount of body fat are best for a given athlete, it is preferable to establish a range of values rather than specific values. It is difficult and potentially dangerous to define an ideal level of weight and/or body fat for each sport or individual participant. Weight is not an accurate estimate of fitness or fatness, and when weight is lost, muscle and fat are lost.
7. An adolescent with menstrual dysfunction attributed to exercise should be encouraged to increase her energy (caloric) intake and modify excessive exercise activity. If an athlete's weight is low, she may be required to gain weight before resuming athletic activity.
8. Estrogen-progesterone supplementation may be considered in the mature amenorrheic athlete.
9. Measurement of BMD may be considered as a tool when making treatment decisions for the amenorrheic athlete.

STAFF
Heather Newland

REFERENCES

1. Yeager KK, Agostini R, Nattiv A, Drinkwater B. The female athlete triad: disordered eating, amenorrhea, osteoporosis. *Med Sci Sports Exerc.* 1993;25:775–777
2. Loucks AB, Heath EM. Induction of low-T_3 syndrome in exercising women occurs at a threshold of energy availability. *Am J Physiol.* 1994; 266:R817–R823
3. Johnson MD. Disordered eating in active and athletic women. *Clin Sports Med.* 1994;13:355–369
4. Benson J, Gillien DM, Bourdet K, Loosli AR. Inadequate nutrition and chronic calorie restriction in adolescent ballerinas. *Phys Sportsmed.* 1985; 13:79–90
5. Loosli AR, Benson J, Gillien DM, Bourdet K. Nutrition habits and knowledge in competitive adolescent female gymnasts. *Phys Sportsmed.* 1986;14:118–130
6. Rosen LW, McKeag DB, Hough DO, Curley V. Pathogenic weight-control behavior in female athletes. *Phys Sportsmed.* 1986;14:79–84
7. Dummer GM, Rosen LW, Heusner WW, Roberts PJ, Counsilman JE. Pathogenic weight-control behaviors of young competitive swimmers. *Phys Sportsmed.* 1987;5:22–27
8. Palla B, Litt IF. Medical complications of eating disorders in adolescents. *Pediatrics.* 1988;81:613–623
9. Ratnasuriya RH, Eisler I, Szmuckler GI, Russell GF. Anorexia nervosa: outcome and prognostic factors after 20 years. *Br J Psychiatry.* 1991;158: 495–502
10. Shangold M, Rebar RW, Wentz AC, Schiff I. Evaluation and management of menstrual dysfunction in athletes. *JAMA.* 1990;263:1665–1669
11. Loucks AB. Effects of exercise training on the menstrual cycle: existence and mechanisms. *Med Sci Sports Exerc.* 1990;22:275–280
12. Loucks AB, Horvath SM. Athletic amenorrhea: a review. *Med Sci Sports Exerc.* 1985;17:56–72
13. Sperling MA, ed. *Pediatric Endocrinology*. Philadelphia, PA: WB Saunders; 1996
14. Sperhoff L, Glass RH, Kase NG. Amenorrhea. In: *Clinical Gynecologic Endocrinology and Infertility.* 5th ed. Philadelphia, PA: Williams & Wilkins; 1994:401–456
15. Emans SJ, Goldstein DP. *Pediatric and Adolescent Gynecology.* 3rd ed. Boston, MA: Little, Brown, and Co; 1990
16. Frisch RE, Gotz-Welbergen AV, McArthur JW, et al. Delayed menarche and amenorrhea of college athletes in relation to age of onset of training. *JAMA.* 1981;246:1559–1563
17. Warren MP. The effects of exercise on pubertal progression and reproductive function in girls. *J Clin Endocrinol Metab.* 1980;51:1150–1157
18. Malina RM. Menarche in athletes: a synthesis and hypothesis. *Ann Hum Biol.* 1983;10:1–24
19. Malina RM. Physical activity, sport, social status and Darwinian fitness. In: Strickland SS, Shetty PS, eds. *Human Biology and Social Inequality.* Cambridge, England: Cambridge University Press; 1998:165–192
20. Loucks AB, Verdun M, Heath EM. Low energy availability, not stress of exercise alters LH pulsality in exercising women. *J Appl Physiol.* 1998; 84:37–46
21. Loucks AB, Callister R. Induction and prevention of low-T_3 syndrome in exercising women. *Am J Physiol.* 1993;264:R924–R930
22. Loucks AB, Heath EM. Dietary restriction reduces luteinizing hormone (LH) pulse frequency during waking hours and increases LH pulse amplitude during sleep in young menstruating women. *J Clin Endocrinol Metab.* 1994;78:910–915
23. Laughlin GA, Yen SS. Nutritional and endocrine-metabolic aberrations in amenorrheic athletes. *J Clin Endocrinol Metab.* 1996;81:4301–4309
24. Loucks AB, Mortola JF, Girton L, Yen SS. Alterations in the hypothalamic-pituitary-ovarian and hypothalamic-pituitary-adrenal axes in athletic women. *J Clin Endocrinol Metab.* 1989;68:402–411
25. Laughlin GA, Yen SS. Hypoleptinemia in women athletes: absence of a diurnal rhythm with amenorrhea. *J Clin Endocrinol Metabol.* 1997;82: 318–321
26. Cann CE, Genant HK, Ettinger B, Gordon GS. Spinal mineral loss in oophorectomized women: determination by quantitative computed tomography. *JAMA.* 1980;244:2056–2059
27. Cann CE, Martin MC, Genant HK, Jaffe RB. Decreased spinal mineral content in amenorrheic women. *JAMA.* 1984;251:626–629
28. Emans SJ, Grace E, Hoffer FA, Gundberg C, Ravnikar V, Woods ER. Estrogen deficiency in adolescents and young adults: impact on bone mineral content and effects of estrogen replacement therapy. *Obstet Gynecol.* 1990;76:585–592
29. Snow-Harter CM. Bone health and prevention of osteoporosis in active and athletic women. *Clin Sports Med.* 1994;13:389–404
30. Drinkwater BL, Bruemner B, Chesnut CH. Menstrual history as a determinant of current bone density in young athletes. *JAMA.* 1990;263: 545–548
31. Drinkwater BL, Nilson K, Ott S, Chestnut CH. Bone mineral density after resumption of menses in amenorrheic athletes. *JAMA.* 1986;256: 380–382
32. Bachrach LK, Guido D, Katzman D, Litt IF, Marcus R. Decreased bone density in adolescent girls with anorexia nervosa. *Pediatrics.* 1990;86: 440–447
33. Dhuper S, Warren MP, Brooks-Gunn J, Fox R. Effects of hormonal status on bone density in adolescent girls. *J Clin Endocrinol Metab.* 1990;71: 1983–1088
34. White CM, Hergenroeder AC, Klish WJ. Bone mineral density in 15 to 21 year old eumenorrheic and amenorrheic subjects. *Am J Dis Child.* 1992;146:31–35
35. Bachrach LK, Katzman DK, Litt IF, Guido D, Marcus R. Recovery from osteopenia in adolescent girls with anorexia nervosa. *J Clin Endocrinol Metab.* 1991;72:602–606
36. Robinson T, Snow-Harter C, Gillis D, Shaw J. Bone mineral density and menstrual cycle status in competitive female runners and gymnasts. *Med Sci Sports Exerc.* 1993;25:549. Abstract
37. Slemenda CW, Johnston CC. High intensity activities in young women: site specific bone mass effects among female figure skaters. *Bone Miner.* 1993;20:125–132
38. Warren MP, Brooks-Gunn J, Hamilton LH, Warren LF, Hamilton WG. Scoliosis and fractures in young ballet dancers: relation to delayed menarche and secondary amenorrhea. *N Engl J Med.* 1986;314:1348–1353
39. Hergenroeder AC, Smith EO, Shypailo R, Jones LA, Klish WJ, Ellis K. Bone mineral changes in young women with hypothalamic amenorrhea treated with oral contraceptives, medroxyprogesterone, or placebo over 12 months. *Am J Obstet Gynecol.* 1997;176:1017–1025
40. Johnson MD. Tailoring the preparticipation exam to female athletes. *Phys Sportsmed.* 1992;20:60–72
41. Steen SN. Nutritional concerns of athletes who must reduce body weight. *Sports Science Exchange, Nutrition.* Oct 1989;2(20)
42. Drinkwater BL, Chesnut CH. Site specific skeletal response to increased calcium in amenorrheic athletes. *Med Sci Sports Exerc.* 1992;24:545
43. Lindsay R, Hart DM, Clark DM. The minimum effective dose of estrogen for prevention of postmenopausal bone loss. *Obstet Gynecol.* 1984; 63:759–763

Medical Concerns in the Female Athlete

Committee on Sports Medicine and Fitness

Pediatrics 2000;106;610
DOI: 10.1542/peds.106.3.610

Medical Concerns in the Female Athlete

Committee on Sports Medicine and Fitness

Pediatrics 2000;106;610
DOI: 10.1542/peds.106.3.610