Although eating disorders have received much attention over the last few years in athletics [1–9], their prevalence was not always well-appreciated. For instance, Cathy Rigby, a 1972 Olympian, battled with an eating disorder that lasted for over 10 years during her gold medal career. This later resulted in cardiac arrest on two different occasions. Despite her status as a world-class athlete, her battle with an eating disorder was not widely known. It was not until the death of Karen Carpenter in 1983 from anorexia nervosa (AN) that the public’s interest in eating disorders began to increase. Later in 1994, Christy Henrich, who had established herself as one of the top gymnasts in the country, died of multiple organ failure at the young age of 22 as a complication of an eating disorder. At this time, professional organizations began to pay close attention to the effects of eating disorders, and with this heightened awareness, professionals as well as the general public began to recognize eating disorders as a major problem in our society.

Around the same time, the American College of Sports Medicine (ACSM) convened the Task Force on Women’s Issues in Seattle, Washington [10,11]. During this conference, members of the ACSM discussed issues related to females and athletics, with specific attention to amenorrhea, osteoporosis, and eating disorders. They called these pathologies collectively the female athlete triad in 1993. Since the creation of the term, much time and effort has been devoted toward research and understanding of the triad. In trying to understand this complicated problem, one must grasp the concept that the three pathologies are interrelated and difficult to explain without the influence of any of the other components.

AMENORRHEA

The first of the three pathologies is amenorrhea. Amenorrhea simply stated is a lack of a menstrual cycle. It can be divided into two different categories: primary and secondary amenorrhea. Primary amenorrhea, or delayed menarche, is the absence of menstruation by the age of 16 in a female who has secondary sex characteristics [12]. Secondary amenorrhea is the absence of three or more
consecutive menstrual cycles after menarche [12]. Even more common is the occurrence of irregular menstrual cycles with decreased frequency, or oligomenorrhea [13]. AN has been linked to both amenorrhea and hypoestrogenism [14,15], whereas oligomenorrhea has been observed in bulimic individuals who have normal body weight [16]. Furthermore, oligomenorrhea has also been reported in individuals who have participated in pathogenic weight control behaviors but do not have an eating disorder [16,17]. Physical training has also been associated with changes in a female’s menstrual cycle [18]. Although the exact etiology of menstrual dysfunction has not been elucidated, excessive body weight loss, low body fat, stress, and amount of training were all originally suggested as the cause [19].

Amenorrhea has not only been known as a marker for AN [20], but other research has shown an important link between female athletes and disrupted menstrual cycles [21]. Low serum estrogen levels also appear to be associated with athletic amenorrhea and osteoporosis. This relationship may exist because much of the body’s estrogen storage lies within adipose tissues, and low body-fat compositions could lead to decreased estrogen stores. It is this body composition that many females and female athletes appear to use as a gauge to assess their activity level. Eating disorders and the avoidance of fatty foods, such as dairy products, may result from this behavior, and this may then contribute to both amenorrhea and secondary osteoporosis. Epling and co-workers [22] further showed that food intake in athletes was reduced following high levels of exercise. West [23] also stated that patterns of eating disorders may lead to menstrual dysfunction and subsequent osteoporosis. Thus maintenance of the body’s total energy stores appears to be crucial to the prevention of amenorrhea, a physical sign of an eating disorder [24].

Important to remember is that amenorrhea does fall along a continuum and demonstrates great individual variability [25]. Some individuals may have several regular cycles but cease for the duration of a year, or may have infrequent, unpredictable cycles. These disruptions in the menstrual cycle can produce effects similar to those of menopause, with decreased ovarian hormone (primarily estrogen) production. This decrease in hormone production has been shown to lead to decreased bone mineral density (BMD) [26–28].

In terms of the etiology of amenorrhea, the literature remains mixed. Frisch and McArthur [29] originally theorized that body fat determined a minimum weight for the onset of a menstrual cycle. Further investigation showed this to be inaccurate [2,16,30–32]. A more widely accepted theory discussed by Loucks and coworkers [33,34] is the “energy availability hypothesis.” This hypothesis states that an individual exercises until she is in a negative caloric state and remains in this state for prolonged periods of time. This occurs when an individual does not ingest enough calories for the amount of exercise she is performing. Loucks and his associates [33,34] showed that dietary stress (low caloric intake) resulted in decreases in cyclical levels of luteinizing hormone (LH), and that LH was depressed in women who have low dietary intakes. The researchers further stated that whether or not dietary intervention to increase
available energy would completely reverse the hypothalamic-pituitary dis-
ruption and subsequent menstrual disturbance remained to be fully determined
[33]; however, amenorrheic athletes are distinctive in having a more extreme
suppression and disorganization of LH pulsatility and a complete suppression
of the amplitude of the diurnal rhythm of leptin [35,36]. 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 [37].

Supporting research has shown that dietary restriction is the culprit for
disruptions in the menstrual cycle, not the level of body fat [38–41]. Amenor-
rhea may be a consequence of severe weight loss, but in a minority of indi-
viduals, may actually precede it [20]. In prepubertal females, menarche may
actually be delayed by severe weight loss or lack of weight gain [20]. In
postmenarchal females, amenorrhea is an indicator of physiological dysfunction
in AN. This may be due to an abnormally low level of estrogen secretion, which
is due in turn to diminished pituitary secretion of follicle-stimulating hormone
(FSH) and LH [20].

It is also believed that athletic amenorrhea is caused by a disruption in the
hypothalamic-gonadal axis, but the exact cause of the disruption is unknown
and is commonly believed to be multifactorial [42,43]. Keizer and Rogol [44]
and Prior and associates [45] concluded that no one factor can be singled out as
the primary cause of menstrual cycle dysfunction, because athletic amenorrhea
appears to result from a combination of nutritional deprivation, physical illness,
stress, and excessive exercise.

Of primary concern to the treating practitioner should be the realization that
early onset and long duration of amenorrhea predisposes an individual to
osteopenia [46] and may be irreversible [47]. It is well understood that most
bone development occurs during early childhood and late adolescence. The
adolescent years are especially important because 60% of peak bone mass is
acquired during these years [48]. Specifically, bone mineral density in normal
female adolescents increases 45% to 60% during the second decade (10%–15%
during stages 4 and 5 in pubertal development), peaks by the end of the third
decade, and then declines at a rate of 1% to 2% per year [48]. Therefore, with
amenorrhea and other menstrual disruptions as physiologic markers for many
pathologies, it is critical to investigate the adolescent who is having difficulty
with her menstrual cycle.

Even though menstrual irregularities have significant effects on the body,
these effects can be reversible [49,50]. Diddle [51] showed that gaining weight,
decreasing the intensity of training, or otherwise altering the physical and
psychological stresses on the athlete usually restores normal menstrual cycling.
More specifically, most athletes who have secondary amenorrhea will experi-
ence a return in their normal menstrual cycles within 1 year, even without an
alteration in their training schedules [52]. Shangold and Levine [53] showed that
future fertility was not impaired as a result of female marathon training.
Drinkwater and colleagues [54] also showed that the resumption of menses
increased the vertebral BMD of the former amenorrheic female athletes, but these increases did not return the BMD to normal levels. Other researchers have confirmed that premature bone loss in amenorrheic women is only partially reversible, even when normal menstruation is restored [54,55]. Therefore, although menstrual irregularities have no negative effects on future fertility, increases in BMD associated with resumption of normal menses may not bring BMD back to normal levels.

With regards to the resumption of menses and eating disorders, Golden and coauthors [32] showed that menses resumed at a mean of 9 months after patients were initially seen and required a weight gain of 2 kg above the weight at which they became amenorrheic. Specifically, a weight of approximately 90% of ideal body weight was the average weight at which resumption of menses occurred, and is a reasonable treatment goal weight. Confirmatory data revealed that 86% of studied patients who achieved this weight gain goal resumed menses within 6 months [32]. Subjects who remained amenorrheic at 1 year had lower levels of LH and FSH at baseline, and lower levels of LH and estradiol at follow-up. The investigators concluded that the resumption of menses required restoration of hypothalamic-pituitary-ovarian function and serum estradiol levels, and was not dependent on the amount of body fat [32].

**OSTEOPOROSIS**

Osteoporosis, as defined by the ACSM, is a disease characterized by low bone mass and microarchitectural deterioration of bone tissue, leading to enhanced skeletal fragility and increased risk of fracture [11,56]. The World Health Organization (WHO) [57] has established guidelines on how to classify BMD using dual energy radiographic absorptiometry (DEXA). Osteoporosis is defined as BMD greater than 2.5 standard deviations below the mean of young adults. Osteopenia is defined by a BMD 1 to 2.5 standard deviations below the mean of young adults. Recently, there has been a change. The International Society for Clinical Densitometry (ISCD) published a position statement [58] that the WHO’s guidelines for osteoporosis should not be used on healthy premenopausal females. They further state that Z-scores should be used instead of T-scores in the diagnosis of osteoporosis in this population.

In conjunction with osteoporosis, there is an increased risk for fracture in the old as well as the young. Some associated risk factors are: corticosteroid or thyroid medications, smoking, a low calcium diet, amenorrhea, a positive family history for osteoporosis [59,60], a sedentary lifestyle, and a lack of hormone replacement therapy postmenopause [60]. Simply being a woman also increases the individual’s chance for developing osteoporosis eight times when compared with men. This is due to a decreased baseline bone mass and an increased level of bone absorption associated with menopause [61]. As such, there is an alarmingly high risk for hip fracture in women; this risk is equal to that of the combined risk for many of the more common cancers [62,63]. Although some of these fractures can cause severe morbidity, some of these complications can prove to be fatal, depending on age and individual comorbidities [64].
Therefore, it is imperative that the female athlete understands that she is already at a higher risk for developing a potentially debilitating disease.

During a process known as “bone remodeling,” bone is constantly being broken down and replenished. Remodeling consists of different cycles of bone resorption and formation, which are commonly known as osteoblastic and osteoclastic activity. Osteoblasts are cells responsible for bone formation [65] during both the remodeling phase and development phase of bone growth. During a remodeling phase, a woven bone is produced, whereas other more mature osteoblasts can produce lamellar bone [65]. One of the products of osteoblasts is osteocalcin, which is an important marker for bone formation [65,66] and is increased when bone turnover is accelerated [65]. Osteoclasts are large and are very important when dealing with bone resorption. With osteoporosis, there is greater osteoclastic activity than osteoblastic activity, which accounts for the decrease in BMD.

Estrogen has direct effects on osteoblasts by increasing cell proliferation and proteins that alter bone remodeling. For instance, estrogen stimulates the synthesis of transforming growth factor-B, insulin-like growth factor-1 (IGF-1), and the IGF-1 binding proteins that also aid in bone formation [67]. Another function of estrogen is that it inhibits the production of cytokines associated with bone resorption [67].

During the critical adolescent years, bone growth is greater than bone resorption. This may account for the greater BMD in younger, healthier individuals. This begins to change around age 25 [68–70]. In females, progressive bone loss, or an increased bone resorption compared with bone formation, starts around age 20. This does not become symptomatic in women until age 45 and in men until 55 [71]. After age 55, one out of three American women will sustain a hip fracture [72]. Individuals who have high osteoclastic activity will have greater bone loss and have a higher risk of osteoporosis. Obviously, then, it is vital that the individual have as much bone mass as possible at the time of menopause to combat the fact that menopause in and of itself is linked to osteoporotic fractures [73]. This only strengthens the point that young female athletes need to acquire the greatest amount of bone mass that they can during their first 2 decades.

Much attention has been directed to the effects of intense exercise on bone mineral density. It is commonly known that disruptions in the menstrual cycle can be a byproduct of exercise [18]. This menstrual irregularity has been linked to lower bone density and a higher incidence of stress fractures when compared with regular controls [19,28,55,74–77]. AN has also been linked to lowered BMD [7,26,78–83]. Cobb et al [78] further looked at 91 competitive female distance runners aged 18 to 26. They found that disordered eating was strongly related to menstrual irregularity, that menstrual irregularity was associated with low BMD, and that disordered eating was associated with low BMD in the absence of menstrual irregularity.

Davies and coauthors [84] looked at 200 young amenorrheic women who had low BMD. The researchers found that the subjects had a mean reduction in
BMD of 15% when compared with normally menstruating, age-matched controls. They also found that 36% of the subjects had suffered at least one stress fracture, despite the fact that they did not have significantly different BMDs when compared with the remainder of the subjects.

**DISORDERED EATING**

The two eating disorders which the American Psychiatric Association (APA) recognizes are AN and bulimia nervosa (BN) [20]. Both are characterized by severe disturbances in eating behavior. AN is characterized by a refusal to maintain a minimally normal body weight [20]. BN is characterized by repeated episodes of binge eating, followed by inappropriate compensatory behaviors such as self-induced vomiting; misuse of laxatives, diuretics, or other medications; fasting; or excessive exercise [20]. For AN, there are subtypes of restriction and binge-eating/purging. For BN, subtypes of purging and nonpurging can also be diagnosed. Along with these criteria, a disturbance in perception of body shape and weight is an essential feature of both disorders [20]. Another classification of eating disorders not otherwise specified (EDNOS) was recently created [20] for those disorders that do not fall within the classic AN or BN categories. Box 1 lists the diagnostic criteria for AN, BN, and the category EDNOS. For a more comprehensive review of the diagnosis of eating disorders, please refer to the *Diagnostic and Statistical Manual of Mental Disorders. 4th edition* (DSM-IV) [20].

According to the ACSM, there are some sport disciplines that are at a greater risk for developing one or more components of the female athlete triad, especially eating disorders [11]. They are as follows:

- **Sports in which performance is subjectively scored** (dance, figure skating, gymnastics)
- **Endurance sports favoring participants who have a low body weight** (distance running, cycling, and cross-country skiing)
- **Sports in which body contour-revealing clothing is worn for competition** (volleyball, swimming, diving, and running)
- **Sports using weight categories for participation** (wrestling, horse racing, martial arts, and rowing)
- **Sports in which prepubertal body habitus favors success** (figure skating, gymnastics, and diving)

Collectively speaking, eating disorders affect 1% to 62% of female athletes [3]. In a recent meta-analysis, Smolack and coworkers [3] examined data from 34 research studies from 1975 to 1999. They found that athletes, especially dancers, were at a greater risk for eating problems versus nonathletes. Elite athletes who participated in sports that emphasized thinness were also at risk of eating problems. Nonelite, non lean-sport participants appear to score better on measures of eating problems than do nonathletes. They also discovered that several groups of athletes were at higher risk versus nonathletes, including college women, dancers, participants in lean sports, elite athletes, and those competing in elite lean sports [3]. There were no differences between athletes
Box 1: American Psychiatric Association diagnostic criteria for eating disorders

Diagnostic Criteria for Anorexia Nervosa

Refusal to maintain body weight at or above a minimally normal weight for age and height (eg, weight loss leading to maintenance of body weight less than 85% of that expected); or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected.

Intense fear of gaining weight or becoming fat, even though underweight.

Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.

In postmenarcheal females, amenorrhea (ie, the absence of at least three consecutive menstrual cycles). A woman is considered to have amenorrhea if her periods occur only following hormone (eg, estrogen) administration.

Specific types

Restricting type: during the current episode of AN, the person has not regularly engaged in binge eating or purging behavior (ie, self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

Binge-eating/purging type: during the current episode of AN, the person has regularly engaged in binge eating or purging behavior.

Diagnostic Criteria for Bulimia Nervosa

Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

Eating, in a discrete period of time (eg, within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.

A sense of lack of control over eating during the episode (eg, a feeling that one cannot stop eating or control what or how much one is eating).

Recurrent and inappropriate compensatory behavior to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.

The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.

Self-evaluation is unduly influenced by body shape and weight.

The disturbance does not occur exclusively during episodes of AN.

Specific types

Purging type: during the current episode of BN, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

Nonpurging type: during the current episode of BN, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuses of laxatives, diuretics, or enemas; in binge-eating; or in inappropriate compensatory behaviors.
and nonathletes in gymnastics, swimming, and running. When the researchers looked at high school participants only, they found no significant difference between athletes and nonathletes with eating disorders. A protective effect of athletic participation was also found among two groups of nonelite, nonlean sports and more specifically, nonelite high school girls [3].

Sundgot-Borgen and Torstveit [8] recently studied a population of elite Norwegian athletes \((n = 1620)\) for the prevalence of eating disorders. They found that more athletes \((13.5\%)\) than controls had subclinical or clinical eating disorders \((AN, BN, anorexia athletica, and EDNOS)\). Eating disorders among female athletes competing in aesthetic sports were higher than observed in endurance \((25\%)\), technical \((17\%)\), or ball game sports \((16\%)\) [8]. They concluded that the prevalence of eating disorders was higher in athletes than their control group \((n = 1696)\), that leanness-dependent and weight-dependent sports were higher versus other sports, and that a higher prevalence was seen in female athletes versus male athletes [8].

Johnson and associates [1] reported on the National Collegiate Athletic Association (NCAA) eating disorder project. All subjects \((N = 1445)\) were Division I athletes competing in the NCAA. They found that 1.1\% of the females and 0\% of the males met the DSM-IV criteria for BN. None of the athletes met the criteria for AN. Binge eating was reported in more than one quarter of both male and female athletes, but female athletes were much more likely to feel out of control during an episode of overeating \((81\% \text{ in females versus } 45\% \text{ in males})\). When the full criteria for a binge were used, more female athletes \((23\%)\) than male athletes \((12\%)\) binged. More females vomited to lose weight than males, and they were more likely to have vomited monthly, weekly, or daily in the preceding 3 months. Self-reporting of disordered eating behaviors was naturally low.

Though true eating disorders have a low reported prevalence in the literature, there is a definite trend toward a higher prevalence of disordered eating in
athletics. Many athletes do participate in pathologic weight practices, but they are subclinical in terms of diagnosis. They do not meet all of the criteria for AN or BN, but rather fall in the EDNOS category. Unfortunately, there has been little standardization between eating disorder protocols over the years, which translates into conflicting data with regards to the prevalence of eating disorders and disordered eating in athletics. More research needs to be conducted to determine a more precise prevalence of these behaviors in athletics.

**IDENTIFICATION AND CLINICAL CORRELATES OF THE FEMALE ATHLETE TRIAD**

Identification of the female athlete triad may be accomplished in many different ways. What may be the easiest way to identify at-risk individuals is through recognition of the clinical correlates. Some individuals may develop lanugo, a fine downy hair that grows on the body due to a lack of adipose tissue, especially on the face [85]. This may be seen on individuals who at a severe caloric deficit. Russell’s sign is the presence of abrasions or small lacerations and calluses on the dorsum of the hand overlying the metacarpophalangeal and interphalangeal joints, due to repetitive contact with the incisors from self-induced vomiting [86,87]. Other oral complications include salivary gland hypertrophy, periodontal disease, and dental caries and enamel loss (the gradual decay and disintegration of a tooth) [88]. A female’s menstrual history can also be very helpful as it is one of the best predictors of current bone density [11,55]. A variety of ECG changes may be seen in individuals who have eating disorders [89].

Further identification of an at-risk individual may be through administration of an eating disorder survey. Though there are many different types of eating disorder questionnaires available, few have been through rigorous testing and validation procedures. According to Allison [90], the most common and widely used surveys are the Eating Disorder Inventory (EDI) [91], the Eating Attitudes Test (EAT) [92], and the Eating Disorder Examination (EDE) [93]. All three have shown clinical utility in the diagnosis of eating disorders.

**Eating Disorder Inventory**

The EDI questionnaire [91,94] was designed to assess psychological characteristics and symptoms common to AN and BN. It contains 64 items that form eight subscales: drive for thinness, bulimia, body dissatisfaction, ineffectiveness, perfection, interpersonal distrust, interoceptive awareness, and maturity fears. A later revised version of the EDI, the EDI-2, included just three of the original subscales: drive for thinness, bulimia, and body dissatisfaction. Both versions have good reliability and internal consistency. Though the EDI has been used diagnostically, it was originally designed for profiling purposes to be able to track changes that occur with treatment.

**Eating Attitudes Test**

The EAT questionnaire [92] was originally designed to assess AN, but now is being used as a tool to screen for eating disorders in general. The EAT is a
40-item, self-report measure, with each item being answered on a 6-point Likert scale (1 = never, 6 = always). Later, a revised version of the EAT [95] was created: the EAT-26. It contains 26 of the original 40 questions, disregarding 14 redundant questions. In terms of scoring, please refer to Garner and Garfinkel’s *Handbook of Treatment for Eating Disorders* [96].

**Eating Disorder Examination**

The Eating Disorder Examination is a semistructured interview for the assessment of general eating disorders [93]. The EDE contains 62 questions that are divided into five subscales: dietary restraint, bulimia, eating concern, weight concern, and shape concern. All questions refer to functioning over the previous 4 weeks, and each question is scored on a 7-point rating scale.

Though not nearly as accurate as an interview with a psychologist trained in the field of eating disorders, these questionnaires and interview can prove to be useful tools in the screening process for eating disorders. The best time to give such a survey may be during the preseason health screening process, because the athlete may be less likely to be defensive about her answers. It is also important to remember that if you are not qualified to diagnose someone with an eating disorder, you will probably be better served using terms like “disordered eating” or “poor dietary habits,” and to use the tools above as guides. If you are qualified to diagnose, the tools described in the earlier paragraphs have been used for the diagnosis of eating disorders for many years, but in no way should they take the place of clinical judgment.

**MANAGEMENT**

Clearly, the best approach to addressing female athlete triad issues is through prevention. Prevention can best be accomplished through athlete education and creation of a caring environment sensitive to these types of concerns. For athletes who have the female athlete triad, treatment needs to include multiple members of the sports medicine team because of the complexity of this pathology. Details of such treatment are beyond the scope of this article. According to the ACSM position statement on female athlete triad [11], any female athlete showing signs of any of the three components needs to be referred to a health care professional, and this health care professional must be a part of a treatment team that typically includes psychologists/psychiatrists, nutritionists, and the primary care/team physician.

**References**


