Position Stand
on
THE FEMALE ATHLETE TRIAD

IOC Medical Commission Working Group Women in Sport
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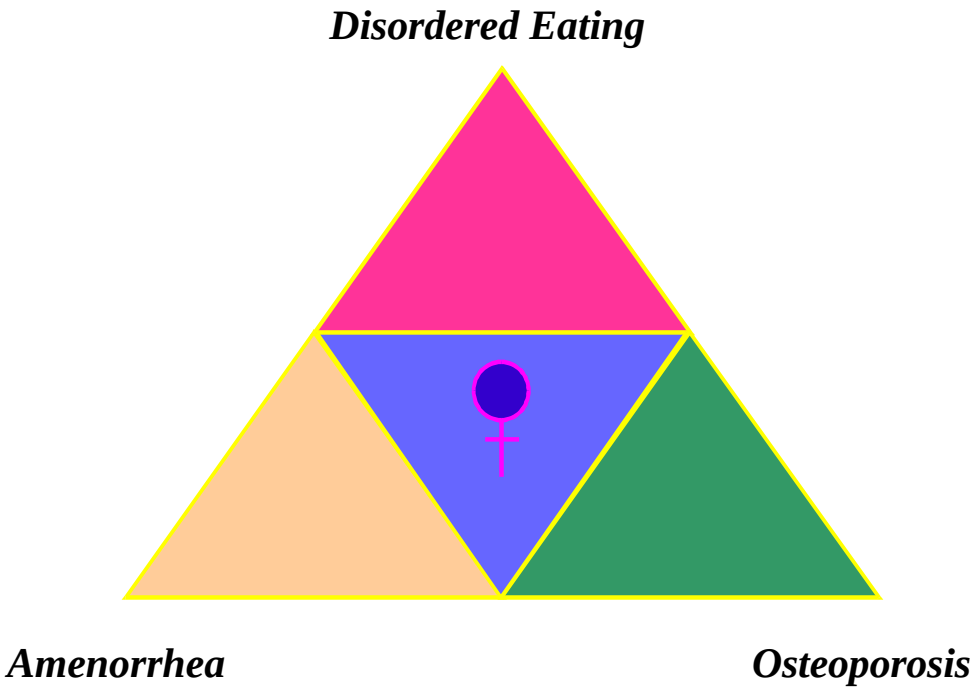

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Girls and women should be encouraged to participate in sports and to strive for excellence, but there is also an obligation on the part of health professionals responsible for their well-being to understand the influence of nutrition on reproductive and skeletal health. Low energy availability, defined as an energy intake inadequate to meet energy expenditure as well as other physiological needs, can disrupt the reproductive cycle and result in amenorrhea (40, 41). There may be no overt signs of an energy deficit other than an irregular or absent menses. Other athletes may respond to pressure to meet unrealistic weight or body fat levels with excessive dieting. In both instances the unintended effect can be a cascade of events labeled the Female Athlete Triad. Some athletes may slip into disordered eating, which in turn can lead to a serious eating disorder such as anorexia nervosa or bulimia nervosa. An inadequate caloric intake and a decrease in endogenous estrogen eventually result in an imbalance in bone remodeling leading to low bone mass or osteoporosis (20, 32). Although any one of these problems can occur in isolation, inadequate nutrition for a woman's level of physical activity often begins a cycle in which all three occur in sequence - hence the term ***"Female Athlete Triad".***

Physicians, trainers, and other healthcare providers should educate athletes and coaches that weight loss does not necessarily ensure improvement in athletic performance. Muscle mass as well as fat is lost during extreme dieting and performance may actually deteriorate. Other side effects of poor nutrition such as fatigue, anemia, electrolyte abnormalities, and depression can also contribute to a poor performance and impair the athlete's health. The Federation Internationale de Medecine du Sport (FIMS) Code of Ethics in Sports Medicine states clearly

that "Sports medicine physicians should oppose training and practices and competition rules as they may jeopardize the health of the athlete." Therefore, it is the responsibility of team physicians to ensure the health and safety of the athletes by: 1) educating athletes, coaches, and parents about the issue, 2) recognizing the symptoms associated with an eating disorder, 3) preventing activities that place pressure on athletes to lose weight, and 4) providing appropriate treatment for athletes who experience one or more of the Triad conditions.

A. Disordered Eating and Low Energy Availability

Definitions

There is a continuum model for disordered eating ranging from abnormal eating behaviors to clinical eating disorders (57). Included in this continuum are anorexia nervosa, bulimia nervosa, and eating disorders not otherwise specified but also eating problems that are subclinical (subthreshold); that is, problems that do not meet diagnostic criteria for an eating disorder. Disordered eating is believed to play a role in amenorrhea or menstrual dysfunction experienced by many athletes. Additionally, some athletes without disordered eating per se also develop amenorrhea through “low energy availability” (43).

Anorexia Nervosa

In anorexia nervosa, the extreme of restrictive eating behaviors, the athlete believes she is overweight even though she is below 85% of expected weight. Amenorrhea is one of the DSM-IV diagnostic criteria for anorexia nervosa as described in Appendix 1 (1).

Bulimia Nervosa

Bulimic behaviors follow a cycle of food restriction or fasting leading to overeating or binge eating followed by compensatory behaviors. Compensatory behaviors may include purging types such as vomiting, and/or the use of laxatives, diuretics, and enemas, and nonpurging types

such as fasting and/or excessive exercise (1). The diagnostic criteria are listed in Appendix 2. Athletes suffering from bulimia nervosa can be at any weight but are usually at “normal” body weight, and often experience menstrual irregularity.

Eating Disorder Not Otherwise Specified (EDNOS)

EDNOS refers to disorders of eating that do not meet the criteria for a specific eating disorder (1). Individuals with EDNOS are often of average weight; however, there is often still a preoccupation with body image and weight, as well as concern regarding eating. Diagnostic criteria for EDNOS are listed in Appendix 3.

Subclinical Eating Problems

In many cases of disordered eating, an athlete may deliberately attempt to lose weight or body fat by restricting her caloric intake, eliminating selected foods from her diet, fasting, and other abnormal eating behaviors without meeting diagnostic criteria for an eating disorder (6, 63). Often these problems involve dietary restriction for the purpose of decreasing weight or body fat and include behaviors from simple dieting to eating patterns resulting in psychological and medical complications that can benefit from treatment.

Low Energy Availability

Low energy availability can be caused by reducing dietary energy intake or by increasing exercise energy expenditure. Athletes may induce a negative energy balance when total energy expenditure exceeds energy intake. The result is an energy deficit. If this deficit is extreme, the body responds to reduce the deficit by suppressing physiological functions that are essential for growth, development and health. While these effects reduce and may even eliminate the energy deficit, they do not increase energy availability.

Low energy availability can result from disordered eating, but it can also occur in the absence of disordered eating. An athlete may be eating normally for a nonathlete but inadvertently or unknowingly ingesting insufficient calories to meet her energy needs as an athlete. The resultant low energy availability is a result of consuming fewer calories than needed to meet the energy costs of daily living plus those expended in exercise. Young athletes must also meet the energy costs of growth (44).

Low energy availability without eating disorders or even disordered eating may account for many cases of amenorrhea in athletes. However, athletes who suffer from disordered eating or eating disorders are at greater risk medically and psychologically and present a much greater challenge for the healthcare professionals who manage them. As a consequence, more of the focus in this section will be on this group.

Prevalence

A number of studies have reported a higher frequency of eating problems in athletes than nonathletes, particularly in athletes competing in sports that emphasize leanness or a low body weight (7, 9, 26, 28, 59, 63). Some studies have suggested a similar risk of eating disorders for female athletes in aesthetic sports (53), running (13, 71) and athletes at lower competitive levels (2) compared with controls. A recent controlled study (64) showed that the prevalence of eating disorders is higher in Norwegian female athletes (20%) than in non-athletic female controls (9%), and more common among those competing in leanness-dependent and weight-dependent sports than in other sports. The prevalence of athletes, who experience a chronic decrease in energy availability without the presence of disordered eating or eating disorders, is not known.

Risk Factors

Although disordered eating involves the full spectrum of dysfunctional eating from abnormal eating behaviors to clinical eating disorders, it is not known whether the different points on the continuum have the same risk factors or causative factors. Any factor that leads an athlete to restrict her caloric intake, whether inadvertent or by intent, could be considered a risk factor for disordered eating and low energy availability. Risk factors can be divided into two categories. The first category involves non-sport factors that place any individual, athlete or nonathlete, at risk for the development of disordered eating. The second category involves factors that are specific to athletes.

Non-Sport Related Factors

Dieting (Restrictive Eating)

Dieting (restrictive eating for the purpose of losing weight) is the primary precursor to the development of eating disorders. Although dieting appears to be necessary for the development of an eating disorder, it is probably not sufficient (72). In most societies, the group most concerned with dieting and weight loss is young females. Consequently, this is also the population with the highest incidence of such disorders (31, 72, 73).

Social Pressure

Eating disorders occur more frequently in young women who equate thinness with social success and/or who engage in activities that place pressure on them to be thin (25). In their seminal work on eating disorders, Garfinkel and Garner (24) proposed that the cause of eating disorders is multi-determined and that there are factors that predispose, precipitate, and perpetuate the disorder. In sports, predisposing factors (e.g., individual, family, culture) set the

stage for the disorder, which is triggered by a precipitating factor (e.g., comment on weight), and then maintained by perpetuating factors (e.g., initial success, approval by coach).

Athlete Specific Factors

Athletes are subject to all of the non-sport related risk factors plus additional factors specific to athletics. These include personality factors, pressure to lose weight through restricted eating and/or frequent weight cycling, early start of sport-specific training, injuries, symptoms of over training, and the impact of coaching behavior (59, 61). Some athletes refer to the development of their eating disorder as "it just happened; I never planned to lose weight" (61). These athletes are inducing an energy deficit when energy expenditure exceeds energy intake. Some of these athletes lose weight and develop an eating disorder (61). Thus, exercise itself could be considered a risk factor, because expending energy in exercise reduces the amount of dietary energy available for other physiological processes such as reproduction and growth.

Belief That Decreasing Body Weight/Fat Will Improve Performance

Included in the risks for disordered eating in the athletic environment is the prevailing notion in some sports that a decrease in body fat/weight can enhance performance. Often an initial loss of weight will lead to a better performance. This initial success can lead the athlete to continue efforts to lose weight and unknowingly slip into an eating disorder. The coach should not pressure an athlete to lose weight nor should the coach be involved in determining the athlete's weight. If the athlete is concerned about her weight or body composition, she should be referred to a dietitian/nutritionist for further evaluation and consultation. The following steps are suggested: 1) Evaluate the athlete's energy and nutrient intake. 2) Measure body composition by objective measurements (i.e., DXA), and then evaluate and discuss the results with the athlete. 3) Depending on these results, her eating and weight history, and her performance level, decisions

are then made regarding possible changes in her dietary intake and training schedule. If the athlete and her healthcare providers decide that changes in her weight/body composition (gain or reduction) are to be attempted, the athlete should be followed closely after the treatment goal has been reached (post follow up 8 weeks, 16 weeks and 12 months). 4) To reiterate, the athlete's healthcare providers in consultation with the athlete will make decisions regarding her weight and body composition. Coaches should not be involved in this process, nor should coaches even suggest weight reduction to athletes. Rather, coaches should express their concern for the athlete's health and optimal performance potential by suggesting that she seek the assistance she needs from appropriate healthcare providers. Coaches should express this concern regarding appropriate referral when the athlete has expressed concern regarding her condition, and/or when the coach believes that the athlete lacks sufficient energy for healthy sport participation and/or when she is engaging in unhealthy eating behavior.

Similarity of "Good Athlete" Traits and Eating Disorder Symptoms

Thompson and Sherman (66) have suggested that some traits that many coaches desire in their athletes (referred to as "good athlete" traits) are similar to traits often found in individuals with eating disorders, such as excessive exercise, perfectionism, and (over) compliance. Leon (38) has suggested that athletes may also evidence psychological traits such as high achievement orientation and obsessive-compulsive tendencies, which are commonly associated with clinical eating disorders but which are also expected and usually essential for competing successfully. Therefore, athletes are at increased risk because eating disorder symptoms may be misperceived by sport personnel as "good athlete" traits. Such attitudes and behaviors are apt to be rewarded in the sport environment. Thus, rather than identifying an individual who is in need of treatment,

sport personnel are apt to increase the “strength” and frequency of eating disorder symptoms through their reinforcement of such attitudes and behaviors.

Competitive Thinness

In some societies, young women compete in terms of thinness. Such competition is even more likely to occur in female athletes. By their nature, athletes tend to be competitive. Also, there are increased opportunities for unhealthy body comparisons in athletics. Athletes get dressed and shower together. In sports that involve revealing suits/uniforms, comparisons are made more easily. Moreover, in sports involving judging or appearance, the risk of an unhealthy comparison between performance and body increases. In such cases, the winner of a competition may be quite lean or thin. A less successful athlete may be inclined to make a body comparison and conclude that she would perform/look better if she were thinner, thereby leading to a process of dietary restriction that may then lead to disordered eating. Worse yet, it may be known to other competitors that the winner engages in pathogenic weight loss methods. Serious, committed athletes are often willing to try anything to enhance performance, even resorting to unhealthy methods, assuming that they will not be at risk. Severe energy deficit and eating disorders can have severe health consequences. Signs, symptoms and medical complications of severe energy deficit and eating disorders are listed in Appendix 4.

Recommendations for Management and Treatment

Assessment

Individuals with disordered eating are characterized by disturbances in eating behavior, body image, emotions, and relationships. Athletes constitute a unique population and special diagnostic considerations should be made when working with this group (5, 60, 64). Identifying disordered eating among athletes must go beyond focusing on those who meet formal diagnoses

for eating disorders to include those in an energy deficit who engage in a myriad of pathogenic weight control behaviors that have clinical significance and can severely compromise health and performance. Also in need of assessment would be those athletes without an overt eating disorder who are in an energy deficit but may not necessarily engage in pathogenic weight loss methods. Questions that are helpful to use at the first consultation with athletes expected to be at risk for disordered eating and the Triad are included in Appendix 5.

It is important to determine whether the athlete's abnormal eating and weight loss behaviors are transient, safely managed behaviors associated with the specific demands of the sport or if the symptoms are more persistent and part of a clinical eating disorder. Therefore, it is not enough to merely document behaviors; the emotional and psychological state of the athlete must also be examined. If, as a result of various signs and symptoms (i.e., menstrual dysfunction, weight loss, suboptimal weight, fatigue, performance decrement, excessive exercise, etc.), an athlete has been identified as not eating enough for her level of physical activity, or is believed to be "at risk" for abnormal eating behavior or an eating disorder by medical, athletic training, or coaching staffs, the following procedures are recommended:

1. Following a medical evaluation, the athlete should be referred to a registered or licensed dietitian, preferably one with experience and expertise in working with athletes with eating problems, for a nutritional assessment and meal planning. The focus should be educational with the aim of helping the athlete understand what her nutritional needs are for good health and performance.

2. For athletes who are unable or unwilling to follow the eating recommendations made by the dietitian in Step 1, a referral to an eating disorder treatment specialist, preferably one with experience working with athletes, should be mandatory.

3. If the eating disorder specialist recommends treatment, the athlete should be considered "injured" and must agree to treatment in order to continue training and competing. Having the athlete in treatment can facilitate recovery by addressing the athlete's eating issues and concerns. Treatment also provides a safeguard in that there is a trained professional involved with the athlete who can help ensure that the athlete's symptoms are not worsening.

4. For those affected athletes who agree to treatment, eligibility to continue training and competing while symptomatic would be determined on an individual basis by treatment staff. At a minimum, the athlete would have to be cleared medically and psychologically (that is, treatment providers have determined that sport participation will not increase the athlete's risk), her training/competition could not be used as a means to lose or control her weight, and she would be required to follow a prescribed set of health maintenance criteria. These criteria would be determined on an individual basis, and would generally include, but not be limited to:

   a. Being in treatment, complying with the treatment plan, and progressing toward therapeutic goals;
   b. Maintaining weight or percent body fat recommended by the treatment team;
   c. Eating enough to comply with the treatment plan regarding weight gain or weight maintenance;
   d. Accepting/Agreeing that the resumption of menses or achieving menarche is needed for good health, as well as a requirement for continuation in sport participation;
   e. Failing to meet the above criteria, the athlete would be withdrawn from training and competition.

5. For those affected athletes who refuse treatment, training and competition would be withheld until they agree to participate in treatment.

In addition, the governing bodies of women's sports should convene meetings to develop minimum physical standards for participation and competition appropriate for protecting the reproductive and skeletal health of participants in their sports, as well as procedures for implementing those standards. Having done so, they should also take action to ensure that these procedures are faithfully practiced.

Prevention of Disordered Eating

Primary Prevention

Because disordered eating and/or low energy availability is the key factor causing other components of the Female Athlete Triad, and because dieting is the primary risk associated with disordered eating, the drive towards dietary restriction by athletes must always be taken seriously. The obvious ways to prevent coaches from suggesting or forcing athletes to lose weight and to discourage athletes from restrictive eating are to educate athletes and coaches regarding the health risks of dietary restriction. Athletes and coaches need to be aware of the negative effects of disordered eating on performance. On the positive side, the benefits of physical and psychological health, sound nutrition, and positive body image should be emphasized. Some specific strategies to decrease the risk of disordered eating and/or low energy availability among athletes would include the following:

1. Recommend healthy eating for enhanced performance.

2. Discourage weight-loss objectives and forbid unhealthy weight-loss practices. In general, the best way to discourage athletes from setting unhealthy weight-loss objectives is for weight-loss objectives to be reviewed and approved only by medical and nutritional personnel, who are knowledgeable regarding the influence of energy availability on reproductive and skeletal health. Also, these weight-loss objectives should be implemented only if the athlete

agrees with the decision to lose weight as prescribed by appropriate healthcare personnel. All weight-loss practices should be specified and monitored by such personnel.

3. Enlist the support of coaches and governing sport organizations/federations. An educative or preventive program will not be successful if it is aimed only at athletes. Without the support of coaches, an educational or preventive program, regardless of content, has little chance of success. In order for coaches to adequately perform this supportive function, many need factual information on nutrition, the energy requirements of exercise, factors determining weight, risks, and causes of disordered eating, menstrual (dys)function, and psychological factors that both negatively and positively affect health and athletic performance. Also coaches have to be knowledgeable about all other factors influencing performance in the specific sport.

Coaches will need the support of their governing organizations and federations. This support could come in the form of providing education regarding disordered eating for coaches and athletes, strong position statements providing coaches with guidelines for prevention, and support to make the difficult and perhaps controversial decisions with respect to affected athletes as to whether they will be in treatment or be allowed to train and compete.

Secondary Prevention

The main goal in secondary prevention should be better identification of athletes with low energy availability in general, and disordered eating in particular, because better identification leads to earlier treatment. Earlier treatment results in fewer health risks for the athlete and an earlier return to training and competition. Improving identification involves education of individuals who are most likely to be in a position to identify disordered eating. In most cases,

this would include coaches and sports medicine personnel, such as team physicians and athletic trainers. Identification could be facilitated with appropriate screening procedures. Certainly, any athlete with amenorrhea, decreased bone mineral density, or overuse injuries should be evaluated. Questions that are appropriate for the first consultation with athletes "at risk" or who may already suffer from disordered eating or eating disorders are listed in Appendix 5. Finally, identification could be facilitated through the de-stigmatization of receiving a diagnosis of disordered eating, as well through proper and supportive management of the athlete following identification. This would make it easier for an affected athlete to come forward and seek treatment rather than feeling as though she must "hide" in order to avoid detection, embarrassment, and removal from training and competition.

Summary

- Disordered eating occurs on a continuum.
- Identifying disordered eating among athletes must go beyond focusing on those who meet formal diagnostic criteria for an eating disorder and should include athletes who are in a negative energy balance, or engage in unhealthy weight control practices that have clinical significance and that can severely compromise health and performance.
- In reviewing the literature on athletes and disordered eating, Brownell and Rodin (8) concluded that athletes have more problems with eating, dieting, and body image than nonathletes, and the problem appears to be greatest in sports in which there is an emphasis on thinness, either for performance or appearance. Athletes most at risk would be those involved not only in sports that emphasize a thin body size or shape (e.g., distance running) but also in sports that utilize weight categories (e.g., rowing, martial

arts), use revealing sport attire (e.g., swimming), are judged (e.g., diving, figure skating, gymnastics), or have an appearance aspect (e.g., rhythmic gymnastics).

- Female athletes at the greatest risk for disordered eating are those individuals who restrict energy intake either to lose weight or maintain a low body weight, those who increase exercise energy expenditure through increased hours of training and/or increase exercise intensity without increasing energy intake, and those athletes who are vegetarian or limit the types of food they eat.
- Additional risk factors for athletes include personality factors, pressure to lose weight, frequent weight cycling, early start of sport-specific training, being injured, overtraining, and coaching behavior (59, 61).
- Many athletes with disordered eating behaviors are in need of help from a healthcare provider to optimize eating behaviors and to redefine goals related to their performance, school work, and personal life.

- Treatments for clinical eating disorders include individual psychotherapy, cognitive group therapy, and nutrition counseling (Appendix 6). Ideally the team should include a registered sport nutritionist, a physician, and a psychologist or psychiatrist specializing in eating disorders.
- Athletes in treatment for disordered eating and eating disorders must meet the following criteria to continue training and competition: agree to comply with treatment strategies as best she can and be closely monitored by the medical and psychological healthcare professionals handling their treatment. Treatment should always take precedence over training and competition; and if the athlete is not able to meet the criteria, competition should not be recommended while the athlete is in active treatment.

- To prevent disordered eating and eating disorders, the athletes have to know and practice healthy eating and make sure energy intake covers energy needs. The team, staff, and parents must be able to recognize the physical symptoms and psychological characteristics that indicate a risk for clinical eating disorders.

B. Amenorrhea

Definitions

The terms eumenorrhea, oligomenorrhea and amenorrhea have each been variously defined for different purposes. If *eumenorrhea* is defined for college-aged women to span $\pm$ 1 standard deviation around the mean, then it ranges from 26-32 days. Cycles of this length are highly regular: eighty-five percent of menstrual cycles 25-34 days in length are followed by another cycle in the same range (46).

The term *oligomenorrhea* is typically used to refer to menstrual cycles longer than 35 days. Such long cycles are highly irregular: only ~25% of menstrual cycles 35-44 days in length are followed by another cycle in that range (46).

The term *amenorrhea* connotes a persistent absence of menstrual cycles. Investigators commonly require three or more months of missed menstrual cycles, depending on how confident they want to be that ovarian follicular development, ovulation and luteal function are absent. All women are amenorrheic before menarche, after menopause, and during pregnancy, but amenorrhea at other times is indicative of pathological changes in diverse physiological systems. The term *secondary amenorrhea* refers to amenorrhea that occurs sometime after the first menstrual cycle and is distinguished from *primary amenorrhea* in which the first menstrual cycle is delayed by some environmental, behavioral or medical factor. The American Society of Reproductive Medicine has recently redefined *primary amenorrhea* as the absence of menstrual

cycles in a girl who has not menstruated by age 15, even though she has undergone other normal changes that occur during puberty (52). This definition does not apply to countries in which sexual development occurs at different rates. Due to improved nutrition, age at the first menstrual cycle declined by five years in developed countries after the middle of the 19th century and is falling rapidly now in some developing countries.

Luteal suppression is an entirely asymptomatic condition evident only by measuring ovarian steroid hormone concentrations in the blood, saliva, or urine over a number of weeks. It should be suspected in short menstrual cycles, but can occur even in eumenorrheic women (41). In luteal suppression, the luteal phase of the menstrual cycle after ovulation is shortened and the production of the hormone progesterone during this phase is reduced. Without more progesterone for a longer time, many women with luteal suppression are also infertile, because the lining of the uterus is shed in the next bleeding episode before a fertilized egg can be implanted.

Prevalence

The estimated prevalence of secondary amenorrhea depends on how amenorrhea is defined: requiring more months without menstrual cycles leads to lower estimates. Large epidemiological studies of college-aged women have defined amenorrhea as no menstrual cycles for three consecutive months resulting in a range of estimates from 2 to 5% (3, 51, 58). Similarly large-scale studies of college-aged athletes using the same definition have not been performed, but in smaller studies of particular sports this definition has led to estimates as high as 44% in dancers (10) and 65% in long-distance runners (23). In general, the highest prevalence is found in endurance, esthetic and weight class sports.

The percentage of girls in the United States who have not menstruated by age 15 is less than 1% (12). By contrast, the percentage of 425 collegiate athletes in 15 sports at seven U.S. universities who had not menstruated by age 16 (as primary amenorrhea was previously defined) was found to be 7.4% overall and an astonishing 22.2% in three aesthetic sports (cheerleading, diving and gymnastics) (6). Luteal suppression is typical of eumenorrheic athletes (41). The occurrence of luteal suppression in one or more of three consecutive months in physically active women has been reported to be ~80% (15).

Etiology

All amenorrheic and oligomenorrheic athletes should be examined medically to ensure that each woman receives appropriate care by ruling out serious medical conditions unrelated to athletics that also disrupt menstrual function (30, 54 Figure 2). Some of these conditions may be disproportionately represented among athletes due to the self-selection of the affected women into activities in which their medical condition confers on them a competitive advantage.

The proximal cause of menstrual disorders in cases that cannot be attributed to medical conditions is cessation of the normal development of ovarian follicles, ovulation and luteal function. Follicular development depends critically on the frequency with which pulses of luteinizing hormone (LH) appear in the blood. Various hypotheses have been proposed to explain how exercise might disrupt LH pulsatility.

*Body Fat and Exercise Stress Hypotheses***.**

Inadequate body fat stores and exercise stress have each been proposed to disrupt menstrual cycles in athletes, but research into these hypotheses has shown that body composition (56) and exercise stress (43) play no role in the disruption of menstrual cycles in athletes.

Energy Availability Hypotheses.

Energy availability is the amount of each day's dietary energy remaining for body functions after some of that energy has been expended in exercise. In the laboratory, any combination of dietary restriction and exercise energy expenditure that reduces energy availability too much disrupts the pulsatile secretion of LH within five days (42). This imposes a limit on the amount by which athletes can lower energy availability to improve performance by reducing fat mass without compromising reproductive and skeletal health.

To maximize their performance in particular sports, many athletes need to modify their body size, body composition or stores of metabolic fuels. Athletes seeking to improve performance by reducing fat mass need special attention to ensure that they do not reduce energy availability too much and to optimize the macronutrient composition of their diets. At modestly restricted energy availabilities it is especially important to consume enough protein and carbohydrate for the remodeling of bone and skeletal muscle and the replenishment of muscle and liver glycogen stores. Replenishing liver glycogen stores for maintaining brain glucose availability may be critical for preserving LH pulsatility (42, 43).

Recently, it has been found that extremely low energy availabilities disrupt LH pulsatility more in habitually sedentary, eumenorrheic women who, under energy-balanced conditions, have luteal phases only 11 days long as determined by urinalysis of LH (42). Because of the time lag between the LH surge and the rises in progesterone and body temperature, an 11-day luteal phase determined by urinalysis of LH is the same length as a 10-day luteal phase determined by basal body temperature. Women with shorter luteal phases, i.e., with clinical short luteal phase, were not included in the experiment. If this finding is confirmed through further research, then testing for luteal phase length may be an inexpensive and convenient way to focus preventive care on the athletes at highest risk.

Amenorrheic athletes display abnormal levels of several metabolic substrates and hormones (14, 36, 37, 40, 41, 48, 67, 75) and low resting metabolic rates (48). Compared to eumenorrheic sedentary women, luteally suppressed eumenorrheic athletes also display abnormal levels of these hormones (16, 36, 37, 41), but the magnitudes of these effects are less extreme than in amenorrheic athletes.

These metabolic abnormalities tell a consistent story of chronic energy and especially carbohydrate deficiency resulting in a reduction in glucose utilization, the mobilization of fat stores, the slowing of metabolic rate, and growth hormone resistance, with more extreme effects in amenorrheic athletes and less extreme effects in eumenorrheic athletes. All these abnormalities have been reproduced experimentally along with the disruption of LH pulsatility by restricting the energy availability of regularly menstruating, habitually sedentary women through dietary restriction alone, through exercise energy expenditure alone, and through a combination of both (42, 43). All these metabolic and reproductive effects have also been prevented in exercising women by increasing their dietary energy intake in compensation for their exercise energy expenditure (42, 43). Dietary supplementation also restores menstrual cycles in amenorrheic exercising monkeys (70) without any moderation of their exercise regimen. Therefore, exercise training appears to have no disruptive effect on LH pulsatility or menstrual cycles beyond the impact of its energy cost on energy availability (43, 70).

Regardless of whether low energy availability disrupts LH pulsatility through changes in metabolic substrates or hormones or both, the research cited above makes clear that this disruption does not depend on cognitive restraint of dietary intake or on the existence of a diagnosable eating disorder or on high scores on the Eating Attitudes Test or the Eating Disorder Inventory questionnaires. Athletes can also become luteally suppressed or amenorrheic without

restricting their diets by failing to increase their dietary energy intake in sufficient compensation for their exercise energy expenditure.

Summary

- Menstrual disorders in female athletes range from entirely asymptomatic luteal suppression to amenorrhea in which ovarian follicular development, ovulation and luteal function are completely absent.
- The prevalence of menstrual disorders is higher in female athletes than in non-athletes, and highest in endurance, esthetic and weight-class sports.
- All athletes with menstrual disorders need to be differentially diagnosed to ensure that potential medical conditions not caused by athletic training receive appropriate medical care (30, 52 Figure 2).
- In most athletes with menstrual disorders, reproductive development and function are disrupted by undernutrition.
- Exercise has no suppressive effect on reproductive function apart from its energy cost, and menstrual disorders can be prevented or reversed by increasing dietary intake in compensation for exercise energy expenditure.

C. Osteoporosis

Definition

Osteoporosis, the third leg of the Triad, is a degenerative disease of the skeleton usually associated with elderly postmenopausal women. However, numerous studies since 1984 have reported low bone density in young amenorrheic female athletes (11, 17, 20, 45, 47, 53). Restricted energy availability and subsequent suppression of bone formation plus the decrease in endogenous estrogen associated with amenorrhea (18, 32) result in a progressive decrease in

bone mass as bone resorption exceeds bone formation during bone remodeling. Over time this imbalance can lead to microarchitectural deterioration and increased skeletal fragility placing the athlete at an increased risk for fractures. While no one has identified the minimum numbers of normal cycles per year required to maintain bone mass, oligomenorrheic athletes and those with a history of oligomenorrhea also have bone density below average for their age (18).

Diagnosis

Other than fractures resulting from minimal trauma or repeated stress fractures, there are no obvious indications that bone loss may have occurred. There should, however, be a high level of suspicion in any case where an athlete has been amenorrheic for longer than six months, had numerous periods of oligomenorrhea, or had numerous stress fractures. The definitive method for determining the status of an athlete's skeletal system is Dual Energy X-ray Absorptiometry (DXA). The interpretation of the results is based on the normal distribution of bone mineral density (BMD) among young normal Caucasian women. The World Health Organization (WHO) has published the following definitions (74):

a. Normal: a BMD above -1 SD of the average BMD of young adult women

b Osteopenia (low bone mass): a BMD between -1 SD and -2.5 SD of young adult women

c. Osteoporosis: more than -2.5 below the average BMD of young adult women women.

Recently, the International Society for Clinical Densitometry (ISCD) has recommended that the bone density of adolescents and premenopausal women be compared to that of average women their own age (Z-Score) rather than using the criteria for postmenopausal women (T-Score) in determining risk for osteoporosis (33). For most amenorrheic athletes, the Z-Score will be the same as the T-Score, which is based on the average bone density for young adult women

and represents the distance in standard deviations from that average value. For those below age 20, the Z-Score is the only meaningful measure as they are still accumulating bone. Athletes with normal menstrual cycles generally have bone densities above average. Not only do amenorrheic athletes generally have a bone density below normal for their age, but they have failed to benefit from the positive effect that physical activity has on bone mass. The ISCD has recently defined a Z-Score of -2.0 or lower as "below the expected range for age" and a Z-Score above -2.0 as "within the expected range for age." These criteria are not suitable for amenorrheic athletes. Any DXA result below Z= -1.0 should be of concern to the athlete and her physician and the athlete should be referred for treatment as suggested in Appendix 7. A Z-Score of -2.5 or below for an amenorrheic athlete, age 20 and above, may be considered evidence of osteoporosis. Hypoestrogenic athletes with BMD in this area are at risk for fractures (22, 68, 69).

The interpretation of BMD values for premenopausal women may change as more data become available. Updates will be posted on the IOC web site. When no DXA is available for determining bone density, it is reasonable to expect that extended periods of amenorrhea, longer than six months, have had a negative effect on the athlete's skeleton. Every effort should be made to correct those conditions which have resulted in the cessation of menses.

Caution

1. The WHO criteria (74), which are based on the likelihood of fractures in Caucasian women, may or may not apply to other ethnic/racial groups where data relating BMD to fractures are not available. Nevertheless, it can serve as a general guideline in assessing the need for intervention.

2. The application of the WHO criteria to the results of other methods of assessing BMD such as ultrasound scans of the calcaneus or use of other peripheral devices is questionable for athletes even when device-specific cut-points are used. The problem with using less sophisticated devices

for measuring bone density in athletes is that they measure bone in the extremities. While they may be useful for average women, they can provide misleading information for athletes. Runners, for example, tend to have high bone density in the calcaneus but at the same time can have low bone density in the hip or spine. Rowers can have above average BMD in the forearm but low values in the hip. The higher density at the peripheral sites appears to be related to the site specific effect of bone loading for each sport (49).

Consequences

1. There are several studies reporting an increase in stress fractures among amenorrheic athletes (4, 34, 39).

2. The combination of amenorrhea and demanding workouts increases the likelihood of stress fractures. In one study (34), 100% of amenorrheic ballet dancers who practiced more than five hours per day had a stress fracture.

3. Premature osteoporotic type fractures in both the axial and appendicular skeleton have occurred in young athletes. On average, BMD at all skeletal sites is lower than average for amenorrheic athletes and anorexic women (53, 55).

4. Should the amenorrhea occur during adolescence, there is concern that these girls will not attain their biological potential for peak bone mass.

5. There are several studies (18, 21, 35) indicating that BMD does not reach normal-for-age levels even when menses resumes. This may place those athletes diagnosed with low bone mass or as osteoporotic prior to menopause at risk for premature osteoporotic fractures.

Treatment

1. It is vital to intervene within the first year of the onset of amenorrhea when bone loss is most rapid. At this time a bone density test will provide a baseline BMD to measure the effectiveness of the intervention.

2. As a pharmacological approach may have negative side effects, the first treatment of choice is to work with the amenorrheic or oligomenorrheic athlete to make changes in nutrition and training (as explained in the previous sections) to encourage resumption of normal menses.

a. The initial BMD will be useful in determining how much time can be spent in attempting to resume menses before using other therapy. If the BMD falls within the osteoporosis area, it is important to take action immediately to prevent further loss.

3. If menses does not resume, pharmacological therapy may be necessary to prevent serious bone loss. There are several options:

a. *Oral contraceptives*

Oral contraceptives are generally well accepted by athletes. Although there is still some question as to how effective OCs are in increasing BMD, they are generally prescribed as a means of preventing or slowing bone loss. One study (29) has reported an increase in the lumbar spine and total body BMD of amenorrheic athletes. A more recent study (54) also found an increase in total body BMD. More studies of longer duration with a larger sample size and BMD measurement at more skeletal sites are needed to determine if OCs can offset the negative effect of amenorrhea on bone.

b. *Nasal spray calcitonin*

One study (19) with a small number of subjects has shown this to be effective in preventing further decrease in BMD. If selected as a treatment, BMD should be retested after 12 months to check the effectiveness for individual athletes.

c. *Calcium*

There is no evidence that increasing calcium intake will prevent bone loss in amenorrheic athletes. However, an inadequate calcium intake is also a risk factor for bone loss. For amenorrheic athletes, the daily calcium intake should be at least 1500 mg/day as recommended for other hypoestrogenic women.

d. **WARNING**

Bisphosphonates and SERMs (Selective estrogen receptor modulators) have not been tested for safety with premenopausal women. The primary concern relative to bisphosphonates is their potential negative consequences during pregnancy. As reported in animal studies, their antiresorptive effect on bone interferes with the maternal need for calcium when the fetal demand is high. In addition, bisphosphonates do cross the placental and are stored in fetal bone with unknown long term consequences. Although these data are from animal studies, the use of bisphosphonates for women of childbearing age should be avoided. Studies of SERMS are less conclusive but do report toxicity effects at pharmacological doses. *At present, the use of bisphosonates and SERMS is contraindicated for premenopausal female athletes.*

There are a number of other pharmaceutical agents under investigation including some that may have the ability to reverse bone loss. When these become approved for use with premenopausal women, they will be added to this document.

None of the interventions for preventing bone loss will correct the effect of inadequate nutrition on bone formation. This is particularly important for adolescent athletes whose accrual of bone in these years will determine their peak bone mass.

Conclusions

Amenorrheic athletes are at risk for a decrease in bone mass which may be irreversible. Musculoskeletal injuries such as stress fractures appear to be more common in amenorrheic athletes. Longitudinal studies are needed to determine if these women are at risk for premature osteoporotic fractures as they age. Athletes need to be educated about the effect of amenorrhea on bone and the long-term consequences for their skeletal health.

Those interested in a broader discussion of the science underlying the recommendations in this document are referred to *The Female Athlete Triad Position Stand* published by the American College of Sports Medicine and available on their web site www.acsm.org.

References

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders,* 4th Ed. Washington, DC: American Psychiatric Association, 1994.

2. Augestad, L. B. and W. D. Flanders. Eating disorder behavior in physically active Norwegian women. *Scand. J. Med. Sci. Sports*. 12:248-255, 2002.

3. Bachmann, G. A. and E. Kemmann. Prevalence of oligomenorrhea and amenorrhea in a college population. *Am. J. Obstet. Gynecol.* 144:98-102, 1982.

4. Barrow, G W, and S. Saha. Menstrual irregularity and stress fractures in collegiate female distance runners. *Am. J. Sports Med*., 16:209-216, 1988.

5. Beals, K. A. and M. M. Manore. The prevalence and consequences of subclinical eating disorders in female athletes. *Int. J. Sport Nutr.* 4:175-195, 1994.

6. Beals, K. A. and M. M. Manore. Disorders of the female athlete triad among collegiate athletes. *Int. J. Sport Nutr. Exerc. Metab*. 12:281-293, 2002.

7. Benson, J. E., Y. Allemann, G. E.Theintz, et al. Eating problems and calorie intake levels in Swiss adolescent athletes. *Int. J. Sport Med*. 11:249-252, 1990.

8. Brownell, K. D.and J. Rodin. Prevalence of eating disorders in athletes. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society,* K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea & Febiger, 1992, pp. 128-145.

9. Byrne, S. and N. McLean. Eating disorders in athletes: A review of the literature. *J. Sci. Med. Sport.* 4:145-159, 2001.

10. Calabrese, L. H., D. T. Kirkendall, M. Floyd, et. al. Menstrual abnormalities, nutritional patterns, and body composition in female classical ballet dancers. *Phys. Sportsmed*.11(2):86-98, 1983.

11. Cann, C.E., Martin, M.C., Genant, H.K., and Jaffe, R.B. Decreased spinal mineral content in amenorrheic women. *JAMA* 251:626-629, 1984.

12. Chumlea WC, Schubert CM, Roche AF et al. Age at menarche and racial comparisons in U.S. girls. *Pediatrics* 2003;111(1):110-3

13. Davis, C. and M. Cowles. A comparison of weight and diet concerns and personality factors among female athletes and non-athletes. *J. Psychosom. Res*. 33:527-536, 1989.

14. De Souza, M. J., M. S. Maguire, C. M. Maresh, W. J. Kraemer, K. R. Rubin, and A. B. Loucks. Adrenal activation and the prolactin response to exercise in eumenorrheic and amenorrheic runners. *J. Appl. Physiol*. 70:2378-2387, 1991.

15. De Souza, M. J., B. E. Miller, A. B. Loucks, et al. High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. *J. Clin. Endocrinol. Metab.* 83:4220-4232. 1998.

16. De Souza, M. J., J. Van Heest, L. M. Demers, and B. L. Lasley. Luteal phase deficiency in recreational runners: evidence for a hypometabolic state. *J. Clin. Endocrinol. Metab*. 88:337-346, 2003.

17. Drinkwater, B.L. Amenorrhea, body weight, and osteoporosis. In: *Eating, Body Weight, and Performance in Athletes.* K.D. Brownell, J. Rodin, and J.H. Wilmore (Eds.) Philadelphia: Lea & Febiger, 1992, pp. 235-247.

18. Drinkwater, B. L., B. Bruemner, and C. H. Chesnut III. Menstrual history as a determinant of current bone density in young athletes. *JAMA*, 263:545-548, 1990.

19. Drinkwater, B. L., N. L. Healy, M. L. Rencken, D. Fontana, and C.H. Chesnut III. Effectiveness of nasal spray calcitonin in preventing bone loss in young

amenorrheic athletes. *J. Bone Min. Res.* 8:S264, 1993.

20. Drinkwater B. L., K. Nilson, C. H. Chesnut III, W. Bremner, S. Shainholtz, and M. Southworth. Bone mineral content of amenorrheic and eumenorrheic athletes. *New Eng. J. Med*. 311:277-281, 1984.
21. Drinkwater B. L., K. Nilson, S. Ott, and C. H. Chesnut III. Bone mineral density following resumption of menses in amenorrheic athletes. *JAMA* 256:380-382, 1986.
22. Dugowson, C. E., B. L.Drinkwater, and J. M. Clark. Nontraumatic femur fracture in an oligomenorrheic athlete. *Med. Sci. Sports Exerc.* 23:1323-1325, 1991.
23. Dusek, T. Influence of high intensity training on menstrual cycle disorders in athletes. *Croat. Med. J.* 42:79-82. 2001.
24. Garfinkel, P. E and D. M. Garner. *Anorexia Nervosa: A Multidimensional Perspective.* New York: Brunner/Mazel, 1982.
25. Garner, D. M., P. E. Garfinkel, W. Rockert, and M. P. Olmsted. A prospective study of eating disturbances in the ballet. *Psychother. Psychosom.* 4:170-175, 1987.
26. Garner, D. M. Eating Disorder Inventory-2: Professional Manual. Odessa, FL; 1991.
27. Harris, R. T. Bulimarexia. *Ann. Intern. Med*. 99:800-807, 1983.
28. Hausenblas, H. A. and A. V. Carron. Eating disorder indices and athletes: An integration. *J. Sport. Exerc. Psychol.* 21:230-258, 1999.
29. Hergenroeder, A.C., Smith, E.O., Shypailo, R., Jones, L.A., Klish, W.J. and K. Ellis. Bone mineral changes in young women with hypothalamic amenorrhea

treated with oral contraceptives, medroxyprogesterone, or placebo over 12 months. *Am. J. Obstet. Gynecol.* 176:1017-1025, 1997.

30. Hoffman, B, and K. D.Bradshaw. Delayed puberty and amenorrhea. *Seminars in Reproductive Medicine* 21:353-362, 2003.

31. Hsu, L. K. G. *Eating Disorders.* New York: Guilford Press, 1990.

32. Ilhe R., and A.B. Loucks. Dose-response relationships between energy availability and bone turnover in young exercising women. *J. Bone Miner. Res*. 19:1231-1240, 2004.

33. International Society for Clinical Densitometry Position Development Conference. Diagnosis of osteoporosis in men, premenopausal women, and children. *J. Clin. Densitom.*7:17-26, 2004.

34. Kadel N.J., C. C. Teitz, and R. A. Kronmal. Stress fractures in ballet dancers. *Am. J. Sports Med.* 20:455-449, 1992.

35. Keen, A. D. and B. L. Drinkwater. Irreversible bone loss in former amenorrheic athletes. *Osteoporosis International* 7:311-315, 1997

36. Laughlin, G. A., and S. S. C. Yen. Nutritional and endocrine-metabolic aberrations in amenorrheic athletes. *J. Clin. Endocrinol. Metab*.81: 4301-4309, 1996.

37. Laughlin, G. A. and S. S. C. Yen. Hypoleptinemia in women athletes: absence of a diurnal rhythm with amenorrhea. *J. Clin. Endocrinol. Metab*. 82: 318-321, 1997.

38. Leon, G. R. Eating disorders in female athletes. *Sports Med*. 4:219-227, 1991.

39. Lloyd, T., S. J. Triantafyllou, E. R. Baker, et al. Women athletes with menstrual irregularity have increased musculoskeletal injuries. *Med. Sci. Sports Exerc.*, 18:374-379, 1986.

40. Loucks, A. B., G. A. Laughlin, J. F. Mortola, L. Girton, J. C. Nelson, and S. S. C.Yen. Hypothalamic-pituitary-thyroidal function in eumenorrheic and *amenorrheic athletes. J. Clin. Endocrinol. Metab.* 75: 514-518, 1992.

41. Loucks, A. B., J. F. Mortola, L. Girton, and S. S. C.Yen. Alterations in the hypothalamic-pituitary-ovarian and the hypothalamic-pituitary-adrenal axes in *athletic women. J. Clin. Endocrinol. Metab.* 68:402-411, 1989.

42. Loucks, A. B. and J. R. Thuma. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. *J. Clin. Endocrinol. Metab.* 88:297-311, 2003.

43. Loucks, A. B., M. Verdun, and E. M. Heath. Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *J. Appl. Physiol*. 84:37-46, 1998.

44. Manore, M. M. Dietary recommendations and athletic menstrual dysfunction. *Sports Med.* 32:887-901, 2002.

45. Marcus R., C., Cann, P. Madvig, et al. Menstrual function and bone mass in elite women distance runners. *Ann. Intern. Med.* 102:158-163, 1985.

46. Matsumoto SN, Nogami Y, and Ohkuri, S. Statistical studies of menstruation: A criticism on the definition of normal menstruation. *Gunma J. Med. Sci.* 1962;11:294-318

47. Myburgh C., L.K. Bachrach, B. Lewis, et al. Low bone mineral density at axial and appendicular sites in amenorrheic athletes. *Med. Sci. Sports Exerc.* 25:1197-1202, 1993.

48. Myerson, M., B. Gutin, M. P. Warren, M.T. May, I. Contento, M. Lee, F. X. PiSunyer, R.N. Pierson, Jr. and J. Brooks-Gunn. Resting metabolic rate and energy balance in amenorrheic and eumenorrheic runners. *Med. Sci. Sports*

Exerc. 23:15-22, 1991.

49. Nattrass, S. M. and B. L. Drinkwater. Does mechanical loading of peripheral skeletal sites affect prediction of axial BMD? *Bone* 23 (5), S315, 1998.
50. O'Connor, P. J., R. D. Lewis, and E. M. Kirchner. Eating disorder symptoms in female college gymnasts. *Med. Sci. Sports Exerc*. 27:550-555, 1995.
51. Pettersson, F., H. Fries, and S. J. Nillius. Epidemiology of secondary amenorrhea. I. Incidence and prevalence rates. *Am. J. Obstet. Gynecol.* 117:80-86, 1973.
52. Practice Committee of the American Society for Reproductive Medicine. Current evaluation of amenorrhea. *Fertil. Steril.* 82:266-272, 2004.
53. Rencken, M., C. H. Chesnut III, and B. L. Drinkwater. Decreased bone density at multiple skeletal sites in amenorrheic athletes. *JAMA* 276:238-240, 1996.
54. Rickenlund, A., K. Carlstrom, B. Ekblom, et al. Effects of Oral Contraceptives on Body Composition and Physical Performance in Female Athletes. *J. Clin. Endocrinol. Metab.* 89:4364-4370, 2004.
55. Rigotti, N.A., S. R. Nussbaum, D. B. Herzog, and R. M. Neer. Osteoporosis in women *with anorexia nervosa. N. Engl. J. Med.* 311:1601-1606, 1984.
56. Sanborn, C. F., B. H. Albrecht, and W. W. Wagner. Athletic amenorrhea: lack of association with body fat. *Med. Sci. Sports Exerc.* 19:207-212, 1987.
57. Shisslak, C. M., M. Crago, and L. Estes. The spectrum of eating disturbances. *Int. J. Eat. Disord.* 18:209-219, 1995.
58. Singh, K. B. Menstrual disorders in college students. *Am. J. Obstet. Gynecol.* 140:299-302, 1981.

59. Smolak, L., S. Murnen, and A. E. Ruble. Female athletes and eating problems: A meta-analysis. *Int. J. Eat. Disord*. 27:371-380, 2000.

60. Sundgot-Borgen, J. Eating disorders, energy intake, training volume and menstrual function in high-level modern rhythmic gymnastic gymnasts. *Int. J. Sport Nutr*. 2:100-109, 1996.

61. Sundgot-Borgen, J. Risk and trigger factors for the development of eating disorders in female elite athletes. *Med. Sci. Sports Exerc*. 26:414-419, 1994.

62. Sundgot-Borgen, J. Disordered Eating. In: *The Female Athlete,* M. L. Ireland and E. Nattiv (Eds.). Philadelphia: Saunders, 2002, pp. 237-248.

63. Sundgot-Borgen, J. and S. Larsen. Pathogenic weight-control methods and self-reported eating disorders in female elite athletes and controls. *Scand. J. Med. Sci. Sports.* 3:150-155, 1993.

64. Sundgot-Borgen, J. and M. K. Torstveit. Prevalence of eating disorders in elite athletes is higher than in the general population. *Clin. J. Sport Med.* 14:25-32, 2004.

65. Thompson, R. A. and R. T. Sherman. *Helping Athletes with Eating Disorders*. Champaign, IL: Human Kinetics, 1993.

66. Thompson, R. A. and R. T. Sherman. "Good athlete" traits and characteristics of anorexia nervosa: Are they similar? *Eating Disorders.* 7:181-190, 1999.

67. Thong, F. S., C. McLean, and T. E. Graham. Plasma leptin in female athletes: relationship with body fat, reproductive, nutritional, and endocrine factors. *J. Appl. Physiol*. 88:2037-2044, 2000.

68. Warren, M. P., J. Brooks-Gunn, L. H. Hamilton, L. F. Warren, and W. G. Hamilton. Scoliosis and fractures in young ballet dancers. *New Eng. J. Med.*

314:1348-1353, 1986.

69. Wilson, J. H. and R. L. Wolman. Osteoporosis and fracture complications in an amenorrheic athlete. *Br. J. Rheumatol*. 33:480-481, 1994.

70. Williams, N. I., D. L. Helmreich, D. B. Parfitt., A.L Caston-Balderraman, and J.L. Cameron. Evidence for a causal role of low energy availability in the induction of menstrual cycle disturbances during strenuous exercise training. *J. Clin. Endocrinol. Metab*. 86:5184-5193. 2001.

71. Wilmore, J. H. Eating and weight disorders in the female athlete. *Int. J. Sport Nutr*. 1:104-117, 1991.

72. Wilson, G. T. The controversy over dieting. In: *Eating Disorders and Obesity: A Comprehensive Handbook,* 2nd Ed., C. G. Fairburn and K. D. Brownell (Eds.). New York: The Guilford Press, 2002, pp. 93-97.

73. Wilson, G. T and K. L. Eldredge. Pathology and development of eating disorders: Implications for athletes. In: *Eating, Body Weight and Athletic Performance: Disorders of Modern Society,* K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea & Febiger, 1992, pp. 115-127.

74. World Health Organization (WHO) Study Group. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis: Report of WHO study group technical report series 843. Geneva, Switzerland: *WHO technical report series* 843:6, 1994.

75. Zanker, C. L. and I. L. Swaine. Bone turnover in amenorrhoeic and eumenorrhoeic women distance runners. *Scand. J. Med. Sci. Sports* 8:20-26, 1998.

Appendix

1. Diagnostic Criteria for Anorexia Nervosa.

2. Diagnostic Criteria for Bulimia Nervosa

3. Diagnostic Criteria for EDNOS.

4. Signs, Symptoms, and Medical Complications of Eating Disorders

5. Questions that can be included in the conversation

6. Treatment of Athletes with Disordered Eating

7. Suggestions for dealing with the amenorrheic athlete

Appendix 1. Diagnostic Criteria for Anorexia Nervosa.

A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., 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)
B. Intense fear of gaining weight or becoming fat, even though underweight
C. 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
D. In post-menarcheal females, amenorrhea, i.e., the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration.)
Specify types: Restricting type: During the episode of anorexia nervosa, the person has not regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics or enemas) Binge eating/purging type: During the current episode of anorexia nervosa, the person has regularly engaged in binge eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics or enemas)

Appendix 2. Diagnostic Criteria for Bulimia Nervosa.

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following: (1) eating, in a discrete period of time (e.g., Within any 2 hour period), an amount of food that is definitely larger than most people would eat during a similar period of time in similar circumstances; and, (2) a sense of lack of control over eating during the episode (e.g., A feeling that one cannot stop eating or control what or how much one is eating).
B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as: self-induced vomiting; misuse of laxatives; diuretics, enemas, or other medications; fasting; or excessive exercise.
C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for three months.
D. Self-evaluation is unduly influenced by body shape and weight.
E. The disturbance does not occur exclusively during episodes of anorexia nervosa.
Specify types: Purging type: The person regularly engages in self-induced vomiting or the misuse of laxatives, diuretics or enemas Non purging type: The person uses other inappropriate compensatory behaviors, such as fasting or excessive exercise, but does not regularly engage in self-induced vomiting or the misuse of laxatives, diuretics or enemas

Appendix 3. Diagnostic Criteria for EDNOS.

A. For females, all of the criteria for anorexia nervosa are met except that the individual has regular menses.
B. All of the criteria for anorexia nervosa are met except that, despite significant weight-loss, the individual's current weight is in the normal range
C. All of the criteria for bulimia nervosa are met except that the binge eating and inappropriate compensatory mechanisms occur at a frequency of less than twice a week or for a duration of less than 3 months
D. The regular use of inappropriate compensatory behavior by an individual of normal body weight after eating small amounts of food (e.g., self-induced vomiting after the consumption of two cookies)
E. Repeatedly chewing and spitting out, but not swallowing, large amounts of food
F. Binge-eating disorder: recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviors characteristic of bulimia nervosa

Appendix 4. Signs, Symptoms, and Medical Complications of Eating Disorders

- **Orofacial**
  - Perimolysis[b]
  - Dental caries[b]
  - Cheilosis[b]
  - Enlargement of the parotid gland[b]
- **Cardiovascular**
  - Postural and nonpastural hypotension
  - Acrocyanosis
  - Electrocardiographic abnormalities: low voltage, prolonged QT interval, prominent U waves
  - Sinus bradycardia
  - Atrial and ventricular arrhythmias
  - Left ventricular changes: decreased mass, decreased cavity size
  - Mitral-valve prolapse
  - Cardiomyopathy (due to ipecac poisoning)
- **Gastrointestinal**
  - Esophagitis hematemesis (including the Mallory-Weiss syndrome) [b]
  - Delayed gastric emptying
  - Decreased intestinal motility
  - Constipation
  - Rectal prolapse
  - Gastric dilatation and rupture[b]
  - Abnormal results on liver-function tests
  - Elevated serum amylase level
- **Endocrine and Metabolic**
  - Hypokalemia (including hypokalemic nephropathy)
  - Hyponatremia, (rarely) hypernatremia
  - Hypomagnesemia
  - Hyperphosphatemia
  - Hypoglycemia
  - Hypothermia
  - Euthyroid sick syndrome
  - Hypercortisolism, elevated free cortisol level in urine
  - Low serum estradiol level
  - Decreased serum testosterone level
  - Amenorrhea, oligomenorrhea
  - Delay in puberty
  - Arrested growth
  - Osteoporosis
  - Stress fractures
  - Lipid abnormalities
  - Obesity [b]
- **Renal**
  - Renal calculi
- **Reproductive**
  - Infertility
  - Insufficient weight gain during pregnancy
  - Low-birth-weight infant
- **Integumentary**
  - Dry skin and hair
  - Hair loss
  - Lanugo
  - Yellow skin due to hypercarotenemia
  - Hand abrasions
- **Neurologic**
  - Peripheral neuropathy
  - Reversible cortical atrophy
  - Ventricular enlargement
- **Hematologic**
  - Anemia, leucopenia, neutropenia,thrombocytopenia
- **Fluids and electrolytes**
  - Dehydration
  - Edema
  - Electrolyte abnormalities
  - Hypokalemia
  - Muscle cramps
  - Metabolic alkalosis
- **Thermoregulation**
  - Hypothermia[a]
- **Others**
  - Significant weight loss (beyond that necessary for optimal sport performance) [a]
  - Frequent and often extreme weight fluctuations[b]
  - Low weight despite eating large volumes[b]
  - Fatigue (beyond that normally expected in training or competition)
  - Muscle weakness
  - More training (aerobic type) than required for her performance enhancement

[a]Especially for anorexia nervosa, [b]Especially for bulimia nervosa. From Sundgot-Borgen J. Disordered Eating. In Ireland ML & Nattiv E (eds): The Female Athlete. Philadelphia, Saunders, 2002, pp 237-248.

Appendix 5. Questions that can be included in the conversation

Questions			
Regarding food	**Regarding weight**	**Regarding the menstruation**	**Regarding the training and injuries**
How do you feel about food? Do you have a "relaxed" relationship with food?	What has been your highest and lowest weight during the last year?	When did you start to menstruate?	Have you changed your training regime (type, load, or intensity)?
What is your eating pattern?*	What do you consider to be your competition weight/match weight?	Has your menstrual cycle been regular after menarche?	Do you do other forms of training than that related to your specific sport?
How many meals do you eat per day?	Have you reduced your weight lately? What did you do to achieve the weight loss?	What has been the longest time period without menstrual bleedings?	Have you experienced a stress fracture or a regular fracture?
Do you have any sort of food you try to avoid? (forbidden food?)	Are you satisfied with your present weight?	When did you have your last menstrual bleeding?	
What did you eat and drink yesterday?	Do other persons have opinions about your weight?	How do you feel about your menstrual cycle?**	
Questions about use of "purging methods" (ask about the past)		Do you use or have you used oral contraceptives?	

*It can be hard for a person with DE to recall what she ate. A person with anorexia nervosa avoids fat and is usually vegetarian. The eating pattern is characterized by the same restrictive intake of "YES-food" every day. A person with bulimia nervosa constantly tries to "avoid the calorie intake" and binge eats in the afternoon and evening.
**An athlete with DE prefers the absence of menstruation (it is considered a failure to have a percentage of body fat required for having regular cycles).

Appendix 6. Treatment of Athletes with Disordered Eating

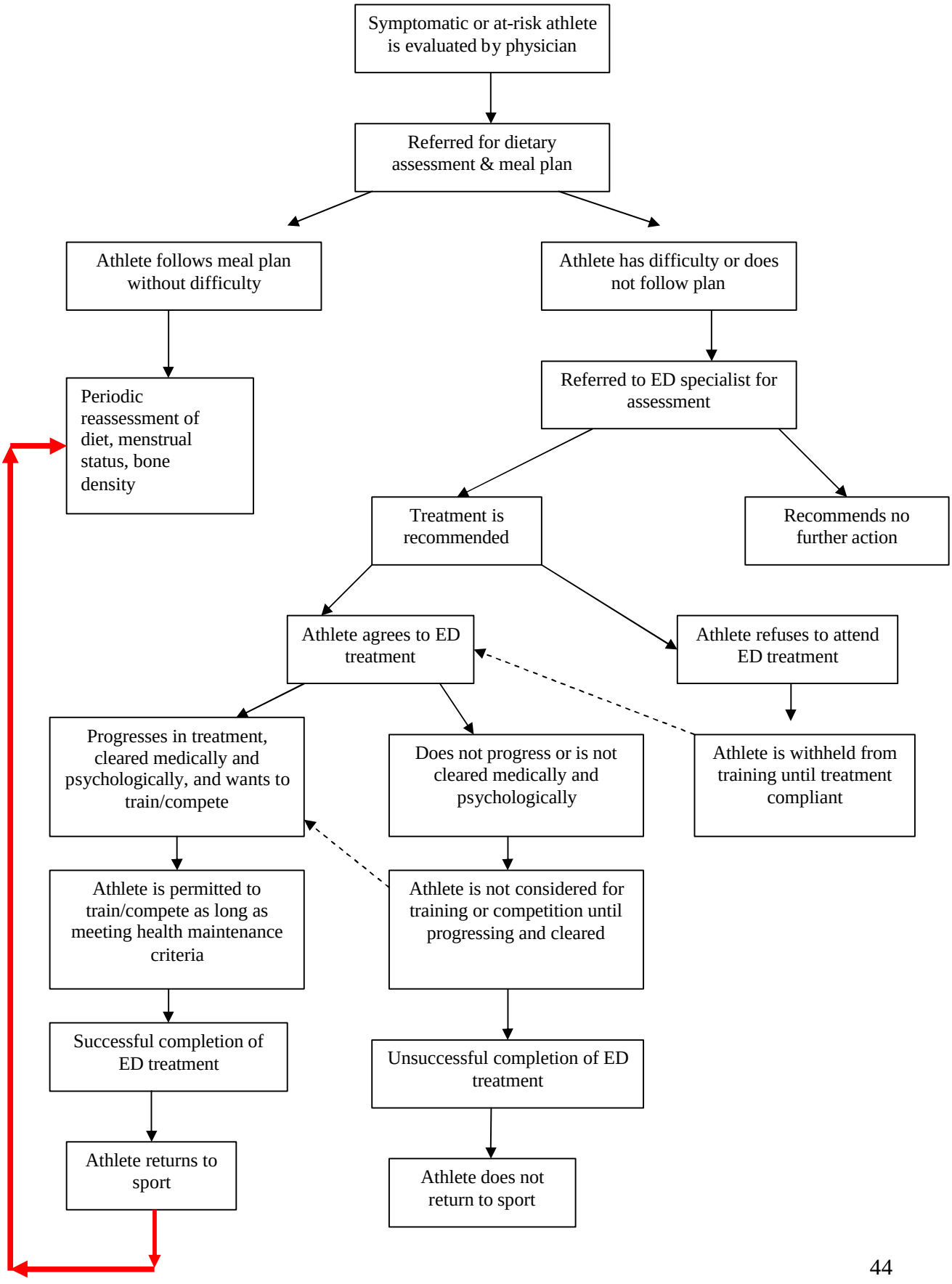
TYPES OF TREATMENT	CONTENTS
Individual psychotherapy	• The therapist works with the DE athlete and intends to: Determine the nature of the individual's eating difficulties and how they might be most effectively changed. Implement a change process. Teach the athlete to deal with how her sport or sport participation may be contributing to the maintenance of the DE.
Group therapy	• The athlete is part of a group made up of other ED athletes. Athletes discover that others have a similar problem. Gives the individual a support group that understands her feelings and eating problem. Provides a safe environment for the athlete to practice the new skills and attitudes she has learned.
Family therapy	• Includes the patient and her immediate family. The family is the focus of treatment A goal is to modify maladaptive family interactions, attitudes, and dynamics to decrease the need for, or the function of, the DE in the family.
Nutritional counseling	• Often part of a multimodal treatment approach. Athletes with DE do not remember what constitutes a balanced meal or "normal" eating. The dietician's primary roles involve providing nutritional information and assisting in meal planning.
Pharmacotherapy	• Can be useful in some cases, especially with patients with bulimic behaviors.

From Sundgot-Borgen J. Disordered Eating. In Ireland ML & Nattiv E (eds): The Female Athlete. Philadelphia, Saunders, 2002, pp 237-248.

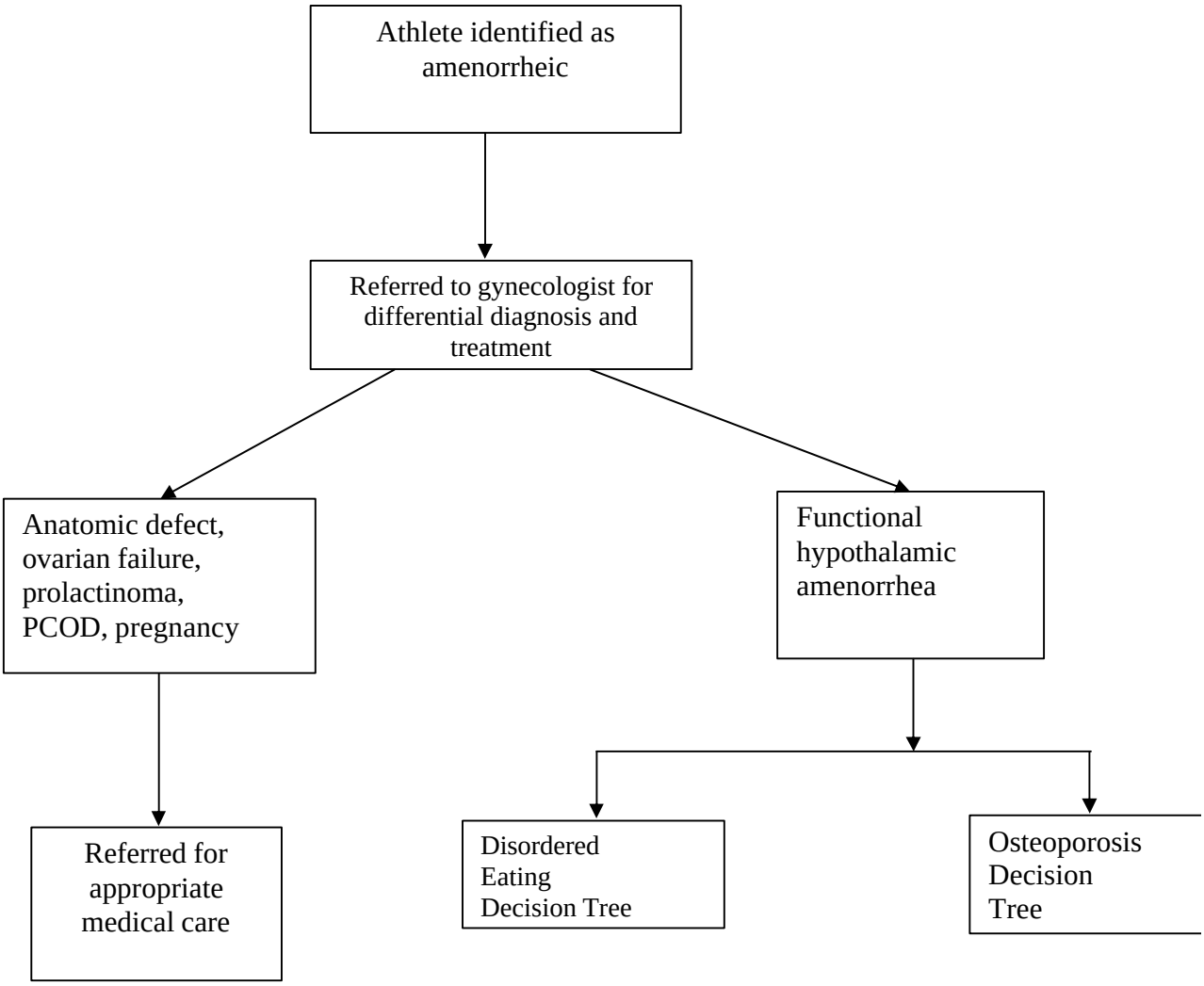
Appendix 7. Suggestions for dealing with the amenorrheic athlete:

Athletes at risk for the medical conditions discussed in the previous sections may be found at any of the various stages of the Triad. One or all of the following Decision Trees may be useful in deciding how to help the athlete. For example, the Disordered Eating Decision Tree would be appropriate for an athlete with poor nutrition or disordered eating who is not yet amenorrheic. Once an athlete becomes amenorrheic, the steps outlined in all three Decision Trees will be helpful in deciding on a course of action. In most cases, the amenorrheic athlete will be progressing through the Disordered Eating Decision Tree at the same time she is undergoing evaluation of her amenorrhea and bone density as described in the Amenorrhea Decision Tree and Osteoporosis Decision Tree.

Disordered Eating Decision Tree



Amenorrhea Decision Tree



Osteoporosis Decision Tree