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Diet and Exercise-Induced Menstrual Dysfunction in the Female Athlete

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DIET AND EXERCISE-INDUCED MENSTRUAL DYSFUNCTION IN THE FEMALE ATHLETE

by

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This Independent Study, submitted by Roberta Jo Lindaas Welp in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

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Signature Roberta Welp

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TABLE OF CONTENTS

LIST OF TABLES

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"It's not the people that lift you up that matter, but the people that keep you up."—Linden Kohtz.

ABSTRACT

In recent years there has been an overwhelming increase in the number of females participating in athletic activities. As members of the sports medicine team, physical therapists must be able to recognize afflictions unique to the female athlete. A cluster of increasingly common disorders has been titled the "female athlete triad." By definition, it is the inter-relatedness of disordered eating, amenorrhea, and osteoporosis. Clinically, it may include other factors such as excessive exercise, various degrees and forms of menstrual dysfunction, and additional issues such as fractures, scoliosis, and general musculoskeletal injury.

The purpose of this study is to provide information to physical therapists and students regarding components of the 'triad', related disorders, secondary pathology, and data concerning evaluation and treatment. By identifying problems early in the course of their progression, physical therapists can facilitate timely and appropriate intervention and prevent or minimize the deleterious effects of the 'triad' and associated disorders.

CHAPTER I

INTRODUCTION

In 1972, legislation was passed stating that no person in the United States shall, on the basis of sex, be subjected to discrimination under any educational program or activity receiving federal assistance. Otherwise known as Title IX, this document is thought to be largely responsible for the dramatic increase in the number of girls and women participating in sports and exercise.[1]

While it is widely accepted that regular physical activity is beneficial in disease prevention, health promotion, and overall well-being, all females are at risk for the development of one or more components of a triad of medical disorders described as the "female athlete triad". Alone, each component of the triad can be worrisome. In combination, they can seriously impact an athlete's health and even be potentially fatal.[1]

The female athlete triad is, by definition, the inter-relatedness of disordered eating, amenorrhea, and osteoporosis.[1,2] However, the "triad" encompasses far more than three distinct entities. The first component, disordered eating, refers to a spectrum of abnormal behaviors ranging from mild dietary abnormalities to the extreme diagnoses of anorexia nervosa and bulimia.[2] It may be influenced by factors outside the realm of its definition, such as caloric and nutritional intake, body weight, body composition, and excessive exercise.

Two chapters are utilized to address these topics some-what individually; one on diet and the other on exercise.

The second element of the triad, as mentioned previously, is amenorrhea. Its position in the triad implies that it is the direct result of disordered eating as well as a contributing factor to the development of osteoporosis. Instead of limiting this paper's content to amenorrhea, the author attempts to provide a thorough explanation of the various forms of menstrual dysfunction in relation to the female athlete.

It is also clear that osteoporosis is not the sole secondary pathology of menstrual dysfunction. The consequences of long-term menstrual dysfunction may further include an increased frequency of stress fractures,[3-11] musculoskeletal injuries,[7] and scoliosis.[8]

The purpose of this study is to provide information to physical therapists and students regarding components of the 'triad', related disorders, secondary pathology, and data concerning evaluation and treatment. By identifying problems early in the course of their progression, physical therapists can facilitate timely and appropriate intervention and prevent or minimize the deleterious effects of the 'triad' and associated disorders.

CHAPTER II

MENSTRUAL DYSFUNCTION

Many of the studies on menstrual dysfunction use terminology regarding a woman's menstrual cycle, related endocrinology, and categories of classification. In order to understand the studies presented as well as their results, it is necessary to have an adequate background in these topics.

Menstrual Cycle

Hormonal activity varies during the menstrual cycle and can be divided into two parts; the *follicular phase* and the *luteal phase*, separated by ovulation. The follicular phase is the stage of growth and maturation of ovarian follicles, one of which becomes the ovulatory follicle. The luteal phase is associated with the presence of a corpus luteum,[12] a small yellow body that develops within the ruptured ovarian follicle.[13] It secretes estradiol and progesterone, which induce the proliferation and secretory changes that occur within the endometrial glands. In the absence of a fertilized ovum, the corpus luteum regresses. The decrease in estradiol and progresterone result in the shedding of the endometrium in the form of menstrual flow.[12]

Regular cycle length differs from source to source but it is typically said to be between 25 and 30,[12] or 23 and 35 days.[14] The follicular phase begins on the first day of menstrual bleeding and normally lasts 12 to 16 days with the luteal

phase persisting 10 to 16[12] or 14 ± 2 days[14] post-ovulation. Regression of the corpus luteum is typically 9 to 11 days after ovulation.[12]

Endocrinology of Menstruation

Gonadotropin-releasing hormone (GnRH, or luteinizing hormone-releasing hormone [LHRH]) is a decapeptide secreted by hypothalamic neurons. It is known to stimulate the synthesis and pulsatile secretion of both *luteinizing hormone* (LH) and *follicle stimulating hormone* (FSH) by the pituitary gonadotroph cells.[12,14] Its secretion is influenced positively by an increase in hypothalamic neurotransmitters such as the catecholemines norepinephrine (NE) and epinephrine (E) and negatively by an increase in endogenous opioid peptides such as β-endorphin. Pulsatile secretion of GnRH appears to be necessary for the maintenance of pituitary responsiveness and normal gonadotropin synthesis and secretion. In addition, data indicates that the frequency of GnRH pulses must fall within a certain narrow range in order to be effective.[12]

LH and FSH are secreted by gonadotroph cells of the anterior pituitary gland. LH is released into circulation in a series of pulses and is thought to be the direct result of pulsatile GnRH release. FSH is secreted in a similar manner. However, it is unknown if its pulses are the direct result of GnRH release. This is due to FSH's long half-life, which is thought to be greater than the interval between pulses.[12] LH and FSH act in concert to produce ovarian follicular maturation, ovulation, and secretion of estradiol and progesterone.[12,14]

The term *estrogen* refers to any natural or artificial substance that induces estrogenic activity. More specifically, it includes the female sex hormones *estradiol* and *estrone*, both of which are produced by the ovaries. Estrone is the least active of the two.[13] The possibility that estradiol inhibits GnRH secretion is supported by data indicating that concentrations of GnRH were increased after ovariectomy.[15] Supportive data further suggests that estradiol decreases the pulse frequency of GnRH.[12] Other investigators have not found these increases following removal of the ovaries.[16] Some have shown that estradiol actually stimulates GnRH secretion. This is evident by an increase in the concentration of GnRH during the LH surge which is also a time of elevated estradiol levels. In addition, plasma estradiol levels increase during the late follicular phase of the menstrual cycle as does the GnRH pulse frequency.[16] From this information, it has be established that the actions of estradiol are time and dose dependent. The inhibitory actions of estradiol remain uncertain.[12]

Progesterone inhibits GnRH secretion by decreasing the frequency of its pulsatile release. The effect is seen as a reduced LH pulse frequency during the luteal phase of the menstrual cycle.[12] In certain circumstances, progesterone can also increase LH secretion, as seen with administration to estrogen-replaced postmenopausal women or ovariectomized animals.[17]

Together, estrogen and progesterone act to augment gonadotroph responsiveness to GnRH. The combined effects are crucial in the production of mid-cycle LH and FSH surge. In addition, progesterone facilitates and prolongs the positive feedback effects of estradiol.[12]

Menstrual Dysfunction Classification

A woman classified as *eumenorrhic* is "normal," fulfilling guidelines explained in the "menstrual cycle" portion of this chapter. Menstrual irregularities common to the female athlete include luteal phase deficiency and anovulation. Anovulation can be further categorized as being either primary amenorrhea, secondary amenorrhea, or oligomenorrhea.[12] Each of these will be outlined to facilitate understanding of the chapters that follow. It should be noted that the actual mechanisms leading to the development of each dysfunction remain unknown.[1,14]

Luteal phase deficiency, as its name implies, is characterized by decreased luteal phase durations and marked progesterone deficits.[14,18] If the time between ovulation and menses is less than 8 days, this denotes what is called a *short luteal phase*. An *inadequate luteal phase* is one of normal duration with subnormal progesterone.[12] A person with luteal phase deficiency may observe a decrease in total cycle length (21 to 23 days)[19] but more frequently her cycle length is normal, causing this irregularity to often go undetected. Some researchers[14] postulate that luteal phase deficiency eventually progresses to amenorrhea. Others state that it is a consistent menstrual pattern for many active women.[20]

Women who experience endometrial shedding more frequently than every 21 days or at intervals of 35 days to 4 months most likely have chronic *anovulation*. They are able to produce estrogen, but not progesterone. Anovulation falls into one of three categories. *Primary amenorrhea* is the

absence of menarche by age 16.[19] It is also commonly called *delayed menarche*. *Secondary amenorrhea*, more commonly referred to as *amenorrhea*, is the cessation of menses a period of time after menarche. Diagnostically, it is the absence of 3 to 12 consecutive menstrual periods. Finally, *oligomenorrhea* refers to long cycles with 35 to 150 days between bleeding.[14]

Factors Affecting Menstrual Dysfunction

Normal puberty in both sexes is characterized by marked maturational events spanning several years. These changes, which prepare the body for reproduction, are known to be affected by environmental factors including nutrition, body weight, stress, altitude, and exercise.[21] The content of this paper will discuss menstrual dysfunction as it relates to nutrition, weight, and exercise. Although it has been determined that weight loss-related amenorrhea and exercise-induced amenorrhea have similar endocrine profiles (suggesting that they have similar hypothalamic mechanisms),[12,22] many studies differentiate between the two forms. Therefore, Chapter III will address how nutrition and weight affect menstruation while Chapter IV will examine the effects of exercise.

CHAPTER III

DIET-INDUCED MENSTRUAL DYSFUNCTION

The information that follows examines the relationship of diet and menstrual dysfunction. It includes discussions related to dietary patterns, caloric intake, body weight, body composition, and the diagnoses of anorexia nervosa and bulimia.

Disordered Eating in the Female Athlete

The phrase "disordered eating", in contrast to the more familiar terminology of "eating disorders," refers to a spectrum of abnormal behaviors ranging from mild dietary abnormalities to the extreme diagnoses of anorexia nervosa and bulimia.[2] Included within this classification are behavioral abnormalities such as: binging; purging; binging and purging; food restriction; prolonged fasting; use of diet pills, diuretics, or laxatives; other abnormal eating behaviors; and abnormal thought patterns such as preoccupation with food, dissatisfaction with one's body, fear of becoming fat, and a distorted body image.[1] The term "disordered eating" has been chosen because it is important to address problematic eating patterns of any degree, considering that not all athletes meet the strict criteria of anorexia or bulimia (see Tables 1. and 2.).[1,23] None-the-less, disordered eating puts them at risk for developing secondary

Table 1.—Diagnostic Criteria for Anorexia Nervosa[23]
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 postmenarcheal 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 type*: **Restricting Type:** during the current 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)

Table 2.—Diagnostic Criteria for Bulimia Nervosa[23]
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 and under similar circumstances (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 3 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 type:* **Purging Type:** during the current episode of Bulimia Nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas **Nonpurging Type:** during the current episode of Bulimia Nervosa, 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 misuse of laxatives, diuretics, or enemas

problems such as menstrual dysfunction, osteoporosis [2] or even death.[24] Furthermore, lesser forms of disordered eating can progress into full-blown eating disorders. [2]

Prevalence

A study of female college athletes participating in a variety of sports reported 32 percent of individuals as having disordered patterns of eating, with at least one athlete identified in each sport surveyed.[25] Although disordered eating is present in all sports, certain sports have higher incidence than others. The prevalence among college gymnasts, for example, has been reported to be as high as 62%.[26] A study of 487 female elite swimmers, reported that between 62% to 77% were skipping meals or eating smaller amounts in attempts to lose weight.[27] Skolnick[2] states the incidence is high among appearance-based and endurance sports. Sundgot-Borgen[28] agrees, stating that the highest prevalence of athletes using pathogenic weight control methods is found among those competing in aesthetic sports (34%), weight dependent sports (32%), and endurance sports (20%). Johnson[24] is more specific, stating that female athletes are at a higher risk in sports "in which subjective judging encourages lean appearance, such a gymnastics, diving, figure skating, dance, and synchronized swimming, as well as sports that emphasize body leanness for optimal performance, such as long distance running, swimming, cross-country skiing, and sports that use weight classifications such as rowing, judo, weightlifting, and taekwondo."[24(p357)]

Contributing Factors

The factors that contribute to the development of disordered eating in the female athlete are multiple. Generally speaking they include personality, environmental, and societal issues. Many athletes, for example, have *personality* traits associated with sport success such as competitiveness, perfectionism, compulsiveness, high achievement expectations, and the drive for self-control.[2,24] Other personality factors that contribute to disordered patterns of eating include poor coping skills, low self-esteem, and lack of identity outside of her sport. A history of physical or sexual abuse will also personally influence the athlete.[24] In addition, athletes tend to have increased body-awareness and are more likely to recognize the typical or optimal weight associated with a high level of performance in their sport.[1,24]

Female athletes can also be influenced by the special demands and characteristics of the athletic *environment*, such as the pressure to succeed in her sport. Coaches, as well as parents, may encourage abnormal behaviors through training techniques like daily or frequent weigh-ins or use of punitive measures and negative reinforcement to detour weight gain or poor performance. In addition, her scholarship and the coach's job may depend on winning.[2] She may feel "overwhelmed or out of control as a result of academic demands, injury, perceived poor sports performance, social relationships, financial concerns, or family issues." The athlete may resort to changing her eating habits, providing her with a seemingly greater sense of control. It is thought that an athlete learns

to block the pain and distractions associated with training and competition and in the same manner is able to block her hunger.[24]

Society is also influential in that it often perpetuates the need to be thin, equating low body weight with control, goodness, power, and beauty.[24] Positive and realistic images of female athletes are a scarcity.

Progression

As mentioned, multiple factors influence the progression towards a focus on achieving or maintaining a certain body weight or percentage of body fat.[1,2] Typically, the athlete attempts to meet her goals in an unrealistic amount of time and without appropriate guidance.[2] Additionally, she may not be able to attain her "ideal" body type given her genetic endowment.[1] These things force her to depend on disordered patterns of eating. The drive to lose weight or to ascertain a certain body size or shape in order to increase competitiveness is basically an "at-any-cost" approach and is generally supported and even demanded by parents, coaches, trainers, and society.[1,2] Skolnick[2] states that "rather than being a solution to their problems, disordered eating is a vicious cycle, with hunger leading to loss of energy, moodiness, and binge eating followed by self-loathing and increased insecurity". Furthermore, disordered eating can result in decreased endurance, strength, reaction time, speed, and ability to concentrate,[24] thereby decreasing athletic ability. For the female athlete, disordered eating is a no-win behavior.

Caloric Intake

Caloric restriction is a behavioral component of disordered eating. It influences body weight, body composition, nutrition, and energy. Restrictive eating behavior may range from "inadvertently failing to meet basal caloric needs (i.e., poor nutrition) to voluntary starvation coupled with extreme exercise regimens."[24]

Although many assume that caloric restriction is the primary dietary influence, it is debatable whether menstrual irregularities are due to below average caloric intake or whether caloric values are typical but insufficient to meet the athlete's needs based on increased demands. Certain studies indicate that the first is true. For example, a study by Marcus et al[9] observed mean caloric intake of 1715 kcal for their eumenorrheic and 1272 kcal for their amenorrheic subjects. Drinkwater et al[29] reports similar findings with mean values of 1965 kcal and 1623 kcal for their eumenorrheic and oligomenorrheic athletes, respectively. Furthermore, it has been reported that professional and student ballet dancers consume fewer calories than the recommended dietary allowance (RDA). Ironically, this figure is intended for the "average" woman who is not participating in a high levels of activity.[55]

Other studies report elevated caloric intake among their female athletes, even those diagnosed with amenorrhea or oligomenorrhea.[31, 32] It has been postulated that their caloric intake may not be high enough to meet the requirements for the amount of energy expended during training. The result has been termed an *energy drain*.[1,14] This energy drain is thought to cause a

"decrease in basal metabolic rate and subsequent hypothalamic dysfunction."[1] It has also been suggested as a contributing factor to the development of athletic amenorrhea.[33]

Vegetarian vs. Nonvegetarian Diet

When considering diet and its influence on menstrual dysfunction, the type of diet must also be investigated. Of particular significance is the vegetarian diet. Although different forms of vegetarianism exist, in general this diet is considered to be one that is high in fiber and low in fat.[55] Increased *dietary fiber*, associated with a vegetarian diet, has been implicated as a cause of increased fecal excretion of estrogen and decreased plasma concentrations of estrogens.[34,35,36] Studies have reported excretions more than twice as great in vegetarians compared to their nonvegetarian counterparts. Mechanisms for this occurrence are not well understood. However, direct estrogen absorption by the fiber and alterations of gut flora have been proposed.[35,36]

The *low fat* content of the vegetarian diet may also implicate decreases in body weight, altered body composition, and, although undemonstrated as of yet, could theoretically cause concentration deficiencies of fat-soluble vitamins.[55] Even though weight loss is known to play a role in menstrual dysfunction (as described in the section that follows), one particular study has shown that vegetarian diets can affect a woman's menstrual cycle more than nonvegetarian diets, even when both result in equal amounts of weight loss. In this study, all subjects lost an average of 1kg of body weight per week. All nine of the vegetarian subjects developed shorter cycles; seven became anovulatory. In

addition, average luteinizing hormone (LH) values were significantly decreased during the midcycle and the luteal phase. Estradiol and progesterone values were significantly lower during the luteal phase. In contrast, the nonvegetarian group was not affected in any of these areas during their cycles. The differences could not be explained by initial weight or extent of weight loss.[37]

Due to the fact that little amounts of meat are ingested in the vegetarian diet, below average *protein* intake may also be a factor in terms of the prevalence of menstrual dysfunction in vegetarians. A confirming study reports that protein constituted a smaller percentage of the total caloric intake of their amenorrheic runners' diets compared to eumenorrheic runners and eumenorrheic nonrunners.[32]

Multiple studies report the connection between vegetarian diets and menstrual dysfunction. A study was performed that analyzed subjects according to their dietary patterns. Subjects described their diets as either "modified vegetarian," "high carbohydrate, low fat," or "balanced four food group". Fourteen (31%) of the 45 vegetarians had secondary amenorrhea; six (14%) of the 44 women who described their diet as "high carbohydrate, low fat" were amenorrheic; only 3 (4%) of the 84 women who consumed a "balanced four food group" diet had less than three menstrual cycles yearly.[38] A study by Brooks et al[39] grouped runners into categories of either amenorrheic or regularly menstruating. The amenorrheic subjects ate five times less meat than the regularly menstruating subjects and had a significantly lower fat consumption. Of the amenorrheic group, 82% were vegetarians, whereas only 13% of the

regularly menstruating runners were vegetarian. A similar study found that 44% of regular menstruating and 100% of amenorrheic runners consumed no red meat.[33] Other literature has also documented that most female runners with amenorrhea are vegetarians.[40]

Body Weight and Composition

Body weight is not static, it varies throughout life. Syndromes associated with weight loss and resultant menarcheal delay or abnormal cyclicity include anorexia nervosa, bulimia, simple dieting with weight loss below ideal body weight, fad diets, and vegetarian diets.[41] Vegetarian diets have previously been examined and anorexia nervosa and bulimia will be subsequently detailed in later sections. This section will focus on weight loss in general.

Hormonal Implications

Multiple hormonal alterations occur as the result of weight loss. Many studies report the effects on the pituitary hormones LH and FSH. Researchers have reported that LH concentrations and the frequency of episodic secretions is reduced with both simple weight loss (1 kg per week)[42] and with losses of 10-12% of body weight.[37] Other studies have reported decreases of both LH [43] and FSH [43,44] basal levels as well as lateness of LH and FSH peaks associated with weight loss.[44] According to some researchers, this may be due to the flattened responses of LH and FSH to GnRH.[43]

Due to the relationship between pituitary (LH and FSH) and ovarian hormones, one should also expect to see altered estrogen and progesterone levels associated with changes in body weight. Researchers have reported

impaired secretion and lower concentrations of progesterone associated with severe[45] and mild weight loss.[42] This often indicates absence of ovulatory cycles and corpus luteum formation.[45] Progressive declines in estradiol concentrations also occur with weight loss,[37] with significant decreases noted after 5.0-8.2 kg weight loss.[46]

Studies have suggested a negative correlation between the extent of weight loss and the extent of hormonal alterations. Greater amounts of weight reduction result in lower hormonal plasma concentrations.[43,47]

The implications of hormonal changes are not fully understood. However, any of the above mentioned hormones may partially account for the menstrual irregularities that develop.

Delayed Menarche

The effects of weight loss and decreased body fat on menstruation are numerous, with several studies implicating a role in delayed menarche. Subjects with extreme weight loss have reportedly been unable to achieve menarche until 87% of ideal body weight was reached.[48] It has been discovered that the onset of menarche in normal girls is correlated not only with weight but also with a calculated amount of body fat around 16 kg. This is approximately equivalent to 22% to 24% body fat. This is because puberty occurs during a "fat spurt, with girls accumulating twice as much fat as boys during the development of reproductive maturity".[21] In terms of weight, a loss of 10% to 15% can reduce the fat/lean ratio to a less than critical level.[49] Another study found that about 22% body fat is needed for the restoration and maintenance of menstrual cycles for

women ages 16 year and over. The same study also reported the weights at which menstrual cycles resumed in post-menarcheal girls ages 16 and older. They are about 10% heavier than the minimal weights for the same height observed at menarche. This may be due to the fact that normal girls become "relatively and absolutely fatter from menarche to reproductive maturity at ages 16 to 18 years".[49]

Information regarding a critical amount of fat suggests that a minimum level of stored, easily mobilized energy is necessary for onset as well as regulation of menstrual cycles.[49] The occurrence of menarche after an absence of training[50] supports the hypothesis that changes in fat/lean ratio may be involved in delayed menarche.

It is not coincidental that delayed menarche occurs most frequently in girls pursuing sports in which there are typically low amounts of body fat.[21] Frisch et al[50] observed ballet dancers. Those dancers reporting absence of menarche were found to be significantly leaner than any other group. It was also noted that each of these girls fell below the critical weight for height as previously described.[49,50] Results may show that 'late bloomers' choose to be ballet dancers or that physical training and poor nutrition cause the thinness that delays puberty.[49] The fact that most of the dancers began training before normal onset of menstruation, in addition to the prevalence of multiple forms of menstrual dysfunction in this sport, suggests that physical activity and caloric restriction contribute to this phenomenon.[50]

Sources state that altered temperature regulation has been reported in both simple weight loss amenorrhea and anorexia nervosa.[41] "The role of the superficial mantle of fat in the body and its importance at puberty may be necessary for the maintenance of temperature homeostasis, and this in turn may be one of the signals involved in neuroendocrine regulation of puberty."[21] Other sources agree, stating that body weight and body fat govern neuroendocrine controls concerned with the onset of menarche and the maintenance of normal menstrual cycles.[49] This can be likened to animal hibernation. Hibernation causes a drop in body temperature and metabolic rate that is associated with gonadal involution.[21]

Other Menstrual Dysfunction

Body weight and composition are also related to other forms of menstrual dysfunction. Frisch and Revelle[51] have found that a weight reduction of more than 13% of ideal body weight (IBW) will cause (secondary) amenorrhea in most women. Pirke et al[46] performed a study in which women were instructed to ingest between 800-1000 kcal/day for six weeks. Absolute weight loss ranged from 5.0 kg to 8.2 kg. Six subjects had regular ovulatory cycles prior to dieting. Of these six, three developed anovulation or had a shortened luteal phase.

Literature often speaks of the inverse relationship between body weight and extent of menstrual irregularities[44] as well as body weight and incidence of amenorrhea[40,42]; the lower the body weight, the greater the extent and incidence of menstrual dysfunction. However, it must be noted that several studies[37,42,46,47] have shown that even mild weight reduction diets will cause menstrual

irregularities in the majority of normal weight young women, even when their body weight does not fall below 100% IBW.

As mentioned previously, body composition can influence menstruation. This is evident in a study by Frisch et al[50] that reported dancers with amenorrhea were significantly leaner than dancers with regular cycles. Although not statistically significant, a separate study noted that subjects in an oligomenorrheic group had less body fat than their eumenorrheic athletes.[31] Others have also confirmed that a reduced body fat is associated with menstrual dysfunction in women athletes.[34] Sanborn et al[40] plotted the prevalence of amenorrhea against an index of fatness for all subjects. They discovered that as body fat decreases, the occurrence of amenorrhea increases.

Some authors have attributed diet-induced menstrual disturbances to the decrease in fat cells which are necessary for peripheral conversion of androstenedione to estrone.[21,47] Preexisting menstrual irregularity may also predispose a woman to diet-related menstrual abnormalities.[47]

Menstrual Dysfunction Associated with Anorexia Nervosa

Previous sections addressed the general topic of diet in terms of menstrual dysfunction. However, certain information reported in studies is specific to the disorders of anorexia nervosa and bulimia. The purpose of this section is to address diet-induced menstrual irregularities as they pertain to the athlete or patient with anorexia. The information is intended for use in conjunction with previously mentioned topics.

Women with anorexia nervosa often have peculiar food preferences, among these, the vegetarian diet. The affects of such diets on menstruation were previously explored.

Anorexia is associated with a drop in body temperature and metabolic rate and altered temperature regulation. As previously described in terms of hibernation, this can be associated with secondary problems such as amenorrhea and delayed menarche.[21]

In terms of hormonal changes, some studies have reported normal FSH and low normal LH.[52] Others report deficiencies of both with LH to a greater extent. In addition to typically low levels of LH and FSH, there is a lack of the normal episodic variation of gonadotropin secretion and, in some cases, a reversion to a prepubertal pattern of secretion with low levels over a 24 hour period.[53] Occasionally adult anorexics will demonstrate another form of immature gonadotropin secretion: nocturnal spurting. Nocturnal spurting is a pattern usually observed only in early puberty.[12] As in prepuberty, the response of the pituitary is immature. FSH response is greater than the LH response.[12,53] Reversion to normal, adult-like patterns in which LH response is greater than FSH occurs with weight gain.[12] Sixty-nine percent of IBW must be reached during recovery from anorexia nervosa in order to obtain pubertal LH secretion patterns. Adult patterns occur at more than 80% of IBW.[54]

Estrogen deficiencies have also been reported in anorexia nervosa.[12] This is due to inadequate gonadotropin stimulation. Reduced stores of body fat may also contribute to low estradiol levels, secondary to "decreased peripheral

aromatization of androgens to estradiol" which occurs in fat cells.[43] Finally, recalling information from descriptives of the vegetarian diet, low estrogen levels may also be attributed to increased fecal excretion associated with diets high in fiber.[34-36]

Findings suggest that amenorrhea in anorexia nervosa is mediated by the central nervous system which alters the signals reaching the hypothalamus. This in turn alters the normal episodic secretions of GnRH which appears to be necessary for normal maturations and cyclicity.[12] More specifically, findings suggest that the "amenorrhea seen in anorexia nervosa is probably due to faulty signals reaching the medial central hypothalamus from the arcuate nucleus, the center most likely responsible for the important episodic stimulation of GnRH."[12(p2682)]

Menstrual Dysfunction Associated with Bulimia

It has been speculated that intermittent dieting can contribute to the development of disturbances of the menstrual cycle in bulimia nervosa.[55] The bulimic individual's weight may fluctuate but usually not to dangerously low levels.[12] Although weight may remain within a certain range, protein-calorie malnutrition must be considered as a possible cause for menstrual disturbances.[12,41]

Bulimia may or may not bring about menstrual dysfunction. One study reported that 14 of its 15 subjects with bulimia had some form of menstrual disturbance. Another source states that about one-half of patients have amenorrhea or oligomenorrhea, despite normal body weight.[56] When present,

menstrual irregularities often are accompanied by adequate estrogen secretion and anovulation.[12]

Patients with bulimia are prone to abnormal follicular development, abnormal plasma progesterone values during the luteal phase, and luteal phase deficiency. Moderate weight disturbances are associated with disturbances in follicular development.[55]

CHAPTER IV

EXERCISE-INDUCED MENSTRUAL DYSFUNCTION

The "female athlete triad," as described previously, typically examines the relationship between diet and menstrual dysfunction. Disordered eating is thought to be the culprit of menstrual problems. However, exercise is often used in place of or in addition to food restriction or pathogenic behaviors as a means of controlling body weight.[1] Furthermore, the athlete may have a seemingly normal diet, but may be experiencing a caloric deficit in relation to energy demands. This previously described "energy drain" is often present in the female athlete and may resemble undernutrition.[41] A large energy drain may delay puberty and affect normal reproductive function in women.[21] Therefore, exercise must also be examined as a precipitating factor in menstrual irregularities.

General Effects of Exercise

The benefits of regular physical activity in disease prevention and health promotion for the majority of girls and women are numerous.[1] For example, physical activity has an effect on maintaining bone mineral content[10] and increasing bone density in areas that are maximally stressed.[1] In terms of the menstrual cycle, active women have been shown to have less dysmenorrhea than sedentary women.[14]

However, physical activity may also have detrimental effects. Exercise can bring about changes similar to those seen with diet alterations. These include decreases in body weight and altered body composition[19,32]; nutrient imbalances[32]; increases in physical[22] and emotional stress[32]; and decreases in hormonal concentrations.[57-59] These factors are thought to act synergistically in promoting various forms of menstrual dysfunction.[19,46] Many of these elements are discussed in Chapter III, Diet-Induced Menstrual Dysfunction. However, studies often report specific hormonal findings associated with exercise. These are provided in the section that follows.

Hormonal Patterns

Several studies report hormonal findings associated with exercise. The following sections outline what is typically found during acute exercise as well as changes that occur with routine exercise and increases in training intensity.

Acute Activity

Hormonal changes occur acutely with exercise and are similar to those seen with food restriction. Concentrations of prolaction,[60] estradiol, progesterone,[61] and testosterone[61] rise during exercise[55] whereas LH frequency and amplitude have been shown to decrease.[58,62] Blood flow to the liver decreases and may affect hormonal metabolism.[41] Following cessation of exercise, hormonal levels are said to return to normal resting levels within an hour or two.[55] Furthermore, the acute hormonal effects are reduced with long-term training.[41] While acute hormonal effects are readily indentified, it is unclear whether they are connected to the development of reproductive abnormalities.

Exercise Intensity

Hormonal changes associated with routine exercise have been shown to increase with elevated workloads. Increased training volume is associated with decreases in progesterone[63] and estradiol levels[64,65]; shortening of the luteal phase[63]; and impaired gonadotropin response to GnRH.[64,65] Cumming et al[58] also report findings that are consistent with the concept that a "high volume of endurance activity exerts a central inhibitory effect on the hypothalamic-pituitary axis".[58(p811)] These hormonal changes may be associated with variations in menstrual function. It must be noted that decreases in mileage have been shown to reverse these changes.[18,55]

Menstrual Dysfunction

Multiple reseachers suggest the relationship between exercise and menstrual dysfunction. The following section provides an explanation of their findings.

Delayed Menarche

It has been shown that delayed menarche and amenorrhea are related to age at onset of training. For example, Frisch et al[34] studied college athletes (swimmers and runners) and found that premenarche-trained athletes had a mean menarcheal age of 15.1 $\pm$ 0.5 years. Furthermore, each year of training before menarche delayed menarche by five months (0.4 years). Postmenarche-trained athletes had a mean menarcheal age of 12.8 $\pm$ 0.2 years, similar to control subjects who reported menarche at 12.7 $\pm$ 0.4 years. Warren[22] reported similar findings in a study using 15 ballet dancers, ages 13-15. The dancers

maintained a high level of activity from early adolescence and were followed for 4.0 years. Mean age of menarche was 15.4 years, significantly different from normal controls (12.5 years).

The relationship between body weight and composition in regards to menarche was previously discussed with the conclusion that a critical body weight and/or percentage of body fat is necessary for the onset of menses. Studies on exercise report that even after an acceptable weight or body composition is obtained, menarche remains delayed. This suggests that other factors (such as exercise) are responsible.[41]

A study by Warren[22] further addresses this issue. The onset of menarche was found to correlate in 10 of 15 subjects with a decrease in exercise and/or forced rest of at least a two-month duration; weight gain was minimal or absent, with no significant changes in body composition. Warren[22] summarizes the relationship among menses, exercise, body weight, and body fat in the case scenarios of two ballet dancers. The first dancer experienced menarche during a vacation. Secondary amenorrhea resumed with return to activity but menses again prevailed during two occasions of rest. The second dancer began her cycle after an injury forced her to rest. No change in body weight or body fat occurred. The girl later developed secondary amenorrhea after resumption of exercise. Menses were restored during a period of dieting and rest that was associated with a 5-kg gain in weight. After returning to dancing, however, amenorrhea resumed.

Other Menstrual Irregularities

Certain researchers have been able to induce menstrual irregularities in their subjects without the use of dietary restrictions or weight loss. Irregularities were largely thought to be the result of intense training. However, researchers admit that factors such as the stress and decreased body fat associated with training may have influenced the results.[14]

The nature of the activity seems to be of little importance. Running, swimming, ballet, gymnastics, rowing, and weight lifting have all been associated with menstrual dysfunction.[9,34,40,50,66] In contrast, intensity of the activity may be a determinant. A study by Sanborn et al[40] revealed a linear relationship between increased mileage in the training of runners and incidence of amenorrhea. Drinkwater et al[29] noted that their amenorrheic and oligomenorrheic subjects differed in mileage run per week at 41.8 miles and 24.9 miles respectively. Lindberg et al[10] also reported that their amenorrheic and oligomenorrheic subjects ran greater distances each week and had a longer duration of training than normal runners. Researchers have not yet discovered a relationship between exercise intensity and menstrual dysfunction in swimmers and cyclists.[40]

Arguments

Certain literature disputes that exercise is a contributing factor to menstrual irregularity. It suggests that perhaps various sports favor a specific physique, one that favors superior athletic performance as well as a delay in pubertal development.[21] Furthermore, some studies have shown that eumenorrheic and amenorrheic runners do not differ significantly in mileage,

pace, or duration of training.[67] This is supported by data demonstrating that amenorrheic runners have a higher incidence of menstrual irregularity prior to onset of training,[32,67] suggesting that exercise is not a factor.

CHAPTER V

SECONDARY PATHOLOGY

The information that follows is intended to provide an overview of the various musculoskeletal pathologies associated with menstrual dysfunction and its precursors. Secondary pathologies included in this papers discussion include alterations in bone mineral density, fractures, general musculoskeletal injury, and scoliosis.

Bone Mineral Density

In the section that follows, the variables that influence bone mineral density will be outlined in terms of current literature. It will include discussions on menstrual dysfunction, body weight and composition, hormones, nutrition, and the specific diagnoses of anorexia nervosa and bulimia.

Menstrual Dysfunction

Amenorrheic subjects have been shown to have significantly lower bone mineral densities than their eumenorrheic counterparts. For example, Drinkwater et al[29] compared the bone mineral densities of these two groups and found that eumenorrheic subjects had densities close to that predicted by an age-based regression equation. In contrast, the amenorrheic subjects had significantly lower bone mineral densities. The average age of these subjects was 24.9 $\pm$ 1.3

years whereas their bone density was equivalent to that of women 51.2 years of age.

Some authors have examined the differences that primary and secondary amenorrheas exert on bone. Ulrich et al[68] found that the bone density of subjects with primary amenorrhea was significantly less than women with secondary amenorrhea. All subjects with primary amenorrhea had ostepenia, whereas only 10 of the 16 subjects with secondary amenorrhea exhibited this effect. Seeman et al[69] also focused on distinguishing between primary and secondary forms of amenorrhea. Researchers discovered that the average bone density of the lumbar spine was 0.88 ± 0.04 g/cm^2 for those with primary amenorrhea versus 1.06 ± 0.03 g/cm^2 for women with secondary amenorrhea. Bone density at the femoral neck was similarly altered with values of 0.80 ± 0.04 g/cm^2 versus 0.92 ± 0.03 g/cm^2 respectively. Therefore, it appears that primary amenorrhea has a more significant effect on bone. This relates well to data indicating that the duration[69,70] as well as the age of onset of amenorrhea correlate with impaired peak bone mass and retarded bone age.[70]

There is some discrepancy as to the type and location of bone that is affected by amenorrhea. Lindberg et al[10] found significant reductions in bone density of cortical and trabecular bone and decreased bone mineral content in trabecular bone of amenorrheic subjects. Ayers et al[70] have reported significant osteopenia in cortical bones. In contrast, other researchers have found that cortical bone mass is not affected by loss of menses.[9] In terms of the locations of affected bones, Warren et al[11] discovered that amenorrhea significantly

lowered bone density of the spine, wrist, and foot. Alterations at the femoral neck have also been reported.[69] Others have argued that vertebral bone is the only area in which density is related to menstrual dysfunction.[71]

Body Weight and Composition

Body weight exerts a mechanical stress on bone that stimulates bone formation. It is essential in the development and maintenance of bone mineral density at all ages. The greatest effect is seen after 18 years.[4] Studies have shown that body mass index is proportionate to bone mineral density in healthy children,[72,73] adolescents,[74] elderly women,[75] and in all studies[76-81] of premenopausal young adult women except one.[82] Furthermore, many studies report significant alterations in bone mineral density of amenorrheic subjects, but further state that the findings could be accounted for or were highly influenced by weight.[11,71,74]

Fat mass, rather than lean body mass, has been said to be the most significant determinant of bone mineral density.[4] Anorexics and highly trained athletes typically have lower body fat composition and body weight. Studies describing the effects of amenorrhea on bone density often use subjects from these groups. This suggests that lower bone densities may be related to body weight and body composition. In addition, because puberty is correlated with weight gain, this may explain the previously mentioned effects that primary amenorrhea has on bone mineral density.

Hormonal Factors

Circulating estrogens have an effect on the maintenance of bone composition because of their effects on calcium metabolism.[7] This is clear in the well-established relationship between postmenopausal estrogen deficiency and osteoporosis. Recent studies indicate that premenopausal estrogen deficiency may also be associated with lower bone density. For example, Dhuper et al[74] divided adolescent girls into categories according to calculated estrogen exposure and discovered that the lowest scoring groups had the lowest spine and wrist bone mineral density. Other studies have found similar results.[11] Subjects in the Dhuper et al[74] study that had low estrogen exposure scores also had significantly lower weight and weight/height ratios. This correlates well with the previously mentioned effects of weight on bone mineral density. In addition, subjects with low estrogen scores were found to have delayed menarche, which is also associated with weight.

Although most agree that low estrogen is the hormonal culprit contributing to loss of bone mineral density, Carmichael et al[4] found that the bone mineral densities of hypoestrogenic subjects did not differ significantly from patients who had normal estrogen levels. Others have also reported that differences in bone mineral densities are not related to estradiol levels.[83] Furthermore, some researchers postulate that it is progesterone levels rather than estrogen that is associated with bone loss.[84] Multiple hypotheses exist. Decreased bone density associated with amenorrhea may be due to the hypoestrogenic status, to the

interaction of low estrogen levels with some other variable, or to a factor that has not yet been identified.[29]

Nutritional Factors

Calcium intake is thought to influence bone mineral density. For example, one study reported that the estimated dietary intake of calcium was significantly higher in the control group compared to the osteoporotic group.[85] Studies have also shown a positive linear correlation between dietary calcium and spinal trabecular bone density in both amenorrheic and eumenorrheic subjects.[86]

Calcium intake may be limited in subjects that demonstrate disordered patterns of eating since food restriction and other pathogenic behaviors may contribute to decreased dietary calcium. Therefore, disordered eating may contribute to menstrual dysfunction as well as alterations in bone mineral density.

It is questionable whether a correlation exists between intestinal absorption of calcium and osteoporosis. Some researchers report decreased intestinal absorption of calcium, whereas others have found values to be normal or even increased.[85]

Dietary fiber may play a role in calcium absorption and calcium balance. Some studies have reported increased calcium excretion associated with increased fiber intake.[87,88] Others have shown no affect of increased fiber on calcium excretion.[89,90] If the hypothesis is true that increased fiber intake may alter calcium balance, then the vegetarian diet must be examined in terms of its contributions to calcium excretion, menstrual dysfunction and associated decreased bone mineral density.

Anorexia Nervosa and Bulimia

Studies have shown that patients with anorexia nervosa have significantly lower bone mineral densities as compared to controls.[4,83] Differences have not been totally accounted for by age, weight, duration of illness, or serum estradiol levels.[83]

Researchers have also examined the differences in bone mineral density of patients with anorexia versus patients with bulimia versus patients with both. Of the three groups, subjects with bulimia had the highest bone mineral densities. Furthermore, subjects with anorexia and bulimia had higher bone mineral densities than those with anorexia alone. Nutritional factors may play a role in these findings. However, researchers claim that the dietary habits of patients with anorexia vary greatly and therefore, cannot fully explain the results.[4]

Fractures

In the section that follows, the variables that influence the incidence of fractures in female athletes will be outlined in terms of current literature. It will include discussions on bone density, menstrual dysfunction, body weight, and nutrition.

Bone Density

Complications of bone loss may include failure to achieve peak bone mass, increased risk of premature fractures, and inability to reach a person's height potential.[4] Of primary concern to the healthcare provider is the incidence of fractures. In a study by Myburgh et al,[66] bone mineral density was significantly lower in athletes with fractures than in control athletes.

Bone mineral density increases until age 35 and then decreases by 1% per year until menopause.[91] Failure to establish the critical peak bone mass before age 35 may place an individual at an increased risk for fractures.[4]

Menstrual Dysfunction

Menstrual irregularities are thought to contribute to decreases in bone mineral density and decreases in bone mineral density have been shown to contribute to fractures.[66] Myburgh et al[66] reported that more of their athletes with fractures had current menstrual irregularity as compared to control athletes. A study by Lloyd et al[7] examined the medical records of collegiate women athletes. They discovered x-ray-documented fractures in 9% of women athletes with regular menses and 24% of women athletes with irregular or absent menses. Barrow and Saha[5] reported that stress fractures occurred in 49% of their very irregular runners, 39% of the irregular runners, and 29% of the regular runners (with very irregular equaling 0-5 menses/year, irregular 6-9 menses/year, and regular 10-13 menses/year). Marcus et al[9] also reported that running-related fractures were more frequent in amenorrheic women. In a study by Lindberg et al,[10] 49% of the amenorrheic runners had stress fractures, whereas no fractures occurred in the normal runners and controls. More astounding was a survey produced by Warren et al[8] indicating a 61% incidence rate of fractures among the ballet dancers assessed. Many of these dancers also had menstrual irregularities, particularly delayed menarche. Duration of amenorrhea has also been shown to positively correlate with fracture rates.[8]

Two studies have implicated age of menarche as being a predictor of incidence of fractures among athletes.[8,11] One of the studies examined multiple variables including bone mineral density of the wrist, spine, and foot; calories ingested and expended; amount of calcium ingested; involvement in high energy activity; age of menarche; and presence of amenorrhea and found that the only variable to correlate with the occurrence of stress fractures was the subject's age at menarche.[11] This correlates well with previously mentioned data stating that primary amenorrhea has a greater effect (compared to secondary amenorrhea) on bone mineral density.[68,69]

Body Weight

It has been previously mentioned that a relationship exists between body weight and bone mineral density. Because of this relationship, one would assume that a correlation would also exist between body weight and fractures. Lloyd et al [31] has confirmed this, reporting that 80% of dancers with recent stress fractures had weights less than 75% of ideal.

Nutritional Factors

Lloyd et al [92] reported that subjects with fractures had a greater incidence of eating disorders. Researchers have been able to rule out menstrual irregularity and bone density as a precursor to the fractures. Athletes with fractures have also reported lower calcium intakes.[66,93]

General Musculoskeletal Injury

Researchers have examined less-specifically how menstrual irregularity may contribute to an athlete's musculoskeletal health. Lloyd et al[7] collected data

from runners in all degrees of competition and discovered that women who had been injured were more likely to have had absent or irregular menses. The authors concluded that premenopausal women who have absent or irregular menses while engaged in vigorous exercise programs are at an increased risk for musculoskeletal injury.[7]

Scoliosis

Scoliosis may also be a secondary pathology in terms of menstrual dysfunction. Specifically, delayed menarche is thought to increase the risks of developing this abnormality in young ballet dancers. There is a higher prevalence of amenorrhea among affected subjects, a longer duration of amenorrhea, and greater amounts of anorectic behavior.[8]

Scoliosis has a high familial incidence. Some researchers argue that decreased upper-to-lower-body ratios and long arm spans commonly noted in dancers could be inherited traits that are also associated with scoliosis.[8]

CHAPTER VI

ASSESSMENT AND TREATMENT

The pre-participation examination for athletes is an opportune time for the physician or health care provider to screen for disordered patterns of eating and diet or exercise-induced menstrual dysfunction. Questions asked during an evaluation should include but should not be limited to a general history, menstrual history, and a nutritional profile. For more information, refer to Table 3. If the health care provider discovers or suspects that disordered eating and/or menstrual dysfunction exists, a physician should perform an in-depth medical evaluation.[24]

Prevention, Assessment and Treatment of Disordered Eating

In order to prevent and detect disordered patterns of eating, athletes, parents, coaches, athletic administrators, training staff, physicians, and any other members of an athlete's comprehensive health care team, including physical therapists, need to be educated regarding the signs and symptoms associated with this problem (see Table 4.). The athlete and healthcare team should also know about the risks associated with these types of behaviors as well as the physiological, psychological, nutritional, and performance effects of disordered eating. Furthermore, individuals should be counseled on appropriate methods

Table 3.—Interview Topics for Evaluation[2,14,24]
General History
History of current and past athletic activity Age of onset of training Medications Surgery Medical illnesses Family history of osteoporosis and thyroid illness
Menstrual History
Frequency and duration of menstrual periods Date of last menstrual period Age of menarche and relationship to training Dysmenorrhea Premenstrual syndrome Sexual activity Contraception and safer sex knowledge and practices Pregnancy Whether changes in cycle length correlate to changes in training Hormonal therapy
Nutritional Profile
Food intake over the past 24 hours A list of "forbidden" foods Patient's highest and lowest weight Satisfaction with current weight Perception of ideal body weight Methods used to control weight

for weight control and ways to avoid disordered patterns of eating. Local and national resources can be used for additional information.[24]

Primary members of the multidisciplinary approach to managing eating disorders include physicians, nutritionists, mental health professionals, trainers, and coaches.[24] Physical therapists also interact with multiple athletes and should be considered an integral part to treatment. The roles of each team member are delineated below.

Table 4.—Signs and Symptoms of Disordered Eating[23,24,44]
Preoccupation with food, calories, and weight
Concerns about being or feeling fat; intense, irrational fear of gaining weight
Weight is average or below average; large weight fluctuations
Secretly eating or stealing food
Disappearing or making trips to the bathroom after meals
Consumption of large amounts of food not consistent with the athletes weight
Bloodshot eyes, especially after trips to the bathroom
Vomiting, or odor of vomit in the bathroom
Periods of severe, caloric restriction
Excessive laxative use
Compulsive, excessive exercise that is not part of the athlete's training regimen
Unwillingness to eat in front of others
Expression of self-deprecating thoughts following eating
Wearing baggy or layered clothing
Mood swings
Preoccupied with the eating behaviors of others
Continuous drinking of diet soda or water
Obsessive compulsive tendencies
Dry skin; brittle hair and nails
Cold intolerance; cold and discolored hands and feet; hypothermia
Menstrual dysfunction: amenorrhea, delayed menarche; psychosexual delay
Bradycardia
Lanugo
Orthostatic blood pressure changes; hypotension; dizziness
Significant weight loss; decreased subcutaneous fat and muscle
Peculiar and ritualistic ways of handling and relating to food
Dawdling while eating; taking small portions and small bites
Denial of behavior
Fatigue
Constipation and/or diarrhea
Sore throat and chest
Bloating; abdominal pain
Face and extremity edema
Swollen parotid glands at the angle of the jaw; chipmunk-like appearance
Erosion of dental enamel
Calluses on the dorsum of the hand

Physicians

Physicians can screen for disordered eating during pre-participation examinations. It is therefore essential that they become familiar with and efficient in conducting medical evaluations for disordered eating. If the physician establishes a diagnosis of disordered eating with or without menstrual irregularities, he/she should perform an in-depth medical evaluation. This may include measurements of pulse, blood pressure, height, weight, and body composition; examination of the skin and hair; palpation and auscultation of the thyroid gland; a breast and pelvic examination; and various special tests.[14] Although the athlete may present with signs and symptoms associated with menstrual irregularities, the physician must rule out differential diagnoses that also lead to amenorrhea.[2,14,19,94] These are provided for the reader in Table 5.

Table 5.—Differential Diagnoses of Amenorrhea [14,19]	
Pregnancy	Hypothalamic tumor
Asherman's Syndrome	Exercise-associated amenorrhea
Ovarian failure	Cushing's disease/syndrome
Ovarian tumors	Congenital adrenal hyperplasia
Polycystic ovary syndrome/disease	Adrenal tumors
Galactorrhea-prolactin-secreting adenoma	Hyperthyroidism
Pituitary tumors	Hypothyroidism
Pituitary failure	Oral contraceptives
Anorexia	Anabolic steroids

He or she should also consider scheduling repeat visits or consultation with a nutritionist, psychologist, or psychiatrist.[2,14] Furthermore, physicians can serve as a resource for educating other physicians as well as trainers, coaches, parents, and athletes.[24]

Nutritionist

A nutritionist can provide further diagnostic screening and educate the athlete on guidelines for healthy nutrition.[14,24] More specifically, the nutritionist's evaluation should include a detailed food and weight/height history; exercise history; a 3-day diary to assess caloric, carbohydrate, protein and nutrient intake; and body composition measurements. The athlete's calorie intake, caloric needs, and healthy weight ranges can be calculated from this data. The patient should be educated in terms of her appropriate weight range, percentage of body fat, and lean muscle mass. In addition, she should be informed regarding the effects of starvation on body composition (loss of muscle mass)[24] and overall athletic performance. Finally, she needs to be instructed to eat regular meals and snacks; be taught how to cook healthy and efficiently; and she should also be told how to make gradual changes in caloric and nutrient intake. Finally, it is recommended that the patient return to the nutritionist every one or two months in order to obtain body composition measurements for assessing progress and to allow for patient feedback and questions.[24]

Mental Health Professional

A mental health professional can address psychological issues for the patient with disordered eating. The therapist can assist the athlete to identify stressors, to learn how she perceives stress, to acknowledge how she defines her eating patterns, and to develop improved coping skills. To meet the goals of therapy, the therapist can decide if individual or group therapy is optimal.[24]

Trainers and Coaches

Trainers and coaches often have a greater frequency of contact with the affected female athlete than do other members of the healthcare team, assuming that she is allowed to continue within her sport. For this reason, their primary role is to monitor the athlete. Furthermore, coaches are encouraged to eliminate methods of control and training that may negatively influence the athlete's diet.

Physical Therapist

Currently, physical therapists are not identified as being part of the primary care team for athletes who have disordered eating.[24] However, due to the multiple secondary problems associated with such a diagnosis (see Chapter V) therapists may become involved in the comprehensive care of these women. Therapists need to keep these complications in mind when formulating treatments and exercise programs. For example, aerobic training is contraindicated, whereas resistive training assists in increasing muscle mass and bone density.[95]

Treatment of Menstrual Dysfunction

Those who work with female athletes and who learn that a woman is experiencing some form of menstrual dysfunction must realize that this is a symptom of something going wrong. It is not a natural response to exercise. Instead, it can be a potentially serious clinical problem.[2] Chapter V discussed some of the ramifications associated with menstrual dysfunction.

Two options offered to athletes in terms of treating menstrual dysfunction are *lifestyle changes* and *hormonal therapy*. The first involves the athlete

decreasing the quantity or intensity of her exercise.[19,94,96] Some authors have reported that a decrease in activity of 10% to 20% can cause menses to resume spontaneously.[96,97] This relates well to the concept of an energy drain, in which energy expenditure exceeds the athlete's caloric intake, resulting in menstrual dysfunction. By decreasing the body's energy needs, this so-called energy drain can be alleviated and menses may resume.[2] Furthermore decreasing an athlete's activity level can help to facilitate weight gain, which has also been associated with the resumption of menses.[19,96] Some believe that most athletes are unwilling to decrease their activities or gain weight and should not be urged to do so against their will.[19] Others believe that athletes naturally have a genuine interest in their health and are willing to accept recommended treatments.[14]

The second treatment option is hormonal therapy. Sources state that women who remain amenorrheic for longer than 6 to 12 months should consider hormone replacement if they are not willing to change their lifestyle.[2,19] This can be offered by cyclic estrogen progesterone therapy or by oral contraceptives. Young athletes may have concerns about the side effects related to hormonal therapy, particularly weight gain, acne, effects on performance, or mood alterations. Some side effects are more common with certain types of formulations.[14] A doctor should be able to assist the athlete in identifying the most appropriate therapy.

The beneficial effects of decreasing activity, resuming menses, and engaging in hormonal therapy will be further discussed in the section that follows.

Prevention and Treatment of Bone Loss

Studies have demonstrated that postmenopausal women experience their greatest bone loss in the first 4 to 6 years following menopause.[2] If young, amenorrheic athletes follow the same course of progression, early intervention is of utmost importance. Authors agree that treatment must be initiated within the first 2 or 3 years of amenorrhea in order to prevent or reverse bone loss.[2]

Chapter V discussed the relationship between menstrual dysfunction and bone density. It is only common sense that eliminating menstrual irregularities would be a treatment of choice. Previously, this paper discussed how alterations in life style or hormonal therapy may assist in the resumption of menses. As anticipated, several authors have reported the beneficial effects of establishing normal menstruation on bone density.[74,96,98] Increases in bone density have been discovered, however no study has reported a complete reversal to normal levels.

Several studies have implicated the effects of contraceptive use in the prevention of bone loss,[69,99] fractures,[5,66] and injury.[7] It must be noted that hormonal therapy, including the use of contraceptives, does not increase bone density but rather prevents further bone loss.[14]

Weight bearing and exercise are factors in the maintenance and development of normal bone mass. It appears that extreme weight-bearing may partially overcome the adverse skeletal effects of estrogen deprivation. For example, a study by Marcus et al[9] compared two groups of amenorrheic runners and found that the group who was more physically active had higher bone

mineral densities than did the less physically active group. However, it must be noted that the bone densities of both groups fell below the cyclic women in the control group. Furthermore, while some superior athletes may be able to partially make up for the skeletal depletion associated with menstrual dysfunction, authors have stated that it is unlikely that the average athlete with menstrual dysfunction can achieve intensity levels needed for this effect.[9]

The benefits of physical exercise on bone density are reliant upon a sufficient intake of calcium.. Bone formation is enhanced at skeletal areas under stress. If dietary calcium is present, it will be used in this process.[19,99] If it is insufficient, calcium may be transferred from other skeletal sites to make up for the shortfall.[99] Researchers have discovered a positive linear correlation between calcium intake and vertebral bone density.[99] Authors vary in their recommendations of calcium intake for women with menstrual dysfunction. Some state that a total of 1500-mg per day is necessary.[2,14] Others state that 1200-mg per day, in addition to normal intakes, is needed to achieve normal bone density in subjects with menstrual irregularities.[99] Myburgh and colleagues[93] suggest following the National Institute of Health (NIH) guidelines for calcium intake by age group, with the added recommendation of 1500 mg/day.[66]

A physical therapist intending to provide a holistic approach to the care of an athlete must be aware of the existence of the 'female athlete triad' and its signs and symptoms in order to make necessary referrals. This is because early intervention is essential in preventing or reversing the progression of the 'triad'. In addition, when treating a patient that is known to have disordered eating, the

therapist must also recognize the potential for menstrual irregularity, diminished bone density, and musculoskeletal injury. Due to the potential for injury, precaution must be used when formulating an exercise program or when selecting the method of treatment.

CHAPTER VII

CONCLUSION

The number of girls and women participating in athletic activities has grown in recent years. Although exercise is typically thought to be beneficial, female athletes in particular are prone to developing a combination of medical disorders that have only recently been described in literature. This "female athlete triad," as some have termed it, is the inter-relatedness of disordered eating, amenorrhea, and osteoporosis.[1,2] When attempting to research the components of the triad, the author of this paper discovered that none of the diagnoses are distinct entities and that factors outside the strict definitions of each pathology also play a role in accurately describing this phenomenon. For example, disordered eating has many factors such as caloric intake, nutritional influences, and alterations in body weight and composition; each must be considered in terms of the contributions they give to the second component of the triad. The author also came to the realization that exercise can additionally influence caloric balance, nutrition, body weight, and body composition in the same manner as dieting. For this reason, individual chapters were devoted to the specific influences of diet and exercise on menstruation. Instead of limiting the information to amenorrhea alone, other menstrual irregularities were also discussed.

A common theme between the two chapters was the concept of an "energy drain". An energy drain is the result of an insufficient caloric intake in relationship to energy demands that the athlete imposes upon her body.[1,14] It is thought to predispose the athlete to menstrual irregularities. The chapters were also in agreement in terms of the influences that body weight and composition can have on menstruation. It appears that a critical amount of body weight or body fat is necessary for the onset of menses.[48, 49] and can also influence the development and extent of menstrual irregularities.

Other factors that theoretically contribute to menstrual irregularity and that were reviewed within the body of this paper include hormonal alterations; intensity and duration of training; caloric and nutritional intake; type of diet (vegetarian vs. nonvegetarian); personality; environmental influences; and society.

In the past, amenorrhea has been thought to be benign, its main complication being infertility. It is now apparent that the consequences of long-term menstrual dysfunction may include early osteoporosis (the third component of the triad) and an increased frequency of stress fractures,[3-5,7-11,66] musculoskeletal injuries,[7] and scoliosis.[8]

As participants in the comprehensive health care of female athletes, physical therapists must be aware of the 'triad' in order to assist in prevention and early detection of its components such that the disorder can be stopped prior to its progression to more serious injuries. Due to the complexity of the triad as well as the nature of its treatment, the duty of the physical therapist is to identify

the problem(s) and make timely and appropriate referrals. In addition, when treating a patient that is known to have disordered eating, the therapist must also recognize the potential for menstrual irregularity, diminished bone density, and musculoskeletal injury. Due to the increased risk for injury, precaution must be used when formulating an exercise program or when selecting the method of treatment.

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