Contemporary Pediatric and Adolescent Sports Medicine *Series Editor:* Lyle J. Micheli

Cynthia J. Stein Andrea Stracciolini Kathryn E. Ackerman *Editors*

The Young Female Athlete



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Series Editor Lyle J. Micheli

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The Young Female Athlete



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The Micheli Center for Sports Injury Prevention



The mission of the Micheli Center for Sports Injury Prevention is at the heart of the *Contemporary Pediatric and Adolescent Sports Medicine* series.

The Micheli Center uses the most up-to-date medical and scientific information to develop practical strategies that help young athletes reduce their risk of injury as they prepare for a healthier future. The clinicians, scientists, activists, and technologists at the Micheli Center advance the field of sports medicine by revealing current injury patterns and risk factors while developing new methods, techniques, and technologies for preventing injuries. The Micheli Center had its official opening in April 2013 and is named after Lyle J. Micheli, one of the world's pioneers in pediatric and adolescent sports medicine. Dr. Micheli is the series editor of *Contemporary Pediatric and Adolescent Sports Medicine*.

Consistent with Dr. Micheli's professional focus over the past 40 years, the Micheli Center conducts world-class medical and scientific research focused on the prevention of sports injuries and the effects of exercise on health and wellness. In addition, the Micheli Center develops innovative methods of promoting exercise in children. The Micheli Center opens its doors to anyone seeking a healthier lifestyle, including those with medical conditions or illnesses that may have previously limited their abilities. Fellow clinicians, researchers, and educators are invited to collaborate and discover new ways to prevent, assess, and treat sports injuries.

Dr. Lyle J. Micheli, Series Editor



Dr. Lyle J. Micheli is the series editor of Contemporary Pediatric and Adolescent Sports Medicine. Dr. Micheli is regarded as one of the pioneers of pediatric and adolescent sports medicine, a field he has been working in since the early 1970s when he co-founded the USA's first sports medicine clinic for young athletes at Boston Children's Hospital.

Dr. Micheli is now director of the Division of Sports Medicine at Boston Children's Hospital, and Clinical Professor of Orthopedic Surgery at Harvard Medical School. He is a past president of the American College of Sports Medicine and is

currently the Secretary General for the International Federation of Sports Medicine. Dr. Micheli co-chaired the International Olympic Committee consensus on the health and fitness of young people through physical activity and sport.

In addition to many other honors, Dr. Micheli has served as Chairperson of the Massachusetts Governor's Committee on Physical Fitness and Sports, on the Board of Directors of the United States Rugby Football Foundation, as Chairman of the USA Rugby Medical and Risk Management Committee, and on the advisory board of the Bay State Games. He has been the Attending Physician for the Boston Ballet since 1977 and is Medical Consultant to the Boston Ballet School.

Dr. Micheli received his undergraduate degree from Harvard College in 1962 and his medical degree from Harvard Medical School in 1966. As an undergraduate student, Dr. Micheli was an avid athlete, competing in rugby, gridiron football, and boxing. Since graduating, Dr. Micheli has played prop for various Rugby clubs including the Boston Rugby Football Club, the Cleveland Blues Rugby Football Club, Washington Rugby Club, and Mystic Valley Rugby Club where he also served as team coach.

Dr. Micheli has authored over 300 scientific articles and reviews related to sports injuries, particularly in children. His present research activities focus on the prevention of sports injuries in children. Dr. Micheli has edited and authored several major books and textbooks.

Foreword

Since I co-founded the USA's first sports medicine clinic for young athletes over 40 years ago, one of the most exciting and gratifying developments has been the emergence of the female athlete. Of course, girls and women were engaged in sports long before the Division of Sports Medicine at Boston Children's Hospital opened in 1974, but their participation and prominence has grown dramatically since then— by "leaps and bounds" one might say. Much of this was the welcome result of the passage of Title IX in 1972, which prohibited discrimination in school sports.

At the same time as I was bearing witness in my professional life to increasing numbers of girls and young women participating in athletics, I was also parenting two daughters who themselves developed a passion for physical activity. My older daughter developed a love of dance, and my younger daughter came to represent the USA in the rough-and-tumble sport of rugby. All this is to say that I have been privileged to enjoy a front row seat to seeing the benefits of sports and other athletic endeavors for the female athlete.

The benefits of sports for women are becoming well known. And it's not just the important health benefits such as stronger hearts and lower rates of stroke, cancer, and obesity. There are also important psychosocial benefits that researchers have discovered. Girls and women who play sports have higher levels of confidence and self-esteem and lower levels of depression. High school girls who play sports are more likely to get better grades in school and more likely to graduate than girls who do not play sports. High school girls who play sports are less likely to be involved in risky lifestyle behavior because they have the confidence to resist peer pressure. Perhaps it's not surprising that women who played sports as girls are more successful in business.

For all these reasons, we need to provide as many opportunities for girls and young women to be as physically active as possible. If and when young female athletes get injured, we need to diagnose and treat their injuries with the most effective means at our disposal. It's also vitally important to *prevent* sports injuries, which is the mission of the institution sponsoring this series, the Micheli Center for Sports Injury Prevention. I'm so pleased that a very large proportion of the athletes

we see at the Micheli Center are girls and young women who are taking proactive steps to prevent injuries by understanding their risk factors and addressing them. They are able to do so with the best methodology and technology available.

Information is an important tool, too, which is where this book comes in. This is a wonderful resource for anyone interested in the sports health of the young female athlete. The information in these pages is the very latest available and written by leaders in the field.

I am very pleased to have *The Young Female Athlete* as a volume in the Contemporary Pediatric Sports Medicine series. I hope you will utilize this excellent resource as you join with me to strive to ensure that girls and young women get to take full advantage of the many benefits available to them in the realm of sporting and athletic endeavor.

Waltham, MA, USA

Lyle J. Micheli, MD

Preface

We are pleased to present *The Young Female Athlete*. Our goal is to provide a compilation of chapters to address some of the most important concerns of the pediatric, adolescent, and young adult female athlete. Using a multidisciplinary approach, and drawing on our years of experience caring for and learning from this population of strong and motivated athletes, we have selected a range of salient topics including normal growth and development, nutrition, sports psychology, and common injuries relevant to the young sportswoman.

Through ongoing research, it is now clear that female athletes are at higher risk of certain conditions and injuries, particularly during periods of rapid growth. We hope to highlight some of the changes and challenges faced by the female athlete as she moves from childhood through adolescence and into early adulthood. The focus of the book is to provide useful information in order to encourage healthy and enjoyable sports participation in the setting of overall fitness, psychological wellbeing, personal development, injury prevention, and long-term health.

Authors were selected for their expertise in different areas of interest. They were asked to describe what we currently know about the issues facing girls in sports and to identify what practitioners in the sports medicine community need to know to more effectively care for young female athletes in the future. In addition, authors were asked to address issues of sports injury prevention and health promotion.

Sports participation by girls has historically been limited and fraught with obstacles. Female athletes have been completely barred, actively discouraged, or simply denied the support that would have allowed physical training and competition. However, the tides have changed dramatically. With the expansion of women's rights, including major advances such as the passage of Title IX, opportunities for girls and women have exploded and continue to grow and develop.

Part of the challenge now facing female athletes, and those who care for them, is to take advantage of the many options available and to create new opportunities to enjoy the numerous benefits of sports participation and physical activity. We hope that this book will contribute to the care of the young female athlete and the ongoing efforts to improve treatment, expand injury prevention, and encourage a lifetime of enjoyable, healthy physical activity.

Boston, MA, USA

Cynthia J. Stein, MD, MPH Kathryn E. Ackerman, MD, MPH Andrea Stracciolini, MD

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Chapter 1 Growth and the Young Female Athlete

Robert M. Malina, Kathryn E. Ackerman, and Alan D. Rogol

Introduction

The body goes through remarkable changes from birth through childhood and adolescence into adulthood. Biological growth and maturation, along with behavioral development, are important aspects "growing up." These three processes, growth, maturation, and development, dominate the daily lives of children and adolescents for approximately the first two decades of life. This chapter will focus predominantly on growth and maturation, and how timing and tempo of these may affect sports involvement and injury risk in young female athletes.

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Definitions

Growth: the increase in the size of the body as a whole and of its parts. As children grow, height and body mass increase. The latter includes changes in skeletal, muscle and fat mass, as well as in bodily organs. Different segments of the body grow at different rates and at different times, resulting in altered body proportions.

Maturation: the progress towards biological maturity. Maturation is a process, while maturity is a state. Maturation occurs in all bodily organs and systems, but maturity varies with each biological system considered. Sexual maturity is fully functional reproductive capability. Skeletal maturity is a fully ossified adult skeleton. Maturation of the nervous and endocrine systems is a major factor underlying sexual, skeletal, and somatic growth and maturation.

Timing and tempo of growth and maturation: Timing refers to the chronological age at which specific events or milestones in growth and maturation occur, while tempo refers to the rate at which the processes of growth and maturation progress. Both timing and tempo vary considerably among individuals.

Development: a psychological, emotional, and behavioral process often subsumed in the term socialization, which is specific to a culture.

Indicators and Patterns of Growth

The study of growth and maturation is based on standardized measurements and observations, which reflect underlying biological and neuroendocrine changes. Time between birth and adulthood has been formally subdivided by Karlberg into phases of normal growth: infancy, childhood, and puberty [1, 2]. During infancy (birth to 6–12 months), growth is rapid but progresses at a decelerating rate. Growth curves often cross percentile lines as the infant moves farther away from the excesses or constraints of the intrauterine environment and toward her genetic potential. The next phase is childhood, during which growth and development are primarily dependent on adequate nutrition, an appropriate psychosocial environment, and the absence of disease. In the context of these environmental factors, one must have sufficient levels of thyroid hormone and insulin-like growth factor 1 (IGF-1), the stable pharmacodynamic marker of growth hormone (GH), to grow normally. Growth rate continues to decelerate until around age 3 years, and then continues at a relatively constant velocity of 4.5–7.0 cm/year and 2.5 kg/year for girls and boys. Growth rate often slows slightly just prior to adolescence [1, 2].

The hypothalamic–pituitary–gonadal (HPG) axis is virtually quiescent until late childhood, when activation from the secretion of estrogen from the ovaries in girls (testosterone from the testes in boys) occurs, well before the outward signs of pubertal development become apparent. The pubertal phase is characterized by a growth spurt of 8–14 cm/year because of the synergistic effects of increasing gonadal steroids and GH secretion [1, 2]. The upper limit of middle childhood is arbitrary given variations in the onset of puberty; some girls as young as 9–10 years old are already in the early stage of puberty/adolescence. The termination of adolescence is also quite variable. Biologically, some girls are sexually mature by 12–13 years of age (i.e., they are biologically adult), even though socially and behaviorally, they remain adolescents. Although chronological age is a commonly used reference point, and children are often divided into age groups, there is great variability among individuals of the same chronological age, especially around the time of puberty [3].

Body Size and Composition

Height and weight are the body dimensions most commonly used to monitor growth status and growth rate. Length is commonly used in place of height during the first 2–3 years of life.

Height

Height in most children is measured yearly, and growth charts represent "smoothed" growth data. However, linear growth is actually episodic with stepwise jumps, as noted by daily measurements over time in both newborns and adolescents [4]. Although less common in girls than in boys, some children show a minor growth spurt between ages 6 and 8. By 9–10 years of age, the rate of height growth accelerates in girls, marking the beginning of the adolescent spurt. From the onset of this growth spurt, the rate of growth in height increases until it reaches its peak [peak height velocity (PHV)], which generally occurs at about 12 years of age in girls [5] and 14 years of age in boys [5].

Children continue to grow in height through adolescence, but after the growth spurt, the rate of growth gradually decreases and eventually reaches zero as individuals attain their adult stature. The timing of each of these events—the growth spurt, PHV, and termination of growth—is variable among individuals.

Weight

Weight, like height, increases throughout childhood, but the rate of increase changes during different phases of growth, decelerating from birth through the second year of life, and then accelerating as the child ages. A growth spurt is also observed in body weight, which generally begins slightly later than that of height. Average age of peak weight velocity in girls is about half a year after PHV. Weight continues to increase into the late teens and early 20s, and unlike height, can continue beyond skeletal maturity [5].

Body Composition

Body weight is a composite of all bodily tissues, but it is often described in terms of its lean (fat-free) and fat components. Thus, body weight=fat-free mass (FFM)+fat mass (FM). Major components of FFM are skeletal muscle and bone mineral. FFM has a growth pattern similar to that of height and weight and demonstrates a clear adolescent spurt. FM increases more gradually during childhood and adolescence. Most other body dimensions (sitting height, leg length, limb circumferences, skeletal breadths) and many major organs follow growth patterns similar to height and weight, although growth rates and the timing of adolescent spurts vary relative to age at PHV [5]. Differential growth rates in specific dimensions influence body proportions and regional distribution of body fat [5, 6].

Body composition changes throughout life, but striking transformations are evident at puberty, resulting from sex-dependent changes in hormones and cytokines, including increases in GH, IGF-1, and reproductive hormone concentrations. GH secretion increases by a similar magnitude in both boys and girls during puberty, but IGF-1 levels are consistently higher in girls, in pre-pubertal and pubertal phases [7, 8]. While reproductive hormones act directly on growth plates of bones, independent of GH, a majority of the sex-related differences in growth and body composition are likely mediated by the effects of reproductive hormones on the GH–IGF-1 axis [8, 9]. For example, in boys, the interactions of testosterone, GH, and IGF-1 together enhance the increase in muscle and loss of fat, leading to the more muscular configuration of the young adult male. In contrast, in pubertal girls, the increase in estrogen attenuates GH secretion and IGF-1, leading to a slowing in the accrual of muscle and an enhancement of fat gain, especially in the gynoid distribution [8].

Bone mineral density (BMD) increases steadily from childhood to adolescence, with a decrease in bone mass relative to bone size before PHV. Peak velocity of bone mineral content occurs about 6 months later than PHV. A majority of bone mass and size are acquired by late adolescence, followed by small increases thereafter [10]. See Chapter 5 for additional details.

Physical Performance

Physical performance is generally measured with standard tests of strength, speed, power, agility, endurance and flexibility, and in the context of sport, with sport-specific skills tests. Sex differences in performances are relatively small during childhood, but are magnified during adolescence as performances of girls typically reach a plateau or improve only slightly. Isometric (static) strength, for example, generally increases in a linear fashion through childhood for both boys and girls. Around age 13, males experience an adolescent spurt and an increase in the rate of strength development, while females continue to experience a linear increase until around age 15. Nevertheless, limited longitudinal data for girls indicate a growth

spurt in static strength that is about one-half the magnitude of that noted for boys. The spurt generally occurs after PHV [3].

Performance on flexibility tasks tends to decline from childhood through midadolescence and then increases. Flexibility in girls usually remains stable or decreases slightly during childhood, increases during adolescence, and plateaus at 14–15 years of age [3, 5, 11, 12]. In general, flexibility tends to be greater in girls than boys.

An adolescent spurt occurs in absolute aerobic power, VO₂max or peak VO₂, in both boys and girls. Peak VO₂ generally increases in boys from childhood through adolescence, but reaches a plateau in girls by 13–14 years. VO₂max starts to increase several years before PHV and continues to increase after. However, due to changes in height and mass, VO₂max per unit body mass actually begins to decline 1 year before PHV and continues after [3].

Indicators and Measurements of Maturation

Maturity Status

Maturity status and progress are traditionally monitored in the skeleton and secondary sex characteristics. Radiographs of the hand and wrist can be used to estimate skeletal maturity using the standards of Greulich and Pyle [13]. This assessment of skeletal maturation, along with current height, can be used to predict adult or mature height. Sexual maturation is based on the development of secondary sex characteristics; in girls, breast bud development (thelarche) is typically the first physically apparent sign of sexual maturation, followed by the appearance of public hair (pubarche).

Tanner described five stages of maturation [14]. Stage 1 (pre-pubertal) indicates lack of overt manifestation of the development of breasts and pubic hair; however, pre-pubertal children of the same chronological age can vary in skeletal age by 4 years or more [5]. Stage 2 marks the onset of puberty, typically starting around a bone age of 11 years in girls, while advancement in breast development and pubic hair distribution in stages 3 and 4 mark progress in puberty. Stage 5 is the mature state [15]. It is important to note that stages have limitations; they provide no information on when the stage was reached (timing) or how long the child has been in the stage (tempo).

Maturity Timing

Ages at PHV and the beginning of menstrual periods (menarche) are indicators of maturity timing. Menarche typically occurs after PHV and on average, 2.6 years after the onset of puberty [16]. The average age of menarche in the United States has declined over the last century, and is now around 12.3 years of age [17]. There are various influences on timing of puberty, including nutrition, race, ethnicity, geography, and other factors.

Measurement of Maturation

Measuring maturation can be challenging. Established indicators used in growth studies and in clinics have limitations: skeletal age determination with X-rays exposes children to low-dose radiation and is dependent on experienced individuals to interpret the films; directly observing secondary sex characteristics is invasive of personal privacy; age at PHV is an "after-the-fact" indicator; and surveys questioning age at menarche, breast bud development, and pubic hair growth are affected by recall accuracy. Other non-invasive estimates using percentage of predicted adult stature at the time of observation and predicted maturity offset (time before PHV) have also been suggested to estimate maturity status and timing, respectively, in youth athletes [18, 19]. However, these too have limitations.

Growth in the Athlete

It is obvious when observing athletes in a variety of sports that different physical factors offer competitive advantages. In some cases, such as taller average height in basketball players, these differences are clearly the result of selection, due to the fact that certain athletes enter or succeed in the sport while others either do not participate or do not progress. However, in other cases, like shorter average height in gymnasts, it has been more difficult to tease out the impact of selection and training. Even when patterns are recognized, causality may be difficult to establish, especially if data are cross-sectional rather than prospective. Despite these limitations, current research indicates that sports training and participation do not appear to affect adult stature or overall rate of growth [3, 20, 21]. In addition, sports training alone does not likely affect skeletal or sexual maturation [3].

Growth data for young female athletes are limited largely to height and weight [21–24]. Although there is variation among sports, female athletes in most sports tend to have heights similar to or greater than the median of U.S. reference data from childhood through adolescence, and weights that are appropriate for their height [3]. Notable exceptions include artistic gymnasts and figure skaters, who are generally shorter than their peers [21, 25]. Young female athletes also tend to be leaner, with lower weightfor-height, compared to non-athletes [3]. Ballet dancers and distance runners tend to have lower weight-for-height, while participants in field events and higher weight categories in weightlifting often have excess weight-for-height [3, 26].

The available height and weight data for young female athletes span decades. There is a need to consider changes in sport rules, training, selection, and other factors when examining small subsets of athletes at different time points. For example, between the 1960s and 1997, the minimum age for female artistic gymnastics to compete internationally was increased from 13 to 16 years. While the average age of participants has increased, heights and weights have changed little from 1987 through 2008 [21], suggesting preferential selection for shorter, leaner athletes. In a

study of body size in elite junior rowers (ages 15–18 years) competing at the World Championships in 1997 versus 2007, female rowers in 2007 were on average 2.1 cm taller [27]. This may simply reflect recruitment of taller athletes to a sport in which height is a competitive advantage.

The BMI is currently the most commonly used index of weight-for-height. Although a great deal of focus is on overweight and obesity in the non-athlete population, low BMI often gets more attention among female athletes. Three grades of low weight-for-height (labeled mild, moderate, and severe thinness) among children and adolescents have been described. The term thinness was selected to avoid confusion with other labels commonly used to define low weight-for-height (e.g., wasting and underweight) in children. Three standard growth curves have been designed to pass through a BMI of 16, 17, and 18.5 at age 18. These points are, respectively, the cut-offs used to define severe, moderate, and mild thinness in adults. Thus the specific BMI cut-off at each age prior to 18 years depends on the standard curves for each grade of thinness [28]. In a sample of over 1000 female artistic gymnasts, figure skaters, divers, distance runners and ballet dancers spanning childhood through young adulthood, mild thinness was reasonably common among female athletes, but prevalence varied by sport and level of competition [29].

On average, female gymnasts are of lower weight than a reference population; however, their weight is appropriate for their diminished height. Some gymnasts are indeed at low weight-for-height. In an evaluation of ages, heights, and weights as reported in the official program of 60 participants 15-20 years of age at the 2008 Beijing Olympic Games, 23 (38 %) were classified as mildly thin and 6 (10 %) were classified as moderately thin; none were severely thin [29]. Allowing for the limited data, some female artistic gymnasts may be at risk for mild or moderate thinness, but the risk is related in part to later maturation. Later maturing girls, on average, tend to have less weight-for-height [5].

Maturation in the Young Female Athlete

There is great interest in understanding how the stages of growth and maturation can be best utilized in sport development programs. However, the available data are extremely limited, and additional studies are needed. The popular Long Term Athlete Development model, a concept of planned, systematic, and progressive training of an athlete from childhood onward, calls for the determination of the time of PHV as a potentially sensitive period for training [30]. As noted earlier, the anthropometric protocol for predicting maturity offset and age at PHV has major limitations.

For male athletes in most sports, there appears to be a competitive advantage to being advanced or at least average in maturity status [3, 31]. For female athletes, the picture is less clear and varies by sport. Girls involved in sports such as basketball, volleyball, rowing, swimming and track, except for some distance runners, generally have a pattern of average growth and maturity [32, 33]. Female athletes typically reach menarche within the normal age range seen in the general population.

However, within this range, gymnasts, ballet dancers, and figure skaters tend to start menstruating later than other athletes and non-athletes [3].

Both male and female gymnasts have been noted to have later ages at PHV, and female gymnasts have been noted to have later maturation in terms of breast and pubic hair development, compared to age-matched controls [3, 34]. In our recent review of the literature, we found that despite the fact that gymnasts are generally shorter than their peers, as well as later and slower in growth, gymnastics training does not appear to affect adult stature [21] nor does such training impact growth rate, timing, or tempo. Data in male gymnasts suggest that observed shorter stature is a result of selection rather than training [20].

Girls who trained in rowing, track, or swimming for approximately 12 h per week for an average of 4 years during puberty and the growth spurt were found to have a slightly later PHV and age of menarche than girls inactive in sport, but the differences were not significant. The interval between PHV and menarche, PHV (cm/year), ages at attaining pubic hair and Tanner breast stages 3, 4 and 5, as well as estimated intervals between adjacent stages were also not different in the athletes versus non-athletes [24]. Studies in gymnasts and ballet dancers have found a 2- to 3-year delay in menarche, as well as oligomenorrhea and secondary amenorrhea, likely secondary to undernutrition disturbing neuroendocrine function [35, 36]. Thus, there is a need for more well-designed studies, accounting for energy availability, using validated non-invasive indicators of maturity status and timing, to better determine the effects of sport involvement alone on maturity. It is also essential to account for the selectivity of sport, specifically differential persistence and dropout, either voluntary or systematic, as in getting "cut" from the team. Data for artistic gymnasts indicate size and maturity differences between those who persist and do not persist in the sport [21].

Body Composition in the Young Athlete

Methods for assessing body composition of athletes in general [37] and of youth athletes [38] are diverse and include densitometry, whole body potassium counting (TBK), total body water (isotope dilution, hydrometry), dual energy X-ray absorptiometry, bioelectrical impedance analysis, magnetic resonance imaging, and skinfold thickness, among others [26, 39].

Allowing for variation associated with sampling and methodology, several trends are apparent in the relative fatness of youth female athletes in several sports. Young female athletes tend to have a lower %Fat than non-athletes of the same chronological age, and overlap among samples of athletes in different sports is considerable. Most estimates based on skinfolds are below the reference range, but a number are at and/or above the reference range compared to estimates based on densitometry and hydrometry. Elevated %Fat is more common in athletic field events (specifically throwers) and several samples of team sport athletes [38, 39].

Training Effects on Body Composition

Studies of the influence of athletic training on body composition have traditionally focused on changes in FFM and FM [40], while more recent studies have focused on bone mineral content (BMC), BMD, and bone microarchitecture [41, 42]. Studies of youth are confounded by difficulties in isolating those changes associated with normal growth and maturation, especially the adolescent spurt and sexual maturation, from those attributed to training [43].

There is a need to study changes in the body composition of youth athletes during the course of a competitive season to better understand the potential impact of these changes on performance; however, few data are available. Studies of changes in FFM and/or %Fat in late adolescence and young adulthood provide some insights. The studies generally compare pre- and post-training means, while duration, intensity, and frequency of training are often variable. One review examined studies of female athletes between 18 and 22 years of age and found that the differences between pre- and post-training means ranged from -1.7 to +1.5 kg for FFM (overall mean +0.3 kg), and from -2.1 to +3.1 % for %Fat (overall mean -0.4 %) [44]. These observed changes are relatively small and may fall within the range of measurement variability. Moreover, it is not clear if these changes associated with training persist, because this information is not typically reported.

Currently, there is considerable interest in the influence of regular sport training on bone health in young athletes. BMD typically doubles between the onset of puberty and young adulthood [45]. While genetic factors greatly affect bone mass, other factors such as nutrition, types of exercise, diseases, medicines, age of menarche, and menstrual regularity also significantly influence bone accrual [46]. Beneficial effects of weight-bearing sports training have been observed in cortical thickness, BMC, and BMD [47-49]. Potential confounding factors are selection for sport and limited control of biological maturity status. For example, athletes and controls are often described as pre-pubertal, although skeletal age can vary by as much as 4 years in pre-pubertal children [5]. BMC and BMD are, on average, greater in weight-bearing female athletes compared to the general population of female youth [48]. Limited longitudinal data indicate training-associated increases in BMC and/or BMD in youth athletes. The long-term effect of sport training on bone is especially apparent in the dominant versus non-dominant arms of racquet sport athletes, highlighting localized increases in bone mineral accretion [50, 51]. Effects of decreased energy availability, as seen in weight-restricted athletes and those with eating disorders, can negate the beneficial effect of weight-bearing [42, 52] (see Chapter 5). It is also interesting to note that nonimpact sports such as swimming, water polo, and cycling during youth and young adulthood are not associated with enhanced BMC or BMD, and swimming may even negatively influence hip bone geometry [53].

Effects of Puberty on Sports Training and Performance

It is not yet well understood how the various changes associated with growth, maturation, and development affect athletic performance, but it is clear that puberty is accompanied by many challenges for the young female athlete, including changes in body size and composition, increased rates of injury, and emotional changes surrounding puberty. These changes, compiled with societal pressures, may contribute to the higher drop-out rate from athletics seen in girls during middle school and high school [54, 55]. During puberty, girls may find it difficult to adapt to their changing bodies and the alterations in their athletic abilities. They may become frustrated by the normal fat gain, breast development, hip widening, and height increases, and they may see at least a temporary decline in performance. These adjustments may be particularly evident for those aesthetic sports such as dance, figure skating, gymnastics, and diving, and can derail a young athlete's confidence [56].

As discussed earlier, performance on various standardized tests typically improves throughout adolescence in boys. However, in girls, performance generally improves until about ages 13–14 years (slightly later in some motor tasks), with little subsequent improvement. Whereas males typically experience an increase in the rate of strength development starting around age 13, females generally demonstrate only a linear increase until around the age of 15. Nevertheless, some girls and particularly athletes often continue to improve in performance through adolescence. This improvement is due to regular activity and probably sport-specific training. Historically, research focused on the performance of adolescent female athletes has not received the attention given to male athletes, though the situation is changing [57, 58].

In addition to the physiologic and emotional challenges that affect young athletes during adolescence, injuries also increase during this time. One study of peripubertal gymnasts demonstrated increased injury rates during periods of rapid growth [59]. Anterior cruciate ligament (ACL) injury rates increase dramatically around ages 12-13 in girls, and female athletes 15-20 years of age account for the largest number of ACL injuries reported [60, 61]. More common in females than males, the difference in ACL injury rates becomes apparent around the time of the growth spurt, peaks during adolescence, and declines in young adulthood [61-63]. This discrepancy is likely multifactorial: during PHV, rapid lengthening of the tibia and femur leads to greater torque at the knee, increasing height results in a higher center of gravity, which requires more challenging muscular control, and increases in weight cause more joint force that is difficult to control during high velocity movements [61, 62]. Unlike boys, whose pubertal increase in testosterone aids in accumulating muscle mass, pubertal girls have difficulty building muscle, making joint control even more difficult [62]. See Chapter 9 for additional information on ACL injuries.

Around PHV, adolescents are more prone to injury because of imbalances of strength and flexibility and changes in biochemical properties of bone [64]. There is a dissociation during puberty between peak BMC and bone mineral area; bones

increase in size more rapidly than they fill in with mineral. BMD decreases before and rebounds after the period of PHV. Thus there is a time of relative skeletal weakness during adolescent growth, with a temporary increase in fracture rate [10]. Also of concern are growth plate injuries. Tendons and ligaments of the growing athlete are relatively stronger than the epiphyseal growth plate. Thus, during sports trauma, such as an ankle inversion, the epiphysis is more likely to be damaged than the ligamentous complex (e.g. fibular growth plate injury rather than a ligamentous tear).

Summary and Conclusions

The human body goes through remarkable transformations throughout the life stages. As young athletes grow and develop, they must also adapt. For sports and medical professionals, awareness of general patterns of growth and maturation allows for improved recognition of the advantages, risks, and challenges that accompany these changes. We can then better support the young athlete as she matures. With girls and young women increasingly involved in intense sports training and participation, additional prospective studies are needed to enhance our understanding of normal variations in growth and maturation and the ways in which they influence and are impacted by sports training and performance.

References

- 1. Karlberg J, et al. Analysis of linear growth using a mathematical model. I. From birth to 3 years. Acta Paediatr Scand. 1987;76(3):478–88.
- 2. Karlberg J. A biologically-oriented mathematical model (ICP) for human growth. Acta Paediatr Scand Suppl. 1989;350:70–94.
- Beunen G, Malina RM. Growth and biologic maturation: relevance to athletic performance. In: Hebestreit H, Bar-Or O, editors. The young athlete. Oxford, UK: Blackwell Publishing Ltd; 2007.
- Lampl M, Veldhuis JD, Johnson ML. Saltation and stasis: a model of human growth. Science. 1992;258(5083):801–3.
- 5. Malina R, Bouchard C, Bar-Or O. Growth, maturation and physical activity. 2nd ed. Champaign, IL: Human Kinetics Press; 2004.
- Malina RM. Variations in body composition associated with sex and ethnicity. In: Heymsfield SB et al., editors. Human body composition. Champaign, IL: Human Kinetics; 2005. p. 271–98.
- Lofqvist C, et al. Reference values for insulin-like growth factor-binding protein-3 (IGFBP-3) and the ratio of insulin-like growth factor-I to IGFBP-3 throughout childhood and adolescence. J Clin Endocrinol Metab. 2005;90(3):1420–7.
- Casazza K, Hanks LJ, Alvarez JA. Role of various cytokines and growth factors in pubertal development. Med Sport Sci. 2010;55:14–31.
- Meinhardt UJ, Ho KK. Regulation of growth hormone action by gonadal steroids. Endocrinol Metab Clin North Am. 2007;36(1):57–73.
- Faulkner RA, et al. Size-corrected BMD decreases during peak linear growth: implications for fracture incidence during adolescence. J Bone Miner Res. 2006;21(12):1864–70.

- Haubenstricker JL, Seefeldt VD. Acquisition of motor skills during childhood. In: Seefeldt V, editor. Physical activity and well-being. Reston, VA: AAHPERD; 1986. p. 41–102.
- 12. Beunen GP, Simons J. Physical growth, maturation and performance. In: Simons J et al., editors. Growth and fitness of Flemish girls: the Leuven growth study. Champaign, IL: Human Kinetics; 1990. p. 69–118.
- Greulich WW, Pyle SI, Brush Foundation Cleveland. [from old catalog], Radiographic atlas of skeletal development of the hand and wrist. Stanford: Stanford University Press; 1950. p. xiii. 190 p.
- 14. Tanner JM. Growth at adolescence, with a general consideration of the effects of hereditary and environmental factors upon growth and maturation from birth to maturity. 2nd ed. Oxford: Blackwell Scientific Publications; 1962. 325 p.
- 15. Tanner JM, et al. Prediction of adult height from height, bone age, and occurrence of menarche, at ages 4 to 16 with allowance for midparent height. Arch Dis Child. 1975;50(1): 14–26.
- 16. Biro FM, et al. Pubertal correlates in black and white girls. J Pediatr. 2006;148(2):234-40.
- 17. Anderson SE, Must A. Interpreting the continued decline in the average age at menarche: results from two nationally representative surveys of U.S. girls studied 10 years apart. J Pediatr. 2005;147(6):753–60.
- Mirwald RL, et al. An assessment of maturity from anthropometric measurements. Med Sci Sports Exerc. 2002;34(4):689–94.
- 19. Malina RM, et al. Maturity status of youth football players: a noninvasive estimate. Med Sci Sports Exerc. 2005;37(6):1044–52.
- 20. Daly RM, et al. Short stature in competitive prepubertal and early pubertal male gymnasts: the result of selection bias or intense training? J Pediatr. 2000;137(4):510–6.
- Malina RM, et al. Role of intensive training in the growth and maturation of artistic gymnasts. Sports Med. 2013;43(9):783–802.
- 22. Malina RM. Physical growth and biological maturation of young athletes. Exerc Sport Sci Rev. 1994;22:389–433.
- Eisenmann JC, Malina RM. Growth status and estimated growth rate of young distance runners. Int J Sports Med. 2002;23(3):168–73.
- 24. Geithner CA, Woynarowska B, Malina RM. The adolescent spurt and sexual maturation in girls active and not active in sport. Ann Hum Biol. 1998;25(5):415–23.
- Leone M, Lariviere G, Comtois AS. Discriminant analysis of anthropometric and biomotor variables among elite adolescent female athletes in four sports. J Sports Sci. 2002;20(6): 443–9.
- 26. Malina RM. Growth and maturation of child and adolescent track and field athletes: Final report, Monaco; 2004.
- 27. Rakovac M, et al. Body size changes in elite junior rowers: 1997 to 2007. Coll Antropol. 2011;35(1):127–31.
- Cole TJ, et al. Body mass index cut offs to define thinness in children and adolescents: international survey. BMJ. 2007;335(7612):194.
- 29. Malina RM, Rogol AD. Sport training and the growth and pubertal maturation of young athletes. Pediatr Endocrinol Rev. 2011;9(1):441–55.
- 30. Balyi I, Way R, Higgs C. Long-term athlete development. Champaign, IL: Human Kinetics; 2013.
- 31. Malina RM. Skeletal age and age verification in youth sport. Sports Med. 2011;41(11): 925–47.
- Malina RM. Physical activity and training: effects on stature and the adolescent growth spurt. Med Sci Sports Exerc. 1994;26(6):759–66.
- Malina RM, Bielicki T. Retrospective longitudinal growth study of boys and girls active in sport. Acta Paediatr. 1996;85(5):570–6.
- Bernink MJ, et al. Height, body composition, biological maturation and training in relation to socio-economic status in girl gymnasts, swimmers, and controls. Growth. 1983;47(1):1–12.
- 35. Georgopoulos NA, et al. Growth, pubertal development, skeletal maturation and bone mass acquisition in athletes. Hormones (Athens). 2004;3(4):233–43.

- 36. Georgopoulos NA, et al. The influence of intensive physical training on growth and pubertal development in athletes. Ann N Y Acad Sci. 2010;1205:39–44.
- Malina RM. Body composition in athletes: assessment and estimated fatness. Clin Sports Med. 2007;26(1):37–68.
- Malina RM, Geithner CA. Body composition of young athletes. Am J Lifestyle Med. 2011;5:262–78.
- 39. Wells JC, Fewtrell MS. Measuring body composition. Arch Dis Child. 2006;91(7):612-7.
- 40. Peltenburg AL, et al. Biological maturation, body composition, and growth of female gymnasts and control groups of schoolgirls and girl swimmers, aged 8 to 14 years: a cross-sectional survey of 1064 girls. Int J Sports Med. 1984;5(1):36–42.
- 41. Christo K, et al. Bone metabolism in adolescent athletes with amenorrhea, athletes with eumenorrhea, and control subjects. Pediatrics. 2008;121(6):1127–36.
- 42. Ackerman KE, et al. Bone microarchitecture is impaired in adolescent amenorrheic athletes compared with eumenorrheic athletes and nonathletic controls. J Clin Endocrinol Metab. 2011;96(10):3123–33.
- 43. Iuliano-Burns S, Mirwald RL, Bailey DA. Timing and magnitude of peak height velocity and peak tissue velocities for early, average, and late maturing boys and girls. Am J Hum Biol. 2001;13(1):1–8.
- 44. Wilmore JH. Body composition in sport and exercise: directions for future research. Med Sci Sports Exerc. 1983;15(1):21–31.
- 45. Katzman DK, et al. Clinical and anthropometric correlates of bone mineral acquisition in healthy adolescent girls. J Clin Endocrinol Metab. 1991;73(6):1332–9.
- 46. Fehily AM, et al. Factors affecting bone density in young adults. Am J Clin Nutr. 1992;56(3):579–86.
- 47. Ferry B, et al. Bone health during late adolescence: effects of an 8-month training program on bone geometry in female athletes. Joint Bone Spine. 2013;80(1):57–63.
- 48. Maimoun L, et al. Peripubertal female athletes in high-impact sports show improved bone mass acquisition and bone geometry. Metabolism. 2013;62(8):1088–98.
- 49. Burt LA, et al. Skeletal adaptations associated with pre-pubertal gymnastics participation as determined by DXA and pQCT: a systematic review and meta-analysis. J Sci Med Sport. 2013;16(3):231–9.
- 50. Ducher G, et al. Effects of repetitive loading on the growth-induced changes in bone mass and cortical bone geometry: a 12-month study in pre/peri- and postmenarcheal tennis players. J Bone Miner Res. 2011;26(6):1321–9.
- 51. Kontulainen S, et al. Good maintenance of exercise-induced bone gain with decreased training of female tennis and squash players: a prospective 5-year follow-up study of young and old starters and controls. J Bone Miner Res. 2001;16(2):195–201.
- Nattiv A, et al. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 2007;39(10):1867–82.
- 53. Tenforde AS, Fredericson M. Influence of sports participation on bone health in the young athlete: a review of the literature. PM R. 2011;3(9):861–7.
- Kimm SY, et al. Decline in physical activity in black girls and white girls during adolescence. N Engl J Med. 2002;347(10):709–15.
- 55. Dumith SC, et al. Physical activity change during adolescence: a systematic review and a pooled analysis. Int J Epidemiol. 2011;40(3):685–98.
- 56. Naughton G, et al. Physiological issues surrounding the performance of adolescent athletes. Sports Med. 2000;30(5):309–25.
- 57. Vescovi JD, et al. Physical performance characteristics of high-level female soccer players 12–21 years of age. Scand J Med Sci Sports. 2011;21(5):670–8.
- Martinez-Lagunas V, Niessen M, Hartmann U. Women's football: player characteristics and demands of the game. J Sport Health Sci. 2014;3:258–72.
- Caine D, et al. An epidemiologic investigation of injuries affecting young competitive female gymnasts. Am J Sports Med. 1989;17(6):811–20.

- Granan LP, et al. The Scandinavian ACL registries 2004–2007: baseline epidemiology. Acta Orthop. 2009;80(5):563–7.
- 61. Renstrom P, et al. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. Br J Sports Med. 2008;42(6):394–412.
- 62. LaBella CR, et al. Anterior cruciate ligament injuries: diagnosis, treatment, and prevention. Pediatrics. 2014;133(5):e1437–50.
- 63. Stracciolini A, et al. Anterior cruciate ligament injuries in pediatric athletes presenting to sports medicine clinic. A comparison of males and females through growth and development. Sports Health. 2015. doi:10.1177/1941738114554768.
- 64. Maffulli N, et al. Aetiology and prevention of injuries in elite young athletes. Med Sport Sci. 2011;56:187–200.

Chapter 2 Sports Nutrition for the Young Female Athlete

Jan P. Hangen and Katrina Schroeder

The 1970s was an important era for the growth of sports opportunities for women. It was also during this period that there was an increase in the number of laboratories focusing on both exercise physiology and sports nutrition. Initially, studies focused mainly on endurance sports and the nutritional status of male participants. Results showed that what an athlete consumed before, during, and after exercise could help to improve performance [1]. The importance of studying both male and female athletes is better recognized today because there are significant differences. For example, food portions, nutrient requirements, and metabolic rates often differ between females and males.

Optimal nutrition enhances athletic training, performance, and recovery, and it also decreases fatigue and injury risk [2]. When addressing nutritional needs of young female athletes, many factors need to be considered. Adolescence is a time of great change in a young woman's body. Females gain an average of 39 pounds and can grow 2–10 inches in height during puberty [3]. Exercise is important during this time in order to help bone mineral accrual and strength, but there can be negative effects on bones if nutritional needs are not met. Overall caloric intake is critical, and there are also specific calcium, vitamin D, and iron requirements for adolescent girls [3]. In addition, caloric and nutrient needs change with training cycles and sports seasons. For example, if an athlete is involved in an endurance

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sport in the fall, she may need to increase her caloric intake. If she is doing more sprint work in the spring, she may need to adjust the number of calories she consumes and focus more on adequate carbohydrate intake.

Periodic anthropometric and dietary assessment by a professional is important as the young athlete grows and matures. Throughout her life cycle, an athlete should strive to maintain a diet to support the short-term goals of her training and performance, as well as the mid- and long-term goals of optimal growth, development, and health. The purpose of this chapter is to offer guidance on dietary assessment, as well as nutrition and hydration recommendations for young female athletes.

Definitions

Macronutrients: Carbohydrate, protein, and fat are the three *macro* (large) nutrients that the body needs from food in order to function.

Micronutrients: Vitamins and minerals are the *micro* (small) nutrients that the body relies on for multiple functions. Examples of micronutrients include vitamins, like folate and beta-carotene, and minerals, such as calcium and iron.

Electrolytes: Minerals that athletes need in order to remain hydrated, especially when sweating. Electrolytes include calcium, potassium, sodium, chlorine, phosphate, and magnesium. These can be found in food as well as specially formulated sports drinks.

Glycemic Index (GI): GI is a relative number that describes the potential of a food to raise blood glucose and subsequently insulin. Foods are compared to white bread or pure glucose to obtain a number called the GI [4]. In general, a food with a high GI has a greater potential to raise blood glucose and be rapidly absorbed, whereas one with a lower GI has a slower rate of digestion and absorption [4, 5].

Epidemiology

The "standard American diet" is often referred to by the fitting abbreviation of SAD. This tends to refer to a diet that is high in simple carbohydrates and saturated fat and low in fruits and vegetables. Teenage girls as a whole are not getting enough fruits and vegetables, with one study showing that only 10 % of girls reached the recommended amounts [6]. In addition, teenage girls tend to be lacking in key nutrients necessary for growth and development, such as calcium, magnesium, potassium, and vitamins D and E, while their diet is higher than other populations in solid fats and added sugars [6]. The prevalence of overweight and obesity reflects these dietary trends, with 15 % of girls ages 2–19 considered obese [7].

Assessment

A complete nutrition assessment of an athlete should include a survey of all available nutritional, medical, biochemical, clinical, and psychosocial data. Dietary assessment analyzes the diet for total calories and content of macronutrients and micronutrients [8, 9]. It is important that the diet recall reflects the athlete's typical intake, with both weekday and weekend intake represented. Under-reporting of energy intake is widespread among some athletes, and over-reporting may be common among those with restrictive eating disorders [10]. Thus, both possibilities should be addressed when interpreting the assessment. Other issues that should be assessed are knowledge and adequacy of portion sizes, frequency of snacking, fluid intake, use of vitamin, mineral and alternative nutrition supplements, weight-control practices, and seasonality of sport activities and food consumption [8, 9]. A comprehensive assessment can help identify the athlete's individual needs.

The following questions can help when formulating individualized and sports-specific recommendations for the young female athlete:

- · Does the athlete desire to reduce body weight to enhance performance?
- Is the athlete at risk for disordered eating, menstrual disturbances, and/or low bone mineral density related to overemphasis on low body weight and inadequate nutrition [11]?
- Is the athlete at risk for low iron status related to a diet low in iron bioavailability (often seen in vegetarian diets) [12]?
- Has energy restriction affected the athlete's growth [11]?
- Does the athlete have optimal food and fluid intake during both short and long training sessions, as well as for optimal recovery after and between training sessions [9]?
- Does the athlete use ergogenic aids or performance-enhancing supplements [13]?
- Does the athlete ingest adequate protein to preserve and/or increase lean body mass during resistance training and to repair muscle in recovery [11]?

Once the diet assessment has been completed, the data need to be compared to nutrient standards in order to offer recommendations to enhance the athlete's nutrition status [9].

Energy Intake

The young female athlete needs a healthy mix of carbohydrate, protein, and fat to ensure proper energy levels. Female athletes have the same basic nutritional needs as non-athletes with some differences, namely increased calories. The total number of calories for athletes also varies depending on the sport [5]. When counseling an athlete about how to design a diet with optimal total calories, the current diet should be analyzed and then compared with a recent weight history, the athlete's sport and training regimen, as well as the frequency and intensity of exercise [5, 9].

Balancing energy intake and expenditure is critical for preventing an energy deficit or excess. Energy deficits can cause short stature, delayed puberty, menstrual dysfunction, loss of muscle mass, and increased susceptibility to fatigue, injury or illness [14]. Energy excess can result in overweight and obesity [2, 14].

Before puberty, caloric needs are similar for boys and girls. For adolescents however, the energy requirements are more variable, depending on age, growth rate, stage of physical maturity, and level of activity [2]. Additional calories are needed during growth spurts, and, on average, during periods of rapid growth, adolescents should consume 500 kcal/day over their normal intake [2]. Consultation with a registered dietitian can help to determine caloric needs, not only for proper growth, but for athletic performance as well.

The percentages of macronutrients may differ depending on the stage of development of the athlete, as well as whether the athlete is growing optimally or in need of catch-up growth. The plan should also address needs for training, competition, preevent, and/or post-event recovery nutrition [5]. In general, teenage girls need the same amount or less macro- and micro-nutrients than their male counterparts with one exception: iron. Female athletes require more iron, and most do not get enough [3].

Carbohydrate

The general guidelines for athletes recommend 50–65 % of calories from carbohydrate, with endurance athletes needing as much as 70 % of total calories from carbohydrate [5]. Female athletes, compared to male athletes, are less likely to consume the recommended amount of carbohydrates. This is often due to a desire to limit total caloric intake in order to reduce body weight and/or body fat [15].

The typical daily diet in the US provides approximately 4–5 g of carbohydrate per kilogram of body weight daily. Carbohydrate recommendations for athletes range from 6 to 10 g/kg/day, with an average intake of 5–7 g/kg/day for general training, increased to a total of 7–10 g/kg/day for those athletes training for endurance events. Each athlete's carbohydrate prescription is best determined individually, based on body weight, sports-specific considerations, energy balance, and adequacy of macronutrients and micronutrients [5].

Carbohydrates should be consumed before, during, and after exercise. Prior to exercise, carbohydrates provide energy to maintain daily activities of living, as well as a pool of calories to spare, or make protein available for muscle growth and repair [5]. Athletes should consume 1–4 g of carbohydrate per kilogram of body weight 1–4 h prior to exercise; no more than 1 g/kg is optimal in the last hour before exercise to help avoid the risk of gastrointestinal distress during activity [5]. High-intensity athletes may require 5–8 g/kg. Recommendations are higher for endurance athletes, who need an average of 7–10 g/kg [5] (Table 2.1).

Table 2.1	Basic sp	ports nutrition	carbohydrate	prescription	summary

• Before exercise: 1–4 g of carbohydrate per kilogram ingested 1–4 h prior to exercise
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- During exercise: 30-60 g of carbohydrate consumed every hour
- After exercise: 1–1.5 g per kilogram of body weight within 30 min after exercise with continued intake of carbohydrate over several hours for a total of 1 g per kilogram of body weight per hour [5]

It is best to avoid pre-exercise meals that are high in protein, as they may take longer to digest and absorb. The protein content of the pre-exercise meal should be approximately 10-15 % [5, 16]. In addition, only about 15-20 % of the calories in the pre-exercise meal should come from fat [5], as fat tends to leave the stomach slowly, thereby delaying the absorption of carbohydrates. Examples of a favorable pre-exercise meal would be whole-wheat toast with peanut butter (or other nut butter), or oatmeal with fruit and milk (Table 2.2).

During exercise, carbohydrates serve to maintain blood glucose stores at higher levels, thus increasing the use of blood glucose when muscle glycogen stores may be lower. Carbohydrates during exercise are especially important for endurance sports such as running, but also with sports such as tennis, basketball, soccer, and cycling that require repeated periods of high-intensity, short-duration efforts [16]. During exercise, it is recommended that athletes consume 30–60 g of carbohydrate per hour. This translates to 120–240 kcal of carbohydrate per hour. The carbohydrate can be obtained from either solid or liquid foods, depending on the athlete's preference [5].

Athletes with more intense training regimens, or higher body weights may not be able to consume enough solid food per day to meet daily caloric and carbohydrate needs; therefore, a high calorie sports beverage may be helpful. Liquid supplements with a high percentage of carbohydrate (18–24 %), as well as glucose polymers, may be useful if an athlete has trouble meeting caloric needs with solid foods [9]. These supplements are not designed to replace food intake, but are for additional calories, carbohydrate, and fluid during heavy training [9].

Energy bars are one of the most popular options for snacks during exercise. The ideal bar is one that has at least 80 % carbohydrate and less than 10 % fat, to help avoid the delayed absorption of carbohydrates [5]. Some protein is also helpful, particularly for endurance athletes, as up to 10 % of the energy during exercise may come from protein stores in the body.

Some athletes prefer to consume liquid supplements. Most sports beverages provide about 15–20 g of carbohydrate per 8–12 oz, thus at least 8 oz every half hour should help maintain an adequate supply of carbohydrates. Both liquid and solid carbohydrates will supply adequate energy during exercise. For comparison, sports gels provide an average of 25 g of carbohydrate, one large banana provides 30 g, and an average sports bar provides 66 g [5, 16].

After exercise, carbohydrates are particularly important in restoring muscle glycogen stores to ensure optimal performance for consecutive exercise sessions. Athletes who train hard over a period of days or several times in 1 day, as well as those who are competing in events that last more than 1 day, need to ensure that they have a recovery snack after each exercise session. Research shows that consuming

Table 2.2	Sample meal	plans for ever	nt fueling
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The night before the event

- · Baked potato with turkey chili, low-fat cheese, and broccoli with a side of fresh fruit
- · Brown rice with stir-fried tofu, mixed vegetables, and pineapple
- · Pasta with meat or vegetarian pasta sauce served with spinach salad and fresh fruit
- · Tortilla filled with sautéed vegetables, meat or scrambled eggs, and salsa
- Salmon with rice pilaf, kale, and fruit salad

The morning of the event

2-3 h before: small to regular-size meal and liquids

- · Cereal with sliced banana and slivered almonds in low-fat dairy or soy milk
- · Homemade breakfast sandwich with two eggs, toast, and tomato with orange juice
- Oven-roasted breakfast potatoes, served with Canadian bacon, banana slices and low-fat dairy or soy milk

· Oatmeal with mixed berries, served with low-fat dairy or soy milk

1-2 h before: small snack and liquids

Granola bar

- Raisins or Banana
- 1/2 bagel with peanut or other nut butter
- Small turkey wrap

• Water or alternating water and sports drink

During the event

- Throughout: alternate drinking water and sports drinks (6–12 oz) every 15 min. Use bars and gels as needed depending on the intensity of the event
- During breaks <2 h: snack on granola bars, pretzels, crackers, oatmeal cookies, trail mix, fig bars, yogurt, and bananas
- During breaks ≥2 h: turkey, ham, or chicken sandwiches, peanut butter and jelly or banana sandwiches, oranges, apples and/or grapes, cheese sticks, pudding cups and squeeze yogurts, as well as the above options

After the event: consume recovery snacks with an approximate ratio of 4-5 g of carbohydrate to 1 g of protein

- Chocolate milk
- Egg sandwich and orange juice
- Baby carrots or pretzels with hummus
- · Low-fat string cheese and a banana
- · Peanut butter sandwich and milk
- Burrito with brown rice, black beans, cheese, and veggies

carbohydrates within 15–30 min after exercise will provide optimal muscle glycogen re-synthesis, whereas delaying the ingestion of carbohydrates by over an hour may reduce muscle glycogen stores up to 50 % or more [16]. The amount of carbohydrate recommended after exercise is 1.0–1.5 g per kilogram of body weight within 30 min after exercise with continued intake of carbohydrate over several hours for a total of 1.0 g per kilogram of body weight per hour [5, 16].

Common recovery foods include pasta, granola, oatmeal raisin cookies, fig bars, potatoes, bananas, and chocolate milk (Tables 2.2 and 2.3). The optimal carbohydrate

High glycemic index				
Glucose and sucrose				
Corn flakes, cheerios, rice krispies				
Waffles, pancakes, and bagels				
• White bread				
Baked potato				
• Honey				
• Banana				
Rice cakes				
English muffins				
Pop-Tarts				
Medium glycemic index				
• Raisins				
Sweet potato				
• Pasta				
• Peas				
• Apple				
Orange juice				
• Oatmeal				
Low glycemic index				
• Milk				
• Yogurt				
Raw celery, peppers, and carrots				
Peanuts and tree nuts				
• Beans				
• 100 % bran cereals				
Bulgur and barley				
Ice cream				

Table 2.3 Examples of low, medium, and high glycemic index foods

Adapted from the International Tables of Glycemic Index and Glycemic Load Values: 2008 [17]

to protein ratio for a recovery snack is 4:1 grams. Although no conclusive data have been found that suggest that the addition of protein aids in restoring muscle glycogen, it may enhance muscle repair [5].

Increasingly, athletes are using glycemic index (GI) to make food choices that may enhance performance. Some propose that lower GI foods are best before exercise to promote sustained and slower absorption of carbohydrates for longer lasting energy. Moderate to high GI foods with faster carbohydrate absorption may be recommended during and after exercise to ensure optimal muscle glycogen stores and enhanced recovery [4, 5]. Recognizing the glycemic index (GI) of individual foods can be beneficial (Table 2.3), as can using combinations of foods to either slow or speed absorption of carbohydrates. Fat, fiber, and protein can all be used to slow carbohydrate absorption. Athletes can experiment with combinations of foods when determining the best pre- and/or post-exercise foods. For example, a pre-event snack of whole grain bread, packed with fiber, may be further slowed in absorption with the addition of a nut butter to supply longer lasting energy. In determining which foods are most beneficial for training and competition, athletes would be wise not only to consider GI, but also to take into account the nutrient value, taste, cost, preparation, and portability of the food [4, 5].

Protein

Protein is important in tissue building and repair, and can also be used as a fuel source by the body. The Dietary Reference Intake for protein is 0.95 g per kilogram for individuals 4–13 years of age, 0.85 g per kilogram for individuals 14–18 years of age, and 0.8 g per kilogram per day for individuals above 18 years of age. These amounts do not take into account differences in physical activity levels. The American College of Sports Medicine, the Academy of Nutrition and Dietetics, and the Dietitians of Canada have all agreed that active individuals have higher protein requirements; their joint position statement, "Nutrition and Athletic Performance," recommends that endurance athletes should consume 1.2–1.4 g of protein per kilogram per day, and that strength or resistance-trained athletes may need to consume up to 1.6–1.7 g per kilogram per day [18].

One of the reasons that athletes may require more protein than sedentary individuals is that protein may contribute 5-15 % of the fuel during exercise. Exercise can cause muscle breakdown that increases the amount of protein needed. Endurance athletes may need more protein as some lose a small amount of protein in the urine, whereas protein is typically not lost this way in healthy individuals who engage in little to no exercise [5]. Other athletes who may require more protein are those who restrict food intake in order to achieve a desired weight, or those who practice some forms of vegetarianism [5, 11]. Adolescent females who are still growing, especially those who have not had their pubertal growth spurt, may also require more protein [5].

Protein requirements remain somewhat controversial among athletes. Many falsely believe that they need more protein than they actually do, and some may consider using protein supplements. These supplements are often marketed as ways to gain extra muscle quickly. However, the primary way by which protein supplements help an athlete gain muscle is through the provision of extra calories, thereby sparing the dietary protein for growth and repair of muscle.

The average American diet generally provides ample protein [5, 12]. Excess protein may be detrimental to optimal performance and may lead to complications such as impaired kidney function [5]. A high protein diet may also indicate a higher fat diet, which in some cases may increase the risk of certain diseases such as cardiovascular disease [12]. In summary, adequate, but not excessive, protein should be encouraged for all athletes.

Fat

Dietary fat provides essential fatty acids the body cannot synthesize on its own, facilitates the absorption of fat-soluble vitamins, and is an energy source when other sources of energy, such as glucose, are depleted [12]. The amount of fat in an athlete's diet varies depending on the total number of calories consumed, the type of sport, and the level of training. In general, female athletes should follow the same guidelines as the general public with 20-35 % of total calories from fat, and saturated fat making up less than 10 % of the total calories. This equates to about 1 g of fat per kilogram of body weight [5, 12]. As with all macronutrients, dietary fat should be individualized, taking into account an athlete's growth stage, nutrition needs, taste preference, physical activity level, energy expenditure, and sports-specific considerations [5].

Athletes in appearance sports, such as gymnastics, dance, and figure skating, may have less than optimal fat intake, and even go well below the recommended range [10, 12]. Very low-fat diets, those with less than 20 % of total calories from fat, are not recommended. Unfortunately, some athletes trying to achieve lower body weight or body fat may end up increasing their intake of carbohydrates and/or protein and failing to consume an adequate amount of dietary fat. For some athletes, especially those who are still growing, a low-fat diet may not provide sufficient energy to meet both the needs of growth and activity. Furthermore, the consumption of very low-fat diets may omit dairy products, meat, fish, poultry, and eggs, leading to deficiencies of calcium, iron, and zinc, as well as causing limited absorption of fat-soluble vitamins [10, 12].

Vitamins and Minerals

Vitamins and minerals are found in a variety of food sources and are necessary for the proper functioning of all body tissues. They are essential for the release of energy from macronutrients (carbohydrates, protein, and fat) by serving as enzymes and cofactors for metabolism. They are called micronutrients because, despite their importance in many of the body's functions, they are only needed in small amounts [5]. Some female athletes mistakenly think they need to take mega-doses of vitamins and minerals to achieve optimal performance [5, 16]. Athletes who consume enough calories in a varied diet with whole foods, and who keep in mind increased caloric needs as physical activity increases, will most likely have sufficient vitamins and minerals for optimal performance.

Female athletes may require more of certain vitamins and/or minerals if they have monotonous and/or restrictive diets, consume vegetarian diets, or have certain medical conditions, such as food allergies or malabsorptive disorders, including lactose intolerance, inflammatory bowel disease, or celiac disease [5, 12].

Adequate iron intake is important for all female athletes. Iron deficiency anemia is a concern for many female athletes, not just those with restrictive eating patterns or vegetarians. Females in general, including athletes, are at higher risk of iron deficiency anemia than their male counterparts [19]. Some young females may require appropriate use of a multivitamin and mineral supplement with iron, or iron supplementation alone [12]. If there is a question of iron deficiency, it is best to consult with a physician or registered dietitian to determine whether supplementation is necessary.

Anemia

Anemia, lack of healthy red blood cells or hemoglobin, can cause fatigue and other health problems. For athletes, it can negatively affect performance and make it difficult to train. There are many different causes of anemia, and iron deficiency anemia is the most common.

The young female athlete may be at risk of iron-deficiency anemia for several reasons, including:

- Blood loss during menstruation.
- Gastrointestinal blood loss in distance runners [20].
- Loss of iron through sweat [21, 22].
- Destruction of blood cells due to repeated impact activities, referred to as "foot strike hemolysis" [23, 24].

Young female athletes also need to make sure that they are getting enough calcium and vitamin D. Calcium is necessary for bone health, and vitamin D helps the body absorb it. Adequate calcium and vitamin D can help reduce the risk of stress fractures from occurring at a young age and osteoporosis from occurring later in life [12]. Girls younger than age 8 years need 1000 mg/day of calcium and girls ages 9–18 need 1300 mg/day [3]. This amount can be easily consumed through food and beverages, such as milk, soymilk, dairy products, cabbage, kale, broccoli, fortified orange juice, and fortified tofu. However, the body can only absorb up to 500 mg at a time, so multiple servings of calcium-rich food daily are ideal.

Adequate vitamin D can be harder to obtain through the diet alone. Dietary sources include fortified milk, fortified orange juice, fortified cereal, salmon, tuna, and mushrooms, and many people get enough vitamin D from sunlight during the summer. However, supplements are often needed during the winter months and in those who consistently use sunscreen or remain indoors. Girls of any age should be getting at least the recommended 600 IU/day of vitamin D [12]. In some individuals, this amount is inadequate to achieve the minimal recommended blood concentration of 30–32 mg/ml of 25-OH vitamin D. Thus checking a blood level in the winter months can help guide dosing.

Many female athletes trying to improve performance seek dietary supplements other than vitamins and/or minerals. These supplements may include protein powders, energy drinks or bars, sports drinks, and other substances claiming to enhance athletic performance, aid in weight loss, or increase muscle. Ideally, supplements should not be used as a substitute for an optimal diet of a variety of whole foods. Health risks of supplements, not regulated by the Food and Drug Administration (FDA), continue to be investigated with no concrete guidelines for safety or efficacy. The best ergogenic aids are proper nutrition and hydration [5, 13].

Hydration

Proper hydration is essential to good sports nutrition. Young female athletes should make a conscious effort to drink fluids throughout the day, as well as before, during, and after exercise. Water functions to regulate body temperature, transport waste products and nutrients, and assist with many biochemical reactions for energy production [5]. As is the case with food, fluid needs also should be individualized. The amount of fluid necessary depends on the individual's age, size, level of physical activity, and the environmental temperature. In general, most athletes need 1–2 cups prior to exercise and 0.5–1 cup every 15–20 min during exercise [12].

Fluids may include plain water, juice, sports beverages, caffeinated and decaffeinated beverages, and soups. Many athletes prefer the taste of sports drinks to water. However, a sports drink is generally not necessary unless exercise lasts more than 60–90 min or occurs in hot weather. In these conditions, carbohydrates and electrolytes in sports beverages may help to improve performance. Sports drinks are best absorbed if they are consumed full strength instead of diluted. They are formulated to empty from the stomach rapidly. Diluting these drinks may not deliver glucose and electrolytes optimally. Drinking carbonated drinks or juice during exercise is not recommended as they may cause cramping, bloating, or diarrhea [5, 12].

A simple way for an athlete to gauge fluid need is to weigh herself before and after a workout. A minimal weight loss usually means that there is adequate fluid intake and replacement. A weight loss of more than 2 pounds suggests inadequate fluid intake and possibly dehydration. For every pound of body weight lost during exercise, 16–20 oz of fluid should be consumed to appropriately replace lost fluids. Gaining weight during exercise may be a sign of excessive drinking, or overhydration, and should be avoided [5, 12].

Symptoms of dehydration may include cramping, dizziness and, in severe cases, heat stroke [5, 18]. An athlete should drink often during exercise and monitor her hydration status by checking her urine color. If urine is light yellow or straw-colored, then the athlete is most likely properly hydrated. However, if it is dark, the urine may be concentrated, suggesting inadequate hydration [5, 12].

Weight Considerations

While some sports require an athlete to lose or maintain weight to optimize performance, other sports require weight gain for a greater competitive advantage. Female athletes participating in appearance or lean sports (e.g. gymnastics, figure skating, dance, running, and swimming) may feel pressure to lose weight to achieve an ideal thinness. Some athletes turn to restrictive eating and the use of unsafe dieting practices to achieve a lower body weight. This may compromise both the athlete's ability to perform, as well as her health [10, 12].

The Female Athlete Triad (Triad) is a syndrome in which low energy availability (with or without intended restrictive eating), amenorrhea, and decreased bone mineral density are present. The Triad is a serious condition with lifelong health consequences, some of which can potentially be fatal [10]. A multidisciplinary approach to the treatment of this disorder is recommended, and includes a medical doctor, dietitian, and a therapist [5]. Additional details about this condition can be found in Chapter 5.

Weight gain is important for those who are underweight, or who require greater strength, as it increases energy and endurance. Weight gain is achieved by ensuring that each meal and snack has healthful, higher calorie foods. The overall goal is to gain about 0.5–1.0 pound per week. Athletes trying to gain weight should consume three meals and 2–3 snacks each day. Skipping meals such as breakfast may make it more difficult to obtain the necessary calories needed to gain weight. If extra calories are needed, there are liquid nutrition supplements available, which contain extra calories and are marketed specifically for weight gain [12].

Summary

Young female athletes, and those who support them, should strive to understand which foods provide the best sources of energy, along with when and how to eat before, during, and after exercise. Nutrition is an important part of sports performance. Energy, macronutrients, micronutrients, and fluids in the proper amounts are essential for optimal growth and activity. The athlete's needs evolve as she grows and matures and as her training cycles and sports seasons change. Each athlete should consider constructing her diet carefully, and professional help from a registered sports dietitian is advised. A dietitian can assess growth, identify athletes who are under- or overweight, monitor athletes with overuse injuries related to poor nutrition, and identify those with or at risk of disordered eating (Table 2.4).

www.eatright.org	
http://www.fueluptoplay60.com/	
www.gssiweb.com	
www.humankinetics.com	
http://www.jissn.com/	
http://btc.montana.edu/olympics/nutrition/	
www.urbanext.illinois.edu/hsnut	
http://www.extension.iastate.edu/ humansciences/sports-nutrition	
http://www.teamusa.org/About-the-USOC/ Athlete-Development/Sport-Performance/ Nutrition/Resources-and-Fact-Sheets	
http://kidshealth.org/parent/nutrition_ center/dietary_needs/feed_child_athlete. html#cat20754	
http://www.youngwomenshealth.org/ nutrition-sports.html	

Table 2.4 Recommended sports nutrition websites

References

- 1. Dunford M. Fundamentals of sport and exercise nutrition. Champaign, IL: Human Kinetics; 2010.
- 2. Hoch AZ, Goossen K, Kretschmer T. Nutritional requirements of the child and teenage athlete. Phys Med Rehabil Clin N Am. 2008;19(2):373–98.
- Story J, Strang M. Guidelines for adolescent nutrition services. Minneapolis, MN: Center for Leadership, Education and Training in Maternal and Child Nutrition, Division of Epidemiology and Community Health, School of Public Health, University of Minnesota; 2005.
- 4. Burke LM, Collier GR, Hargreaves M. The glycemic index a new tool in sport nutrition? Int J Sport Nutr. 1998;8:401–15.
- 5. Rosenbloom C. Sports nutrition: a practice manual for professionals. 4th ed. Chicago (IL): American Dietetic Association; 2006.
- Moore L, Singer M, Qureshi M, et al. Food group intake and micronutrient adequacy in adolescent girls. Nutrients. 2012;4:1692–708. doi:10.3390/nu4111692.
- Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999–2010. JAMA. 2012;307(5):483–90.
- Burke L, Deakin V. Clinical sports nutrition. 3rd ed. Sydney, Australia: The McGraw Hill Companies; 2007.
- 9. Burke L. Practical sports nutrition, vol. 1. Champaign, IL: Human Kinetics; 2007.
- Nattiv A, Loucks AB, Manore MM, Sanborn CF, Sundgot-Borgen J, Warren MP. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 2007;39:1867–82.

- 11. Bar-Or O. Nutritional considerations for the child athlete. Can J Appl Physiol. 2001;26(Suppl):S186–91.
- Clark N. Nancy Clark's sports nutrition guidebook. 4th ed. Champaign, IL: Human Kinetics; 2008.
- 13. Maughan RJ, Depiesse F, Meyer H. The use of dietary supplements by athletes. J Sports Sci. 2007;25:S102–13.
- Meyer F, O'Connor H, Shirreffs SM, International Association of Athletics Federations. Nutrition for the young athlete. J Sports Sci. 2007;25 Suppl 1:S73–82.
- Burke LM, et al. Guidelines for daily carbohydrate intake: do athletes achieve them? Sports Med. 2001;31(4):267–99.
- Burke LM, Kiens B, Ivy JL. Carbohydrates and fat for training and recovery. J Sports Sci. 2004;22:15–30.
- 17. Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. Diab Care. 2008;31(12):2281–3.
- Position of the American Dietetic Association, Dietitians of Canada, and the America College of Sports Medicine: nutrition and athletic performance. J Am Diet Assoc. 2009;100: 1543–56.
- 19. Suedekum NA, Dimeff RJ. Iron and the athlete. Curr Sports Med Rep. 2005;4(4):199-202.
- Nachtigall D, Nielsen P, Fischer R, Engelhardt R, Gabbe EE. Iron deficiency in distance runners. A reinvestigation using Fe-labelling and non-invasive liver iron quantification. Int J Sports Med. 1996;17(7):473–9.
- DeRuisseau KC, Cheuvront SN, Haymes EM, Sharp RG. Sweat iron and zinc losses during prolonged exercise. Int J Sport Nutr Exerc Metab. 2002;12(4):428–37.
- 22. Waller MF, Haymes EM. The effects of heat and exercise on sweat iron loss. Med Sci Sports Exerc. 1996;28(2):197–203.
- 23. Shaskey DJ, Green GA. Sports haematology. Sports Med. 2000;29(1):27-38.
- Janakiraman K, Shenoy S, Sandhu JS. Intravascular haemolysis during prolonged running on asphalt and natural grass in long and middle distance runners. J Sports Sci. 2011;29(12): 1287–92.

Chapter 3 Resistance Training for Young Female Athletes

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Introduction

In 1976 researchers demonstrated that injuries sustained by female athletes, aside from those related to different biological structures, are essentially no different than those of males, and "well-trained" female athletes are no more prone to injury than their male counterparts [1]. Research has advanced greatly since that time; the evidence continues to mount regarding the health and fitness-related benefits of resistance training for girls and young women, and the concept of training female athletes for injury prevention has received much deserved attention. Currently, most

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school-aged girls participate in developmentally appropriate resistance training during physical education classes as part of a contemporary curriculum designed to prepare children to live physically active lives [2]. Moreover, a growing number of YMCAs, fitness facilities, and sports training centers now specialize in the fitness market for girls and young women by offering programs specifically designed to enhance muscular fitness, improve sports performance, and reduce the risk of sportsrelated injuries.

Experts in the fields of sports medicine, pediatric exercise science, physical education, and biomechanics recommend that exercise programs for girls and young women include resistance training activity [3–5]. Early teachings by Micheli et al. [6], now supported by recent literature [7], suggest that acute and overuse injuries could be reduced by 15–50 % in female athletes by addressing risk factors, such as poor muscle strength, muscle imbalances, and flexibility deficits. The National Athletic Trainers' Association also estimates that more than half of all sports-related overuse injuries in young athletes may be preventable with simple approaches that include preseason and in-season exercise programs built upon a foundation of resistance training [8]. Furthermore, obesity treatment and prevention, via the promotion of safe and effective physical activity participation, are additional benefits that may arise from resistance training [9–15].

This chapter focuses on dispelling the myths associated with resistance training for girls and young women, followed by a discussion of the observable benefits of resistance training in this population, including muscular fitness development, improvement of bone mass, physical activity promotion, sports injury reduction, and obesity prevention.

Definitions

Resistance training: A method of conditioning, also called *strength training*, that involves the progressive use of a wide range of resistive loads, different movement velocities, and a variety of training modalities, including free weights (barbells and dumbbells), weight machines, resistance bands, suspension systems, and medicine balls, to improve muscle strength, muscular power, and local muscular endurance.

Muscular Fitness: An inclusive term that refers to muscle strength, muscular power, and local muscular endurance.

Weightlifting: A competitive sport that involves the performance of the snatch and clean and jerk lifts. Weightlifting training refers to a variety of explosive multi-joint exercises that are performed by controlled movements, requiring a high degree of technical skill and qualified instruction.

Concentric muscle contraction: Activation of a muscle while the muscle shortens. During this type of contraction, the external force on the muscle is less than the force the muscle is generating.

Eccentric muscle contraction: Active contraction of a muscle occurring simultaneously with lengthening of the muscle.

Peak height velocity (PHV): A somatic indicator of physical maturity that reflects the maximum velocity of growth in stature during adolescence.

Importance of the Issue

There are many myths and concerns about resistance training in children and adolescents. One of the main objectives of this chapter is to examine and dispel these myths.

Myth 1: Resistance Training Is Unsafe for Youth

There is no evidence that resistance training will negatively impact growth and maturation in children and adolescents [16]. Injury to the growth cartilage has not been reported in any prospective youth resistance training research studies. Growth cartilage injuries described in case reports from the 1970s and 1980s typically resulted from improper lifting techniques, poorly chosen training loads, or lack of qualified adult supervision [3]. A recent review of the literature indicated that there is a relatively low risk of injury in children and adolescents who follow age-appropriate resistance training guidelines, which include qualified supervision, constructive feedback on exercise technique, and a sensible progression of training loads and volume [12].

Examination of data from resistance training-related injuries in patients presenting to United States emergency rooms demonstrated that many injuries were likely related to accidents and poor technique; notably, two-thirds of the injuries sustained by 8- to 13-year-old patients were to the hand and foot and were most often related to "dropping" and "pinching" in the injury descriptions. The incidence of joint sprains and muscle strains was higher in the older age groups [17]. Quatman et al. found that males may suffer more exertional-type resistance injuries to the trunk during resistance training, while females may be more susceptible to lowerextremity injuries resulting from accidents during resistance training [18]. These studies collectively support the assertion that resistance training for young females can be safe and effective when conducted with qualified guidance and supervision.

Furthermore, resistance training designed to enrich the motor learning environment in young females may help children with low motor skill confidence and competence catch up to their peers, as well as serve to reduce injury risk factors [7]. The recently published "Position statement on youth resistance training: the 2014 International Consensus" is an excellent resource, as it describes resistance training guidelines for children and adolescents and highlights considerations for young females [5].

Myth 2: Resistance Training Will Not Increase Muscle Strength in Girls

Resistance training can effectively increase girls' muscle strength. Malina reviewed 22 reports dealing with experimental resistance training protocols in both boys and girls before and during early puberty. He found that most programs used weight machines and free weights, 2- to 3-day protocols, and 8- to 12-week durations. Significant improvements in muscular strength during childhood and early adolescence were achieved. Importantly, in the ten studies that systematically monitored injuries, only three injuries were reported, with estimated injury rates of 0.176, 0.053, and 0.055 per 100 participant hours in the respective programs [16]. A meta-analysis investigating the effects of resistance training in children and adolescents did not reveal any significant sex differences in the efficacy of resistance training. However, the authors noted that there was a distinct imbalance in the number of male and female participants (1162 males versus only 317 females) [19].

Myth 3: Resistance Training Creates Bulky Muscles

Muscular strength development is a multidimensional fitness component that is influenced by a combination of muscular, neural, and biomechanical factors. For example, a potential factor for increasing strength during childhood may be related to the maturation of the central nervous system, improvements in motor unit recruitment, firing frequency, synchronization, and neural myelination [5]. In preadolescents, proper resistance training can enhance strength without concomitant muscle hypertrophy. Such gains in strength can be attributed to the aforementioned neurological mechanisms [20]. These mechanisms may account for the increase in strength in populations with low androgen concentrations, including females and preadolescent boys [21]. Assessments of muscular strength in children and adolescents indicate that strength increases in a relatively linear fashion throughout childhood for both boys and girls. As children reach the onset of puberty, they experience rapid growth along with observable non-linear gains in muscular strength. During this period, sex differences in muscular strength begin to emerge, with boys demonstrating accelerated gains as a result of the adolescent spurt, and girls appearing to continue to develop in a more linear fashion [5]. The accelerated gains in strength in males during this time period is largely from increased hormonal concentrations, including testosterone, growth hormone and insulin-like growth factor playing a more significant role.

Magazine cover images of female bodybuilders, especially those who use exogenous steroids, may be one culprit for the persistence of myths regarding resistance training and muscle bulk in females. Physical education (PE) teachers and youth coaches may be able to start early (before high school) and educate children about the benefits of sensible strength training and hormonal differences that limit large gains in muscle hypertrophy in girls. Thought also needs to be given to how strength training is explained and offered to girls. While some girls may shy away from "weightlifting" with "bulky" boys in an afterschool program, girls, especially non-athletes, may to be more interested in fitness activities and toning exercises. Introducing strength training in a non-threatening and educational PE class, for example allowing girls to train with friends in small groups, may promote safe and effective resistance training.

Myth 4: Resistance Training Is Only Beneficial for Athletes

From a public health perspective, all children and adolescents, not just young athletes, should participate in a variety of strength building physical activities as part of play, sports, physical education, planned exercise, and recreation. However, it is crucial that training programs take into account the fitness level of each participant and start at an appropriate point. Young sedentary girls, as well as young athletic girls, should be instructed by qualified pediatric exercise specialists on proper exercise technique and progressive training. The goals of well-designed resistance training programs for youth should not be limited to increasing muscle strength. Since basal muscle strength and neuromuscular control is needed to run, jump, hop, skip, and kick proficiently [22], exercise recommendations for young females should include a variety of enjoyable, age-appropriate activities with resistance training exercises and games as the core. This targeted approach early in childhood may serve to enhance a girl's physical abilities throughout the growth process and into her adult years. Teaching girls about physical fitness, improving coordination, demonstrating safe training procedures, and providing an engaging and enjoyable program promotes a positive attitude toward resistance training and physical activity in general [4]. While enhancing the physical abilities of girls in order to improve sports performance is not a novel concept, it is worth highlighting that a training model that emphasizes muscular strength and motor skill development early in life also serves to provide a developmental approach that supports overall physical and psychosocial development [23-25].

Myth 5: There Is Only One Right Way to Perform Resistance Training Exercises

There is not one optimal combination of sets, repetitions, and exercises that will result in favorable adaptations in muscular strength, fitness performance, and body composition in all youth. By periodically altering the training variables over time, the training stimulus will remain effective and adaptations to the training program will be maximized [26–29]. Strength building throughout all aspects of a training program is

critical. There exists no minimum age requirement for participation in youth resistance training programs provided participants are able to follow directions and safety rules [3]. Most 7- and 8-year-old girls are ready for some type of resistance exercise [27]. Resistance training with free weights (i.e., dumbbells and barbells) (Fig. 3.1), medicine balls, elastic bands, and one's own body weight are beneficial for youth who need to enhance motor skill performance, improve balance, and increase muscle strength and power as part of an integrative neuromuscular training program [12, 25, 26, 30].



Fig. 3.1 Resistance training using proper technique and spotting



Fig. 3.1 Resistance training using proper technique

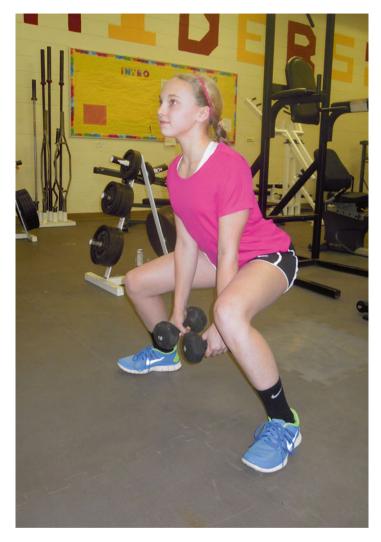


Fig. 3.1 Resistance training using proper technique

Resistance Training and Injury Prevention for the Young Female Athlete

Anatomical changes during growth and development include widening of the pelvis, increase in the Q angle (Fig. 3.2), and changes in center of gravity [31]. See Chapter 1 for additional details on growth and development. Researchers have hypothesized that following the onset of puberty and the initiation of peak height velocity (PHV) core stability decreases due to increased tibia and femur length, increased overall body mass, and increased height of the center of mass. A key finding is that these

Fig. 3.2 The Q angle is the angle formed from the intersection of a line drawn from the anterior superior iliac spine to the central patella with a second line from the central patella to the tibial tubercle



changes are not accompanied by increases in strength or muscle recruitment at the hip and trunk [32, 33]. In other words, as female athletes reach maturity, decreased core stability and deficient core strength may cause increased dynamic lower extremity valgus load during sport specific tasks, placing them at increased risk for injury [34–36]. Intervention efforts with targeted training that includes increasing strength, improving neuromuscular control, and incorporating skill development, can be implemented for females during preadolescence and adolescence to help decrease injury risk factors [25, 28, 37–40]. If these programs are initiated early enough in the maturational years, they are highly effective in reducing injury [7].

A recent meta-analysis of 14 ACL injury prevention studies in female athletes concluded that programs were more successful in preventing knee injuries when training commenced before the onset of neuromuscular deficits and peak knee injury incidence, optimally during early adolescence [7]. Quatman-Yates and colleagues studied changes in lower extremity strength across maturational stages for female student athletes. They found that hip abduction and hamstring-quadriceps ratio strength decreased from prepubertal to pubertal stages, lending further support to strength training for females during preadolescence with the goal of injury prevention

[41]. Neuromuscular training conducted during preadolescence or earlier increases neuromuscular function related to improved biomechanics that may lead to a reduction of ACL injuries as girls progress in growth and sport [31, 36, 37, 39–48].

While some female athletes appreciate the aesthetics of a strong, muscular build, the performing artist athlete may actually fear the development of this type of body habitus. Female performing artists, including dancers, gymnasts, and figure skaters, are a unique group of athletes, who may feel challenged by what can appear to be opposing goals: the need to build strength while also meeting the aesthetic requirements of the discipline, namely appearing thin and sleek. However, data on female ballet dancers have shown that supplemental resistance training for hamstrings and quadriceps can lead to improvements in leg strength, without interfering with key artistic and physical performance requirements [49].

Helping these athletes understand the benefits of resistance training, dispelling the myths associated with weight training, and designing protocols that are conducted in environments familiar to the athlete (e.g. the dance studio, gym, or ice rink) may allow them to feel more comfortable with these training programs. Training protocols should be designed to correct imbalances unique to the discipline. For the ballet dancer, this means conducting resistance training in a parallel lower extremity position, as compared to the turned out position. This technique encourages improvement in strength of the relatively weaker adductor and internal rotator muscles of the lower extremity. Resistance training in this population should include dynamic concentric and eccentric muscle actions, and should be performed through full ranges of motion in keeping with the unique demands of the performing artist athlete. A progressive suspension training protocol in the dance studio, gym, or ice rink, incorporated into training programs throughout the week, is an example of a program that builds strength, keeps young female athletes interested and energized, occurs in the comfort of familiar environments, and can be done along side their peers.

Implementing evidence-based injury prevention protocols in schools and sports training facilities may ultimately promote safe and successful physical activity into adulthood [4, 50]. Without interventions that target deficits in muscular fitness and motor skills early in life, girls will be less likely to engage in the recommended amount of daily physical activity and more likely to experience negative health outcomes [11]. Age-related interventions taught by trained pediatric exercise specialists are needed to prevent the subsequent decline and disinterest in physical activity and upsurge in high-risk behaviors [51]. A population-wide approach to injury prevention should be started early in life, and novel strategies are needed if the lifelong benefits of exercise are to be fully realized [12].

The Benefit of Resistance Training on Bone Mineral Density in the Young Female

Childhood through late adolescence is a critical time period for bone mass accrual, and physical activities characterized by considerable loading have been shown to have the greatest osteogenic, or bone building, effects on the growing skeleton [52].

Resistance training for females during this time period may be an important component of exercise when considering future bone health, in particular for certain groups at high risk for suboptimal bone health, including bony stress injuries, eventual osteopenia, and even osteoporosis [53].

Children who participate in competitive sports and strength building exercise have higher bone mineral density (BMD) than those who do not [54, 55]. Bass et al. reported that prepubertal female gymnasts, whose training mainly involves high-impact and resistance training, had significantly higher BMD than age-matched controls. Specifically, BMD Z-scores increased as the duration of training increased (r=0.32-0.48, p ranging between <0.04 and <0.002). During 12 months, the increase in areal BMD (g/cm²/year) of the total body, spine, and legs in the active prepubertal gymnasts was 30–85 % greater than in prepubertal controls (all p < 0.05). In the retired gymnasts, the areal BMD was 0.5–1.5 SD higher than the predicted mean in controls at all sites, except the skull (p ranging between <0.06 and <0.0001) [56]. This supports initiating resistance training in girls early in childhood, maximizing the benefits attributable to resistance training. One possible motivator for the athlete herself is that increased BMD, developed through regular strength-promoting exercise, provides protection against fractures, translating to increased sports participation and decreased time lost from injury [57].

Potential Benefits of Resistance Training for Obese or Overweight Girls and Adolescents

Overweight and obese children may enjoy resistance training because it is not aerobically taxing and provides an opportunity for participants to enhance fitness performance while gaining confidence in their ability to be physically active [58]. Regular participation in a resistance training program influences favorable changes in body composition in children and adolescents who are obese or at risk for obesity [15]. This may be critical, in particular, for young, developing females. Sung and colleagues evaluated the effects of a low energy diet with or without resistance training on blood lipid profiles in obese children. Fat free mass increased significantly in the training group. Serum total cholesterol decreased in both the training group and control groups, with the LDL:HDL ratio significantly decreased in the training group [59]. Van der Heijden examined the effects of a 12-week strength-training program on obese adolescents (six males and six females) and reported significant improvements in strength, lean body mass, and hepatic insulin sensitivity [60]. Others examined the effects of an 8-week resistance and power-training program on overweight and obese children (22 males and 26 females) and reported significant improvements in body composition and muscular fitness [61]. In a study evaluating the effects of resistance training on lean body mass and BMD, 82 school age obese/overweight children were randomly assigned to receive a balanced low-energy (900-1200 cal) diet plus resistance training or diet alone. After the short study period of just 6 weeks, the children in the training group showed significantly larger increases in lean body

mass and total bone mineral content via dual energy X-ray absorptiometry (DXA) measurements than those in the control group [62]. These findings, along with other reports [63–65], suggest that resistance training, or a program that combines resistance training and aerobic exercise, may be an attractive and beneficial alternative to aerobic activity alone in obese children and adolescents.

Conclusion

There are substantial benefits of resistance training for both young female athletes and non-athletes. These include:

- Improving muscular strength, power, and local muscular endurance.
- Preventing sports injuries.
- Improving BMD.
- Improving body composition.
- Improving metabolic health (i.e. insulin sensitivity).
- Improving balance and coordination.
- Promoting a positive attitude toward physical fitness.

It is important to recognize the benefits of initiating developmentally appropriate resistance training for girls during childhood, at a time when they may be more responsive to training protocols and before neuromuscular deficits become engrained. Finally, the early introduction of resistance training protocols by qualified fitness professionals who understand the unique physical and psychosocial characteristics of girls and adolescents may promote longstanding healthy behaviors and a lifelong interest in regular physical activity.

References

- 1. Haycock CE, Gillette JV. Susceptibility of women athletes to injury. Myths vs reality. JAMA. 1976;236(2):163–5. Epub 1976/07/12.
- 2. Physical Ed for Lifelong Fitness re. Human Kinetics; copyright 2011 Publisher Human Kinetics PO BOX 5076 Champagne II 61825 Pages 147–162.
- Faigenbaum AD, Kraemer WJ, Blimkie CJ, Jeffreys I, Micheli LJ, Nitka M, et al. Youth resistance training: updated position statement paper from the national strength and conditioning association. J Strength Cond Res. 2009;23(5 Suppl):S60–79. Epub 2009/07/22.
- Faigenbaum AD, Lloyd RS, Myer GD. Youth resistance training: past practices, new perspectives, and future directions. Pediatr Exerc Sci. 2013;25(4):591–604. Epub 2013/11/12.
- Lloyd RS, Faigenbaum AD, Stone MH, Oliver JL, Jeffreys I, Moody JA, et al. Position statement on youth resistance training: the 2014 International Consensus. Br J Sports Med. 2013. Epub 2013/09/24.
- 6. Micheli LJ. Preventing injuries in team sports: what the team physician needs to know. In: Chan K, Micheli L, Smith A, Rolf C, Bachl N, Frontera W, Alenabi T, editors. FIMS team physician manual. Hong Kong: CD Concepts; 2006.
- Myer GD, Sugimoto D, Thomas S, Hewett TE. The influence of age on the effectiveness of neuromuscular training to reduce anterior cruciate ligament injury in female athletes: a metaanalysis. Am J Sports Med. 2012;41:203–15. Epub 2012/10/11.

- 3 Resistance Training for Young Female Athletes
- Valovich McLeod TC, Decoster LC, Loud KJ, Micheli LJ, Parker JT, Sandrey MA, et al. National Athletic Trainers' Association position statement: prevention of pediatric overuse injuries. J Athl Train. 2011;46(2):206–20. Epub 2011/03/12.
- Faigenbaum AD, Chu DA, Paterno MV, Myer GD. Responding to exercise-deficit disorder in youth: integrating wellness care into pediatric physical therapy. Pediatr Phys Ther. 2013;25(1):2–6. Epub 2013/01/05.
- 10. Faigenbaum AD, Gipson-Jones TL, Myer GD. Exercise deficit disorder in youth: an emergent health concern for school nurses. J Sch Nurs. 2012;28:252–5. Epub 2012/03/20.
- Faigenbaum AD, Myer GD. Exercise deficit disorder in youth: play now or pay later. Curr Sports Med Rep. 2012;11(4):196–200. Epub 2012/07/11.
- 12. Faigenbaum AD, Myer GD. Resistance training among young athletes: safety, efficacy and injury prevention effects. Br J Sports Med. 2010;44(1):56–63. Epub 2009/12/01.
- Myer GD, Faigenbaum AD, Stracciolini A, Hewett TE, Micheli LJ, Best TM. Exercise deficit disorder in youth: a paradigm shift toward disease prevention and comprehensive care. Curr Sports Med Rep. 2013;12(4):248–55. Epub 2013/07/16.
- 14. Stracciolini A, Myer G, Faigenbaum AD. Exercise-deficit disorder in children: are we ready to make this diagnosis? Phys Sportsmed. 2013;41(1):94–101.
- Schranz N, Tomkinson G, Olds T. What is the effect of resistance training on the strength, body composition and psychosocial status of overweight and obese children and adolescents? A Systematic review and meta-analysis. Sports Med. 2013;43(9):893–907. Epub 2013/06/05.
- 16. Malina RM. Weight training in youth-growth, maturation, and safety: an evidence-based review. Clin J Sport Med. 2006;16(6):478–87. Epub 2006/11/23.
- Myer GD, Quatman CE, Khoury J, Wall EJ, Hewett TE. Youth versus adult "weightlifting" injuries presenting to United States emergency rooms: accidental versus nonaccidental injury mechanisms. J Strength Cond Res. 2009;23(7):2054–60. Epub 2009/10/27.
- Quatman CE, Myer GD, Khoury J, Wall EJ, Hewett TE. Sex differences in "weightlifting" injuries presenting to United States emergency rooms. J Strength Cond Res. 2009;23(7): 2061–7. Epub 2009/10/27.
- 19. Behringer M, Vom Heede A, Yue Z, Mester J. Effects of resistance training in children and adolescents: a meta-analysis. Pediatrics. 2010;126(5):e1199–210. Epub 2010/10/27.
- Granacher U, Goesele A, Roggo K, Wischer T, Fischer S, Zuerny C, et al. Effects and mechanisms of strength training in children. Int J Sports Med. 2011;32(5):357–64. Epub 2011/03/08.
- Ozmun JC, Mikesky AE, Surburg PR. Neuromuscular adaptations following prepubescent strength training. Med Sci Sports Exerc. 1994;26(4):510–4. Epub 1994/04/01.
- 22. Malina B, Bar-Or O. Growth maturation and physical activity. Champaign, IL: Human Kinetics; 2004.
- 23. Faigenbaum AD, McFarland JE, Schwerdtman JA, Ratamess NA, Kang J, Hoffman JR. Dynamic warm-up protocols, with and without a weighted vest, and fitness performance in high school female athletes. J Athl Train. 2006;41(4):357–63. Epub 2007/02/03.
- 24. Lloyd RS, Oliver J. The youth physical development model: a new approach to long-term athletic development. Strength Cond J. 2012;34(3):61–72.
- Faigenbaum ADMG, Farrell A, et al. Neuromuscular training and sex-specific fitness performance in 7-year-old children: an exploratory investigation. J Athl Train. 2014;49:145–53.
- Faigenbaum A. Youth strength training: programs for health, fitness and sport. Champaign, IL: Human Kinetics; 2009.
- Faigenbaum AD. Resistance training for overweight and obese youth: beyond sets and reps. Obes Weight Manag. 2009;5:282–5.
- Myer GD, Faigenbaum AD, Chu DA, Falkel J, Ford KR, Best TM, et al. Integrative training for children and adolescents: techniques and practices for reducing sports-related injuries and enhancing athletic performance. Phys Sportsmed. 2011;39(1):74–84. Epub 2011/03/08.
- 29. Chu DA. Plyometrics. Champaign, IL: Human Kinetics; 2013.
- Faigenbaum AD, Farrell A, Fabiano M, Radler T, Naclerio F, Ratamess NA, et al. Effects of integrative neuromuscular training on fitness performance in children. Pediatr Exerc Sci. 2011;23(4):573–84. Epub 2011/11/24.

- Hewett TE, Myer GD. The mechanistic connection between the trunk, hip, knee, and anterior cruciate ligament injury. Exerc Sport Sci Rev. 2011;39(4):161–6. Epub 2011/07/30.
- Myer GD, Chu DA, Brent JL, Hewett TE. Trunk and hip control neuromuscular training for the prevention of knee joint injury. Clin Sports Med. 2008;27(3):425–48. ix. Epub 2008/05/28.
- 33. Ford K, Myer G, Hewett TE. Increased trunk motion in female athletes compared to males during single leg landing. Med Sci Sports Exerc. 2007;39(5):S70.
- Hewett T, Snyder-Mackler L, Spindler KP. The drop-jump screening test: difference in lower limb control by gender and effect of neuromuscular training in female athletes. Am J Sports Med. 2007;35(1):145. author reply 6-7. Epub 2007/01/02.
- 35. Hewett TE, Myer GD, Ford KR, Slauterbeck JR. Preparticipation physical examination using a box drop vertical jump test in young athletes: the effects of puberty and sex. Clin J Sport Med. 2006;16(4):298–304. Epub 2006/07/22.
- Ford KR, Shapiro R, Myer GD, Van Den Bogert AJ, Hewett TE. Longitudinal sex differences during landing in knee abduction in young athletes. Med Sci Sports Exerc. 2010;42(10): 1923–31. Epub 2010/03/23.
- 37. Myer GD, Stroube BW, DiCesare CA, Brent JL, Ford KR, Heidt Jr RS, et al. Augmented feedback supports skill transfer and reduces high-risk injury landing mechanics: a doubleblind, randomized controlled laboratory study. Am J Sports Med. 2013;41(3):669–77. Epub 2013/02/02.
- Myer GD, Brent JL, Ford KR, Hewett TE. A pilot study to determine the effect of trunk and hip focused neuromuscular training on hip and knee isokinetic strength. Br J Sports Med. 2008;42(7):614–9. Epub 2008/03/01.
- Myer GD, Ford KR, Brent JL, Hewett TE. The effects of plyometric vs. dynamic stabilization and balance training on power, balance, and landing force in female athletes. J Strength Cond Res. 2006;20(2):345–53. Epub 2006/05/12.
- Myer GD, Ford KR, Palumbo JP, Hewett TE. Neuromuscular training improves performance and lower-extremity biomechanics in female athletes. J Strength Cond Res. 2005;19(1):51–60. Epub 2005/02/12.
- Quatman-Yates CC, Myer GD, Ford KR, Hewett TE. A longitudinal evaluation of maturational effects on lower extremity strength in female adolescent athletes. Pediatr Phys Ther. 2013;25(3):271–6. Epub 2013/04/27.
- 42. Hewett TE, Lindenfeld TN, Riccobene JV, Noyes FR. The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. Am J Sports Med. 1999;27(6):699–706. Epub 1999/11/24.
- Ford KR, Myer GD, Hewett TE. Longitudinal effects of maturation on lower extremity joint stiffness in adolescent athletes. Am J Sports Med. 2010;38(9):1829–37. Epub 2010/06/05.
- 44. Hewett TE, Myer GD, Ford KR. Decrease in neuromuscular control about the knee with maturation in female athletes. J Bone Joint Surg Am. 2004;86-A(8):1601–8.
- 45. Myklebust G, Engebretsen L, Braekken IH, Skjolberg A, Olsen OE, Bahr R. Prevention of anterior cruciate ligament injuries in female team handball players: a prospective intervention study over three seasons. Clin J Sport Med. 2003;13(2):71–8. Epub 2003/03/12.
- 46. Gilchrist J, Mandelbaum BR, Melancon H, Ryan GW, Silvers HJ, Griffin LY, et al. A randomized controlled trial to prevent noncontact anterior cruciate ligament injury in female collegiate soccer players. Am J Sports Med. 2008;36(8):1476–83. Epub 2008/07/29.
- 47. Myer GD, Ford KR, Brent JL, Hewett TE. Differential neuromuscular training effects on ACL injury risk factors in "high-risk" versus "low-risk" athletes. BMC Musculoskelet Disord. 2007;8(39):39.
- Myer GD, Ford KR, McLean SG, Hewett TE. The effects of plyometric versus dynamic stabilization and balance training on lower extremity biomechanics. Am J Sports Med. 2006;34(3):445–55. Epub 2005/11/12.
- 49. Stalder MA, Noble B, Wilkinson JG. The effects of supple- mental weight training for ballet dancers. J Appl Sport Sci Res. 1990;4(3):95–102.
- 50. Myer GD, Faigenbaum AD, Ford KR, Best TM, Bergeron MF, Hewett TE. When to initiate integrative neuromuscular training to reduce sports-related injuries and enhance health in youth? Curr Sports Med Rep. 2011;10(3):155–66. Epub 2011/05/31.

- 3 Resistance Training for Young Female Athletes
- Faigenbaum AD, Stracciolini A, Myer GD. Exercise deficit disorder in youth: a hidden truth. Acta Paediatr. 2011;100(11):1423–5. Epub 2011/09/08.
- Gunter KB, Almstedt HC, Janz KF. Physical activity in childhood may be the key to optimizing lifespan skeletal health. Exerc Sport Sci Rev. 2012;40(1):13–21. Epub 2011/09/16.
- 53. Nattiv A, Kennedy G, Barrack MT, Abdelkerim A, Goolsby MA, Arends JC, et al. Correlation of MRI grading of bone stress injuries with clinical risk factors and return to play: a 5-year prospective study in collegiate track and field athletes. Am J Sports Med. 2013;41(8):1930–41. Epub 2013/07/05.
- Cassell C, Benedict M, Specker B. Bone mineral density in elite 7- to 9-yr-old female gymnasts and swimmers. Med Sci Sports Exerc. 1996;28(10):1243–6. Epub 1996/10/01.
- Morris FL, Naughton GA, Gibbs JL, Carlson JS, Wark JD. Prospective ten-month exercise intervention in premenarcheal girls: positive effects on bone and lean mass. J Bone Miner Res. 1997;12(9):1453–62. Epub 1997/09/01.
- 56. Bass S, Pearce G, Bradney M, Hendrich E, Delmas PD, Harding A, et al. Exercise before puberty may confer residual benefits in bone density in adulthood: studies in active prepubertal and retired female gymnasts. J Bone Miner Res. 1998;13(3):500–7. Epub 1998/04/03.
- 57. Specker BL. The significance of high bone density in children. J Pediatr. 2001;139(4):473–5. Epub 2001/10/13.
- 58. Faigenbaum AD, Myer GD. Pediatric resistance training: benefits, concerns, and program design considerations. Curr Sports Med Rep. 2010;9(3):161–8. Epub 2010/05/14.
- Sung RYYC, Chang SK, Mo SW, Woo KS, Lam CW. Effects of dietary intervention and strength training on blood lipid level in obese children. Arch Dis Child. 2002;86(6):407–10.
- 60. van der Heijden GJ, Wang ZJ, Chu ZD, Sauer PJ, Haymond MW, Rodriguez LM, et al. A 12-week aerobic exercise program reduces hepatic fat accumulation and insulin resistance in obese, Hispanic adolescents. Obesity (Silver Spring). 2010;18(2):384–90. Epub 2009/08/22.
- 61. McGuigan MTM, Newton R, Pettigrew S. Eight weeks of resistance training can significantly alter body composition in children who are oberweight or obese. J Strength Cond Res. 2008;22(6):1–6.
- 62. Yu CC, Sung RY, So RC, Lui KC, Lau W, Lam PK, et al. Effects of strength training on body composition and bone mineral content in children who are obese. J Strength Cond Res. 2005;19(3):667–72. Epub 2005/08/13.
- 63. Schranz N, Tomkinson G, Parletta N, Petkov J, Olds T. Can resistance training change the strength, body composition and self-concept of overweight and obese adolescent males? A randomised controlled trial. Br J Sports Med. 2013. Epub 2013/08/16.
- 64. Shaibi GQ, Cruz ML, Ball GD, Weigensberg MJ, Salem GJ, Crespo NC, et al. Effects of resistance training on insulin sensitivity in overweight Latino adolescent males. Med Sci Sports Exerc. 2006;38(7):1208–15. Epub 2006/07/11.
- 65. Lee S, Bacha F, Hannon T, Kuk JL, Boesch C, Arslanian S. Effects of aerobic versus resistance exercise without caloric restriction on abdominal fat, intrahepatic lipid, and insulin sensitivity in obese adolescent boys: a randomized, controlled trial. Diabetes. 2012;61(11):2787–95. Epub 2012/07/04.

Chapter 4 Mental Skills Training: Games Girls Play— The Why, What, and When of Mental Fitness

Caroline Silby

Today, 3.2 million girls play high school sports, and women represent 41.7 % of National Collegiate Athletic Association (NCAA) athletes [1]. For many, the benefits of this increased participation have been life changing.

Girls who participate in sports have higher levels of self-esteem and decreased risk of depression [2]. Girls who participate in sports perform better in school and are more likely to graduate from high school. They are less likely to join gangs or use drugs. In general they begin having sex at a later age, and they are less likely to have unprotected sex and unintended pregnancy [3, 4]. Nationwide, girls who participate on one or two school sports teams are significantly less likely ever to have used marijuana, cocaine, or other illegal drugs [5]. Furthermore, compelling data from the 1980s and 1990s, showed that the growth of the female workforce in a particular state mirrored the sports opportunities afforded to girls in that state [6]. Athletic participation for girls has also been associated with a 7 % lower risk of obesity nearly 20–25 years later [7]. In addition, female athletes are more achievement oriented, more independent, more emotionally stable and more assertive than their non-athletic counterparts [8]. Even with these well-documented life-long benefits, significant material challenges remain in getting girls involved in sport and keeping them involved. By the age of 14 years old, girls drop out of sports at a rate two times greater than that of boys [9].

In an effort to continue to improve the quality of the sports experience for girls and to provide them with opportunities to compete at the highest levels, it is important to consider both the personality of the athlete, as well as the environment in which the athlete lives and trains. Investigations of training environments revealed that in ego-oriented training climates, mistakes are punished, the most talented athletes are more highly valued, collaborative learning is discouraged, and rivalries

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are encouraged. In that type of environment, female athletes were found to have more stress, less enjoyment of the sport, lower levels of self-esteem, and poorer body image than those who train in mastery climates. Mastery climates are sport environments in which mistakes are seen as part of the learning process, the contributions of each team member are valued, collaborative learning is encouraged, and importance is placed on effort [10, 11].

In a study of ten Olympic Champions (winners of 32 medals), coach and family influences were particularly important and affected athletes through teaching and modeling of certain psychological lessons [12]. Furthermore, athletes who play for trained coaches (i.e. coaches holding the appropriate certifications or licensure endorsed by the sport's national governing body) enjoy their sport experience more, evaluate their coach and teammates more positively, show significant increases in general self-esteem over the course of the sport season, and are roughly five times less likely than those playing for untrained coaches to drop out of the program the following season [13–15].

Psychological attributes such as self-confidence and the ability to cope with and interpret anxiety-related symptoms in a positive way are commonly accepted as being major contributors to success in sports [16, 17]. Interestingly, distinct differences have been found between highly successful performers (i.e. Olympic medalists) and performers who failed to achieve the level expected of them, with regards to focus, commitment, use of competition simulation, imagery, and post-competition recovery and planning [18]. In examining specific psychological characteristics of U.S. Olympic Champions, common characteristics include a high level of motivation and commitment, a positive or optimistic outlook, the ability to focus and concentrate, resiliency, and sport intelligence (creativity and ingenuity) [12].

Female athletes seeking to enhance performance typically define three attributes of mental fitness they want to strengthen:

- 1. Control—athletes want to feel more in-control of their performances and daily training.
- 2. Consistency—athletes want to perform more consistently at their highest levels, as well as consistently manage emotions on a daily basis.
- 3. Confidence—athletes want to believe in their abilities to succeed in their athletic pursuits.

The goal is to assist female athletes in strengthening these personal attributes as a way to ultimately develop into healthy, happy, successful adults, who also have sport outcomes that match their capabilities. These attributes and skills can be implemented into training, competition, and post-competition recovery.

The Why of Mental Fitness Training

In a survey conducted by this author, competitive athletes indicated that 50-99.9 % of their performance is influenced by attitude. However, in the same survey, they reported spending 0-5 % of their time working on their attitude. This suggests that

the athletes recognize the importance of attitude, but tend not to work on improving it. Most often athletes do not work on their attitude because they simply do not know how to go about it.

High-level athletes also know that attitude is important. Michael Jordan's famous quote illustrates the importance he placed on emotional skills like resiliency, openness to learning, and reframing:

"I've missed more than 9,000 shots in my career. I've lost almost 300 games. 26 times, I've been entrusted to take the game winning shot and missed. I've failed over and over again in my life. And that is why I succeed."

Michael Jordan

In a study of over 300 Olympians representing 13 countries, athletes viewed psychological factors as more important to their success than physical talent. The most frequently cited attributes were drive and ambition (29 %), determination (20 %), confidence (15 %), focus (11 %), natural talent (8 %), and the ability to "relax" (4 %) [19].

Researchers have evaluated psychological skills training programs to assess their effectiveness in relation to the performance enhancement process, and 38 of the 45 studies examined (85 %) had found positive performance effects [20]. A number of studies [21–23] have concluded that sport performance significantly improved as a consequence of psychological skills training. It is relevant to note that a performance enhancement of 3 % can make a significant difference at the elite level; it can mean the difference between not making the Olympic team and winning a gold medal [24].

The What of Mental Skills Training

Control

Regardless of the sport or level of participation, athletes and performers share a common set of worries. When asked, most athletes can easily identify their worries, which include stressors such as receiving unpleasant input from peers, fans, or coaches, experiencing pain or injury, making a physical or mental error, and receiving a "bad" call from officials [25, 26] (see Fig. 4.1). Yet, if an athlete is asked to identify one action taken that contributed to the creation of a positive outcome, she will likely respond, "I don't know." If an athlete cannot identify even one action that contributed to her success, how can she compete consistently well at the highest levels? More importantly, how can she feel in control of her performance? In essence, the athlete has to hope she performs well, as opposed to creating a systematic way of giving herself the best chance to create the desired level of play. It is especially important for female athletes to make connections between personal actions, behaviors, and successful competitive results, as female athletes tend to have a more external attribution style, which means that they will often attribute performance success to luck, and performance failure to personal flaws [27, 28].

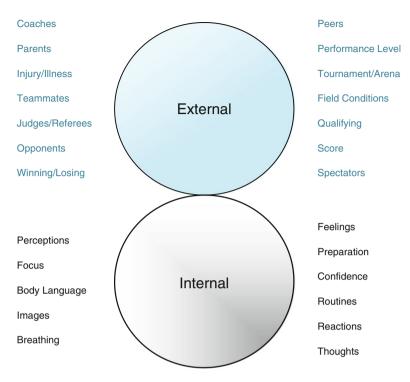


Fig. 4.1 Pressure loop: external and internal control factors in sport

Consistency

Elite athletes generally interpret their arousal and anxiety as more facilitative than non-elite athletes [29]. Eubank and Collins found that individuals who view their anxiety as facilitative are more likely to apply problem-focused and emotion-focused coping strategies [30]. While there are many anxiety coping strategies available to athletes, Tamres et al. found that female athletes used emotion-focused coping strategies, such as seeking support for emotional reasons, rumination, and positive self-talk, more frequently than males [31]. Most athletes who experience significant performance inconsistency show similar inconsistencies in daily training and fluctuations in the effort they put forth to manage "imperfect" emotions such as fear, worry, isolation, and stress. Consistency, both in relation to performance and managing one's emotions, can be created by identifying what is important, accepting imperfect feelings, and using factual assessments.

Identification of What Is Important

There is inherent "noise" associated with any competitive sport. In order for athletes to put filters on this noise and respond to the right cues, they can be invited to identify "TBUs" (true but useless pieces of information). There will be many distractions in sport; perhaps someone else is physically stronger, or maybe an athlete did not get the playing time she felt was deserved, or maybe she does not like her coach. These are all examples of TBUs. In spite of these facts, there are still positive actions the athlete can take to create success.

Acceptance of Imperfect Feelings

Feelings such as doubt, worry, and fear are a common part of the competitive sport experience, and acceptance of these "imperfect" feelings is the pre-condition for growth and change. We can be filled with doubt, AND we can be competent. We can feel stressed, AND we can solve our problems. Understanding that we are capable of holding two opposing emotions at the same time allows an athlete to use emotions as information and as cues to take positive action. For example, "I feel worried that my legs are tight, so I'm going to contract and release the muscles." Athletes should be discouraged from making JUDGMENTS about the feelings, such as, "I feel worried that my legs are tight, so I'm probably going to swim slowly."

The following is an example of acceptance by World Heavyweight Champion Buster Douglas:

"Doubt is always ringing my doorbell. I just open the door and say, 'How you doin', Doubt? Look everybody it's my old friend Doubt.' I've come to realize that doubt will always be there." - Buster Douglas on defeating Mike Tyson for the undisputed world heavyweight championship in February 1990.

Use of Factual Assessment

The stories athletes tell themselves about their training and performance shape their belief systems. Interestingly, what tends to be upsetting to female athletes is not usually the FACTS, for example, "I missed five series on beam," but rather the stories they tell themselves about those facts. For example, "I am falling behind in my training" or "I am not going to perform well at the meet." The way an athlete frames a situation impacts her ability to learn from her experiences, especially in situations where she fails to meet expectations. Reframing a situation is the ability to look at a problem through a different lens, or frame of reference, in order to make the emotional impact of an event less severe or to make finding a solution easier [32]. Female athletes want to know that they have earned others' belief in them. Therefore, when reframing a situation, it is crucial to stick to the facts.

An example of reframing through the use of factual appraisal comes from the author's early work with athletes. A junior level skater was preparing for the National Championships. During morning practice, despite multiple attempts, she was unable to execute her combination jump for the technical program. Because she was frustrated, the story she was telling herself was that she was not capable or prepared to compete. In response, she was asked to calculate the number of times she had successfully completed the combination, and the facts showed that she had been doing the jump at least five times per day, well over 8000 times in total. She was asked to identify another activity that she did as often, and she indicated that she brushed her teeth several times per day. When asked, "Have you ever brushed your teeth and had the toothbrush slip and jam into your gum line?" She responded, "Sure." She was then asked, "Well, do you wake up the next morning worried that you don't know how to brush your teeth?" She laughed. When asked "Why not?" she responded, "Well, I know I know how to brush my teeth." With that perspective, she was better able to reframe the situation and devise a plan to complete the jump successfully during competition.

The exercise of reframing provides an athlete with an opportunity to look at the facts, and tell an accurate story about herself and her competitive situation. From there, the athlete is able to proactively devise a game plan and identify positive actions to take that would give her the best chance to create the desired result.

Confidence

Sport confidence refers to the certainty an individual athlete possesses about her ability to be successful in sport [33]. Sport confidence is often measured and referred to as a trait, meaning it is a relatively stable characteristic. However, research suggests that it also contains state properties, indicating that it can fluctuate in intensity and over time [34]. Self-confidence is rooted in both belief and expectation. Across research it has been consistently found that athletes who possess higher levels of sport confidence tend to be more skilled and more efficient when using cognitive resources necessary for sporting success [35]. Confidence also plays a significant role in the athlete's coping processes—an athlete who possesses a strong belief in her ability is able to peak under pressure and cope successfully with adverse situations during competition [35].

Several researchers have investigated differences between males and females in sport confidence, and a relatively consistent finding is that male athletes demonstrate higher levels of confidence than females [36]. Hays et al. also found that confidence in female athletes is influenced by external factors such as social support and time spent with a personal coach, whereas male athletes derived confidence from a belief in the coach's ability to create an effective training plan [35]. Horn and Glenn found that anxious female athletes preferred coaches who gave strong support and positive feedback [37].

There is much research indicating marked declines in girls' self-confidence during the adolescent years [38]. Bond and Neideffer found that self-esteem scores progressively declined with age in female athletes, while the opposite was true in male athletes [39]. Similarly, Carol Gilligan has reported that girls lose confidence in their ability to express their needs and opinions as they move into the early adolescence [40]. Mary Pipher, in her acclaimed book, "Reviving Ophelia," outlined a variety of ways in which young female adolescents lose confidence in themselves leading to anxiety and depression [41].

In sports, early adolescence is often the "perfect storm" for female athletes. It is in this timeframe that there is a collision between increased expectations for performance and decreased confidence, leading to much performance inconsistency, frustration, and overall displeasure with the sport experience. It is therefore not surprising that at the age of 14 years, girls drop out of sports at a rate two times greater than that of boys [9].

There are numerous strategies used to enhance confidence, including mindfulness training, goal setting, commitment to a tactical plan, relaxation/imagery, and preand post-competition routines. Yet, most often athletes attribute differences in goal attainment and missed expectations to an overactive mind. Simply put, they say that they "think too much."

Self-Talk

Self-talk has been defined as "dialogue in which the individual interprets feelings and perceptions, regulates and changes evaluations and convictions, and gives herself instruction and reinforcement" [42]. While there is a body of experimental and field-based research supporting the use of positive self-talk [43–46], other research has found positive performance associations with negative self-talk [47] or no effect with positive or negative self-talk [48, 49]. The use of cue/key words to aid in learning and performance has shown more positive results. Cue words have been found to aid in the learning of different skills, such as ground strokes [50], volleying skills [51], and tennis and figure skating skills [52].

Zinsser et al. differentiate between self-talk that is motivational as opposed to instructional [53]. Some have proposed that the motivational function of self-talk assists athletes in building confidence and drive; it can increase effort and help control arousal and anxiety [16]. On the other hand, self-talk of an instructional nature is theorized to improve performance through attending to and executing actions based upon technique or tactics.

Informational self-talk can include both internal and external cues, which athletes can use to create a mental pacing of the performance/game. Mental pacing allows athletes predetermined opportunities to physically and mentally push or relax throughout the performance/game while also connecting mind and body into the present moment. It helps athletes achieve "flow," which is described by Susan Jackson as "a harmonious experience where mind and body are working together effortlessly" [54]. Providing mental pacing allows an athlete to be proactive in her execution of a predetermined tactical plan, thus minimizing questioning and avoiding response to the wrong cues, while increasing decisiveness, execution, and control.

The cues that create mental pacing of the performance/game can either be internal cues directing the athlete's attention to her body movements, or external cues directing attention to the action of the moment, but not specifically to body movement and skill execution. In a series of studies conducted by Wulf and colleagues [55, 56], the researchers found that participants who were taught to direct attention to an external cue, as opposed to their own body movement, performed better than those directed to focus on their body and feel their movement. Internal cues assist athletes in narrowing focus and serve as reminders to execute a certain technique, form, or feeling. External cues tend to be actions that simulate the mindbody connection that occurs when an athlete is naturally performing well, when her mind is quiet, and she is in a state of "flow."

The When of Mental Skills Training

One of the most frequently asked questions related to mental fitness training is "At what point is it appropriate to start mental skills training?" While not always exact, the following are commonly used references to frame an athlete's capacity to learn skills and principles:

- Pre-School (1–5 years)
- Pre-Adolescence (6–11 years)
- Early Adolescence (12–15 years)
- Maturing Adolescence (16+years)

The pre-school years are marked by allowing athletes to move their bodies in as many different ways as possible, providing an environment of fun and sharing in that fun. According to Carol Dweck, children's attitudes and behaviors regarding achievement and failure are already in place by pre-school [57]. Furthermore, providing descriptive feedback to 1- to 3-year olds by focusing on controllable aspects of performance, such as effort, predicted their later desire to take on new challenges, which in turn influenced these children's math achievement 7 years later [57, 58]. By providing process-oriented feedback and making use of emotional control strategies such as time-outs, it is possible to lay the groundwork for mental training in the pre-school years.

Pre-adolescence is a time period when athletes have the capacity to express their thoughts and feelings about sports participation, learn correct movement patterns, and respond to strategies for learning. While Marshall and Comalli found that most students are not formally taught much about their brain until at least middle school, mental strengths can be taught at this stage by providing opportunities for girls to openly discuss the actions that create success [59]. It is important to put emphasis on reasons for happiness, provide descriptive feedback on controllable aspects of performance, and help children identify personal actions that contribute to positive outcomes.

Early adolescence marks the time when female athletes are at high risk of experiencing performance inconsistency, burnout, injury, and dropout. This is the stage of athletic development where a collision occurs between increased expectation and decreased confidence, leading to performance inconsistency, sensitivity to criticism, feelings of frustration, resistance to learning, and a sense of staleness. If an athlete confronts all of these challenges without strategies to navigate them, it is likely she will quit her sport. Given that these performance challenges are predictable, this is the time to continue to build upon the foundation of skills laid earlier by proactively arming girls with a more advanced skill set to manage these challenges before they occur.

Maturing adolescence is marked by uncertainty about the future, search for one's place in the world, and separation from immediate family. At this stage, athletes often struggle to balance their commitment to sports with their responsibilities as students, friends, and members of their families and communities. Recognition of only one dimension of personality leaves athletes vulnerable to high anxiety and depression [60]. The physical changes that accompany this time period can wreak havoc on the performance as well as the confidence of female athletes. It is also during this time period that overtraining, injury, and underperformance commonly emerge. While males are likely to show increases in their strength and speed, females typically experience some difficulty emotionally and physically adjusting to their new body size and composition. The physical and psychological symptoms of overtraining and underperformance are related, making cause and effect difficult to distinguish. It is critically important that athletes are both physically and psychologically prepared to navigate both. If mental fitness strategies have previously been introduced and implemented, the athlete can continue to hone those skills and transfer them to the new situations and self-perceptions that emerge during this stage of development.

Summary

Athletes seem to know that in order to create sport outcomes that match their capabilities, some sort of mental fitness training must be practiced. Typically, athletes are seeking to feel more in control of their performance levels, perform consistently to their abilities, and feel confident about their ability to deliver high-level performances in critical moments. Through mental training, athletes can develop the coping skills of focusing on the controllable aspects of performance, finding perspective through factual appraisals, responding to appropriate cues, and increasing confidence through the use of self-talk and cue words. Ultimately, the foundation for mental training can be laid in early childhood when we begin to teach emotional control. As children mature, more advanced coping strategies can be introduced and honed to meet the specific cognitive, emotional, and performance challenges presented by each stage in a young woman's development.

Sports participation, like many other endeavors throughout life, is an adventure; it is about figuring out what brings an athlete happiness, what gets her engaged, and what challenges her. Developing mental fitness skills will not guarantee a young woman success, but it can provide her with useful tools and the best chance of reaching her goals while enjoying the process.

References

- 2011–2012 High School Athletics Participation Survey, National Federation of State High School Associations, 1981-1982-2011-2012 Student Athlete Participation, NCAA Sports Sponsorship and Participation Rates Report.
- Colton M, Gore S. Risk, resiliency, and resistance: current research on adolescent girls. Bethesda MD: Ms. Foundation for Women; 1991.
- 3. Miller K, Sabo DF, Melnick MJ, Farrell MP, Barnes GM. The Women's Sports Foundation Report: Benefits: why sports participation for girls and women. Women's Sports Foundation, The Women's Sports Foundation Report: health risks and the Teen Athlete. East Meadow, NY: Women's Sports Foundation; 2000.
- Gibbons T, Hill R, McConnell A, Forster T, Moore J. The path to excellence: a comprehensive view of development of U.S. Olympians who competed from 1984-1998. Colorado Springs, CO: United States Olympic Committee; 2002.
- 5. Sabo D, Miller K, Farrell M, Barnes G, Melnick M. The Women's Sports Foundation Report: sport and teen pregnancy. East Meadow, NY: Women's Sports Foundation; 1998.
- 6. Stevenson B. Beyond the classroom: using title IX to measure the return to high school sports. Rev Econ Stat. 2010;92(2):284–301. 08.
- 7. Kaestner R, Xu X. Title IX, girls' sports participation, and adult female physical activity and weight. Eval Rev. 2010;34:52–78.
- 8. Williams J. Personality traits of champion level fencers. Res Q. 1970;41(3):446-53.
- 9. Sabo D, Veliz P. Girls drop-out at a different rates depending on where they live. Go out and play: Youth sports in American. East Meadow, NY: Women's Sports Foundation; 2008.
- Duda JL, Ntoumanis N. After-school sport for children: implications of a task-involving motivational climate. In: Mahoney JL, Eccles J, Larson R, editors. After school activities: contexts of development. Mahwah, NJ: Erlbaum; 2005. p. 311–30.
- Walling MD, Duda JL, Chi L. The perceived motivational climate in sport questionnaire: construct and predictive validity. J Sport Exer Psychol. 1993;15:172–83.
- 12. Gould D, Dieffenbach K, Moffett A. Psychological characteristics and their development in Olympic champions. J Appl Sport Psychol. 2002;14:172–204.
- Barnett NP, Smoll FL, Smith RE. Effects of enhancing coach-athlete relationships on youth sport attrition. Sport Psychol. 1992;6:111–27.
- 14. Smith RE, Smoll FL, Curtis B. Coach effectiveness training: a cognitivebehavioral approach to enhancing relationship skills in youth sport coaches. J Sport Psychol. 1979;1:59–75.
- Smoll FL, Smith RE, Barnett NP, Everett JJ. Enhancement of children's self-esteem through social support training for youth sport coaches. J Appl Psychol. 1993;78:602–10.
- 16. Hardy L, Jones G, Gould D. Understanding psychological preparation for sport: theory and practice of elite performers. New York: Wiley; 1996.
- 17. Mellalieu SD, Hanton S, Jones G. Emotional labeling and competitive anxiety in preparation and competition. Sport Psychol. 2003;17:157–74.
- 18. Orlick T, Partington J. Mental links to excellence. Sport Psychol. 1988;2:105-30.
- 19. Wann DL. The head and shoulders psychology of success project: an examination of perceptions of Olympic athletes. N Am J Psychol. 2012;14(1):123–38.
- Weinberg RS, Comar W. The effectiveness of psychological intervention in competitive sports. Sports Med. 1994;18:406–18.
- Daw J, Burton D. Evaluation of a comprehensive psychological skills training program for collegiate tennis players. Sport Psychol. 1994;8:37–57.
- Kendall G, Hrycaiko D, Martin GL, Kendall T. The effects of an imagery rehearsal, relaxation, and self-talk package on basketball game performance. J Sport Exer Psychol. 1990;12(2):157–66.
- 23. Lerner BS, Ostrow AC, Yura MT, Etzel EF. The effects of goal-setting and imagery training programs on the free-throw performance of female collegiate basketball players. Sport Psychol. 1996;10:382–97.

- 24. Birrer D, Morgan G. Psychological skills training as a way to enhance an athlete's performance in high-intensity sports. Scand J Med Sci Sports. 2010;20 Suppl 2:78–87.
- 25. Anshel MH. Coping styles among adolescent competitive athletes. J Soc Psychol. 1996;136:311–24.
- Anshel MH, Kaissidis AN. Coping style and situational appraisals as predictors of coping strategies following stressful events in sport as a function of gender and skill level. Br J Psychol. 1997;88:263–76.
- Bird AM, Williams JM. A developmental-attributional analysis of sex-role stereotypes for sport performance. Dev Psychol. 1980;16:312–22.
- 28. Lenney E. Women's self-confidence in achievement settings. Psychol Bull. 1977;84(1):1-13.
- 29. Hanton S, Jones G. The acquisition and development of cognitive skills and strategies: I. Making the butterflies fly in formation. Sport Psychol. 1999;13(1):1–21.
- Eubank MR, Collins DJ. Coping with pre-and in-event fluctuations in competitive state anxiety: a longitudinal approach. J Sports Sci. 2000;18:121–31.
- 31. Tamres LK, Janicki D, Helgeson VS. Sex differences in coping behavior: a meta-analytic review and an examination of relative coping. Pers Soc Psychol Rev. 2002;6:2–30.
- 32. Carr A. Family therapy: concepts, process and practice. Chichester: Wiley; 2000.
- Munroe-Chandler K, Hall C, Fishburne G. Playing with confidence: the relationship between imagery use and self-confidence and self-efficacy in youth soccer players. J Sports Sci. 2008;23:1–8.
- Thomas O, Lane A, Kingston K. Defining and contextualizing robust sport confidence. J Appl Sport Psychol. 2011;23:189–208.
- 35. Hays KF. Performance anxiety. In: Hays KF, editor. Performance psychology in action: a casebook for working with athletes, performing artists, business leaders, and professionals in high risk occupations. Washington, DC: American Psychological Association; 2009. p. 101–20.
- Hays K, Maynard I, Thomas O, Bawden M. Sources and types of confidence identified by world class sport performers. J Appl Sport Psychol. 2007;19:434–56.
- 37. Horn T, Glenn S. The relationship between athletes' psychological characteristics and their preference for particular coaching behaviors. Paper presented at the meeting of the North American Society for the Psychology of Sport and Physical Activity, Knoxville, TN; 1988.
- 38. American Association of University Women. Shortchanging girls, shortchanging America: full data report. Washington, DC: American Association of University Women; 1990.
- 39. Bond JW, Nideffer RM. Attentional and interpersonal characteristics of elite Australian athletes. Excel Aust Sport Comm. 1992;8:101–11.
- 40. Gilligan C. Making connections: The relational world of adolescent girls at the Emma Willard School. Cambridge, MA: Harvard University Press; 1990.
- 41. Pipher M. Reviving Ophelia: saving the selves of adolescent girls. New York: Ballantine Books; 1994.
- Hackfort D, Schwenkmezger P. Anxiety. In: Singer RN, Murphey M, Tennant LK, editors. Handbook of research on sport psychology. New York: Macmillan Publishing Company; 1993. p. 328–64.
- Dagrou E, Gauvin L, Halliwell W. Effects of positive, negative and neutral self-talk on motor performance. Can J Sport Sci. 1992;17:145–7.
- 44. Van Raalte JL, Brewer BW, Lewis BP, Linder DE. Cork! The effects of positive and negative self-talk on dart throwing performance. J Sport Behav. 1995;18:50–7.
- 45. Van Raalte JL, Brewer BW, Rivera PM, Petitpas AJ. The relationship between observable selftalk and competitive junior tennis players' match performances. J Sport Exerc Psychol. 1994;16:400–15.
- Mahoney MJ, Avener M. Psychology of the elite athlete: an exploratory study. Cognit Ther Res. 1977;1(2):135–41.
- Highlen PS, Bennett BB. Elite divers and wrestlers: a comparison between open-and closedskill athletes. J Sport Psychol. 1983;5(4):390–409.
- 48. Dagrou E, Gauvin L, Halliwell W. The mental preparation of athletes in the Côte d'Ivoire: current practices and research perspectives. Int J Sport Psychol. 1991;22(1):15–34.

- 49. Cohn PJ, Rotella RJ, Lloyd JW. Effects of a cognitive-behavioral intervention on the preshot routine and performance in golf. Sport Psychol. 1990;4(1):33–47.
- 50. Ziegler SG. Effects of stimulus cueing on the acquisition of groundstrokes by beginning tennis players. J Appl Behav Anal. 1987;20(4):405–11.
- 51. Smith PJK, Johnston D. A comparison of augmented verbal cues and self-talk regimes in learning a tennis volley. J Sport Exer Psychol. 2000;22:S101.
- 52. Ming S, Martin GL. Single-subject evaluation of a self-talk package for improving figure skating performance. Sport Psychol. 1996;10(3):227–38.
- 53. Williams JM, Zinsser N, Bunker L. Cognitive techniques for building confidence and enhancing performance. Mountain View, CA: Mayfield; 2001.
- 54. Jackson SA, Csikszentmihalyi M. Flow in sports. Champaign, IL: Human Kinetics; 1999.
- 55. Totsika V, Wulf G. The influence of external and internal foci of attention on transfer to novel situations and skills. Res Q Exerc Sport. 2003;74(2):220–32.
- 56. Wulf G, Shea C, Park JH. Attention and motor performance: preferences for and advantages of an external focus. Res Q Exerc Sport. 2001;72(4):335–44.
- 57. Dweck Carol S. The perils and promises of praise. Educ Leadersh. 2007;65:34-9.
- 58. Gunderson E, et al. Parent praise to 1-to 3-year-olds predicts children's motivational frameworks 5 years later. Child Dev. 2013;84(5):1526–41.
- 59. Marshall PJ, Comalli CE. Young children's changing conceptualizations of brain function: implications for teaching neuroscience in early elementary settings. Early Educ Dev. 2012;23(1):4–23.
- Brewer BW, Van Raalte JL, Linder DE. Athletic identity: Hercules' muscles or Achilles heel? Int J Sport Psychol. 1993;24:237–54.

Chapter 5 The Female Athlete Triad

Maria L. Eguiguren and Kathryn E. Ackerman

Introduction

The Female Athlete Triad (Triad) is a relatively new entity that has been described and studied primarily in the past two decades. Due to ongoing research and efforts to increase awareness of this condition, athletes, coaches, physicians, and other providers are now more familiar with the Triad and its health consequences. However, the complete pathophysiology of the Triad is not fully understood, and debate continues about how clinicians and the sports community should provide care for athletes with this disorder. This chapter provides an overview of the definitions, epidemiology, clinical presentation, current management, and future directions of the Triad.

Definitions

The Female Athlete Triad: A syndrome that involves the interrelationship of decreased energy availability, menstrual dysfunction, and poor bone health.

Energy availability: The amount of dietary energy remaining, after exercise training, for all other physiological functions each day.

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Components	Definitions
Low energy availability	Low energy availability = (energy intake – exercise energy expenditure)/fat free mass <45 kcal/kg per day or inadequate kcal/kg to sustain normal bodily functions
	Disordered eating: various abnormal eating behaviors, including restrictive eating, fasting, frequently skipped meals, diet pills, laxatives, diuretics, enemas, overeating, binge-eating and then purging (vomiting)
	Eating disorder: a clinical mental disorder defined by DSM-5 and characterized by abnormal eating behaviors, an irrational fear of gaining weight, and false beliefs about eating, weight, and shape
Menstrual disorders	<i>Primary amenorrhea</i> : Lack of menstruation by age 15 in the presence of normal secondary sexual characteristics
	Secondary amenorrhea: absence of menstrual cycles for \geq 3 months after menarche
	Oligomenorrhea: menstrual cycles at intervals >35 days
	Anovulation: absence of ovulation due to follicular impairment
	Luteal phase defect: luteal phase <11 days and/or low progesterone level
Low bone mineral density	Low BMD: Z-score between -1 and -2 with secondary clinical risk factors for fractures ^a
	Osteoporosis: Z-score ≤ -2 with a significant fracture history ^b

Table 5.1 Definitions of the components of the female athlete triad in young females [6, 7]

^aClinical risk factors for fracture include: chronic malnutrition, eating disorders, hypogonadism, glucocorticoid exposure, previous fractures, and hyperparathyroidism

^bSignificant fracture history: (1) two or more long bone fractures by age 10 years, and/or (2) three or more long bone fractures at any age up to age 19 years

Amenorrhea: Absence of menstruation by the age of 15 in those with normal secondary sexual characteristics (primary amenorrhea) or absence of menses for ≥ 3 months after menarche (secondary amenorrhea).

Oligomenorrhea: Menstrual cycles at intervals >35 days.

Z-score: A measure of bone mineral density, which indicates the standard deviation of a measurement obtained in a particular patient compared to a sex- and age-matched control group.

Osteoporosis: A condition of decreased bone mineral density resulting in an increased risk of fracture. In children and adolescents it is defined as a Z-score ≤ -2 with a significant fracture history.

In 1992, the American College of Sports Medicine (ACSM) first described a triad of disordered eating, amenorrhea, and osteoporosis in female athletes [1–5] (see Table 5.1). The clinical consequences, need for treatment, and further questions about the Triad were then presented in the ACSM's 1997 Position Stand on the Female Athlete Triad [8]. Subsequent research suggested that many patients do not necessarily have all the components of the Triad simultaneously, and that previous conceptual models of the condition failed to identify many women at risk of developing the disease [8–12].

Therefore, in 2007 the ACSM published an updated position stand, defining the Triad as an interrelationship of energy availability, menstrual function, and bone

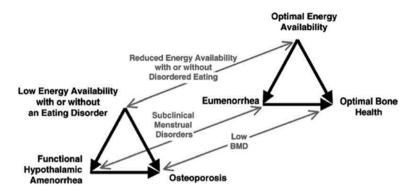


Fig. 5.1 The female athlete triad spectra along which female athletes are distributed: energy availability, menstrual function, and bone mineral density. An athlete's condition moves along each spectrum at a different rate based on her diet and exercise habits [6]. From De Souza MJ, Nattiv A, Joy E, et al. Br J Sports Med 2014;48:289

health, noting that an athlete may be at different points along spectra from optimal health to significant disease (Fig. 5.1) [6]. At the ideal end, an athlete has optimal energy availability, eumenorrhea, and optimal bone health. Farther along the spectra, an athlete may have decreased energy availability with or without disordered eating, subclinical menstrual disorders, including oligomenorrhea, luteal deficiency, and anovulation, and/or low bone mineral density (BMD). At the pathologic end are the combination of low energy availability with or without an eating disorder, functional hypothalamic amenorrhea (absence of menses caused by suppression of the hypothalamic–pituitary–ovarian axis without a known anatomic or organic disease cause), and osteoporosis [6].

Low Energy Availability

Energy availability is the energy required to maintain bodily functions, and it is defined as the dietary energy intake minus the energy used during exercise (exercise energy expenditure) normalized to fat free mass [6]. The current recommendation for daily energy availability is above 45 kcal/kg of fat free mass; below this level alterations in bone can be seen. Metabolic and reproductive changes are observed below 30 kcal/kg of fat free mass in adult women [6, 13, 14]. The appropriate energy availability range for growing adolescents has not been rigorously tested; therefore, the authors recommend that 45 kcal/kg of fat free mass/day be used as a guideline for adolescent female athletes, while also closely monitoring growth and development.

Some female athletes may inadvertently underconsume calories, while others may have a disordered eating pattern or a true eating disorder as the cause of their low energy availability. The revised criteria of anorexia nervosa in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) include restriction of energy intake relative to requirements leading to significantly low body weight, intense fear of gaining weight or of becoming fat, and disturbance in the way one's body weight or shape is experienced [15, 16]. Amenorrhea is no longer a criterion for the diagnosis of anorexia nervosa, and the qualification of one's weight being <85 % ideal body weight was also removed [16]. Bulimia nervosa is defined as recurrent episodes of binge eating followed by recurrent inappropriate compensatory behaviors (e.g., vomiting, laxative abuse, diuretic abuse, and overexercising) at least once a week for 3 months in order to prevent weight gain, and self-evaluation that is excessively influenced by body shape and weight [16]. Eating disorder not otherwise specified (EDNOS) was a catch-all category used in the past for those who did not fit neatly into a specific category;

instead, the DSM-5 includes subcategories, such as binge-eating disorder (binging without inappropriate compensatory behaviors), atypical anorexia nervosa (if the patient is not underweight), and purging disorder [16].

Menstrual Dysfunction

The etiology of amenorrhea is quite extensive; however, amenorrhea related to energy deficiency in athletes is considered functional hypothalamic amenorrhea (FHA). FHA is the suppression of the hypothalamic–pituitary–ovarian axis in the absence of an identifiable anatomic or organic cause, and can be related to decreased energy availability, exercise, and/or stress [17]. There are various menstrual irregularities along the Triad menstrual spectrum such as amenorrhea, oligomenorrhea, and subclinical menstrual disorders (luteal phase defects and anovulation). Table 5.1 shows the definitions of these menstrual cycle abnormalities.

Bone Impairment

There are different definitions referring to low BMD and osteoporosis based on age. The World Health Organization (WHO) uses dual energy X-ray absorptiometry (DXA)-derived T-scores to define osteopenia and osteoporosis in postmenopausal women and men \geq 50 years of age. This method compares the patient's BMD to normative data of sex-matched individuals in their 20s (the time of peak bone mass) [18]. In recent studies in children, adolescents, and premenopausal women, Z-scores are preferred over T-scores because a Z-score indicates the standard deviation of a measurement obtained in a particular patient compared to a sex- and age-matched control group [19]. According to the International Society for Clinical Densitometry (ISCD), a Z-score \leq -2.0 SD is considered "below expected range for age," and a Z-score >-2.0 SD is "within the expected range for age." In children and adolescents, diagnosis of osteoporosis is used only when the BMD Z-score is \leq -2.0 and there is a clinically significant fracture history. For premenopausal women, "osteoporosis" is defined as a BMD Z-score \leq -2.0 plus risk factors for fracture or

secondary causes of osteoporosis [20, 21]. However, due to the expected higher BMD in athletes participating in weight bearing sports, the ACSM defines the term "low BMD" in premenopausal women as a Z-score between -1 and -2 in the presence of secondary clinical risk factors for fracture. ACSM reserves the term "osteoporosis" for those with a Z-score ≤ -2.0 along with secondary clinical risk factors for fractures (Table 5.1) [6].

Epidemiology

The prevalence of the Triad varies widely among athletes. In general, the estimated prevalence of all three components of the Triad simultaneously is low. However, the individual components are common in recreational and elite-level female athletes of all ages [10, 22, 23]. In a systematic review of the literature, Gibbs et al. found that the prevalence of all three components of the Triad in female athletes ranged from 0 % to 15.9 %. The prevalence of any two components ranged from 2.7 % to 27 %, and 16 % to 60 % for any single component [24]. In a study by Hoch et al., up to 78 % of high school varsity female athletes were found to have one or more component of the Triad [25].

Although energy restriction is common in athletes, it is difficult to assess its prevalence because of a lack of simple and accurate methods to measure energy intake and expenditure. One study, which was based on self-report questionnaires and 3-day food diaries, showed that the prevalence of low energy availability (defined as <45 kcal/kg fat free mass) in high school female athletes was 36 % [25]. In their review, Gibbs et al. reported that the prevalence of eating disorders among female athletes in general ranged from 0 % to 48 %, and the prevalence of clinical disordered eating ranged from 7.1 % to 89.2 % in 17 studies [24].

The prevalence of menstrual irregularities in athletes was examined in 34 studies using self-reported methods and/or hormonal assessments [24]. The prevalence of primary amenorrhea ranged from 0 % to 56 %, secondary amenorrhea from 1 % to 60 %, and of oligomenorrhea from 0.9 % to 52.5 % [24]. The prevalence of luteal phase defects ranged from 5.9 % to 43 %, and of anovulation from 12 % to 30 % [24]. The wide range in each category likely reflects the menstrual dysfunction definitions used, methodologies of assessment, subjects' ages, exercise volume, sport practiced, etc.

Individual estimates of low BMD, defined as a Z-score ≤ -2.0 , ranged from 0 % to 15.4 % in various studies, while the prevalence of low BMD, defined as a Z-score between -1.0 and -2.0, ranged from 0 % to 39.8 % [24]. BMD can have significant variability depending on sport. In a study of 93 high school female runners, ages 13–18 years, 11.8 % and 28 % met the -2 and -1 BMD Z-score cutoffs, respectively. Total hip and lumbar spine BMD were significantly lower in runners with menstrual irregularity, after adjusting for body mass index (BMI) and lean tissue mass [26].

Even though the Triad can be observed in any exercising woman, the risk is higher in those practicing leanness, aesthetic, or endurance sports [6, 27, 28] (Table 5.2). For instance, 70.1 % of athletes competing in leanness sports are at risk

Endurance sports	Cross-country skiing, cycling, rowing, running, speed skating, swimming	
Aesthetic sports	Cheerleading, dance, figure skating, gymnastics, synchronized swimming	
Weight-class sports	Boxing, judo, kickboxing, lightweight rowing, mixed martial arts, taekwondo, weightlifting, wrestling	
Anti-gravitational sports	Cycling, swimming, synchronized swimming	

Table 5.2 Examples of leanness sports: sports in which leanness and/or a specific body weight are considered important for performance

of developing the Triad compared to 55.3 % of athletes in other sports [29]. Moreover, the prevalence of all three components of the Triad is higher in athletes participating in sports emphasizing leanness (1.5 % to 6.7 %) versus those in non-lean sports (0-2 %) [24].

Pathophysiology

Low Energy Availability

Low energy availability results from failing to meet nutritional requirements for a given amount of physical activity. For instance, studies among female soccer players found that the prevalence of low energy availability was highest during mid-season [30] and was associated with a low carbohydrate diet and lower energy dense meals [31, 32]. These findings suggest that restrictions in dietary intake (with or without disordered eating or eating disorders), in addition to increases in exercise activity, can lead to low energy availability.

Elite athletes are at increased risk for eating disorders, especially those athletes in sports and activities emphasizing leanness, including dance [33]. Athletes generally start to diet to meet the body paradigm specific to their sport, to improve performance, and to conform to sociocultural "ideals" for thinness [34, 35]. Although many athletes have eating disorders, others are not consciously restricting food but are simply unaware of the dietary intake needed. In fact, athletes often lack the appetite necessary to promote food intake to compensate for energy expenditure from intense exercise [36, 37]. Appetite is regulated by several hormones including ghrelin, which stimulates food intake, and hormones such as peptide YY (PYY), pancreatic polypeptide (PP), and glucagon-like peptide 1 (GLP-1), which suppress food intake [38]. Some studies have shown that acute bouts of exercise suppress acylated ghrelin and increase PYY, PP, and GLP-1 [39, 40]. This phenomenon is called "exercise-induced anorexia" [39]. However, these studies have only analyzed the hormonal response immediately after exercise training.

Chronic low energy stores in athletes are insufficient to maintain physiological processes. In an effort to conserve energy, the body experiences certain physiological adaptations, such as reproductive function inhibition, resulting in menstrual dysfunction [14, 41].

Menstrual Irregularities

Menstrual function in those with the Triad can range from eumenorrhea to amenorrhea. In female athletes with the Triad, amenorrhea is caused by a disruption in the hypothalamic–pituitary–ovarian (HPO) axis [42]. It has been shown that luteinizing hormone (LH) pulse frequency decreases below an energy availability threshold of 30 kcal/kg of lean body mass [14]. As a consequence, gonadal steroid release from the ovaries is disrupted, resulting in a hypoestrogenic state. In some cases, chronic energy deficiency can reduce ovarian steroid secretion without disrupting menstrual cycles. This may lead to subclinical menstrual disorders such as luteal deficiency and anovulation [43, 44].

Low energy availability also affects other metabolic and gastrointestinal hormones that may be involved in the regulation of the HPO axis. The adipocytokine leptin, as well as insulin, ghrelin, and PYY, all have the ability to cross the blood–brain barrier and are not only involved in appetite regulation, but also have been hypothesized to regulate GnRH pulsatility [45, 46]. Ghrelin and PYY are elevated, while leptin and insulin are suppressed in female athletes with amenorrhea [41, 47–51].

Bone Impairment

Bone mass accrual occurs principally during puberty, and by 26 years of age young women reach 99 % of their total bone mineral content [52]. Weight-bearing exercise typically has a beneficial effect on bone accretion when proper nutrition is present [53]. However, in athletes with the Triad, bone health is compromised due to low energy availability and the subsequent decrease in gonadal steroids and other hormones [37, 54]. In fact, bone accrual in adolescent runners was found to be suppressed when compared to non-runners, putting this group at special risk for fractures and future complications [55].

It is well established that estrogen inhibits osteoclast activity, and low levels of estrogen increase bone resorption. However, studies have also shown that energy deficiency itself causes detrimental effects on bone health, independent of estrogen [54, 56]. The mechanisms involve changes in metabolic hormones, such as leptin, insulin-like growth factor 1 (IGF-1), PYY, cortisol (increased in low energy states including Triad), and nutritional deficiencies critical to bone formation, such as calcium and vitamin D [54, 56–62]. One study by Ihle et al. concluded that there is an uncoupling of bone resorption and formation within 5 days of low energy availability, and that extreme energy restriction causes a more pronounced effect on bone remodeling, suggesting a dose–response relationship [63].

Although DXA is the gold standard for measuring BMD, it does not evaluate other parameters of bone such as bone geometry and microarchitecture [37]. Recent research has focused on the assessment of bone quality, mineral content, and strength by using new imaging techniques such as high-resolution peripheral quantitative computed tomography (HR-pQCT). It has been shown that bone microarchitecture

is impaired in adolescent amenorrheic weight-bearing athletes compared to eumenorrheic athletes and healthy controls [64]. Athletes presented greater total area, trabecular area, and cortical perimeter compared to non-athletes; however, amenorrheic athletes had lower cortical area and thickness, lower trabecular number, and higher trabecular separation at the weight-bearing tibia [64]. Also, amenorrheic athletes have lower estimated bone strength (by finite element analysis) at the non-weightbearing distal radius than non-athletes, and lose the strength advantage of weightbearing exercise seen in eumenorrheic athletes at the distal tibia [65].

Symptoms/Diagnosis

Patients with the Triad may present in a variety of ways, and coaches, teammates, families, and physicians should be alert for early warning signs. Signs and symptoms may include weight loss, ritualistic eating, isolating behavior, excessive concern with body image, mood changes, fatigue, frequent illness and injury, decreased exercise performance, and prolonged recovery time after injury. Individuals suffering from Triad may notice weakness, bowel changes, lightheadedness, menstrual irregularity, or difficulty concentrating; many other changes can also be seen, as decreased energy availability can lead to a variety of musculoskeletal, cardiovascular, gastrointestinal, renal, and neuropsychiatric symptoms [66].

A pre-participation examination may pick up on signs of the Triad if the right questions are asked. The Female Athlete Triad Coalition recommends the screening questions listed in Table 5.3 [67]. Additionally, physicians should also have the Triad on their radars when assessing female athletes in clinic with related problems, including amenorrhea or recurrent injury, particularly stress fractures. An athlete who presents with one component of the Triad should be evaluated for the others [6].

Low energy availability may be suggested by a low BMI ($<17.5 \text{ kg/m}^2$) or in adolescents, <85 % of expected body weight. In general, absolute BMI cutoffs are not recommended in adolescents; the BMI percentile method for calculating estimated body weight is preferred. This compares the adolescent's weight to that at

- 1. Have you ever had a menstrual period?
- 2. How old were you when you had your first menstrual period?
- 3. When was your most recent menstrual period?
- 4. How many periods have you had in the past 12 months?
- 5. Are you presently taking any female hormones (estrogen, progesterone, birth control pills)?
- 6. Do you worry about your weight?
- 7. Are you trying to or has anyone recommended that you gain or lose weight?
- 8. Are you on a special diet or do you avoid certain types of foods or food groups?
- 9. Have you ever had an eating disorder?
- 10. Have you ever had a stress fracture?
- 11. Have you ever been told you have low bone density (osteopenia or osteoporosis?)

Table 5.3
 Female Athlete Triad Coalition pre-participation examination screening questions [67]

the 50th BMI percentile, the expected body weight [67, 68]. Using growth charts to assess changes in weight and BMI is helpful. Some patients with inadequate energy availability may not have significantly low body weight, but may have fallen off their growth curve, have low triiodothyronine, have a low heart rate, hypotension, hypothermia, orthostatic changes, or other signs of poor nutrition [67, 69]. A sports dietitian and/or exercise physiologist can be instrumental in estimating dietary intake and energy expenditure.

Menstrual dysfunction can be assessed initially with screening questions followed by a more thorough workup. In addition to the questions included in Table 5.3, it is important to assess familial menstrual history in order to determine hereditary patterns. When evaluating primary or secondary amenorrhea, etiologies such as thyroid disorders (hyper- or hypothyroidism), pituitary tumors (e.g., prolactinoma), and polycystic ovarian syndrome should be considered. A thorough medication review should be performed, including questions regarding antipsychotic agents, combined oral contraceptive pills, depot medroxyprogesterone acetate injections, and possible intrauterine devices, all of which may disrupt menses [17]. If the treating physician is not comfortable evaluating FHA, consultation with an endocrinologist is recommended. The medical workup for amenorrhea is shown in Fig. 5.2 [67].

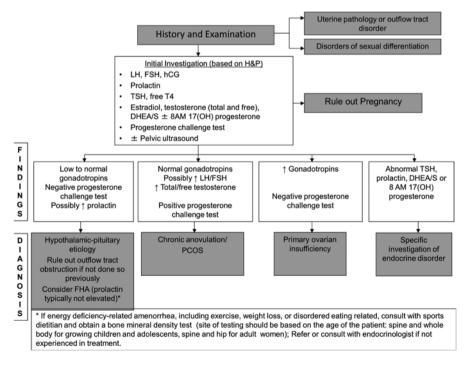


Fig. 5.2 Amenorrhea Algorithm from 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad [67]. *DHEA/S* dehydroepiandrosterone sulfate, *FHA* functional hypothalamic amenorrhea, *FSH* follicle-stimulating hormone, *hCG* human chorionic gonadotropin, *LH* luteinizing hormone, *PCOS* polycystic ovarian syndrome, *TSH* thyroid-stimulating hormone. From De Souza MJ, Nattiv A, Joy E, et al. Br J Sports Med 2014;48:289

Bone health can be assessed by obtaining the patient's history of fractures, family history of osteoporosis and other possible metabolic bone diseases, and obtaining a DXA scan as needed. The authors recommend obtaining a DXA when there is a history of an eating disorder; a BMI ≤ 18.5 kg/m², < 85 % estimated weight, or recent weight loss of ≥ 10 %; menarche ≥ 16 years of age; history of ≥ 6 months of amenorrhea; two or more stress reactions/fractures; one high risk stress reaction/fracture (e.g., femoral neck stress injury) or low energy non-traumatic fracture; or at least 1 year after a prior low BMD DXA result [67]. Depending on results, further laboratory testing may be warranted (e.g., 25-hydroxy vitamin D, calcium, phosphorus, magnesium, parathyroid hormone, thyroid-stimulating hormone, urinary calcium and creatinine, etc.)

Treatment

A multidisciplinary approach is optimal to treat and prevent further complications of the Triad. The treatment team often involves a primary care and/or sports physician, a dietitian, and a mental health professional. Broadening the team to provide more support can be quite helpful and may include family members, coaches, athletic trainers, and/or exercise physiologists.

The primary goal of treatment is to increase energy availability and body weight to achieve normal gonadal and other hormonal levels. This is typically done with both diet and exercise modification. Modest exercise reduction (10–20 %) and an increase in caloric intake (20–30 % over baseline energy needs) offer a good beginning strategy [6, 67]. If energy availability can be accurately estimated, the target should be \geq 45 kcal/kg of fat free mass per day [67]. If the athlete's diet is significantly restricted, caloric intake should be increased slowly to avoid raising the patient's fear of weight gain and to avoid the negative sequelae of "refeeding syndrome," a condition that involves potentially fatal shifts in fluids and electrolytes that can occur when caloric intake increases too quickly in a malnourished patient.

Weight gain to achieve a BMI >18.5 kg/m² or >90 % of ideal body weight is recommended to restore menstrual function and increase BMD [6, 67, 70]. Some athletes may need to achieve even higher weight goals to restore normal menstrual function, because of their greater amount of lean muscle mass and relatively lower amount of adipose tissue. In addition, getting BMD to a normal range may not be possible depending on the timing, severity, and duration of energy restriction, but stopping bone loss and achieving some BMD improvement is common [71].

Because decreased energy availability associated with the Triad often involves maladaptive eating and training behaviors, many athletes are reluctant to adhere to suggested activity and dietary modifications. Therefore, psychological counseling may be necessary and may help uncover the drive behind the disorder. If a true eating disorder is suspected, more intensive therapy with an eating disorder treatment team may be needed. Also, because progress is sometimes slow, pharmacological therapy may be considered as an adjunct to lifestyle changes.

Oral contraceptive pills (OCPs) containing estrogen and progestin are commonly prescribed to athletes with amenorrhea, but evidence regarding the effects on BMD

is inconclusive. Different studies have demonstrated an improvement, no change, or a decrease in BMD [67, 72, 73]. Transdermal estrogen may have a better impact on bone than OCPs secondary to differing effects on IGF-1, a bone trophic hormone. Oral estrogen decreases systemic IGF-1, while transdermal formulations maintain or increase concentrations of this growth factor important for bone formation and remodeling [74]. Studies involving postmenopausal women found that transdermal estrogen (+/– progesterone) is more effective than OCPs in increasing BMD and decreasing fracture risk [75, 76]. Transdermal estrogen and oral progesterone therapy also improved spine and hip BMD in adolescents with anorexia nervosa [77]. Cyclic progesterone is suggested for those on transdermal estrogen to avoid the negative effects of unopposed estrogen on the uterine lining. However, further research on the efficacy of transdermal estrogen and oral progesterone on BMD in Triad patients is needed.

Small studies have tested subcutaneous leptin analog injections for recovering menstrual cycles and improving BMD in those with FHA. While some patients did resume menses and had significant increases in BMD, leptin's side effect of weight loss is of great concern. Therefore, larger studies with detailed dosing adjustments are warranted [78, 79].

Additionally, adequate calcium and vitamin D are important for bone health. Calcium ingestion via food products is ideal, but often not achieved in restricted diets, and serum 25-hydroxy vitamin D levels may be lower than the recommended 30-32 ng/mL cutoff. Therefore 1300 mg/day of elemental calcium in divided doses in adolescents (1000 mg in premenopausal women \geq 19 years old), and at least 600 IU of vitamin D daily are recommended [74, 80]. Higher vitamin D dosing may be necessary to achieve adequate blood levels.

In general, bisphosphonates are not recommended for BMD treatment in premenopausal populations, except in extreme circumstances and under the guidance of a bone metabolism specialist, such as an endocrinologist [67]. Antidepressant medications such as selective serotonin reuptake inhibitors (SSRIs) may be useful in certain Triad patients. Studies have found SSRIs to be effective in the treatment of bulimia nervosa, significantly reducing the frequency of binge eating and purging. The efficacy is less clear in anorexia nervosa [81, 82]. There is a potential advantage to using antidepressants in Triad patients in order to treat comorbid conditions such as anxiety, depression, and obsessive compulsive disorder [67]. However, SSRIs have been linked to weight loss in some individuals along with negative effects on BMD, so further research is warranted [67, 83, 84].

Future Directions

In 2014 the Female Athlete Triad Coalition expanded on the prior ACSM position statements by publishing updated research findings and suggesting evaluation and return to play guidelines for Triad [67]. In addition, the International Olympic Committee (IOC) published a consensus statement on Relative Energy Deficiency in Sport (RED-S), highlighting the effects of energy deficit on not only the

reproductive and musculoskeletal systems, but others as well. Their paper also called for more research in other sport populations including male athletes, minorities, and athletes with disabilities [66]. The Female Athlete Triad Coalition and the IOC publications included their own Triad assessment and return to play models, both of which are currently being tested in adolescent sport populations.

As knowledge about the Triad increases, more questions have emerged that need to be answered: Should athletes with disordered eating or eating disorders have a different approach to weight recovery than non-athletes? Are there more accurate, faster, clinical tools for assessing energy availability? What other cardiovascular, immunologic, neurologic, and psychological effects occur from energy deficiency? Are there other adjunctive hormonal or other medical therapies that may be helpful in treating the Triad? Are there different energy availability set points for adolescents? These questions and others are currently being explored.

Summary

The female athlete triad and its individual components can occur in female athletes at any age and in any sport. Early awareness and education during adolescence can help prevent a variety of difficulties, ranging from unsatisfying sports performance to lifelong health problems. Particular attention should be paid to athletes participating in high risk sports, which include leanness activities. Recent research has further illucidated the complex hormonal pathways contributing to the Triad and the multiple negative consequences of decreased energy availability. As guidelines from the ACSM, the Female Athlete Triad Coalition, the IOC, and other professional medical organizations are refined, information about detection and treatment must be more widely disseminated to improve the care of these young athletes.

References

- Yeager KK, et al. The female athlete triad: disordered eating, amenorrhea, osteoporosis. Med Sci Sports Exerc. 1993;25(7):775–7.
- 2. Carbon RJ. Exercise, amenorrhoea and the skeleton. Br Med Bull. 1992;48(3):546-60.
- 3. Myburgh KH, et al. Low bone density is an etiologic factor for stress fractures in athletes. Ann Intern Med. 1990;113(10):754–9.
- 4. Highet R. Athletic amenorrhoea. An update on aetiology, complications and management. Sports Med. 1989;7(2):82–108.
- 5. Otis CL. Exercise-associated amenorrhea. Clin Sports Med. 1992;11(2):351-62.
- Nattiv A, et al. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 2007;39(10):1867–82.
- 7. Klein DA, Poth MA. Amenorrhea: an approach to diagnosis and management. Am Fam Physician. 2013;87(11):781–8.
- Otis CL, et al. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 1997;29(5):i–ix.

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- 9. Nattiv A, et al. The female athlete triad. The inter-relatedness of disordered eating, amenorrhea, and osteoporosis. Clin Sports Med. 1994;13(2):405–18.
- 10. Nichols JF, et al. Prevalence of the female athlete triad syndrome among high school athletes. Arch Pediatr Adolesc Med. 2006;160(2):137–42.
- 11. Quah YV, et al. The female athlete triad among elite Malaysian athletes: prevalence and associated factors. Asia Pac J Clin Nutr. 2009;18(2):200–8.
- 12. Burrows M, et al. The components of the female athlete triad do not identify all physically active females at risk. J Sports Sci. 2007;25(12):1289–97.
- Loucks AB. Energy availability, not body fatness, regulates reproductive function in women. Exerc Sport Sci Rev. 2003;31(3):144–8.
- Loucks AB, Thuma JR. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. J Clin Endocrinol Metab. 2003;88(1):297–311.
- 15. Attia E, et al. Feeding and eating disorders in DSM-5. Am J Psychiatry. 2013;170(11): 1237–9.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Washington, DC: APA Press; 2013.
- Gordon CM. Clinical practice. Functional hypothalamic amenorrhea. N Engl J Med. 2010;363(4):365–71.
- Czerwinski E, et al. Current understanding of osteoporosis according to the position of the World Health Organization (WHO) and International Osteoporosis Foundation. Ortop Traumatol Rehabil. 2007;9(4):337–56.
- Gordon CM, et al. Dual energy X-ray absorptiometry interpretation and reporting in children and adolescents: the 2007 ISCD Pediatric Official Positions. J Clin Densitom. 2008;11(1):43–58.
- Crabtree NJ, et al. Dual-energy X-ray absorptiometry interpretation and reporting in children and adolescents: The revised 2013 ISCD pediatric official positions. J Clin Densitom. 2014;17(2):225–42.
- Gordon CM, Leonard MB, Zemel BS. 2013 pediatric position development conference: executive summary and reflections. J Clin Densitom. 2014;17(2):219–24.
- Javed A, et al. Female athlete triad and its components: toward improved screening and management. Mayo Clin Proc. 2013;88(9):996–1009.
- Beals KA, Hill AK. The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. Int J Sport Nutr Exerc Metab. 2006;16(1):1–23.
- Gibbs JC, Williams NI, De Souza MJ. Prevalence of individual and combined components of the female athlete triad. Med Sci Sports Exerc. 2013;45(5):985–96.
- 25. Hoch AZ, et al. Prevalence of the female athlete triad in high school athletes and sedentary students. Clin J Sport Med. 2009;19(5):421–8.
- Barrack MT, Rauh MJ, Nichols JF. Prevalence of and traits associated with low BMD among female adolescent runners. Med Sci Sports Exerc. 2008;40(12):2015–21.
- Beals KA, Manore MM. Disorders of the female athlete triad among collegiate athletes. Int J Sport Nutr Exerc Metab. 2002;12(3):281–93.
- 28. Movaseghi S, et al. Clinical manifestations of the female athlete triad among some Iranian athletes. Med Sci Sports Exerc. 2012;44(5):958–65.
- Torstveit MK, Sundgot-Borgen J. The female athlete triad: are elite athletes at increased risk? Med Sci Sports Exerc. 2005;37(2):184–93.
- Reed JL, De Souza MJ, Williams NI. Changes in energy availability across the season in Division I female soccer players. J Sports Sci. 2013;31(3):314–24.
- Reed JL, et al. Exercising women with menstrual disturbances consume low energy dense foods and beverages. Appl Physiol Nutr Metab. 2011;36(3):382–94.
- Reed JL, et al. Nutritional practices associated with low energy availability in Division I female soccer players. J Sports Sci. 2014;32(16):1499–509.
- Smolak L, Murnen SK, Ruble AE. Female athletes and eating problems: a meta-analysis. Int J Eat Disord. 2000;27(4):371–80.
- 34. Sundgot-Borgen J. Risk and trigger factors for the development of eating disorders in female elite athletes. Med Sci Sports Exerc. 1994;26(4):414–9.

- 35. Sundgot-Borgen J, Torstveit MK. Aspects of disordered eating continuum in elite highintensity sports. Scand J Med Sci Sports. 2010;20 Suppl 2:112–21.
- Loucks AB, Kiens B, Wright HH. Energy availability in athletes. J Sports Sci. 2011;29 Suppl 1:S7–15.
- 37. Mallinson RJ, De Souza MJ. Current perspectives on the etiology and manifestation of the "silent" component of the Female Athlete Triad. Int J Womens Health. 2014;6:451–67.
- 38. Stensel D. Exercise, appetite and appetite-regulating hormones: implications for food intake and weight control. Ann Nutr Metab. 2010;57 Suppl 2:36–42.
- Martins C, et al. Effects of exercise on gut peptides, energy intake and appetite. J Endocrinol. 2007;193(2):251–8.
- 40. Schubert MM, et al. Acute exercise and hormones related to appetite regulation: a metaanalysis. Sports Med. 2014;44(3):387–403.
- 41. Scheid JL, De Souza MJ. Menstrual irregularities and energy deficiency in physically active women: the role of ghrelin, PYY and adipocytokines. Med Sport Sci. 2010;55:82–102.
- De Souza MJ, Metzger DA. Reproductive dysfunction in amenorrheic athletes and anorexic patients: a review. Med Sci Sports Exerc. 1991;23(9):995–1007.
- Williams NI, et al. Estrogen and progesterone exposure is reduced in response to energy deficiency in women aged 25–40 years. Hum Reprod. 2010;25(9):2328–39.
- Bullen BA, et al. Induction of menstrual disorders by strenuous exercise in untrained women. N Engl J Med. 1985;312(21):1349–53.
- Wade GN, Jones JE. Neuroendocrinology of nutritional infertility. Am J Physiol Regul Integr Comp Physiol. 2004;287(6):R1277–96.
- 46. Hill BR, et al. 24-hour profiles of circulating ghrelin and peptide YY are inversely associated in normal weight premenopausal women. Peptides. 2012;38(1):159–62.
- Laughlin GA, Yen SS. Nutritional and endocrine-metabolic aberrations in amenorrheic athletes. J Clin Endocrinol Metab. 1996;81(12):4301–9.
- 48. Ackerman KE, et al. Higher ghrelin and lower leptin secretion are associated with lower LH secretion in young amenorrheic athletes compared with eumenorrheic athletes and controls. Am J Physiol Endocrinol Metab. 2012;302(7):E800–6.
- De Souza MJ, et al. Fasting ghrelin levels in physically active women: relationship with menstrual disturbances and metabolic hormones. J Clin Endocrinol Metab. 2004;89(7):3536–42.
- Scheid JL, et al. Elevated PYY is associated with energy deficiency and indices of subclinical disordered eating in exercising women with hypothalamic amenorrhea. Appetite. 2009;52(1):184–92.
- 51. Russell M, et al. Peptide YY in adolescent athletes with amenorrhea, eumenorrheic athletes and non-athletic controls. Bone. 2009;45(1):104–9.
- 52. Harel Z, et al. Bone mineral density in postmenarchal adolescent girls in the United States: associated biopsychosocial variables and bone turnover markers. J Adolesc Health. 2007;40(1):44–53.
- 53. Barkai HS, et al. Influence of sports participation and menarche on bone mineral density of female high school athletes. J Sci Med Sport. 2007;10(3):170–9.
- 54. De Souza MJ, et al. The presence of both an energy deficiency and estrogen deficiency exacerbate alterations of bone metabolism in exercising women. Bone. 2008;43(1):140–8.
- 55. Barrack MT, Rauh MJ, Nichols JF. Cross-sectional evidence of suppressed bone mineral accrual among female adolescent runners. J Bone Miner Res. 2010;25(8):1850–7.
- De Souza MJ, Williams NI. Beyond hypoestrogenism in amenorrheic athletes: energy deficiency as a contributing factor for bone loss. Curr Sports Med Rep. 2005;4(1):38–44.
- 57. Ackerman KE, Misra M. Bone health and the female athlete triad in adolescent athletes. Phys Sportsmed. 2011;39(1):131–41.
- 58. Scheid JL, et al. Estrogen and peptide YY are associated with bone mineral density in premenopausal exercising women. Bone. 2011;49(2):194–201.
- 59. Ackerman KE, et al. Cortisol secretory parameters in young exercisers in relation to LH secretion and bone parameters. Clin Endocrinol (Oxf). 2013;78(1):114–9.
- 60. De Souza MJ, et al. Clinical tests explain blunted cortisol responsiveness but not mild hypercortisolism in amenorrheic runners. J Appl Physiol. 1994;76(3):1302–9.

5 The Female Athlete Triad

- De Souza MJ, et al. Adrenal activation and the prolactin response to exercise in eumenorrheic and amenorrheic runners. J Appl Physiol. 1991;70(6):2378–87.
- De Souza MJ, et al. Luteal phase deficiency in recreational runners: evidence for a hypometabolic state. J Clin Endocrinol Metab. 2003;88(1):337–46.
- 63. Ihle R, Loucks AB. Dose-response relationships between energy availability and bone turnover in young exercising women. J Bone Miner Res. 2004;19(8):1231–40.
- 64. Ackerman KE, et al. Bone microarchitecture is impaired in adolescent amenorrheic athletes compared with eumenorrheic athletes and nonathletic controls. J Clin Endocrinol Metab. 2011;96(10):3123–33.
- 65. Ackerman KE, et al. Cortical microstructure and estimated bone strength in young amenorrheic athletes, eumenorrheic athletes and non-athletes. Bone. 2012;51(4):680–7.
- 66. Mountjoy M, et al. The IOC consensus statement: beyond the Female Athlete Triad—Relative Energy Deficiency in Sport (RED-S). Br J Sports Med. 2014;48(7):491–7.
- 67. De Souza MJ, et al. 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad: 1st international conference held in San Francisco, California, May 2012 and 2nd international conference held in Indianapolis, Indiana, May 2013. Br J Sports Med. 2014;48(4):289.
- Le Grange D, et al. Calculation of expected body weight in adolescents with eating disorders. Pediatrics. 2012;129(2):e438–46.
- 69. Golden NH, et al. Eating disorders in adolescents: position paper of the Society for Adolescent Medicine. J Adolesc Health. 2003;33(6):496–503.
- Arends JC, et al. Restoration of menses with nonpharmacologic therapy in college athletes with menstrual disturbances: a 5-year retrospective study. Int J Sport Nutr Exerc Metab. 2012;22(2):98–108.
- Fredericson M, Kent K. Normalization of bone density in a previously amenorrheic runner with osteoporosis. Med Sci Sports Exerc. 2005;37(9):1481–6.
- 72. Miller BE, et al. Sublingual administration of micronized estradiol and progesterone, with and without micronized testosterone: effect on biochemical markers of bone metabolism and bone mineral density. Menopause. 2000;7(5):318–26.
- Vescovi JD, Jamal SA, De Souza MJ. Strategies to reverse bone loss in women with functional hypothalamic amenorrhea: a systematic review of the literature. Osteoporos Int. 2008;19(4):465–78.
- 74. Nazem TG, Ackerman KE. The female athlete triad. Sports Health. 2012;4(4):302-11.
- 75. Ettinger B, et al. Effects of ultralow-dose transdermal estradiol on bone mineral density: a randomized clinical trial. Obstet Gynecol. 2004;104(3):443–51.
- Warming L, Ravn P, Christiansen C. Levonorgestrel and 17beta-estradiol given transdermally for the prevention of postmenopausal osteoporosis. Maturitas. 2005;50(2):78–85.
- 77. Misra M, et al. Physiologic estrogen replacement increases bone density in adolescent girls with anorexia nervosa. J Bone Miner Res. 2011;26(10):2430–8.
- Chou SH, et al. Leptin is an effective treatment for hypothalamic amenorrhea. Proc Natl Acad Sci U S A. 2011;108(16):6585–90.
- Welt CK, et al. Recombinant human leptin in women with hypothalamic amenorrhea. N Engl J Med. 2004;351(10):987–97.
- Institute of Medicine, 30 Nov 2010. Institute of medicine of the national academies. Dietary reference intakes tables and application. http://www.iom.edu/Activities/Nutrition/ SummaryDRIs/DRI-Tables.aspx. Retrieved 27 Mar 2014.
- Thiel A. Are psychotropic drugs necessary for the treatment of anorexia and bulimia nervosa? Psychother Psychosom Med Psychol. 1997;47(9–10):332–45.
- Zhu AJ, Walsh BT. Pharmacologic treatment of eating disorders. Can J Psychiatry. 2002;47(3): 227–34.
- Couturier J, et al. Bone mineral density in adolescents with eating disorders exposed to selective serotonin reuptake inhibitors. Eat Disord. 2013;21(3):238–48.
- Tsapakis EM, et al. The adverse skeletal effects of selective serotonin reuptake inhibitors. Eur Psychiatry. 2012;27(3):156–69.

Chapter 6 Overuse Injuries in Young Female Athletes

Genevra Stone, Cassidy M. Foley, and Ellen Geminiani

Introduction

Along with the many benefits of sports participation, unfortunately injuries also occur. Most injuries can be classified as either acute/traumatic injuries or overuse injuries. This chapter focuses on some of the most common overuse injuries experienced by young female athletes.

Overuse injuries result from repetitive stress or load to the musculoskeletal tissues without adequate rest to allow for tissue recovery. It is estimated that approximately 50 % of injuries in young athletes are overuse, and many may be preventable [1]. Lack of uniformity in injury definition, classification, and reporting methods leads to underestimation of the total impact of overuse injuries [2].

A recent study found that athletic injuries differ between adolescent females and males by type, diagnosis, and body area. Physically active females ages 5–17 years have a higher rate of overuse injuries and are more likely to sustain injuries to the lower extremity and spine compared to their male counterparts [3]. Additionally, studies have demonstrated differences in joint angles, muscular strength, and proprioceptive ability between male and female athletes [3].

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Definitions

Tendinopathy: A nonspecific term that refers to tendon injury; it encompasses conditions such as tendinitis and tendinosis. Histologically, tendinitis is marked by inflammation of the tendon and typically develops over a short period of time. Tendinosis represents a more chronic degenerative process within the tendon [3].

Physis: Also known as the growth plate, this is the area where the bone lengthens.

Apophysis: A secondary ossification center and the bony attachment site of a musculotendinous unit. Rapid bone growth that exceeds the growth rate of the muscle and tendon places particular traction stress at the apophysis [4].

Apophysitis: Inflammation and microtrauma at the apophysis that develop due to repetitive traction forces at this site.

Epiphysis: The articular end of a long bone, which during growth is separated from the bone shaft by an area of cartilage.

Epiphysitis: Inflammation of the epiphysis due to compression or shearing in this region of growing bone.

Eccentric exercises: Exercises that strengthen while the muscles lengthen.

Risk Factors

Risk factors for overuse injuries are often categorized as intrinsic factors, related to the athlete herself, or extrinsic factors, related to the conditions of sport participation [5]. Intrinsic risk factors can include anatomic malalignment, joint hypermobility, the adolescent growth spurt, hormonal irregularities, and biomechanical alterations [6]. Extrinsic risk factors include rules, equipment, play surface, and environment, including weather [7]. Training errors related to frequency, volume, intensity, and technique are other extrinsic factors frequently responsible for overuse injuries [5]. In addition, over-scheduling injuries have been described due to excessive planned physical activity without adequate rest and recovery time [8]. Pressure from coaches and parents represents an additional extrinsic factor described in the literature [9]. Each athlete's individual risk factors should be considered to help prevent injury.

History of prior injury is an important risk factor for overuse injury, which should always be addressed when evaluating the young athlete. Additionally, overuse injuries may be more common during periods of rapid growth, when the athlete is adapting to tremendous changes and imbalances in body structures, strength and flexibility [6].

Overuse Injuries

Patellofemoral Knee Pain

Patellofemoral knee pain is one of the most common complaints among female athletes with a lifetime incidence of 19.6 % compared to 7.4 % in males [10, 11]. It is estimated to account for up to 25 % of knee injuries in athletes [12]. Patellofemoral knee pain is a complex of symptoms affecting the anterior knee that results from the position and movement of the patella and retinaculum, which is not attributable to other intra-articular or peripatellar pathology. It is, therefore, a diagnosis of exclusion.

The patella is a sesamoid bone within the quadriceps tendon. It is an important part of the extensor mechanism as it glides in the trochlea of the distal femur and assists with leverage in knee extension. Articular cartilage of the patella and the trochlea aid with the gliding motion, and soft tissue structures balance and stabilize the patella within the femoral groove. Forces superiorly (quadriceps tendon), inferiorly (patellar tendon), medially (medial retinaculum, vastus medialis) and laterally (lateral retinaculum, vastus lateralis, iliotibial band) act together to maintain position of the patella in the trochlear groove [13].

The pathophysiology of patellofemoral pain is not clearly understood and is most likely multifactorial. Forces acting on the patella can create a lateral deviation and misalignment with a tight lateral retinaculum, iliotibial band, and/or vastus lateralis that overpowers the medial forces from the vastus medialis [14]. Other studies have demonstrated a delay in firing of the vastus medialis as a factor in lateral tracking [15]. Poor flexibility in the gastrocnemius and hamstring has been identified in patellofemoral pain patients [16] along with tightness and weakness in the quadriceps [17], all of which can increase the compressive forces on the patella. Other causative factors described include excess pronation [18], weakness of the hip stabilizers [19], and increased physical activity [20]. It has also been observed that adolescent girls with increased knee abduction (valgus stress) during landing may be at increased risk for both patellofemoral pain and ACL injury [21].

Athletes typically report insidious onset of pain over the anterior knee. Pain is usually worse with increased activity, especially running, squatting, prolonged sitting ("theater sign") or going upstairs or downstairs. Some patients experience a "giving out" sensation, which likely represents poor quadriceps control due to weakness, imbalance, or misfiring of the muscles; this sensation should be distinguished from true mechanical symptoms. Physical examination should initially evaluate for structural causes of knee pain including tendinitis, ligament instability, and internal derangement. Important elements of the physical exam include palpation of the patellar borders, mobility of the patella, and strength of the surrounding muscles. A detailed evaluation of the patient's lower extremity flexibility, strength, and biomechanics is necessary to determine the contributing factors in the pathophysiology of patellofemoral pain [22].

The majority of patients respond well to conservative treatment, which may includes physical therapy, activity modification, and orthotic support. Physical therapy should focus on improving flexibility and strength. Counseling the patient regarding temporary activity modification and cross training may help decrease the load on the patellofemoral joint. In some patients orthotics may provide some pain relief, especially if there is excess pronation [13]. Many patients may benefit from bracing or taping techniques, but the data on the efficacy of these approaches are inconclusive [23]. Rarely, conservative measures are not adequate, and surgical interventions, such as lateral release or tibial tubercle osteotomy, are considered for patients with persistent symptoms.

Stress Fractures

Stress fractures are a common overuse injury with a higher lifetime incidence in female athletes (9.7 % in females vs 6.5 % in males) [24]. Like other overuse injuries, stress fractures result from repetitive stress without adequate recovery. Normal bone undergoes a continuous process of repair and remodeling when under strain and tension. Excessive bone strain causes an accumulation of microdamage and possible crack initiation. Fracture can develop when normal bone healing is overwhelmed by increased demands or when there is reduced bone healing that cannot respond to normal strain on the bone [25].

Bones of the lower extremity are most commonly affected, with sites of injury varying based on the specific sport. Athletes will typically describe an insidious onset of pain that worsens during or after activity. Symptoms often follow an increase in training volume or intensity. Physical examination usually reveals bony tenderness, with or without associated soft tissue swelling. Symptoms often worsen with provocative measures, such as hopping on one foot or placing a vibrating tuning fork against the affected bone [26]. Clinical suspicion from patient history and physical exam may prompt further diagnostic evaluation with plain X-rays. However, X-rays are often negative, especially in the early stages of stress injuries. While callus formation can sometimes be seen on plain films, visible changes generally take several weeks to develop. Therefore, in the setting of high clinical suspicion, additional imaging with an MRI or bone scan can confirm the diagnosis.

In general, treatment of stress fractures consists of rest, proper rehabilitation, and gradual return to activity. The location of the injury is important because of the risk of delayed healing or nonunion. Stress fractures that typically heal well can be treated with rest from sports activity and immobilization as needed for pain control [27]. Once the athlete is pain free, close follow up is important to determine the timing and rate of safe progression back to activities [28]. In general, when there is no pain at rest, the athlete may begin light activity such as pool exercises or elliptical training with a slow progression to full impact physical activity and sports participation.

Some stress fractures have an increased risk of delayed healing or nonunion and possible long-term sequelae. These high risk stress injuries include fractures of the

pars interarticularis of the spine, tension side of the femoral neck, patella, anterior tibia, medial malleolus, talus, tarsal navicular, the metaphyseal–diaphyseal junction of the fifth metatarsal, and the sesamoids [6].

Stress fracture of the pars intrarticularis, known as spondylolysis, is a common cause of back pain in adolescent athletes. During growth, spinal alignment changes with an increase in lumbar lordosis, which results in greater stress on the posterior elements and pars intrarticularis and an increased risk of stress fracture [28]. Sports requiring repetitive extension of the spine, such as gymnastics, dance, and figure skating, carry an elevated risk, but spondylolysis can be seen in many different sports. A high index of suspicion must be maintained in all adolescent athletes with back pain. Additional details about this condition can be found in Chapter 7.

Other high risk stress injuries occur on the tension side of the femur and the anterior tibia. In athletes with anterior hip or groin pain, a femoral stress injury should be suspected and an MRI may be warranted based on the history and physical examination, even in the presence of negative radiographs [6]. See Chapter 8 for additional details. The "dreaded black line" anterior tibial stress fracture (see Fig. 6.1) is another stress fracture recognized for its tendency towards delayed healing or nonunion. Surgical intervention is necessary on rare occasions, but conservative treatment is often successful if adequate time for rest is allowed [29].



Fig. 6.1 Black line stress fractures

Prevention of stress fractures comes from identifying and modifying risk factors. Field et al. found that older age at menarche, a maternal family history of osteoporosis, and greater than 8 h per week of sports participation were risk factors for stress injuries. In addition, high impact activities, specifically basketball, running, and gymnastics/cheerleading were identified to significantly increase risk for stress fracture in adolescent girls [30]. Of these, training schedules, rest periods, and amount of high impact activities are potentially modifiable.

Osteochondritis Dissecans

Osteochondritis dissecans (OCD) is a disorder of subchondral bone and the overlying articular cartilage. It is hypothesized that vascular occlusion of subchondral bone leads to local inflammation and subsequent poor bone health [31]. It is an overuse injury due to repeated microtrauma that can be found at multiple joints throughout the body, including knees, ankles and elbows. The OCD lesion may remain intact, or the cartilage and bone may become a loose body in the joint. Patients may then present with decreased range of motion or mechanical symptoms such as locking, catching, or clicking.

OCD of the knee occurs most frequently at the lateral aspect of the medial femoral condyle. Males are affected more often than females, and these lesions are usually seen in older adolescents. The typical age range for patients with OCD lesions of the knee is 12–19 years [32]. Kessler et al. did not find any patients under the age of six with OCD of the knee in a 4-year study of 192 patients. This same study found that African Americans had the highest risk compared to other racial/ethnic groups [32].

OCD of the talus represents one type of osteochondral lesion of the talus that begins as a bony lesion with development of subsequent overlying articular cartilage abnormality [33]. Presentation is most common in the second decade with a range of presentation from 10 to 40 years [34]. Symptoms include pain and swelling with weight bearing activities, especially running [33].

OCD can also be seen in the elbow. Particularly in gymnasts and pitchers, OCD may result from repetitive compression and shearing forces applied to the radiocapitellar joint. These forces damage the subchondral bone's blood supply at the humeral capitellum [35]. Athletes generally present with poorly localized, activityrelated elbow pain.

OCD of the capitellum must be distinguished from osteochondrosis of the capitellum, also known as Panner's disease. Panner's disease is typically self-limited, may be related to overuse, and presents in children under 10 years of age [35]. Radiographically, Panner's disease demonstrates involvement of the entire capitellar epiphysis. The disease typically has a benign course. Recovery is achieved by rest and activity modifications to avoid pain.

Treatment for OCD lesions is based on the stability of the lesion. Plain radiographs may be used to diagnose and follow an OCD lesion, but MRI is generally required to better characterize the size and stability of the lesion. A localized lesion with flattening or radiolucency, or a more defined fragment with intact overlying cartilage, may be treated conservatively and followed closely for healing. Large lesions may require surgical intervention if they do not show healing with conservative management. Unstable lesions, where the cartilage over the subchondral bone has become cracked or compromised, allowing synovial fluid underneath the cartilage surface, generally require surgical stabilization. If a loose body has formed, surgical intervention is necessary to replace and stabilize the fragment if possible.

Apophysitis

Open growth plates are one of the many factors that put immature athletes at particular risk of certain types of injuries. Repetitive stress across the physis can result in pain, inflammation, and even fracture. In particular, during growth spurts, the cartilage cells of the hypertrophic zone of the physis become more active and are more susceptible to injury [35]. Apophysitis results from traction at the attachment site of the tendon to a secondary ossification site of bone (apophysis). Injury results from repetitive micro-trauma and muscle-tendon imbalance. Apophysitis differs from epiphysitis, which is a compression or shearing injury at the region of growing bone [28, 35].

Overuse injuries affecting the growth plates typically occur in active adolescents between the ages of 8-15 years. Because female adolescents tend to experience their growth spurt 1-2 years before males, they may also develop these conditions earlier than their male counterparts [35].

Apophysitis can occur in many different areas of the body, and symptoms vary by location. Pain is often insidious in onset and progressively worsens with activity. Spinous process apophysitis can cause extension-based back pain and tenderness along the posterior elements of the spine. Medial epicondyle apophysitis leads to elbow pain and is one of the many different injuries commonly referred to as "Little League Elbow." Iliac crest apophysitis can cause lateral hip pain and tenderness along the iliac crest. Apophysitis at the tibial tubercle, known as Osgood-Schlatter disease, is a very common cause of knee pain in a growing athlete, and calcaneal apophysitis leads to heel pain and is commonly known as Sever's disease. A similar condition at the base of the fifth metatarsal is known as Iselin's disease.

The diagnosis of apophysitis can often be made clinically. Plain films may show widening of growth plates, but in many cases they show no abnormality. Comparison of the affected and unaffected sides may allow for recognition of normal growth plates or identification of subtle abnormalities. Because symptoms of apophysitis can overlap with other conditions and treatments differ based on diagnosis, care must be taken to rule out other injuries.

Treatment of apophysitis is usually conservative. Rest, ice, compression, elevation, and NSAIDs are generally recommended [36]. NSAIDs are often used in the acute phase to assist in decreasing pain and inflammation [36]. Correcting the athlete's muscle imbalances and increasing flexibility are very important components of treatment and prevention of recurrence [28]. Although there is limited research to support the use of acupuncture, it may offer symptomatic relief to some patients. Symptoms of apophysitis tend to resolve as the athlete ages and the growth plates fuse at skeletal maturity [36].

Epiphysitis

One example of another kind of repetitive injury in the growing athlete is "Gymnast's Wrist." This condition results from compression of the distal radial physis with weight bearing. Pain is gradual in onset and worse with wrist extension, tumbling, vaulting and back walkovers [35]. Patients may have normal range of motion, but experience tenderness and swelling over the distal radial physis. Symptoms may present at an older age than typical physeal injuries secondary to delayed skeletal maturity in gymnasts and aesthetic athletes. Widening and/or sclerosis of the radial physis may be evident on radiographs. Similar changes may be noted in the ulnar growth plate and carpal bones. Permanent growth plate changes may result in shortening of the radius and a positive ulnar variance. The radial physis will typically respond to 4 weeks of rest when radiographs are negative. However, with more severe involvement healing may require 6 months or more of rest [35].

Tendinopathy

Athletes in general, and aesthetic and performing arts athletes in particular, frequently develop tendinopathy. The term tendinopathy includes tendinitis, which involves inflammation, and tendinosis, which is degeneration of the tendon without inflammation. Tendinosis often results from a more prolonged injury in older athletes [3]. Like many overuse injuries, athletes will present typically with insidious onset of pain which progresses over time. Examination reveals pain over the involved tendon often along with pain and/or weakness with resisted muscle testing. Common tendons involved include the Achilles tendon, in addition to the peroneals and posterior tibial tendons; these tendons contribute to dynamic ankle stabilization and can become overworked with repeated movements. Conventional therapies, including anti-inflammatory medications, physical therapy and orthotic devices, do not have strong scientific evidence showing efficacy; however, these treatments may provide some pain relief. Treatment with extra-corporeal shock wave therapy, injections with corticosteroid or blood products, and other interesting new treatments are being studied. The most effective treatment studied seems to be eccentric exercises implemented slowly at low intensity with gradual progression [37].

Overuse Injuries in Aesthetic and Performing Arts Athletes

Dancers, gymnasts, and figure skaters are among the athletes who combine the artistry of performance with athletic skills. The repetitions needed to master both the artistic and athletic elements make the aesthetic athlete particularly susceptible to overuse injuries. Errors in technique, combined with repetitive training, may put these athletes at increased risk of overuse injuries. Proper body alignment is an essential factor along with balanced strength and flexibility to avoid potentially harmful movement patterns. Several injuries are particularly common in aesthetic athletes.

Snapping Hip

The "snapping hip" is a common complaint among dancers, skaters and other athletes who perform repeated hip flexion, hyperextension and rotation. Often the snapping causes no pain at first and may not be recognized by the athlete as a problem. Coaches and teachers also may dismiss the problem, not understanding the importance of proper technique, alignment, flexibility and strength. These factors must be addressed to stop the condition from progressing and also to prevent maladaptive compensation, which can result in other injuries [38]. A detailed description of snapping hip can be found in Chapter 8.

Os Trigonum

Os trigonum syndrome refers to a bony and soft tissue compression resulting in posterior impingement of the ankle. The lateral tubercle of the talus can be more elongated (called a Steida process) creating a source for posterior impingement. An os trigonum results from a stress fracture/fracture of the Steida process or a failed fusion of the secondary ossification center of the lateral tubercle [39]. Clinical symptoms include stiffness and pain, particularly in the plantar flexed position, such as en pointe or relevé in dance. Swelling may develop anterior to the Achilles, and tendinitis of the flexor hallucis longus may occur concurrently [40]. Diagnosis is based on clinical history and plain X-rays, however, presence of an os trigonum on plain film is not clinically relevant without symptoms [39]. Treatment is initially conservative with rest, ice, NSAIDs, physical therapy and possible ultrasound guided injection. Failed conservative management warrants consideration for surgical excision [40].

Flexor Hallucis Longus Tendonopathy

The flexor hallucis longus (FHL) plays an important role in pointe work as well as in pushing off during steps and jumps [38]. The FHL can easily become overworked as it compensates for other weak muscles and improper foot mechanics. Proper instruction on weight distribution on the feet while standing flat, in relevé (raising oneself up onto the metatarsal heads or toes), and en pointe is important so that the foot remains aligned in all positions.

Lace Bite

For the skater, the anterior tibialis and extensor digitorum group are commonly susceptible to the compressive forces along the anterior ankle from the skating boot with repeated bending at the ankle. This condition is often referred to as "lace bite." Adaptation of the skating boot, protective padding, and the standard conservative treatment measures should be implemented [41]. It is also important to assess the overall ankle strength and stability since excess boot pressure can develop if the peroneals and posterior tibialis groups lack adequate strength.

Hypermobility

Hypermobility is a term to describe joints that stretch beyond the normal range. Increased length and elasticity of ligaments lead to increased translation at joint surfaces. Most commonly the hypermobility is benign, and individuals with flexible joints have no other symptoms. Some individuals, however, have "joint hypermobility syndrome" in which their ligamentous laxity leads to pain and other complications. Less commonly, hypermobility is associated with other systemic complications as in the setting of hereditary collagen or connective tissue defects, such as Ehlers–Danlos and Marfan syndromes. Anyone suspected of having a connective tissue disorder should be referred for genetic counseling and monitored by a rheumatologist or clinician experienced in these systemic conditions.

It is estimated that between 4–13 % of the healthy population have hypermobile joints [42, 43], and this hypermobility is more common in females. It is also more common in younger populations, and its prevalence decreases sharply through the adolescent years and then more slowly during the adult years [44], Many athletes suffer no ill effects of their hypermobility. On the contrary, hypermobility is an asset in many activities such as dance, gymnastics, and throwing sports.

Beighton's scoring system is the classic method of diagnosing joint hypermobility. There are five criteria, and because four apply to the right and left limbs individually, there are a maximum of nine points. The criteria are: passive dorsiflexion of the fifth finger beyond 90°, passive apposition of the thumb to the flexor aspect of the forearm, hyperextension at the elbow beyond 10°, hyperextension at the knee beyond 10°, and the ability to rest palms flat on the floor with trunk flexed and knees extended [45]. A Beighton score ≥ 4 is commonly used to diagnose hypermobility. However, debate continues about the most useful cut off points and the criteria for diagnosis of hypermobility. This scoring system may not be appropriate for all populations [46].

Joint hypermobility syndrome is the diagnosis of hypermobility in the setting of musculoskeletal complaints with no systemic symptoms [47]. Features may include joint pain, easy bruising, ligament and/or tendon rupture, and limb dislocations. Fatigue and muscle weakness have also been identified as symptoms [48]. The pain is thought to be due to improper joint alignment during activity [49]. Another potential contributing factor is impaired sensory feedback in the affected joints leading to excessive joint trauma [47].

Patients with joint hypermobility syndrome are at increased risk of overuse and joint instability injuries [50]. Some of the more frequent injuries include synovitis, chondromalacia patellae, shoulder instability, dorsal wrist ganglion cysts, and pes planus [51, 52]. As infants, some individuals with hypermobility may have been diagnosed with congenital hip dysplasia. As these patients age, it has been suggested that the increased translation at the articular surface will predispose them to premature osteoarthritis; however, this hypothesis has not been supported by the research [53–55].

Joint hypermobility syndrome in some individuals may be effectively treated with exercise, proper body mechanics, avoidance of aggravating activities, and joint protection (e.g., supportive bracing and taping) [56]. Nonoperative therapeutic treatment with exercise regimens should be maximized when treating these patients in order to enhance joint stability and to prevent musculoskeletal injury. Weightlifting and strength training can help stabilize the joint by developing stronger muscles to maintain proper joint alignment. In particular, the shoulder and knee joints benefit from increased muscle tone in the rotator cuff and quadriceps, respectively [57]. Resistance training should consist of open kinetic chain (distal end moves freely) and closed kinetic chain (distal end meets resistance) exercises. Other physical therapy involving balance and proprioception exercises may also be helpful in injury prevention. Water therapy may also be helpful for some individuals, especially if pain is prohibiting progress.

For acute episodes of pain, NSAIDs or acetaminophen have often been used. Rest and abstinence from symptom-inducing activity may offer additional pain relief. In the event of acute injury (such as joint subluxation), injury-specific physical therapy for treatment is advised [58].

For most, education, activity modification, and exercise therapy are important tools in symptom management. However, in some individuals, joint subluxation and repeated episodes of instability may lead to chronic pain and soft-tissue or osseous lesions, especially in the shoulder (e.g., rotator cuff injury, glenoid rim defect) and the hip (e.g., labral tear). Consultation with a pain service may be helpful in some cases. Alternative pain management options should be considered including biofeedback, acupuncture, and massage, in addition to physical therapy and pain medications if needed. If conservative measures are unsuccessful, consultation with an orthopedic surgeon regarding surgical intervention may be necessary to improve symptoms and function. Hypermobility syndrome itself is not a contraindication to surgery; however, surgical techniques may need to be modified due to the nature of the tissues in patients with hypermobility syndrome [59]. Patients should also be counseled that because the underlying cause of joint laxity cannot be altered, hypermobility may affect surgical outcomes.

Conclusion

Many injuries in the young female athlete are due to overuse, resulting from repetitive stress on the musculoskeletal system that overwhelms the body's healing mechanisms. Especially during times of rapid growth, the young athlete is at particular risk of injury. These injuries generally build up gradually over time, and thus there are many opportunities to intervene, prevent progression, and allow for healing. When injuries do occur, complete rehabilitation is important to help athletes heal fully and reduce the risk of future injuries.

References

- 1. Valovich McLeod TC, et al. National Athletic Trainers' Association position statement: prevention of pediatric overuse injuries. J Athl Train. 2011;46(2):206–20.
- Clarsen B, Myklebust G, Bahr R. Development and validation of a new method for the registration of overuse injuries in sports injury epidemiology: the Oslo Sports Trauma Research Centre (OSTRC) overuse injury questionnaire. Br J Sports Med. 2013;47(8):495–502.
- Maffulli N, Wong J, Almekinders LC. Types and epidemiology of tendinopathy. Clin Sports Med. 2003;22(4):675–92.
- 4. Hoang QB, Mortazavi M. Pediatric overuse injuries in sports. Adv Pediatr. 2012;59(1): 359-83.
- 5. Outerbridge AR, Micheli LJ. Overuse injuries in the young athlete. Clin Sports Med. 1995;14(3):503–16.
- 6. Difiori JP, et al. Overuse injuries and burnout in youth sports: a position statement from the American Medical Society for Sports Medicine. Br J Sports Med. 2014;48(4):287–8.
- 7. Kerssemakers SP, et al. Sport injuries in the paediatric and adolescent patient: a growing problem. Pediatr Radiol. 2009;39(5):471–84.
- Ruedl G, et al. Sport injuries and illnesses during the first Winter Youth Olympic Games 2012 in Innsbruck, Austria. Br J Sports Med. 2012;46(15):1030–7.
- 9. Brenner JS. Overuse injuries, overtraining, and burnout in child and adolescent athletes. Pediatrics. 2007;119(6):1242–5.
- 10. Ivkovic A, et al. Overuse injuries in female athletes. Croat Med J. 2007;48(6):767-78.
- 11. DeHaven KE, Lintner DM. Athletic injuries: comparison by age, sport, and gender. Am J Sports Med. 1986;14(3):218–24.
- Taunton JE, et al. A retrospective case-control analysis of 2002 running injuries. Br J Sports Med. 2002;36(2):95–101.
- 13. Earl JE, Vetter CS. Patellofemoral pain. Phys Med Rehabil Clin N Am. 2007;18(3):439-58. viii.
- Fredericson M, Powers CM. Practical management of patellofemoral pain. Clin J Sport Med. 2002;12(1):36–8.
- 15. Van Tiggelen D, et al. Delayed vastus medialis obliquus to vastus lateralis onset timing contributes to the development of patellofemoral pain in previously healthy men: a prospective study. Am J Sports Med. 2009;37(6):1099–105.
- 16. Waryasz GR, McDermott AY. Patellofemoral pain syndrome (PFPS): a systematic review of anatomy and potential risk factors. Dyn Med. 2008;7:9.
- 17. Lankhorst NE, Bierma-Zeinstra SM, van Middelkoop M. Risk factors for patellofemoral pain syndrome: a systematic review. J Orthop Sports Phys Ther. 2012;42(2):81–94.
- Barton CJ, et al. Foot and ankle characteristics in patellofemoral pain syndrome: a case control and reliability study. J Orthop Sports Phys Ther. 2010;40(5):286–96.

- 6 Overuse Injuries in Young Female Athletes
- Prins MR, van der Wurff P. Females with patellofemoral pain syndrome have weak hip muscles: a systematic review. Aust J Physiother. 2009;55(1):9–15.
- 20. Thomee R, Augustsson J, Karlsson J. Patellofemoral pain syndrome: a review of current issues. Sports Med. 1999;28(4):245-62.
- 21. Myer GD, et al. High knee abduction moments are common risk factors for patellofemoral pain (PFP) and anterior cruciate ligament (ACL) injury in girls: is PFP itself a predictor for subsequent ACL injury? Br J Sports Med. 2015;49:118.
- 22. Morelli V, Braxton Jr TM. Meniscal, plica, patellar, and patellofemoral injuries of the knee: updates, controversies and advancements. Prim Care. 2013;40(2):357–82.
- 23. D'Hondt NE, et al. Orthotic devices for treating patellofemoral pain syndrome. Cochrane Database Syst Rev 2002;(2):Cd002267.
- 24. Wentz L, et al. Females have a greater incidence of stress fractures than males in both military and athletic populations: a systemic review. Mil Med. 2011;176(4):420–30.
- Pepper M, Akuthota V, McCarty EC. The pathophysiology of stress fractures. Clin Sports Med. 2006;25(1):1–16. vii.
- 26. Dissmann PD, Han KH. The tuning fork test—a useful tool for improving specificity in "Ottawa positive" patients after ankle inversion injury. Emerg Med J. 2006;23(10):788–90.
- Chen YT, Tenforde AS, Fredericson M. Update on stress fractures in female athletes: epidemiology, treatment, and prevention. Curr Rev Musculoskelet Med. 2013;6(2):173–81.
- 28. d'Hemecourt P. Overuse injuries in the young athlete. Acta Paediatr. 2009;98(11):1727-8.
- 29. Young AJ, McAllister DR. Evaluation and treatment of tibial stress fractures. Clin Sports Med. 2006;25(1):117–28. x.
- Field AE, et al. Prospective study of physical activity and risk of developing a stress fracture among preadolescent and adolescent girls. Arch Pediatr Adolesc Med. 2011;165(8):723–8.
- O'Loughlin PF, Heyworth BE, Kennedy JG. Current concepts in the diagnosis and treatment of osteochondral lesions of the ankle. Am J Sports Med. 2010;38(2):392–404.
- 32. Kessler JI, et al. The demographics and epidemiology of osteochondritis dissecans of the knee in children and adolescents. Am J Sports Med. 2014;42(2):320–6.
- Zanon G, DI Vico G, Marullo M. Osteochondritis dissecans of the talus. Joints. 2014;2(3):115–23.
- 34. Vannini F, et al. Treatment of juvenile osteochondritis dissecans of the talus: current concepts review. Joints. 2014;2(4):188–91.
- 35. Frush TJ, Lindenfeld TN. Peri-epiphyseal and overuse injuries in adolescent athletes. Sports Health. 2009;1(3):201–11.
- Atanda Jr A, Shah SA, O'Brien K. Osteochondrosis: common causes of pain in growing bones. Am Fam Physician. 2011;83(3):285–91.
- 37. Kaux JF, et al. Current opinions on tendinopathy. J Sports Sci Med. 2011;10(2):238-53.
- 38. Solomon R, et al. The young dancer. Clin Sports Med. 2000;19(4):717-39.
- Nault ML, Kocher MS, Micheli LJ. Os trigonum syndrome. J Am Acad Orthop Surg. 2014;22(9):545–53.
- Kadel N, Micheli LJ, Solomon R. Os trigonum impingement syndrome in dancers. J Dance Med Sci. 2000;4(3):99–102.
- 41. Smith AD. The young skater. Clin Sports Med. 2000;19(4):741–55.
- 42. Biro F, Gewanter HL, Baum J. The hypermobility syndrome. Pediatrics. 1983;72(5):701-6.
- Seçkin U, Tur BS, Yilmaz O, Yagci I, Bodur H, Arasil T. The prevalence of joint hypermobility among high school students. Rheumatol Int. 2005;25(4):260–3. doi:10.1007/s00296-003-0434-9.
- 44. Grahame R. The hypermobility syndrome. Ann Rheum Dis. 1990;49(3):199-200.
- 45. Beighton P, Solomon L, Soskolne CL. Articular mobility in an African population. Ann Rheum Dis. 1973;32:413–8.
- 46. Clinch J, Deere K, Sayers A, Palmer S, Riddoch C, Tobias JH, Clark EM. Epidemiology of generalized joint laxity (hypermobility) in fourteen-year-old children from the UK: a population-based evaluation. Arthritis Rheum. 2011;63(9):2819–27. doi:10.1002/art.30435.
- Simpson MR. Benign joint hypermobility syndrome: evaluation, diagnosis, and management. J Am Osteopath Assoc. 2006;106(9):531–6.

- Mato H, Berde T, Hasson N, Grahame R, Maillard S. A review of symptoms associated with Benign Joint Hypermobility Syndrome in children. Pediatr Rheumatol Online J. 2008;6 Suppl 1:155. doi:10.1186/1546-0096-6-S1-P155.
- Soep JB. Rheumatic diseases. In: Hay Jr WW, Levin MJ, Deterding RR, Ross JJ, Sondheimer JM, editors. CURRENT diagnosis & treatment: pediatrics. 21st ed. New York: McGraw-Hill; 2012 (Chapter 29).
- Adib N, Davies K, Grahame R, Woo P, Murray KJ. Joint hypermobility syndrome in childhood. A not so benign multisystem disorder? Rheumatology (Oxford). 2005;44(6):744–50. doi:10.1093/rheumatology/keh557.
- Johnson SM, Robinson CM. Shoulder instability in patients with joint hyperlaxity. J Bone Joint Surg Am. 2010;92(6):1545–57. doi:10.2106/JBJS.H.00078.
- McKeon KE, London DA, Osei DA, Gelberman RH, Goldfarb CA, Boyer MI, Calfee RP. Ligamentous hyperlaxity and dorsal wrist ganglions. J Hand Surg Am. 2013;38(11): 2138–43. doi:10.1016/j.jhsa.2013.08.109.
- 53. Bird HA, Barton L. Joint hyperlaxity and its long-term effects on joints. J R Soc Health. 1993;113(6):327–9. doi:10.1177/146642409311300613.
- 54. Chen HC, Shah SH, Li YJ, Stabler TV, Jordan JM, Kraus VB. Inverse association of general joint hypermobility with hand and knee osteoarthritis and serum cartilage oligomeric matrix protein levels. Arthritis Rheum. 2008;58(12):3854–64. doi:10.1002/art.24319.
- 55. Dolan AL, Hart DJ, Doyle DV, Grahame R, Spector RD. The relationship of joint hypermobility, bone mineral density, and osteoarthritis in the general population: the Chingford Study. J Rheumatol. 2003;30(4):799–803.
- Scheper MC, Engelbert RH, Rameckers EA, Verbunt J, Remvig L, Juul-Kristensen B. Children with generalised joint hypermobility and musculoskeletal complaints: state of the art on diagnostics, clinical characteristics, and treatment. Biomed Res Int. 2013;2013:121054. doi:10.1155/2013/121054.
- 57. Turner JK, Schlesinger P. Approach to the adolescent with arthritis. In: Imboden JB, Hellmann DB, Stone JH, editors. CURRENT diagnosis & treatment: rheumatology. 3rd ed. New York: McGraw-Hill; 2013 (Chapter 5).
- 58. Everman DB, Robin NH. Hypermobility syndrome. Pediatr Rev. 1998;19(4):111–7. doi:10.1542/pir.19-4-111.
- 59. Hakim A, Grahame R. Joint hypermobility. Best Pract Res Clin Rheumatol. 2003;17(6): 989–1004. doi:10.1016/j.berh.2003.08.001.

Chapter 7 Spondylolysis

Pierre A. d'Hemecourt, Laura E. Gould, and Nicole M. Bottino

Introduction

Back pain in the female athlete is a common complaint. It may represent a simple muscular or ligamentous injury that will often resolve spontaneously. However, when pain is prolonged, a number of more significant considerations should be entertained, including disk pathology, stress fractures, and bone tumors. One common cause of back pain in the adolescent athlete that should be considered is spondylolysis.

Spondylolysis is an overuse injury to the posterior elements of the spine where a stress fracture occurs at the pars interarticularis (pars), between the facet joints. Repeated hyperextension and torsion of the spine are the main mechanisms for this injury. In this type of motion, the superior facet places an increased load on the pars [1]. Over time, there is enough stress across the pars that a stress fracture forms.

These stress fractures can occur unilaterally or bilaterally, and can take place at any level of the lumbar spine. The preponderance of cases, roughly 85-95 %, are seen at L5. The second most common site is L4, with 5-15 % of cases occurring at this location [2]. In 4 % of people experiencing spondylolysis, stress injuries occur at multiple levels [3]. Bilateral involvement occurs in 80 % of cases, and can result in spondylolisthesis, a slippage of one vertebra anteriorly over the level below it [1].

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Definitions

Pars interarticularis: the area of the vertebrae located between the inferior and superior articular processes of the facet joints. The pars is situated in the posterior column of the spine, which is defined as the bony and ligamentous structures posterior to the vertebral body (Fig. 7.1).

Spondylolysis: a stress fracture in the pars interarticularis of the vertebral posterior elements (Fig. 7.2).

Spondylolisthesis: the slippage that occurs when a vertebra becomes displaced in relation to the vertebra below it (Fig. 7.3).

Facet joint: also known as zygapophyseal joint, the articulation between the superior articular process of one vertebra with the inferior articular process of the vertebra superior to it.

Stress fracture: also known as fatigue fracture, a fracture in the bone as a result of repetitive loading of a specific area over a period of time.

Lordosis: the normal sagittal inward curvature of the lumbar spine. When it is excessive it is referred to as hyperlordosis (sway back). Factors that contribute to hyperlordosis are anterior pelvic tilt and core imbalance of strength and flexibility.

Kyphosis: the normal thoracic outward curvature. At times it can be excessive and is referred to as round back. Excessive kyphosis may secondarily increase lumbar lordosis in order to maintain horizontal vision.

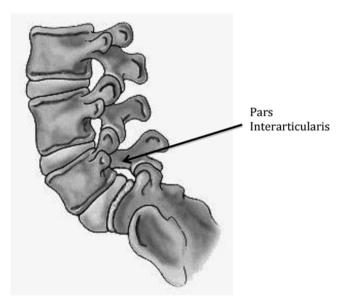


Fig. 7.1 Pars interarticularis

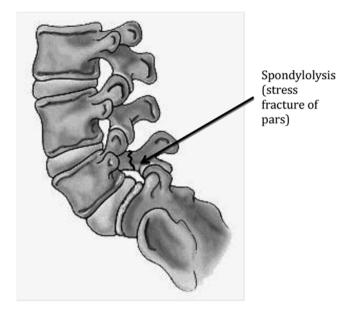


Fig. 7.2 Spondylolysis

Fig. 7.3 Spondylolisthesis



Epidemiology and Risk Factors

Spondylolysis and spondylolisthesis are the most common causes of back pain in young athletes. While the most common cause of back pain in adults is disk pathology, Micheli and Wood have reported the rate of spondylolysis to be as high as 47 % in the adolescent athletic population with back pain [4]. When looking at the adolescent population alone, the prevalence of back pain is 45 % in the athletic adolescent in comparison to 15 % in the sedentary adolescent [5]. Anyone who cares for young athletes should know how to diagnose, treat, and prevent this injury. The ability to identify risk factors for this condition is an important place to begin.

During growth acceleration, the female athlete experiences several anatomic changes that increase stress on the posterior elements of the spine. With the adolescent growth spurt, the anterior pelvic tilt increases. Lordosis of the spine progressively increases, which has been shown to result in back pain [14]. This pain may be due to increased compression of the posterior column and facet joints.

The rate of bony growth during this time exceeds that of musculotendinous growth. The subsequent muscular imbalance and tightness are seen particularly with the hip flexors and the thoracolumbar fascia. These changes, combined with weakness of the abdominal musculature and weakness of the gluteus maximus, have been shown to increase lumbar lordosis and are associated with back pain [6]. Athletic activity, particularly gymnastics, is an additional factor that has been shown to contribute to increased lordosis [7]. In female gymnasts participating in greater than 300 h per year, there was a noted increase in lordosis and kyphosis.

Rapid linear growth during the growth spurt occurs with a much slower acquisition of bone mineralization; therefore there is temporarily a lower bone density [8]. The combination of these factors follows an order of increased linear growth with decreased flexibility and temporary bone acquisition and increased anterior pelvic tilt. See Chapter 1 for additional details on normal growth.

Genetics also contribute to spondylolysis risk to a lesser extent. A study by Roche showed that in the prevalence of spondylolysis, the racial/ethnic breakdown includes 6.4 % of Caucasian men, 2.8 % of African American men, 2.3 % of Caucasian women, and 1.1 % of African American women [9]. Interestingly, Canadian Inuit Eskimos show the highest prevalence, with a reported rate as high as 50 % [11]. Spondylolysis has a familial association. One study reported spondylolysis in 19 % of first-degree relatives [10].

In the past, sex was thought to be a factor in the development of spondylolysis because the condition was seen two to three times more commonly in males than females; however, this is no longer the case [11]. As females have become more active in organized and competitive sports, the development of spondylolysis is now just as common in females as it is in males. In addition, sports and activities such as gymnastics, figure skating, ballet, and other forms of dance, have both a high level of female athlete participation and a higher incidence of spondylolysis than other sports [12].

Some spinal deformities have been found to be risk factors in the development of spondylolysis. The presence of Scheuermann's kyphosis, a fixed severe thoracic

outward curvature, has been noted to increase the incidence of spondylolysis by 30-50 % [13, 14]. This is likely due to an increase in lordosis that occurs with Scheuermann's kyphosis. Spina bifida occulta has also been related to an increased incidence of spondylolysis by 3.7 % [15].

The biomechanics of specific sports are also predisposing factors for spondylolysis. Flexion, extension, rotation, and shear forces all play a role in the development of spondylolysis [16]. Sports that require a combination of repetitive extension and rotation of the spine place the greatest stress on the pars and have the highest incidence of spondylolysis [17]. The initiation of a pars fracture also involves a tensile force on the ventral aspect of the pars [18]. Sports and activities with the highest risk for the development of spondylolysis include dance, gymnastics, figure skating, wrestling, diving, and football, specifically the lineman position. All of these sports involve extremes of the motions mentioned above [19, 20]. The incidence of spondylolysis has been reported to be as high as 40 % in diving and 32 % in ballet dancing [21].

Spondylolisthesis

Bilateral spondylolysis occurs in 80 % of cases and can result in slippage of one vertebra over another, a condition known as spondylolisthesis [1]. The Wiltse Classification is used to classify the etiology of spondylolisthesis. There are five types described, with type II being the most common type seen with a sports-related mechanism. Type I is dysplastic, type II is isthmic (related to overuse of the pars seen most frequently in the female athlete), type III is degenerative (usually presenting during middle age at the L4-5 level), type IV is traumatic, and type V is a pathologic fracture. Thirty-two percent of type I fractures are likely to progress with increasing slippage. Conversely, only 4 % of type II are likely to progress [22].

The Myerding Classification is used to classify the actual degree of slippage that occurs in spondylolisthesis. It is based on the percentage of slippage of the involved vertebrae on the vertebrae below it. Grade I is less than or equal to 25 %, grade II is 26–50 %, grade III is 51–75 %, grade IV is 76–100 %, and grade V is greater than 100 %. Grade V is also referred to as spondyloptosis [23].

Clinical Presentation

In the adolescent athlete, back pain typically begins during sports activities, particularly those involving lumbar extension. The pain generally develops gradually with increased activity. Spondylolysis is an overuse injury that develops over time; however, patients often recall a particular injury or competition when the pain began. The location of pain is usually described as either central or off to one side with radiation to the buttocks or proximal extremity. In the early stages, the pain resolves after activity. However, as the spondylolysis progresses, it can persist after activity, even at rest. Neurologic signs are rare and are more often present in a high-grade



Fig. 7.4 Stork test

spondylolisthesis or disk herniation. However, sciatic symptoms occasionally occur, presumably secondary to the site of the fracture being one wall of the neuroforamina.

There are a number of common physical examination findings associated with spondylolysis. Patients typically do not have tenderness with palpation along the spine unless there is overlying spasm of the paraspinous musculature. Patients generally have no pain with flexion, but significant pain with hyperextension. Hyperextension of the spine with the patient standing on one leg (Stork test) helps to illicit information about the side of the spondylolysis (Fig. 7.4); pain on the weight-bearing side is indicative of a fracture [24]. In the athlete with spondylolisthesis, there may be a palpable step off with higher grades of slippage. Patients also commonly present with tight hamstrings. The strength and flexibility of the athlete should always be assessed in order to identify any abnormalities and to structure a regimented physical therapy program. A tight iliopsoas combined with weak gluteal and abdominal muscles can result in an anteriorly rotated pelvis with increased lordosis contributing to the progression of the spondylolysis [25].

Differential Diagnosis

There are several other diagnoses that the clinician must consider when seeing the female athlete with extension-based back pain. These include lumbar disk injuries, sacroiliac instability, lordotic low back pain, and segmentation abnormalities.

While lumbar disk-related pathology is uncommon in children and young adolescents, it may occur. Typically, patients present with worsened pain on flexion, such as during sitting. However, central disk protrusion may elicit pain on extension, causing some clinical confusion.

Another entity involving the disk in the young athlete is referred to as atypical Scheuermann's disease. Typical Scheuermann's disease is a kyphosis, which involves pathologic changes in the upper thoracic spine; this condition occurs when the disk compresses the soft growth plate of the vertebral endplate and causes wedging in the bone. When these changes occur in the upper lumbar and lower thoracic spine, they are often painful and result in a more flat back appearance, called atypical Scheuermann's.

Athletes with any type of lumbar injury may develop atrophy of the lumbar extensor musculature due to pain inhibition and disuse. This atrophy may result in some degree of sacroiliac instability, which typically presents with pain at the superior buttocks adjacent to the L5 region. Pain often increases with lumbar flexion as well as extension. A sacroiliac (SI) provocation maneuver, such as the thigh thrust or thigh compression test, can be helpful to distinguish between SI instability and spondylolysis. This maneuver involves having the supine patient flex both her hip and knee to 90°. The clinician then pushes the thigh posteriorly into the pelvis. Pain at the posterior superior iliac spine marks a positive test for SI instability.

Another possible diagnosis to consider is lordotic low back pain, which generally presents with diffuse, multilevel back pain and significant lordosis. Palpation may reveal tender posterior elements along the entire lumbar spine, including the facet joints and spinous processes. Apophysitis of the growth cartilage of the spinous processes can result in discomfort and tenderness to light palpation directly over the spinous processes, which would not be expected with simple spondylolysis. Most commonly, these diagnoses are made after spondylolysis has been ruled out.

Abnormalities of segmentation of the lower lumbar spine are common. These include lumbar super-segmentation with a lumbarized S1 (i.e., "L6" is not incorporated into the sacrum), which is not commonly associated with back pain. Conversely, incomplete segmentation of L5 is associated with a unilateral bony bridge from L5 that remains in continuity with the sacrum and creates a pseudoar-throsis. This pseudoarthrosis is a common cause of back pain in the athlete and often presents in a similar fashion to spondylolysis or SI pain with pain on hyper-extension. It is identified by plain radiographs, and the pain often correlates with the area of pseudoarthrosis.

Diagnostic Testing

In order to confirm the diagnosis of spondylolysis, imaging is often needed if the pain is more than 3–4 weeks in duration. Plain radiographs may be the first line of imaging although they are not sensitive for picking up the disease process. The oblique view may yield the "collar on the scotty dog" but has been shown to be only 32 % sensitive and adds unnecessary radiation [26]. Many clinicians will now

simply avoid the initial radiographs in cases of prolonged extension-based back pain in the young athlete and go straight to advanced imaging. However, plain radiographs with anteroposterior and lateral views are useful in detecting spondylolisthesis and transitional vertebrae (incomplete segmentation of vertebrae).

If suspicion is high, the most sensitive test is single-photon emission computed tomography (SPECT) [27]. SPECT scanning can also evaluate osseous healing as it is able to evaluate metabolic activity of bone [28]. In addition, it can be helpful in looking at other etiologies of pain such as SI pain, osteoid osteomas, and transitional vertebrae pseudoarthrosis [29]. However, there is a significant exposure to radiation with bone scans, which should be considered.

Magnetic resonance imaging (MRI) is commonly used for detecting spondylolysis. MRI can detect bone marrow edema in the pedicle and pars region to identify the disease process at an early stage [30]. The MRI techniques involve a sagittal STIR image that looks at pedicle edema, as well as axial T1 and T2 sequences. The STIR images demonstrate the acuity of the pars involvement much like the bone scan. Early detection of a stress reaction through MRI may prevent exacerbation of the condition and development of a pars defect [31]. MRI tends to be a preferred method of imaging in young athletes, as it does not involve radiation exposure. It can also help detect other pain generators, such as disk pathology or other lesions in the bone or soft tissue. At this time, many clinicians will use the MRI as the first line of imaging in athletes with persistent extension-based back pain.

Despite its many advantages, MRI is not as accurate as computed tomography (CT) in demonstrating bony detail. Once a fracture is detected, CT can classify lesions as early, progressive, or chronic. This characterization of the fracture can help to predict which fractures will heal. One study demonstrated healing in 73 % of early fractures, while only 39 % of progressive fractures healed, and none of the terminal or bilateral fractures healed [3]. Unfortunately, CT scanning involves radiation exposure. The amount of radiation is best minimized by scanning only the vertebral level of concern.

In the authors' experience, a limited CT of the affected level is used at times if the fracture is not responding in a clinically favorable manner. If the patient has persistent pain after 6–8 weeks of treatment, a CT may help provide more information for prognosis. An initial CT at the time of diagnosis is usually not needed.

Treatment

There is some controversy and variability in the treatment of spondylolysis. Treatment regimens vary in terms of brace utilization, activity modification, and physical therapy. Research does not support the superiority of one specific treatment over others; however, many studies have not adequately differentiated patients by age and fracture acuity. Furthermore, bony union is desirable, but fracture healing has not been correlated with successful outcomes [32]. Nonetheless, the clinician should attempt to gain a bony union when able. It is likely that the younger athlete

with an early phase fracture will heal with a bony union. Those with a subacute or more chronic fracture may not attain a bony union, but usually do well with a stable fibrous union [33]. When considering treatment options, the principals remain the same, and each case should be evaluated and treated based on presentation.

Most treatment plans begin with activity modification. Some providers opt to restrict all sports and physical activities until the patient is pain-free, while others may choose to restrict only those activities that cause pain. Some providers suggest additional time out of sports even after the patient's pain has resolved. Most protocols utilizing only activity modification will recommend that the athlete remains out of sports for 3–4 months [34]. In one study on pediatric soccer players, those athletes who did not adhere to activity modification demonstrated diminished athletic performance [35]. When the Boston Overlap Brace (BOB) protocol is used as described below, the athlete will typically be out of sports for 4–6 weeks before returning to the athletic arena as tolerated.

The use of a brace in treating spondylolysis remains somewhat controversial. There has not been a definitive study demonstrating improved healing with or without bracing. Nonetheless, one retrospective study showed the utilization of the hard, customized BOB allowed early return to sports at 4–6 weeks while continuing brace use [36]. Bracing may consist of either a hard lumbosacral orthosis (LSO), the most common of which is the BOB, or a less rigid transitional brace.

There are three basic protocols for brace utilization. The first and one of the more commonly used regimens does not involve using a brace unless the patient has persistent symptoms after 3–4 months [37]. However, this approach often involves a more prolonged period of activity modification.

The second bracing protocol utilizes a smaller transitional brace along with activity modification for 6–8 weeks. At that time the athlete is assessed for continued pain and dysfunction. If pain persists, especially on lumbar hyperextension, the more rigid BOB is then utilized. This bracing would be combined with activity modification and maintained for an additional 6–8 weeks [2].

The third bracing protocol is the one the authors most often recommend. This utilizes the BOB after confirmation of the diagnosis. The patient is generally placed in the brace for 23 h/day, coming out only to shower and begin anti-lordotic physical therapy exercises. The athlete is reevaluated at 4–6 weeks [34]. If she is pain-free at that time and demonstrates no pain on lumbar extension, the brace is trimmed laterally and the athlete is allowed to return to sports in the brace. It is emphasized to the athlete that this return to activity is dependent on remaining pain-free, wearing the BOB brace, and continuing with a regular physical therapy program. At this point, some providers will allow the athlete to come out of the brace at night. Most athletes are able to participate in their sport and activities in the brace, but some sports such as gymnastics and dance will be significantly limited by brace wear. At this 4–6 week mark, the core stabilization is advanced to incorporate all of the lumbar extensors, while limiting hyperextension. With brace wear, it is crucial to avoid erector spinae and multifidi atrophy.

For most athletes using this protocol, the brace is continued for 3–4 months. However, in some circumstances, shorter treatment periods of 6–8 weeks may be considered; these shorter periods of brace use may be utilized in cases of stress reaction with no identified fracture, or subacute fracture with minimal edema on the T2 MRI signal. Early fractures may heal in as little as 3 months, whereas more chronic fractures can take up to 6 months to heal [38].

The athlete is reevaluated at 3–4 months from brace initiation to identify any residual pain as well as to assess strength and flexibility. If the athlete is pain-free and demonstrates good strength and flexibility, the brace is weaned off over the next 2–3 weeks. A smaller, less restrictive Velcro transitional brace for sports only may be considered. Most importantly, education regarding repetitive extension-based activity should be given in order to prevent recurrence.

Patients with persistent pain at this 3- to 4-month point should be evaluated for adherence to brace use and physical therapy. Providers should also consider persistent fracture and other pain generators, such as facet arthropathy or SI joint pain. Surgical consideration may be necessary at this stage. In patients with persistent pain and with non-union of the fracture, a bone stimulator may be considered to advance healing. While bone stimulators have not been shown to expedite healing in the acute phase of spondylolysis, there is evidence to suggest improved bony healing in chronic cases [39].

When considering other potential contributors to the pain, PT modifications and steroid injections can be utilized. PT can be directed at peri-pelvic strengthening as well as SI stabilization. Alternatively, SI or facet injections can be both diagnostic and therapeutic.

There are a few complications that can arise despite treatment. In general, the outcome of unilateral pars fractures is good [2]. However, up to 25 % of cases can develop a stress response or fracture in the contralateral side leading to bilateral fractures. In addition, patients with bilateral pars defects can also develop spondylo-listhesis. These often do not progress after diagnosis.

Surgery is sometimes considered in cases of spondylolysis and spondylolisthesis. Surgery is reserved for truly refractory cases despite full conservative management of at least 6–12 months of treatment. Other considerations for surgery include progression of spondylolisthesis, a related neurologic deficit, or refractory radicular symptoms [40]. The surgical treatment may involve a fusion or in some cases a direct pars repair. Indications for a direct repair include minimal disk degeneration and less than 3 mm of spondylolisthesis [40]. Return to sports after surgery is often in the 6–12 month range.

Conclusion

Spondylolysis is an overuse injury to the posterior elements of the spine in which a stress fracture occurs at the pars interarticularis of the vertebra. Repeated hyperextension and torsion of the spine, which are common movements in many different types of sports, are the main mechanisms for this injury. The young female athlete

is at particular risk for spondylolysis, especially during times of rapid growth with concurrent core weakness and muscle tightness.

When spondylolysis does occur, the best treatment starts with early detection to avoid prolongation of symptoms and progression of the injury. Treatment centers on limitation of extension and initiation of anti-lordotic and core stability exercises. Activity modification is usually a temporary but important element of treatment.

These stress injuries are best prevented with good core stabilization and limitation of hyperextension in the growing athlete. Therefore, it is necessary to recognize risk factors and treat the core instability, poor biomechanics, and muscular imbalances that can predispose young athletes to injury. Many of these risk factors can be identified through a thorough preparticipation exam.

References

- Labelle H, Roussouly P, Berthonnaud E, Dimnet J, O'Brien M. The importance of spino-pelvic balance in L5-S1 developmental spondylolisthesis. A review of pertinent radiologic measurements. Spine. 2005;30(6, suppl):S27–34.
- 2. Standaert DC, Herring S. Spondylolysis: a critical review. Br J Sports Med. 2000;34: 415–22.
- Morita T, Ikata T, Katoh S, Miyake R. Lumbar spondylolysis in childrens and adolescents. J Bone Joint Surg Br. 1995;77(4):620–5.
- Micheli LJ, Wood R. Back pain in young athletes. Significant differences from adults in causes and patterns. Arch Pediatr Adolesc Med. 1995;149:15–8.
- Kujala UM, Taimela S, Erkintalo M, Salminen JJ, Kaprio J. Low-back pain in adolescent athletes. Med Sci Sports Exerc. 1996;28:165–70.
- 6. Gilchrist RV, Frey ME, Nadler SF. Muscular control of the lumbar spine. Pain Physician. 2003;6(3):361–8.
- 7. Wojts EEM, Ashton-Miller JA, Huston LJ, et al. The association between athletic training time and the sagittal curvature of the immature spine. Am J Sports Med. 2000;28(4):490–8.
- 8. Bass S, Delmas P, Pearce G, et al. The differing tempo of growth in bone size, mass, and density in girls is region-specific. J Clin Invest. 1999;104(6):795–804.
- 9. Roche MB, Rowe GG. The incidence of separate neural arch and coincident bone variations; a survey of 4,200 skeletons. Anat Rec. 1951;109:233–52.
- Merbs CF. Patterns of activity-induced pathology in a Canadian Inuit population, Archaeological Survey of Canada, vol. 119. Ottawa, ON: National Museums of Canada; 1983. p. 120–8.
- 11. Nadler SF, et al. Hip muscle imbalance and low back pain in athletes: influence of core strengthening. Med Sci Sports Exerc. 2002;34(1):9–16.
- 12. Kujala UM, et al. Lumbar mobility and low back pain during adolescence. A longitudinal three-year follow up study in athletes and controls. Am J Sports Med. 1997;25(3):363–8.
- Ogilvie JW, Sherman J. Spondylolysis in Scheuermann's disease. Spine (Phila Pa 1976). 1987;12:251–3.
- 14. Greene TL, Hensinger RN, Hunter LY. Back pain and vertebral changes simulating Scheuermann's disease. J Pediatr Orthop. 1985;5:1–7.
- Sakai T, Sairyo K, Takao S, et al. Incidence of lumbar spondylolysis in the general population in Japan based on multi-detector CT scans from 2000 subjects. Spine (Phila Pa 1976). 2009;34:2346–50.

- Farfan H, Osteria V, Lamy C. The mechanical etiology of spondylolysis and spondylolisthesis. Clin Orthop. 1976;117:40–55.
- 17. Sairyo K, Katoh S, Komatsubara S, Terai T, Yasui N, Goel VK, et al. Spondylolysis fracture angle in children and adolescents on CT indicates the fracture producing force vector: a biomechanical rationale. Internet J Spine Surg 2005;1(2).
- Terai T, Sairyo K, Goel VK, Ebraheim N, Biyani A, Faizan A, et al. Tensile stress at the ventral aspect of the pars interarticularis causes the initial defect of the pediatric lumbar spondylolysis. 38th annual meeting of the Japanese Society for Spine Surgery and Related Research. J Bone Joint Surg Br. 2009;20:390.
- McTimoney CA, Micheli LJ. Current evaluation and management of spondylolysis and spondylolisthesis. Curr Sports Med Rep. 2003;2:41–6.
- 20. Rossi F, Dragoni S. Lumbar spondylolysis: occurrence in competitive athletes. Updated achievements in a series of 390 cases. J Sports Med Phys Fitness. 1990;30(4):450–2.
- Seitsalo S, Osterman K, Poussa M. Scoliosis associated with lumbar spondylolisthesis: a clinical survey of 190 young patients. Spine (Phila Pa 1976). 1988;13:899–904.
- McPhee IB, O'Brien JP, McCall IW, Park WM. Progression of lumbosacral spondylolisthesis. Australas Radiol. 1981;25:91–5.
- Hu SS, Bradford DS. Spondylolysis and spondylolisthesis. In: Weinstein SL, editor. The pediatric spine. Philadelphia, PA: Lippincott Williams & Wilkins; 2001. p. 433–51.
- 24. Weiker GG. Evaluation and treatment of common spine and trunk problems. Clin Sports Med. 1989;8(3):399–417.
- 25. Janda V. Muscles and motor control in low back pain: assessment and management. In: Twomey LT, editor. Physical therapy of the low back. New York, NY: Churchill Livingstone; 1987. p. 253–78.
- Saifuddin A, White J, Tucker S, Taylor BA. Orientation of lumbar pars defects: implications for radiological detection and surgical management. J Bone Joint Surg Br. 1998;80(2): 208–11.
- 27. Bellah RD, Summerville DA, Treves ST, Micheli LJ. Low-back pain in adolescent athletes: detection of stress injury to the pars interarticularis with SPECT. Radiology. 1991;180(2): 509–12.
- Van den Oever M, Merrick MV, Scott JH. Bone scintigraphy in symptomatic spondylolysis. J Bone Joint Surg Br. 1987;69(3):453–6.
- 29. Drubach L, Connolly L, d'Hemecourt P, et al. Assessment of the clinical significance of asymptomatic lower extremity uptake abnormality in young athletes. J Nucl Med. 2001;42:209–12.
- Hollenberg GM, Beattie PF, Meyers SP, Weinberg EP, Adams MJ. Stress reactions of the lumbar pars interarticularis: the development of a new MRI classification system. Spine. 2002;27(2):181–6.
- Cohen E, Stuecker RD. Magnetic resonance imaging in diagnosis and follow-up of impending spondylolysis in children and adolescents. Early treatment may prevent pars defects. J Pediatr Orthop B. 2005;14(2):63–7.
- Klein G, Mehlman C, McCarty M. Nonoperative treatment of spondylolysis and grade I spondylolisthesis in children and young adults: a meta-analysis of observational studies. J Pediatr Orthop. 2009;29:146–56.
- Beutler WJ, Fredrickson BE, Murtland A, Sweeney CA, Grant WD, Baker DB. The natural history of spondylolysis and spondylolisthesis: 45-year follow-up evaluation. Spine. 2003;28:1027–35. discussion 1035.
- 34. Standaert CJ, Herring SA. Expert opinion and controversies in sports and musculoskeletal medicine: the diagnosis and treatment of spondylolysis in adolescent athletes. Arch Phys Med Rehabil. 2007;88(4):537–40.
- El Rassi G, Takemitsu M, Woratanarat P, et al. Lumbar spondylolysis in pediatric and adolescent soccer players. Am J Sport Med. 2005;33(11):1688–93.

7 Spondylolysis

- 36. d'Hemecourt PA, Zurakowski D, Kriemler S, Micheli LJ. Spondylolysis: returning the athlete to sports participation with brace treatment. Orthopedics. 2002;25:653–7.
- 37. Sairyo K, Sakai T, Yasui N, et al. Conservative treatment for pediatric lumbar spondylolysis to achieve bone healing using a hard brace: what type and how long?: clinical article. J Neurosurg Spine. 2012;16(6):610–4.
- Sairyo K, Sakai T, Yasui N. Conservative treatment of lumbar spondylolysis in childhood and adolescence: the radiological signs which predict healing. J Bone Joint Surg Br. 2009;91:206.
- 39. Stasinopoulos D. Treatment of spondylolysis with external electrical stimulation in young athletes: a critical literature review. Br J Sports Med. 2004;38(3):352–4.
- 40. Radcliffe K, Kalantar S, Reitman C. Surgical management of spondylolysis and spondylolisthesis in athletes: indications and return to play. Curr Sport Med Rep. 2009;8(1):35–40.

Chapter 8 Hip Injuries in the Young Female Athlete

Sasha Carsen and Yi-Meng Yen

Introduction

Growing sports participation among children and adolescents has been met with improvement in awareness, understanding, diagnosis, and management of hip injury for the young female athlete. Advances in imaging technology have also increased understanding of hip and pelvis pathology [1–4].

Injuries around the hip can be the result of a single traumatic event or repetitive microtrauma. The majority of injuries around the hip in children are soft tissue, apophyseal, or bony injuries that require supportive management with only a small minority of injuries ever requiring surgical intervention. Important considerations in the young female athlete include their skeletal maturity, status of their growth plates, physiological maturity, and underlying bone health. This chapter focuses on common injuries that occur to the hip joint and to surrounding structures in the pediatric, adolescent, and young adult female athlete.

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Definitions

Athletic pubalgia: a non-specific term that refers to pain in the area of the groin.

Coxa saltans: also known as snapping hip, a condition that refers to a snap or pop of the hip as it moves through particular positions. It is most often due to a tendon or other soft tissue snapping over a bony area.

Labral tear: a tear of the fibrocartilagenous ring that is located along the perimeter of the acetabulum known as the labrum.

Femoroacetabular impingement (FAI): Abnormal contact or rubbing of the hip joint due to the shape of the proximal femur, the acetabulum, or both, which can cause pain and damage to the joint.

Slipped capital femoral epiphysis (SCFE): A fracture through the growth plate of the proximal femur.

Physical Examination and Diagnostic Evaluation

As with all injuries, it is important to understand the history and determine whether symptoms followed acute trauma or developed over time. Awareness of the mechanism of injury and the energy involved allows for better understanding of the subsequent changes to the hip joint structures. Relevant elements of the history unique to the female athlete's hips include a menstrual history, known hip disease or pathology (e.g. dysplasia or impingement), and past history of stress fracture or other injury. Family history can be important, in particular if there is a family history of hip dysplasia or early-onset hip disease. Location and character of pain should be identified, though at times they can be difficult to interpret because of the variable radiation and referral patterns of pain in and around the hip. The classic description of intra-articular hip pain is the "C sign" where the patient uses her hand in the shape of the letter C to indicate that the area of pain is deep in the hip, anterior and posterior to the greater trochanter [5] (Fig. 8.1). It is important to remember that pathology of the hip can sometimes be expressed as symptoms of the knee or lower back. Knee pain without findings or pathology in the knee should always prompt evaluation of the hip.

A complete examination of the hip includes evaluation of the pelvis, as well as joints above and below the hip, specifically the lumbar spine and knee. Observation of the athlete's gait can provide insight, as a Trendelenburg gait (weak abductor gait) may point to a more chronic weakness or inhibition of the abductors, as opposed to an acutely antalgic or pain-avoidance gait. Trendelenburg sign, a single leg stance with a sagging pelvis to the contralateral side, may point to a more chronic

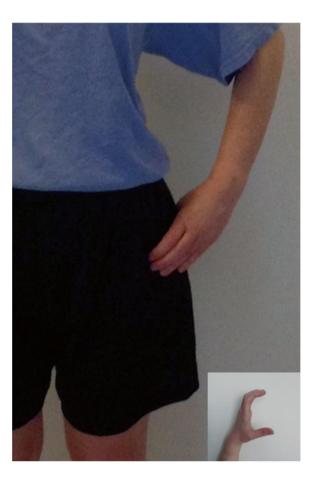


Fig. 8.1 The C sign is commonly used by patients to indicate that pain is felt deep in the hip

weakness or inhibition of the abductors (Fig. 8.2). A detailed neurovascular examination of the lower extremities should be included, and testing for general ligamentous laxity may also provide helpful information, especially in the young female athlete. Please see Chapter 6 for more information on ligamentous laxity.

Asking the patient to identify the area of pain or recreate the painful movement can provide useful information as part of the physical examination. Tests for the hip include checking range of motion and evaluating for tightness or contractures in muscles crossing the joint. The log roll test of the lower extremity refers to gently rolling the limb such that rotation occurs through the hip, and is relatively specific for intra-articular pathology [5, 6]. The impingement or FADIR (Flexion, Adduction, Internal Rotation) test is quite sensitive for hip injuries and disorders, but not specific [5, 6]. The FABER (Flexion, Abduction, External Rotation) test



Fig. 8.2 Trendelenburg Sign: pelvis sagging on contralateral side

can be used to stress the hip, the sacroiliac (SI) joint, and the hip adductors (Fig. 8.3).

Standard radiographs are typically the first line of imaging, starting with an AP radiograph of the pelvis, which provides full visualization of both hip joints [7]. A lateral radiograph of the hip in question is required in order to obtain orthogonal visualization. In the setting of acute injury, a frog-leg lateral or cross-table lateral is most appropriate. If assessing possible impingement morphology, a Dunn lateral view is best, while possible dysplasia may require a false-profile view. Discussion with a radiologist can help to determine the most appropriate views (Figs. 8.4, 8.5, and 8.6).



Fig. 8.3 The FADIR or impingement test is sensitive, but not specific, for hip injuries

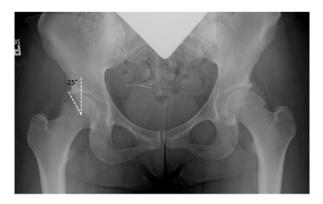


Fig. 8.4 The AP, Dunn lateral, and false-profile are common radiographic views used to evaluate the hip joint. The AP radiograph demonstrates a center edge angle of 25° on the right side, measured by taking the angle between a line drawn from the center of the femoral head to the lateral edge of the acetabulum and a perpendicular line from the center



Fig. 8.5 The Dunn lateral radiograph shows a large cam deformity on the right side (*circled*) as well as on the left, at the femoral head-neck junctions



Fig. 8.6 The false profile view of the hip

Muscle Injury

Muscle strains and contusions are common in the young athlete [8]. They can result from trauma or be associated with previous injury, fatigue, weather conditions, and uneven surfaces, among other factors [9]. Diagnosis is largely via history and physical examination. Muscle strains occur most commonly around the hip when muscles are fired while being lengthened, known as eccentric contraction. Muscle strain usually presents with sudden onset, localized pain and tenderness of the affected muscle, and varying degrees of ecchymosis. The athlete is often unable to continue the activity immediately after the injury. A contusion, on the other hand, is due to direct contact, and is more likely to allow a return to play. Treatment for muscle injury involves early compression and icing to control bleeding and swelling, as well as use of non-steroidal anti-inflammatories (NSAIDs). Immobilization and partial weight-bearing can be used initially to help provide comfort and protection, followed by early range of motion, gentle stretching, and progression to strengthening when muscle pain is absent [10]. Functional activities can start once strength has returned. Surgical intervention is rarely indicated.

Hip Bursitis

Hip bursitis refers to the inflammation of one of the major bursa, typically either the iliopsoas bursa or the greater trochanteric bursa. Causes of bursitis include chronic microtrauma, arthritis, regional muscle dysfunction, overuse, and acute injury [11].

The iliopsoas bursa is the largest synovial bursa in the body, located between the iliopsoas tendon and the lesser trochanter, extending upward into the iliac fossa beneath the iliacus muscle. Iliopsoas bursitis causes pain in the anterior groin, tenderness to palpation over the lesser trochanter and pain with resisted hip flexion [12].

The greater trochanteric bursa is located on the lateral side of the greater trochanter and cushions the gluteal tendons, iliotibial band, and tensor fascia latae. Symptoms of trochanteric bursitis include pain on the lateral aspect of the hip and thigh, pain when moving from sitting to standing, and pain while walking up stairs. Pain may be elicited on examination by having the patient lie on her side and reproduce the motion of riding a bike.

Bursitis can cause erythema, swelling, and warmth over the involved area. Diagnosis is usually clinical and imaging is often unnecessary. Plain radiographs can be used to rule out other pathology, and in chronic cases may show calcifications. Ultrasound or magnetic resonance imaging (MRI) can be used to localize areas of inflammation if necessary. Treatment of isolated bursitis involves rest, ice, NSAIDs, gentle stretching, and physical therapy. Corticosteroid injections may help to decrease inflammation and pain. In recalcitrant cases, surgical bursectomy or tendon release may be helpful [13–16].

Athletic Pubalgia

Athletic pubalgia is a general term for pain around the groin, which can refer to a variety of conditions including osteitis pubis, adductor dysfunction, sports hernia, or other pelvic/abdominal muscular injury.

Adductor dysfunction causes tenderness localized to the adductor longus insertion, and on examination there is pain with adduction against resistance and with stretch in abduction. This is usually treated with non-operative management including rest and physical therapy. Occasionally, the use of injected corticosteroids can give relief [17], while adductor tenotomy (tendon release) has been shown to provide long-term relief in recalcitrant cases [18, 19]. Osteitis pubis is inflammation at the pubic symphysis, which causes localized tenderness to palpation. Chronic osteitis pubis can cause resorption at the pubic symphysis visible even on plain radiographs [20, 21] and a "secondary cleft sign" on MRI, which refers to fluid signal extending inferolaterally from the pubic symphysis, indicating inflammation and possible chronic maceration at the joint [22]. In acute cases, edema can be seen around the pubis on MRI [23]. Osteitis pubis is generally treated with rest and physical therapy [24], and with injections [25] and surgical intervention [26] in recalcitrant cases.

While the term athletic pubalgia can encompass a variety of conditions, the most common is the "sports hernia" [27]. It is a syndrome that results from weakness of the posterior wall of the inguinal canal that results in both nerve irritation and disruption and instability of the muscular attachments [28, 29]. Athletic pubalgia can be thought of as an overuse injury [30] in which the muscular attachments of the pelvis are unbalanced and lead to a loss of postural control and increases in the shear forces across the pelvis. Most patients report insidious unilateral, dull, achy pain in their groin that is significantly exacerbated by activities such as running, cutting, or twisting. Coughing, sneezing, and other Valsalva-type maneuvers usually worsen the pain. Physical examination can show tenderness over the conjoined tendon, adductor origin, distal rectus insertion, and inguinal canal. It is important to distinguish athletic pubalgia from an inguinal or femoral hernia. Newer MRI imaging can be useful, showing rectus abdominis tendon injury and subtle abnormalities of the myofascial layers of the abdominal wall [31]. Prevention with core and hip stability and flexibility is paramount in decreasing the incidence of athletic pubalgia [32]. Non-operative measures include rest, anti-inflammatories, strengthening of core musculature, and postural control [33]. Referral to a general surgeon should be considered if non-operative treatment has failed after 3-6 months and other diagnoses have been excluded.

Snapping Hip (Coxa Saltans)

A "snapping hip" refers to the finding of a "snap" or "pop" of the hip, often audible or even visible, as it is brought though a specific range of motion. The most common cause is a tendon or soft tissue snapping over a bony surface. Snapping hip, or coxa saltans, is typically atraumatic in origin, and can be classified as being external, internal, or intra-articular. Snapping hip can be painless, mildly uncomfortable, or very limiting. It is often exacerbated by activities such as dancing or running up or downhill.

History and physical examination are typically diagnostic for the location and cause of the snapping hip. Coxa saltans externa is most common, and involves the posterior border of the iliotibial band or the anterior border of the gluteus maximus tendon snapping over the greater trochanter when the hip is flexed from an extended position or with internal and external rotation while the hip is extended. The snapping is often visible as the iliotibial band snaps over the greater trochanter [34]. Patients with external snapping hip can often reproduce the symptoms voluntarily.

More commonly painful and audible is coxa saltans interna, which is caused by the iliopsoas tendon snapping over the iliopectineal eminence near the anterior acetabulum or over the femoral head [35]. Internal snapping hip occurs when moving the hip from a flexed and abducted position to an extended and adducted position. This can oftentimes be seen by dynamic ultrasonography. Finally, intra-articular snapping hip, which is less predictable, can be secondary to a synovial plica or intraarticular pathology, such as a loose body or labral/chondral flap [36].

Initial management includes physical therapy for stretching of the iliotibial band or iliopsoas. Anti-inflammatory medications, rest, and bursal injections [13] can also be effective. If conservative management does not relieve the symptoms, surgical intervention may be considered. Open surgical approaches involve lengthening or release [37, 38] and have also been described in adolescents [35]. Retrospective study of open surgical correction of coxa saltans interna has shown high patient satisfaction rates, despite a relatively high complication rate [39]. Arthroscopic treatment has shown some good outcomes in both interna and externa [14, 40]. Intra-articular snapping can be caused by a loose body, chondral flap, labral tear or synovial plica; these conditions require further diagnostic work-up such as MRI and are far more likely to require surgical intervention [41].

It should be emphasized that physical therapy, rehabilitation, and non-invasive techniques are successful in the treatment of snapping hip for the majority of patients. Dancers in particular should be treated non-operatively with physical therapy. Laible et al., in the largest series of dancers treated for snapping hip, found that non-operative treatment was ultimately successful in all dancers [42].

Avulsion Injury

Avulsion injuries around the pelvis are quite common in skeletally immature athletes, particularly in those approaching skeletal maturity [43]. This is due to the inherent weakness of the open apophysis relative to the stronger tendon attachment. Avulsions are most often secondary to sudden forceful muscle contraction as can be seen with kicking, jumping, and rapid acceleration and deceleration sports, such as soccer, gymnastics, sprinting, rugby, and hockey.

Common sites of avulsion injury around the hip and pelvis include the ischial tuberosity (hamstring attachment), the anteroinferior iliac spine (rectus femoris attachment), anterior superior iliac spine (sartorius attachment), iliac crest (abdominal musculature attachment), lesser trochanter (iliopsoas attachment), or greater trochanter (abductor muscle attachment) [44].

Clinical presentation typically follows a traumatic incident with an acute onset of localized pain and the description of a "pop." Palpation and passive stretching of the muscle is typically quite painful, and patients will assume a position that places the least amount of tension on the involved muscle. Radiographs of the region assist with diagnosis. They are used to gauge the size of the fragment and the degree of displacement, and they serve as a baseline to monitor for future healing or displacement.

Principles of treatment include relative rest, icing, and possibly reduced weight-bearing. The vast majority (>90 %) can be successfully treated non-operatively, with mean times of healing and return to sports under 3 months [45]. Rehabilitation is progressive, with return to sport only once full pain-free range of motion is restored. Fragment displacement greater than 2 cm can be a relative indication for surgery. Also, avulsions of the ischial tuberosity are at a greater risk of requiring surgical intervention [45]. Due to the risks of proximal hamstring syndrome and chronic irritation of the nearby sciatic nerve, there is an ongoing debate regarding both the timing and indicators for intervention in ischial tuberosity avulsions [46–48].

Iliac Crest Apophysitis

Apophysitis of the iliac crest is an inflammatory overuse injury of the adolescent athlete with an open iliac crest apophysis. It involves pain along the iliac crest, which can be very tender to palpation. Generally there does not tend to be a specific injury, but rather an insidious onset and development of pain. It can often be linked to a recent change in training technique or an increase in intensity. Radiographs are usually normal or show only mild widening of the iliac apophysis. Treatment begins with relative rest and activity modification, cold therapy, anti-inflammatory medication, and physical therapy for muscle-stretching exercises. Return to full activity is dependent on symptoms and is widely variable, but can range from 6 weeks to several months. Recalcitrant aphophysitis has shown some potential benefit from bone stimulator treatment [49], though its use is still investigational.

Stress Fractures and Injuries

The young female athlete is at particular risk for stress injury, especially during adolescence. In addition to the physiological changes that occur, there are increasing levels of training and competition during roughly the same time period. When treating the young female athlete, one must always have a low threshold for suspecting a stress injury or fracture. In the hip and pelvis, in particular, such a diagnosis is important in order to help prevent or mitigate the effects of a potentially significant injury. Stress fractures of the femoral neck are seen most commonly in distance runners and young people enlisted in the military [50, 51], but the incidence of femoral neck stress fractures specifically in young female athletes has also been reported to be significant [52].

Stress fractures occur due to abnormal forces on normal bone (fatigue fractures) or normal forces on abnormal bone (insufficiency fractures) [53]. Overuse and stress injuries are a result of repetitive microtrauma damage that exceeds the body's ability to repair and remodel. The most common sites of stress fracture in the hip are in the

femur, pubic rami, iliac crest, and sacroiliac joints [54]. Stress fractures of the femoral neck are particularly important because they often go unrecognized and untreated and can lead to the potentially devastating consequence of an acute femoral neck fracture [55, 56]. Female athletes are at a particular risk of developing stress fractures in the setting of the Female Athlete Triad (Triad), the interrelationship of decreased energy availability with or without an eating disorder, menstrual irregularity, and low bone mineral density [56–58]. The Triad should always be considered when evaluating a young female athlete and her risk for stress fracture. See Chapter 5 for additional information on the Triad. Additional risk factors for stress fracture include chronic glucocorticoid use, smoking, endocrine disorders, malabsorption syndromes, and calcium deficiencies [59–61].

Stress fractures of the hip often present with only subtle symptoms, such as vague groin discomfort worsened with activity, making the diagnosis difficult. Patients often provide a history of a recent increase in training frequency or intensity. As the fracture worsens, pain may occur earlier or even at rest. On physical examination, pain may be reproduced at the extremes of range of motion, in particular with impingement testing or simple internal rotation [62]. A positive Trendelenburg test, inability to straight leg raise against resistance, or inability to hop on the affected side should raise suspicion of a possible stress fracture [63]. Plain radiographs are often negative in the acute setting, and advanced imaging, such as MRI or bone scan, may be required.

Management of a hip stress fracture is based upon the location, chronicity and causative factors, but they require urgent orthopedic or sports medicine consultation and immediate non-weight-bearing status on the affected side. Femoral neck stress fractures in the young female are most likely to occur on the compression (inferior and medial) side of the femoral neck, and are most often treatable non-operatively [64, 65]. Non-weight-bearing and relative rest are important until radiographic and clinical union is achieved. Assessment and treatment of any underlying predisposing disorder is important to allow both healing as well as to prevent recurrence.

Acute Fractures and Dislocations

Fractures around the hip and pelvis are relatively rare in the adolescent and pediatric population. These fractures usually result from high-energy trauma or fall from a height, rather than from participation in sports. However, due to increased joint laxity, traumatic hip dislocation can occur secondary to a low-energy injury in children, and can result from sports injuries or falls from relatively low heights [66, 67].

Most hip dislocations occur posteriorly [67, 68], and the hip is usually held in flexion, adduction, and internal rotation. Neurovascular status must be documented thoroughly, and mechanism of injury and possible blunt trauma to the ipsilateral knee should be noted. Radiographs of the pelvis and/or hips are generally diagnostic; however, advanced imaging, such as computed tomography (CT) scan or MRI, can be helpful when radiographs are negative but fracture or spontaneously reduced dislocation is suspected. CT and MRI can also be used to detect bony fragments within the joint and concomitant injuries.

Fracture of the pelvis or femur and dislocation of the hip require immediate orthopedic consultation to evaluate the need for urgent surgical intervention. For example, traumatic hip dislocation requires an emergent closed reduction within the first 6 h after injury to minimize the risk of avascular necrosis [66, 68]. Closed reduction should be performed in a controlled environment by experienced clinicians. Open reduction is occasionally required if the hip is irreducible or if soft tissue or bony fragments are interposed between the head and acetabulum.

The major complication after a dislocated hip is avascular necrosis of the femoral head [66, 67, 69, 70]. Other complications include sciatic nerve injury, post-traumatic osteoarthritis, coxa magna, heterotopic ossification, and recurrent dislocation.

Pathological and Neoplastic Conditions

Although rare, pathological conditions in and around the hip can be a cause of hip pain. Adolescents and young adults are at a relatively increased risk of such lesions as they happen with greater frequency in the periods of maximal growth. The vast majority of these lesions are benign, such as non-ossifying fibromas and bone islands; many are found incidentally and do not require any management other than reassurance. Atypical pain or symptoms, especially night pain or constitutional symptoms, such as fevers, warrant further investigations, starting with plain radiographs. Simple (unicameral) bone cysts, chondroblastomas, and osteoid osteomas are examples of benign lesions that require assessment by an orthopedic surgeon. Malignant lesions include Ewing's sarcoma, osteosarcoma, and metatstatic disease, all of which require urgent referral to an orthopedic oncology team [71]. If the history or the physical examination is concerning, advanced imaging, such as MRI, CT scan, or bone scan, may be indicated.

Developmental Dysplasia of the Hip

Dysplasia of the hip exists along a spectrum, with the dislocated hip existing at the extreme. More common, however, is the mildly dysplastic hip that is minimally symptomatic through childhood and is diagnosed during adolescence or young adulthood as a relative under-coverage of the femoral head and a potential source of pain. Relative under-coverage can lead to relative instability of the hip, labral pathology and tearing, and even early osteoarthritic changes due to abnormal hip loading and biomechanics. Young active females are the prototypical group to develop symptoms, and there is often a family history of hip dysplasia or early arthritis of the hip [72]. In addition to physical examination, preliminary assessment requires radiographs of the pelvis and hips. On the PA pelvis radiograph,

a centre-edge angle (measuring from the center of the femoral head to the edge of the weight-bearing acetabulum) of less than 25°, or a roof-arc angle of greater than 10°, are indicators of possible under-coverage, as is an anterior center-edge angle of less than 20° on a false-profile lateral view of the hip [7]. Suspected dysplasia or under-coverage should be referred to an orthopedic surgeon, preferably one with experience in hip preservation. There may be a role for hip preserving techniques such as re-directional osteotomies.

Labral Tears

The acetabular labrum refers to the fibrocartilagenous ring that runs along the outside edge of the acetabulum. It functions to deepen the acetabulum and provide enhanced joint stability, as well as create a type of fluid seal of the hip [73-75]. The labrum is subject to significant stress and can be torn, especially in the setting of anatomic morphologies like femoroacetabular impingement (FAI) or dysplasia [76]. Labral tears can lead to pain, clicking, and/or locking of the hip, and they are most frequently seen in the antero-superior labrum [77]. Labral tears are identified most commonly and easily through the use of MR-arthrograms, but they may also be detected without contrast in the setting of an experienced musculoskeletal radiologist and a high-powered MRI [78, 79]. Isolated symptomatic labral tears are far less common than tears that accompany an underlying anatomic predisposition, such as FAI or dysplasia. Therefore, radiographs are important in assessing the anatomy of the hip. Arthroscopic management and repair of symptomatic labral tears is now generally the standard of care for the young athlete, with good reported results [80-83]. In some cases with significant underlying anatomic abnormality, there may be a role for open surgery.

Femoroacetabular Impingement (FAI)

FAI refers to dynamic impingement of the hip joint secondary to osseous morphology of the proximal femur, the acetabulum, or both. Impingement exists near the end of the range of motion of the hip, such that the bone of the femoral head–neck junction abuts the anterior rim of the acetabulum. This impingement can cause damage of the hip joint and pain with repetitive motion. FAI has not only been implicated in the development of osteoarthritis, but it is also recognized as a significant cause of hip pain in the adolescent population [84]. It is the most commonly found underlying pathoetiology for labral tears and early cartilage damage [85, 86]. FAI morphology includes relative over-coverage from the acetabulum (pincer impingement), a "bump" on the femoral head–neck side (cam impingement), or, as is seen most often, both (mixed impingement) (Fig. 8.7) [87].

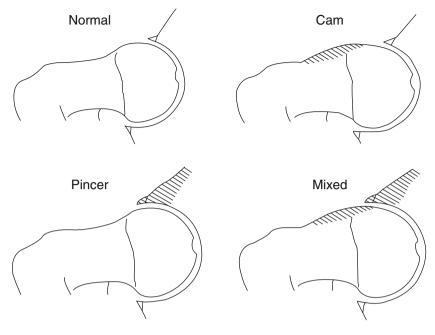


Fig. 8.7 Morphology of the hip

Patients often present with insidious onset of groin pain that worsens with activities. A concomitant labral tear is also common. FAI is a dynamic finding, and decreased hip flexion, limited internal rotation, and a positive impingement test (flexion, adduction, and internal rotation) are typical. Radiographic imaging should include an AP of the pelvis and orthogonal views of the hip, including Dunn lateral views of the hips if cam impingement is suspected [7]. MRI and MR-arthrogram are important for evaluation of the intra-articular cartilage and labrum [88]. Treatment of FAI depends on severity and associated pathology. Non-operative treatment includes physical therapy with focus on hip muscle flexibility and strength, pelvic tilt, posture, and core strengthening. Surgical management may be necessary for recalcitrant or more severe cases in order to address both the soft tissue pathology, such as labral tears, as well as the underlying osseous morphology [85]; results in the adolescent population have been positive and encouraging [89].

SCFE

Slipped capital femoral epiphysis (SCFE) is a shear fracture through the physis of the proximal femur. The femoral head is displaced posteriorly, and the femoral neck and remainder of the lower extremity move anteriorly and externally rotate [90]. Though SCFE is a fracture through the growth plate, it can be acute or chronic, and

patients most commonly present without any history of trauma [91]. This condition is often described in overweight, African American adolescent males; however, it can also be seen infrequently in young female athletes [4]. Patients may present with pain in the groin with activity or weight-bearing, but radiating pain felt more in the knee or lower back is also common. SCFE is clinically classified as either stable or unstable, based on the patient's ability to bear weight on the affected limb [90]. Diagnosis requires imaging, followed by urgent orthopedic consultation for possible surgical intervention. Delayed diagnosis is linked to poorer outcomes [92]. In the setting of high clinical suspicion but negative X-rays, an MRI can be used to rule out SCFE with higher sensitivity [90]. Endocrine workup may also be helpful to rule out possible underlying conditions.

Summary

Hip injuries in the young female athlete are becoming better recognized using modern imaging modalities such as MRI and targeted ultrasound to supplement a thorough history and physical examination. It is important to correlate the athlete's symptoms and history to her physical examination findings and imaging results. Intra-articular analgesic and corticosteroid injections can provide added diagnostic benefit in questionable cases where the articular nature of the injury is unclear. Most pathology of the pelvis and hip in the young female athlete can be treated non-operatively, with activity modification, physical therapy, medications, and injections. However, a high level of suspicion must be maintained for abnormal bony morphology and the underlying presence of the Triad. Structured and progressive rehabilitation and resolution of pain through full range of motion and weightbearing is important prior to return to sport.

References

- 1. Heyworth BE, Voos JE, Metzl JD. Hip injuries in the adolescent athlete. Pediatr Ann. 2007;36(11):713–8.
- Jacoby L, Yen YM, Kocher MS. Hip problems and arthroscopy: adolescent hip as it relates to sports. Clin Sports Med. 2011;30(2):435–51.
- 3. Kocher MS, Tucker R. Pediatric athlete hip disorders. Clin Sports Med. 2006;25(2):241–53. viii.
- 4. Kovacevic D, Mariscalco M, Goodwin RC. Injuries about the hip in the adolescent athlete. Sports Med Arthrosc Rev. 2011;19(1):64–74.
- Byrd JWT. Physical examination. In: Byrd JWT, editor. Operative hip arthroscopy. 2nd ed. New York, NY: Springer; 2005. p. 36–50.
- Byrd JW. Evaluation of the hip: history and physical examination. N Am J Sports Phys Ther. 2007;2(4):231–40.
- Clohisy JC, Carlisle JC, Beaule PE, Kim YJ, Trousdale RT, Sierra RJ, Leunig M, Schoenecker PL, Millis MB. A systematic approach to the plain radiographic evaluation of the young adult hip. J Bone Joint Surg Am. 2008;90 Suppl 4:47–66.

- Frank JB, Jarit GJ, Bravman JT, Rosen JE. Lower extremity injuries in the skeletally immature athlete. J Am Acad Orthop Surg. 2007;15(6):356–66.
- 9. Best TM. Muscle-tendon injuries in young athletes. Clin Sports Med. 1995;14(3):669-86.
- Kary JM. Diagnosis and management of quadriceps strains and contusions. Curr Rev Musculoskelet Med. 2010;3(1-4):26–31.
- Shbeeb MI, Matteson EL. Trochanteric bursitis (greater trochanter pain syndrome). Mayo Clin Proc. 1996;71(6):565–9.
- 12. McGrory B. Stinchfield resisted hip flexion test. Hosp Phys. 1999;35:41-2.
- Blankenbaker DG, De Smet AA, Keene JS. Sonography of the iliopsoas tendon and injection of the iliopsoas bursa for diagnosis and management of the painful snapping hip. Skeletal Radiol. 2006;35(8):565–71.
- Ilizaliturri Jr VM, Camacho-Galindo J. Endoscopic treatment of snapping hips, iliotibial band, and iliopsoas tendon. Sports Med Arthrosc. 2010;18(2):120–7.
- Slawski DP, Howard RF. Surgical management of refractory trochanteric bursitis. Am J Sports Med. 1997;25(1):86–9.
- Zoltan DJ, Clancy Jr WG, Keene JS. A new operative approach to snapping hip and refractory trochanteric bursitis in athletes. Am J Sports Med. 1986;14(3):201–4.
- 17. Schilders E, Bismil Q, Robinson P, O'Connor PJ, Gibbon WW, Talbot JC. Adductor-related groin pain in competitive athletes. Role of adductor enthesis, magnetic resonance imaging, and entheseal public cleft injections. J Bone Joint Surg Am. 2007;89(10):2173–8.
- Akermark C, Johansson C. Tenotomy of the adductor longus tendon in the treatment of chronic groin pain in athletes. Am J Sports Med. 1992;20(6):640–3.
- Maffulli N, Loppini M, Longo UG, Denaro V. Bilateral mini-invasive adductor tenotomy for the management of chronic unilateral adductor longus tendinopathy in athletes. Am J Sports Med. 2012;40(8):1880–6.
- McCarthy B, Dorfman HD. Pubic osteolysis. A benign lesion of the pelvis closely mimicking a malignant neoplasm. Clin Orthop Relat Res 1990; (251): 300–7.
- 21. McGuigan LE, Edmonds JP, Painter DM. Pubic osteolysis. J Bone Joint Surg Am. 1984;66(1):127–9.
- Brennan D, O'Connell MJ, Ryan M, Cunningham P, Taylor D, Cronin C, O'Neill P, Eustace S. Secondary cleft sign as a marker of injury in athletes with groin pain: MR image appearance and interpretation. Radiology. 2005;235(1):162–7.
- Morelli V, Espinoza L. Groin injuries and groin pain in athletes: part 2. Prim Care. 2005;32(1):185–200.
- Hiti CJ, Stevens KJ, Jamati MK, Garza D, Matheson GO. Athletic osteitis pubis. Sports Med. 2011;41(5):361–76.
- O'Connell MJ, Powell T, McCaffrey NM, O'Connell D, Eustace SJ. Symphyseal cleft injection in the diagnosis and treatment of osteitis publis in athletes. AJR Am J Roentgenol. 2002;179(4):955–9.
- 26. Williams PR, Thomas DP, Downes EM. Osteitis pubis and instability of the pubic symphysis. When nonoperative measures fail. Am J Sports Med. 2000;28(3):350–5.
- Minnich JM, Hanks JB, Muschaweck U, Brunt LM, Diduch DR. Sports hernia: diagnosis and treatment highlighting a minimal repair surgical technique. Am J Sports Med. 2011;39(6):1341–9.
- Ahumada LA, Ashruf S, Espinosa-de-los-Monteros A, Long JN, de la Torre JI, Garth WP, Vasconez LO. Athletic pubalgia: definition and surgical treatment. Ann Plast Surg. 2005;55(4):393–6.
- 29. Litwin DE, Sneider EB, McEnaney PM, Busconi BD. Athletic pubalgia (sports hernia). Clin Sports Med. 2011;30(2):417–34.
- Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29(4):521–33.
- Zoga AC, Kavanagh EC, Omar IM, Morrison WB, Koulouris G, Lopez H, Chaabra A, Domesek J, Meyers WC. Athletic pubalgia and the "sports hernia": MR imaging findings. Radiology. 2008;247(3):797–807.

- 32. Meyers WC, Yoo E, Devon ON, Jain N, Horner MA, Lauencin C, Zoga AC. Understanding "Sports Hernia" (Athletic Pubalgia): the anatomic and pathophysiologic basis for abdominal and groin pain in athletes. Oper Tech Sports Med. 2007;15(4):165–77.
- 33. Diesen DL, Pappas TN. Sports hernias. Adv Surg. 2007;41:177-87.
- 34. Schaberg JE, Harper MC, Allen WC. The snapping hip syndrome. Am J Sports Med. 1984;12(5):361–5.
- Dobbs MB, Gordon JE, Luhmann SJ, Szymanski DA, Schoenecker PL. Surgical correction of the snapping iliopsoas tendon in adolescents. J Bone Joint Surg Am. 2002;84-A(3):420–4.
- Allen WC, Cope R. Coxa saltans: the snapping hip revisited. J Am Acad Orthop Surg. 1995;3(5):303–8.
- Taylor GR, Clarke NM. Surgical release of the 'snapping iliopsoas tendon'. J Bone Joint Surg Br. 1995;77(6):881–3.
- White RA, Hughes MS, Burd T, Hamann J, Allen WC. A new operative approach in the correction of external coxa saltans: the snapping hip. Am J Sports Med. 2004;32(6):1504–8.
- Hoskins JS, Burd TA, Allen WC. Surgical correction of internal coxa saltans: a 20-year consecutive study. Am J Sports Med. 2004;32(4):998–1001.
- Voos JE, Rudzki JR, Shindle MK, Martin H, Kelly BT. Arthroscopic anatomy and surgical techniques for peritrochanteric space disorders in the hip. Arthroscopy. 2007;23(11):1246. e1–5.
- Atlihan D, Jones DC, Guanche CA. Arthroscopic treatment of a symptomatic hip plica. Clin Orthop Relat Res 2003; (411): 174–7.
- Laible C, Swanson D, Garofolo G, Rose DJ. Iliopsoas syndrome in dancers. Orthop J Sports Med. 2013;1(3):1–6.
- 43. Howard FM, Piha RJ. Fractures of the apophyses in adolescent athletes. JAMA. 1965;192:842–4.
- 44. Rossi F, Dragoni S. Acute avulsion fractures of the pelvis in adolescent competitive athletes: prevalence, location and sports distribution of 203 cases collected. Skeletal Radiol. 2001;30(3):127–31.
- 45. Heyworth BE, Bonner B, Yen YM, Millis MB, Kocher MS, Micheli LJ. Results of non-operative and operative management of apophyseal avulsion fractures of the hip and pelvis in adolescent athletes. Presentation: Pediatric Orthopaedic Society of North America, Jan 5, 2014, Los Angeles, CA.
- Cohen S, Bradley J. Acute proximal hamstring rupture. J Am Acad Orthop Surg. 2007;15(6):350–5.
- Gidwani S, Jagiello J, Bircher M. Avulsion fracture of the ischial tuberosity in adolescents-DOUBLEHYPHENan easily missed diagnosis. BMJ. 2004;329(7457):99–100.
- Wootton JR, Cross MJ, Holt KW. Avulsion of the ischial apophysis. The case for open reduction and internal fixation. J Bone Joint Surg Br. 1990;72(4):625–7.
- 49. Kivel CG, d'Hemecourt CA, Micheli LJ. Treatment of iliac crest apophysitis in the young athlete with bone stimulation: report of 2 cases. Clin J Sport Med. 2011;21:144–7.
- Bennell KL, Malcolm SA, Thomas SA, Wark JD, Brukner PD. The incidence and distribution of stress fractures in competitive track and field athletes. A twelve-month prospective study. Am J Sports Med. 1996;24(2):211–7.
- 51. Fullerton Jr LR, Snowdy HA. Femoral neck stress fractures. Am J Sports Med. 1988;16(4):365–77.
- Brukner P, Bennell K. Stress fractures in female athletes. Diagnosis, management and rehabilitation. Sports Med. 1997;24(6):419–29.
- 53. Paluska SA. An overview of hip injuries in running. Sports Med. 2005;35(11):991-1014.
- Matheson GO, Clement DB, McKenzie DC, Taunton JE, Lloyd-Smith DR, MacIntyre JG. Stress fractures in athletes. A study of 320 cases. Am J Sports Med. 1987;15(1):46–58.
- 55. Johansson C, Ekenman I, Tornkvist H, Eriksson E. Stress fractures of the femoral neck in athletes. The consequence of a delay in diagnosis. Am J Sports Med. 1990;18(5):524–8.

- 56. Okamoto S, Arai Y, Hara K, Tsuzihara T, Kubo T. A displaced stress fracture of the femoral neck in an adolescent female distance runner with female athlete triad: a case report. Sports Med Arthrosc Rehabil Ther Technol. 2010;2:6.
- 57. Haddad FS, Bann S, Hill RA, Jones DH. Displaced stress fracture of the femoral neck in an active amenorrhoeic adolescent. Br J Sports Med. 1997;31(1):70–2.
- Putukian M. The female triad. Eating disorders, amenorrhea, and osteoporosis. Med Clin North Am. 1994;78(2):345–56.
- 59. Bennell KL, Malcolm SA, Wark JD, Brukner PD. Models for the pathogenesis of stress fractures in athletes. Br J Sports Med. 1996;30(3):200–4.
- Haddad FS, Mohanna PN, Goddard NJ. Bilateral femoral neck stress fractures following steroid treatment. Injury. 1997;28(9-10):671–3.
- Melhus H, Michaelsson K, Holmberg L, Wolk A, Ljunghall S. Smoking, antioxidant vitamins, and the risk of hip fracture. J Bone Miner Res. 1999;14(1):129–35.
- 62. Browning KH, Donley BG. Evaluation and management of common running injuries. Cleve Clin J Med. 2000;67(7):511–20.
- Noakes TD, Smith JA, Lindenberg G, Wills CE. Pelvic stress fractures in long distance runners. Am J Sports Med. 1985;13(2):120–3.
- 64. Maezawa K, Nozawa M, Sugimoto M, Sano M, Shitoto K, Kurosawa H. Stress fractures of the femoral neck in child with open capital femoral epiphysis. J Pediatr Orthop B. 2004;13(6):407–11.
- 65. St Pierre P, Staheli LT, Smith JB, Green NE. Femoral neck stress fractures in children and adolescents. J Pediatr Orthop. 1995;15(4):470–3.
- Mehlman CT, Hubbard GW, Crawford AH, Roy DR, Wall EJ. Traumatic hip dislocation in children. Long-term followup of 42 patients. Clin Orthop Relat Res 2000; (376): 68–79.
- 67. Vialle R, Odent T, Pannier S, Pauthier F, Laumonier F, Glorion C. Traumatic hip dislocation in childhood. J Pediatr Orthop. 2005;25(2):138–44.
- Kutty S, Thornes B, Curtin WA, Gilmore MF. Traumatic posterior dislocation of hip in children. Pediatr Emerg Care. 2001;17(1):32–5.
- 69. Barquet A, Vecsei V. Traumatic dislocation of the hip with separation of the proximal femoral epiphysis. Report of two cases and review of the literature. Arch Orthop Trauma Surg. 1984;103(3):219–23.
- 70. Offierski CM. Traumatic dislocation of the hip in children. J Bone Joint Surg Br. 1981;63-B(2):194–7.
- Ruggieri P, Angelini A, Montalti M, Pala E, Calabro T, Ussia G, Abati CN, Mercuri M. Tumours and tumour-like lesions of the hip in the paediatric age: a review of the Rizzoli experience. Hip Int. 2009;19 Suppl 6:S35–45.
- Nunley RM, Prather H, Hunt D, Schoenecker PL, Clohisy JC. Clinical presentation of symptomatic acetabular dysplasia in skeletally mature patients. J Bone Joint Surg Am. 2011;93 Suppl 2:17–21. doi:10.2106/JBJS.J.01735.
- 73. Ferguson SJ, Bryant JT, Ganz R, Ito K. The acetabular labrum seal: a poroelastic finite element model. Clin Biomech (Bristol, Avon). 2000;15(6):463–8.
- Ferguson SJ, Bryant JT, Ganz R, Ito K. The influence of the acetabular labrum on hip joint cartilage consolidation: a poroelastic finite element model. J Biomech. 2000;33(8):953–60.
- 75. Ferguson SJ, Bryant JT, Ito K. The material properties of the bovine acetabular labrum. J Orthop Res. 2001;19(5):887–96.
- Hunt D, Clohisy J, Prather H. Acetabular labral tears of the hip in women. Phys Med Rehabil Clin N Am. 2007;18(3):497–520.
- 77. Lewis CL, Sahrmann SA. Acetabular labral tears. Phys Ther. 2006;86(1):110-21.
- Burnett RS, Della Rocca GJ, Prather H, Curry M, Maloney WJ, Clohisy JC. Clinical presentation of patients with tears of the acetabular labrum. J Bone Joint Surg Am. 2006;88(7): 1448–57.
- 79. Kelly BT, Williams 3rd RJ, Philippon MJ. Hip arthroscopy: current indications, treatment options, and management issues. Am J Sports Med. 2003;31(6):1020–37.

- 80. Byrd JW, Jones KS. Hip arthroscopy in athletes: 10-year follow-up. Am J Sports Med. 2009;37(11):2140-3.
- 81. Ikeda T, Awaya G, Suzuki S, Okada Y, Tada H. Torn acetabular labrum in young patients. Arthroscopic diagnosis and management. J Bone Joint Surg Br. 1988;70(1):13–6.
- Kocher MS, Kim YJ, Millis MB, Mandiga R, Siparsky P, Micheli LJ, Kasser JR. Hip arthroscopy in children and adolescents. J Pediatr Orthop. 2005;25(5):680–6.
- Siparsky PN, Kocher MS. Current concepts in pediatric and adolescent arthroscopy. Arthroscopy. 2009;25(12):1453–69.
- Sink EL, Gralla J, Ryba A, Dayton M. Clinical presentation of femoroacetabular impingement in adolescents. J Pediatr Orthop. 2008;28(8):806–11.
- 85. Ganz R, Gill TJ, Gautier E, Ganz K, Krugel N, Berlemann U. Surgical dislocation of the adult hip a technique with full access to the femoral head and acetabulum without the risk of avascular necrosis. J Bone Joint Surg Br. 2001;83(8):1119–24.
- Ganz, R, Parvizi J, Beck M, Leunig M, Notzli H, Siebenrock KA. Femoroacetabular impingement: a cause for osteoarthritis of the hip. Clin Orthop Relat Res 2003; (417): 112–20.
- Lavigne M, Parvizi J, Beck M, Siebenrock KA, Ganz R, Leunig M. Anterior femoroacetabular impingement: Part I. Techniques of joint preserving surgery. Clin Orthop Relat Res 2004; (418): 61–6.
- Leunig M, Podeszwa D, Beck M, Werlen S, Ganz R. Magnetic resonance arthrography of labral disorders in hips with dysplasia and impingement. Clin Orthop Relat Res 2004; (418): 74–80.
- Philippon MJ, Yen YM, Briggs KK, Kuppersmith DA, Maxwell RB. Early outcomes after hip arthroscopy for femoroacetabular impingement in the athletic adolescent patient: a preliminary report. J Pediatr Orthop. 2008;28(7):705–10.
- Loder RT, Aronsson DD, Weinstein SL, Breur GJ, Ganz R, Leunig M. Slipped capital femoral epiphysis. Instr Course Lect. 2008;57:473–98.
- Peck D. Slipped capital femoral epiphysis: diagnosis and management. Am Fam Physician. 2010;82(3):258–62.
- Kocher MS, Bishop JA, Weed B, Hresko MT, Millis MB, Kim YJ, Kasser JR. Delay in diagnosis of slipped capital femoral epiphysis. Pediatrics. 2004;113(4):e322–5.

Chapter 9 ACL Injuries in the Female Athlete

Benedikt L. Proffen and Martha M. Murray

Introduction

Injury to the anterior cruciate ligament (ACL) is relatively common, particularly in young women, and it has potentially serious short- and long-term consequences. A number of risk factors, such as being female and playing specific types of sports, predispose some individuals to ACL injury. Fortunately, there are training techniques that can help reduce the risk of ACL injuries, and effective surgeries for ACL reconstruction if an ACL injury occurs. This chapter will review the risk factors for ACL injury, existing prevention strategies, the overall mechanics of ACL reconstructive surgery in its current form, and some new directions for treatment of athletes with ACL injuries.

Definitions

Anterior cruciate ligament (ACL): The anterior cruciate ligament (ACL) is a major ligament of the knee (Fig. 9.1). It stabilizes the shinbone (tibia) relative to the thighbone (femur), especially during activities that involve twisting and turning.

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Fig. 9.1 The anterior cruciate ligament (ACL) is inside of the knee joint. It courses from the front of the tibia to the back of the femur

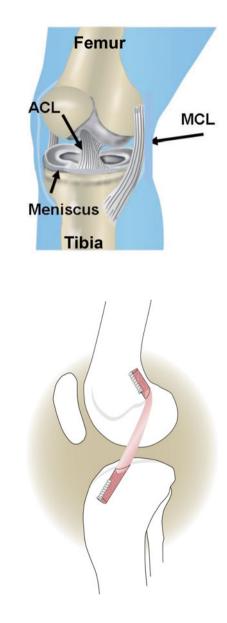


Fig. 9.2 When the ACL is torn, it is typically treated by replacing the torn tissue with a graft of tendon and fixing the tendon to the tibia and femur to secure it

ACL reconstruction: An operation involving a graft of tendon taken from elsewhere in the patient (typically the same knee or the thigh on the injured side) or from a cadaver which is placed through tunnels in the bone to replace or "reconstruct" the torn ACL (Fig. 9.2).

Allograft and autograft: An allograft is a graft that is taken from a cadaver. An autograft is tissue taken from the patient herself.

Epidemiology

The ACL is one of the most commonly injured ligaments in the knee joint with an estimated 400,000 ACL tears per year in the USA alone [1]. The incidence is particularly high in adolescents [2]. Scandinavian national registries estimate the annual incidence of primary ACL reconstruction at 76 per 100,000 girls and 47 per 100,000 boys in the age group of 10–19 years [3]. Most ACL injuries are sports related [4], and in women's sports, gymnastics, soccer, and basketball have the highest rates of ACL injuries. Overall, ACL injuries make up a higher proportion of total injuries in women's sports than in men's [5].

ACL tears most frequently occur when an athlete is turning or changing direction on a planted foot. The knee drops toward the ground, stretching the ACL, and if enough force is applied, the ACL is torn (Fig. 9.3). While most people think of this happening with high-impact trauma, such as in professional football or basketball, the fact is that 80 % of all ACL injuries are non-contact injuries; they occur when no one else is near the athlete, typically when the player is quickly changing direction, cutting, pivoting, or stopping suddenly [6].

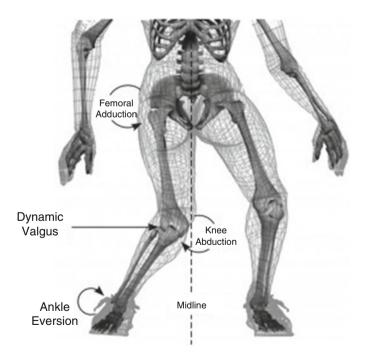


Fig. 9.3 The typical mechanism of an ACL tear. The foot is fixed (planted) on the ground, and when the player goes to change direction (pivot), the knee drops toward the ground and the ACL is torn. Exercises to teach players to not let their knee drop down toward the ground are a key component of most ACL injury prevention strategies (Used with permission from "The ACL Handbook," Eds. Murray, Vavken, and Fleming, Springer, 2013)

Girls and women are at higher risk of sustaining an ACL tear than boys and men participating in the same activities. This increased risk is estimated to be about 3:1 (Fig. 9.4) [7]. Multiple risk factors have been proposed to explain this difference. These include anatomical factors: smaller bones of the knee, which allow less room for the ACL; wider hips, resulting in greater angulation of the femur; hormonal factors with cyclic rise and fall of estrogen and progesterone [8]; and neuromuscular factors [9], including development of knee valgus (knee dropping toward midline) during impact on landing (Fig. 9.3). While anatomic factors are difficult to change, and the research around hormonal factors is inconclusive, the neuromuscular risk factors are of great interest as they are modifiable with proper training. Programs that teach female athletes to land in a safer, non-valgus position, to focus on keeping the knees in position over the toes when landing, to land softly on the toes rather than the whole foot, and to land on both feet when possible, have been very effective in reducing rates of ACL injury (See Prevention Section later in this chapter for links to specific programs).

Sports-Specific Risks of ACL Tears

Not all sports are equal in terms of ACL injury risk. Sports that involve jumping, landing, or abrupt changes of speed and/or direction, place athletes at a higher risk for ACL injury (Fig. 9.4). These include sports such as soccer, basketball, and football. For female athletes, the injury rate per 1000 athlete-exposures is highest in gymnastics, soccer, and basketball [4, 5].

Another question that surrounds ACL injuries is whether the risk of injury changes with age. Over the past few years there has been a 400 % increase in ACL injuries reported in children and adolescents, and it is currently estimated that 50 % of all patients with an ACL tear are between the ages of 15 and 25 years (Fig. 9.5) [3]. For women, the peak age of ACL injury is between 15 and 19 years of age [10, 11]. One of the most likely reasons for this increase may be secondary to young women having just completed their adolescent growth spurt at that age. When girls get taller during the growth spurt, they do not automatically develop larger muscle bulk as boys do. Therefore, young women in particular need to train in order to strengthen their leg muscles, improve the control over their knees, and protect their ACLs. Until that muscle strength comes in, young women are at increased risk for injury.

Once an athlete has an ACL tear, the risk for another ACL tear is higher than for someone who has never had a tear. The baseline risk for an initial ACL tear is 35 out of 100,000, or 0.035 %. However, once an athlete sustains an ACL tear, the risk of tearing the contralateral ACL within the next 2 years is in the range of 8-16 % [12]. This may be due, in part, to a return to high-risk activities, including participation in cutting and pivoting sports. Other risk factors, including major differences in limb strength at the time of return to sport, are also likely to play a role [13].

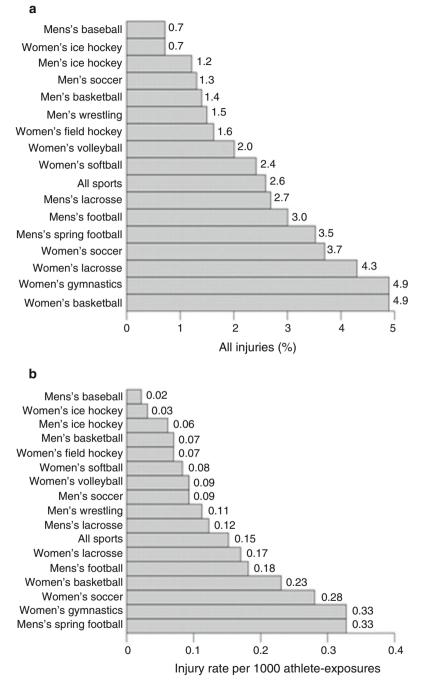


Fig. 9.4 Chart for specific sports demonstrating the relative risk for male and female athletes participating in the same sports for sustaining an ACL tear (Reprinted with permission from Renstrom, P., et al., British Journal of Sports Medicine, 2008; 42:394–412.)

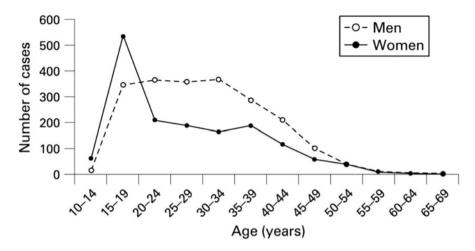


Fig. 9.5 The epidemiology of ACL injury as a function of age and gender. Note that the *curve* for women has a sharp peak between the ages of 15 and 19—the time of high school sports and just after completion of the adolescent growth spurt (Reprinted with permission from Renstrom, P., et al., British Journal of Sports Medicine, 2008; 42:394–412.) [5]

Symptoms/Diagnosis

What if injury prevention strategies do not work? What happens when an athlete tears her ACL? The common story is a non-contact injury—a plant and pivot, a layup for a basket, landing from a jump—and the athlete feels the knee shift and often feels or hears a pop. Swelling occurs within the first few hours of the injury as the ends of the ACL bleed into the joint. An athlete with a complete ACL tear typically cannot walk off the field.

As with any major knee injury, evaluation by a physician is essential for diagnosis and treatment. On exam, tests like the Lachman test or pivot shift exam evaluate for abnormal movement of the tibia relative to the femur (Fig. 9.6). If the injury is acute and the knee is very swollen or painful, these tests may have to wait for a few weeks until the swelling subsides to provide accurate information. Imaging of the knee with an MRI can often confirm the diagnosis of an ACL tear, and can also provide information about injuries to other structures, including other ligaments of the knee and the fibrocartilage menisci between the bones.

Early treatment of an ACL injury involves taking weight off the injured knee by using crutches until the knee feels well enough to walk on. If the knee feels unstable, a functional ACL brace can help stabilize the knee from the outside until it becomes more stable or until surgery is performed to reconstruct or replace the torn ACL.



Fig. 9.6 Lachman test. To evaluate for ACL tear, the femur is stabilized while anterior translation of the tibia is assessed

Treatment

For most young athletes, treatment of a torn ACL involves surgery to improve the stability of the knee. For sports that do not involve cutting and pivoting, such as running or cycling, some athletes may be able to get back to their prior level of activity without surgery. But for activities that involve turning and twisting (e.g., soccer, basketball, skiing), there is an increased risk of injury to other structures in the knee with return to sport without surgical stabilization of the knee. For this reason, most young athletes opt for surgery.

Unlike other tissues that live outside of the fluid joint environment, the ACL typically does not heal after it tears, even with surgery to repair it. For this reason, the gold standard of surgical treatment is to reconstruct, or replace, the torn ends of the ligament with a graft of tendon taken either from elsewhere in the knee (autograft) or using a donor tendon (allograft) and placing it where the ACL used to be (Fig. 9.2). The graft is anchored to the tibia and femur using one of several devices.

The two most common autograft choices are two of the hamstring tendons from the posterior aspect of the knee or the central section of the patellar tendon, with parts of the patella and tibia attached to each end (bone–patellar tendon–bone or BPTB graft). Overall, there is little difference in outcomes for these two grafts—both have a high rate of improving knee stability and allowing patients a return to sport. The only significant difference is a higher incidence of kneeling pain after a patellar tendon graft [14].

For athletes who still have open growth plates, there are special concerns. Graft tunnels should be kept central and as small as possible through the growth plate, and the surgeon needs to avoid placing bone or fixation devices across the growth plate [15]. Thus, for most patients in their early teens, hamstring grafts are selected rather than BPTB grafts. For very young patients (girls younger than 12 years and boys younger than 14 years), the surgeon may suggest a "growth-plate sparing" operation, where the growth plate, or physis, is avoided altogether [16]. These procedures may not be available everywhere and should be performed by a specialist in pediatric orthopedics.

The use of cadaver grafts, or allografts, is another option; however, recent studies have suggested that the failure rates for these grafts in a young, active population is twice as high as that for the autografts (hamstring tendon or BPTB) [14]. Therefore, these grafts are typically not used as first-line treatment for adolescent athletes, although they can be used to supplement autograft tissue if the harvested tendons are small. There are advantages and disadvantages to all graft sources. Each patient should discuss graft choices with her orthopedic surgeon to help determine which is best for her.

The surgery is typically done athroscopically, with multiple small incisions and a camera to guide the work inside the knee. The use of this minimally invasive technique results in less pain for the patient postoperatively, and a faster initial recovery. Unless there are other reasons for urgent surgery (e.g., a displaced meniscal tear or injury to other ligaments), surgery is typically delayed from 4–6 weeks after injury to allow swelling to decrease and motion to improve. This is thought to lessen the risk of postoperative stiffness in the knee and to facilitate recovery and rehabilitation after surgery [17].

Healing of the graft within the bone tunnels takes time, and thus, rehabilitation after an ACL tear is lengthy, with most patients not returning to sport until 6–9 months after reconstructive surgery [18]. During that postoperative period, strengthening of the knee is paramount to protect the graft. Focus is on hamstring, hip external rotator and core strengthening in particular, and is often done on a progressive, graded basis. The Multicenter Orthopaedic Outcomes Network has a goal-oriented rehabilitation protocol which has been used with great success [19]. The need for a lengthy rehabilitation in addition to the surgical costs and hospital stay makes the ACL tear a particularly expensive injury, with an estimated average life-time cost of \$38,000 [20].

Even after reconstruction, there remains a risk of re-rupture. The risk of re-rupturing a surgically treated ACL is approximately 6 % (2–8 %), which is about half the risk of tearing the ACL of the contralateral knee [21]. Most of these graft ruptures occur within 12 months after surgical reconstruction. In fact, during this first year, the risk of re-rupture can be as high as the risk of a contralateral injury (12 %) [21]. Another study looking at 612 patients undergoing ACL reconstruction found that factors increasing the risk of repeat ACL injury included a contact mechanism of index injury and a return to competitive sports that required side-stepping, pivoting, or jumping [22].

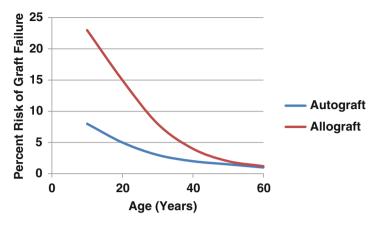


Fig. 9.7 Relative risk for graft failure as a function of age and graft choice (Used with permission from Kaeding CC, Aros B, Pedroza A, et al. Allograft versus autograft ACL reconstruction: Predictors of Failure from a MOON Prospective Longitudinal Cohort. *Sports Health.* Jan-Feb 2011;3(1):73–81)

Both patient age and graft selection have been shown to affect rates of ACL tears, with higher graft failure rates in younger patients and in those patients with an allograft reconstruction [23]. These factors have been found to be multiplicative. For example, a 14 year old with an allograft ACL reconstruction has a 22 % chance of tearing her graft, while the same patient has only a 6.6 % chance of tearing an autograft. In contrast, a 40 year old patient has only a 2.6 % chance of tearing her allograft, and only a 0.6 % chance of tearing an autograft (Fig. 9.7) [23].

ACL Injury Prevention Strategies

Understanding the risk factors associated with ACL tear allows for better identification of individuals at increased risk. Risk factors can be reliably used by doctors, parents, and coaches to identify players who might be at higher risk for an ACL tear due to their muscle development or limb alignment. The benefit of identifying highrisk individuals is that a number of exercises have been developed and assembled to create highly effective ACL injury prevention programs (Fig. 9.8) [24]. In addition, these programs can be used by an entire team, to reduce the risk among all the participating athletes. Such programs consist of simple balance board exercises and postural training, which typically require only 30 min twice a week. Recent evidence has shown that for every 40 high school students enrolled in such programs, one ACL tear can be prevented [25]. And after all, the most effective treatment for any disease is its prevention.



Fig. 9.8 The FIFA 11+ ACL injury prevention program. Additional details on the program can be found at http://f-marc.com/11plus/ (reprinted under the permission of the creative commons license http://creativecommons.org/licenses/by-nc-nd/3.0/)

Excellent descriptions of such programs, including pictures, background information, and physician information, can be found online at the Boston Children's Hospital webpage (http://childrenshospital.org/cfapps/research/data_admin/Site2226/mainpageS2226P9.html) or the PEP program website (http://smsmf.org/pep-program). The International Federation of Football Association, FIFA (Fédération Internationale de Football Association), has endorsed a warm-up and exercise program to reduce ACL injury, called FIFA 11+ (Fig. 9.8). The program can be downloaded from the FIFA website (http://f-marc.com/11plus/), and has been shown to be effective in reducing ACL tears [26, 27]. It also has been proven to work in sports other than soccer, for example in elite level male basketball players [24].

Future Directions

Improvement of injury prevention protocols and implementation of validated programs have a high likelihood of preventing many non-contact ACL injuries. In addition, advances in surgical technique—making the surgery less invasive and the rehabilitation quicker—are also in the pipeline. Techniques such as bio-enhanced primary ACL repair, where the ACL is repaired using a biologically active scaffold implanted between the two torn ends of the ligament may also play a role in the future [29]. Finally, prevention of the posttraumatic osteoarthritis, which affects the majority of patients within the first two decades after ACL tear [28], will be of paramount importance for this young patient population.

Summary

ACL injuries are common and important injuries, especially for the female athlete. It is necessary for the athlete to recognize that injury prevention programs can help minimize the chances of sustaining an ACL tear, and patients and parents should encourage coaches to institute these programs, particularly for high-risk sports like gymnastics, soccer, and basketball. ACL reconstructive surgery is very effective at restoring gross stability to the knee; however, patients remain at a higher risk for a second ACL injury than their non-injured counterparts. Improvements in surgery and prevention of posttraumatic osteoarthritis are currently in progress, and research in these areas will continue over the next decade to help improve the care of all patients with ACL injuries. Additional information on this topic can be found in "The ACL Handbook," edited by Murray, Vavken, and Fleming and available at www.springer.com [30].

References

- Junkin DM, Johnson DL, Fu FH, et al. Knee ligament injuries. In: Kibler WB, editor. Orthopaedic knowledge update 4: sports medicine. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2009. p. 135–53.
- Myer GD, Ford KR, Hewett TE. Rationale and clinical techniques for anterior cruciate ligament injury prevention among female athletes. J Athl Train. 2004;39(4):352–64.
- 3. Granan LP, Forssblad M, Lind M, Engebretsen L. The Scandinavian ACL registries 2004-2007: baseline epidemiology. Acta Orthop. 2009;80(5):563–7.
- 4. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. J Athl Train. 2007;42(2):311–9.
- Renstrom P, Ljungqvist A, Arendt E, Beynnon B, Fukubayashi T, Garrett W, et al. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. Br J Sports Med. 2008;42(6):394–412.
- 6. Yu B, Garrett WE. Mechanisms of non-contact ACL injuries. Br J Sports Med. 2007;41 Suppl 1:i47–51.
- Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury-reduction regimen. Arthroscopy. 2007;23(12):1320–5. e6.
- 8. Hewett TE, Zazulak BT, Myer GD. Effects of the menstrual cycle on anterior cruciate ligament injury risk: a systematic review. Am J Sports Med. 2007;35(4):659–68.
- 9. Hewett TE, Myer GD, Ford KR. Decrease in neuromuscular control about the knee with maturation in female athletes. J Bone Joint Surg Am. 2004;86-A(8):1601–8.
- Vavken P, Murray M. Treating anterior cruciate ligament tears in skeletally immature patients. Arthroscopy. 2011;27(5):704–16.
- 11. Vavken P, Murray MM. Translational studies in anterior cruciate ligament repair. Tissue Eng Part B Rev. 2010;16(1):5–11.
- Wright RW, Magnussen RA, Dunn WR, Spindler KP. Ipsilateral graft and contralateral ACL rupture at five years or more following ACL reconstruction: a systematic review. J Bone Joint Surg Am. 2011;93(12):1159–65.
- 13. Di Stasi S, Myer GD, Hewett TE. Neuromuscular training to target deficits associated with second anterior cruciate ligament injury. J Orthop Sports Phys Ther. 2013;43(11):777–92. A1-11.
- Borchers JR, Pedroza A, Kaeding C. Activity level and graft type as risk factors for anterior cruciate ligament graft failure: a case-control study. Am J Sports Med. 2009;37(12):2362–7.
- Frosch KH, Stengel D, Brodhun T, Stietencron I, Holsten D, Jung C, et al. Outcomes and risks of operative treatment of rupture of the anterior cruciate ligament in children and adolescents. Arthroscopy. 2010;26(11):1539–50.
- Beynnon BD, Johnson RJ, Abate JA, Fleming BC, Nichols CE. Treatment of anterior cruciate ligament injuries, part I. Am J Sports Med. 2005;33(10):1579–602.
- Ramski DE, Kanj WW, Franklin CC, Baldwin KD, Ganley TJ. Anterior cruciate ligament tears in children and adolescents: a meta-analysis of nonoperative versus operative treatment. Am J Sports Med. 2014;42:2769.
- Tjong VK, Murnaghan ML, Nyhof-Young JM, Ogilvie-Harris DJ. A qualitative investigation of the decision to return to sport after anterior cruciate ligament reconstruction: to play or not to play. Am J Sports Med. 2014;42(2):336–42.
- 19. Wright RW, Haas AK, Anderson J, Calabrese G, Cavanaugh J, Hewett TE, Lorring D, McKenzie C, Preston E, Williams G, the MOON Group. Anterior cruciate ligament reconstruction rehabilitation MOON guidelines. Sports health: a multidisciplinary approach. Rosemont, IL: American Academy of Orthopaedic Surgeons; 2014.
- Mather 3rd RC, Koenig L, Kocher MS, Dall TM, Gallo P, Scott DJ, et al. Societal and economic impact of anterior cruciate ligament tears. J Bone Joint Surg Am. 2013;95(19): 1751–9.

- 21. Wright RW, Dunn WR, Amendola A, Andrish JT, Bergfeld J, Kaeding CC, et al. Risk of tearing the intact anterior cruciate ligament in the contralateral knee and rupturing the anterior cruciate ligament graft during the first 2 years after anterior cruciate ligament reconstruction: a prospective MOON cohort study. Am J Sports Med. 2007;35(7):1131–4.
- Salmon L, Russell V, Musgrove T, Pinczewski L, Refshauge K. Incidence and risk factors for graft rupture and contralateral rupture after anterior cruciate ligament reconstruction. Arthroscopy. 2005;21(8):948–57.
- 23. Kaeding CC, Aros B, Pedroza A, Pifel E, Amendola A, Andrish JT, et al. Allograft versus autograft ACL reconstruction: predictors of failure from a MOON Prospective Longitudinal Cohort. Sports Health. 2011;3(1):73–81.
- Longo UG, Loppini M, Berton A, Marinozzi A, Maffulli N, Denaro V. The FIFA 11+ program is effective in preventing injuries in elite male basketball players: a cluster randomized controlled trial. Am J Sports Med. 2012;40(5):996–1005.
- 25. Yoo JH, Lim BO, Ha M, Lee SW, Oh SJ, Lee YS, et al. A meta-analysis of the effect of neuromuscular training on the prevention of the anterior cruciate ligament injury in female athletes. Knee Surg Sports Traumatol Arthrosc. 2010;18(6):824–30.
- 26. Fuller CW, Junge A, Dvorak J. Risk management: FIFA's approach for protecting the health of football players. Br J Sports Med. 2011;46(1):11–7.
- 27. Gatterer H, Ruedl G, Faulhaber M, Regele M, Burtscher M. Effects of the performance level and the FIFA "11" injury prevention program on the injury rate in Italian male amateur soccer players. J Sports Med Phys Fitness. 2012;52(1):80–4.
- Lawrence JT, Argawal N, Ganley TJ. Degeneration of the knee joint in skeletally immature patients with a diagnosis of an anterior cruciate ligament tear: is there harm in delay of treatment? Am J Sports Med. 2011;39(12):2582–7.
- Murray MM, Fleming BC. 2013. Use of a bioactive scaffold to stimulate anterior cruciate ligament healing also minimizes posttraumatic osteoarthritis after surgery. Am J Sports Med 41:1762–1770.
- Murray MM. The ACL Handbook Knee Biology, Mechanics, and Treatment. Murray MM, Vavken P, Fleming BC (Eds), New York, Springer, 2013.

Chapter 10 Concussion and the Female Athlete

Cynthia J. Stein and William P. Meehan III

Introduction

Concussion is a traumatic brain injury that affects over a million Americans each year [1]. Differences in males and females with regard to concussion are now emerging in the literature. Some studies suggest that female athletes may be at higher risk of concussion [2, 3], and may differ from males in terms of symptom profile [4, 5] as well as recovery time [6].

Concussion is generally initiated by a blow to the head or body, causing rapid rotation of the brain. Symptoms, such as headache, confusion, difficulty with concentration, and memory impairment, result from the disturbance of normal brain function. For most athletes, concussion causes short-term difficulties that resolve spontaneously; however, some athletes suffer prolonged changes after concussion. The purpose of this chapter is to review concussion risks, symptoms, management, and areas of research that are important to the young female athlete.

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Definition

Concussion: A type of traumatic brain injury, which is "a complex pathophysiological process affecting the brain, induced by biomechanical forces" [7]. It can be caused by a direct blow to the head or by transmission of force to the head leading to a shaking or rotation of the brain. Clinical symptoms are due primarily to a disturbance of normal brain function, rather than a structural injury. This disturbance has been attributed to a variety of changes, described as a neurometabolic cascade, involving ion shifts, neuronal depolarization, release of neurotransmitters, alterations in glucose metabolism, and changes in cerebral blood flow patterns [8, 9].

Epidemiology

The Centers for Disease Control and Prevention have determined that concussion is a public health problem, and the magnitude and effects of this issue are underestimated by existing surveillance systems [10]. It is estimated that there are 1.7 million traumatic brain injuries each year in the USA [11], and the majority of these are concussions [10]. Seventy percent of sports- and recreation-related traumatic brain injuries evaluated in the emergency department (ED) are among 10–19-year-olds [12], and females account for 31 % of all pediatric (age 0–19) concussion visits to the ED [13].

Bicycles and sports are the cause of 25-30 % of all mild traumatic brain injuries sustained by patients aged 5–14 years and cared for in the ED [13, 14]. Concussion is the cause of close to 15 % of all sports-related injuries in high school athletes, and 90 % of recorded injuries to the head and face in these athletes [15]. The estimated cost of these injuries, in terms of direct medical cost, plus indirect costs such as loss of productivity, is \$76.5 billion per year [16].

Football has more concussions, in terms of absolute numbers, than any other sport [2]. It has both a high rate of concussion and a large number of participating athletes, who are almost exclusively male. Therefore, in order to accurately compare risk in female and male athletes, it is important to look beyond the absolute numbers and to evaluate those sports played with similar rules by both males and females. Some studies have shown that in these sports—soccer, basketball, and softball/baseball—the risk of concussion is higher for female athletes [2, 3]. For example, one study showed that in high school sports, the overall concussion rate was 25 per 100,000 athlete-exposures (AEs). However, in gender comparable sports, the rates are higher among female athletes (17 per 100,000 AEs) than among males (10 per 100,000 AEs) [2]. Not surprisingly, rates of concussion are higher in contact and collision sports. For female high school athletes, the highest rates of concussion are found in lacrosse, soccer, and field hockey. For male high school athletes, football, ice hockey, and lacrosse have the highest incidence [2].

One study showed that the risk of concussion was over 50 % higher in girls' soccer (34 per 100,000 athletic exposures) than in boys' soccer (19 per 100,000 AEs) [2]. This relationship has been noted in both high school and college athletics; the incidence of concussion was over 60 % higher for female high school soccer players compared to their male counterparts (36 per 100,000 AEs in females vs. 22 in males.) [3] Among college soccer players, women continue to have an elevated risk compared to men (63 vs. 49 per 100,000 AEs). This apparent increased risk for female athletes has also been observed in basketball and in softball/baseball [3].

Concussion incidence rates are generally higher in competition than in practice, except for cheerleading where the incidence is higher in practice [2, 17]. Player-to-player contact is the most common mechanism of injury, causing 70 % of concussions, followed by player-to-surface contact, which is involved in 17 % of injuries [2].

Second Impact Syndrome

In rare cases, additional injury after concussion results in second impact syndrome [18]. This devastating syndrome was first described by Saunders and Harbaugh in 1984 [19]. It occurs when a person with a concussion suffers another injury, often minor, that is followed by cerebral edema, cerebral herniation, and death [19–21]. Cerebral edema, herniation, and catastrophic injury have been well described after a single episode of trauma to the brain, prompting some authors to question whether second impact syndrome is a separate clinical entity or whether a single blow to the head results in the catastrophic outcomes, arguing that the previous injury may be coincidental [22, 23]. Animal models, however, have demonstrated a window of increased vulnerability during which the brain is more susceptible to additional injury [24, 25]. Given these findings in animal models, and case reports of second impacts with devastating outcomes, it is crucial that concussed athletes avoid returning to sports or other high-risk activities until they have completely recovered.

Symptoms

Headache is the most commonly reported symptom of concussion [26, 27]. The pathophysiology of many post-concussion headaches remains unknown [28], but most appear to be migraine- or tension-type headaches [29]. Other common symptoms include dizziness, poor balance, nausea, fatigue, confusion, difficulty with concentration and memory, sensitivity to light and noise, sleep disturbance, sadness, and other emotional changes. Symptoms can vary widely by individual and by injury. Female athletes report more concussion symptoms overall [4], and one study showed female athletes tend to have more cognitive, emotional, and sleep symptoms than male athletes [5]. A separate study of high school athletes found that females report more neurobehavioral symptoms (related to sleep, fatigue, drowsiness, or nervousness) and somatic symptoms (such as headache, nausea, light and noise sensitivity, and balance difficulties) after sport-related concussion [30].

The post-concussion symptom scale included in the Sport Concussion Assessment Tool 3 (SCAT3) [7] is a frequently used tool for athletes to rate the severity of the most common concussion symptoms on a scale of 0 (none) to 6 (severe). After injury, the symptom severity score can be tallied for the athlete and used as a clinical measure to track recovery.

The classic presentation of concussion involves the development of symptoms after a rapid rotational acceleration of the brain, which is then followed by a gradual resolution over time. However, in some cases, symptoms can take hours to evolve [7] and may not be easily or immediately recognized. In addition, most concussion symptoms are not specific to concussion and can be due to a variety of other illnesses, injuries, and medications, which underscores the importance of proper medical history taking. Other etiologies of symptoms should always be considered [31], especially in cases without a clear history of a traumatic initiating event.

Diagnosis

Many sport-related concussions go undiagnosed [32, 33]. Most concussions do not involve obvious signs, such as loss of consciousness or convulsions [17, 27, 34, 35], and symptoms may be unrecognized or intentionally minimized by the athlete. One study found that a third of athletes evaluated for concussion had experienced a prior blow to the head that led to concussion-like symptoms, but that was not diagnosed as concussion [33]. Because symptoms can be subtle or change over time, the athlete, along with her coaches, parents, athletic trainers, and team physicians, must maintain a high index of suspicion for sport-related concussions.

The diagnosis of concussion is predominately based on the history and physical examination. Tools that have been developed to aid in diagnosis include symptom inventories, balance evaluations, and neurocognitive assessments. Instruments, such as the SCAT3, are available online [7]. This tool includes sideline cognitive assessments and a commonly used balance assessment, the balance error scoring system (BESS) [36]. Because there is great variability in balance and cognitive performance among individuals, these assessments are most helpful when compared to baseline measurements recorded at the start of the season prior to injury.

Different types of computer-based neurocognitive testing are available and are becoming more widely used to analyze and monitor changes that occur with concussion [37]. While these tests cannot be used to diagnose concussion, they offer objective measures of cognitive function and provide an additional tool that can be used in concussion management and return-to-play decisions. Some athletes show persistent deficits in their level of cognitive functioning, even after they report resolution of their symptoms, suggesting incomplete recovery [38–40]. The use of computerized neurocognitive testing allows clinicians to detect this possibly incomplete recovery and avoid potentially returning an athlete to play prematurely.

Scores vary by individual and can be affected by many different factors; therefore, preseason baseline scores are helpful in interpretation of these tests.

Because concussion is a functional problem as opposed to a gross structural injury to the brain, currently available imaging, such as magnetic resonance imaging (MRI) and computed tomography (CT), cannot detect concussion. Routine imaging is not recommended for the young athlete with concussion; however, imaging may be useful and necessary in some cases to rule out other causes of symptoms.

Risk Factors

Athletes involved in contact sports are at higher risk for sustaining concussions than those who participate in non-contact activities; [2, 15] however, concussions can occur in all types of sports and activities, including non-contact sports, such as swimming, golf [12], cheerleading, track [2], and dance [41]. A history of prior concussion is associated with an increased risk of a sustaining another concussion [17, 26, 34]. It is not clear if this elevated risk is due to genetic or structural factors that predispose individual athletes to concussion or whether this risk is associated with style of play, time spent in active participation, or changes to the physiology of the brain after the initial injury. Also, athletes with relatively low body mass index may have an elevated risk of concussion compared to their peers [17].

Some studies suggest that female athletes are at higher risk of sustaining concussions compared to male athletes involved in similar activities [2, 3, 42, 43]. Multiple hypotheses have been developed to explain this difference in risk. Some suggest that smaller head size, weaker neck muscles, or less dynamic head–neck stabilization may predispose the female athlete to concussion [44]. Others suggest that more accurate reporting by female athletes or increased attention by trainers and coaches to female athletes' complaints may explain some of the differences between reported rates of concussion between males and females. However, given that female athletes appear to have greater deficits on neuropsychological testing following injury compared to males [4], a physiological explanation seems more likely.

Treatment Guidelines

The most recent consensus statement from the 4th International Conference on Concussion in Sport held in Zurich (2012) [7] replaced earlier recommendations from Zurich (2008) [45], Prague (2004) [46], and Vienna (2001) [47]. Previously used grading systems (I/II/III) and classifications of concussion (simple/complex), have been superseded by a more individualized approach to concussion management, followed by a graduated return-to-play protocol.

If an athlete is suspected of sustaining a concussion, she should be removed from play and evaluated immediately. Because symptoms can develop and change over time, there should be no return-to-play on the day of injury [7]. During recovery, athletes must remain out of activities that involve risk of direct or indirect injury to the head.

The foundation of concussion management is physical and cognitive rest [7, 48]. However, the optimal amount of rest is not known and likely varies from person to person and injury to injury. In terms of physical rest, athletes should avoid strenuous activity and resistance training, which often worsen symptoms during the initial phases of treatment. After a period of rest and with continued recovery, some light exercise such as walking or stationary bicycling may be reintroduced gradually followed by more vigorous activities, as long as the athlete does not experience worsening of current symptoms or onset of new symptoms.

The Zurich guidelines outline six stages of progression from complete rest to full activity: [7] (1) No activity, (2) Light aerobic exercise, (3) Sport-specific exercise, (4) Non-contact training drills, (5) Full contact practice, and (6) Return to play. The guidelines recommend that the return-to-play protocol be initiated when the athlete is asymptomatic at rest. The athlete can then advance through the stages if she remains symptom free. If her symptoms reoccur at any stage, she should return to rest for at least 24 h and restart at a previous symptom-free level. As outlined in the guidelines, each stage should take at least 24 h [7].

Also for those with persistent symptoms, the guidelines suggest that light exercise may be beneficial, recommending a "sensible approach" that avoids exacerbation of symptoms [7]. Beyond being enjoyable for the athlete, it is hypothesized that exercise during recovery may improve sleep and decrease stress. However, few studies have looked at methods of physical activity advancement [49, 50], and the optimal timing of initiation, frequency, and intensity are not yet known.

Cognitive rest is also important in recovery, as many daily activities can worsen concussion symptoms and should be avoided or significantly limited. These include schoolwork, reading, computer work, video games, text messaging, and games that require concentration, such as chess. For students, academic accommodations are often used to adjust school schedules and academic workloads [51]. There is evidence to support the use of cognitive rest [52]; however, the amount of cognitive rest required is quite variable among injured athletes. Gradual return is recommended, but specific steps have not been well studied.

Most athletes will recover quickly from a sport-related concussion and will not require any treatment beyond physical and cognitive rest. Some athletes, however, suffer persistent symptoms and additional treatment may be recommended [53]. Common post-concussion symptoms, such as insomnia, headache, cognitive dysfunction, balance difficulties, and emotional changes, can continue to interfere with daily activities. A variety of medications, vestibular and other physical therapies, as well as educational and emotional support programs can be utilized to address these issues and improve quality of life during recovery. Many of these treatments are considered experimental and are not approved by the US Food and

Drug Administration for the management of concussion. Therefore, they should be used by providers who are experienced in concussion management. It is not yet known which treatment strategies work best with female or male athletes, or if significant differences exist.

Recovery Time

Most athletes recover from sport-related concussions within a few days to weeks [2, 15, 26, 27]. Over 95 % of high school athletes will recover from concussion within a month [15, 27]. Unfortunately, some have a more prolonged recovery. Factors associated with longer recovery include history of prior concussion [17, 26], amnesia at the time of injury [26, 39, 54], and decreased computerized neurocognitive test scores after injury [55, 56]. Interestingly, brief loss of consciousness is not associated with a prolonged recovery [57]. However, loss of consciousness, especially if it lasts longer than a few seconds, may indicate other injuries, such as an intracranial hematoma, and should be evaluated immediately by a medical professional.

Female athletes report more symptoms and demonstrate greater deficits on neuropsychological testing after concussion compared to male athletes [4]. In addition, some data suggest that female athletes may experience longer symptom duration than their male counterparts [6], but other studies have found no difference in time to recovery in males and females [30].

Variations in recovery time are not well understood and are the subject of ongoing research. Currently there is no known way to predict duration of symptoms, and recovery times vary from person to person and injury to injury. Therefore, athletes and their families should be counseled on the natural course of concussion and may need support in managing expectations, especially around return to play.

Cumulative Effects

For most athletes who experience concussion, symptoms resolve with time and rest, and they are able to return to full activities. However, some people who sustain multiple concussions or multiple sub-concussive blows to the head do suffer long-term effects [26, 58–60]. There is growing awareness and concern surrounding chronic traumatic encephalopathy (CTE), a degenerative neurological disorder that is believed to result from repetitive brain injury. Symptoms attributed to CTE include memory loss, difficulty with impulse control, depression, and dementia [61]. CTE has been found postmortem in contact sports athletes, but the association specifically with concussion or sub-concussive injuries is not yet well understood [62].

It has been shown that people who have had multiple concussions are at higher risk of additional concussions and longer recovery times [26]. Prolonged symptoms can include decreased processing speed [59], cognitive impairment, and memory difficulties [60]. Athletes, especially those with a history of multiple concussions, should understand the risk involved before making decisions about return to play.

Prevention

In some cases, changes to and enforcement of the rules of play may decrease the risk of concussions [63, 64]. Protective equipment, such as helmets and mouth guards, are crucial in preventing skull fractures, dental injuries, and catastrophic brain injuries. Therefore, all athletes participating in sports that require their use should wear properly fitting, undamaged personal protection equipment. However, there is little convincing evidence, that helmets, mouth guards, or other personal protection devices significantly reduce the risk of sport-related concussions.

There is some evidence to suggest that athletes are less likely to suffer a concussion when they anticipate an oncoming collision [65], and it has been hypothesized that stronger neck muscles, which can better stabilize the head, may reduce the risk of concussion by decreasing the acceleration of the brain after impact [43, 66]. Preliminary evidence suggests that increased neck strength does reduce the risk of concussion, and a 5 % reduction in the odds of concussion was estimated for every 1 pound increase in measured neck strength [67]. It is not yet known whether increased core strength and stability might also make the athlete better able to avoid or react to an impact. However, where there seem to be few risks and multiple potential benefits of neck and core stabilization, the athlete may want to ensure that these exercises are included in her regular fitness program.

Future Directions

Many questions about concussion have yet to be answered. Researchers are working to learn what puts some athletes at greater risk than others and which factors affect severity and duration of symptoms. We do not yet know why female athletes may be at higher risk, or what genetic, physical or behavioral elements may play a role in concussion risk and recovery. Efforts are being made to better understand the underlying pathophysiology of concussion, to improve methods of diagnosis and treatment, to predict recovery time and risk of long-term sequelae, and, perhaps most importantly, to discover and develop effective methods of preventing sport-related concussions.

Summary

Concussion is a traumatic injury to the brain that causes functional impairment. Female athletes may be at higher risk for concussion than male athletes involved in similar activities. Female athletes also may experience different types of symptoms and show more changes on neurocognitive testing after injury, compared to their male counterparts. For most athletes, concussion symptoms resolve quickly and completely. Unfortunately, some athletes suffer severe symptoms and prolonged recovery. Treatments exist to mitigate symptoms, and guidelines have been created to help athletes successfully return to full sports participation. Some factors, such as playing contact sports and history of prior concussion, are known to put athletes at higher risk of concussion, but other risk factors have not been well defined. Additional research is needed to better understand which factors put the female athlete at particular risk and to identify optimal methods of prevention, diagnosis, and treatment of sport-related concussions.

References

- Centers for Disease Control and Prevention. Nonfatal traumatic brain injuries from sports and recreation activities--United States, 2001-2005. MMWR Morb Mortal Wkly Rep. 2007; 56(29):733–7.
- Marar M, et al. Epidemiology of concussions among United States high school athletes in 20 sports. Am J Sports Med. 2012;40(4):747–55.
- Gessel LM, et al. Concussions among United States high school and collegiate athletes. J Athl Train. 2007;42(4):495–503.
- Covassin T, et al. The role of age and sex in symptoms, neurocognitive performance, and postural stability in athletes after concussion. Am J Sports Med. 2012;40:1303.
- Covassin T, et al. Sex and age differences in depression and baseline sport-related concussion neurocognitive performance and symptoms. Clin J Sport Med. 2012;22(2):98–104.
- Berz K, et al. Sex-specific differences in the severity of symptoms and recovery rate following sports-related concussion in young athletes. Phys Sportsmed. 2013;41(2):58–63.
- McCrory P, et al. Consensus statement on concussion in sport the 4th International Conference on Concussion in Sport held in Zurich, November 2012. J Sci Med Sport. 2013;16(3): 178–89.
- Giza CC, Hovda DA. The neurometabolic cascade of concussion. J Athl Train. 2001;36(3): 228–35.
- Shrey DW, Griesbach GS, Giza CC. The pathophysiology of concussions in youth. Phys Med Rehabil Clin N Am. 2011;22(4):577–602. vii.
- National Center for Injury Prevention and Control. Report to Congress on Mild traumatic brain injury in the United States: steps to prevent a serious public health problem. Atlanta, GA: Centers for Diseases Control and Prevention; 2003.
- 11. Faul M, et al. Traumatic brain injury in the United States: Emergency Department visits, hospitalizations and deaths 2002-2006. Atlanta, GA: National Center for Injury Prevention and Control Centers for Disease Control and Prevention; 2010.
- Centers for Disease Control and Prevention. Nonfatal traumatic brain injuries related to sports and recreation activities among persons aged </=19 years--United States, 2001-2009. MMWR Morb Mortal Wkly Rep. 2011;60(39):1337–42.

- Meehan 3rd WP, Mannix R. Pediatric concussions in United States emergency departments in the years 2002to 2006. J Pediatr. 2010;157(6):889–93.
- 14. Bazarian JJ, et al. Mild traumatic brain injury in the United States, 1998–2000. Brain Inj. 2005;19(2):85–91.
- 15. Meehan 3rd WP, et al. Assessment and management of sport-related concussions in United States high schools. Am J Sports Med. 2011;39(11):2304–10.
- 16. Finkelstein E, Corso PS, Miller TR. The incidence and economic burden of injuries in the United States. Oxford: Oxford University Press; 2006. xiii, 187 p.
- 17. Schulz MR, et al. Incidence and risk factors for concussion in high school athletes, North Carolina, 1996-1999. Am J Epidemiol. 2004;160(10):937–44.
- CDC. Sports-related recurrent brain injuries--United States. MMWR Morb Mortal Wkly Rep. 1997;46(10):224–7.
- 19. Saunders RL, Harbaugh RE. The second impact in catastrophic contact-sports head trauma. JAMA. 1984;252(4):538–9.
- 20. Byard RW, Vink R. The second impact syndrome. Forensic Sci Med Pathol. 2009;5(1): 36-8.
- 21. Cantu RC. Second-impact syndrome. Clin Sports Med. 1998;17(1):37-44.
- 22. McCrory P. Does second impact syndrome exist? Clin J Sport Med. 2001;11(3):144-9.
- 23. McCrory PR, Berkovic SF. Second impact syndrome. Neurology. 1998;50(3):677-83.
- 24. Longhi L, et al. Temporal window of vulnerability to repetitive experimental concussive brain injury. Neurosurgery. 2005;56(2):364–74. discussion 364–74.
- Meehan WP 3rd, Zhang J, Mannix R, Whalen MJ. Increasing recovery time between injuries improves cognitive outcome after repetitive mild concussive brain injuries in mice. Neurosurgery. 2012;71(4):885–91.
- Guskiewicz KM, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study. JAMA. 2003;290(19):2549–55.
- 27. Meehan 3rd WP, d'Hemecourt P, Comstock RD. High School concussions in the 2008-2009 Academic Year: mechanism, symptoms, and management. Am J Sports Med. 2010;38(12): 2405–9.
- 28. Packard RC. Epidemiology and pathogenesis of posttraumatic headache. J Head Trauma Rehabil. 1999;14(1):9–21.
- 29. Haas DC. Chronic post-traumatic headaches classified and compared with natural headaches. Cephalalgia. 1996;16(7):486–93.
- 30. Frommer LJ, et al. Sex differences in concussion symptoms of high school athletes. J Athl Train. 2011;46(1):76–84.
- 31. Sroufe NS, et al. Postconcussive symptoms and neurocognitive function after mild traumatic brain injury in children. Pediatrics. 2010;125(6):e1331–9.
- McCrea M, et al. Unreported concussion in high school football players: implications for prevention. Clin J Sport Med. 2004;14(1):13–7.
- Meehan 3rd WP, et al. The prevalence of undiagnosed concussions in athletes. Clin J Sport Med. 2013;23(5):339–42.
- Guskiewicz KM, et al. Epidemiology of concussion in collegiate and high school football players. Am J Sports Med. 2000;28(5):643–50.
- McCrory PR, Berkovic SF. Concussive convulsions. Incidence in sport and treatment recommendations. Sports Med. 1998;25(2):131–6.
- 36. Guskiewicz KM. Assessment of postural stability following sport-related concussion. Curr Sports Med Rep. 2003;2(1):24–30.
- 37. Meehan 3rd WP, et al. Computerized neurocognitive testing for the management of sport-related concussions. Pediatrics. 2012;129(1):38–44.
- Lovell MR, et al. Grade 1 or "ding" concussions in high school athletes. Am J Sports Med. 2004;32(1):47–54.
- 39. Lovell MR, et al. Recovery from mild concussion in high school athletes. J Neurosurg. 2003;98(2):296–301.
- 40. Field M, et al. Does age play a role in recovery from sports-related concussion? A comparison of high school and collegiate athletes. J Pediatr. 2003;142(5):546–53.

- 41. Stein CJ et al. Dance-related concussion: a case series. J Dance Med Sci. 2014;18(2):53-61.
- 42. Covassin T, Elbin RJ. The female athlete: the role of gender in the assessment and management of sport-related concussion. Clin Sports Med. 2011;30(1):125–31.
- Dick RW. Is there a gender difference in concussion incidence and outcomes? Br J Sports Med. 2009;43 Suppl 1:i46–50.
- 44. Tierney RT, et al. Gender differences in head-neck segment dynamic stabilization during head acceleration. Med Sci Sports Exerc. 2005;37(2):272–9.
- 45. McCrory P, et al. Consensus statement on concussion in sport: the 3rd International Conference on Concussion in Sport held in Zurich, November 2008. Br J Sports Med. 2009;43 Suppl 1:i76–90.
- 46. McCrory P, et al. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. Br J Sports Med. 2005;39(4):196–204.
- 47. Aubry M, et al. Summary and agreement statement of the First International Conference on Concussion in Sport, Vienna 2001. Recommendations for the improvement of safety and health of athletes who may suffer concussive injuries. Br J Sports Med. 2002;36(1):6–10.
- Reddy CC, Collins MW. Sports concussion: management and predictors of outcome. Curr Sports Med Rep. 2009;8(1):10–5.
- 49. Leddy JJ, et al. Rehabilitation of concussion and post-concussion syndrome. Sports Health. 2012;4(2):147–54.
- Leddy JJ, Willer B. Use of graded exercise testing in concussion and return-to-activity management. Curr Sports Med Rep. 2013;12(6):370–6.
- McGrath N. Supporting the student-athlete's return to the classroom after a sport-related concussion. J Athl Train. 2010;45(5):492–8.
- 52. Brown NJ, et al. Effect of cognitive activity level on duration of post-concussion symptoms. Pediatrics. 2014;133(2):e299–304.
- 53. Meehan 3rd WP. Medical therapies for concussion. Clin Sports Med. 2011;30(1):115-24. ix.
- 54. McCrea M, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. JAMA. 2003;290(19):2556–63.
- 55. Lau B, et al. Neurocognitive and symptom predictors of recovery in high school athletes. Clin J Sport Med. 2009;19(3):216–21.
- 56. Erlanger D, et al. Symptom-based assessment of the severity of a concussion. J Neurosurg. 2003;98(3):477–84.
- 57. Lovell MR, et al. Does loss of consciousness predict neuropsychological decrements after concussion? Clin J Sport Med. 1999;9(4):193–8.
- 58. Collins MW, et al. Cumulative effects of concussion in high school athletes. Neurosurgery. 2002;51(5):1175–9. discussion 1180–1.
- 59. Gronwall D, Wrightson P. Cumulative effect of concussion. Lancet. 1975;2(7943):995-7.
- Guskiewicz KM, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. Neurosurgery. 2005;57(4):719–26. discussion 719–26.
- McKee AC, et al. Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury. J Neuropathol Exp Neurol. 2009;68(7):709–35.
- 62. Solomon GS, Zuckerman SL. Chronic traumatic encephalopathy in professional sports: retrospective and prospective views. Brain Inj. 2015;29:164.
- Roberts WO, et al. Fair-play rules and injury reduction in ice hockey. Arch Pediatr Adolesc Med. 1996;150(2):140–5.
- Harmon KG, et al. American Medical Society for Sports Medicine position statement: concussion in sport. Br J Sports Med. 2013;47(1):15–26.
- Lincoln AE, et al. Video incident analysis of concussions in boys' high school lacrosse. Am J Sports Med. 2013;41(4):756–61.
- 66. Viano DC, Casson IR, Pellman EJ. Concussion in professional football: biomechanics of the struck player-part 14. Neurosurgery. 2007;61(2):313–27. discussion 327–8.
- 67. Collins CL et al. Neck strength: a protective factor reducing risk for concussion in high school sports. J Primary Prev. 2014: epub.

Chapter 11 Management of Cardiovascular Concerns in Female Athletes

Jennifer A. Michaud Finch and Aaron L. Baggish

Introduction

For decades, girls and women were discouraged from participating in competitive sports or vigorous exercise training for fear that physical activity could harm the reproductive system and ultimately diminish fertility. Fortunately, numerous visionary women rejected these recommendations [1]. The last 30 years in particular have seen a tremendous increase in female athletes' participation in a wide variety of sporting arenas. Effective care of the athlete requires an understanding of the cardiovascular demands of exercise, a familiarity with training-related cardiovascular adaptations, and a structured approach to the athlete with symptoms suggestive of cardiovascular disease [2]. This chapter is written to provide the clinician with a basic foundation of knowledge in these principal areas of patient care with emphasis on the differences between female and male athletes.

Definitions

Stroke volume (SV): The quantity of blood ejected from the heart during each contraction.

Cardiac output (CO): The product of stroke volume and heart rate.

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 VO_2 : The volume of oxygen consumed by the body. The relationship between CO and VO_2 is quantified by the Fick equation $[VO_2=cardiac output \times (\Delta arterial-venous O_2)]$: used to quantify the relationship between cardiac output and VO_2 .

Syncope: A sudden, transient loss of consciousness with inability to maintain postural tone, followed by complete and spontaneous recovery.

Palpitations: The awareness of forceful or subjectively abnormal heartbeats.

Overview of Relevant Exercise Physiology

There is a direct relationship between exercise intensity (external work) and the body's demand for oxygen. Oxygen demand is met by increasing pulmonary oxygen uptake (VO₂). Peak oxygen consumption (peak VO₂) is the amount of oxygen uptake/utilization that occurs at an individual's peak level of exercise. The cardio-vascular system is responsible for transporting oxygen-rich blood from the lungs to the skeletal muscles, a process that is quantified as cardiac output (liters per minute). In the healthy human, there is a direct and inviolate relationship between VO₂ and cardiac output.

Cardiac output (CO) may increase fivefold to sixfold during maximal exercise effort. Coordinated autonomic nervous system function, characterized by rapid and sustained parasympathetic withdrawal coupled with sympathetic activation, is required for this to occur. Heart rates may range from <40 beats per minute at rest to >200 beats per minute in a young, maximally exercising athlete. Heart rate increase is responsible for the majority of CO augmentation during exercise. Maximal heart rate varies innately among individuals, decreases with age, and does not increase with exercise training.

In contrast, stroke volume (SV), both at rest and during exercise, may increase significantly with prolonged exercise training. Cardiac chamber enlargement and the accompanying ability to generate a large SV are direct results of exercise training and are the cardiovascular hallmarks of the endurance-trained athlete. SV rises during exercise as a result of increases in ventricular end-diastolic volume and, to a lesser degree, sympathetically medicated reduction in end-systolic volume.

Hemodynamic conditions, specifically changes in CO and peripheral vascular resistance, vary widely across sporting disciplines. Although there is considerable overlap, exercise activity can be classified into two forms with defining hemodynamic differences. Isotonic exercise, or endurance exercise, involves sustained elevations in CO, with normal or reduced peripheral vascular resistance. Such activity represents a primary volume challenge for the heart that affects all four chambers. This form of exercise underlies activities such as long-distance running, cycling, rowing, and swimming. In contrast, isometric exercise, or strength training, involves activity characterized by increased peripheral vascular resistance and normal or only slightly elevated CO. This increase in peripheral vascular resistance causes transient, but potentially marked, increases in systolic hypertension and left ventricular afterload. Strength training is the dominant form of exercise in activities such as

weightlifting, track and field throwing events, and American-style football. Many team sports, such as soccer, lacrosse, basketball, hockey, and field hockey, involve significant elements of both endurance and strength exercise [4].

Cardiac Remodeling in Female Athletes

Long-term training is associated with left ventricular (LV) remodeling, including increases in chamber size, wall thickness, and mass. This remodeling is considered a physiologic adaptation to the increased hemodynamic load imparted by exercise [5, 7–9]. The term "the athlete's heart" is often used to describe the numerous attributes of exercise-induced cardiac remodeling. Despite persistence of this term in the literature, it has no specific definition and does not convey with certainty any specific aspect of cardiac structure and function. We therefore discourage its use in favor of more specific descriptors of cardiac morphologic findings.

The impact of exercise training on LV structure has been the topic of extensive study [22, 40, 41, 42, 44]. Early ECG investigations demonstrated increased electrical voltage suggestive of LV enlargement in trained athletes [10]. Subsequent work with 2-dimensional echocardiography confirmed underlying LV hypertrophy and dilation [11]. Italian physician-scientists have contributed a great deal to our understanding of LV structure in athletes using data derived from their long-standing pre-participation screening program. Pelliccia et al. [12] reported echocardiographic LV end-diastolic cavity dimensions in a large group of Italian elite female athletes representing 27 different sports. LV end-diastolic cavity dimensions ranged from 40 to 66 mm (mean 49±4 mm). Most demonstrated absolute values within normal limits (i.e., end-diastolic diameter \leq 54 mm), but 8 % exceeded this, and 1 % (four athletes) had cavity dimensions in the range of primary dilated cardiomyopathy (DCM) (\geq 60 mm). In this study, LV wall thickness was far less likely to exceed clinically accepted upper limits of normal and ranged from 6-12 mm (mean 8.2 ± 0.9 mm). Maximum LV wall thickness measured >10 mm in only a small minority of female athletes. Compared to sedentary controls (of comparable age, body size, and racial composition), the female athletes showed larger LV cavity dimensions (average +6 %), increased wall thickness (average +14 %), and larger LV mass normalized to body size (average +25 %) [12] (Fig. 11.1).

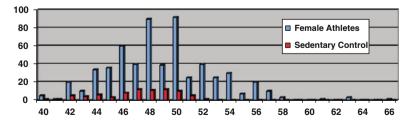


Fig. 11.1 Distributions of LV cavity dimensions (mm) in 600 elite female athletes compared to 65 sedentary controls. Adapted from Pelleccia, JAMA 1996

It may be difficult to differentiate normal physiologic adaptation from structural cardiovascular disease based solely on LV dimensions. As mentioned above, some highly trained female athletes may have dilated LV cavity dimensions (i.e., end-diastolic dimensions \geq 54 mm) and, occasionally, marked dilatation (>60 mm) that overlap into a distinctively pathologic range observed in patients with DCM [14]. This morphologic finding raises a differential diagnosis between extreme cardiac adaptation to intensive exercise training and a pathologic cardiac condition with potential for adverse clinical consequences.

Sharma et al. [13] reported a low incidence (0.4 %) of LV wall thickness >12 mm among 720 female and male elite adolescent athletes, and confirmed that increased LV wall thickness is associated with increased chamber size in young athletes. It must be emphasized that LV wall thickness in excess of 13 mm is a rare finding in healthy female athletes. This finding should therefore prompt consideration of pathological hypertrophy and further diagnostic workup. In contrast, LV dilatation accompanied by mild increases in LV wall thickness is common among female endurance athletes and should not necessarily be considered pathology. The correct identification of physiologic LV dilation may prevent the unnecessary withdrawal of athletes from competition, and the unjustified loss of the varied benefits derived from sport.

Overall, much of the variability of the LV dimensions seen in female athletes is associated with body size, greater chronological age, and lower resting heart rate (which reflects the intensity and duration of athletic conditioning) [12]. In addition, sport type impacts cardiac remodeling, with athletes participating in endurance disciplines (e.g., cycling, cross-country skiing, rowing, canoeing) often demonstrating the largest LV cavity dimensions. Gender itself is an independent determinant. Although much of the difference between male and female LV dimensions are correlated with differences in body size, other mechanisms possibly implicated are the lower absolute blood pressure response to exercise and lower availability of anabolic androgenic hormones in female versus male athletes.

Electrocardiography in Female Athletes

In addition to the morphological adaptations discussed above, there are characteristic patterns of electrical remodeling that are commonly captured on the 12-lead electrocardiogram (ECG). Sinus bradycardia, first degree atrioventricular (AV) block, incomplete right bundle branch block, early repolarization, and isolated voltage criteria for left ventricular hypertrophy (LVH) are common features of normal electrical remodeling in the female athlete's heart and do not usually require further evaluation [24, 43, 50]. In a cross-sectional analysis, such benign ECG patterns were observed among 40 % of trained competitive athletes participating in endurance sports [23, 47]. In contrast, ECG findings that warrant further cardiovascular evaluation include widespread T wave changes, complete left bundle branch block, left atrial enlargement when coupled with voltage criteria for left ventricular hypertrophy (LVH), right ventricular hypertrophy when coupled with right axis deviation (>120 degrees), ST segment depression, pre-excitation, prolonged heart rate, corrected QT interval (>500 ms), and pathologic Q waves, as none of them are normal features associated with physiologic cardiac remodeling.

Although the ECG increases the ability to detect underlying cardiovascular conditions that place athletes at increased risk for adverse events during sport participation, the ECG is a screening and diagnostic tool with inherent limitations in both sensitivity and specificity. Even if properly interpreted, an ECG will not detect all clinically relevant cardiovascular conditions. Our group evaluated 510 collegiate athletes using medical history, physical examination, ECG, and transthoracic echocardiography [17]. While 16 % demonstrated ECG abnormalities, only three male athletes (<1 %) and no female athletes were ultimately found to have conditions that warranted restriction from participation [17]. In a similar single university experience [48], 964 consecutive athletes underwent prospective collection of medical history, physical examination, 12-lead electrocardiography, and 2-dimensional echocardiography. ECGs were classified as abnormal in a third of athletes, and 10 % of those were classified as distinctly abnormal. Compared with females, male athletes were nearly three times more likely to have a distinctly abnormal ECG pattern [48].

It is important to note that the two studies discussed above used the 2005 European Society of Cardiology ECG interpretation criteria, which did not address some of the benign adaptive patterns frequently noted in trained athletes. Revised criteria [25, 50] have been developed, resulting in improved ECG sensitivity and specificity among athlete cohorts. Weiner, et al. [49] compared diagnostic accuracy of the European Society of Cardiology's 2010 ECG criteria to the 2005 criteria in 508 university athletes. Use of the revised criteria reduced the number of participants with abnormal ECG findings from 16.3 to 9.6 %, primarily due to the reclassification of participants with isolated QRS voltage criteria for LVH from abnormal to normal [49].

Clinical Care of the Female Athlete

Although participation in sport and regular exercise promotes good health, athletes are not immune to cardiovascular symptoms and disease. Sudden death in a young athlete is a tragic event that attracts substantial interest within the general and medical communities [15, 45, 46]. These deaths often assume a high public profile because of the youth of the victims and the generally held perception that trained athletes constitute the healthiest segment of society. Sudden death during sport is most commonly caused by occult cardiovascular disease [28], and many of the key cardiovascular diseases responsible for sudden death first manifest as symptoms during exercise. The following sections address the most common cardiovascular issues encountered in the clinical care of the female athlete.

Chest Pain

Chest pain is a common complaint seen in athletes across the entire age spectrum and the underlying etiologies of chest pain are both myriad and referable to many organ systems [38]. The term "chest pain" encompasses vague sensations that carry a low likelihood of cardiac etiology to typical angina that is commonly associated with pathologic underlying cardiovascular disease. Athletic patients presenting with chest pain most often experience their symptoms during physical exertion. In patients under 35 years of age, congenital valvular heart disease, genetic cardiomyopathies, such as hypertrophic cardiomyopathy (HCM), and coronary anomalies/ malformations are the leading causes of cardiac chest pain. In contrast, among patients over 35 years of age, obstructive coronary artery disease due to atherosclerosis is the most common cause of exertional chest pain [26, 27].

Chest pain can be divided into two categories: chest pain resulting from myocardial ischemia and chest pain from other causes. Myocardial ischemia results from an imbalance between myocardial oxygen demand and coronary blood flow. The most common causes of ischemic chest pain in athletes include atherosclerotic coronary artery disease (typically in older patients), anomalous coronary arteries (typically in younger patients), and genetic cardiomyopathies, most often HCM. Ischemic chest pain can also be caused by less frequent entities including coronary vasospasm, coronary artery dissection, valvular heart disease, including aortic stenosis (both congenital and acquired), severe anemia, and thyrotoxicosis.

Congenital coronary artery anomalies originating from the contralateral Sinus of Valsalva and following a course between the aorta and pulmonary artery are the most common cause of underlying ischemic chest pain in young athletes (Fig. 11.2) and are an important cause of exercise-induced sudden death [8, 29-31]. Preparticipation screening, whether confined to standard medical history and physical examination or inclusive of a resting or exercise 12-lead ECG, has limited capacity to identify coronary artery anomalies. Therefore, anomalous coronary lesions should be suspected in the athlete who presents with chest discomfort during or immediately after exertion, even if the athlete has been previously screened with a pre-participation exam. In our experience, "refractory asthma," more precisely defined as exertional dyspnea that fails to respond to conventional treatment for reactive airway disease, is a common presentation among athletes with high-risk coronary anomalies. When this diagnosis is suspected, direct, noninvasive imaging of the coronary anatomy is required. Transthoracic echocardiographic imaging is capable of accurately defining coronary anatomy in 90 % of athletes [31, 48]. Given the absence of radiation exposure and the widespread accessibility of this technique, we recommend this as the initial diagnostic imaging test in this setting. If transthoracic echocardiography does not yield definitive images of the coronary origins and proximal course, conventional invasive arteriography [32], magnetic resonance imaging [30, 33, 34], or computed tomography [35] should be considered, based on institutional preferences and insurance reimbursement patterns.

Chest pain that is unrelated to myocardial ischemia is common and often referred to as "atypical chest pain." While few studies have examined this type of pain in

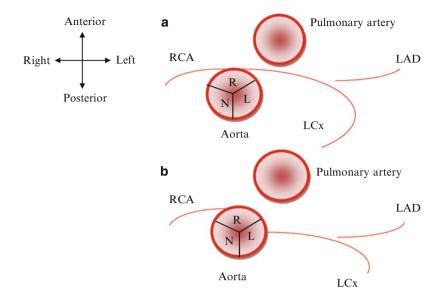


Fig. 11.2 Panel (a) Anomalous origin of a coronary artery from the opposite sinus with an interarterial course. Panel (b) Normal coronary anatomy. *RCA* right coronary artery, *LAD* left anterior descending artery, *LCx* left circumflex artery. *N* non-coronary cusp, *R* right coronary cusp, *L* left coronary cusp. Adapted from Lim, J. C. E. et al., *Nat. Rev. Cardiol.* 2011

athletes, the literature pertaining to the general population has relevance. Two studies examining atypical chest pain leading to hospital admission found that the causes fell into five categories: musculoskeletal, cardiac, gastrointestinal, respiratory, and miscellaneous. Musculoskeletal causes were the most common (27 %) [36], and in our experience, this finding also applies to athletes with chest pain. A careful physical examination of the chest wall can be relied on to differentiate musculoskeletal chest pain from other causes.

There are a few important cardiac conditions that can cause chest pain without myocardial ischemia. The most common are pericarditis, myocarditis, and aortic dissection. Pericarditis and myocarditis are inflammatory diseases of the heart lining and muscle respectively, which are most often caused by infectious agents (most commonly viruses). Both conditions often follow clinically appreciable infectious syndromes and are typically characterized by positional chest discomfort, fatigue, and in some cases palpitations. Aortic dissection, acute tearing of the aortic wall, is a surgical emergency that classically presents with "tearing" chest or back pain, often localized to the mid-scapular region. Among athletes, aortic dissection most commonly occurs in individuals with underlying aortopathy and is usually precipitated by intense isometric actions (i.e., lifting heavy weight) or direct blunt chest wall trauma (Table 11.1).

Table 11.1 Clinical cf	Table 11.1 Clinical characteristics of key cardiac causes of chest pain in young athletes	young athletes	
	Typical clinical presentation	Evaluation	Management
Coronary anomaly	Exertional chest discomfort at high levels of exertion	Resting 12-lead ECG often normal	Sport restriction as a bridge to surgical procedure
	Exertional shortness of breath often mislabel as "asthma"	Maximal exercise testing with or without imaging may show inducible ischemia	including coronary "unroofing" or
	Can manifest as syncope or cardiac arrest without premonitory symptoms	Direct imaging should be pursed in all cases of suspect anomalous coronary anatomy	re-implantation
Pericarditis	Often manifests within days to weeks of a clear non-cardiac viral infection	Resting ECG is diagnostic	NSAIDs ± colchicine
	Positional chest pain that is most severe when lying supine and relieved at least in part by sitting forward	2D-echo should be ordered to document the presence or absence of pericardial fluid	Activity restriction until symptom resolution
Myocarditis	Similar infectious link as above with pericarditis	Similar infectious link as above with pericarditis BCG variable without clear pathonomic patterns	Activity restriction until symptom resolution
	Symptoms variable (chest pain, dyspnea, exercise intolerance)	Reduced global or segmental function by 2D-echo and/or MRI	Cardiomyopathy medical regimen in cases of marked LV or RV dysfunction
Aortic dissection	"Tearing" pain localized in the back or scapula	ECG non-diagnostic unless dissection occludes coronary artery	Emergent cardiac surgery
		Urgent aortic imaging with CT scan or transesophageal echocardiography	

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Syncope

Syncope is one of the most common cardiovascular presentations among athletes. In a large series of young Italian athletes, approximately 6 % experienced some form of syncope during their athletic careers [18]. Etiologies of syncope in the female athlete range from the benign neurally mediated collapse to life-threatening pathologic conditions, including structural heart disease and primary arrhythmias.

Most syncope in athletes is attributable to neurally mediated mechanisms involving predominant and often excessive vagal tone related to chronic exercise training. Syncope frequently occurs in athletes outside of sports participation. Typical triggers include anxiety, sudden postural changes, and painful stimuli. While often recurrent, non-exertional, neurally mediated syncope in the athlete is usually a benign condition.

A closely related and common fainting syndrome is "post-exertional" syncope, often informally referred to as exercise-associated collapse. Post-exertional syncope occurs following abrupt cessation of exercise, most commonly moderate to high intensity endurance exercise, and is caused by a rapid reduction in central venous return. This reduction in venous return, caused by both the cessation of skeletal muscle contraction and altered sympathetic/parasympathetic balance, causes transient cerebral hypoperfusion. The affected athlete typically reports presyncopal feelings of warmth, lightheadedness, or diaphoresis, which begin within seconds of exercise termination and rapidly culminate in a loss of consciousness lasting from several seconds to a minute. Athletes predisposed to this condition should be counseled to avoid abrupt termination of exercise and in some cases may benefit from maneuvers to augment vascular volume, including hydration, dietary sodium augmentation, and use of commercially available compression stockings.

Syncope that occurs during intense exercise, either with or without prodromal symptoms, should raise suspicion for underlying cardiac disease. Traumatic injury secondary to syncope should further increase the index of suspicion for underlying disease. Current American College of Cardiology/American Heart Association (ACC/AHA) guidelines recommend that the approach to the athlete with exertional syncope begin with a meticulous history, physical examination, resting 12-lead electrocardiogram and echocardiogram [58]. The medical history should characterize potential triggers, the timing and duration of the event, and the risks associated with future episodes of loss of consciousness. Physical examination should be directed toward signs, most often pathologic murmurs, of occult heart muscle and valve diseases. The resting 12-lead ECG should be inspected for abnormalities of conduction (QT prolongation, pre-excitation, pathologic right bundle branch block with early precordial ST elevations suggestive of Brugada syndrome) and findings suggestive of structural heart disease (left bundle branch block, LVH with repolarization abnormalities, diffuse T-wave inversion). Transthoracic echocardiography is recommended to exclude structural and valvular heart disease in individuals with syncope, especially if any abnormality is detected during medical history, physical examination or ECG interpretation.

The comprehensive assessment of true exertional syncope often extends far beyond these basic measures and must be tailored to exclude underlying structural and electrical heart disease. We recommend such evaluations be conducted under the supervision of a cardiovascular specialist with expertise in the care of athletic patients. Provocative and customized exercise testing is often high yield and should be designed to approximate the exercise conditions in which the syncope occurred. Careful attention should be given to the exercise ECG for the detection of explanatory arrhythmias. When lab-based exercise testing is inconclusive, ambulatory rhythm monitoring may be necessary. There are numerous ambulatory rhythm monitoring devices available, and the choice should be dictated by the frequency and duration of syncope on an individual patient basis. The use of tilt-table testing in athletes, due to high rates of false-positive testing, is of limited value. In very select cases, most often in the setting of a documented arrhythmia syndrome, invasive electrophysiologic (EPS) study may be both diagnostic and potentially therapeutic [19].

Management of the athlete with syncope is dictated by cause. Individuals with significant structural or valvular heart disease should be managed with appropriate sport restriction, medication, EPS with or without ablation, implantable defibrillator placement, or surgery on the basis of specific pathology [39]. Neurally mediated post-exertional syncope can often be avoided by mandating an active cool down period after exertion and by paying attention to hydration and supplemental salt intake. In athletes with recurrent neurally mediated syncope despite these first-line treatments, postural training or pharmacologic therapy may be reasonable; however, these should be prescribed only after careful consideration of rules delineating banned substances in athletes.

Palpitations

Palpitations are a frequent complaint in athletes. The heightened vagal tone that accompanies routine exercise training commonly results in marked sinus bradycardia, and the relatively lengthy time period between sinus beats may facilitate an increase in ectopic atrial and ventricular beats. Slow resting heart rate coupled with the keen body awareness that is typical among athletic patients, often lead to sensing of these spontaneous depolarizations. In the majority of cases, resting palpitations that are suppressed with exercise, particularly among athletes with no findings during medical history, physical examination, and 12-lead ECG, are a benign phenomenon that requires no further evaluation [21].

The athlete presenting either with palpitations that are exacerbated by exercise or with other findings suggestive of occult cardiac disease requires a comprehensive evaluation by a cardiovascular specialist with expertise in the care of athletic patients. A broad differential diagnosis must be considered. Noninvasive cardiac imaging, customized exercise testing designed to stimulate the demands of training and competition in an attempt to trigger and capture the cause of palpitations, and ambulatory rhythm monitoring play key roles in the evaluation.

The choice of a specific ambulatory rhythm-monitoring device is crucial and requires individualized decision making. For symptoms that predictably recur within a 24-h period, simple Holter monitoring may be adequate. If the athlete experiences less frequent, more intermittent symptoms, she is best evaluated with a continuous loop recorder or event (patient-triggered) monitor. For those with very infrequent or elusive symptoms (>1 month between symptoms), an implantable loop recorder may be required.

Palpations due to benign atrial and/or ventricular ectopy that occur at rest, are suppressed by exercise, and are not accompanied by primary structural or electrical diseases, are best managed conservatively. Often counseling geared toward patient reassurance coupled with avoidance of potential triggers including alcohol, caffeine, and stimulants are sufficient. Among athletic patients with palpitations caused by pathology, disease-specific therapies designed to reduce the burden of arrhythmia (e.g., pharmacologic suppression, intracardiac ablation) and to reduce the risk of sudden cardiac death (e.g., sports restriction, implantable defibrillator placement) are often indicated.

Sudden Cardiac Death

Occult cardiovascular disease is the most common cause of sudden death in young athletes. The vast majority of these deaths in US athletes <35 years of age are due to congenital or genetic cardiac conditions [14, 26, 45–47]. Data establishing the true prevalence of sudden cardiac death (SCD) in athletes are plagued by imprecision, with reported figures ranging from 1:24,000 to 1:300,000 [27, 28, 45, 46]. Such variable findings are attributed to geographic differences in the prevalence of causal diseases, characteristics of the population studies, case ascertainment techniques, and other factors.

The incidence of SCD in high school and college athletes was first reported by Van Camp et al. [47] and was estimated to be 1:300,000 (1:212,000 for male athletes and 1: 1:300,000 for female athletes). Over a 10-year period of observation, SCD was found to be more common among male football and basketball athletes, more common in college athletes than in high school athletes, and more common among male athletes compared to females [47]. A subsequent registry-based series of SCD in young competitive US athletes over a 25-year period suggests an overall lower rate of SCD [26]. This study, the first to present autopsy data, suggested that HCM was the single most common cause of athletic deaths (responsible for 1/3 of cases), followed by congenital coronary artery abnormalities, particularly those of wrong aortic sinus origin (Fig. 11.3) [26].

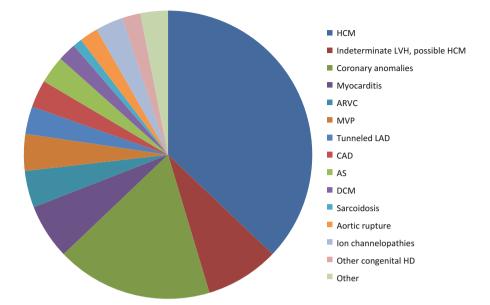


Fig. 11.3 Distribution of cardiovascular causes of sudden death in 1435 competitive athletes. *HCM* hypertrophic cardiomyopathy, *LVH* left ventricular hypertrophy, *ARVC* arrhythmogenic right ventricular cardiomyopathy, *MVP* mitral valve prolapse, *LAD* left anterior descending artery, *CAD* coronary artery disease, *AS* aortic stenosis, *DCM* dilated cardiomyopathy. Adapted from Maron. Minneapolis Heart Institute Foundation

A more recent study of National Collegiate Athletic Association (NCAA) athletes in the United States reported an incidence of SCD ranging from 1.05 to 3.45 per 100,000 athlete-years (Division 3 and 1 athletes, respectively), but no autopsies were performed and the causes of death were not verified [46]. SCD was 2.3 times more common in male athletes than in female athletes, and cardiac deaths were most common in basketball, football, swimming, lacrosse, and cross-country.

SCD incidence rates varied considerably by sport with the highest incidence observed in Division I basketball athletes. Looking specifically at basketball players, the rate of SCD was approximately 5 1/2 times higher in males (1:7000) than females (1:38,000). Race/ethnicity also appeared to affect risk in these basketball athletes, with the highest rate of SCD among black males (1:4000) compared to white males (1:13,000) [46].

The notion that underlying cardiovascular disease accounts for a substantial portion of athletic SCD has led to recommendations for pre-participation cardiac disease screening [16]. ACC/AHA joint guidelines recommend a pre-participation cardiovascular screening protocol for competitive athletes consisting of 12 items (8 personal and family history questions and 4 physical examination maneuvers), and do not require an ECG. A positive response or finding in one or more of the 12 items should trigger a referral for more in-depth cardiovascular evaluation. Parental verification of the responses for medical history questions is regarded as essential for middle school and high school students (Table 11.2).

Fam	nily History
1.	Premature sudden cardiac death
2.	Heart disease in surviving relatives less than age 50 years
Pers	conal History
3.	Heart murmur
4.	Systemic hypertension
5.	Fatigue
6.	Syncope/near syncope
7.	Exertional chest pain
8.	Excessive or unexplained exertional dyspnea
Phy	sical Exam
9.	Cardiac auscultation for heart murmurs (in supine and standing positions)
10.	Palpitations of peripheral pulses (bilateral) to detect diminished femoral pulses
11.	Visual inspection for signs of Marfan Syndrome
12.	Measurement of brachial blood pressure (bilateral) sitting position

 Table 11.2 The 12-element AHA recommendations for preparticipation cardiovascular screening of competitive athletes

Adapted from Maron et al., Circulation 2007

In contrast to the ACC/AHA guidelines, European pre-participation screening guidelines recommend mandatory inclusion of the 12-lead ECG. This difference is based on the concept that medical history and physical examination lack sufficient accuracy to detect the pathologic conditions that are of most relevance. Current ACC/AHA guidelines acknowledge this possibility but continue to avoid a national mandate for ECG inclusion due to concerns about financial cost and anticipated high rates of false positives.

At the present time, the definitive scientific and economic data required to settle this controversy are lacking. As previously mentioned, our group prospectively evaluated over 500 US collegiate athletes [17], and we found that screening with history and physical examination alone correctly identified fewer than half of the athletes with potentially important cardiac findings and failed to detect any of the athletes with structural abnormalities, including two athletes with cardiomyopathic conditions that necessitated sports restriction. Adding ECG to the history and examination improved the overall sensitivity and negative predictive value of athlete screening to 99.8 % and led to the detection of all participants with abnormalities that required sport restriction. However, ECG use was associated with a 16 % rate of false positive screening when the 2005 European Society of Cardiology criteria, the only athlete-specific criteria available at the time of the study, were applied. Refinements in ECG criteria have now been shown to markedly reduce the rates of false positive testing and have renewed interest in the use of ECG-inclusive screening in settings with the necessary clinical expertise [49–51].

Future Directions

Several clinical issues in sports cardiology require further attention. First, a strategy for differentiating adaptive from pathological cardiomyopathy that integrates basic clinical factors with modern diagnostic tests (imaging, cardiopulmonary exercise testing, and genetic assessment) should be developed and validated for clinical use. Second, consensus committee guidelines for the management of cardiovascular disease in athletes require timely updates as dictated by diagnostic and therapeutic advances. Third, prospective, longitudinal study of the impact of pre-participation ECG screening should be conducted to end the ongoing debate about how best to reduce incidence of sport-related sudden cardiac death.

Summary

Our understanding of the athlete's heart has progressed, however a number of unanswered questions remain. The physiologic cardiac adaptations to endurance and strength training that have been well-defined in male athletes need to be better studied in female athletes, and we need to continue to refine our understanding of the ways that these physiologic changes are reflected on the ECG and other cardiac screening tools.

Caring for the young female athlete requires an understanding of exercise-related cardiovascular demands and adaptations, as well as a structured approach to screening asymptomatic athletes and evaluating those athletes who present with symptoms. With growing participation of female athletes, future prospective sports cardiology studies need to include larger numbers of girls and women of various demographics and sports disciplines in order to better address the needs of these athletes.

References

- 1. Sangenis P. Women in sport, cardiovascular issues. Encyclopedia of sports medicine. IOC Medical Commission Publication. London: Blackwell and Science; 2000. p. 241–8.
- Lawless CE, Olshansky B, Washington RL, et al. Sports and exercise cardiology in the United States: cardiovascular specialists as members of the athlete healthcare team. J Am Coll Cardiol. 2014;63(15):1461–72.
- 3. Prior D, LaGerche A. The athlete's heart. Heart. 2012;98:947-55.
- 4. Baggish A, Wood M. Athlete's heart and cardiovascular care of the athlete scientific and clinical update. Circulation. 2011;123:2723–5.
- 5. Pluim BM, et al. The athlete's heart. A meta-analysis of cardiac structure and function. Circulation. 1999;100:336–44.
- Maron BJ. Structural features of the athlete heart as defined by echocardiography. J Am Coll Cardiol. 1986;7:190–203.

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- 7. Spirito P, Pelliccia A, et al. Morphology of the "athlete's heart" assessed by echocardiographic in 947 elite athletes representing 27 sports. Am J Cardiol. 1994;74:802–6.
- 8. Venerando A, Rulli V. Frequency and meaning of the electrocardiographic anomalies found in Olympic marathon runner and walkers. J Sports Med Phys Fitness. 1964;4:135–41.
- 9. Roeske WR, et al. Noninvasive evaluation of ventricular hypertrophy in professional athletes. Circulation. 1976;53:286–91.
- Pelliccia A, Maron BJ, et al. Athlete's heart in women. Echocardiographic characterization of highly elite female athletes. JAMA. 1996;276:211–5.
- Sharma S, Maron BJ, et al. Physiologic limits of left ventricular hypertrophy in elite junior athletes: relevance to differential diagnosis of athlete's heart and hypertrophic cardiomyopathy. J Am Coll Cardiol. 2002;40:1431–6.
- Baggish AL, et al. Differences in cardiac parameters among elite rowers and subelite rowers. Med Sci Sports Exerc. 2010;42:1215–20.
- Maron BJ, Thompson PD, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes 2007 update. Circulation. 2007;115:1643–55.
- 14. Corrado D, et al. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. JAMA. 2006;296:1593–601.
- Baggish AL, Hutter Jr AM, Wang F, Yared K, Weiner RB, Kupperman E, Picard MH, Woods MJ. Cardiovascular screening in college athletes with and without electrocardiography: a cross-sectional study. Ann Intern Med. 2010;152:269–75.
- Colivicchi F, Ammirati F, Santini M. Epidemiology and prognostic implications of syncope in young competitive athletes. Eur Heart J. 2004;25(19):1749–53.
- Colivicchi F, Ammirati F, Biffi A, Verdile L, Pelliccia A, Santini M. Exercise-related syncope in young competitive athletes without evidence of structural heart disease. Clinical presentation and long-term outcome. Eur Heart J. 2002;23(14):1125–30.
- 18. Lawless CE, Briner W. Palpitations in athletes. Sports Med. 2008;38(8):687-702.
- 19. Pluim BM, et al. The athlete's heart. A meta-analysis of cardiac structure and function. Circulation. 2000;101:336–44.
- Pelliccia A, Maron BJ, Culasso F, Di Paolo FM, Spataro A, Biffi A, Caselli G, Piovano P. Clinical significance of abnormal electrocardiographic patterns in trained athletes. Circulation. 2000;102:278–84.
- Corrado D, et al. 12 Lead ECG in the athlete: physiological versus pathological abnormalities. Br J Sports Med. 2009;43:669–76.
- 22. Corrado D, et al. Recommendations for interpretation of 12-lead electrocardiogrtam in the athlete. Eur Heart J. 2010;31:243–59.
- 23. Maron BJ. Sudden death in young athletes. N Engl J Med. 2003;349:1064-75.
- 24. Maron BJ, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. J Am Coll Cardiol. 1998;32:1881–4.
- Maron BJ, Shirani J, et al. Sudden death in young competitive athletes: clinical, demographic and pathologic profiles. JAMA. 1996;276:199–204.
- Basso C, Maron BJ, Corrado D, Thiene G. Clinical profile of congenital coronary artery anomalies with origin from the wrong aortic sinus leading to sudden death in young competitive athletes. J Am Coll Cardiol. 2000;35:1493–501.
- Zeppilli P, Dello Russo A, Santini C, et al. In vivo detection of coronary artery anomalies in asymptomatic athletes by echocardiographic screening. Chest. 1998;114:89–93.
- 28. Pelliccia A, Spataro A, Maron BJ. Prospective echocardiographic screening for coronary artery anomalies in 1,360 elite competitive athletes. Am J Cardiol. 1993;72:978–9.
- 29. Serota H, Barth III CW, Seuc CA, et al. Rapid identification of anomalous coronary arteries in adults: the "dot and eye" method. Am J Cardiol. 1990;65:891–8.
- McConnell MV, Ganz P, Selwyn AP, et al. Identification of anomalous coronary arteries and their anatomic course by magnetic resonance coronary angiography. Circulation. 1995;92: 3158–62.

- Post JC, van Rossum AC, Bronzwaer JGF, et al. Magnetic resonance angiography of anomalous coronary arteries. A new gold standard for delineating the proximal course? Circulation. 1995;92:3163–71.
- 32. Mousseaux E, Hernigou A, Sapoval M, et al. Coronary arteries arising from the contralateral aortic sinus: electron beam computed tomographic demonstration of the initial course of the artery with respect to the aorta and right ventricular outflow tract. J Thorac Cardiovasc Surg. 1996;112:836–40.
- Spalding L, et al. Cause and outcome of atypical chest pain in patients admitted to hospital. J R Soc Med. 2003;96(3):122–5.
- 34. Perron A. Chest pain in athletes. Clin Sports Med. 2003;22:37-50.
- 35. Pelliccia A, et al. European Society of Cardiology consensus document: recommendations for competitive sports participation in athletes with cardiovascular disease. Eur Heart J. 2005;26:1422–45.
- Urhausen A, Monz T, Kindermann W. Sport-specific adaptation of left ventricular muscle mass in athlete's heart. Int J Sports Med. 1996;17:S145–51.
- Maron B. Structural features of the athlete's heart as defined by echocardiography. J Am Coll Cardiol. 1986;7(1):190–203.
- Pelliccia A, Maron M, Maron B. Assessment of the left ventricular hypertrophy in a trained athlete: differential diagnosis of physiologic athlete's heart from pathologic hypertrophy. Prog Cardiovasc Dis. 2012;54:387–96.
- Tikkanen JT, Anttonen O, Junttila MJ, Aro AL, Kerola T, Rissanen HA, Reunanen A, Huikuri HV. Long-term outcome associated with early repolarization on electrocardiography. N Engl J Med. 2009;361:2529–37.
- 40. Baggish A, et al. Training-specific changes in cardiac structure and function: a prospective and longitudinal assessment of competitive athletes. J Appl Physiol. 2008;104(4):1121–8.
- Maron B, et al. Incidence and cause of sudden death in U.S. college athletes. J Am Coll Cardiol. 2014;63:1636–43.
- 42. Harmon K, et al. Incidence of sudden cardiac death in national collegiate athletic association athletes. Circulation. 2011;123:1594–600.
- 43. Van Camp SP, Bloor CM, Mueller FO, Cantu RC, Olson HG. Nontraumatic sports death in high school and college athletes. Med Sci Sports Exerc. 1995;27:641–7.
- 44. Magalski A, et al. Cardiovascular screening with electrocardiography and echocardiography in collegiate athletes. Am J Med. 2011;124(6):511–8.
- 45. Weiner R, et al. Performance of the 2010 European Society of Cardiology criteria for ECG interpretation in athletes. Heart. 2011;97:1573–7.
- 46. Drezner JA, et al. Electrocardiographic interpretation in athletes: the 'Seattle Criteria'. Br J Sports Med. 2013;47:122–4.
- 47. Malhotra A, et al. 103 Prevalence and significance of anterior T wave inversions in females. Heart. 2014;100:A60. doi:10.1136/heartjnl-2014-306118.103.
- Dougals P, ACCF/ASE/AHA/ASNC/HFSA/HRS/SCAI/SCCM/SCCT/SCMR, et al. Use criteria for echocardiography. J Am Soc Echocardiogr. 2011;24:229–67.
- 49. Strickberger SA, et al. AHA/ACCF scientific statement on the evaluation of syncope: from the American Heart Association Councils on Clinical Cardiology, Cardiovascular Nursing, Cardiovascular Disease in the Young, and Stroke, and the Quality of Care and Outcomes Research Interdisciplinary Working Group; and the American College of Cardiology Foundation: In Collaboration With the Heart Rhythm Society: Endorsed by the American Autonomic Society. Circulation. 2006;113:316–27.
- Brosnan M, et al. The Seattle Criteria increases the specificity of preparticipation ECG screening among elite athletes. Br J Sports Med. 2014;48:1144–50.
- Wasfy M. ECG findings in competitive rowers: normative data and the prevalence of abnormalities using contemporary screening recommendations. Br J Sports Med. 2015;49:200.

Chapter 12 Physical Activity and Chronic Disease Prevention

Hank Dart, Nhi Nguyen, and Graham A. Colditz

Introduction

It is clear that there are many benefits of sports participation for the young female athlete, including increased self-esteem, higher academic performance, decreased risk of depression, and lower rates of drug use [1-3]. In addition, there are also significant long-term health benefits that come with regular physical activity.

Approximately 12 % of all mortality in the USA is related to the lack of regular physical activity, and generally recommended levels of physical activity provide a 20 % reduction in all-cause mortality [4, 5] (Fig. 12.1). Studies have consistently shown that physical activity reduces the risk of major chronic diseases in a dose-dependent relationship (Table 12.1). The strongest evidence exists for diabetes, car-diovascular disease, colon cancer, breast cancer, and osteoporosis. There is also emerging evidence of the protective effect of physical activity on dementia. In the following sections, we briefly review the evidence for each of these conditions.

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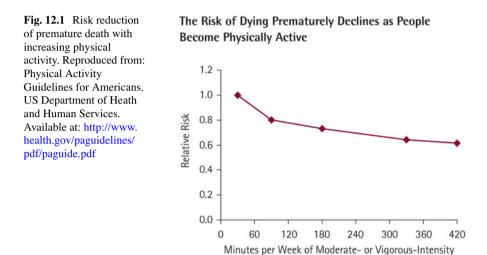
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Despite demonstrated and robust health benefits, less than half (47 %) of adult women in the USA engage in recommended levels of aerobic physical activity [6, 7]. In girls aged 12–15 years old, only 23 % meet the recommended guidelines for youth of 60 min of moderate to vigorous-intensity activity every day [8]. Just under 9 % of girls get zero moderate or vigorous-intensity activity in the course of a week.

Increasing physical activity levels is a major goal of Healthy People 2020, which aims to set achievable targets for improving the nation's health [9].



Physical Activity

Table 12.1 Health benefits associated with regular physical activity	vity
Children and adolescents	
Strong evidence	
 Improved cardiorespiratory and muscular fitness 	
Improved bone health	
• Improved cardiovascular and metabolic health biomarkers	
Favorable body composition	
Moderate evidence	
Reduced symptoms of depression	
Adults and older adults	
Strong evidence	
Lower risk of early death	
Lower risk of coronary heart disease	
Lower risk of stroke	
Lower risk of high blood pressure	
	(continued)

• Lo	wer risk of adverse blood lipid profile
• Lo	ower risk of type 2 diabetes
• Lo	ower risk of metabolic syndrome
• Lo	ower risk of colon cancer
• Lo	ower risk of breast cancer
• Pr	evention of weight gain
• W	eight loss, particularly when combined with reduced calorie intake
• In	proved cardiorespiratory and muscular fitness
• Pr	evention of falls
• Re	educed depression
• Be	etter cognitive function (for older adults)
Mode	erate to strong evidence
• Be	etter functional health (for older adults)
• Re	educed abdominal obesity
Mode	erate evidence
• Lo	ower risk of hip fracture
• Lo	ower risk of lung cancer
• Lo	ower risk of endometrial cancer
• W	eight maintenance after weight loss
• In	creased bone density
• In	proved sleep quality
Reprodu	uced from: US Department of Health & Human Services, 2008 Physical

Reproduced from: US Department of Health & Human Services. 2008 Physical Activity Guidelines for Americans

Definitions

Physical activity: bodily movement produced by the contraction of skeletal muscle that increases energy expenditure above the basal level [10].

Exercise: a form of physical activity that is planned, structured, repetitive, and purposeful with a main objective of improvement or maintenance of one or more components of physical fitness [11].

Type 2 Diabetes

Diabetes affects over 11 % of females in the USA, with the vast majority of cases being preventable type 2 diabetes [12]. Once very rare in childhood, type 2 diabetes is now seen with increasing frequency in obese youth under age 20 [13]. All told, the economic cost of diabetes is approximately \$245 billion each year in the USA alone [14].

Strong evidence supports physical activity as a key intervention for preventing type 2 diabetes (DM2). One analysis of ten prospective cohort studies reported a 30 % reduction in the risk of developing DM2 with moderate physical activity, such as brisk walking, compared with being sedentary [15]. Regular exercise has also been shown to reduce hemoglobin A1c (HbA1c), which is an indicator of average blood glucose levels over the previous 60–90 days [16–19].

In DM2, genetics play a substantial role in insulin resistance and impaired insulin secretion. Moderate physical activity on a long-term basis increases the translocation of insulin-responsive glucose transporter (GLUT4) from intracellular stores to cell surface. GLUT4 promotes glucose uptake, which probably explains the overall increase in insulin sensitivity [20]. In addition, favorable changes also occur in skeletal muscles including increased mitochondrial enzyme activity, thereby improving muscle energetics, and muscle capillary recruitment. However, there is little or no increase in muscle capillaries in patients with microvascular complications [21]. Although exercise can transiently increase urinary protein excretion, there is no evidence of increased progression of chronic kidney disease [22].

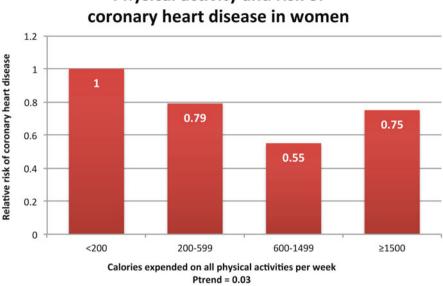
The American Diabetes Association recommends at least 150 min of moderateintensity aerobic activity per week for prevention of DM2, along with strengthtraining multiple days per week. Efforts to lose weight or maintain weight loss may require greater levels of aerobic activity. Regular activity is also a key part of managing DM2 as well as helping to reverse it [22–24].

Cardiovascular Disease

Cardiovascular disease is a major health burden in the USA. More than a third of adult women have some type of cardiovascular disease, which includes heart disease, stroke, and related conditions [25]. Cardiovascular disease kills approximately 400,000 women each year—more than all cancers combined [25].

Despite their high prevalence, heart disease and stroke are largely preventable through a mix of healthy lifestyle choices and therapeutic interventions to control risk factors like diabetes, unhealthy blood lipids, and hypertension [26]. Though heart disease and stroke usually do not present until older age, risk factors can begin to accrue early in life. A 2012 analysis by May et al. found that 14 % of US adolescents 12–19 years old had hypertension or prehypertension; 22 % had high or borderline high low-density lipoprotein (LDL) cholesterol; and 15 % had diabetes or prediabetes. Fully 37 % of normal weight adolescents had at least one cardiovascular disease risk factor, while 43 % of obese adolescents did [27].

Regular physical activity has been demonstrated to lower the risk of cardiovascular disease, with the American Heart Association classifying activity as a "useful and effective" Class I lifestyle intervention [26]. Separate analyses of the Women's



Physical activity and risk of

Fig. 12.2 Risk reduction of coronary heart disease with physical activity. Data source: Lee et al., 2001

Health Study and Nurses' Health Study found significant benefits from regular walking, with 1 h or more a week lowering risk by 30-50 % compared to those who did not walk regularly (Fig. 12.2) [28, 29]. Results from the Nurses' Health Study also showed a lower risk of total stroke and, especially, ischemic stroke with increasing amounts of regular physical activity and walking [30].

Regular activity reduces serum triglyceride and increases high-density lipoprotein cholesterol (HDL) with variable effect on LDL. Within 4 weeks of beginning an exercise program, blood pressure can lower as much as 5-15 mmHg in patients with primary hypertension. There is also increasing evidence that regular exercise reduces serum C-reactive protein, which plays an important role in atherosclerosis [31–33].

For adults, the American Heart Association strongly recommends 30 min of moderate-intensity aerobic activity at least 5 days per week for a total of 150 min, or 25 min of vigorous aerobic activity at least 3 days per week for a total of 75 min, or a combination of the two. A weaker recommendation is given for moderate to high intensity muscle strengthening activity at least 2 or more days per week for additional health benefits [34].

Cardiac events during exercise are uncommon, with the highest risk in those who are untrained and take part in vigorous-intensity activity [10]. The overall benefits of physical activity far outweigh the possible associated risks in the majority of individuals [10].

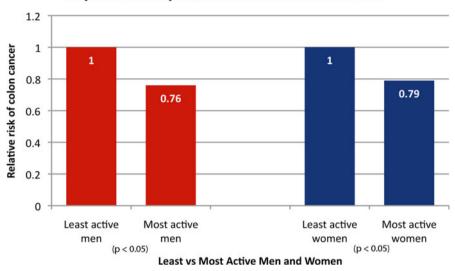
Sudden cardiac death in youth and young adult athletes is a rare, but often high profile, event, affecting fewer than 100 athletes each year [35]. Hypertrophic cardiomyopathy and coronary artery abnormalities have been identified as primary causes, among others [35, 36]. Multiple professional medical and athletic organizations endorse pre-participation cardiovascular screening for young competitive athletes to identify those at high risk of cardiac events [36]. The American Heart Association currently recommends that such screening involve a targeted personal history, family history, and physical examination [37, 38]. For more information on cardiac conditions and risks, see Chapter 11.

Cancer

Cancer is rare in youth and young adults, and becomes increasingly common with age. Over three quarters of all cancers are diagnosed at age 55 years or older [39]. Early life, however, is an important period for establishing lifelong healthy habits that can reduce adult cancer risk. Regular physical activity lowers the risk of numerous cancers, including colon cancer, breast cancer, endometrial cancer, and possibly lung cancer.

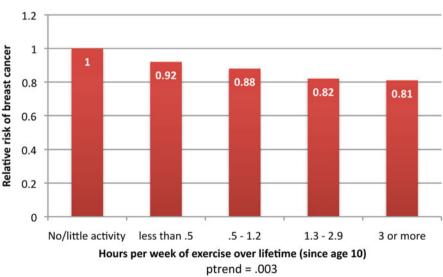
Colon and rectal cancer: Cancer of the colon and rectum is the third most common cancer among American women, after lung and breast cancers [39]. The benefit of physical activity on colon cancer risk has been extensively studied, with consistent reports of 30–40 % reduction in colon cancer risk in adults who increase their physical activity [40]. A meta-analysis by Wolin et al., of 52 observational studies, found that, compared to the least active women, the most physically active women experienced a 21 % lower risk of colon cancer (Fig. 12.3) [41]. However, no association has been found for rectal cancer [42, 43]. It is estimated that 30–60 min of moderate to vigorous physical activity per day is needed to protect against colon cancer [40].

Breast cancer: Breast cancer is the most common cancer among women both in the USA and worldwide [39, 44]. In the USA and other high-income countries, approximately 22 % of cases are diagnosed in women under 50 years old, and about 8 % in those under age 40. Early interest in the effect of physical activity on breast cancer resulted from the association of activity with weight and hormone metabolism. However, physical activity is now recognized as an independent risk factor. Numerous studies have reported that moderate to vigorous physical activity is associated with a decreased breast cancer risk of 20–30 % among both premenopausal and postmenopausal women [45, 46]. A study by Bernstein et al. linked lifetime regular exercise habits from the age of 10 years with reduced adult breast cancer risk (Fig. 12.4) [47]. Women who increase their physical activity after menopause may also experience a reduced risk [48, 49]. In addition, research also suggests that regular activity in early life, between the ages of 12–22 years, may be particularly beneficial when it comes to later life breast cancer risk [50]. Most studies suggest



Physical Activity Level and Risk of Colon Cancer

Fig. 12.3 Risk reduction of colon cancer with physical activity. Data source: Wolin et al., 2009



Lifetime Exercise and Risk of Breast Cancer

Fig. 12.4 Risk reduction of breast cancer with lifetime exercise. Data source: Bernstein et al., 2005

that 30–60 min per day of moderate- to high-intensity physical activity is associated with a reduction in breast cancer risk [40, 51].

Endometrial cancer: Epidemiologic studies consistently report an inverse association between physical activity and endometrial cancer. These studies suggest that women who are physically active have a 20–40 % reduced risk of endometrial cancer in a dose-dependent relationship [40].

Lung cancer: Lung cancer is the leading cause of cancer death in both women and men in the US, with more than 85 % of deaths caused by tobacco smoking [52, 53]. Overall, studies suggest a 20 % risk reduction in lung cancer with physical activity [40, 51]. However, smoking is an important risk factor, and tobacco use is associated with lack of activity and unhealthy dietary habits. Therefore, it is difficult to isolate effects of these factors in relation to the risk of developing lung cancer. For example, one study found that higher levels of physical activity among current and former smokers were associated with a lower risk of lung cancer, but no association was found in nonsmokers. The authors suggested these discrepancies may be due to a residual confounding effect of smoking [54].

Physical activity may lower cancer risk through a number of potential causal mechanisms, including improved immune function and the regulation of selected hormones [55, 56]. Although the optimal intensity, duration, and frequency of physical activity to prevent cancer are unknown, standard recommendations of 150–300 min or more of moderate intensity physical activity each week should provide benefits.

Osteoporosis

Osteoporosis is a major health burden in the US. Sixteen percent of women age 50 or older suffer from osteoporosis of the femoral neck or lumbar spine, while over 75 % exhibit some level of compromised bone strength [57].

Marked by a significant fracture risk due to poor bone strength, osteoporosis is a condition most often of later life. Yet modifiable lifestyle factors in youth and young adulthood can have an important impact on later adult risk. Physical activity is a particularly important factor in both achieving optimal bone mineral density (BMD) and maintaining bone mass throughout adulthood.

Approximately 90 % of a woman's peak bone mass (PBM) develops by the time she reaches age 18, with most of this occurring in the adolescent years [58]. The remainder accumulates between the ages of 20–30 [58]. Bone mass is slowly and inevitably lost after this. Optimizing PBM in early life offers one of the most important opportunities to avoid or delay osteoporosis in later adulthood [58].

Studies of identical twins attribute 60-70 % of variability in the magnitude of peak bone mass to genetics, while 30-40 % of variability is thought to be related to environmental factors such as diet, exercise, diseases, and medications [59].

Lack of physical activity in adolescence has been shown to be an independent risk factor of low bone mass in healthy premenopausal women [60]. Results from studies in children consistently report that weight-bearing physical activity increases

both bone size and BMD during pre-puberty and early adolescence [61]. Additionally, in a 20-year longitudinal study, Foley et al. found a positive correlation between measures of childhood fitness and later adult bone health [62].

While regular activity in adolescence and the young adult years have undeniable benefits, the young female athlete also needs to be cautious about the Female Athlete Triad (Triad), which is a medical condition that involves at least one of three components of low energy availability, menstrual dysfunction, and poor bone health [58, 63]. Though any female may be affected, athletes in sports that stress leanness (such as gymnastics, long-distance running, and Nordic skiing) are more prone to the condition, which, if untreated, can advance to disordered eating, amenorrhea, and osteoporosis, with both short-term and lasting effects [64]. The initial focus of treating Triad conditions is to improve an athlete's energy availability, with increased calorie intake, lower energy output, or a combination of both [63, 64]. On a population level, coaches, trainers, parents, and others directly involved with athletes need to be educated on the health risks related to the Triad and approaches to help avoid them [64]. For additional information on the Triad, see Chapter 5.

In adulthood, bone loss begins to occur around age 40. Rates of loss vary from woman to woman but are on the order of 0.5 % per year, with a higher rate of loss in the years surrounding menopause [65]. Regular physical activity can help limit bone loss and reduce the risk of fractures. A 2010 Cochrane Collaboration metaanalysis of 43 randomized controlled trials by Howe et al. found that regular exercise in postmenopausal women had a "relatively small statistically significant, but possibly important, effect [on bone density]" [66]. Women in the exercise group had 0.85 % less bone loss at the spine and 1.03 % less bone loss at the trochanter compared with controls. A meta-analysis by Moayyeri of 13 prospective cohort studies found that regular exercise lowered the risk of osteoporotic hip fracture by 38 % compared to those who did not exercise regularly [67]. The role of regular activity in the prevention of falls in the elderly remains unclear.

Evidence is lacking on the exact exercise program that will optimize bone health and lower the risk of osteoporosis [66]. Generally recommended levels and types of physical activity that promote overall health and chronic disease prevention are likely to also have bone health benefits. Within the 60 min of daily activity recommended for youth in the Physical Activity Guidelines for Americans, it is recommended to include bone-strengthening activities, such as jumping jacks, running, and weightlifting, at least 3 days of the week [68]. A 2004 position stand by the American College of Sports Medicine (ACSM) highlights approaches that may help optimize bone health in youth and adults. The ACSM recommends that, to maximize peak bone mass, children and adolescents need to perform high intensity impact activities, such as plyometrics, gymnastics, and jumping, and moderate intensity strength training 3 or more days per week for 10-20 min [69]. Running, soccer, and basketball are likely beneficial as well. The Canadian Academy of Sports and Exercise Medicine recommends that youth perform at least 60 min of weight-bearing activity per day, mostly at moderate to high intensity, and including 15 min sessions of jumping three times per week [70].

Adult activity is aimed at limiting bone loss. ACSM recommendations focus on weight-bearing endurance activities and activities that include jumping, such as running, walking with periods of jogging, and playing basketball, tennis, and volleyball, 3–5 times per week, in addition to strength training 2–3 times per week, for a total of 30–60 min per day [69]. A moderate or high intensity level is recommended for bone-loading forces [69].

Alzheimer's Disease and Dementia

With the aging of the US population, cognitive decline will become an increasing burden on the nation's health system. In 2000, Alzheimer's Disease (AD) had a prevalence of 4.5 million. By 2050, this is expected to balloon to 13.2 million [71]. Identifying effective approaches to lowering the risk or delaying the onset of AD and dementia is essential, and growing evidence supports the benefits of healthy behaviors in preventing cognitive decline [72, 73].

Though there is some variance in study results, the overall body of evidence points to regular physical activity playing an important role in helping maintain cognition and in preventing both AD and dementia [74–76]. A 2003 meta-analysis of 18 intervention studies found that fitness training improved performance on cognitive tasks by 0.5 standard deviations compared to 0.16 standard deviations in controls [76]. Other reviews have demonstrated reductions in AD and dementia risk with physical activity, but some studies have found no risk reduction from physical activity [74, 75].

Regular activity likely works to reduce cognitive decline though multiple mechanisms, such as the mitigation of coronary heart disease, stroke, and diabetes risk factors, as well as improved blood perfusion and brain volume [75].

Though results of studies to date do not point to clear guidelines for the type and duration of activity that has the greatest cognitive benefits, practical guidelines for the prevention of AD were discussed at the International Conference on Nutrition and the Brain in Washington, DC in July 2013 and subsequently published in 2014 [72]. The recommendations for physical activity included 40 min of brisk walking (or other moderate-intensity aerobic activity) three times per week.

Amounts of Physical Activity for Disease Prevention and Overall Health

Young athletes, by definition, are physically active and many exceed recommended activity guidelines for health and wellness. However, with variances between athletes, sports, and sports programs, it is important to detail current activity guidelines for both adults and youth. Though there are immediate benefits from physical activity, the majority of prevention and health benefits accrue when it is practiced throughout life—in addition to, and often well after, participation in organized

Moderate-intensity physical activity (approximately 3–6 METs)	Vigorous-intensity physical activity (approximately >6 METs)	
Requires a moderate amount of effort and noticeably accelerates the heart rate.	Requires a large amount of effort and causes rapid breathing and a substantial increase in heart rate.	
Examples of moderate-intensity exercise include:	Examples of vigorous-intensity exercise include:	
Brisk walking	Running	
• Dancing	Walking/climbing briskly up a hill	
Gardening	Fast cycling	
Housework and domestic chores	Aerobics	
Traditional hunting and gathering	Fast swimming	
• Active involvement in games and sports with children/walking domestic animals	• Competitive sports and games (e.g., traditional games, football, volleyball, hockey, basketball)	
General building tasks (e.g., roofing, thatching, painting)	Heavy shovelling or digging ditches	
• Carrying/moving moderate loads (<20 kg)	• Carrying/moving heavy loads (>20 kg)	

Table 12.2 Examples of moderate-intensity and vigorous-intensity physical activity

Metabolic Equivalents (METs) are commonly used to express the intensity of physical activities. MET is the ratio of a person's working metabolic rate relative to their resting metabolic rate. One MET is defined as the energy cost of sitting quietly and is equivalent to a caloric consumption of 1 kcal/kg/h. It is estimated that compared with sitting quietly, a person's caloric consumption is three to six times higher when being moderately active (3–6 METs) and more than six times higher when being vigorously active (>6 METs)

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athletics. Certain activities such as tennis, swimming, and cycling can be part of an active lifestyle throughout all stages of life.

Youth should get at least 60 min of moderate intensity or vigorous activity each day, largely from aerobic activities such as brisk walking, running, cycling, and active sports (see Table 12.2). Muscle-strengthening and bone-strengthening activities three or more times per week are an important part of the physical activity routine [10]. It is important to guard against the health risks of the Triad by promoting healthy levels of athletic training combined with a healthy diet that includes adequate calories. Overtraining should also be avoided, and adequate time for healing and tissue repair must be allotted.

Adults should avoid inactivity and work to be regularly active, getting at least 150 min of moderate intensity activity each week. Getting 300 min or more per week has added health benefits and may be necessary for weight control and sustained weight loss. Even greater amounts have further health benefits [10]. Exercise programs should also include strength-training two or more times per week.

Counseling Patients

Though many young athletes will reach or exceed recommended levels of physical activity, many will not, and it is important to counsel them on healthy levels and types of physical activity. The focus should be on choosing enjoyable activities that provide health benefits but also lay a solid foundation for lifelong physical activity. Some young female athletes will also require counseling related to the Triad.

Patient counseling on physical activity can be done in less than 5 min and should be directed at healthy patients as well as those with chronic disease or related conditions. The 5A's brief intervention model, which has been used successfully for smoking cessation, can be modified for physical activity counseling (Table 12.3). It can be helpful to understand the stages of change through which individuals can move forward or back (Trans-theoretical model). Even if patients are not ready to change, providers may help patients move forward from one stage to the next toward the goal of increasing physical activity or taking a healthier approach to physical activity.

General Messages for Patients

Youth

- Aim for 60 min a day of moderate or vigorous activities, such as running, brisk walking, and playing basketball, and get three or more muscle-strengthening and bone-strengthening sessions each week.
- Choose activities you enjoy—whatever gets the body moving.
- Avoid long periods of sitting—even on days that include a lot of physical activity.
- Keep screen time (TV, computer, phone) to under 2 h a day. Less is even better.
- Being on a sports team may not provide all the activity you need to be healthy. Be active on top of sports if need be.
- If you are very active in sports (especially sports that place a priority on being thin), be sure to get enough calories to keep your weight healthy and your bones healthy.

Adults

- Any level of activity is better than none, and it is important to avoid inactivity.
- Try to get at least 150 min of moderate intensity physical activity per week, like brisk walking. More is even better and can help with losing weight or keeping it off.

Table 12.3 5A's for adult physical activity counseling	Table 12.3	5A's for adult	physical activity	counseling
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Ask all patients about their current level of activity.

- Ask about the type, frequency, and duration of exercise, including occupational, household, and leisure-time activities.
- Determine whether their activity level is below, at, or above the minimum recommended levels of 150 min per week of moderate-intensity aerobic activity, with two or more strength-training session per week.
- Document this information in the patient's chart.

Advise all patients to be physically active.

• Deliver a clear, strong, and personalized message advising the patient to get at least 30 min of exercise per day.

Assess the patient's attitudes toward physical activity.

- Understanding the patient's readiness to change can help both the provider and the patient set realistic physical activity goals.
- If the patient is unwilling to consider becoming more active, provide information, and stress the benefits of increased physical activity.
- If the patient is interested in increasing activity, provide motivational support and assistance as described below.
- For patients who are currently active, provide encouragement and assistance, and note that increasing the duration or intensity of activity can yield additional health benefits.

Assist patients who want to become more active.

- Identify potential barriers, and help patients come up with solutions that will work for them.
- For patients who have been inactive for a while, counsel them to initiate activity slowly and then gradually increase.
- Discuss exercise safety and help tailor the mode, intensity, duration, and frequency of activity to meet the needs of individual patients.

Arrange follow-up.

- Follow up is necessary to support successful behavior change, to reinforce messages, and to examine barriers that persist or arise.
- Offer positive feedback and congratulate patients on their successes. Provide additional encouragement and practical advice.
- Reinforcement is important for helping patients maintain their activity levels. Studies suggest that the number, type, and duration of reinforcements are central to the success of behavioral interventions.
- · Help patients reassess their goals and obstacles.
- Try to add three or more muscle-strengthening sessions per week.
- Avoid long periods of sitting—even on days that include a lot of physical activity.
- Keep screen time (TV, computer, phone) to under 2 h a day. Less is even better.
- If starting a new exercise program, start slowly and build from there.
- Choose activities you enjoy-whatever gets the body moving.

Conclusion

Regular activity is one of the most important contributors to wellness, quality of life, and disease prevention (Table 12.1). Relatively modest amounts of activity can lower the risk of heart disease, stroke, diabetes, osteoporosis, dementia, and numerous cancers. Though there are immediate benefits from physical activity, the majority of prevention and health benefits accrue when it is practiced lifelong—in addition to, and often well after, participation in organized athletics.

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References

- Babiss LA, Gangwisch JE. Sports participation as a protective factor against depression and suicidal ideation in adolescents as mediated by self-esteem and social support. J Dev Behav Pediatr. 2009;30(5):376–84.
- 2. Kwan M, et al. Sport participation and alcohol and illicit drug use in adolescents and young adults: a systematic review of longitudinal studies. Addict Behav. 2014;39(3):497–506.
- 3. Women's Sports Foundation. Play fair: a title IX playbook for victory. East Meadow, NY: Women's Sports Foundation; 2009.
- 4. Woodcock J, et al. Non-vigorous physical activity and all-cause mortality: systematic review and meta-analysis of cohort studies. Int J Epidemiol. 2011;40(1):121–38.
- 5. Powell KE, et al. Physical activity and the incidence of coronary heart disease. Annu Rev Public Health. 1987;8:253–87.
- U.S. Department of Health and Human Services. Preventing chronic diseases: investing wisely in health. 2008.
- 7. National Center for Health Statistics. Health, United States, 2013: with special feature on prescription drugs. Hyattsville, MD: National Center for Health Statistics; 2014.
- 8. Fakhouri TH et al. Physical activity in U.S. youth aged 12–15 years, 2012. NCHS Data Brief 2014; (141): 1–8.
- U.S. Department of Health and Human Services. Healthy people 2020. Accessed on August 10, 2013. Available from: http://www.healthypeople.gov/2020/topicsobjectives2020/overview.aspx?topicid=33
- 10. US Department of Health and Human Services. 2008 physical activity guidelines for Americans. 2008.
- World Health Organization. Global recommendations on physical activity and health. Geneva: WHO Press; 2010.
- Centers for Disease Control and Prevention. National diabetes statistics report: estimates of diabetes and its burden in the United States, 2014. Atlanta. GA: Centers for Disease Control and Prevention; 2014.
- D'Adamo E, Caprio S. Type 2 diabetes in youth: epidemiology and pathophysiology. Diabetes Care. 2011;34 Suppl 2:S161–5.
- American Diabetes Association. Economic costs of diabetes in the U.S. in 2012. Diabetes Care. 2013;36(4):1033–46.
- 15. Jeon CY, et al. Physical activity of moderate intensity and risk of type 2 diabetes: a systematic review. Diabetes Care. 2007;30(3):744–52.
- 16. Boule NG, et al. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. JAMA. 2001;286(10):1218–27.

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- Snowling NJ, Hopkins WG. Effects of different modes of exercise training on glucose control and risk factors for complications in type 2 diabetic patients: a meta-analysis. Diabetes Care. 2006;29(11):2518–27.
- Chudyk A, Petrella RJ. Effects of exercise on cardiovascular risk factors in type 2 diabetes: a meta-analysis. Diabetes Care. 2011;34(5):1228–37.
- 19. Umpierre D, et al. Physical activity advice only or structured exercise training and association with HbA1c levels in type 2 diabetes: a systematic review and meta-analysis. JAMA. 2011;305(17):1790–9.
- McAuley KA, et al. Intensive lifestyle changes are necessary to improve insulin sensitivity: a randomized controlled trial. Diabetes Care. 2002;25(3):445–52.
- Jensen TE, Richter EA. Regulation of glucose and glycogen metabolism during and after exercise. J Physiol. 2012;590(Pt 5):1069–76.
- American Diabetes Association. Standards of medical care in diabetes--2013. Diabetes Care. 2013;36 Suppl 1:S11–66.
- 23. Colberg SR, et al. Exercise and type 2 diabetes: the American College of Sports Medicine and the American Diabetes Association: joint position statement. Diabetes Care. 2010;33(12): e147–67.
- 24. Buse JB, et al. Primary prevention of cardiovascular diseases in people with diabetes mellitus: a scientific statement from the American Heart Association and the American Diabetes Association. Circulation. 2007;115(1):114–26.
- American Heart Association. Statistical fact sheet 2014 Update: women & cardiovascular disease. 2014. Accessed on Jul 15, 2014. Available from: http://www.heart.org/idc/groups/ heart-public/@wcm/@sop/@smd/documents/downloadable/ucm_462030.pdf
- 26. Mosca L, et al. Effectiveness-based guidelines for the prevention of cardiovascular disease in women--2011 update: a guideline from the American Heart Association. Circulation. 2011;123(11):1243–62.
- May AL, Kuklina EV, Yoon PW. Prevalence of cardiovascular disease risk factors among US adolescents, 1999-2008. Pediatrics. 2012;129(6):1035–41.
- Lee IM, et al. Physical activity and coronary heart disease in women: is "no pain, no gain" passe? JAMA. 2001;285(11):1447–54.
- 29. Manson JE, et al. A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women. N Engl J Med. 1999;341(9):650–8.
- 30. Hu FB, et al. Physical activity and risk of stroke in women. JAMA. 2000;283(22):2961-7.
- 31. Fletcher GF, et al. Statement on exercise: benefits and recommendations for physical activity programs for all Americans. A statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. Circulation. 1996;94(4):857–62.
- 32. Fletcher GF, et al. Exercise standards. A statement for healthcare professionals from the American Heart Association. Writing Group. Circulation. 1995;91(2):580–615.
- 33. Smith JK, et al. Long-term exercise and atherogenic activity of blood mononuclear cells in persons at risk of developing ischemic heart disease. JAMA. 1999;281(18):1722–7.
- 34. American Heart Association. American Heart Association recommendations for physical activity in adults. Dallas, TX: American Heart Association; 2013.
- Maron BJ, et al. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation. 2009;119(8):1085–92.
- Asif IM, Rao AL, Drezner JA. Sudden cardiac death in young athletes: what is the role of screening? Curr Opin Cardiol. 2013;28(1):55–62.
- 37. Maron BJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. Circulation. 2007;115(12): 1643–55.
- 38. American heart Association. Preparticipation cardiovascular screening of young competitive athletes: policy guidance. 2012. Accessed on Jul 16, 2014. Available from: http://www.heart. org/idc/groups/ahaecc-public/@wcm/@adv/documents/downloadable/ucm_443945.pdf

- 39. American Cancer Society. Breast cancer facts & figures 2013-2014. Atlanta, GA: American Cancer Society; 2013.
- Lee IM. Physical activity and cancer prevention--data from epidemiologic studies. Med Sci Sports Exerc. 2003;35(11):1823–7.
- Wolin KY, Yan Y, Colditz GA. Physical activity and risk of colon adenoma: a meta-analysis. Br J Cancer. 2011;104(5):882–5.
- 42. Friedenreich C, et al. Physical activity and risk of colon and rectal cancers: the European prospective investigation into cancer and nutrition. Cancer Epidemiol Biomarkers Prev. 2006;15(12):2398–407.
- 43. World Cancer Research Fund/American Institute for Cancer Research. Continuous update project report. Food, nutrition, physical activity, and the prevention of colorectal cancer. Washington, DC: American Institute for Cancer Research; 2011.
- 44. International Agency for Research on Cancer. GLOBOCAN 2012: estimated cancer incidence, mortality and prevalence worldwide in 2012 - breast cancer. 2012. Accessed on Jul 31, 2014. Available from: http://globocan.iarc.fr/Pages/fact_sheets_cancer.aspx
- 45. Friedenreich CM. Physical activity and breast cancer: review of the epidemiologic evidence and biologic mechanisms. Recent Results Cancer Res. 2011;188:125–39.
- 46. Norat T, Chan D, Lau R, Aune D, Viera R. The Associations Between Food, Nutrition and Physical Activity and the Risk of Breast Cancer. WCRF/AICR Systematic Literature Review Continuous Update Project Report. London: World Cancer Research Fund/American Institute for Cancer Research; 2010.
- 47. Bernstein L, et al. Lifetime recreational exercise activity and breast cancer risk among black women and white women. J Natl Cancer Inst. 2005;97(22):1671–9.
- Schmidt ME, et al. Physical activity and postmenopausal breast cancer: effect modification by breast cancer subtypes and effective periods in life. Cancer Epidemiol Biomarkers Prev. 2008;17(12):3402–10.
- Eliassen AH, et al. Physical activity and risk of breast cancer among postmenopausal women. Arch Intern Med. 2010;170(19):1758–64.
- 50. Maruti SS, et al. A prospective study of age-specific physical activity and premenopausal breast cancer. J Natl Cancer Inst. 2008;100(10):728–37.
- 51. IARC. IARC handbooks of cancer prevention. Weight control and physical activity, vol. 6. Lyon: IARC; 2002.
- 52. World Cancer Research Fund/American Institute for Cancer Research. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Washington, DC: American Institute for Cancer Research; 2007.
- 53. Berkey CS, et al. Dairy consumption and female height growth: prospective cohort study. Cancer Epidemiol Biomarkers Prev. 2009;18(6):1881–7.
- 54. Leitzmann MF, et al. Prospective study of physical activity and lung cancer by histologic type in current, former, and never smokers. Am J Epidemiol. 2009;169(5):542–53.
- 55. McTiernan A, et al. Effect of exercise on serum estrogens in postmenopausal women: a 12-month randomized clinical trial. Cancer Res. 2004;64(8):2923–8.
- 56. McTiernan A, et al. Effect of exercise on serum androgens in postmenopausal women: a 12-month randomized clinical trial. Cancer Epidemiol Biomarkers Prev. 2004;13(7): 1099–105.
- 57. Looker AC et al. Osteoporosis or low bone mass at the femur neck or lumbar spine in older adults: United States, 2005–2008. NCHS Data Brief 2012; (93): 1–8.
- 58. Thein-Nissenbaum J. Long term consequences of the female athlete triad. Maturitas. 2013;75(2):107–12.
- 59. Pocock NA, et al. Genetic determinants of bone mass in adults. A twin study. J Clin Invest. 1987;80(3):706–10.
- 60. Hawker GA, et al. A clinical prediction rule to identify premenopausal women with low bone mass. Osteoporos Int. 2002;13(5):400–6.
- 61. Borer KT. Physical activity in the prevention and amelioration of osteoporosis in women: interaction of mechanical, hormonal and dietary factors. Sports Med. 2005;35(9):779–830.

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- 62. Foley S, et al. Measures of childhood fitness and body mass index are associated with bone mass in adulthood: a 20-year prospective study. J Bone Miner Res. 2008;23(7):994–1001.
- 63. De Souza MJ, et al. 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad: 1st International Conference held in San Francisco, California, May 2012 and 2nd International Conference held in Indianapolis, Indiana, May 2013. Br J Sports Med. 2014;48(4):289.
- Nattiv A, et al. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 2007;39(10):1867–82.
- 65. Duque G, Troen BR. Chapter 117. Osteoporosis. In: Hazzard WR, Halter JB, editors. Hazzard's geriatric medicine and gerontology. New York, NY: McGraw-Hill Medical Pub. Division; 2009. p. xxviii, 1634 p., 12 p. of plates.
- 66. Howe TE et al. Exercise for preventing and treating osteoporosis in postmenopausal women. Cochrane Database Syst Rev 2011; (7): CD000333.
- 67. Moayyeri A. The association between physical activity and osteoporotic fractures: a review of the evidence and implications for future research. Ann Epidemiol. 2008;18(11):827–35.
- 68. United States. Department of Health and Human Services. 2008 physical activity guidelines for Americans be active, healthy, and happy! Washington, DC: U.S. Department of Health and Human Services; 2008.
- 69. Kohrt WM, et al. American College of Sports Medicine Position Stand: physical activity and bone health. Med Sci Sports Exerc. 2004;36(11):1985–96.
- Fletcher JA. Canadian Academy of Sport and Exercise Medicine position statement: osteoporosis and exercise. Clin J Sport Med. 2013;23(5):333–8.
- Hebert LE, et al. Alzheimer disease in the US population: prevalence estimates using the 2000 census. Arch Neurol. 2003;60(8):1119–22.
- Barnard ND, et al. Dietary and lifestyle guidelines for the prevention of Alzheimer's disease. Neurobiol Aging. 2014;35(S2):S74–8.
- Polidori MC, Nelles G, Pientka L. Prevention of dementia: focus on lifestyle. Int J Alzheimers Dis. 2010;2010, 393579.
- Rolland Y, Abellan v K G, Vellas B. Physical activity and Alzheimer's disease: from prevention to therapeutic perspectives. J Am Med Dir Assoc. 2008;9(6):390–405.
- 75. Harrington M et al. The AlzRisk Database. Alzheimer Research Forum. 2012 Accessed on Jul 18, 2014. Available at: http://www.alzrisk.org
- Colcombe S, Kramer AF. Fitness effects on the cognitive function of older adults: a meta-analytic study. Psychol Sci. 2003;14(2):125–30.

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