Chapter 8

The exercising female

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CHAPTER CONTENTS

LEARNING OBJECTIVES

After studying this chapter, you should be able to:

1. Understand the basic physiological fluctuations associated with the menstrual cycle, menopause and pregnancy.
2. Evaluate how these fluctuations might influence physical performance and the physiological responses to performance.
3. Understand and critique the female athlete triad.
4. Understand how disordered eating and/or aberrations in menstrual cycle functioning influence the long-term risk of osteoporosis.
5. Analyse the increased risk of cardiovascular disease associated with being sedentary in postmenopausal women.
6. Reflect upon how the physiological alterations during pregnancy impact upon the potential to exercise.
7. Describe the considerations that need to be given to exercise prescription for premenopausal, postmenopausal and pregnant women.

INTRODUCTION

At first sight, the immediate physiological responses of female exercisers normally differ from those of their male counterparts only in a manner dictated by body size and organ differences. However, the cyclical nature of the menstrual cycle on a monthly and lifespan basis provides some special considerations for the exercising female. The rhythmic nature of reproductive hormonal fluctuation has been seen to influence both performance (e.g. muscle strength, time to fatigue) and physiological responses to exercise (e.g. blood lactate production, carbohydrate metabolism, minute ventilation, heart rate); however, these aspects will not be covered in this chapter (for review see Constantini et al 2005). Of more importance in relation to the exercising female and exercise prescription are the considerations that need to be given to low and high fit pre- and postmenopausal women in order to avoid the 'female athlete triad' of disordered eating, osteoporosis and amenorrhoea. In addition, of course, of great relevance to the exercising female are the special considerations pertinent to exercise training during and following pregnancy. This chapter will review the reproductive physiology of the female menstrual cycle and pregnancy and the interrelationship between reproductive hormone fluctuations, physical activity and health.

PHYSIOLOGY OF THE EXERCISING FEMALE

The menstrual cycle

The female menstrual cycle is characterized by cyclical fluctuations in the anterior pituitary hormones (the gonadotrophins), follicle-stimulating hormone (FSH) and luteinizing hormone (LH), and the ovarian steroid hormones oestrogen and progesterone (Fig. 8.1). Each menstrual cycle lasts approximately 26 to 35 days and is simplistically divided into two phases by the mid-cycle occurrence of ovulation. The first half of the cycle is characterized by the growth of a primordial follicle within the ovary and higher concentrations of FSH and LH. Towards the end of the phase oestrogen reaches its peak concentrations. This phase, referred to as the *follicular phase*, begins on the first day of menstrual bleeding (day one) and continues until ovulation. Ovulation, or the release of the ovum into the fallopian tubes, occurs at approximately day 14 and continues until the next menstrual blood loss. The second half of the cycle is characterized by the presence of both oestrogen and progesterone and is referred to as the *luteal phase*. In addition, the 4 to 5 days of menstrual blood loss are referred to as *menses*, whilst the 72 hours prior to menses are known as *premenses*.

The rhythmic nature of the menstrual cycle is controlled by a series of feedback loops between the hypothalamic–pituitary axis and the ovaries (Fig. 8.2). The hypothalamus and anterior pituitary glands (the hypothalamic–pituitary axis) communicate principally through the portal blood vessels that run between them. At the onset of puberty, or *menarche*, the existing pulsatile release of gonadotrophin-releasing hormone (GnRH) from the median eminence of the hypothalamus increases in magnitude and rhythm. This maturation in GnRH cycling enhances the sensitivity of the anterior pituitary to the gonadotrophins, produces the first period in female adolescents (menarche) and then controls the cycle on a monthly basis. The average age of menarche in the UK is approximately 12 years. Each month the pulsatile release of GnRH causes the synthesis and release of FSH and LH from the anterior pituitary gland. FSH released into the circulation targets receptor cells in the ovaries that respond by releasing 17-β-oestradiol

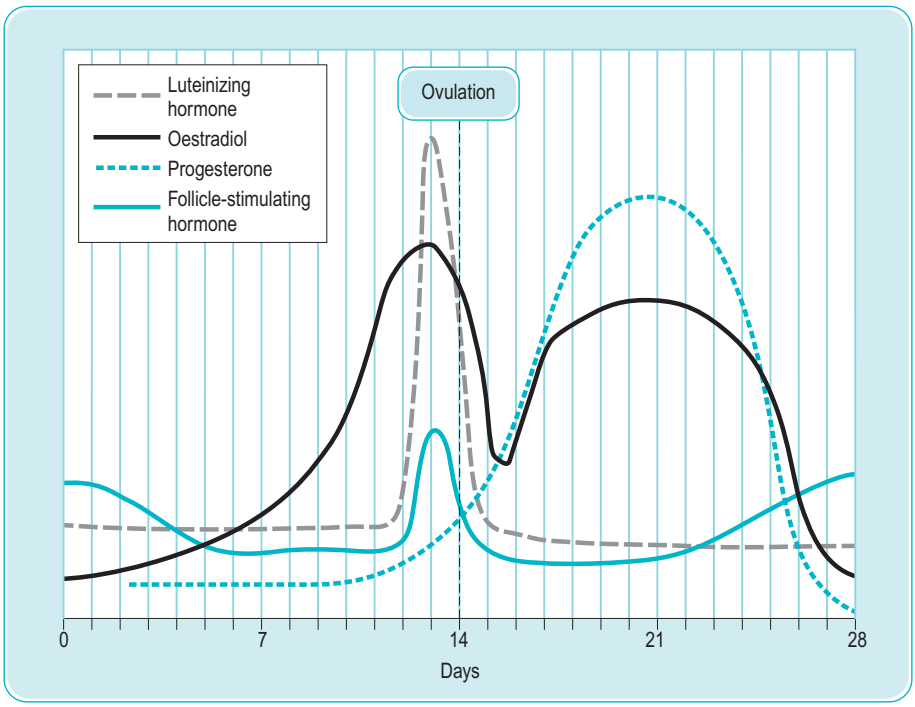

Figure 8.1 The menstrual cycle.

into the circulation. Circulating oestradiol concentrations are monitored via the hypothalamic–pituitary axis via a positive feedback loop leading to a mid-cycle surge in LH that is responsible for the release of the ovum from the ovary. At this stage the remaining cells within the ovary, the *corpus luteum*, begin to secrete 17-β-oestradiol and progesterone.

The circulating concentrations of oestradiol and progesterone at this stage of the cycle act via a negative feedback loop to the hypothalamic–pituitary axis to inhibit the release of FSH and LH from the anterior pituitary. However, the corpus luteum is dependent upon LH for its survival, so once LH is suppressed the corpus luteum begins to involute and die. As oestradiol and progesterone concentrations thus begin to decrease, the feedback loop inhibiting FSH and LH release from the anterior pituitary is removed, the lining of the womb (the endometrium) is shed in menses, and the cycle begins again. It is noteworthy that these feedback loops work in exactly the same way when concentrations of oestrogen and progesterone are elevated through consumption of the oral contraceptive pill, or by contraceptive implants.

The menstrual cycle normally continues from menarche (approximately age 11–14 years) to the climacteric or menopause (approximately age 53–56 years), during which time females have exposure to high levels of oestrogen for approximately 14–16 days each month. Females experiencing regular and healthy menstrual cycles are referred to as being *eumenorrhoeic*, whilst those experiencing irregular menstrual cycles are referred to as being *oligomenorrhoeic*. A loss of menstrual cycles for a period of 6 months or more following successful menarche is referred to as *secondary*

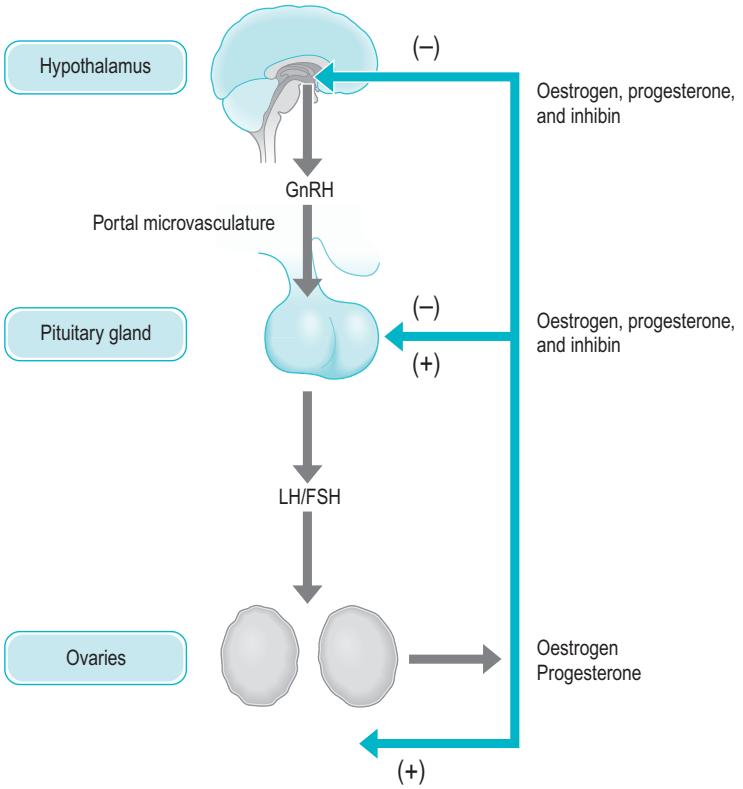

Figure 8.2 The rhythmic nature of the menstrual cycle.

amenorrhoea. If this loss is non-pathological and deemed to be associated with participation in high-intensity or high-volume physical exercise, it has been referred to as *athletic amenorrhoea*. Some women experience painful menses usually due to cramps initiated by high concentrations of prostaglandins. This disorder is known as *dysmenorrhoea* and differs from the little understood syndrome of premenstrual tension, or *premenstrual syndrome* (PMS), that occurs in the few days prior to menses. The physiology of the menstrual cycle can be reviewed in Guyton & Hall (2006).

The menopause

The cessation of menstruation occurs primarily because of a decline in the number of primordial follicles within the ovary. Initially this results in increasing failure to ovulate and a compensatory increase in gonadotrophin secretion. Despite this effort to maintain hormonal concentrations there is a gradual decline in hormone production and the falling oestrogen levels commonly result in hot flushes and night sweats (vasomotor symptoms), sleep disturbance and vaginal dryness. Levels of the steroid hormones begin to fall approximately 5–10 years prior to the menopause. During this time the luteal phase of the cycle may shorten, menses may become irregular and menstrual blood flow may be lighter. Eventually the cycle will become anovulatory (lack of ovulation), as follicular maturation becomes inadequate to trigger ovulation.

The physiology of the menopause can be reviewed in Guyton & Hall (2006) and may have some impact upon cardiovascular and bone health in sedentary women.

Pregnancy

If the ovum released into the fallopian tubes following ovulation is fertilized by ejaculated sperm and then is successfully implanted into the endometrial lining, the menstrual cycle will cease throughout pregnancy. The corpus luteum is maintained for the first 3 to 4 months of pregnancy and continues to secrete oestrogen and progesterone, an action that will inhibit further secretion of the gonadotrophins. Following this time the high levels of these hormones required throughout pregnancy are produced by the placenta. The chorion of the placenta also secretes human chorionic gonadotrophin (hCG). This hormone provides a stimulus for the continued production of progesterone from the corpus luteum, which is necessary for the continued connection of the embryo and fetus to the endometrium. The placenta produces oestrogen from about 4 weeks into pregnancy, and takes over from the corpus luteum in the production of both oestrogen and progesterone from approximately 4 months. At this stage the production of hCG is greatly reduced.

Both the placenta and the ovaries also secrete relaxin. This hormone relaxes the symphysis pubis and the ligaments of the sacroiliac and sacrococcygeal joints in order to aid delivery. The physiology of pregnancy can be reviewed in Guyton & Hall (2006).

SPECIAL CONSIDERATIONS FOR THE EXERCISING FEMALE

The female athlete triad

The 'female athlete triad' describes a model (Fig. 8.3) utilized to identify exercising females presenting with one or more of three disorders – amenorrhoea, disordered eating and osteoporosis. These disorders are interlinked and can seriously impact upon exercise performance and long-term health. A female may enter the triad most likely displaying amenorrhoea or disordered eating, as osteoporosis is more likely to be a consequence of the former disorders. Importantly, identifying an exercise performer as having entered the triad implies that the cause of the identified disorder is related to participation in high-volume or high-intensity physical activity, and not to another departure from normality (a different pathology, or pregnancy).

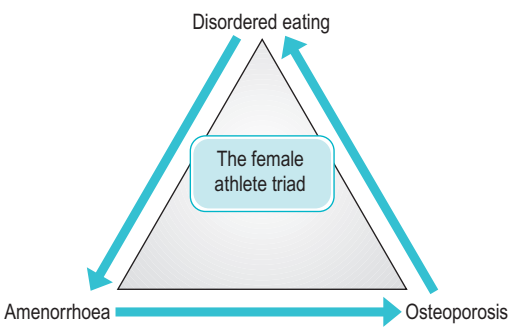

Figure 8.3 The female athlete triad.

In the general population the incidence of secondary amenorrhoea is approximately 5%. However, in a population of female exercise participants the incidence has been reported to be anywhere between 10% and 80%. It is important to note that the female athlete triad is not only found in elite female athletes. On the contrary, disorders which are part of the triad have been identified in club level athletes and in exercising females who utilize exercise training as a means to control body mass (Torstveit & Sundgot-Borgen 2005). The loss of oestrogen production in athletic or secondary amenorrhoea has been associated with infertility, an increased risk of osteopenia and/or osteoporosis plus related stress fractures, and an increased risk of cardiovascular disease. Whereas infertility is reversible on reattainment of menstruation, the participant must understand that bone loss and disease risk may be controllable, but are not fully retrievable.

Athletic amenorrhoea

The aetiology of athletic amenorrhoea appears to be multifactorial in nature. Primarily, amenorrhoea is caused by down-regulation of the hypothalamic–pituitary axis. The pulse frequency of GnRH is decreased whilst its amplitude is increased (Warren & Shantha 2000) such that the pulsatile release of LH from the anterior pituitary is inhibited. Altered LH pulsatility may impact upon the cycle by inducing luteal phase suppression. In effect, the lowered production of LH means that the corpus luteum involutes earlier, oestrogen and progesterone production decrease and menses occurs at an earlier point in the cycle (shortened luteal phase). A healthy luteal phase should have duration of greater than 10 days. The complete down-regulation of LH secretion from the anterior pituitary results in failure to develop a dominant follicle within the ovary, and thus anovulation (lack of ovulation). The pathophysiology of athletic amenorrhoea can be reviewed in Redman & Loucks (2005).

A popular theory explaining the aetiology of amenorrhoea has been that females who lose weight through exercise, and/or participate in sports where aesthetics require a low body mass (e.g. ballet, gymnastics), display an exceedingly low percentage body fat, which in turn leads to amenorrhoea. Historically, it was felt that 17% body fat was required to initiate menarche, whilst 22% body fat was required to maintain menstruation (Frisch & McArthur 1974). However, this theory has been thoroughly refuted, although the identification of a low percentage body fat does serve to signpost that athletic amenorrhoea may be present. In other words low body fat may be correlated with the prevalence of amenorrhoea; however, it is not the cause of amenorrhoea. Other theories have reported down-regulation of the hypothalamic–pituitary axis as a result of elevated β-endorphins following high duration exercise, and indeed administration of a β-endorphin antagonist (naloxone) has been seen to reverse amenorrhoea (Szabo et al 1987). The GnRH pulse generator may also be suppressed by the hypothalamic–adrenal axis as increased levels of corticotrophin-releasing factor (CRF) and adrenocorticotrophic hormone (ACTH) have both been seen to decrease GnRH pulse frequency. Certainly ACTH and cortisol are elevated during exercise and ACTH and cortisol rhythms have been seen to be dysfunctional in amenorrhoeic athletes, probably as a result of sustained (not rhythmic) activation of CRF (De Souza et al 1994). Thus one cause of down-regulation of the hypothalamic–pituitary axis may be stress-induced by competitive action.

Bullen et al (1984) attempted to initiate down-regulation of the hypothalamic–pituitary axis in a prospective study that involved women endurance training for 4.5 hours per week at 85% maximal heart rate (approximately 70% $\dot{V}O_2$max) over

2 to 3 months. Although diminished urinary oestradiol concentrations were found in half of the group participants, no change in cycle length or ovulatory capacity occurred. This and similar studies of the day concluded that moderate-intensity exercise was not a great enough stimulus to induce down-regulation of the axis. Bullen et al (1985) randomly assigned 28 college women to either a weight-loss training group, or a weight maintaining group for a study that covered two consecutive menstrual cycles. Weight loss was limited to 0.45 kg/week and training consisted of running 6.4 km/day during the first week, increasing to 16 km/day by the fifth to eighth week at heart rates corresponding to 70–80% $\dot{V}O_2$max. In addition, all participants engaged in 3.5 hours of moderate-intensity sports activity daily. Forty-four per cent of the participants proceeded from abnormal luteal function in cycle 1 to amenorrhoea in cycle 2, with a significantly greater number presenting as amenorrhoeic in the weight-loss group. It thus appeared that more vigorous intensity and/or higher volume physical activity was more likely to induce amenorrhoea.

Interestingly, Bullen et al (1985) did not match energy balance (energy consumption less energy utilization) between the two groups of participants. In fact the weight-loss group necessarily consumed many fewer calories than their energy expenditure. Historically, researchers in this area began to ponder the impact of a negative energy balance upon the hypothalamic–pituitary axis. It was contended that a 'metabolic arrest' was induced by a negative energy balance aimed at conserving energy (Bonen 1994). Indeed, maintenance of the luteal phase of the cycle requires a basal metabolic rate of between 0.7 and 1.0 kcal/min more than that of the follicular phase (Solomon et al 1982). Certainly some hibernating animals are seen to conserve energy during hibernation by temporarily 'switching off' the reproductive cycle. Given the prevalence of amenorrhoea in females with anorexia nervosa, it was contended that the down-regulation of the hypothalamic–pituitary axis may thus be related to energy balance.

Loucks et al (1998, 2003) have conducted well-controlled prospective studies to evaluate the impact of energy availability and exercise stress upon LH pulsatility. Eumenorrhoeic women participating in these studies were admitted to a hospital environment where exercise and energy intake were closely controlled. Energy availability was standardized per kilogram of lean body mass (kgLBM) by controlling nutritional intake and energy expenditure through exercise at an energy cost of 30 kcal·kgLBM^{-1}. Thus energy availability was either balanced at 45 kcal·kgLBM^{-1} or deprived at 10 kcal·kgLBM^{-1}. LH pulsatility, measured via repetitive 10-minute blood sampling, was suppressed whether energy availability was reduced by dietary restriction alone, or by energy expenditure alone. Supplementing the diet to replace the energy cost of exercise prevented the disruption of LH pulsatility. Loucks & Thuma (2003) expanded these data by investigating the dose–response effect of restricted energy availability on LH pulsatility in exercising women. Within 5 days of the start of exercising, LH pulsatility was disrupted below a threshold of energy availability between 20 and 30 kcal·kgLBM^{-1}·d^{-1}, a response that was most extreme in women who already displayed shortened luteal phases.

It would thus appear that maintenance of a healthy menstrual cycle in exercising women is entirely possible as long as energy availability is maintained. However, in the women studied by Loucks & Thuma (2003), normal LH pulsatility was demonstrated in women whose energy availability was restricted by 33% to 30 kcal·kgLBM^{-1}·d^{-1}. Loucks & Thuma thus contend that 30 kcal·kgLBM^{-1}·d^{-1} appears to be sufficient energy availability to preserve normal reproductive function, and that screening for athletes/participants with decreased luteal phase lengths will

identify females who are at greatest risk of developing amenorrhoea if energy intake is further restricted.

Disordered eating

Of course, restricted energy availability is brought about by participating in high training intensities or volumes whilst restricting dietary intake. It should be noted that although some female athletes may partake in unhealthy dietary practices such as using laxatives etc., the second corner of the 'female athlete triad' is termed 'disordered eating' and not 'eating disorders'. Disordered eating ranges from abnormal eating behaviours to clinical eating disorders such as anorexia nervosa and bulimia nervosa. Clinical eating disorders are identified using the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV: APA 1994) criteria, whilst disordered eating can be identified with the Eating Disorder Inventory Drive for Thinness and Body Dissatisfaction subscales (Espelage et al 2003). Exercising females may be driven to achieve unrealistic and unhealthy body mass through energy restriction, restriction of high fat or high carbohydrate foods, purging, use of diet pills, laxatives or diuretics and over-exercising, and may achieve high scores on the Drive for Thinness and Body Dissatisfaction scales. To be classified as having an eating disorder the participant must show a disturbance of eating habits or weight control behaviour that could result in impairment of physical health or psychosocial functioning (Fairburn & Harrison 2003).

The causes of disordered eating are complicated but, in the population at large, might include social, psychological and physiological factors. In female exercise participants additional factors include expectations of athletic perfection and a belief that low body mass induces better performance. The prevalence of disordered eating in athletic female populations ranges from 10% to 32%, although establishing a correct figure is hampered by the secretive nature of disordered eating and the differing diagnostic criteria used throughout the literature (Sudi et al 2004). The prevalence of pathological eating disorders in exercising females is quite low (approximately 1–3%). However, the markers of nutritional deficiency found in amenorrhoeic female exercise participants, such as increased insulin-like growth factor binding protein-1, cortisol and growth hormone (GH) and low tri-iodothyronine (T_3) and insulin-like growth factor-1, are very similar to the levels found in anorexic patients (Laughlin et al 1998). Additionally, the low levels of leptin found in these women are due to low energy availability, and not the stress of exercise itself.

Osteoporosis

For the exercising female who becomes amenorrhoeic the risk of stress fractures and future osteoporosis significantly increases. The risk of osteoporosis has been found to be related to the life-long exposure to oestrogen, and so given the monthly production of oestrogen that should occur between the years of menarche and the menopause, long periods of amenorrhoea will significantly decrease life-long exposure.

In both sexes oestrogen acts largely at endosteal sites (the inner surface of bone), whereas testosterone in the male also acts upon periosteal sites (the fibrous membranes covering the bone) leading to a larger cortical apposition. After the third decade, bone mass tends to be stable for some 15 years, following which (after approximately 45 years of age) an age-related bone loss ensues. The rate of bone loss

for the female will increase during the menopausal years and thus, in order to ensure an adequate bone mass in older years, females are best served by reaching a good peak in bone mass, maintaining a eumenorrhoeic menstrual cycle and having a later menopause. See Borer (2005) for more detailed review.

The hormone oestrogen acts to inhibit bone resorption and thus decreases the rate of bone turnover. Any loss of oestrogen, such as that occurring with down-regulation of the hypothalamic–pituitary axis or with the menopause, leads to resorption being favoured and a subsequent loss of bone mineral. The mechanism by which oestrogen exerts its action is an inhibition of production of the cytokines, interleukin-1 (IL-1) and -6 (IL-6), and tumour necrosis factor alpha (TNF-α) in the marrow. These cytokines induce osteoclastogenesis when unchecked, and thus elevate the rate of bone resorption. Oestrogen also acts to stimulate production of the resorption inhibitor tumour growth factor beta (TGF-β) (for review see Riggs 2000). For amenorrhoeic female exercise participants who are displaying a low energy availability, suppression of the bone trophic hormones IGF-1 and T_3 can also lead to inadequate bone formation.

For the premenopausal female exercise participant who becomes amenorrhoeic, the loss of oestrogen has been seen to increase the prevalence of stress-induced fractures and scoliosis, and these conditions are highly associated with low bone mass. Indeed, amenorrhoeic female athletes have been seen to have 10–20% lower bone mineral densities than their eumenorrhoeic counterparts, with rates of loss as high as 3–5% per year in the first year of amenorrhoea. Importantly, the mechanical effect of physical activity upon bone mineral density still exists in amenorrhoeic exercising females and amenorrhoeic athletes have been seen to have a greater bone mineral density than non-exercising amenorrhoeic women (see also Chapter 7). Resumption of menses has been seen to increase bone mineral density by an initial 6–9.7% per annum, but this rate of increase slowed and then ceased following the first year of resumption (see Warren & Perlroth 2001 for review). In the long term, even after regaining menses and increasing bone mineral density, amenorrhoeic participants remain significantly below normal controls when bone mineral density is examined.

Osteoporosis has existed as the third point of the triangle since the triangle's inception in 1993. Osteoporosis is defined as a bone mineral density that is more than 2.5 SD below the mean of young adults, whilst a bone mineral density between 1 and 2.5 SD below this mean defines osteopenia. Khan et al (2002) re-evaluated a number of journal articles in the literature that had reported the prevalence of osteoporosis in premenopausal exercising females and females diagnosed with anorexia nervosa using dual X-ray absorptiometry in the lumbar spine. The prevalence of osteoporosis reported within the literature was no higher than 13% in the exercising females and 21% in the anorexic females. However, when reanalysed using the above strict SD criteria for osteoporosis and osteopenia, all of the females originally classified as having osteoporosis were reclassified as having osteopenia, mild osteopenia or as having normal bone mineral density.

It would thus appear that osteopenia would be better placed within the female athlete triad and indeed Khan et al (2002) call for osteoporosis to be replaced by osteopenia/osteoporosis as the third corner of the triad. This would perhaps mean that a greater prevalence of bone mass irregularities would be seen in exercising females, and thus the triad would potentially have better clinical utility. What is clear is that female exercise participants who present with osteopenia are at a greater risk of developing osteoporosis later in life than a participant with normal bone mass. Physical activity and optimal nutrition are the key factors to managing osteopenia, and as both of these factors are related to amenorrhoea and disordered

eating, defining the triad as a continuum of amenorrhoea, disordered eating and osteopenia/osteoporosis might well be more appropriate.

Physical activity guidelines for the female athlete triad

It is imperative that the existence of any corner of the female athlete triad is identified using valid tools. As previously discussed, evaluation must utilize correct diagnostic criteria and these must be undertaken by medical personnel. The diagnosis of athletic amenorrhoea must rule out other pathology and pregnancy, and should be based upon a detailed medical history, a physical examination, a pelvic examination and laboratory tests. Disordered eating and/or eating disorders should be diagnosed with a full medical history, use of valid diagnostic criteria from the DSM (APA 1994) and other questionnaires and by interview with a psychologist. Bone mineral density should be measured using valid tools, whilst other endocrinological disorders should be identified via laboratory tests. It should not be the role of the coach, trainer or selector to clinically identify an aspect of the triad. However, it is extremely important that coaches, trainers, selectors and parents/spouses are aware of (a) the risk factors associated with entering the female athlete triad, and (b) the signs and symptoms of disordered eating/eating disorders (pathological eating behaviours, mood changes, fatigue, constipation, light-headedness, etc.), amenorrhoea (loss of menstruation, shortened luteal phase) and low bone mass (recurrent stress fractures). For a detailed review of early identification see Waldrop (2005).

Treatment of the exercising female again depends upon the extent to which she has progressed down the continuum of the triad. Assuming low bone mass is present the reader is referred to Bassey & Dinan (2001) and Chapter 6 of the present volume for guidelines on treatment and exercise modification. Management of the female in the triad as a whole centres on changing both eating and exercise behaviour. Changing eating behaviour must be managed with psychological intervention, perhaps using cognitive behaviour change or a 'stages of change' approach. The exercise intervention requires a complete analysis of training history, training intensity, duration and frequency. It is recommended that training volume is decreased initially by 10%, and that this decrease might be either in intensity or in duration. The optimal requirement is a gain of 2–3 kg in body mass. The recommended approach to this management is that a stabilization period of 3 months might involve reduction of exercise volume, whilst following this time an evaluation and counselling period can explore exercise behaviour and knowledge (Prior et al 1992). Certainly many exercising females believe that low body mass will enhance performance, even in the light of low nutritional status and power-to-weight ratio; the endeavour must be to help the triad sufferer adjust her perspective on these factors.

During the evaluation and counselling stage of treatment it may be advisable to increase calcium intake, utilizing 1500 mg/day calcium + 400–800 IU/day vitamin D. If stress fractures are present then the exercise regimen will need to be changed to one using non-weight-bearing activities. Overall, energy availability will also need to be assessed, remembering that athletes can be in a negative energy balance and yet successfully maintain menstruation (and thus oestrogen levels) as long as they do not drop below a threshold of 30 $kcal \cdot kgLBM^{-1} \cdot d^{-1}$ (Loucks & Thuma 2003). It is recommended that the participant should be reassessed every 3–6 months, whilst bone density examinations should be repeated at intervals of 1–2 years.

For the extra complications in team management of the female athlete triad, the reader is referred to the detailed analysis by Joy et al (1997a, 1997b).

THE PREMENSTRUAL SYNDROME AND DYSMENORRHOEA

The prevalence of the premenstrual syndrome (PMS) ranges from 30% to 63% of populations across the age span, whilst premenstrual mood changes have been reported in up to 97% of women. The validity of these numbers is difficult to ascertain given the wide-ranging diagnostic criteria used throughout the literature. Clinical diagnosis of PMS requires a prospective self-report of at least one of six affective complaints (including depression, anxiety and irritability) and one of four somatic complaints (breast tenderness, abdominal bloating, headache and swelling of extremities) in at least two cycles (Warren & Shanghold 1997). The aetiology of these so-called 'moliminal' symptoms is unclear, but it is thought to relate to imbalances in oestrogen and progesterone concentrations late in the cycle, potentially causing a disturbance in calcium regulation (Bertone-Johnson et al 2005). Certainly calcium supplementation has been found to relieve symptoms, whilst serotonin reuptake inhibitors have also been observed to aid treatment.

The evidence suggesting that exercise might alleviate PMS is contentious. Timonen & Procope (1971) reported that active university students had fewer premenstrual emotional symptoms than their less active counterparts, but that self-reports of moliminal symptoms did not differ between the two groups. However, in a comparison of sedentary women who took up regular running, runners training for a marathon and sedentary women, Prior et al (1986) reported decreased breast tenderness, fluid retention and stress in the new runners, decreased breast tenderness, fluid retention, depression and anxiety in the marathon runners and no changes in the non-exercise controls. Lustyk et al (2004) suggested that women with the worst PMS symptoms may reduce these by exercising, while women who exercise often or never do not associate exercise with their symptoms.

The prevalence of dysmenorrhoea (pain accompanying menstruation) in the general population ranges from 47% to 80%, and 58% of these sufferers also complain of PMS. Symptoms include lower abdominal pain that may radiate to the lower back or legs, headache, nausea and vomiting. The aetiology of dysmenorrhoea is again unclear; however, there is a firm belief that raised prostaglandin concentrations lead to uterine contractions and ischaemia (Tzafettas 2006). Certainly prostaglandin inhibitors and oral contraceptives providing progesterone to inhibit prostaglandin synthesis in endometrial cells have been found to alleviate symptoms.

Attempting to identify whether exercise participation might decrease the incidence of dysmenorrhoea is difficult given that an exercise-induced change in mood state might very well influence the experience of and reporting of painful sensations. The effect of physical activity upon the severity of dysmenorrhoea certainly requires more investigation.

PREGNANCY

The physiological alterations experienced throughout pregnancy are driven by the need to support both maternal and fetal well-being and functioning. Maternal system alterations are controlled by the changes in gestational hormones previously reviewed. In the first two trimesters of pregnancy, increased concentrations of oestrogen and progesterone promote β-cell hyperplasia and thus greater insulin sensitivity and secretion. This in turn promotes an increased maternal adiposity until late in gestation when the fetal energy requirements tend towards an adipose tissue decrease (Boden 1996). Weight gain in pregnancy is approximately 12 kg (uterus: 1.0 kg, breasts: 1.5 kg, placenta: 0.7 kg, amniotic fluid 0.8 kg, maternal fluid gain:

2.0 kg, maternal fat: 2.5 kg: fetus: 3.5 kg; Clark 1992), which throughout the period of pregnancy has an impact upon balance, posture and locomotion. Certainly the abdominal protrusion, upward displacement of the diaphragm, flattening of the lower back and forward displacement of the centre of gravity are thought to provide some explanation for why approximately 50% of pregnant women suffer with low back pain. Additionally, the increased secretion of the reproductive steroid hormones and relaxin are associated with ligamentous and connective tissue laxity, which may predispose the pregnant female to ligament strain and further pain.

The endocrine responses to pregnancy lead to significant changes in the cardiovascular system. Increased aldosterone secretion leads to sodium and water retention that cause a 40–50% increase in blood volume (a major contribution to the 2 kg maternal fluid gain), whilst red cell mass increases by 17–25% (Longo 1983). Both systolic and diastolic blood pressures fall during the first trimester but rise again towards term. Cardiac output rises by approximately 40% during the first trimester due to an increased heart rate and stroke volume (enhanced left ventricular end-diastolic volume and contractility). Much of this increased cardiac output is directed towards the uteroplacental circulation, whilst blood flow to the skin and breasts is also increased. Renal blood flow increases by up to 80% in the first trimester but again falls towards term. The third trimester is associated with a reduced venous return because of the mechanical depression of the inferior vena cava by the gravid uterus. This is particularly a problem in the supine position and pregnant women are advised not to lie, and certainly not to exercise, in this position.

The above-mentioned physiological alterations are associated with an increase in resting $\dot{V}O_2$ of 20–30%, whilst the increased insulin secretion leads to reduced lipolysis and a greater reliance upon carbohydrate metabolism. Certainly metabolic homeostasis requires an additional 300 kcal per day. Late in pregnancy insulin resistance develops and there is a reduced carbohydrate use, increased lipolysis and greater use of maternal fat stores. The increased insulin resistance may well develop into gestational diabetes; however, this disorder does not always continue postpartum. The increase in resting $\dot{V}O_2$ is mostly due to fetal oxygen consumption and is aided by the increased red cell mass and a 40% increase in minute ventilation. Minute ventilation is increased solely via an increase in tidal volume as a result of a progesterone-mediated increase in CO_2 sensitivity. The increase in pulmonary ventilation leads to an increase in arterial PO_2 to approximately 100 mmHg, whilst arterial PCO_2 drops to 30–32 mmHg (Templeton & Kelman 1976), a respiratory alkalosis not entirely compensated for.

Participation in steady-state exercise during pregnancy has been seen to induce physiological responses similar to those observed at rest. Maternal heart rate, stroke volume, cardiac output, tidal volume and minute ventilation are all greater, whilst PCO_2 and pH are lower than would be expected in the non-pregnant state. Blood pressure responses are unchanged at a given absolute work rate. Net $\dot{V}O_2$ of non-weight-bearing activities is also unchanged, whilst weight-dependent activities elicit an increased oxygen cost. In late pregnancy stroke volume and cardiac output may well be reduced during exercise due to the compression of the vena cava by the gravid uterus. For more detailed review see Davies et al (2003).

Absolute $\dot{V}O_2$max is unchanged throughout pregnancy, unless physical activity levels are particularly reduced. Expression of $\dot{V}O_2$max relative to body mass, however, indicates a declining maximal aerobic capacity throughout pregnancy. Maximal heart rate has been seen to be reduced during late pregnancy probably due to blunted sympathoadrenal responses to exercise, and as resting heart rate is increased there is a reduced heart rate reserve. It would appear that the $\dot{V}O_2$ at the

ventilatory threshold is not affected by being pregnant; however, peak exercise respiratory exchange ratio, blood lactate concentration and excess post-exercise oxygen consumption following maximal exercise are all reduced in pregnancy. This is thought to be due to a decreased ability to exercise anaerobically because of reduced availability of carbohydrates as a result of decreased maternal liver glycogen stores or glycogenolysis and blunted sympathoadrenal responses to exercise. In prolonged strenuous exercise these maternal factors may well decrease availability to the fetus leading to decreased birthweight. The reader is referred to a further review by Morris & Johnson (2005).

The two major concerns for the fetus during maternal exercise are fetal hypoxia and fetal hyperthermia. Fetal oxygen delivery is aided by compensatory mechanisms that favour the fetus against the mother; these include haemoconcentration of maternal blood during exercise, redistribution of uteroplacental blood flow and an increased uteroplacental arteriovenous oxygen difference (Wolfe et al 1994). The fetus is protected from heat gain by further maternal compensations such as enhanced peripheral vasodilatation, reduced sweating threshold and increased ventilatory heat loss. The fetal temperature is usually approximately 0.5°C higher than the maternal core temperature, and thus prevention of an increase in maternal core temperature acts to protect the fetus during exercise.

The fetal heart rate increases during maternal aerobic exercise and takes approximately 20 minutes to return to resting values post exercise. The magnitude of increase is dependent upon exercise intensity and duration. The effect of heavy-intensity exercise upon fetal heart rate has been evaluated in a small number of studies indicating minimal responses. Exercise in the supine or semi-supine position results in fetal bradycardia and should, as previously noted, be avoided in late pregnancy.

Physical activity guidelines for the pregnant female

Exercise prescription for the pregnant female is dependent upon pre-pregnancy fitness level and/or physical activity status. Whether previously sedentary or active, all pregnant women should be examined by medical personnel prior to beginning their exercise programme and may be screened using the Physical Activity Readiness Medical Examination (PARmed-X) for Pregnancy questionnaire (Canadian Society for Exercise Physiology 2002). Contraindications to exercising during pregnancy have been published by the American College of Obstetricians and Gynecologists (2002). General advice for exercise during pregnancy is that participation in a wide range of recreational activities is safe as long as the participant avoids abdominal trauma, exercising in the heat or in the supine position, exercise above an altitude of 6000 feet and breath-holding activities such as scuba diving. Vigorous activity should be avoided in the third trimester. On the whole it is the mother that will benefit from long-term exercise but the fetus that will pay the price for her over-exercising.

The use of heart rate training zones to set levels of exercise intensity requires adjustment during pregnancy because of the reduced HR reserve (HRR). Wolfe (Canadian Society for Exercise Physiology 2002) recommends that the upper end of the age-related non-pregnant (60–75% maximal HRR) training zone be lowered for the pregnant state. Given that the non-pregnant training zone represents a width of approximately 20 beats/minute, the width of the pregnant zone should be decreased to 15 beats/minute by decreasing the top end by 5 beats/minute. Those women who were sedentary prior to pregnancy are recommended by the American College of Sports Medicine (ACSM 2006) to begin aerobic training at 20–39% HRR,

with low-impact activities such as walking or swimming. RPE is a valid tool to utilize during pregnancy as an adjunct to heart rate monitoring. The ACSM recommend 'light' to 'somewhat hard' (11–13) on the Borg 6–20 scale. In the absence of medical or obstetric complications, 30–40 minutes or more of moderate-intensity activity is recommended on most, if not all days per week, whilst a minimum of 15 minutes per session is required for a conditioning stimulus. Previously sedentary women can then increase duration by 1–2 minutes per week during the second trimester, to a maximum of 30 minutes.

Exercise for muscle strength and endurance and flexibility during pregnancy is complicated by the potential ligament laxity associated with the release of relaxin and the need to avoid raising blood pressure by a large magnitude. Flexibility exercises need to be controlled and non-ballistic, whilst strength training should consist of high repetition but low resistance exercises conducted in the upright position and with avoidance of the Valsalva manoeuvre (breath holding). Supine exercises should be avoided and abdominal exercises should be avoided so as not to aggravate any tearing of the linea alba. Balance training in pregnancy should be avoided due to the obvious risks of falls and related complications.

The rate of return to physical activity following delivery is dependent upon postpartum health and mode of delivery (particularly a caesarean). Following an uncomplicated normal delivery the participant may return to aerobic exercise once vaginal bleeding has stopped and her postpartum check-up is normal. She should attempt to start training at a similar HRR as when pregnant. Following caesarean section, the participant is advised not to return to aerobic exercise for at least 10 weeks, or until all complications have healed. Resistance exercises can be started after vaginal bleeding has ceased and Kegel exercises for the pelvic floor are recommended. These exercises involve contractions of the vaginal muscles for repeated 10 second periods. Abdominal exercises can be conducted in the supine position but only if diastasis recti (division of the abdominal muscles felt with the fingertips) has healed. Exercise has no detrimental effects upon milk composition, milk volume or maternal health.

THE POSTMENOPAUSAL FEMALE

The physiological changes that occur with ageing have been reviewed in Chapter 6, and indeed the postmenopausal female exercise participant will (on the whole) be over 50 years of age. Consequently, physical performance and physiological responses to exercise will vary according to age and physical activity/fitness status. However, the postmenopausal female brings with her a number of factors related specifically to the loss of oestrogen following the menopausal transition.

A recent UK birth cohort study indicated that at 53 years of age body mass index (BMI), waist circumference, total cholesterol (TC), low-density lipoprotein (LDL) and HbA_{1c} concentration (blood marker of glucose control) varied between pre- and postmenopausal women (all being greater post menopause), and that TC and HbA_1c increased across the menopausal transition before and after adjustment for BMI, smoking, lifestyle and socioeconomic circumstances (Kuh et al 2005). In addition, Zaydun et al (2006), in a study of 3149 women, added that being postmenopausal significantly increased the risk of arterial stiffness independently of age, hypertension, hypercholesterolaemia, diabetes mellitus, obesity and smoking, whilst Taddei et al (1996) associated the menopause with endothelial dysfunction. Each of these reported variations is associated with an increased risk of cardiovascular disease following

the menopausal transition, whilst studies by Rossi et al (2005a, 2005b) have also indicated that the menopausal transition might well increase the risk of both hypertension and type 2 diabetes.

Participation in physical activity in postmenopausal women has been shown to have positive effects upon risk factors associated with cardiovascular disease. In cross-sectional analyses of physical activity status, LDL and high-density lipoprotein (HDL) cholesterol levels have been more favourable in active than non-active women, irrespective of HRT utilization (Green et al 2004). Physical activity interventions have resulted in decreases in BMI and waist circumference, decreases in LDL, fasting blood glucose, HbA_{1c}, C-reactive protein (CRP: marker of inflammation) and TC concentrations, and increases in insulin sensitivity and arterial compliance in women, whether utilizing or not utilizing HRT. These results have been seen in studies as short as 2 and 12 weeks in duration and in those which have utilized walking as the exercise stimulus. Changes in fat mass, especially visceral adiposity, lipoprotein subfractions and markers of glucose control (HbA_{1c}) and inflammation (CRP), are all indicative of a decrease in the risk of cardiovascular disease and this decrease appears to be independent of HRT utilization (Green et al 2004). HRT use may have an additive effect upon the favourable alterations in these risk factors; however, this evidence needs to be weighed against the known risks of HRT.

The loss of oestrogen following the menopause also places the exercising female at risk of osteopenia and osteoporosis. The mechanisms for this increase in risk have been covered above. In brief, oestrogen deficiency accelerates bone resorption, and thus women who have low bone mass prior to the menopause have a significantly greater risk of developing osteoporosis in later years. Comparisons of pre- and postmenopausal athletes suggest that even participation in vigorous physical activity does not prevent the loss in bone mineral density that occurs at the menopause. Indeed, data from the Nurses' Health Study (Feskanich et al 2002) suggested that the risk of hip fracture was reduced by 60–70% in women using hormone replacement therapy (HRT), regardless of physical activity level, compared to sedentary women not using HRT. Among those women not using HRT, those in the highest quintile of physical activity (>24 METs, hours per week) also had a 67% reduction in hip fracture risk, suggesting an additional benefit of physical activity above the effect upon bone mass.

The effect of physical activity upon bone mass and bone mineral density in postmenopausal women has received considerable attention over the last three decades. The influence of physical activity upon bone turnover in hypo-oestrogenic conditions has become of even greater importance in the last decade due to the newly observed detrimental health effects of HRT. The recent attention to HRT-induced coronary events and incidence of breast cancer has induced contentious results and reports (for review see Tormey et al 2006). What is clear, however, is that HRT is no longer recommended for the attenuation of bone loss in postmenopausal women. Recently recommended courses of action are bisphosphonates, selective oestrogen receptor modulators, raloxifene and, of course, physical activity (Delaney 2006).

Physical activity guidelines for the postmenopausal female

Exercise prescription for the postmenopausal woman and the seven components of fitness discussed in Chapter 1 does not differ from the details discussed

in other chapters within this book. The factors indicating an increased risk of cardiovascular disease at the menopausal transition (e.g. markers of glucose control, inflammation, insulin sensitivity, visceral adiposity, LDL, TG and TC, endothelial dysfunction and arterial stiffness) will respond more to physical activity aimed at enhancing cardiorespiratory endurance (see Asikainen et al 2004 for further review). This means, for these special considerations, the postmenopausal and indeed perimenopausal female should be partaking in high-volume, low–moderate-intensity, longer duration aerobic activities. The reader is referred to Chapters 1, 2, 3, 6 and 7 for further guidelines that relate to females of differing ages or relevant health conditions.

The special considerations for the postmenopausal woman do not only include the menopause-related increase in the risk of cardiovascular disease. Indeed, the postmenopausal woman, if already sedentary, may well also be at an increased risk of osteopenia, osteoporosis and related stress fractures and falls. The reader is referred to Chapter 6 for further information regarding guidelines for increasing muscle strength and balance in an older population. Bassey & Dinan (2001) provide guidelines on the recommendations for safely increasing bone mineral density using exercise or physical activity.

The literature evaluating the influence of physical activity upon bone mass in postmenopausal women has utilized a range of different exercise modes, intensities and durations. Walking interventions have been seen to provide only modest effects at best. Daily walking at an intensity equivalent to 50% $\dot{V}O_2$max, for a duration of 1 hour per day with at least 8000 steps, 4 days per week over 12 months induced an increased bone mineral density of the lumbar spine in women with osteopenia (Yamazaki et al 2004). An evaluation of elite female runners not using HRT before, during and after the menopause revealed an expected age-related decrease in bone mineral density from the femoral neck and spine, but a reduced rate of bone loss in the femoral trochanter and almost no bone loss in the calcaneus (Tomkinson et al 2003). It would thus appear that oestrogen loss has a greater effect upon the femoral neck and spine than the trochanter, even when mechanical stress is constant.

Given that bone responds positively to stresses of multiple amplitudes and directions, physical activity interventions in postmenopausal women have attempted to use multiple exercise modes to increase bone mass. The Erlangen Fitness Osteoporosis Prevention study (Engelke et al 2006), cited in Chapter 7, examined the impact of a vigorous combined high-impact, strength and endurance programme over 2 years upon bone mineral density in postmenopausal women with osteopenia. Bone mineral density increased by 1.3% at the lumbar spine, whilst no change occurred in the hip and femoral neck. At 3 years bone mineral density was increased in the spine, hip and calcaneus but not in the forearm. Similar studies using high-impact but low-volume training have reported identical results. Stengel et al (2005) compared power training to strength training in postmenopausal women over a period of 12 months. As noted in Chapter 7, the power-trained women maintained bone mineral density at the spine and total hip, whereas the strength-trained women lost density at both sites. Hip bone mineral density has also been seen to be maintained in postmenopausal women participating in jumping whilst wearing a weighted vest and it thus appears that for women in a hypo-oestrogenic condition bone responds best to higher-volume and high-speed mechanical stresses. Of course if osteoporosis is already present, the use of exercise to increase bone mineral density should be tailored to protect the low starting point.

KEY POINTS

1. The menstrual cycle, pregnancy and menopause are associated with major physiological and endocrinological fluctuations that indicate key considerations for exercise prescription.
2. The 'female athlete triad' describes a unique set of disorders that place the exercising female at risk of decreased health and performance.
3. Disordered eating that induces a negative energy balance can lead to loss of the secretion of oestrogen (often indicated by amenorrhoea) and thus increase the risk of stress fractures and osteopenia/osteoporosis.
4. Negative energy balance can be avoided in exercising females, typically by a modest reduction in training volume coupled with an altered eating pattern.
5. Pregnancy requires attention to the type and intensity of exercise undertaken and can influence fetal safety or birthweight if guidelines are not followed.
6. The menopausal transition may well be associated with a detrimental cardiovascular disease profile, and with progressive decline in bone strength, in sedentary females.
7. Consideration given to each of the key points above will ensure that the female can exercise or train effectively and safely.

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