Review Article

Musculoskeletal Considerations for Exercise and Sport: Before, During, and After Pregnancy

Lauren E. Borowski, MD
Elizabeth I. Barchi, MD
Julie S. Han, MD
David A. Friedman, MD
Cordelia W. Carter, MD

ABSTRACT

There is little written in the orthopaedic literature regarding common musculoskeletal problems that women encounter in relation to pregnancy and their clinical and surgical management. Exercise and other physical activity are generally recommended for most women before, during, and after pregnancy. Unfortunately, a variety of musculoskeletal issues may keep women from starting, continuing, or resuming a healthy exercise regimen throughout a notable portion of their reproductive years. Untreated and undertreated orthopaedic conditions in female athletes may therefore have further unintended negative effects on maternal and fetal health. This article reviews the existing literature on musculoskeletal health considerations before, during, and after pregnancy to provide practical information to orthopaedic surgeons who treat women of all ages and athletic abilities.

According to recent census data, more than 75 million women of childbearing age (15 to 50 years old) are living in the United States. In addition, since the introduction of Title IX legislation in 1972, the Women's Sports Foundation reports that the number of girls and women participating in sport has increased by a factor of 10, from less than 4 of every 100 to 4 in 10. This translates to a 990% increase in girls participating in high school sport and a 545% increase in girls and women participating in college sport. As a result, the chances are good that a practicing orthopaedic surgeon will encounter a female patient who is, was, wants to be, or will be pregnant and physically active at some point in her life. Despite this, very little is known empirically about the risks and benefits of participation in sport and other physical activities during childbearing years, which, for some women, may span 4 decades.

This lack of data likely stems at least in part from a collective desire to protect vulnerable research populations—women in their reproductive years and children chief among them. However, as a consequence, physicians (orthopaedic surgeons included) may dispense information to patients regarding physical activity and exercise during pregnancy that is based on expert opinion rather than scientifically acquired data. Perhaps worse, perinatal counseling is typically "protectionist"—meaning that women may be counseled against performing a certain type or intensity of exercise not because it is known to have negative health consequences,

From the Department of Orthopedic Surgery, Division of Sports Medicine, NYU Grossman School of Medicine (Borowski, Barchi, Han, Carter), and the Department of Obstetrics and Gynecology, NYU Grossman School of Medicine, New York, NY (Friedman).

None of the following authors or any immediate family member has received anything of value from or has stock or stock options held in a commercial company or institution related directly or indirectly to the subject of this article: Borowski, Barchi, Han, Friedman, and Carter.

J Am Acad Orthop Surg 2021;29:e805-e814

DOI: 10.5435/JAAOS-D-21-00044

but simply because any health consequences, if they exist, are unknown. This may result in the loss of well-established benefits to participation in physical activity for women of childbearing age, including optimization of bone health, cardiovascular health, mental health, and protection against diabetes and metabolic syndrome. Furthermore, exercise during pregnancy is seen to have minimal risk and may have substantial benefits including lower incidence of excessive weight gain, gestational diabetes, gestational hypertension, preterm birth, caesarean sections, and lower birth weight.[1]

Although many activities are considered safe in pregnancy, some activities, such as contact sport, skiing, and scuba diving are considered unsafe in pregnancy. These activities are summarized in Figure 1. There are warning signs, such as vaginal bleeding and leakage of amniotic fluid, for discontinuation of exercise in pregnancy.[2] Although many women will be able to enjoy an appropriate level of physical activity, there are relative and absolute contraindications to aerobic exercise in pregnancy. Patients such as heavy smokers and the morbidly obese may wish to proceed with caution, whereas those with conditions such as placenta previa after 26 weeks of gestation and preeclampsia or pregnancy-induced hypertension should avoid higher intensity aerobic exercise.[2]

For many women, failure to continue training or competing in their desired sport because of pregnancy-related concerns may result additionally in the loss of self-identity, and in those considered elite athletes, a loss of financial income, sponsorships, and even careers. The purpose of this article is to review the existing literature on musculoskeletal health as it relates to women in their childbearing years, from recreational to elite athletes, and to highlight issues related to the prenatal, perinatal, and postnatal periods that are most salient for the orthopaedic surgeon. The definitions for the terms used in reference to exercise and physical activity are seen in Table 1.

Preconception

Perhaps the most important factor that may negatively affect a female athlete's ability to conceive is the presence of an eating disorder. It has been reported that athletes have higher rates of eating disorders than nonathletes. The sport most affected include endurance and aesthetic sport (eg, cross-country and gymnastics) and those with weight categories. Additional risk factors for an eating disorder in athletes—which may be used as screening tools in the office setting—are listed in Table 2.[3] The constellation of amenorrhea, osteoporosis, and an eating disorder is known as the female athletic triad. The effects are not always reversible; therefore, it is essential

Figure 1

Chart showing the physical activities in pregnancy

Table 1. **Adapted From the 2016 Evidence Summary From the International Olympic Committee Expert Group Meeting on Exercise and Pregnancy in Recreational and Elite Athletes**

Physical activity	Bodily movement produced by the contraction of skeletal muscles resulting in a greater energy expenditure than while at rest
Exercise	Repetitive physical activity, often structured and planned, usually performed to improve fitness and health
Sport	Contest or game in which people perform physical activities based on set rules and compete against others
Fitness	Qualities relating to the ability to perform physical activity and exercise
Athlete	A person who performs regular strenuous exercise for fitness and competition but is not part of a national team
Elite athlete	A person on a national team or other high-level representative team in a sport organization

to recognize athletes with eating disorders or amenorrhea and refer for appropriate treatment.[4]

However, a full-blown eating disorder is not needed to disrupt the neuroendocrine axis and interfere with fertility. Rather, the syndrome of relative energy deficiency in sport has been characterized as impaired physiological function resulting from relative energy deficiency (in some cases because of disordered eating or systematic underfueling) such that the body cannot function optimally for maintenance of health and peak athletic performance.[5] This syndrome may affect both male and female athletes, with symptoms such as bone stress injury and impaired immunity. In women, it may additionally manifest as menstrual dysfunction, which may "confound conception"—leading to frank infertility or, conversely, to unexpected or misdated pregnancy.

In the specific setting of the elite female athlete who may wish to train and compete well into her reproductive years, counseling on "strategic conception" may be useful so that an athlete may plan her pregnancy in a way that is minimally disruptive to her competition schedule. Importantly, the data that exist on pregnancy in elite athletes suggest that they can get pregnant and deliver healthy babies at rates commensurate with nonathletic controls.[6] Alternatively, counseling regarding delayed conception through the use of strategic fertility preservation techniques (eg, egg harvest and cryostorage of gametes and/or embryos for use in retirement) may allow a female athlete to achieve both personal and professional goals.

Postconception: Considerations During the Peripartum and Postpartum Periods

Existing Musculoskeletal Conditions

Although many musculoskeletal conditions arise in the setting of pregnancy because of the unique physical and physiologic changes that a female body undergoes, a few conditions exist that may be present before conception that may be exacerbated during pregnancy.

It has been postulated that physiologic adaptations of pregnancy may play a role in mitigating the long-term manifestations of pain syndromes. Gutierrez et al,[7] using a rat model, suggested that a change in the descending inhibitory/facilitating balance on spinal nociception neurotransmission during the puerperium may exert a protective effect against injury-induced hypersensitivity.

Joint Instability

During pregnancy, the release of certain hormones leads to an increase in joint laxity beginning approximately the 10th week of gestation and can last up to 4 to 12 weeks postpartum.[8] These changes are thought to be due to the increased levels of the hormone, relaxin, which is produced at peak levels approximately weeks 10 to 12 of gestation.[9,10] Relaxin stimulates collagenase which helps prepare the pelvis for delivery by decreasing the

Table 2. **Common Risk Factors for Eating Disorder in Female Athletes**

• Early sport-specific training
• Overuse and overtraining syndromes
• Positive injury history
• Pressure to lose weight
• Negative sport culture and/or coaching behavior

Recognition of eating disorders and referral for appropriate care are essential for both optimization of the athlete's health and performance and for optimization of her fertility and the outcomes of future pregnancies: prepregnancy underweight is associated with both preterm births and low birthweight babies (Bø, 2016).

tensile strength of ligaments resulting in increased mobility of joints.[11] Estrogen increases the relaxin receptor sensitivity and therefore increases its effects.[11]

The morbidity from increased joint laxity is still unclear. Marnach et al[12] found no correlation between peripheral joint laxity and relaxin levels. Meanwhile, Schauberger et al[8] discovered an increase in peripheral joint laxity as pregnancy progressed, yet no correlation existed between peripheral joint laxity and relaxin levels. In addition, Sundelin et al[13] found that women with joint hypermobility syndrome/Ehlers-Danlos syndrome did not seem to have an increased risk of adverse pregnancy outcomes. To our knowledge, no study has been performed investigating pregnancy-related ligamentous laxity and a female athlete's risk of joint instability, which might be postulated to manifest as patellofemoral or shoulder subluxation and/or dislocation events.

Back Pain

Low back pain (LBP) is common within the general cohort with an estimated prevalence as high as 85% to 90% in a lifetime.[11] LBP is also common in athletes, especially those who participate in gymnastics, diving, weightlifting, and racquet sports. Proposed etiologies for LBP are numerous and risk factors include poor muscle endurance, muscular imbalance, altered muscle-firing rates of the erector spinae, and decreased flexibility of the spine.[11]

LBP associated with pregnancy can occur at any time during pregnancy but most frequently appears approximately at 22 weeks of gestation.[11] The prevalence of LBP during pregnancy has been reported between 24% and 84%, and some reports suggest that up to 85% of women with pregnancy-related LBP will have LBP in subsequent pregnancies.[11] Wang et al[14] found that LBP during a previous pregnancy, LBP during menstruation, and history of nonpregnancy-related LBP were independent predictors for the existence of LBP during pregnancy. They also found that a markedly larger proportion of African American women experienced LBP compared with women of other ethnicities.[14] The question has been raised as to whether the use of epidural labor analgesia is a causative factor in LBP. This has been investigated and is not considered to be a cause of new or long-term back pain.[14]

Pregnancy-related back pain typically presents with a dull, achy pain in the lower back that tends to worsen throughout the day.[11] Activities that require repetitive forward flexion of the spine often aggravate symptoms.[15] Pregnancy-related LBP is often associated with several biomechanical and muscular changes during pregnancy. Sihvonen et al[16] reported women with reduced activity in the paraspinal musculature at L4 and L5 during the first trimester noted more pain and disability throughout the pregnancy. In addition, Gutke et al[17] found decreased muscular endurance of the back flexors and extensors as well as reduced hip extensor strength in women with pregnancy-related LBP. Noren et al[18] also noted lower endurance of the back extensors and hip abductors in association with pregnancy-related LBP.

Degenerative spondylolisthesis has a markedly higher incidence in women who have had children compared with nulliparous women.[19] The most common location for degenerative spondylolisthesis is L4-L5 and can progress during pregnancy.

In the event of "red flags" such as progressive sensorimotor deficit, saddle anesthesia, or new incontinence or retention, MRI or ultrasonography is the recommended imaging modality.[11] The American College of Obstetricians and Gynecologists guidelines on diagnostic imaging during pregnancy states that there have been no observed, adverse effects to the fetus after ultrasonography, MRI, and imaging, resulting in less than five rads of radiation exposure. The American College of Obstetricians and Gynecologists also recommends MRI over radiographs when indicated.[1]

Exercise is generally accepted to help reduce the risk of pregnancy-related LBP; however, there is little evidence to support this. Some studies have reported that individualized treatment programs were more effective at reducing pain intensity and sick leave compared with back school education.[11] Stuge et al[20] noted improvement of symptoms with therapy focusing on stabilizing the hip abductors, hip adductors, gluteus maximus, transverse and oblique abdominis, erector spinae, lumbar multifidus, quadratus lumborum, and latissimus dorsi. LBP during pregnancy may limit a woman's opportunities for physical activity.

Exercising in water may benefit these patients because the buoyancy provides a reduction in osteoarticular load.[1] Interestingly, female athletes experience pregnancy-related LBP (and pelvic girdle pain, discussed below) at rates similar to their nonathletic peers.[3]

Conditions Unique to Pregnancy

Pregnancy-Related Pelvic Girdle Pain

The pelvic girdle consists of the pubic symphysis and right and left sacroiliac joints. Pregnancy-related pelvic girdle pain (PPGP) is often thought to be because of joint laxity. However, the asymmetry of this laxity, especially in the sacroiliac joints, may be the true underlying issue.

PPGP is often a self-limited problem with 93% of women having resolution of their pain by 3 months postpartum. 1% to 2% of women remain symptomatic at 1-year postpartum.[21] The average gestational age at which this pain usually begins is approximately 18 weeks, and pain severity tends to peak between 24 and 36 weeks gestation.[21]

PPGP can be hard to locate precisely, and it varies in its presentation ranging from a stabbing, dull ache to a burning or shooting pain in the sacral, gluteal, or pubic symphysis areas. On examination, the patient may want to avoid hip abduction and adduction due to pain. Patients may also have an altered gait. Generally no singular nerve root distribution is identified on examination.

Stabilization exercise programs have been considered a mainstay in treatment of PPGP. In 2018, Shiri et al performed a meta-analysis of four randomized control trials for pelvic girdle pain, which showed no protective effect of exercise on pelvic girdle pain like it does on LBP. However, it was shown that exercise could decrease the severity of pelvic girdle pain and the amount of sick leave.[22] Sakamoto et al[23] reported the use of a realignment device in addition to exercise programs to be helpful in decreasing pain in the immediate postpartum period, but no differences were observed between exercise programs done with or without the device at 13 weeks postpartum. As noted above, although one might expect to see a protective effect of physical activity for prevention of LBP and PPGP, female athletes experience these pain syndromes at rates commensurate with nonathletes.

Transient Osteoporosis of Pregnancy

Pregnancy- and lactation-associated osteoporosis is a rare but serious form of osteoporosis that presents in the last trimester of pregnancy or immediately postpartum. The lumbosacral spine and hips tend to have the most notable decrease in bone density, and women can present with pain and subsequent fractures in these areas (Figure 2). Deficits in osteoblast function contribute to a low bone remodeling state that has been seen in women with pregnancy- and lactation-associated osteoporosis at approximately 1 year postpartum.[24]

The clinical presentation of women with pregnancy-associated osteoporosis is generally more severe than premenopausal women with idiopathic osteoporosis.[24] They are more likely to have multiple fractures, particularly of the vertebrae. These patients most commonly present with LBP. A retrospective study by Gehlen et al[25] in 2019 showed that women had symptoms for an average of 3.3 months before diagnosis. The women in that study had an average of 5.4 vertebral fractures seen at the time of diagnosis. Given the young age of these women, most do not have previous bone mineral density scores available. Currently, no data exist on whether female athletes are more or less vulnerable than nonathletes for developing this condition. One might postulate that those athletes with previous stress fractures and/or disordered eating may have lower bone mineral density scores before pregnancy, rendering them more vulnerable. However, it is also known that weight-bearing activity is associated with an increase in bone mineral density and may be protective against pathologic fracture.

The physiology of women changes during pregnancy and lactation to meet the fetal requirements for mineral and bone development.[26] The fetal demand for calcium and phosphorus, particularly in the third trimester, can potentially lead to maternal hypocalcemia and hypophosphatemia.[26] The physiologic adaptations to meet fetal and neonatal requirements differ from pregnancy to lactation. During pregnancy, the efficiency of maternal intestinal absorption of calcium doubles to help meet fetal needs.[26] During lactation, skeletal resorption increases to augment calcium levels in breast milk.[26] In the absence of very low mineral intake or an underlying bone mineral density deficiency, few long-term consequences of these adaptations to the maternal skeleton exist.[26,27]

The appropriate treatment of these patients is unclear, given that increases in bone density progressively occur after weaning from lactation. Treatment includes the use of medications such as calcitonin, bisphosphonates, teriparatide, and strontium ranelate as well as procedures such as vertebroplasty and kyphoplasty. Bisphosphonates should not be given if the patient plans to conceive again within 1 year, and women should stop

Figure 2

Radiograph showing mild widening of the pubic symphysis seen in a woman at 2 weeks postpartum.

treatment at least 6 months before they plan to conceive again. If a woman becomes pregnant during treatment, they should stop bisphosphonates immediately.[28] Calcium and vitamin D serum levels should be optimized for anyone suffering a low-trauma fracture.[27] Weight-bearing activities are also encouraged to help build bone density. Knowledge that bone density increases after pregnancy and that subsequent pregnancies are rarely affected can inform discussions of prognosis.

Pubic Symphysis Diastasis

Some widening of the symphysis pubis is a normal occurrence that happens during pregnancy. This widening of several millimeters is generally asymptomatic (Figure 3). Separation of the pubic symphysis is considered pathologic when the distance is greater than 10 mm.[29] Pubic symphysis diastasis has been associated with multiparity, fetal macrosomia, precipitous labor, prolonged labor, shoulder dystocia, forceps delivery, maternal connective tissue disorder, and previous pelvic pathology or trauma.[29] The overall incidence is not well known, ranging from 1 in 300 to 1 in 30,000.[30] This condition can occur during antepartum, intrapartum or, most commonly, postpartum.

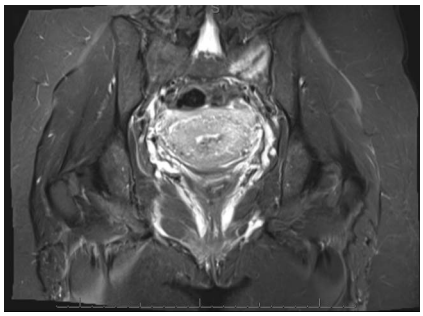
These patients may present with pain on ambulation, urinary dysfunction, and pelvic instability. Imaging of the area can be misleading because the degree of separation on imaging does not directly correlate with the severity of the patient's pain postpartum. For this reason, the diagnosis is generally considered to be a clinical one and is supported by imaging.[29] The sacroiliac joints and sacrum can also be affected by pelvic instability that accompanies pubic symphysis diastasis, and imaging of the pelvis with a CT scan may be needed to see the extent of injury, particularly any posterior ring involvement.[31]

Treatment of pubic symphysis diastasis is largely considered to be nonsurgical and usually includes some combination of bed rest, physical therapy, pelvic binder or sling, and pain medications. Some protocols include weight bearing immediately with physical therapy, whereas others emphasize nonweight bearing and bed rest. Surgical treatment—open reduction with internal and/or external fixation—may be considered for large gaps in the pubic symphysis (>40 mm) and for patients with recalcitrant pain or notable pelvic instability; however, little evidence regarding treatment of these injuries comes from randomized control trials.[31]

Carpal Tunnel Syndrome

Carpal tunnel syndrome (CTS) occurs because of the compression or traction of the median nerve as it travels through the volar wrist into the hand. CTS is the most common mononeuropathy in pregnancy with studies reporting the prevalence ranging from 2% to 62%.[32] Meems et al[33] showed the severity of symptoms increased with each gestational term, and most women reported symptoms after 32 weeks. Increased fluid retention seemed to increase the likelihood of patients reporting CTS symptoms.[33,34]

Figure 3



MRI of 35-year-old female with left hip pain that began immediately postpartum showing incomplete sacral stress fracture

Presenting symptoms for CTS include tingling, numbness, and pain in the thumb, index, and middle fingers as well as the radial aspect of the ring finger. Other signs include diminished grip strength, hand dexterity, decreased two-point discrimination, and burning sensations in the hand. Atrophy of the thenar musculature tends to be a late sign.[32] Symptoms are frequently exacerbated by repetitive motions and certain sleep positions. CTS can have detrimental effects on sleep, particularly in the last month of pregnancy.[33] The clinical diagnosis of CTS in pregnant patients is the same as it is in the general cohort. Although electrodiagnostic testing, including nerve conduction tests and electromyography, are safe in pregnancy and can be helpful for diagnostic and prognostication purposes, they can often be avoided.

Most women will have resolution of their symptoms shortly after birth, but approximately one of six women will continue to have symptoms at 12 months postpartum.[34] Women with early onset of symptoms (before third trimester), severe CTS symptoms, and high scores on a depression severity index tend to have CTS symptoms that persist at 12 months postpartum or longer.[34] The approach to treatment of CTS in pregnant women is similar to the general cohort. Wrist splints worn at nighttime are generally the first-line of treatment. If the use of the splints fails to improve symptoms, a local corticosteroid injection (CSI) is both

safe and efficacious. Surgical decompression is considered only if conservative treatments fail or electrodiagnostic studies show notable impairment. No data exist demonstrating an increased risk of incidence or severity of CTS in athletic women versus their nonathletic peers.

de Quervain Tenosynovitis

de Quervain tenosynovitis is an overuse disease involving repetitive strain of the abductor pollicis longus and extensor pollicis brevis tendons passing under a thickened overlying extensor retinaculum.[21,35,36] Inciting activities include gripping, lifting, and twisting motions of the wrist which involve a repetitive ulnar deviation of the wrist with thumb extension or abduction.[37,38] These culprit activities are more common in the postpartum period because of infant care, which includes lifting the infant and wringing a washcloth.[35] Increased fluid retention because of hormonal changes during pregnancy and lactation may contribute to the increased incidence of de Quervain tenosynovitis in the peripartum period.[21]

The presenting clinical picture is characterized by radial-sided wrist pain that may radiate down the thumb and into the forearm.[21,37,38] On physical examinationin, pain is reproduced to palpation of the first dorsal compartment of the wrist located near the radial styloid.[38,39] There may be swelling and/or crepitus to palpation in this region.[36] Finkelstein test is a useful diagnostic tool. The diagnosis is clinical.

Initial treatment is nonsurgical and includes bracing treatment with thumb spica splint, nonsteroidal antiinflammatory drugs, physical therapy, and CSI.[37] CSI is an effective first-line treatment option for de Quervain tenosynovitis, and the utility of adjunctive treatment options has not been definitively established.[37] Surgical treatment with first dorsal compartment release has a reported 91% cure rate but its associated costs, invasiveness, and risks make/ nonsurgical treatment the first-line option for de Quervain tenosynovitis.[36]

There have been no studies specifically looking at the incidence of de Quervain in athletes who use their hands and wrists in sport such as those who play racquet sport or whose sport involves throwing. This is an area ripe for research.

Diastasis Rectus Abdominis

The abdominal rectus muscles are separated by a fascia called the linea alba, which is composed of the aponeuroses of the external oblique, internal oblique, and transverse abdominal muscles.[40,41] Diastasis rectus abdominis (DRA) is a separation of the two muscle bellies of the rectus abdominis resulting in a widened linea alba.[21,41] Increasing intra-abdominal pressure and stretch against the abdominal wall from the growing fetus and softening of connective tissue because of hormonal changes in pregnancy predisposes pregnant women to developing DRA.[21,40-42] DRA is found in more than half of pregnant women with prevalence rates ranging from 27% to 100%, and the risk increases with each trimester.[21,42,43]

Patients will present with complaints of a visual or palpable gap in the abdomen with or without a protrusion of the abdomen, which may be coupled with other concurrent symptoms such as back pain or, in severe cases, herniation of the abdominal viscera.[21,40,42] The widened gap between the rectus abdominis muscles at the linea alba will be visible and/or palpable on clinical examinationin findings. Beer et al[41] established a quantitative classification of normal inter-rectus distance (IRD) by measuring the distance between the medial borders of the two rectus abdominis muscle bellies at three different reference points using an ultrasonography machine (Table 3). However, no consensus exists on the definition of normal values of IRD and when it is considered pathologic.[40,41]

Nonsurgical management including physical activity, muscular training, and physical therapy in the antepartum period seems to have a protective effect against DRA, although it is not known if the rate of DRA in female athletes is lower than in nonathletes.[40,43,44] A small study by Chiarello et al[43] found that nonexercising pregnant women have higher occurrence and size of DRA compared with women who participate in an abdominal exercise program consisting of abdominal muscle strengthening and pelvic floor exercise programs during pregnancy. The type of exercise may be relevant as another study by Gilleard et al found DRA in all pregnant women despite participation in various exercise activities such as cycling, aerobics, walking, weight training, and swimming. Although no current benchmark of treatment of DRA exists, abdominal strengthening exercise programs have been shown to be effective

Table 3. Normal Widths of the Linea Alba in Nulliparous Women[41]

Reference Point	Width (mm)
At the xyphoid	15
3 cm above the umbilicus	22
2 cm below the umbilicus	16

in decreasing the incidence of diastasis recti abdominus and decrease the IRD in women who gave birth vaginally or by cesarean birth.[1,45]

A systematic review by Akram et al found the existing literature on surgical repair of DRA (open, laparoscopic, and endoscopic) to be low-quality evidence and concluded that no consensus exists on indication for repair. Surgical repair is done mostly for cosmetic reasons and is usually performed with abdominoplasty.[40]

Pelvic Floor Dysfunction

The pelvic floor muscles include the pubococcygeus, puborectalis, and iliococcygeus muscles, which are collectively referred to as the levator ani.[46] The pelvic floor functions to support the pelvic organs and maintain urinary, anal, and vaginal continence.[46] Pelvic floor dysfunction (PFD) occurs when these muscles are weakened and the function of the pelvic floor is compromised. PFD is comprised of a variety of disorders including relaxing PFD such as urinary incontinence, pelvic organ prolapse, fecal incontinence, and nonrelaxing or hypertonic PFD, which includes pelvic myofascial pain, dyspareunia, and vulvodynia.[46,47] PFD is common in women with one in four women reporting symptoms of urinary incontinence, fecal incontinence, or pelvic organ prolapse.[46] Stress-related urinary incontinence has been reported in a notable percentage of female athletes even before pregnancy, with the range reported to be between 28% and 80%.[3] Increased age, weight, and parity are risk factors for developing symptoms of PFD.[46] Pregnancy and childbirth stretches and weakens the pelvic floor muscles, which can lead to PFD.[47] The reported rates of postpartum PFD in athletes are similar to the general cohort. It should be noted that a cesarean delivery does not eliminate the risk of PFD.[48]

The signs and symptoms of PFD are varied, and women with PFD can present with a wide range of symptoms. Urinary incontinence presents with involuntary leakage of urine with activities that increase intra-abdominal pressure such as laughing, coughing, exercising, or sneezing.[47] Similarly, fecal incontinence involves an involuntary loss of stool. Pelvic organ prolapse presents with pelvic pressure, fullness, or problems voiding or defecating.[47] Signs and symptoms of nonrelaxing or hypertonic PFD can include tenderness of pelvic floor muscles with painful myofascial trigger points, vaginal pain with sexual intercourse, and vulvar pain.[47]

Physical therapy has been shown to be an effective first-line treatment option for the disorders associated with PFD.[46,47] Pelvic floor physical therapy (PFPT) involves strengthening of pelvic floor muscles through exercise programs, manual therapy, behavioral education, and electrical stimulation.[47] Strengthening of the pelvic floor muscles improves urinary incontinence by increasing pelvic floor muscle contraction to prevent involuntary urine leakage with activities.[49] Similarly, PFPT helps decrease episodes of fecal incontinence when added in combination with electrical stimulation or biofeedback.[47] PFPT is recommended with level 1, grade A evidence for pelvic organ prolapse as many studies demonstrate improved outcomes and reduction of symptoms.[47] PFPT is an effective first-line treatment of nonrelaxing or hypertonic PFD.[47] In women with dyspareunia, PFPT is part of a multidisciplinary treatment approach to increase muscle relaxation and proprioception.[47]

Summary

Although women of childbearing age are a common sight in the offices of orthopaedic surgeons, musculoskeletal conditions that are unique to pregnancy have not traditionally been an important part of orthopaedic education and research efforts. This review of the scant existing literature on pregnancy-related musculoskeletal concerns that arise in women who participate in exercise and sport during pregnancy may serve as an introduction to the practicing orthopaedic surgeon who is likely to encounter a peripartum athlete in the office. Although surgical treatment is rarely required in this setting, an understanding of fetus-protecting imaging recommendations is useful, as is awareness of conditions—such as transient osteoporosis of pregnancy—that may require coordinated multidisciplinary care.

References

References printed in **bold type** are those published within the past 5 years.

1. Mota P, Bø K: ACOG committee opinion no. 804: Physical activity and exercise during pregnancy and the postpartum period. *Obstet Gynecol* 2021;137:376.

2. Artal R: Exercise in pregnancy: Guidelines. *Clin Obstet Gynecol* 2016; 59(3):639-644.

3. Bø K, Artal R, Barakat R, et al: Exercise and pregnancy in recreational and elite athletes: 2016/2017 evidence summary from the IOC expert group meeting, Lausanne. Part 5. Recommendations for health professionals and active women. *Br J Sports Med* 2018;52: 1080-1085.

4. Committee opinion no.702: Female athlete triad. *Obstet Gynecol* 2017;129:e160-e167.

5. Mountjoy M, Sundgot-Borgen J, Burke L, et al: The IOC consensus statement: Beyond the female athlete triad—relative energy deficiency in sport (RED-S). *Br J Sports Med* 2014;48:491-497.

6. Sundgot-Borgen J, Sundgot-Borgen C, Myklebust G, Sølvberg N, Torstveit MK: Elite athletes get pregnant, have healthy babies and return to sport early postpartum. *BMJ Open Sport Exerc Med* 2019;5: e00065.

7. Gutierrez S, Hayashida K, Eisenach JC: The puerperium alters spinal cord plasticity following peripheral nerve injury. *Neuroscience* 2013;228:301-308.

8. Schauberger CW, Rooney BL, Goldsmith L, Shenton D, Silva PD, Schaper A: Peripheral joint laxity increases in pregnancy but does not correlate with serum relaxin levels. *Am J Obstet Gynecol* 1996;174:667-671.

9. Bani D: Relaxin: A pleiotropic hormone. *Gen Pharmacol* 1997;28:13-22.

10. Kristiansson P, Svärdsudd K, Von Schoultz B: Serum relaxin, symphyseal pain, and back pain during pregnancy. *Am J Obstet Gynecol* 1996;175:1342-1347.

11. Noon ML, Hoch AZ: Challenges of the pregnant athlete and low back pain. *Curr Sports Med Rep* 2012;11:43-48.

12. Marnach ML, Ramin KD, Ramsey PS, Song SW, Stensland JJ, An KN: Characterization of the relationship between joint laxity and maternal hormones in pregnancy. *Obstet Gynecol* 2003;101:331-335.

13. Sundelin HEK, Stephansson O, Johansson K, Ludvigsson JF: Pregnancy outcome in joint hypermobility syndrome and Ehlers–Danlos syndrome. *Acta Obstet Gynecol Scand* 2017;96:114-119.

14. Wang S-M, Dezinno P, Maranets I, Berman MR, Caldwell-Andrews AA, Kain ZN: Low back pain during pregnancy. *Obstet Gynecol* 2004;104: 65-70.

15. Gross GA, George JW: Orthopedic injury in pregnancy. *Clin Obstet Gynecol* 2016;59(3):629-638.

16. Sihvonen T, Huttunen M, Makkonen M, Airaksinen O: Functional changes in back muscle activity correlate with pain intensity and prediction of low back pain during pregnancy. *Arch Phys Med Rehabil* 1998;79: 1210-1212.

17. Gutke A, Östgaard HC, Öberg B: Association between muscle function and low back pain in relation to pregnancy. *J Rehabil Med* 2008;40: 304-311.

18. Norén L, Östgaard S, Johansson G, Östgaard HC: Lumbar back and posterior pelvic pain during pregnancy: A 3-year follow-up. *Eur Spine J* 2002;11:267-271.

19. Sanderson PL, Fraser RD: The influence of pregnancy on the development of degenerative spondylolisthesis. *J Bone Joint Surg Br* 1996;78:951-954.

20. Stuge B, Lærum E, Kirkesola G, Vøllestad N: The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: A randomized controlled trial. *Spine (Phila Pa 1976)* 2004;29:351-359.

21. Thabah M, Ravindran V: Musculoskeletal problems in pregnancy. *Rheumatol Int* 2015;35:581-587.

22. Shiri R, Coggon D, Falah-Hassani K: Exercise for the prevention of low back and pelvic girdle pain in pregnancy: A meta-analysis of randomized controlled trials. *Eur J Pain* 2018;22:19-27.

23. Sakamoto A, Nakagawa H, Nakagawa H, Gamada K: Effects of exercises with a pelvic realignment device on low-back and pelvic girdle pain after childbirth: A randomized control study. *J Rehabil Med* 2018;50:914-919.

24. Cohen A, Kamanda-Kosseh M, Dempster DW, et al: Women with pregnancy and lactation–associated osteoporosis (PLO) have low bone remodeling rates at the tissue level. *J Bone Miner Res* 2019;34: 1552-1561.

25. Gehlen M, Lazarescu AD, Hinz C, et al: Long-term outcome of patients with pregnancy and lactation-associated osteoporosis (PLO) with a particular focus on quality of life. *Clin Rheumatol* 2019;38: 3575-3583.

26. Kovacs CS: Maternal mineral and bone metabolism during pregnancy, lactation, and post-weaning recovery. *Physiol Rev* 2016;96: 449-547.

27. Kovacs CS, Ralston SH: Presentation and management of osteoporosis presenting in association with pregnancy or lactation. *Osteoporos Int* 2015;26:2223-2241.

28. Jia P, Wang R, Yuan J, et al: A case of pregnancy and lactation-associated osteoporosis and a review of the literature. *Arch Osteoporos* 2020;15:94.

29. Shnaekel KL, Magann EF, Ahmadi S: Pubic symphysis rupture and separation during pregnancy. *Obstet Gynecol Surv* 2015;70:713-718.

30. Smith MW, Marcus PS, Wurtz LD: Orthopedic issues in pregnancy. *Obstet Gynecol Surv* 2008;63:103-111.

31. Amorosa LF, Amorosa JH, Wellman DS, Lorich DG, Helfet DL: Management of pelvic injuries in pregnancy. *Orthop Clin North Am* 2013;44:301.

32. Oliveira GAD, Bernardes JM, Santos ES, Dias A: Carpal tunnel syndrome during the third trimester of pregnancy: Prevalence and risk factors. *Arch Gynecol Obstet* 2019;300:623-631.

33. Meems M, Truijens S, Spek V, Visser LH, Pop VJ: Prevalence, course and determinants of carpal tunnel syndrome symptoms during pregnancy: A prospective study. *BJOG* 2015;122:1112-1118.

34. Meems M, Truijens SEM, Spek V, Visser LH, Pop VJM: Follow-up of pregnancy-related carpal tunnel syndrome symptoms at 12 months postpartum: A prospective study. *Eur J Obstet Gynecol Reprod Biol* 2017;211:231-232.

35. Goel R, Abzug JM: de Quervain's tenosynovitis: A review of the rehabilitative options. *Hand (N Y)* 2015;10:1-5.

36. Peters-Veluthamaningal C, van der Windt DA, Winters JC, Meyboom-de Jong B: Corticosteroid injection for de Quervain's tenosynovitis. *Cochrane Database Syst Rev* 2009:CD005616.

37. Ippolito JA, Hauser S, Patel J, Vosbikian M, Ahmed I: Nonsurgical treatment of de Quervain tenosynovitis: A prospective randomized trial. *Hand (N Y)* 2020;15:215-219.

38. Schned ES: DeQuervain tenosynovitis in pregnant and postpartum women. *Obstet Gynecol* 1986;68:411-414.

39. Ilyas AM, Ilyas A, Ast M, Schaffer AA, Thoder J: de Quervain tenosynovitis of the wrist. *J Am Acad Orthop Surg* 2007;15:757-764.

40. Akram J, Matzen SH: Rectus abdominis diastasis. *J Plast Surg Hand Surg* 2014;48:163-169.

41. Beer GM, Schuster A, Seifert B, Manestar M, Mihic-Probst D, Weber SA: The normal width of the linea alba in nulliparous women. *Clin Anat* 2009;22:706-711.

42. Boissonnault JS, Blaschak MJ: Incidence of diastasis recti abdominis during the childbearing year. *Phys Ther* 1988;68:1082-1086.

43. Chiarello CM, Falzone LA, McCaslin KE, Patel MN, Ulery KR: The effects of an exercise program on diastasis recti abdominis in pregnant women. *J Womens Health Phys Therap* 2005;29:11-16.

44. Khan KM, Bø K, Dooley M, et al: Exercise and pregnancy in recreational and elite athletes: 2016/17 evidence summary from the IOC Expert Group Meeting, Lausanne. Part 3—exercise in the postpartum period. *Br J Sports Med* 2017;51:1516-1525.

45. Gruszczyńska D, Truszczyńska-Baszak A: Exercises for pregnant and postpartum women with diastasis recti abdominis–literature review. *Adv Rehabil* 2018;32.

46. Louis-Charles K, Biggie K, Wolfinbarger A, Wilcox B, Kienstra CM: Pelvic floor dysfunction in the female athlete. *Curr Sports Med Rep* 2019;18:49-52.

47. Wallace SL, Miller LD, Mishra K: Pelvic floor physical therapy in the treatment of pelvic floor dysfunction in women. *Curr Opin Obstet Gynecol* 2019;31:485-493.

48. Van Geelen H, Ostergard D, Sand P: A review of the impact of pregnancy and childbirth on pelvic floor function as assessed by objective measurement techniques. *Int Urogynecol J* 2018;29:327-338.

49. Brostrøm S, Lose G: Pelvic floor muscle training in the prevention and treatment of urinary incontinence in women–what is the evidence? *Acta Obstet Gynecol Scand* 2008;87:384-402.