INTRODUCTION — In the past two decades, a large quantity of data has provided evidence that historical, restrictive guidelines for exercise in pregnancy can and should be modified. In the absence of medical or obstetrical complications, pregnant women should be encouraged to continue and maintain an active lifestyle during their pregnancies. Regular aerobic exercise during pregnancy appears to maintain or improve physical fitness [1]. Maternal physiological adaptations to pregnancy should be considered, however, and some modification in exercise routines or activity patterns may be necessary because of the normal anatomical changes in pregnancy and fetal requirements.

Current information on physiological responses to exercise during pregnancy, including physiological variables that are evaluated during exercise, will be reviewed here. These variables are assessed to determine fitness, ability to engage in various forms of exercise, and corresponding metabolic adjustments. Recommendations for exercise during pregnancy and the puerperium are discussed separately. (See "Recommendations for exercise during pregnancy and the postpartum period" and "Exercise physiology").

ANATOMICAL CHANGES — Pregnancy results in anatomical changes or conditions which could have an impact on a woman's ability to engage in physical activities. The most significant pregnancy induced change is the expansion of the uterus to accommodate the growing fetus and products of conception. The uterus expands from a strictly pelvic organ before 12 weeks to become an abdominal organ for the remainder of gestation.

The expanding uterus causes an anterior cephalad shift in the center of gravity and progressive lumbar lordosis. Lumbar lordosis leads to low back pain in 40 to 50 percent of all pregnant women [2]. To compensate for the lumbar lordosis, pregnant women increase the anterior flexion of the cervical spine, resulting in kyphosis. Prominent kyphosis can be associated with paresthesias and motor weakness over the distribution of the ulnar and/or median nerve [3].

Although prominent lordosis and kyphosis can interfere with the ability to engage in physical activities, physical therapy, weight control, and specific exercise routines can minimize or prevent these problems. Avoiding excessive weight gain is important because a 20 percent weight gain will increase the biomechanical force exerted on a joint by as much as 100 percent, thereby increasing the risk of joint injuries during certain physical activities [4]. Physical therapy and exercises that strengthen muscles at major joints may prevent or minimize this risk. (See "Weight gain in pregnancy").

Laxity of joints varies from one individual to another. Most pregnant women experience a widening of 10 mm of the pubic symphysis beginning at 10 to 12 weeks of gestation, and 1 in 37 women will also experience pubic symphysis regional pain, which is occasionally incapacitating [5]. (See "Musculoskeletal changes and pain during pregnancy").

Soft tissue edema of the lower extremities affects as many as 80 percent of pregnant women by term [4]. Edema can cause nerve entrapment with related compression neuropathies, most commonly affecting the median nerve (carpal tunnel syndrome). Compression of the common peroneal nerve may occur at the fibular head. Nerve compression can be exacerbated during labor/delivery. (See "Neurologic disorders complicating pregnancy", section on 'Neuropathy").
**ENERGY NEEDS** — For normal weight pregnant women (body mass index [BMI] 20 to 28 kg/m(2)), approximately 300 extra kilocalories (Kcal) per day are required to meet the metabolic needs of pregnancy after the first trimester [6]. The demand is unevenly distributed: approximately 390 Kcal/day at 20 to 30 weeks of gestation versus 250 Kcal/day at 30 to 40 weeks of gestation. Recommendations for weight gain in pregnancy have been published by the Institute of Medicine and are described in detail separately. (See "Weight gain in pregnancy".

However, these recommendations did not account for energy intake and energy requirements of active versus sedentary women. The caloric requirement is higher when daily energy expenditure is increased through exercise. Depending on the activity and intensity of exercise, an additional 350 to 500 cal/hour intake may be required [7].

In the laboratory, an estimate of the increased requirement can be made based upon the metabolic equivalent (MET), a unit of resting oxygen uptake. One MET is defined as 3.5 mL O2 uptake/kg per min, which is the resting oxygen uptake in a sitting position; it is roughly equivalent to 1 Kcal per kg body weight per hour. As an example, a 70 kg woman who briskly walks (3 to 4 METs) for one-half hour per day would increase her caloric requirement by 105 to 140 Kcal. During weight-bearing exercise, such as walking, the caloric requirement will progressively increase with the increase in weight during the course of the pregnancy. (See "Exercise assessment and measurement of exercise capacity in patients with coronary heart disease", section on 'Peak exercise capacity'.)

Carbohydrate requirements — Pregnant women should consume diets high in complex carbohydrates and avoid intake of large quantities of concentrated carbohydrates over brief periods of time.

Carbohydrate metabolism in the later part of pregnancy is directed toward supplying glucose and amino acids to the growing fetus, while providing extra free fatty acids, ketones, and glycerol as sources of maternal fuel. Adequate carbohydrate intake is important. Pregnant women utilize carbohydrates at a greater rate, both at rest and during exercise, than nonpregnant women [8,9]. Increased carbohydrate metabolism coupled with lower glycogen reserves could result in lower blood glucose concentrations, which predispose exercising pregnant women to hypoglycemia [10]. Indeed it has been demonstrated that hypoglycemic levels can be achieved during prolonged exercise of 45 to 60 min at 60 percent of VO(2) max (figure 1) [9]. (See "Maternal endocrine and metabolic adaptation to pregnancy", section on 'Glucose metabolism'.)

Increased tissue storage of fat occurs at rest; exercise induces higher triglyceride levels, but no change in protein utilization [10]. The amount of carbohydrate metabolized during exercise is commensurate with the level of intensity and there appears to be preferential use of carbohydrates during nonweight-bearing exercise during pregnancy, possibly due to the anaerobic component of this type of activity [11]. (See "Exercise physiology".)

**CARDIOVASCULAR ADAPTATION** — Pregnancy induces profound alterations in maternal hemodynamics, including increases in blood volume, heart rate, and stroke volume, and a decrease in systemic vascular resistance (figure 2A-C) [6,12,13]. These hemodynamic changes establish the circulatory reserve necessary to provide nutrients and oxygen to both mother and fetus at rest and during exercise. (See "Maternal cardiovascular and hemodynamic adaptations to pregnancy".)

Pregnancy is characterized by an increase in cardiac output by 30 to 50 percent in singleton gestation; cardiac output is even higher in multiple gestation. Pregnancy is also characterized by a 20 percent increase in heart rate and a decrease of 5 to 10 mmHg in mean arterial pressure followed by a gradual increase to prepregnancy levels. These hemodynamic changes establish the circulatory reserve necessary for oxygen and nutrient supply to the fetus.

The immediate physiologic response that an individual at sea level will experience at altitude is hyperventilation and an increased cardiovascular response. The ventilatory response to altitude
is central to the preservation of oxygen transport. Exercise and pregnancy can exacerbate these responses. During submaximal exercise, however, no alteration in exercise efficiency or response occurs for most variables when moderate altitude and sea level data are compared [14]. Both cardiac output and stroke volume are elevated at moderate altitude at rest, but not during exercise, suggesting a lower reserve for both variables at moderate altitude.

Conflicting evidence exists concerning maternal heart rate response to steady-state submaximal exercise during pregnancy [15,16]. Both blunted and normal responses to weight-bearing and nonweight-bearing exercise have been reported, thus making use of heart rate monitoring to guide exercise intensity during pregnancy difficult. The upper limit in heart rate of 140 bpm during exercise suggested in the past was an arbitrary target set to correspond to a VO2 of 60 percent for women in their reproductive age range, and not based on high quality evidence [17]. It has been suggested that perceptual responses to exercise be used instead. One option is the Borg's 15-point rating of perceived exertion (RPE) Scale. An even simpler and more practical approach is the "talk test," in which moderate exercise intensity is defined as the ability to carry on a normal conversation during exertion or strenuous exercise.

Posture and position — Body posture is an important consideration for the pregnant woman, both at rest and during exercise. After the first trimester, the supine position causes relative obstruction of venous return resulting in decreased cardiac output. Therefore, supine positions should be avoided while resting and during exercise. (See "Maternal cardiovascular and hemodynamic adaptations to pregnancy", section on 'Supine hypotensive syndrome'.)

Motionless standing is also associated with a significant decrease in cardiac output. In one study of 7722 pregnancies, newborns of women who worked in the third trimester weighed 150 to 400 g less than newborns of mothers who remained at home, with the greatest reduction among women who worked in a predominantly standing position, women who were underweight and had a low pregnancy weight gain, and those who were hypertensive [18]. Compared with the left lateral position, cardiac output fell 9 percent in the supine position and 18 percent while standing near term [19]. Motionless standing for prolonged periods of time should be avoided.

RESPIRATORY STATUS — Maternal respiratory adaptation to pregnancy is discussed separately (figure 3A-B). (See "Changes in the respiratory tract during pregnancy".)

During treadmill exercise in pregnancy, arteriovenous 02 differences are decreased [16]. Because of increased resting oxygen requirements and increased work of breathing brought about by physical effects of the enlarged uterus on the diaphragm, there is decreased oxygen available for the performance of aerobic exercise during pregnancy. Both subjective workload and maximum exercise performance are decreased [20,21], although some experts feel pregnant women do not show changes in maximum aerobic power or acid-base balance during exercise when compared to their nonpregnant state [6,22,23]. However, it is generally agreed that aerobic training during pregnancy increases exercise capacity compared to sedentary pregnant controls [24-27].

The anatomical changes that characterize pregnancy also have an effect on the respiratory system during exercise. Pregnant women experience obstructive symptoms secondary to excessive hyperemia, edema and secretions developing during pregnancy. The growing uterus causes a cephalad displacement of the diaphragm that results in an expansion of the chest circumference. These changes lead to an increase in inspiratory capacity of 300 mL (ie, tidal volume minus inspiratory volume) and a reduction in functional residual capacity. During exercise in normal pregnant subjects, there are diminished responses for every respiratory function, reflecting a decrease in pulmonary reserve and inability to exercise anaerobically. During mild exercise, the respiratory frequency of pregnant women is higher than in nonpregnant controls (figure 4) [20], and minute ventilation is slightly increased, while oxygen consumption and tidal volume are comparable for the same type of exercise (figure 4 and figure 5). Pregnant subjects demonstrate a more efficient ventilatory response to moderate exercise (5 to 6 MET) when compared to non-pregnant control subjects, as demonstrated by a trend toward
lower ventilatory equivalent (figure 5). This more efficient response has been attributed to the primary respiratory alkalosis of pregnancy.

**THERMOREGULATORY CONTROL** — Basal metabolic rate and heat production are increased during pregnancy. Maintenance of euhydration is critical to heat balance during prolonged exercise because loss of fluid such as sweat may compromise heat dissipation.

The increase in body temperature during exercise is directly related to the intensity of the exercise. During moderate intensity aerobic exercise in thermoneutral conditions, core temperature of nonpregnant women rises an average of 1.5ºC during the first 30 minutes of exercise and then plateaus if exercise is continued [9]. A steady-state of heat production versus heat dissipation is accomplished via increased conductance of heat from core to periphery via the cardiovascular system, as well as through evaporative cooling (ie, perspiration). If heat production exceeds heat dissipation capacity, as with exercise in hot, humid conditions or during very high intensity exercise, the core temperature will continue to rise. Given the increase in body surface during pregnancy, heat dissipation occurs more rapidly; however, the risk of dehydration increases, as well. Studies have reported that, in response to 60 minutes of prolonged exercise at an intensity of 55 percent of VO2 max, the rectal temperature in pregnant women rose by only 0.6ºC [9].

Possible teratogenic effects — Data regarding the effects of exercise on core temperature during pregnancy are limited [6,9,28]. Fetal temperatures are approximately 1 degree C higher than maternal temperatures (fetal temperature is 0.5 degrees higher than intrauterine temperature which is 0.8 degrees higher than maternal oral temperature). In animal studies, an increase in maternal core temperature greater than 1.5 degree C during embryogenesis is associated with major congenital malformations (eg, neural tube defects, microphthalmia, arthrogryposis, abdominal wall defects, limb deficiencies). These data coupled with human studies suggest that maternal hyperthermia (related to exposure to a hot tub, sauna, or fever) in excess of 39 degrees C during the first 45 to 60 days of gestation may also be teratogenic in humans [29]. Hyperthermia associated with exercise, however, has not been definitively associated with teratogenic consequences in humans.

**FETAL RESPONSE TO MATERNAL EXERCISE** — There is good evidence that the fetus is able to tolerate maternal exercise and that any responses are transient with no lasting adverse effects. Adverse events are unlikely to occur in the uncomplicated gestation. Nevertheless, healthcare providers who prescribe exercise for pregnant women should be cognizant of all potential complications.

Transient maternal hypoxia — Transient maternal hypoxia can cause fetal tachycardia and an increase in fetal blood pressure, as demonstrated in animal studies. These fetal responses are protective mechanisms for the fetus to facilitate transfer of oxygen and decrease CO2 tension across the placenta. Acute alterations result in transient fetal heart rate changes, whereas chronic hypoxia may result in intrauterine growth restriction. (See "Antepartum fetal heart rate assessment", section on 'Cardiovascular response to hypoxia'.)

Fetal heart rate — Exercise induces significant cardiovascular changes. One concern is that selective redistribution of maternal blood flow to the exercising muscles and away from the splanchnic organs could potentially have an adverse effect on the fetus. Although a sustained significant reduction of blood flow to the uterus could lead to hypoxia or asphyxia, no adverse association between maternal exercise and fetal well-being has been demonstrated [20,24,30-35].

The most commonly analyzed fetal response to maternal exercise is a change in the fetal heart rate (FHR). Maternal exercise is generally associated with an increase of FHR by 10 to 30 beats per minute, but fetal bradycardia may occur [30]. Fetal heart rate decelerations and bradycardia have been reported in 8.9 percent of exercising patients [6]. The mechanism leading to fetal
bradycardia is likely related to vagal reflex, cord compression, or fetal head malposition. No lasting effects on the fetus have been reported.

Umbilical blood flow — Doppler velocimetry studies during maternal exercise are technically difficult [36]. Most measurements have been taken before and after exercise, by which time any exercise-induced changes could have returned to normal.

Fetal growth — Epidemiological studies have suggested a link between strenuous physical activities, deficient diets, and lower birth weights, including a decreased risk of giving birth to a large for gestational age infant.

A meta-analysis including 30 studies concluded that the differences in birth weights for newborns of mothers that exercised during pregnancy are minimal when compared with controls. However, women who engaged in strenuous exercise during the third trimester were more likely to deliver newborns that weighed 200 to 400 g less than matched controls [37]. This discrepancy may be explained, in part, by inadequate caloric intake among exercising women in the former studies, although this mechanism has not been proven. Other uncontrolled studies performed in elite athletes have also yielded conflicting results [38,39].

Women who are diet-conscious often do not take in minimum amounts of required nutrients. The combined energy requirements of pregnancy and exercise coupled with caloric restriction may lead to poor weight gain and fetal growth restriction.

Premature labor — There are insufficient data to provide risk assessment of premature labor among exercising mothers. Sociodemographic differences among women with different levels of physical activity may account for reported associations between activity and preterm birth [40]. (See "Risk factors for preterm labor and delivery").

Most pregnant women with increased levels of physical activity are not at increased risk of preterm delivery [41,42]. However, these data do not address the role of activity restriction in the management of women at high risk for preterm labor and birth. Anecdotal clinical observations suggest that these women may have labor triggered by exercise.

**SUMMARY AND RECOMMENDATIONS**

- Pregnancy results in multiple anatomical changes or conditions which can impact a woman's ability to engage in physical activities. (See 'Anatomical changes' above.)
- For normal weight pregnant women (body mass index [BMI] 20 to 28 kg/m(2)), approximately 300 extra kilocalories (Kcal) per day are required to meet the metabolic needs of pregnancy after the first trimester. Depending on the activity and intensity of exercise, an additional 350 to 500 cal/hour intake may be required. (See 'Energy needs' above.)
- Conflicting evidence exists concerning maternal heart rate response to steady-state submaximal exercise during pregnancy. Both blunted and normal responses to weight-bearing and nonweight-bearing exercise have been reported, thus making use of heart rate monitoring to guide exercise intensity during pregnancy difficult. (See 'Cardiovascular adaptation' above.)
- During mild exercise, the respiratory frequency of pregnant women is higher than in nonpregnant controls (figure 4), and minute ventilation is slightly increased, while oxygen consumption and tidal volume are comparable for the same type of exercise (figure 4 and figure 5). (See 'Respiratory status' above.)
- Basal metabolic rate and heat production are increased during pregnancy so maintenance of euhydration is particularly critical to heat balance during prolonged exercise. Hyperthermia associated with exercise has not been definitively associated with teratogenic consequences in humans, but is a theoretical concern. (See 'Thermoregulatory control' above.)
• There is good evidence that the fetus is able to tolerate maternal exercise and that any responses are transient with no lasting adverse effects. (See 'fetal response to maternal exercise' above.)

• Maternal exercise is generally associated with an increase of FHR by 10 to 30 beats per minute, but fetal bradycardia may occur. The mechanism leading to fetal bradycardia is likely related to vagal reflex, cord compression, or fetal head malposition. No lasting effects on the fetus have been reported. (See 'Fetal heart rate' above.)

• Women who engage in strenuous exercise during the third trimester are more likely to deliver newborns weighing 200 to 400 g less than matched controls; there are no to minimal effects from mild exercise. Fetal growth delay may be due to inadequate caloric intake among exercising women. (See 'Fetal growth' above.)

• Most pregnant women who exercise during pregnancy are not at increased risk of preterm delivery. (See 'Premature labor' above.)

REFERENCES

17 Artal, R. Personal communication.