The interactions of exercise and pregnancy: A review

Frederik K. Lotgering, M.D., Ph.D., Raymond D. Gilbert, Ph.D., and Lawrence D. Longo, M.D.

Loma Linda, California

Increasing numbers of women engage in relatively strenuous exercise during pregnancy. The interaction of the increased metabolic demands of physical activity with those of pregnancy is poorly understood. We review what is known and what is not known of the extent to which pregnancy affects a woman's ability to perform strenuous activity and the degree to which exercise affects the pregnant woman, the fetus, and the infant. (Am. J. Obstet. Gynecol. 149:560, 1984.)

When the stress of strenuous physical activity is superimposed upon that of pregnancy, the metabolic demands of the gravid uterus may come in conflict with those of the exercising muscles. This may result in reduced exercise performance of the mother and/or adverse effects, including acute fetal distress. It is also possible that the repeated stress of daily exercise results in fetal growth retardation.

Because little is known about this field we will review present knowledge of the physiology of exercise during pregnancy. The two main questions which we will consider concern first the extent to which pregnancy affects a woman's ability to perform strenuous activity and then the extent to which exercise affects the pregnant woman, the fetus, and the infant. Ancillary questions include the following: (1) To what extent does total maternal oxygen consumption during physical activity differ from that in nonpregnant individuals? (2) Is one's physical working capacity affected by pregnancy? (3) To what extent are uterine blood flow and uterine oxygen consumption altered by exercise? (4) What is the significance of temperature changes for the mother and fetus? (5) To what degree does exercise affect maternal and fetal respiratory blood gases? (6) Is there evidence for acute fetal distress during exercise? (7) Is fetal outcome affected by repeated strenuous physical activity during pregnancy? In this review we will briefly consider these questions. A more complete exposition is given elsewhere.¹

Before dealing with these issues, however, we would like to point out some of the problems associated with the study of the physiologic effects of exercise during pregnancy. First, pregnancy affects maternal body weight, dimensions, composition, and, consequently, the baseline values of many physiologic variables relevant to the study of exercise. Second, these changes also affect the physiologic burden of a given exercise regimen on the individual. This is obvious in weight-bearing exercise (e.g., treadmill, step test). However, the amount of energy required to pedal a bicycle ergometer (non-weight-bearing exercise) may also increase because of fluid accumulation in the legs and perhaps because of other factors. Third, the physiologic response to a fixed exercise regimen shows a wide variation between individuals. By standardizing the exercise level to a percentage of the individual's maximal oxygen consumption rather than to a fixed external task this apparent variation can be minimized. Although this standardization is commonly used in physiologic studies, no one has used it in pregnant women. Fourth, because many variables affect both pregnancy and exercise, few, if any, studies on the combined subject can be considered well controlled. Fifth, only a limited number of studies have been reported under the most strenuous circumstances, i.e., exhaustive exercise near term. Therefore, it is not known to what extent exercise responses differ as a result of pregnancy per se. For similar reasons there are no data on fetal outcome under these circumstances. Sixth, most reliable physiologic data are derived from animal studies. However, the results of such studies may be of limited applicability to humans because quadrupeds (in which most such studies are performed) are less subject

¹From the Division of Perinatal Biology, Departments of Physiology and Obstetrics and Gynecology, School of Medicine, Loma Linda University.

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Reprint requests: Lawrence D. Longo, M.D., Division of Perinatal Biology, School of Medicine, Loma Linda University, Loma Linda, California 92350.
Table I. Oxygen consumption during bicycle exercise in the sitting position

<table>
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<tr>
<th>Author, year</th>
<th>Subjects</th>
<th>Rest Absolute (mL · min⁻¹)</th>
<th>Rest Pregnant-nonnepregnant (%)</th>
<th>Exercise Absolute (mL · min⁻¹)</th>
<th>Exercise Pregnant-nonnepregnant (%)</th>
<th>Exercise-rest Absolute (mL · min⁻¹)</th>
<th>Exercise-rest Pregnant-nonnepregnant (%)</th>
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<tr>
<td>Ueland et al.,' 1973</td>
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<td>13</td>
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Maternal oxygen consumption

During pregnancy resting oxygen consumption increases with advancing gestational age to a maximum value near term of 16% to 32% above nonpregnant values.2, 3 This higher value results largely from the increased uterine tissue mass, including that of the fetus. Maternal oxygen consumption in sheep is increased only 4% above that of the nonpregnant state after subtracting for the total uterine contents,4 and there is no evidence that this is any different in humans. Thus, the metabolic rate of the other maternal tissues is virtually unaffected by pregnancy despite a slight increase in cardiac and respiratory work.

Because oxygen consumption increases with both exercise level and gestational age, the most pronounced increases in oxygen consumption can be expected during maximal exercise near term. Submaximal exercise during late gestation is associated with approximately 10% higher absolute values for oxygen consumption than is exercise during the nonpregnant state, both during weight-bearing exercise in humans5 and goats6 and during non-weight-bearing exercise.2, 3, 5, 7–11

The amount of oxygen required for exercise can be calculated by subtracting the resting oxygen consumption from the total oxygen consumption during and following exercise. During pregnancy higher values have been reported for treadmill exercise,5, 12 as would be expected because of the pregnancy weight increase. In contrast, non-weight-bearing exercise during pregnancy does not consistently increase the oxygen requirements (Table I). This suggests that exercise efficiency is not greatly affected by pregnancy, either by body position and composition or by the metabolic and endocrinologic changes.

Because all "normal" weight-bearing activities during pregnancy require a higher energy output, some training effect seems inevitable unless a more sedentary life style is adopted. In the nonpregnant individual physical training increases maximal oxygen consumption up to 33%. To what extent maximal oxygen consumption is affected by pregnancy is largely unknown. In pregnant ewes oxygen consumption can increase fivefold to sixfold with maximal exercise,13 but whether this increase is greater than that in nonpregnant sheep is unknown. A controlled study in rats showed a nonsignificant increase of about 8% in maximal oxygen consumption in both sedentary rats and in animals trained before pregnancy.14 Rats that were trained both prior to and during pregnancy had 23% higher maximal oxygen consumption values than sedentary rodents, but the training effect prior to pregnancy accounted for 13% of the total.

In addition, two case reports suggest that maximal oxygen consumption increases about 20% in pregnant women who maintain fairly strenuous activity during pregnancy.15, 16

Physical working capacity

If one could extrapolate from the above observations at submaximal work levels in humans, a given maximal task during pregnancy would require about a 10% higher absolute value for maximal oxygen consump-

Fig. 1. Physiologic changes for several functions in response to 40 minutes of exercise at 70% maximal oxygen consumption in pregnant sheep. a: Uterine blood flow (percentage of control value); b: hematocrit (percentage of control value); c: uterine oxygen consumption (percentage of control value); d: maternal and fetal temperatures. Values are means ± SEM (n = 8 in a to c and 6 in d).

This important variable is clearly indicated, particularly in humans.

Uterine oxygen consumption

Uterine blood flow increases with advancing gestational age, although flow decreases somewhat per kilogram of total uterine contents. The flow increase results from a decrease in uterine vascular resistance secondary to vasodilatation associated with increasing concentrations of estrogens produced by the fetoplacental unit and prostaglandins (E2 and L2) produced by the vessel wall. Although the dilated uterine vasculature is less sensitive to the effects of vasoconstrictive agents during pregnancy than in the nonpregnant state, it responds to circulating prostaglandins and catecholamines and to sympathetic stimulation. Uterine blood flow may decrease spontaneously up to 20%, and reductions up to 50% have been reported in response to alkalosis and hyperthermia and to a variety of other stresses. It has been suggested that these vasoconstrictive occurrences are catecholamine-mediated, but this has not yet been demonstrated.

During exercise the redistribution of cardiac output has been thought to result from vasodilatation in exercising muscles, mediated by local metabolic factors, and from sympathetic vasoconstriction in tissues with a high resting flow and low oxygen extraction, such as the splanchnic bed and nonworking muscles. Because the uterine vasculature during pregnancy is sensitive to sympathetic stimulation and catecholamine release, one would also expect a reduction in uterine blood flow during maternal exercise. Using the disappearance of sodium 24 injected into the myometrium, Morris et al. suggested a 25% reduction in flow to the pregnant human uterus during mild short-term bicycle exercise in the supine position. However, this probably represents an overestimate. In the supine position the presence of a large pregnant uterus may well affect uterine blood flow. In addition, animal data show that the myometrium is more sensitive to catecholamines than is the placenta, resulting in flow redistribution within the uterus, favoring coticelary flow at the expense of myometrial flow.

Lotgering et al. studied uterine blood flow at different levels (percentage of maximal oxygen consumption) and durations of exercise in sheep. As shown in Fig. 1, uterine blood flow decreased immediately at the onset of exercise, was significantly below control values throughout the exercise period, and returned to control levels within 10 minutes of recovery. Flow decreased 13% during a 10-minute exercise period at 70% maximal oxygen consumption, 17% during a 10-minute exercise period at 100% maximal oxygen consumption, and 24% near the end of a 40-minute exercise period at 70% maximal oxygen consumption.
Regression analysis showed a significant decrease in flow with exercise time, and flow varied linearly with heart rate, where heart rate is linearly related to the level of exercise. Thus, it is likely that uterine blood flow decreases with both the level and the duration of exercise. Hohimer et al.,25 Clapp,27 and Chandler and Bell28 also have reported decreases in uterine blood flow of up to 36% during exercise. Other investigators24, 29 have concluded that uterine blood flow in sheep remains constant during treadmill exercise. However, their measurements were made shortly after, rather than during, exercise, and uterine flow returns rapidly to control levels when exercise is discontinued.28

Although a reduction in uterine blood flow suggests a reduction in the supply of oxygen and nutrients to the uterus and/or a reduction in oxygen consumption, this result is not necessarily true during exercise, because exercise is associated with a marked rise in maternal hematocrit. During exercise plasma filtrate is forced across the capillary membrane in exercising muscles, resulting in a decrease of up to 14% in plasma volume in man30 and up to 20% in pregnant sheep,26 while the red blood cell mass remains constant.26 This hemoco­ncentration is associated with an increase in hemoglobin concentration (shown as hematocrit in Fig. 1, b) and thus of blood oxygen-carrying capacity. Consequently, the reduction in oxygen delivery (oxygen content \times flow) to the uterus is much smaller than the decrease in blood flow would suggest.26 In addition, in sheep the blood flow is redistributed within the uterus, favoring the placental cotyledons at the expense of the myometrium,24, 25 and oxygen extraction is increased.31 The net result of these compensatory mechanisms is a constant oxygen uptake by the uterus as a whole (Fig. 1, c)27, 28, 31 and by the fetus.27

**Temperature changes**

During exercise the total heat production may increase as much as 20 times resting values. Only 20% to 25% of the added energy expenditure is used for external work, while the remaining 75% to 80% is transferred into heat. Although most of the heat is lost to the environment, some is stored, resulting in increased body temperature. Body temperature increases with the level and duration of exercise. This is accompanied by marked circulatory changes, including increased blood flow to the skin to provide adequate cooling. If heat loss is reduced because of high ambient temperature and/or humidity, the working capacity of an individual will be markedly decreased.21

Under normal resting conditions the temperature of the fetus is about 0.5°C higher than that of the mother in humans32 and sheep.31, 32 Most of the fetal heat is transferred to the mother across the placenta and a smaller proportion is transferred across the fetal skin, amniotic fluid, and uterine wall.32, 33 Recent theoretical studies34 suggest that the maternal body temperature is the major determinant of fetal temperature, while changes in uterine blood flow or fetal metabolism are quantitatively less important.

In studies of exercising sheep from our laboratory,31 the fetal temperature lagged behind the rapidly changing maternal temperature at the onset and cessation of exercise (Fig. 1, d). Consequently the fetal-maternal temperature difference during the onset of exercise was reduced or even reversed, while a larger temperature difference existed following exercise. These changes were more pronounced with higher levels of exercise during which the maternal temperature increases more rapidly. Return of the fetal temperature to control values was slow, requiring more than 1 hour following prolonged (40 minutes) exhaustive exercise at 70% maximal oxygen consumption (Fig. 1, d).

Among the possible physiologic implications of increased body temperature are increased metabolism (Q_{10} effect*), rightward shifts of the maternal and fetal oxyhemoglobin dissociation curves, and a reduction of uterine blood flow. However, the quantitative aspects of these changes are not yet fully understood and require further study.1

**Respiratory blood gases**

Knowledge of the maternal and fetal temperatures is essential for the correct interpretation of fetal blood gas measurements. Blood obtained anaerobically and analyzed for respiratory blood gases at a temperature below that of the body shows a rise in pH and a fall in oxygen and carbon dioxide tensions.30 The failure to correct for a 1°C increase has been estimated to result in about a 1.9 and 2.7 torr underestimate for fetal oxygen and carbon dioxide tensions, respectively, and a pH that is falsely 0.015 units too high.31 All factors which change maternal temperature, uterine or umbilical blood flow, or fetal metabolism may affect the temperature gradient, especially in the non-steady state. Unfortunately, this consideration is often not taken into account in studies of fetal blood gas values.

When the proper temperature corrections are made, maternal oxygen tension and oxygen content increase and carbon dioxide tension decreases as a result of exercise-induced hyperventilation and hemoconcentration. As shown in Fig. 2, in sheep exercised to

*The interdependent physical, chemical, and metabolic processes of the human body may vary with small differences in temperature. This change in activity with temperature is usually expressed as Q_{10}. This is a factor by which the velocity of a reaction at given temperature is multiplied to give the velocity of that reaction at a temperature of 10°C higher. The Q_{10} of physical reactions is very nearly 1.
exhaustion at 70% maximal oxygen consumption, maternal oxygen tension increased 13% and oxygen content increased 25%, while carbon dioxide tension decreased 28%. Consequently the oxygen-carrying capacity of the maternal blood was increased and the oxygen transport across the placenta was enhanced.

Although the reduced uterine blood flow during exercise will tend to lower the placental oxygen transport, both theoretical analysis and observations in our laboratory during acute embolization of the uterine vascular bed suggest only minimal decreases in fetal oxygen tension when uterine blood flow is approximately 60% or more of its normal resting value. Several studies in exercising sheep have reported reductions in fetal arterial oxygen and carbon dioxide tensions of as much as 25%. Failure to correct the blood gas values for the temperature changes largely explains the differences in results among these studies and the smaller changes observed in our studies. In these studies fetal arterial oxygen and carbon dioxide tensions and oxygen content decreased with the level and the duration of exercise, but the values differed significantly from control only when the ewes were run to exhaustion. During prolonged (40 minutes) exhaustive exercise at 70% maximal oxygen consumption fetal aortic oxygen tension decreased 3.0 torr (from 26.2 to 23.2 torr); carbon dioxide tension, 4.5 torr (from 54.1 to 49.6 torr); and oxygen content, 1.5 ml • dl⁻¹ (from 5.8 to 4.3 ml • dl⁻¹), while pH increased 0.02 units. The percent changes are shown in Fig. 2. Theoretical calculations suggest that about 30% of the decrease in oxygen saturation can be accounted for by the temperature and Bohr shifts of the oxyhemoglobin dissociation curve, whereas the remaining 70% of the decrease in oxygen saturation is associated with the 3 torr decrease in oxygen tension. Although fetal oxygen tension and content decrease with exercise, this decrease does not necessarily indicate that the fetal metabolic demands are not met or that the fetus is in "distress."

**Other fetal responses**

Fetal hypoxia and/or "distress" may be associated with changes in any of the following fetal variables: heart rate, blood pressure, cardiac output distribution, hematocrit, and catecholamine concentration.

Several authors have studied the fetal heart rate prior to and following a mild to moderate exercise stress of short duration. They noted only small changes in mean fetal heart rate and heart rate patterns, changes that were inconclusive as to possible "distress." However, it is conceivable that the fetal responses to maternal exercise recover rapidly following such a minimal stress. Recently Dale et al. reported transient fetal bradycardia for about 3 minutes during short-term, moderately strenuous exercise in three subjects. In contrast, Sibley et al. reported a slightly higher fetal heart rate during short-term treadmill exercise as compared to baseline and recovery values, but the mean values were not compared statistically. This is also true for a study in which higher fetal heart rates were found during the recovery from moderately strenuous exercise (1.5 miles of jogging) in seven women. In neither study did the fetal heart rate pattern show any signs of "distress." One study reported on the changes in fetal heart rate both during and following 25 minutes of bicycle exercise at about 70% maximal oxygen consumption. The auscultated heart rate demonstrated only a mean increase of 4 bpm during and following exercise, which is physiologically insignificant. In sheep, one investigator observed an increased fetal heart rate, but others have reported no significant changes during either short-term or prolonged exhaustive exercise.

Animal studies show that the other fetal cardiovascular variables are also largely unaffected by maternal exercise. Fetal arterial blood pressure is unaffected by short-term as well as prolonged exercise in sheep. Cardiac output and cardiac output distribution are unaffected by exhaustive short-term exercise, but cardiac output has not been studied during prolonged exercise in sheep. One study reported a 10% reduction in umbilical blood flow during prolonged exhaustive exercise. However, this observation needs to be confirmed because the reported resting value of 354 ml • min⁻¹•kg⁻¹ was well above accepted normal values. In addition, fetal hematocrit and red blood cell and plasma volumes are unaffected by prolonged exercise.

The number of studies of fetal cardiovascular responses to maternal exercise is limited, but the available...
evidence suggests the absence of hypoxia or "stress." In addition, fetal catecholamine concentrations are not significantly increased in fetal sheep during either short-term$^{31}$ or prolonged exercise.$^{33}, 55, 56$ However, extrapolating these responses from fetal sheep to the human fetus may not be legitimate for the reasons detailed above.

**Fetal outcome**

The effect on fetal outcome of a single factor such as exercise is easily obscured by the wide normal variation in outcome caused by a multitude of variables, including genetic and socioeconomic factors, nutrition, environmental factors, "stress," and so forth. Thus, the question of whether physical activity affects fetal outcome can be answered only by large, well-controlled prospective epidemiologic studies. We know of no such study in pregnant women.

However, several studies concerning this subject are nonetheless worthy of discussion. Clapp and Dickstein$^{57}$ prospectively studied the effects of maternal exercise on fetal outcome in 336 pregnant women, divided into five activity groups on the basis of interview data. They concluded that preconceptional exercise had no effect on fetal outcome but that regular vigorous exercise during pregnancy increased the incidence of low-birth weight infants. However, as the authors pointed out, the findings of their study must be interpreted with caution because of the limited accuracy of the data. In contrast, a high level of voluntary daily exercise or a high "physical fitness score" was found not to be associated with low Apgar scores or low birth weights in humans,$^{58}, 59$ and adverse effects on fetal outcome were not observed in two small prospective studies in which the expectant mothers participated in mild exercise programs.$^{52}, 54$ The effects of strenuous exercise during human pregnancy have been studied mainly in women who were highly physically active prior to pregnancy,$^{16}, 60-67$ and all of these studies report normal or improved fetal outcomes. Although these studies were retrospective and/or uncontrolled, with the women in excellent condition prior to pregnancy, they suggest the absence of major negative effects of strenuous exercise on fetal outcome in healthy women. Strenuous exercise is normal during later gestation in many large game mammals and their predators as well as in some domestic animals.$^{68}$ This suggests that strenuous activity during pregnancy does not adversely affect fetal outcome in healthy individuals of many species.

In contrast, adverse effects of maternal physical activity on fetal outcome have been reported in both humans and laboratory animals. The most commonly mentioned negative effect is low birth weight. Several studies have reported a decrease of as much as 400 gm and an increased incidence of low-birth weight infants in offspring of working mothers.$^{69-72}$ However, this may reflect the poor nutritional status of these women,$^{70}$ or other selection factors rather than represent the effect of physical activity per se. Weight reduction has also been reported in pregnant laboratory animals that were forced to exercise strenuously during pregnancy. An 8% reduction was reported in forcefully exercised mice$^{73}$ and guinea pigs.$^{74}$ Fetal weight in rats forced to swim was 6% lower than normal$^{75}$; however, rats forced to run did not show growth retardation.$^{76}$ A 12% to 20% reduction in fetal weight was also reported in exercised pygmy goats,$^{6}$ but this study may not have been well controlled. In contrast, no reduction in birth weights was observed in swine moderately exercised throughout gestation.$^{77}$ Because fetal growth retardation has been associated not only with increased maternal physical activity but also with restriction of activity,$^{79}$ one cannot exclude the possibility that the "stress" of fear and handling related to forced exercise in a laboratory environment rather than the exercise per se contributed to the growth retardation. A variety of other adverse effects has also been suggested, including increased perinatal mortality and increased incidence of prematurity.$^{60}$ However, further well-matched prospective studies are necessary to confirm any such effects. This is also true for the possible teratogenic effect of increased temperature during exercise in early pregnancy.

It is apparent that exercise might have positive or negative health effects during pregnancy. Fig. 3 presents a scheme of the manner in which this might occur. The ordinate indicates such health effects without numerical values. The product of exercise intensity and duration as a rough approximation of overall work

![Fig. 3. Diagrammatic representation of how exercise intensity and duration might have positive or negative effects on certain physiologic functions or outcomes during pregnancy. (See text for details.)](image-url)
or effort is shown on the abscissa. Line A indicates those effects which are beneficial and tend to increase as a function of exercise intensity and duration. Line B indicates the opposite effects. Line C indicates that for some functions there may be a positive effect at low to moderate exercise levels but that this decreases or reverses at higher levels. Finally, line D suggests that for some functions there is no discernible effect. In addition, the figure suggests that training may shift the curves upward and to the right, while, in contrast, other stresses such as hypoxia, malnutrition, or certain disease states may compete for the subject’s physiologic reserves, shifting the curve downward and to the left.

Comment

Animal studies show that physical activity during pregnancy results in marked cardiovascular adjustments in the mother, including a reduction in uterine blood flow. However, because of simultaneous hemocconcentration and increased oxygen extraction, uterine oxygen consumption remains constant. In contrast to relatively profound physiologic changes in the mother, the changes in the fetus are small. The oxygen tension and oxygen content in the fetal arterial blood decrease slightly, but other fetal variables, including catecholamine concentrations, heart rate, blood pressure, cardiac output, blood flow distribution, and blood volume, remain virtually constant. This suggests that acute exercise does not represent a major hypoxic or other stress to the fetus. Little is known about the physiologic effects of chronic exercise on the fetus. The most likely possible effect is a small reduction in birth weight, at least in some species. Again we wish to stress that one must be careful in extrapolating these animal data to humans. Large, well-controlled prospective epidemiologic studies are necessary to establish possible positive or negative effects in humans. Finally, additional physiologic studies are needed for a more complete understanding of the remarkably effective homeostatic mechanisms in both mother and fetus during maternal exercise.

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