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Maternal and fetal cardiovascular responses to strenuous bicycle exercise

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In a longitudinal study we investigated some cardiovascular responses to strenuous bicycle exercise in 33 healthy women during pregnancy and the postpartum period. The exercise electrocardiogram demonstrated depression of the ST segment in 12% of women in the absence of clinical signs of ischemia, and the incidence of these changes was unaffected by pregnancy. In spite of slightly different blood pressures at rest during the first and second trimesters of pregnancy, the blood pressure response to exercise at approximately 75% VO_{2max} was virtually unaffected by pregnancy. After a maximal bicycle test, the fetal heart rate was increased by an average of 4 beats/min, without a change in pattern. Tocodynamometry suggested a transient increase in uterine activity after maximal exercise in 6% of the tests. These findings support the view that strenuous exercise of limited duration is not harmful to the healthy mother and fetus. (Am J Obstet Gynecol 1992;166:854-9.)

Key words: Pregnancy, maximal exercise, blood pressure, electrocardiogram, fetal heart rate

With ever-increasing numbers of pregnant women wanting to participate in sport activities, the question as to how safe maternal exercise is for mother and fetus becomes more important. With regard to the mother, cardiovascular adjustments to strenuous physical activities could be dangerous because of the added burden to an already hyperdynamic maternal circulation. On the other hand, the low resistance of the dilated vascular bed in pregnancy could have a protective effect on the circulation and depress the maternal blood pressure response to exercise.

Physical exercise of the mother presents a potential threat to fetal health because uterine blood flow is known to decrease progressively with exercise intensity. Results of early studies in healthy pregnant women suggested that even mild to moderate exercise may occasionally be associated with fetal bradycardia during exercise, but this finding was later dismissed as representing an artifact. Animal studies have demonstrated that several compensatory mechanisms act
together to preserve fetal oxygen consumption even during exhaustive exercise. These observations provide the justification for a study of maternal and fetal responses to strenuous exercise in healthy pregnant women.

In this study in healthy pregnant women with uncomplicated pregnancy, we assessed some physiologic responses to strenuous bicycle exercise relevant to the safety of the mother and her fetus: maternal electrocardiogram (ECG), blood pressure, uterine contractility, and fetal heart rate (FHR).

**Material and methods**

We studied 33 women at 16, 25, and 35 weeks' gestation and 7 weeks after delivery. In each study period we measured maternal blood pressure and recorded the ECG at rest and at increasing levels of bicycle exercise until maximal aerobic power was reached; we also recorded FHR and uterine contractility at rest and during recovery. All women were healthy and had uncomplicated singleton pregnancies. The study was approved by the Hospital and University Ethics Committee, and all women included in the study gave their informed consent.

Exercise was performed in an air-conditioned room at 21° C and 55% humidity. All participants underwent a physical and obstetric examination before each test to exclude any abnormality. In addition, we recorded FHR by means of Doppler ultrasound and uterine contractility by external tocodynamometry (model III fetal monitor, Corometrics, Wallingford, Conn.) in the second and third trimesters of pregnancy to ensure fetal well-being.

After 20 minutes of rest in the semisupine position, each woman was asked to sit on a bicycle ergometer (model II FE ergometer 400 L, Mijnhardt, Bunnik, The Netherlands). She was connected to a gas flow meter (Oxycon 4, Mijnhardt) for a study of maximal oxygen consumption on which we reported separately. The volunteer was also connected to an ECG monitor (model RM 102, Honeywell, Best, The Netherlands) and a bipolar ECG, lead CM9, from manubrium to the left precordial position (V5), was continuously recorded (with a Cardiostat, Siemens, Stockholm). We measured blood pressure at 3-minute intervals with a standard sphygmomanometer (Erka-sphygmomanometer, Bad Tolz, Germany) and auscultation using Korotkoff sounds I and V to indicate systolic and diastolic blood pressures, respectively. After 5 minutes of baseline measurements at rest on the ergometer, the woman started to exercise. Three minutes of warming up at 30 W was followed by stepwise increments in exercise intensity of 10 W every 30 seconds until maximal oxygen consumption (VO2max) was reached. This was followed by 5 minutes of cooling down at 10 W. The cardiotocogram could not be recorded reliably during exercise, so it was recorded during 20 minutes of recovery with the woman in a semisupine position.

The ECGs were evaluated according to previously established criteria. Mean blood pressures were calculated as \( \frac{1}{3} \times (\text{Systolic pressure} + 2 \times \text{Diastolic pressure}) \). The cardiotocograms were evaluated in a blinded fashion with the use of the Fisher score. In addition, we calculated the average basal FHR during the last 5 minutes of rest and the first 5 minutes of recovery with the woman in a semisupine position and measured the frequency of contractions.

For each test period and each variable under consideration we computed mean values and standard errors of the mean. Analysis of variance and Student t tests were used to assess differences between paired variables. A p value of <0.05 was taken as the level of significance.

**Results**

The 33 women who participated in the study, 23 nulliparous and 10 parous, remained healthy, with uncomplicated pregnancies, throughout the study period, and they were delivered of healthy infants. Maternal age at the time of delivery was 30.9 ± 0.7 years (mean ± SE), gestational age was 40.3 ± 0.2 weeks, and birth weight was 3.43 ± 0.08 kg. Birth weight for gestational age, corrected for parity and fetal sex, was between the 10th and 90th percentiles of the reference curve in 28, <10th percentile in four, and >90th percentile in one infant.

All subjects were studied at 16 ± 1.0, 25.3 ± 0.7, and 35.0 ± 0.6 weeks of pregnancy and at 6.7 ± 1.4 weeks after delivery. Mean body weight at 16 weeks' gestation was 68.0 ± 1.7 kg, not different from the postpartum control value of 67.6 ± 1.9 kg, but it increased significantly with advancing gestational age to 71.8 ± 1.8 and 75.3 ± 1.8 kg at 25 and 35 weeks' gestation, respectively.

The values of heart rate at rest and at maximal exercise have been previously reported. Heart rate at rest increased significantly with advancing gestational age from 87 ± 2 beats min⁻¹ at 16 weeks to 89 ± 2 beats min⁻¹ at 25 weeks and to 94 ± 2 beats min⁻¹ at 35 weeks' gestation, as compared with 83 ± 2 beats min⁻¹ post partum. Heart rate showed a linear increase with exercise intensity to a maximum of 174 ± 2 beats min⁻¹ throughout pregnancy and 178 ± 2 beats min⁻¹ post partum, as shown in Fig. 1. Analysis of the resting ECGs for abnormal changes in conduction, repolarization, and rhythm showed no anomalies in 30 women; two demonstrated sinus arrhythmia and one had an ectopic atrial rhythm. During exercise, 28 women had a normal ECG throughout pregnancy and post partum; four subjects showed...
depression of the ST segment of ≥0.1 mV, accompanied by an inverted T wave in one subject. Sinus arrhythmia disappeared during exercise in the two women who showed this abnormality at rest, but the ectopic atrial rhythm persisted in one subject. The ECG abnormalities during exercise in these women occurred consistently in all pregnancy and postpartum test periods. None of the 33 women had signs or symptoms suggestive of cardiac ischemia.

Mean values of systolic, diastolic, and mean arterial pressures at rest and at approximately 75% \( \text{VO}_2\text{max} \) are shown in Table I. Systolic blood pressures at rest appeared to be unaffected by pregnancy, whereas diastolic and mean pressures were slightly lower during the first and second trimesters of pregnancy as compared with values obtained in the third trimester and post partum. Systolic and mean arterial pressures showed a linear increase with exercise intensity (Fig. 1). At approximately 75% \( \text{VO}_2\text{max} \) systolic and mean arterial pressures were significantly increased by an average of 36% and 17%, respectively, whereas diastolic pressures increased marginally by an average of 2%. Mean values of exercise intensity and systolic blood pressure were similar and values of diastolic pressure were slightly lower in the first and second trimesters than in the postpartum period. During the third trimester the average exercise intensity at the time of the measurement was 5% higher and the systolic blood pressure during exercise was 4% higher than in the postpartum period, whereas the diastolic pressure was similar. Except for a marginal difference at 16 weeks' gestation, calculated mean blood pressures during ex-

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**Fig. 1.** Rise in heart rate and systolic and mean blood pressures at increasing levels of exercise intensity and absence of change in diastolic blood pressure. ○, First trimester; □, post partum.
Table I. Effect of pregnancy on blood pressure at rest and during strenuous bicycle exercise

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>Exercise intensity (% (V_{O_2\text{max}}))</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systolic</strong></td>
<td><strong>Mean</strong></td>
</tr>
<tr>
<td>At rest</td>
<td></td>
</tr>
<tr>
<td>16 wk</td>
<td>110.0 ± 1.8</td>
</tr>
<tr>
<td>25 wk</td>
<td>113.0 ± 1.7</td>
</tr>
<tr>
<td>35 wk</td>
<td>114.8 ± 1.9</td>
</tr>
<tr>
<td>Post partum</td>
<td>112.9 ± 1.6</td>
</tr>
<tr>
<td>During exercise</td>
<td></td>
</tr>
<tr>
<td>16 wk</td>
<td>150.3 ± 2.7</td>
</tr>
<tr>
<td>25 wk</td>
<td>153.9 ± 3.0</td>
</tr>
<tr>
<td>35 wk</td>
<td>158.0 ± 3.0*</td>
</tr>
<tr>
<td>Post partum</td>
<td>151.5 ± 2.7</td>
</tr>
</tbody>
</table>

Values are mean ± SE. Exercise values are significantly increased above resting values \((p < 0.01)\) for all variables except for diastolic blood pressure.

* \(p < 0.01\), compared with postpartum control values \((n = 33)\).
† \(p < 0.05\), compared with postpartum control values \((n = 33)\).

exercise in pregnancy were not different from the postpartum value.

Mean FHR during the first 5 minutes after exercise showed a small but significant increase relative to the resting control value observed in the second and third trimesters; in the second trimester it rose from 143.9 ± 0.9 to 148.7 ± 1.5 beats • min⁻¹ and in the third trimester from 140.7 ± 1.4 to 144.2 ± 1.8 beats • min⁻¹. The Fisher score could not be established in 10 of the 33 subjects at 25 weeks’ gestation because of poor quality of tracings, whereas all 20-minute tracings except one were satisfactory at 35 weeks’ gestation. Average scores after exercise were not significantly different from those obtained at rest, both at 25 weeks’ gestation (9.7 ± 0.2 and 9.9 ± 0.1, respectively) and at 35 weeks of pregnancy (9.5 ± 0.2 and 9.6 ± 0.2, respectively). At 25 weeks of pregnancy 22 of the remaining 23 fetuses had an optimal score of 8 to 10 points both at rest and after exercise. In one case the score changed from optimal (8 points) at rest to suboptimal (7 points) after exercise. At 35 weeks 30 fetuses were scored as optimal both before and after exercise, one changed from optimal (8 points) to suboptimal (7 points), and one changed from suboptimal (7 points) to optimal (10 points).

Uterine activity was recorded reliably from all subjects in both trimesters. At 25 weeks’ gestation 32 of the 33 women showed no uterine activity before or after exercise; in one woman uterine contractility after exercise was not present at rest but was recorded after exercise. At 35 weeks’ gestation 27 women had no uterine contractility either before or after the bicycle exercise, three women showed some uterine contractility both before and after exercise, and three women showed a few irregular contractions after exercise, which subsided spontaneously. None of the women noticed the recorded contractions or progressed into labor.

Comment

We observed depression of the ST segment in 12% of the pregnant women studied; similar or much higher figures of up to 75% have been observed in other studies in healthy pregnant women. The cause of these repolarization abnormalities remains unknown; most women with ST depression during exercise testing have angiographically normal coronary arteries. It has been suggested that the ECG changes could be due to altered sympathetic regulation, and one might speculate that ECG abnormalities during exercise may occur more frequently in pregnant women because pregnancy may affect sympathetic regulation. Our findings do not suggest that pregnancy markedly increases the incidence of repolarization abnormalities or arrhythmias in exercising women, but our study does not rule out a possible effect of pregnancy. Our data seem to support, however, the suggestion that exercise electrocardiography is of limited value in healthy pregnant or nonpregnant women.

We used sphygmanomanometry and auscultation for the measurement of arterial blood pressure. This method has known limitations with respect to accuracy and precision, in the nonpregnant and pregnant state. However, in this longitudinal study the errors inherent in the method of blood pressure measurement may be expected to have been similar in all test periods, and for that reason it seems unlikely that they have affected pregnancy trends. In the first and second trimesters of pregnancy we found lower diastolic pressures at rest as compared with those observed in the third trimester.
and post partum but no significant differences in systolic blood pressure. This is in agreement with the literature and is thought to reflect a reduction in systemic vascular resistance. Systolic arterial pressure increases with exercise intensity, whereas diastolic pressure increases only slightly. Some authors reported a higher systolic pressure response to exercise in pregnant women than in nonpregnant ones, whereas others observed no difference. We found that absolute values of diastolic and mean arterial pressure during exercise were slightly lower during the first and second trimesters of pregnancy, which may reflect the lower resting values observed during these periods. Systolic blood pressure during exercise differed from that in the post-partum period only in the third trimester of pregnancy, when it was significantly increased by 4%, or 6 mm Hg. This may reflect the cumulative effect of the 2% higher systolic pressure at rest and the 5% higher exercise intensity during the third trimester in this study rather than a physiologically important difference in response. The observation that the blood pressure response to exercise is virtually unaffected by pregnancy in spite of the hyperdynamic circulation suggests that the pressure response is governed by local demand.

The FHR response to moderately strenuous maternal exercise has been investigated repeatedly since it was proposed as a clinical test for uteroplacental insufficiency. We consistently observed an elevated basal FHR after the maximal bicycle test. This probably reflects the increase in body temperature rather than fetal distress and confirms similar observations after less strenuous exercise. Although abnormal heart rate patterns, including bradycardia, have been reported during exercise, other studies have adduced evidence that these abnormal patterns are artifacts caused by rhythmic maternal movements during exercise. During recovery from exercise such artifacts are less likely to occur. Fetal bradycardia has been reported to persist occasionally for 4 minutes after exercise, and one study reported a 19% incidence of fetal bradycardia (defined as a heart rate <110 beats/min for ≥10 seconds) within a few minutes after maximal maternal exercise before return to a normal heart rate. One may only speculate as to what may have caused these changes. We were unable to record FHR reliably during the bicycle test but found the pattern unchanged after exercise and 5 minutes of cooling down. We did not measure FHR within 5 minutes of maximal effort. However, with the use of invasive techniques it has been demonstrated that even exercise at the point of exhaustion has little effect on FHR in experimental animals and that fetal blood gas values remain within normal limits.

We observed uterine contractility after exercise in some women. This is in agreement with observations by others who reported either occasional slight increases or no change in uterine contractility in response to physical activity, without progression into labor.

In conclusion, we obtained no evidence that strenuous exercise of limited duration in pregnancy markedly modulates the ECG or blood pressure response or affects the FHR pattern. The safety to the healthy mother and fetus of strenuous exercise of long duration remains to be studied.

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