Effects of acute and chronic maternal exercise on fetal heart rate

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Webb, Katherine A., Larry A. Wolfe, and Michael J. McGrath. Effects of acute and chronic maternal exercise on fetal heart rate. J. Appl. Physiol. 77(5): 2207–2213, 1994.—Maternal-fetal effects of cycle ergometer conditioning (heart rate of 145 beats/min at 25 min/day for 3 days/wk) were studied during the second and third pregnancy trimesters. Subjects were 22 previously sedentary women and 16 nonexercising pregnant control women. Fetal heart rate (FHR) characteristics were studied before, during, and after 15 min of upright cycling at a maternal heart rate target of 145 beats/min at the end of both the second and third trimesters. Despite higher cycling power outputs in the exercised group, mean FHR responses were similar in both groups and conformed to 1) gradual increase in FHR baseline during exercise, 2) normal variability, and 3) normal reactivity. Fetal bradycardia was observed during (n = 1) and after (n = 2) exercise in three isolated tests. The timing of these events suggested that the likelihood of significant fetal hypoxia is highest in the immediate postexercise period. These results also support the hypothesis that physically conditioned women can perform at higher exercise power outputs than sedentary women without inducing fetal hypoxic stress. Further study is recommended to examine possible fetal and placental adaptations to maternal aerobic conditioning.

pregnancy; physical conditioning; gestational age; fetal bradycardia

The study of fetal heart rate (FHR) responses to maternal exercise has at least two important applications. First, studies of laboratory animals (10, 19, 20) indicate that strenuous maternal exercise can result in reduced uterine blood flow caused by redistribution of cardiac output toward contracting maternal skeletal muscle. This effect and the attendant risk of fetal hypoxia also appear to be related to both the intensity and duration of maternal exercise (10). For ethical reasons, parallel studies of healthy human subjects require the use of noninvasive methodologies. In this regard, FHR can be determined using safe and reliable methods and deviations from normal FHR patterns are correlated with fetal hypoxia. Depending on the degree of fetal hypoxia, these reactions can include tachycardia, bradycardia, increased frequency of FHR decelerations, loss of FHR accelerations, and reduction in FHR variability (13). Reliable data on FHR responses to different modalities, intensities, and durations of maternal exercise will help determine the safe limits for maternal occupational and recreational activity.

As described in recent reviews (11, 19), substantial knowledge gaps remain concerning the effects of acute maternal exercise on FHR characteristics. Clarification is needed concerning the true incidence of exercise-induced fetal bradycardia under different exercise conditions and the effect of maternal exercise on FHR accelerations (a sign of fetal well-being) and long-term FHR variability (a correlate of intact fetal central nervous system function). Many of these knowledge gaps are the result of methodological problems in previous studies. These include the use of inadequate FHR recording methods, lack of standardization of FHR analysis criteria, failure to accurately quantify the exercise stimulus, and the study of heterogeneous subject samples that include both obstetric patients and apparently healthy women (19).

Another important application of human FHR studies is to determine the effects of maternal physiological condition on fetal well-being during and after acute exercise stress. The majority of existing studies have focused on the detection of fetal hypoxia in association with single exercise bouts. However, recent investigations confirmed that aerobic conditioning can improve maternal metabolic and cardiopulmonary reserve (11, 20) and may augment maternal fetoprotective capacities. The possibility that chronic maternal exercise may also cause fetal and/or placental adaptations that will increase fetal tolerance for hypoxic stress (8, 19) has not been examined in any systematic way.

The purpose of the present study was to examine, using a controlled longitudinal design, the effects of acute and chronic maternal exercise on FHR characteristics (i.e., FHR baseline and periodic features) before, during, and after an acute bout of submaximal exercise.

METHODS

Subjects. Pregnant subjects were recruited via newspaper advertisements, posted announcements, and contact with local obstetricians in Kingston, Ontario, Canada. Prospective subjects were screened medically by the obstetricians monitoring their pregnancies. Specific exclusion criteria included smoking before or during pregnancy, regular participation in recreational or occupational physical activity before or during pregnancy, presence of metabolic or cardiopulmonary diseases or conditions, maternal obesity or eating disorders, presence of either absolute or relative contraindication to exercise in pregnancy (1, 20), and taking of medications other than prenatal vitamins. Qualifying subjects then chose voluntarily to participate as members of a physical conditioning (exercised) group or a control group that remained sedentary. Written informed consent was obtained before participation in data collection. The study design and informed consent form were approved by an institutional human ethics committee.

Study design. The experimental group participated in a supervised physical conditioning program during the second (Tm2) and third (Tm3) trimesters and detrained during the immediate 3-mo postpartum period. The control group remained sedentary during the same time periods. Data collection was conducted at the start of Tm2 (entry/preconditioning), after both Tm2 and Tm3 (postconditioning), and 3-mo postpartum (nonpregnant control). Subjects in both groups en-
tered the study on a staggered time schedule, and each subject was studied for a 9-mo time span.

Physical conditioning program. The physical conditioning sessions were conducted 3 days/wk by a qualified instructor and included both aerobic and muscular conditioning components (20). Aerobic conditioning consisted of upright stationary cycling at 70% of age-predicted maximal heart rate (HR; 140–150 beats/min). Exercise duration was increased during Tm2 from 14 to 25 min/session and was held constant during Tm3. Exercise records were kept by the class instructor for each subject and included class attendance and cycling power output, steady-state HR during cycling, and exercise duration for each conditioning session. The total duration of the exercise program was 20 wk.

Measurements. Measurements at each data collection time included evaluation of physical characteristics (body weight, the sum of 4 skinfold thicknesses, resting electrocardiogram, echocardiogram, lung function, and blood pressure). Skinfold sites were biceps, triceps, subscapula, and suprailium. Exercise metabolism and cardiopulmonary function were studied using a three-stage submaximal cycle ergometer test. Metabolism and respiratory function were evaluated using open-circuit spirometry, and cardiac output was measured via the indirect CO2 rebreathing method. Maternal HR responses were determined electrocardiographically. Power output at HR of 150 beats/min (PWC150) was then calculated as an index of aerobic working capacity (14).

FHR studies. Studies of FHR during maternal exercise were conducted at Kingston General Hospital, a university-affiliated level III perinatal referral center, at Tm2 and Tm3. Specific times for these tests were 28 ± 1 wk (range of 24–31 wk) and 37 ± 1 wk (range of 36–38 wk) of gestation, respectively. Subjects avoided heavy meals, caffeine intake, and physical exertion for ≥3 h before testing. FHR was recorded using a Doppler ultrasound cardiotocograph (model 8040-A, Hewlett-Packard). Each exercise test session included continuous recording of FHR baseline at rest in the sitting position for 20 min, during 15 min of upright cycling (where feasible) at a target pulse rate of 140–150 beats/min, and for 20 min of postexercise recovery in the sitting position. Maternal IHR was monitored electrocardiographically and recorded during each minute of the protocol. Maternal blood pressure was determined by auscultation at 5-min intervals. To ensure accurate evaluation of FHR recordings obtained during exercise (12), cycling was interrupted for 10–20 s after both 5 and 10 min of cycling. The obtained exercise FHR recordings were considered acceptable for analysis if they were continuous with data obtained during nonexercising periods.

Specific criteria for the analysis of obtained FHR tracings were as described by Wolfe et al. (19). Variables evaluated include FHR baseline (normal range of 120–160 beats/min), baseline variability (normal range of >5 beats/min; protocol scoring options were 0, 3, 5, 6, 10, 15, 20, and 25 beats/min), presence of normal reactivity (FHR acceleration of >15 beats/min for >15 s), frequency of FHR deceleration (transient FHR reduction of >15 beats/min from previous FHR baseline for >15 s), and incidence of bradycardia (FHR baseline of <120 beats/min for >2 min) or tachycardia (FHR baseline of >160 beats/min for >2 min). FHR baseline during exercise was estimated to the nearest 5 beats/min at the start of exercise; at 5, 10, and 15 min of exercise; and immediately (0–30 s) after exercise. Postexercise FHR baseline was measured on a minute-by-minute basis as the predominate FHR of each minute. These analyses were conducted by an experienced observer without knowledge of the identities of the subjects, group assignments, or the trimester of FHR recording.

Pregnancy outcome. Pregnancy outcome data were obtained from medical charts recorded at delivery by qualified medical personnel. Variables examined included infant birth weight, length of labor and its phases, Apgar scores, and perinatal complications (19).

Statistical analyses. Physical characteristics of the exercised and control group at entry were compared using unpaired Student's t-test. Changes in maternal physical characteristics and responses to exercise during the experimental period were analyzed using analysis of variance for repeated measures. Student-Newman-Keuls multiple range tests were used to compare main effect means when significant F ratios were obtained. Time sequence analyses of changes in FHR from preexercise baseline values were conducted using analysis of variance for repeated measures. Where applicable, group and trimester differences were examined using Student-Newman-Keuls multiple range tests. The results of all statistical tests were considered significant if P < 0.05.

RESULTS

Subjects. Twenty-seven members of the exercised group and 22 members of the control group completed the 9-mo experimental protocol. Reasons for dropping out from the exercised group included moving from the area (n = 4), lack of interest (n = 1), and an obstetric complication unrelated to exercise participation (n = 1). One woman from the control group was also unable to complete the protocol due to perinatal medical complications. Other reasons for dropping out from the control group were moving from the area (n = 2) and lack of interest (n = 1).

A total of 87 technically satisfactory tests were conducted regardless of group assignment or trimester. These data were used for frequency analyses of changes in FHR. Satisfactory FHR recordings were available at both Tm2 and Tm3 for 22 exercised and 16 control subjects. These data were used for statistical comparisons between groups and across trimesters.

At entry, the exercised and control groups exhibited similar mean values for physical characteristics including age (30.2 ± 0.9 vs. 29.1 ± 0.9 yr), parity (0.7 ± 0.9 vs. 0.5 ± 0.5), body height (163.9 ± 0.9 vs. 165.2 ± 0.8 cm), sum of four skinfold thicknesses (65 ± 4 vs. 64 ± 4 mm), and PWC150 (70 ± 4 vs. 67 ± 4 W). However, the control group was significantly heavier than the exercised group (68.7 ± 2.5 vs. 63.3 ± 1.2 kg). Because the sum of four skinfolds was similar between groups, this was probably due to greater fat-free body mass in the subjects of the control group. Mean gestational age on entry to the study was 16 ± 2 wk in the exercised group and 17 ± 2 wk in the control group.

Maternal physical conditioning responses. Records of actual exercise performed by the exercised group in supervised classes are summarized in Table 1. These confirmed good compliance with the exercise regimen and

<table>
<thead>
<tr>
<th>Variable</th>
<th>Tm2</th>
<th>Tm3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise heart rate, beats/min</td>
<td>145±1</td>
<td>147±1</td>
</tr>
<tr>
<td>Exercise duration, min/session</td>
<td>18.2±0.3</td>
<td>24.5±1.1*</td>
</tr>
<tr>
<td>Attendance, %</td>
<td>82±2</td>
<td>76±3</td>
</tr>
</tbody>
</table>

Values are means ± SE; n = 22 subjects. EG, exercised group. * Significantly greater 3rd trimester (Tm3) compared with 2nd trimester (Tm2), P < 0.05.
achieve an HR target of ~145 beats/min was significantly higher in the exercised group in both tests and confirmed a higher aerobic conditioning status compared with the control group.

**FHR baseline characteristics.** An overview of FHR responses to maternal exercise was provided by comparing the 20-min pre- and postexercise windows between groups and between trimesters (Table 3). Within both groups and in both trimesters, values for mean FHR baseline were 3–7 beats/min higher postexercise compared with those preexercise. These changes reached statistical significance during both Tm2 and Tm3 in the exercised group and during Tm3 in the control group.

Mean values for both pre- and postexercise baseline were 5–7 beats/min lower in Tm3 compared with corresponding values in Tm2 in both groups. Changes were significant for both pre and postexercise means in the exercised group and for preexercise in the control group. FHR variability did not change significantly within exercise tests (post- vs. preexercise) or between trimesters. There were no between-group differences for absolute values of FHR baseline or baseline variability. However, analysis of change in pre-to postexercise data revealed a significant difference between groups for FHR variability in Tm3. This was the result of the combined effect of a small decrease in the control group and a small increase in the exercised group.

The frequency of transient FHR accelerations was higher in Tm3 compared with that in Tm2 (overall effect of $P < 0.05$ within both groups). Changes between Tm2 and Tm3 reached statistical significance in the control group for preexercise and in the exercised group for postexercise (Table 3). There were no between-group differences in absolute values. However, change scores in pre-to postexercise were significantly different between groups in Tm3. This resulted from the combined statistical effects of a small decrease in the control group and small increases in the exercised group.

The frequency of transient FHR decelerations was

| TABLE 3. FHR 20 min before and 20 min after maternal aerobic exercise |
|-----------------------------|-----------------------------|-----------------------------|
|                             | Tm2                        | Tm3                        |
|                             | Preexercise | Postexercise | Change in pre to postexercise | Preexercise | Postexercise | Change in pre to postexercise |
| Group                      |            |              |                          |            |              |                          |
| CG                         | 145±2      | 148±3        | 3±2                      | 139±2†     | 143±2*       | 4±1                      |
| EG                         | 144±1      | 150±2*       | 6±2                      | 137±2†     | 144±2†       | 7±1                      |
| **FHR baseline, beats/min**|             |              |                          |            |              |                          |
| CG                         | 6.3±0.5    | 6.2±0.4      | -0.1±0.4                 | 6.9±0.5    | 6.3±0.4      | -0.6±0.4§               |
| EG                         | 6.1±0.3    | 5.9±0.3      | -0.2±0.4                 | 5.9±0.2    | 6.4±0.3      | 0.5±0.3                 |
| **Accelerations, no./20 min**|             |              |                          |            |              |                          |
| CG                         | 1.5±0.5    | 1.4±0.5      | -0.1±0.7                 | 4.7±0.6†   | 3.1±0.7      | -1.6±0.8§               |
| EG                         | 2.0±0.5    | 2.3±0.5      | 0.3±0.7                  | 3.6±0.6    | 4.2±0.6†     | 0.6±0.7                 |
| **Decelerations, no./20 min**|             |              |                          |            |              |                          |
| CG                         | 1.6±0.4    | 1.5±0.3      | 1.0±0.5                  | 1.7±0.5†   | 0.8±0.3      | -0.9±0.6§               |
| EG                         | 2.2±0.4    | 2.5±0.5      | 0.2±0.4                  | 0.6±0.2†   | 0.1±0.4†     | 0.5±0.4                 |

Values are means ± SE; $n =$ 16 in CG and 22 in EG. FHK, fetal heart rate. Significantly different at $P < 0.05$: * postexercise mean compared with preexercise mean within group; † mean at Tm3 compared with corresponding mean at Tm2 within group; ‡ greater CG mean compared with EG mean within trimester; and § mean change in pre- to postexercise in CG compared with that in EG during Tm3.
TABLE 4. Exercise-induced deviations from normal FHR baseline at Tm2 and Tm3

<table>
<thead>
<tr>
<th>Group</th>
<th>Mild Tachycardia (160-180 beats/min)</th>
<th>Moderate Tachycardia (180-200 beats/min)</th>
<th>Mild Bradycardia (100-120 beats/min)</th>
<th>Moderate Bradycardia (80-100 beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tm2</td>
<td>Tm3</td>
<td>Tm2</td>
<td>Tm3</td>
</tr>
<tr>
<td>CG</td>
<td>1/18 (6%)</td>
<td>0/20 (0%)</td>
<td>0/18 (0%)</td>
<td>0/20 (0%)</td>
</tr>
<tr>
<td>EG</td>
<td>2/22 (9%)</td>
<td>1/27 (4%)</td>
<td>0/22 (0%)</td>
<td>1/27 (4%)</td>
</tr>
<tr>
<td>CG</td>
<td>3/18 (16%)</td>
<td>0/20 (0%)</td>
<td>1/18 (6%)</td>
<td>0/20 (0%)</td>
</tr>
<tr>
<td>EG</td>
<td>7/22 (32%)</td>
<td>1/27 (4%)</td>
<td>0/19 (0%)</td>
<td>1/27 (4%)</td>
</tr>
</tbody>
</table>

Values are frequencies; n = 16 in CG and 22 in EG. No deviations were observed before exercise (20 min).

lower at Tm3 compared with that at Tm2 within the exercised group for both pre- and postexercise values (Table 4). Changes in scores pre- to postexercise for FHR decelerations were significantly greater in the control group compared with the exercised group in Tm3, primarily due to significantly higher preexercise values in the control group.

Data from minute-by-minute analysis of the effects of maternal exercise on FHR baseline are presented in Figs. 1 and 2. Values increased significantly from preexercise to the end of the 15-min exercise bout in the exercised and control groups at both Tm2 and Tm3. Absolute values recorded immediately before exercise, during exercise, and for 20 min after exercise were similar to one another in the exercised and control groups during both Tm2 (Fig. 1A) and Tm3 (Fig. 1B). FHR values were higher during Tm2 vs. during Tm3 in both the control (Fig. 2A) and exercised (Fig. 2B) groups. Differences were statistically significant immediately before exercise in both groups and at 5, 10, and 15 min of exercise in the control group. In both groups, values in late recovery were also significantly higher at Tm2 vs. Tm3, suggesting a slower recovery to preexercise baseline in mid- vs. late gestation.

**FHR baseline deviations.** The incidence of tachycardia (FHR > 160 beats/min) and bradycardia (FHR < 120 beats/min) during and after exercise in 87 technically satisfactory tests was low during both Tm2 and Tm3 (Table 4). The most common response was tachycardia rather than bradycardia. During all tests, normal reactive FHR patterns were observed before exercise and normal baseline variability persisted during and after exercise. Note that 21 of the 87 tests provided continuous or partly continuous recordings during exercise.

Three instances of exercise-induced fetal bradycardia were documented. The fetus of one exercised subject exhibited moderate bradycardia after 10 min of exercise during a Tm3 test. At this point exercise was discontinued. Mild to moderate tachycardia was then observed for the first 10 min of recovery. This individual delivered a healthy infant weighing 2,806 g at 38 wk with no labor complications. All other instances of significant FHR deceleration were observed during recovery from exercise.

Two instances of mild postexercise bradycardia were observed. In both cases, bradycardia occurred suddenly on exercise cessation and recovered after 5 and 13 min, respectively. The first of these women was a Tm3 exercised subject who delivered a healthy infant weighing 3,380 g at 43 wk gestation. The second woman was a Tm2 control subject who subsequently gave birth to a healthy 3,600-g child at 40 wk gestation. However, labor compli-
FIG. 2. Effects of advancing gestational age on fetal heart rate responses during and after maternal exercise in control (A) and exercised (B) groups. Values are means ± SE; n, no. of subjects in each group.
* Significantly higher mean value for end of Tm2 compared with that for end of Tm3, P < 0.05.

Pregnancy outcome. Data on pregnancy outcome from the present study are not included here. Overall study results have been published in a preliminary report (4). Briefly, within the present study sample, both groups gained ~10 kg between entry and the end of Tm3 and lost a similar amount of body mass between the end of Tm3 and the postpartum assessment. No significant differences existed between groups for gestational age at delivery (39.8 ± 0.3 vs. 39.6 ± 0.3 wk), infant birth weight (3,521 ± 81 vs. 3,630 ± 99 g), Apgar scores, labor duration (7.0 ± 1.0 vs. 7.4 ± 1.4 h), or frequency of perinatal complications. No subjects in either group delivered infants with low birth weights (<2,500 g). As described above, none of the observations of bradycardia or tachycardia was associated with an abnormal pregnancy outcome.

DISCUSSION

The present study was conducted to characterize the effects of maternal aerobic conditioning on FHR responses to an acute bout of submaximal exercise performed by healthy pregnant women. Particular attention was paid to a controlled longitudinal design, proper characterization of the subject sample, accurate quantification of the acute exercise stimulus during testing, and close monitoring of the physical conditioning regimen. A nonrandomized study design was chosen to promote subject compliance with the assigned treatments (i.e., exercise and sedentary control). However, subjects in both groups were recruited from a homogeneous population of sedentary women and were not admitted to the study if they displayed specific exclusion criteria. Statistical analysis of physical characteristics on entry to the study confirmed that the two groups were similar from a physiological viewpoint.

The current state of knowledge on FHR responses to maternal exercise was described in detail in a recent review from this laboratory (19). The results of the present study confirmed that the most common FHR response to sustained submaximal exercise is a moderate increase in FHR baseline that gradually returns toward preexercise baseline during postexercise recovery (2, 6, 7, 16). The cause of exercise-induced FHR elevation has not been identified (see Ref. 19 for detailed discussion of proposed mechanisms). A higher FHR baseline, both at rest and in association with maternal exercise, has also been reported in late vs. midgestation (2, 6, 7, 16).

It has been hypothesized that this is a reflection of greater vagal/parasympathetic tone in late vs. midgestation. In this regard, studies of laboratory animals suggest that sympathetic control of fetal cardiac function is established before midgestation, whereas parasympathetic control is not well developed until the last one-third of gestation (13, 17).

Also of specific interest in this study was the incidence of significant but transitory deviations from the normal FHR range that occurred during or after maternal exercise. In theory, maternal exercise may result in shunting of cardiac output toward contracting maternal muscle and away from the uterus, resulting in reduced oxygen delivery to the fetus. The initial fetal response to acute hypoxia or asphyxia is bradycardia (see Ref. 19 for a detailed description of the mechanism of fetal bradycardia). Fetal bradycardia during or after maternal exercise has been reported in several studies (2, 3, 9, 18, 19). According to a recent review (11), the published incidence of fetal bradycardia in association with maternal exercise tests is ~15–16%. However, when FHR monitors that employ Doppler ultrasound are utilized, apparent episodes of fetal bradycardia during exercise may, in fact, be motion artifact related to maternal stepping or pedaling rhythm during treadmill exercise or cycle ergometry, respectively (12). Thus, the true incidence of exercise-induced fetal bradycardia has probably been overesti-
nated. In the present study, particular care was taken to avoid or recognize motion artifact by briefly discontinuing pedaling at 5 and 10 min of exercise. With this procedure and with detailed analysis of the 21 technically acceptable FHR records, it appears that fetal bradycardia does not usually occur during submaximal maternal exercise. The single occurrence of fetal bradycardia observed in the study persisted on exercise cessation and was compensated for by moderate tachycardia during the postexercise period.

From a hemodynamic viewpoint, the immediate postexercise period differs significantly from that of exercise. It is well established from previous studies of nonpregnant subjects that HR declines markedly on exercise cessation but peripheral vascular conductance remains high due to continued vasodilation of skeletal muscle vascular beds. Thus, hypotension, reduced venous return, diminished arterial blood flow, and reduced uterine blood flow may ensue in some pregnant subjects during the first few minutes of recovery, and this may lead in turn to transient FHR deceleration. In this study, two fetuses exhibited mild transient bradycardia that began suddenly on exercise cessation and another showed a 1-min FHR deceleration below 120 beats/min in the immediate postexercise period. Because all three rapidly returned to a normal baseline as recovery progressed, the most likely explanation for these findings is fetal hypoxia caused by maternal hypotension and reduced uterine blood flow. Additional study involving measurement of maternal blood pressure and uterine blood flow in addition to FHR is needed to confirm this hypothesis.

In summary, the incidence of fetal bradycardia in the present study was only 1.1% during and 2.3% after maternal exercise. This low incidence of fetal deceleratory responses to exercise despite a probable reduction in uterine blood flow can be attributed to the operation of several maternal-fetal adaptive mechanisms. These may include hemoconcentration of maternal blood during exercise, redistribution of uterine blood flow to favor the placental vs. myometrial vasculature, and augmented uterine arteriovenous oxygen extraction (19). The use of a submaximal vs. maximal testing protocol may also have contributed to our findings (5).

The incidence of mild tachycardia (FHR of 160-180 beats/min) was higher during and after Tm2 exercise tests than during Tm3 tests. This was expected, since average FHR baseline declines as gestation progresses as a result of maturation of the fetal parasympathetic nervous system (13, 17). Thus, these findings likely result from upward drift during exercise from a higher preexercise baseline during Tm2 tests. The incidence of moderate tachycardia (FHR, 180-200 beats/min) was low in both trimesters and in both groups. Tachycardia under the present conditions is probably the result of augmented fetal sympathoadrenal catecholamine output and may be a compensatory mechanism during recovery from fetal hypoxia (13). Note that moderate fetal tachycardia was observed more frequently (10 vs. 3 occurrences) after exercise compared with during exercise (Table 4). In the present study, evidence also exists for a higher frequency of FHR accelerations (an indicator of fetal well-being) both before and after exercise in Tm3. An increase in FHR accelerations in the resting state with advancing gestational age has also been reported (15) and probably reflects a greater level of fetal activity in late pregnancy (19).

To our knowledge, the present study is the first to examine maternal physical conditioning effects on FHR responses to acute maternal exercise using a controlled prospective design. Results suggest that FHR responses to HR-targeted submaximal maternal exercise were similar in conditioned and unconditioned subjects despite the fact that the former were exercising at a significantly higher work rate. Theoretical advantages of maternal physical conditioning included less redistribution of maternal cardiac output toward skeletal muscle and away from splanchnic vascular beds, more efficient maternal heat dissipation, and greater utilization of fat vs. carbohydrate at any given submaximal exercise power output. Thus, a higher absolute maternal work rate may be required in conditioned women to induce the same level of fetal hypoxic stress.

Historically, little scientific attention has been given to the possibility that fetal and/or placental adaptations to chronic maternal exercise may result in fetoprotective adaptations. However, the recent report of Clapp et al. (8) indicated that increases in placental volume during the 1st 24 wk of pregnancy were augmented significantly in physically active women who continued to exercise regularly during pregnancy. Improvement in placental vascularity (and diffusing capacity) in response to aerobic conditioning may help to account for our observation that increases in FHR in response to acute maternal exercise were similar between groups despite significantly higher maternal exercise work rates in the exercised group. Such changes may also help maintain normal FHR variability in response to maternal exercise.

In the present study, a significant between-group effect was also observed in Tm3 for pre- to postexercise changes in FHR accelerations that resulted from the combined effects of small increases after exercise in the experimental group and small decreases after exercise in the control group. A significant between-group difference was also observed for pre- to postexercise changes in FHR variability during Tm3 exercise tests. Again, this resulted from nonsignificant trends to decrease variability in the control group and to increase variability in the exercised group. Finally, significant reductions in FHR decelerations were observed within the exercised group both before and after exercise in Tm3 vs. Tm2. All of these findings suggest a beneficial effect of maternal physical conditioning but should also be viewed with caution, since changes were less than the error of the measurement method and were also too small in magnitude to have any important clinical significance. However, considered together, they support the need for future studies to examine fetal and placental adaptability to chronic maternal exercise and to test the hypothesis that fetal tolerance for maternal exercise is improved after physical conditioning.

In summary, the results of the present study confirmed that the normal FHR response to submaximal maternal exercise is a moderate increase in FHR baseline with gradual recovery toward preexercise baseline within
~20 min of exercise cessation. Transient fetal bradycardia was observed occasionally in association with maternal exercise, and this seems to occur most often in the immediate postexercise period. This may be due to maternal venous pooling, maternal hypotension, and a temporary reduction in uterine blood flow. Moderate fetal tachycardia may also occur during or after maternal exercise, presumably in response to augmented fetal sympathetic-adrenal catecholamine output during recovery from transient hypoxia. Results were also consistent with the hypothesis that physically conditioned women can perform at higher exercise power outputs than sedentary women without inducing fetal hypoxic stress. Additional study is indicated to further examine fetal and/or placental adaptations to maternal physical conditioning.

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