EXERCISE IN HUMAN PREGNANCY

CYCLING AND SWIMMING IN THE LAST TRIMESTER

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EXERCISE IN HUMAN PREGNANCY

CYCLING AND SWIMMING IN THE LAST TRIMESTER

INSPANNING IN DE ZWANGERSCHAP

FIETSEN EN ZWEMMEN IN HET LAATSTE TRIMESTER

PROEFSCHRIFT
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OP GEZAG VAN DE RECTOR MAGNIFICUS
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Science sans conscience égale science de l'inconscience
Mc Solaar; prose combat

Aan Ard en mijn ouders

CONTENTS

CHAPTER 1

GENERAL INTRODUCTION

Physical exercise and sports are generally accepted as part of a healthy lifestyle. In pregnancy, however, women are often advised to refrain from physically demanding exercise because it is thought to be potentially harmful to the pregnant woman and her fetus. The recent edition of Williams Obstetrics[96] states that; ".... in women who were previously sedentary, aerobic activity more strenuous than walking is not recommended." Such an advise may create a dilemma for many pregnant women.

In earlier decades, studies on fetal responses to maternal exercise were limited to laboratory animals in which invasive techniques could be used. Studies in exercising pregnant sheep showed that compensatory physiologic mechanisms protect the developing fetus, such as maternal hemoconcentration, redistribution of blood flow within the uterus from the muscle to the placenta, and increased oxygen extraction.[60] The results obtained in animal experiments suggest that maternal exercise has no harmful effect on fetal health and provide justification for further study of physiologic responses to exercise in human pregnancy. In the past decade the subject gradually moved from the laboratory animal to the human being.

Despite the reassuring results of animal experiments unanswered questions remain and new issues arise. Many exercising women report that they feel their baby moving less and that they are aware of increased uterine contractions. This could indicate a true change in fetal condition and uterine contractility, or could be based on subjective impression only. The fetal heart rate response to maternal exercise has been studied since in 1961 Hon and Wohlgemuth[46] proposed that it could serve as a clinical test of fetal health and uteroplacental sufficiency. Most authors have reported on fetal

heart rate and uterine contractility before and after, but not during physical exercise.[67,89] Extrapolation of these data to the exercise period itself may not be justified because in instrumented sheep most fetal variables appear to return to baseline levels within a few minutes.[59] Studies in pregnant women during the exercise period by means of external transducers are sensitive to motion artifacts.[79] The study of fetal heart rate and uterine contractility during maternal exercise requires a direct method of measurement. With the current techniques this can only be realized in term pregnant women with ruptured membranes. To the best of our knowledge there is only one report on this approach used in two term women, in whom no adverse fetal responses to brief exercise were observed.[5] Further studies in a larger group of women over longer periods of exercise, are needed to support or refute these preliminary observations.

Maximal exercise testing has been used to study physical performance in pregnancy.[61] Physical performance can be assessed objectively by determination of maximal oxygen uptake ($\dot{V}O_{2max}$) during an increasing exercise test. The upper limit for O_2 utilization is determined by the pumping limit of the heart, by the potential for O_2 extraction by the exercising tissue, and by the limit of ventilation.[94] The level of exercise $\dot{V}O_2$ above which aerobic energy production is supplemented by anaerobic mechanisms is defined as the anaerobic threshold.[94] Energy production above the anaerobic threshold is reflected in an exponential increase in blood lactate concentration. Traditional methods for detection of the anaerobic threshold rely on visual inspection of graphical plots of ventilatory equivalents and end-tidal gas concentrations.[12] Because of the large inter- and intra-observer variation in the visual assessment of the ventilatory threshold a more reproducible method is preferred, e.g. a mathematical method like the V-slope method[12] or serial blood lactate measurements. However, the V-slope method used in nonpregnant individuals, cannot be reliably applied to exercise data obtained in pregnant women. Although maximal aerobic power appears to remain largely unaltered during pregnancy,[61] differences with the nonpregnant state at a submaximal level are likely. To answer the question if the anaerobic threshold changes in pregnancy, an adaptation of the existing mathematical method is required.

Most studies on endurance exercise in pregnant women have been performed at low exercise intensities,[15,20,21,75] but competitive endurance exercise is usually performed at an intensity of 70-80 % of maximal oxygen uptake ($\dot{V}O_{2max}$), or at a heart rate

(HR) between 160-180 beats per minute.[7] Little is known about the physiologic effects of strenuous endurance exercise in pregnancy. A meta-analysis of 18 studies showed that pregnant women can exercise without apparent adverse effects three times a week for approximately 40 minutes at a heart rate of up to about 145 beats per minute.[57] There is evidence that women perform rapidly progressive maximal weightbearing and nonweightbearing exercise equally well in pregnancy as in the nonpregnant state. Peak oxygen uptake ($\dot{V}O_{2peak}$) during cycling [61,80] and running[61] was reported to be unaffected by gestation. This led us to postulate that women are quite capable of performing strenuous endurance exercise in pregnancy.

Many people are discovering the benefits of working out in water, which combines exercise with comfort. Because water supports weight, pregnant women will feel more comfortable and less clumsy. Also the risk of injuries due to connective tissue and joint laxity in pregnancy is reduced in water exercise. For these reasons, swimming is generally recommended to maintain a good physical condition during gestation.[54] However, literature on the physiologic adaptations of pregnant women to exercise in water is scarce. Only one study was published in which $\dot{V}O_{2peak}$ during swimming was reported as 17% lower during gestation than post partum.[74] Whether this effect can be attributed to a different physiologic response to a water environment or if the pregnant participants did not push themselves as hard as after delivery is not clear.

Also diving using selfcontained underwater breathing apparatus (SCUBA) is increasingly popular among women and more women may be expected to seek obstetric advise on the safety of diving while pregnant. The underwater environment is unique because of the need of a life-support system and the concomitant physiologic changes in breathing. Questions arise concerning the effects on the pregnant women and the fetus of altered hydrostatic pressure and gas transfer. Scientific data to answer these questions can be derived from several experimental studies in animals and from a limited number of observations in pregnant women.

Based on the considerations presented above the objectives of this thesis are:

- To assess the physiologic responses of fetal heart rate and uterine contractility to moderately strenuous exercise in term pregnant women.

- To determine if the anaerobic threshold is affected by pregnancy.
- To test the hypothesis that pregnant and nonpregnant women are equally capable of performing endurance exercise.
- To explore what is known about immersion, swimming, and diving in pregnancy.
- To test the hypothesis that swim $\dot{V}O_{2peak}$ is not reduced by pregnancy and is lower than cycle $\dot{V}O_{2peak}$.

The studies related to these objectives are described in Chapters 2 to 5 of this thesis and followed by a general discussion (Chapter 6).

CHAPTER 2

FETAL HEART RATE AND UTERINE CONTRACTILITY DURING MATERNAL EXERCISE AT TERM[1]

2.1 *Introduction*

The fetal heart rate (FHR) response to maternal exercise has been studied repeatedly since in 1961 Hon and Wohlgemuth[46] proposed that it could serve as a clinical test for uteroplacental insufficiency. Nonetheless, the question as to what is the normal FHR response to maternal exercise has remained unanswered. This may be attributed to several factors. First, it is difficult to measure fetal heart rate accurately during maternal exercise and, second, the physiologic burden imposed by the exercise and the physiologic alterations in fetal behavioral states must be taken into account.

Because of motion artifacts induced by maternal exercise, most authors have reported on fetal heart rates measured not during but before and after exercise.[37,46,67,89] Conclusions from this data cannot be extrapolated to the exercise period itself, because in sheep most fetal variables are known to return to baseline levels within a few minutes of recovery from even exhausting exercise.[59] Fetal heart rate during maternal exercise has usually been studied by means of an external transducer,[3,4,28,50] a method that is sensitive to motion artifacts. Indeed, in one study artifact was demonstrated; FHR was

[1]The main substance of this chapter was published in: WEM Spinnewijn, FK Lotgering, PC Struijk and HCS Wallenburg. Fetal heart rate and uterine contractility during maternal exercise at term. Am. J. Obstet. Gynecol. 1996;174:43-48.

identical to the pedaling frequency on the cycle ergometer or the stepping rate at the treadmill.[79] Such artifacts might explain the periods of severe fetal bradycardia observed during maternal exercise in several studies.[3,28,50] However, in two studies the occurrence of bradycardia was also reported. In one study bradycardia was observed in 16 of 32 fetuses during or after maternal exercise when B-mode echocardiography was used to study the FHR,[17] using similar technique another study reported bradycardia following exercise in 2 of 12 women.[67] Although echocardiography does not carry the same risk of artifact as the use of external transducers, the data show that many of the periods of fetal bradycardia were of short duration and may be described as spikes or variable decelerations rather than as bradycardia. Recently, the use of direct fetal monitoring was reported; fetal bradycardia was not observed during 4.5 minutes of maternal exercise at approximately 60% of maximal aerobic capacity.[5] In instrumented sheep, fetal bradycardia has been occasionally reported in relation to maternal exercise and severe hypoxemia,[32] but in other studies FHR was reported unchanged, or slightly increased, without evidence of bradycardia or decelerations,[18,59] and severe hypoxemia was absent even during exhaustive maternal exercise.[59]

Some women report increased uterine contractility during physical exercise. Uterine contractility has been studied with the use of external tocodynamometry before and after maternal exercise.[89] However, tocodynamometry is rather inaccurate and recovery values provide little information about the effects of exercise itself.

In an effort to determine the response of the FHR and uterine contractility to maternal exercise, we studied a group of healthy women admitted for elective induction of labor in whom the fetal heart rate was recorded from a scalp electrode and intrauterine pressure through an intra-amniotic fluid-filled catheter after artificial rupture of the membranes, before, during, and after moderately strenuous exercise on the cycle ergometer.

2.2 *Methods*

Subjects

Between October 1991 and January 1993 we studied 30 healthy pregnant women at a gestational age between 38 and 42 weeks. All women carried uncomplicated singleton pregnancies, with the fetus in the vertex position and the occiput engaged in the pelvis. All women opted for elective induction of labor, which is an accepted procedure in the department,[90] and had a cervix favorable for induction. The women gave informed consent to participate in the study, approved by the Hospital and University Ethics Committee.

Study Protocol

The women were admitted at about 8.00 a.m. to the airconditioned labor suite, kept at 23°C and 60% humidity. Maternal heart rate was continuously recorded and stored as 60-sec average values (Polar Sport tester, Polar Electro, Kempele, Finland). After artificial rupture of the membranes, a fluid-filled pressure catheter was introduced into the amniotic cavity and an electrode was attached to the fetal scalp; both were connected to a FHR monitor (HP 8040 A, Hewlett-Packard, Boeblingen, Germany). After the pressure transducer was calibrated a 20-minute baseline cardiotocogram was recorded at a paper speed of 3 cm per minute, with the woman in the semirecumbent position. Immediately thereafter the woman was seated on the cycle ergometer (Ergoline 900, Mijnhardt, Bunnik, The Netherlands) placed at her bedside and 5 minutes later she started to pedal at a rate of 60-80 revolutions per minute and an initial workload of 50 Watt. The workload was subsequently increased by 10 W every 30 sec until a maternal heart rate of approximately 140 beats per minute was reached, after which the woman continued to exercise for a total of 20 minutes. Immediately following the exercise period the woman returned to the semirecumbent position for recovery. After 20 minutes of recovery, she was returned to the care of the attending obstetrician and labor was induced with the use of incremental intravenous doses of oxytocin.

Measurements and calculations

We divided each 60-minutes cardiotocogram into three 20-minute periods (baseline, exercise, and recovery) and separated the FHR tracings from the uterine pressure recordings to avoid recognition of the exercise period by the observers. After random numbers were assigned to the 20-minute FHR tracings, the fetal cardiograms were assessed by three independent, experienced observers. They classified the cardiograms according to Fischer et al.,[35] as a measure of fetal well-being, and Nijhuis et al.,[77] as a measure of the fetal behavioral state. The tocogram was later reunited with the cardiogram to allow subtyping of decelerations. The Fischer score takes into account the baseline heart rate, variability (bandwidth and zero-crossings), accelerations, and decelerations. In this score, each variable is assigned a well-defined subscore of 0 (poor), 1 (intermediate), or 2 (good) for each of the 5 variables, so that the total score ranges from 0 to 10. Fetal condition is considered critical if the score is below 5, questionable if the score is between 5 and 7, and optimal if the score is 8 or more. The three observers also classified the cardiotocogram into FHR patterns A, B, C, and D according to Nijhuis, which reflect fetal behavioral states 1F to 4F. FHR pattern A has a small band width and isolated accelerations; it is associated with behavioral state 1F, characterized by incidental body movements in the absence of the eye movements. FHR pattern B has a wider band width and frequent accelerations; it is associated with behavioral state 2F, characterized by periodic movements in the presence of eye movements. FHR pattern C also has a wider band width, but no accelerations; it is associated with behavioral state 3F, characterized by absence of body movements in the presence of eye movements. FHR pattern D has large and long-lasting accelerations; it is associated with behavioral state 4F, characterized by continuous body movements and eye movements. We present the median values of Fischer scores and subscores, and Nijhuis patterns, assigned by the three observers. One observer assessed all 20 minute tocograms with the use of an xy-data tablet (Summagraphics, Fairfield, CT) and measured the frequency, basal pressure, peak pressure and time pressure integral of contractions for each period, and calculated peak minus basal pressures.

Statistical analysis

We used the median value and range for each variable under consideration for descriptive statistics. We used SPSS/PC + V 5.02 (SPSS, Chicago, IL) to perform the Friedman and Wilcoxon signed-rank test to analyze differences between paired variables, and the Fisher exact test for differences in behavioral states between periods. A p-value of < 0.05 was taken as the level of significance.

2.3 *Results*

Of the 30 women enrolled in the study, six were nulliparous, 24 parous; maternal age was 31 (23-37) years. Twenty-six women completed the exercise test. Two volunteers withdrew because they found the test too demanding, one woman developed strong uterine contractions and decided to stop after 14 minutes of exercise, and in one participant the scalp electrode became detached. We report on the cardiotocograms of the 26 volunteers who completed the test.

Obstetric complications did not occur within 3 hours after the exercise test and all 30 women were delivered vaginally of healthy infants, 16 boys and 14 girls, with a median 1-minute Apgar-score of 9 (range 4-10), a median 5-minute Apgar-score of 10 (range 7-10) and a median birth weight of 3620 gm (2690-4800 gm). Twenty-seven women had a spontaneous delivery and three women were assisted by vacuum extraction (n=2) or by forceps (n=1) because of failure to progress. Maternal heart rate was 92 (79-116) beats per minute at rest and increased to a peak value of 140 (130-152) beats per minute during exercise.

A typical example of a cardiotocogram before, during, and after maternal exercise is shown in Fig. 2.1. Maternal exercise did not significantly affect the basal FHR, which was 140 (130-160) beats per minute at rest, 145 (130-170) beats per minute during exercise, and 145 (130-170) beats per minute during recovery.

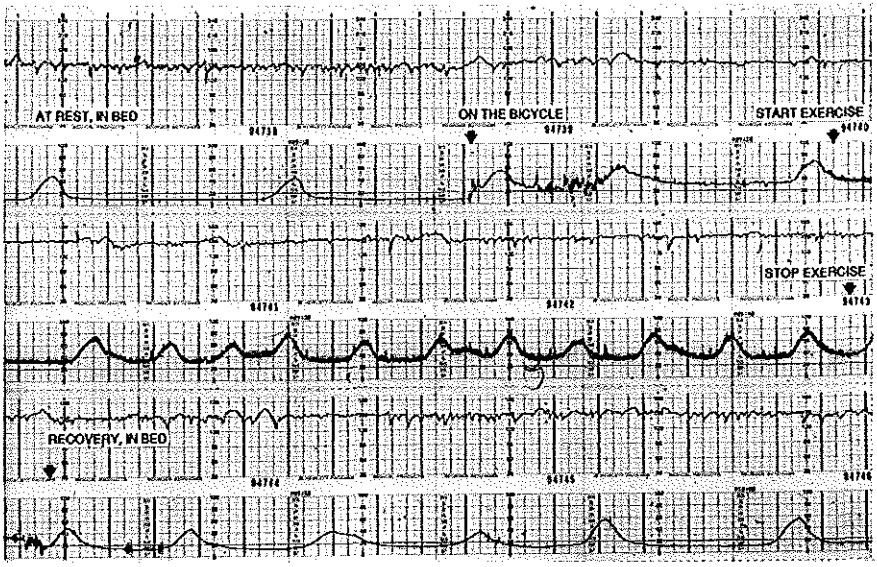

Fig. 2.1. Typical example of cardiotocogram before, during, and after maternal exercise

Table 2.1. Fischer scores of fetal heart rate before, during, and after maternal exercise (n=26).

Fischer score		Number of fetuses		
		Rest	Exercise	Recovery
10		17	13	13
9	"Optimal"	6	6	5
8		0	3	3
7		3	2	2
6	"Questionable"	0	1	3
5		0	0	0
4	"Critical"	0	1	0

As shown in Table 2.1., the Fischer score at rest was optimal ($\geq$ 8) in 23, and questionable (5-7) in 3 fetuses. There were no significant differences in Fischer scores between periods. One fetus had a "critically" low Fischer score of 4 during exercise; at rest and during recovery the Fischer scores were 7 and 6, respectively, and three hours after the exercise a healthy 3740 gm girl was born with Apgar scores of 9 and 10 after 1 and 5 minutes, respectively.

Table 2.2. Fetal heart rate before, during, and after maternal exercise (n=26).

	Number of fetuses								
	Rest			Exercise			Recovery		
Fischer subscore	2	1	0	2	1	0	2	1	0
Basal heart rate	26	0	0	22	4	0	23	3	0
Bandwidth	23	3	0	23	3	0	21	5	0
Zero crossings	26	0	0	25	1	0	24	2	0
Accelerations	24	1	1	20	0	6*	21	1	4
Decelerations	22	3	1	20	6	0	20	6	0

* $p < 0.05$ compared with control values at rest.

As shown in Table 2.2 significantly more fetuses had no heart rate accelerations during exercise than at rest or during recovery; no significant differences between periods were observed for the other criteria on which the Fischer score is based.

When we assessed FHR patterns according to Nijhuis et al.,[77] at the onset of the recording heart rate pattern A was present in 7, B in 13, C in 2, and D in 4 fetuses. Fig. 2.2. shows that the heart rate pattern changed 10 times while the mother was still at rest, 15 times during exercise, and 11 times during recovery. A dominant pattern A, B, C, and D was present at rest in 7, 13, 3, and 3 cases; during exercise in 9, 14, 2, and 1 cases; and during recovery in 9, 14, 2, and 1 cases, respectively. The changes in FHR patterns occurred in apparently random order, without clustering of (state) changes at the transitions from rest to exercise or from exercise to recovery, and without significant differences between periods.

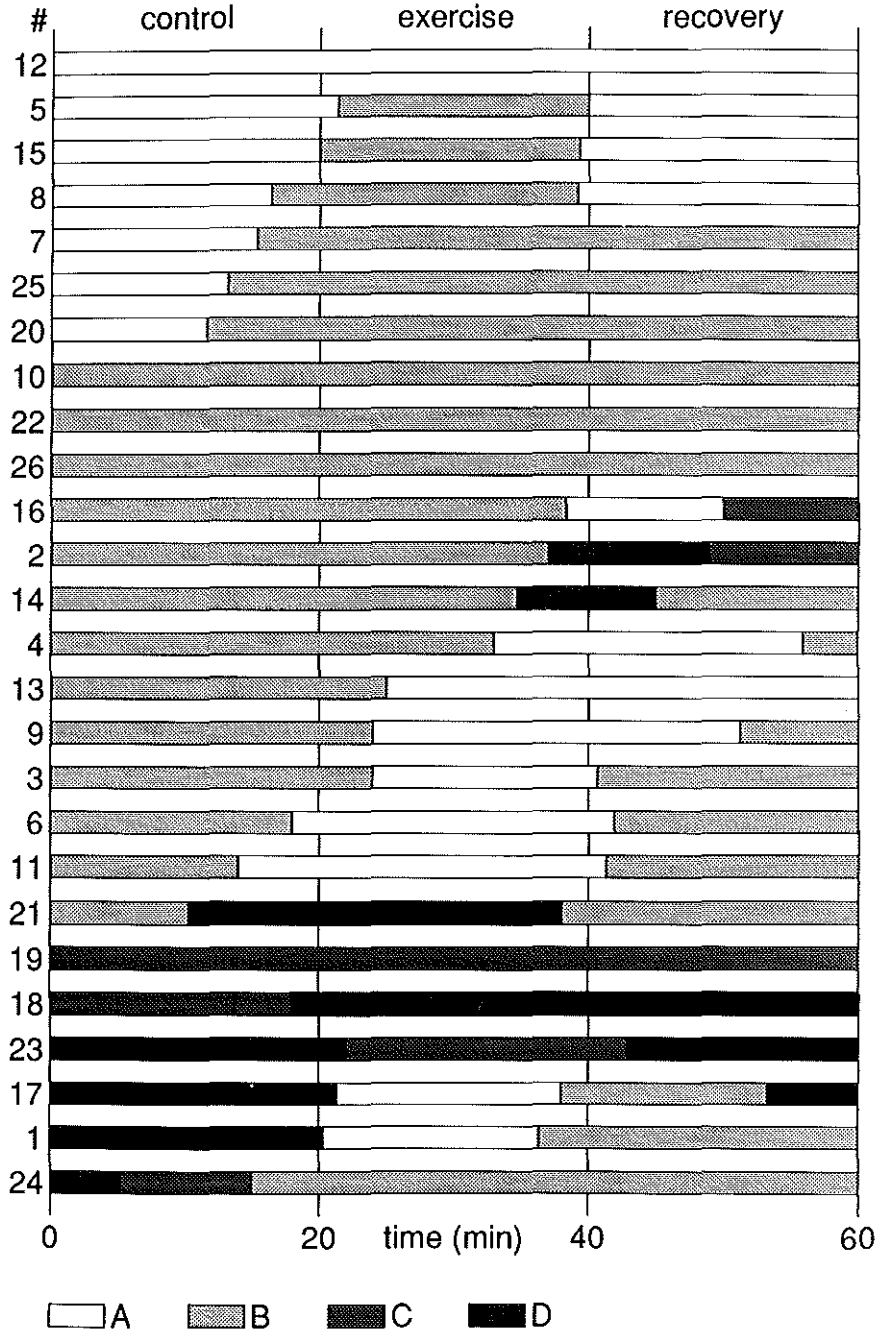

Fig. 2.2. Effect of maternal exercise on FHR patterns *A* to *D*, representing fetal behavioral states 1F to 4F.

Table 2.3. Uterine contractility before, during, and after maternal exercise (n=25).

Contractions	Rest	Exercise	Recovery
Frequency (# / 20 min)	2 (0-7)	11 (6-18)*	3 (0-10)*
Basal pressure (mm Hg)	10 (1-20)	28 (2-39)*	10 (2-24)
Peak pressure (mm Hg)	25 (3-69)	47 (11-70)*	30 (5-61)
(Peak-basal) pressure (mmHg)	15 (0-57)	15 (8-47)	19 (0-52)
Time pressure integral (mmHg / contraction)	7 (0-39)	9 (4-31)	12 (0-31)
Total time pressure integral (mmHg / 20 min)	25 (0-136)	94 (46-343)*	38 (0-188)

Values are medians with ranges in brackets; * $p < 0.05$ compared with control values at rest.

The effect of exercise on uterine contractility is shown in Table 2.3. We report on 25 pressure recordings because in one case the pressure transducer failed. Exercise was associated with a significant increase in the frequency of contractions, from 2 (0 to 7) contractions per 20-minute period at rest to 11 (6 to 18) contractions per 20-minute period during exercise. During recovery the frequency of 3 (0 to 10) contractions per 20-minute period was not different from that at rest. We did not change the position of the pressure transducer when the volunteer moved from bed to ergometer and vice versa. The estimated position of the catheter tip was approximately 25 cm higher with the woman on the ergometer than on the bed, which explains why both basal and peak pressures were increased significantly by 18 mm Hg during exercise compared with the control period at rest. Peak minus basal pressure differences were not significantly different between periods. Because of the increase in contraction frequency during exercise in the absence of a significantly different time pressure integral of a contraction, the total time pressure integral was significantly higher during the exercise period than at rest or during recovery.

2.4 *Discussion*

Our aim was to describe the responses of FHR and uterine contractility to moderately strenuous maternal exercise. Because the response to exercise may vary with the type, intensity and duration of exercise, we controlled for these variables in accordance with the recommendations of the American College of Obstetricians and Gynecologists (ACOG)[1] and imposed cycle exercise at a target heart rate of 140 beats per minute, which corresponds to approximately 60% peak HR, for 20 minutes, including warming-up. Recently, less restrictive ACOG recommendations have been published.[2] The results from our study should not be extrapolated beyond the limits that we set out, i.e. not to women who are less advanced in their pregnancy, or to women who perform exercise of a different type or at a more strenuous level.

In an attempt to differentiate between healthy and compromised fetuses on the basis of the heart rate pattern, Fischer et al. described a score based on five criteria with 0 to 2 points each, which add up to a total score between 0 and 10 points.[35] Visual classification of heart rate patterns appears to be superior to computer analysis of these patterns in identifying the underlying behavioral states, since in near-term fetuses the occurrence of noncoincidence is less than 15%.[91] To limit the effect of the large interobserver and intraobserver variation in the interpretation of scoring cardiotocograms,[58] we used the median value of the subscores assigned by three experienced observers to describe each period in each volunteer. Only in one of the 26 cases we observed a FHR pattern during the exercise that could suggest fetal hypoxemia in a fetus that was born in good health three hours later. We did not observe a single case of fetal bradycardia during exercise or recovery. As mentioned in the introduction, some of the observations of fetal bradycardia during exercise reported in the literature may have resulted from artifact, or from incorrect nomenclature. It seems unlikely that the absence of fetal bradycardia in our study, in contrast to others,[3,17,28,50,67] could have resulted from differences in the duration or intensity of exercise.

Many women report that they feel their baby moving less when they exercise. This could indicate either a real change in fetal movements or a subjective impression. To the best of our knowledge, fetal behavioral states have not been studied in relation to maternal exercise. Heart rate patterns A to D describe the fetal behavioral state rather

than the state of well-being per se. In accordance with Nijhuis et al.,[77] we observed predominantly patterns A (27%), and B (50%) at rest. In spite of the significant increase during exercise in the number of fetuses without heart rate accelerations, the changes in FHR patterns occurred in an apparently random order, as shown in Fig. 2.2. During exercise the dominant heart rate patterns remained A (35%) and B (54%), corresponding with behavioral states 1F and 2F. This suggests that the changes in fetal movements that women may experience during exercise do not reflect a consistent change in fetal behavioral pattern but rather a subjective impression. This impression may be caused by distraction or by increased awareness of fetal quiescence as an otherwise normal state change.

Some women report an increase in the number of uterine contractions during physical exercise. Again, this may indicate either a true increase in uterine contractility or a subjective impression. External tocodynamometry has been used to study uterine contractility during[31] and after[89] exercise. It was reported that during cycling 50% of women experienced uterine contractions,[31] but, because uterine activity at rest was not mentioned, the extent to which the exercise affected myometrial activity cannot be derived from this study. During recovery from exercise uterine activity was reported unchanged as compared to the control period at rest.[89] However, our study shows that with regard to uterine contractility, the recovery period is not representative of the exercise period itself. We found that the peak minus basal pressure difference was unaffected by exercise, so that the intensity of uterine contractions was unaltered by the physical activity. Uterine contractility during exercise at term was increased as a result of a 5.5-fold higher frequency of contractions, associated with a 4-fold higher time-pressure integral. During recovery, the contraction frequency was still 1.5-fold higher than in the control period. The slightly increased number of contractions during the recovery period may have resulted from the preceding exercise but it could also be caused by the rupture of the membranes, 40 to 60 minutes earlier.

The mechanisms that cause the increase in uterine contractility during the exercise period and restrict it largely to that period are unknown. It might be speculated that some hormonal mechanism may be the cause, but mechanical stimulation of the uterus during movements might also explain the sudden onset and end of uterine contractions during exercise. Our data confirm the impression that exercise indeed

increases uterine contractility. However, the results of this study should not be extrapolated to women in whom the uterus is less likely to respond to various stimuli than in our study in term pregnant women admitted for elective induction of labor with a ripe cervix, i.e. to preterm women with an unripe cervix.

CHAPTER 3

ANAEROBIC THRESHOLD AND RESPIRATORY COMPENSATION IN PREGNANT WOMEN[2]

3.1 *Introduction*

O_2 uptake ($\dot{V}O_2$) and CO_2 output ($\dot{V}CO_2$) at rest are slightly increased during pregnancy, whereas during exercise peak $\dot{V}O_2$ and peak power are unaffected, and peak $\dot{V}CO_2$ is lower during gestation.[61] The increased $\dot{V}O_2$ and $\dot{V}CO_2$ at rest during pregnancy reflect the needs of the growing conceptus. The fact that peak $\dot{V}O_2$ is unaffected by pregnancy suggests that muscle mass and physical fitness are not altered by gestation. However, the observation that peak $\dot{V}CO_2$ is reduced during pregnancy cannot be explained easily.

It is known that minute ventilation ($\dot{V}E$) is increased during pregnancy through an increase in tidal volume, an effect that has been attributed to high circulating levels of progesterone[29] and results in a lower arterial PCO_2. Given the hyperbolic relationship between $\dot{V}E$ and arterial PCO_2,[94] higher values of $\dot{V}E$ for a given $\dot{V}CO_2$ are therefore to be expected during pregnancy. However, this does not explain the observation of a lower peak $\dot{V}CO_2$ relative to peak $\dot{V}O_2$ during pregnancy.

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[2]The main substance of this chapter was published in: Frederik K. Lotgering, Piet C. Struijk, Marieke B. van Doorn, Wilma E.M. Spinnewijn and Henk C.S. Wallenburg. Anaerobic threshold and respiratory compensation in pregnant women. J. Appl. Physiol. 1995; 78(5): 1772-1777.

Below the anaerobic threshold (AT), the relationship between $\dot{V}CO_2$ and $\dot{V}O_2$ is virtually linear, largely metabolic in origin, and little affected by $\dot{V}E$.[12] Above AT, $\dot{V}CO_2$ increases more steeply relative to $\dot{V}O_2$, predominantly as a result of bicarbonate buffering of lactic acid, and VE is closely coupled to $\dot{V}CO_2$.[12] The respiratory compensation (RC) point marks the onset of RC for metabolic acidosis, VE rises more rapidly than does $\dot{V}CO_2$, and therefore the behavior of $\dot{V}CO_2$ no longer reflects solely metabolic and buffering events.[12] We propose that a reduction in $\dot{V}CO_2$ relative to $\dot{V}O_2$ during exercise in pregnant compared to nonpregnant women most likely reflects a reduction in the buffering of lactic acid above AT during gestation. A reduction in RC of metabolic acidosis above the RC point would result in extreme respiratory acidosis and is unlikely because it is not in keeping with the known respiratory drive in pregnant women and the higher peak $\dot{V}E$ observed in pregnant than in postpartum women.[61]

To explore why $\dot{V}CO_2$ at peak exercise is lower during pregnancy than postpartum despite little change in peak $\dot{V}O_2$, we examined in greater detail our data from a longitudinal study of $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$ during cycle tests with rapidly progressing exercise intensities throughout pregnancy and after delivery,[61] and determined the AT and RC points as well as the slopes of $\dot{V}CO_2$ vs. $\dot{V}O_2$ both below and above the AT and RC points.

3.2 *Methods*

Subjects

We studied 33 healthy women at 16, 25, and 35 weeks of pregnancy and 7 weeks after delivery. All women were healthy and had uncomplicated singleton pregnancies. The physical fitness of the subjects who entered the study was variable, ranging between women with a sedentary lifestyle and competitive sportswomen. The study was approved by the Hospital and University Ethics Committee, and all women included in the study gave informed consent.

Study protocol

Details of subjects, study protocol, and data on peak values of heart rate (HR), $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ have been previously reported.[61] In short, a physical and obstetric examination was performed to confirm the health of all individuals participating in the study. After 20 minutes of rest, the subject was seated on a cycle ergometer and connected to an electrocardiogram monitor, a gas flow meter and mixing chamber, and an O_2 and CO_2 analyzer (Oxycon-4, Mijnhardt, Bunnik, The Netherlands). After 5 minutes of rest on the cycle ergometer, during which baseline measurements were taken, the women started to exercise. Three minutes of warming up at 30 W were followed by stepwise increments in exercise intensity of 10 W every 30 seconds until peak aerobic power was achieved. This was followed by 5 minutes of cooling down at 10 W. Each woman underwent an initial test to become acquainted with the experimental circumstances; the data obtained in this test were discarded. All women subsequently were studied at 16.1 ± 1.0 weeks (trimester 1), 25.3 ± 0.7 weeks (trimester 2), 35.0 ± 0.6 weeks (trimester 3) of pregnancy and at 6.7 ± 1.4 weeks after delivery. Peak aerobic power was defined by subjective maximal effort in the presence of at least two of the following objective criteria: 1) an increase in $\dot{V}O_2$ of $<5\%$ in response to an increase in exercise intensity; 2) an increase in HR of $<5\%$ in response to an increase in exercise intensity; and 3) a respiratory exchange ratio of >1.0.

Measurements and calculations

We continuously measured HR, $\dot{V}E$, $\dot{V}O_2$, $\dot{V}CO_2$ and exercise intensity. Thirty-second average values of all variables were calculated on-line and were stored on diskettes for later analysis.

If we assume linear interdependence of the variables $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$, and the presence of an AT point and a RC point, according to Beaver et al,[12] the relationship between $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$ can be described as a line with two breakpoints in a three-dimensional space. To determine the lines and the breakpoints in a three-dimensional trilinear model, we used nonlinear regression analysis for each test as described in the APPENDIX. We normalized the data of all individual tests to a scale of 0 (rest) to 1 (peak) for $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$ to avoid a slight distortion that might occur during regression analysis by $\dot{V}E$, which was almost 50-fold higher than

$\dot{V}O_2$ and $\dot{V}CO_2$. We used the values at rest and at peak exercise as well as the data obtained at each step of incremental exercise, after exclusion of the first minute of incremental exercise to avoid distortion caused by the capacity effect of changing tissue CO_2 stores, according to Beaver et al.[12] After the normalized values of the AT and RC points were calculated, the normalized values were converted back to conventional units of VE, $\dot{V}O_2$, and $\dot{V}CO_2$, i.e. liters per minute.

Statistical analysis

For each test period and each variable under consideration, we computed means ± SE. We used SPSS/PC + V 4.01 (SPSS, Chicago, IL) to perform Friedman two way analysis of variance and Wilcoxon signed-rank test to analyze differences between paired variables. A p-value of < 0.05 was taken as the level of significance. Linear regression analysis was used where appropriate.

3.3 Results

The 33 women, 23 nulliparous and 10 parous, completed all tests. They remained healthy throughout the study period and delivered healthy infants. Mean age at the time of delivery was 30.9±0.7 years, gestational age was 40.3±0.2 weeks, and birthweight was 3.43±0.08 kg. 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.

Mean heart rate at rest was significantly increased during pregnancy above postpartum control values (87±2, 89±2, 94±2 and 83±2 beats per minute, at 16-, 25-, 35-weeks gestation and postpartum, respectively), whereas peak heart rate values were slightly, but significantly, reduced during pregnancy (174±2, 174±2, 174±2, and 178±2 beats per minute, at 16-, 25-, 35-weeks gestation and postpartum, respectively). Peak power was not significantly different from postpartum control values during the first and second trimester of pregnancy, but was slightly reduced at 35-weeks gestation (202±7, 196±7, 191±7, and 199±7 W at 16-, 25-, 35- weeks gestation and postpartum, respectively).

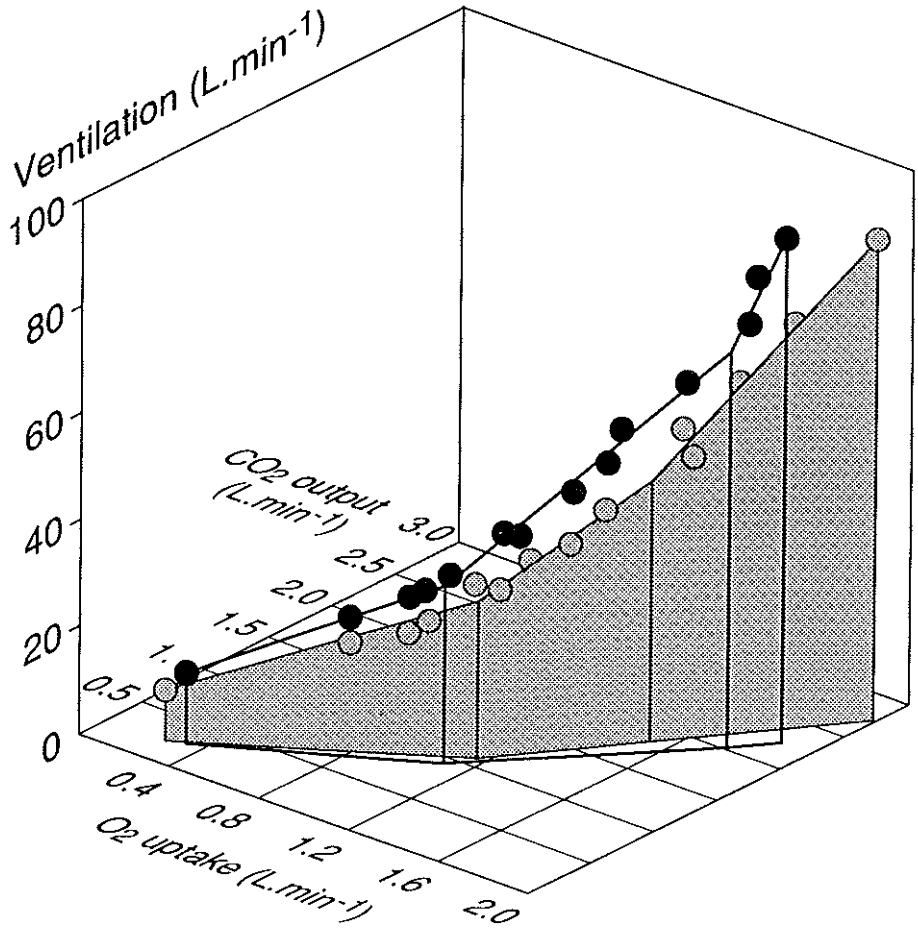

Fig. 3.1. Trilinear relationship among ventilation, O_2 uptake, and CO_2 output in a woman at 16 weeks gestation (shaded circles) and postpartum (filled circles).

When we assumed a trilinear relationship between $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$, regression analysis showed a good fit for all tests: the median value of the average distance from the data points to the regression line of a test was 1.2 (0.6-2.2), 1.0 (0.6-2.2), 1.3 (0.6-2.2) and 1.1 (0.6-2.7) % of normalized values at 16-, 25-, 35-weeks gestation, and postpartum, respectively. Fig. 3.1. shows an example of the trilinear relationship between $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$ in a single volunteer at 16-weeks gestation,

and postpartum. Fig. 3.2. shows the same trilinear relationship in the same volunteer projected onto two-dimensional planes. In addition, it shows that a visually correct fit is maintained for the derived variables: $\dot{V}E/\dot{V}O_2$ vs. $\dot{V}O_2$, $\dot{V}E/\dot{V}CO_2$ vs. $\dot{V}CO_2$, and $\dot{V}CO_2/\dot{V}O_2$ vs. $\dot{V}O_2$.

We found a clearly discernible AT and RC point in 125 of 132 tests (95%), and in all 33 volunteers two breakpoints were found in at least three of the four tests. In seven tests breakpoints were found that might suggest a bilinear rather than a trilinear relationship among variables. In three of these tests the AT point was found between the resting value and the lowest value measured during incremental exercise, in three tests the AT and RC points were found to coincide, and in one test the RC point was found between the peak value and the highest value measured during incremental exercise below the peak. These breakpoints are not necessarily incorrect nor do they markedly distort the overall picture. Therefore, these breakpoints have not been excluded from further analysis.

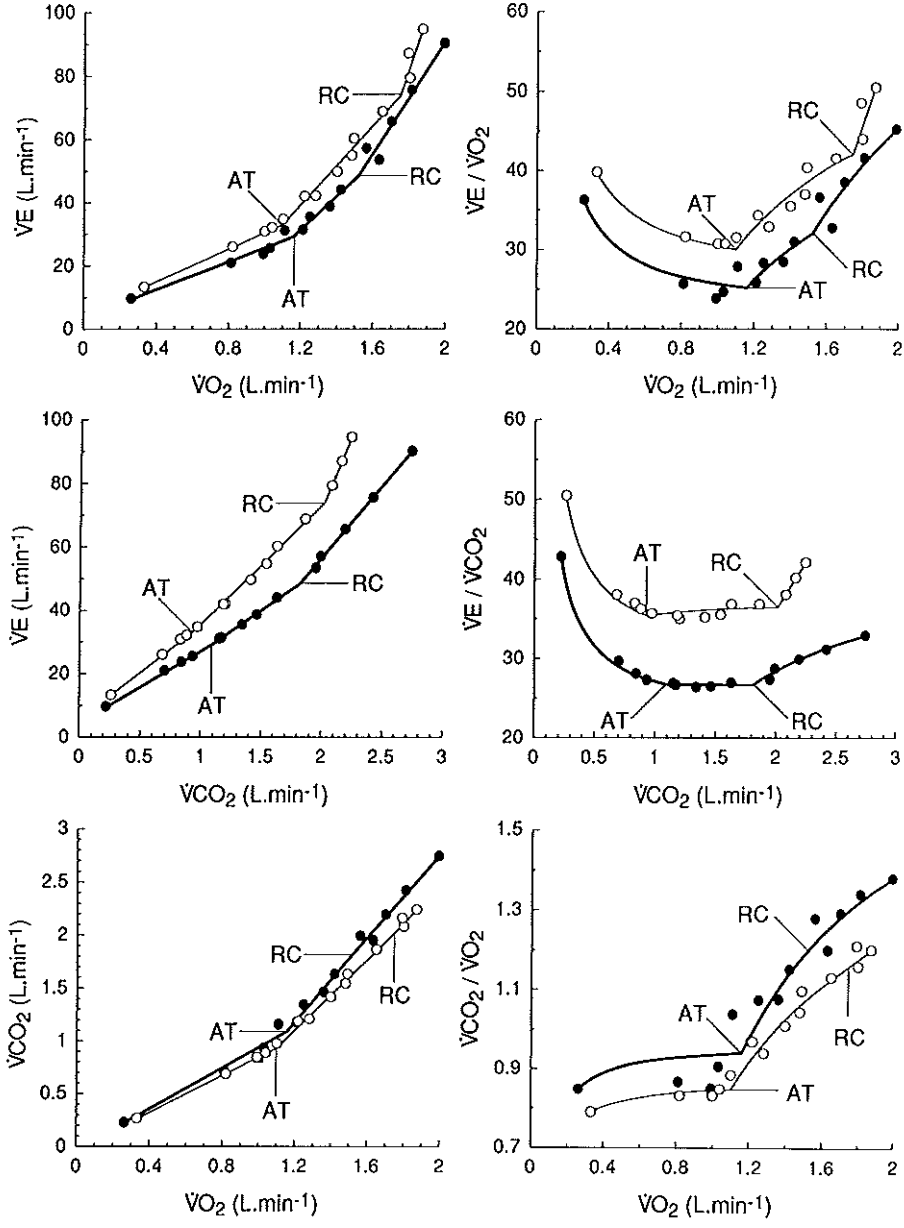

Fig. 3.2. Trilinear relationship among $\dot{V}E$, $\dot{V}O_2$ and $\dot{V}CO_2$ and derived variables in a woman at 16 weeks gestation (○) and postpartum (•). RC, respiratory compensation; AT, anaerobic threshold.

Table 3.1. Effect of pregnancy on values at rest and at peak cycle exercise as well as at anaerobic threshold and onset of respiratory compensation (n=33).

	$\dot{V}O_2$ (l/min)	$\dot{V}CO_2$ (l/min)	$\dot{V}E$ (l/min)	Breathing rate (breaths/min)	Vt (liters)
At rest					
16 weeks	0.29±0.01+	0.23±0.01*	11.4±0.3*	15.7±0.4	0.74±0.02*
25 weeks	0.29±0.01*	0.24±0.01*	11.5±0.3*	15.6±0.4	0.75±0.02*
35 weeks	0.31±0.01*	0.25±0.01*	12.3±0.3*	15.7±0.4	0.80±0.02*
postpartum	0.27±0.01	0.21±0.01	9.5±0.2	16.1±0.4	0.60±0.02
Anaerobic threshold					
16 weeks	1.24±0.05	1.05±0.04	35.8±1.1*	21.1±0.6	1.72±0.06*
25 weeks	1.19±0.03	1.01±0.04	35.1±0.8*	21.8±0.5	1.64±0.05*
35 weeks	1.21±0.04	1.02±0.04	37.1±1.0*	22.3±0.6	1.71±0.07*
postpartum	1.17±0.03	0.98±0.03	29.8±0.8	21.9±0.6	1.39±0.05
Respiratory compensation					
16 weeks	1.84±0.07	1.88±0.08	62.6±2.7	30.0±1.0	2.10±0.06*
25 weeks	1.83±0.07	1.88±0.08	63.8±2.7*	30.3±1.0	2.11±0.07*
35 weeks	1.79±0.07	1.83±0.07	63.8±2.4*	30.0±1.0	2.16±0.08*
postpartum	1.81±0.06	1.92±0.07	54.8±2.2	28.8±1.0	1.93±0.07
At peak					
16 weeks	2.20±0.08	2.56±0.09*	95.6±3.2*	45.2±1.3	2.13±0.07*
25 weeks	2.16±0.08	2.51±0.08*	94.6±2.9*	44.1±1.3	2.14±0.07*
35 weeks	2.15±0.08	2.46±0.09*	96.1±3.5*	42.7±1.1	2.20±0.08*
postpartum	2.19±0.08	2.70±0.09	89.5±3.0	43.9±1.6	2.02±0.06

Values are means ± SE; * $p < 0.01$; + $p < 0.05$. Compared with postpartum control values.

Table 3.1. shows the mean values of $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, breathing rate and Vt at rest and at peak exercise as well as at the two calculated breakpoints (AT and RC) in the relationship between $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$. The AT and RC points were found at exercise intensities of approximately 50 and 80% peak $\dot{V}O_2$, respectively, with no significant differences between the four test periods (53.4±4.3, 52.2±5.6, 52.3±4.8, and 50.5±6.4% peak $\dot{V}O_2$ for AT, and 79.3±8.4, 79.0±9.4, 76.9±8.0, and 78.2±6.9% peak $\dot{V}O_2$ for RC, at 16-, 25-, 35-weeks gestation, and postpartum respectively). O_2 uptake at the AT and RC points correlated with peak $\dot{V}O_2$ in all four test periods ($0.73<r<0.91$) and, therefore, with physical condition. $\dot{V}E$ was significantly higher during gestation at rest and throughout incremental exercise. This was accomplished by a significantly higher Vt, without a significantly different breathing rate during pregnancy compared with postpartum. $\dot{V}O_2$ and $\dot{V}CO_2$ were higher

during pregnancy than postpartum at rest and not significantly different between periods at the AT and RC points. At peak effort $\dot{V}O_2$ was similar and $\dot{V}CO_2$ was lower in pregnant than in postpartum women.

Table 3.2. Effect of pregnancy on the slopes of the trilinear relationship between VE, O_2 uptake, and CO_2 output (n=33).

	Slope below AT	Slope between AT and RC	Slope above RC
$\dot{V}E$ vs $\dot{V}O_2$			
16 weeks	25.1 * (22.4-30.2)	44.3 * (30.1-59.1)	95.3 (54.5-170)
25 weeks	25.9 * (23.9-30.2)	47.1 * (28.9-56.8)	85.4 (56.6-245)
35 weeks	26.9 * (23.9-34.3)	48.6 * (31.2-60.9)	86.4 (48.9-180)
postpartum	22.9 (19.3-26.1)	39.4 (23.9-52.1)	86.1 (46.8-178)
$\dot{V}E$ vs $\dot{V}CO_2$			
16 weeks	29.6 * (27.2-32.6)	31.3 * (24.5-37.4)	49.2 * (33.7-67.4)
25 weeks	30.8 * (27.9-34.2)	33.0 * (25.2-37.3)	48.5 * (34.5-77.4)
35 weeks	31.7 * (29.3-37.0)	34.0 * (25.1-39.4)	49.0 (32.0-64.7)
postpartum	26.3 (23.4-30.8)	26.2 (20.3-32.5)	42.7 (26.4-65.7)
$\dot{V}CO_2$ vs $\dot{V}O_2$			
16 weeks	0.87 (0.74-0.94)	1.37 * (1.21-1.62)	1.94 (1.47-2.88)
25 weeks	0.85 (0.78-0.96)	1.42 * (1.06-1.56)	1.83 (1.44-3.16)
35 weeks	0.84 (0.77-0.95)	1.36 $^+$ (1.18-1.75)	1.76 $^+$ (1.26-2.58)
postpartum	0.87 (0.75-0.94)	1.44 (1.15-1.75)	2.14 (1.61-2.71)

Values are medians with 10th and 90th percentiles in brackets. * $p < 0.01$, $^+$ $p < 0.05$. Compared with postpartum control values. AT = anaerobic threshold, RC = respiratory compensation.

Table 3.2. shows the slopes of the lines in relation to AT and RC points. The data are presented as medians with 10th to 90th percentiles to exclude some extreme values of slopes that resulted from the questionable presence of an AT or RC point in 7 of the 132 tests. Above AT, the slopes of $\dot{V}E$ vs. $\dot{V}O_2$ and $\dot{V}CO_2$ vs. $\dot{V}O_2$ were significantly steeper than below AT for all periods, whereas the slopes of $\dot{V}E$ vs. $\dot{V}CO_2$ were not different from those below AT. Above the RC point, the slopes showed a significant further increase for all three relations and all four periods. Consequently, the $\dot{V}E$ vs.

$\dot{V}O_2$ and $\dot{V}CO_2$ vs. $\dot{V}O_2$ relationships are trilinear, with an AT and RC point, whereas $\dot{V}E$ vs. $\dot{V}CO_2$ is in fact bilinear with no AT point.

Pregnancy significantly increased the slopes of $\dot{V}E$ vs. $\dot{V}CO_2$ throughout incremental exercise, whereas for $\dot{V}E$ vs. $\dot{V}O_2$ it increased the slopes below and above AT, but not above the RC point. Most notably, however, the slopes of $\dot{V}CO_2$ vs. $\dot{V}O_2$, that were similarly steep between periods below AT, were significantly shallower above AT during gestation. Above the RC point the slopes of $\dot{V}CO_2$ vs. $\dot{V}O_2$ were significantly more shallow than postpartum only during the third trimester of pregnancy.

3.4 *Discussion*

The anaerobic threshold is defined as the level of exercise $\dot{V}O_2$ above which aerobic energy production is supplemented by anaerobic mechanisms.[94] Traditional methods for AT detection rely on visual inspection of graphical plots of ventilatory equivalents and end-tidal gas concentrations.[12] The large inter- and intra-observer variation in the visual assessment of the ventilatory threshold led us, like others,[12,78] to search for a mathematical method, applicable to the data from our longitudinal study of $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$ during incremental exercise in pregnancy and after delivery as previously reported.[61] The fact that we had not measured end-tidal PCO_2 or arterial blood gas values in that study, and had stored only 30-seconds average values, limited our options to determine AT.

The physiologic basis for our analysis of the data was provided by the V-slope method of Beaver et al.,[12] who start their analysis by considering $\dot{V}E$ vs. $\dot{V}CO_2$ as a bilinear relationship to detect an RC point. Subsequently, the data below the RC point are selected to derive AT as the breakpoint of the relationship of $\dot{V}CO_2$ vs. $\dot{V}O_2$ which is bilineair below the RC point. When we tried this method, exclusion of the data above the RC point resulted in such a reduction of our 30-seconds average data points that the AT point could not be identified reliably in several cases. By modifying the V-slope method into a single routine, in which we consider the relationship between the three variables $\dot{V}E$, $\dot{V}O_2$, and $\dot{V}CO_2$ as a line in a three-dimensional space with two breakpoints, AT and RC, there was no need to exclude any of our data points and we were able to detect reliably two breakpoints in 95% of the tests. There are three reasons

why we feel that our mathematical modification of the V-slope method is justified. First, we very consistently found two breakpoints for the $\dot{V}E$ vs. $\dot{V}O_2$ and $\dot{V}CO_2$ vs. $\dot{V}O_2$ relationships and only one breakpoint, the RC point, for $\dot{V}E$ vs. $\dot{V}CO_2$, as one would expect. Second, the breakpoints were found at exercise intensities that one would expect for the AT and RC points, and very close in terms of percent peak $\dot{V}O_2$ to the AT and RC points found by Beaver et al.[12] Third, a visually correct fit was maintained for derived variables, as shown in Fig. 3.2.

Our data show that the marked reduction in peak $\dot{V}CO_2$ relative to peak $\dot{V}O_2$ during pregnancy results from a shallower slope of $\dot{V}CO_2$ vs. $\dot{V}O_2$ above AT, compared with postpartum. This most likely reflects a reduction in the buffering of lactic acid by bicarbonate. Because our data does not allow identification of the underlying cause, we may only speculate about the possible mechanisms. A reduction in the rate of lactic acid production is unlikely as maximal power was virtually unaffected by gestation. Increased utilization of lactate during pregnancy by the liver[93] or by the fetoplacental unit - for which it is the second most important substrate[11] - might possibly explain the observed reduction in maximal $\dot{V}CO_2$ in pregnancy.

CHAPTER 4

RESPIRATORY AND METABOLIC RESPONSES TO ENDURANCE CYCLE EXERCISE IN PREGNANT AND POSTPARTUM WOMEN[3]

4.1 *Introduction*

Competitive endurance exercise is usually performed at an intensity of 70-80% of $\dot{V}O_{2max}$, or at a HR between 160-180 beats per minute.[7] Because most studies on endurance exercise in pregnant women were performed at lower exercise intensities,[15,20,21,75] little is known about the physiologic effects of strenuous endurance exercise in pregnancy. However, it has been suggested in the literature that the physiologic responses of pregnant women to endurance exercise are different from those in nonpregnant women with regard to glucose metabolism[15,20,21,75] and thermoregulation.[21] In contrast, there is evidence that women perform rapidly progressive maximal weightbearing and nonweightbearing exercises equally well in pregnancy as in the nonpregnant state: peak $\dot{V}O_2$ during cycling[61,83,ch 5.2.] and running[61] are unaffected by gestation. This suggests that women, like females of several other species, are quite capable of performing strenuous endurance exercise in pregnancy.

To test the null hypothesis that the ability of women to perform strenuous endurance exercise in late pregnancy is equal to that in the nonpregnant state, we undertook a longitudinal study in women at 30-34 weeks of pregnancy and 8-12 weeks

[3]The main substance of this chapter was accepted for publication in F.K. Lotgering, W.E.M. Spinnewijn, P. Struijk, H.C.S. Wallenburg. Respiratory and metabolic responses to endurance exercise in pregnant and postpartum women. Int. J. Sports Med.

after delivery, who performed cycle exercise intended to last for 40 minutes at 70% peak $\dot{V}O_2$.

4.2 Methods

Subjects

For the study we recruited 22 healthy women at 30-34 weeks gestation and 8-12 weeks postpartum. All women had uncomplicated singleton pregnancies. The physical fitness of the subjects was variable and ranged from women with a sedentary lifestyle to those who participated in competitive sports. All volunteers were familiar with cycling, and they were all on a mixed diet. The study was approved by the Hospital and University Ethics Committee, and all women recruited gave informed consent.

Study protocol

Each subject underwent a total of five tests: three maximal and two endurance cycle tests. The initial test, a maximal test, was performed to allow the subjects to become acquainted with the experimental circumstances; the results of this test were discarded. Thereafter, at 30-34 weeks of pregnancy, a set of tests was performed: a maximal test followed by an endurance test. The same set of tests was repeated at 8-12 weeks postpartum. The maximal and endurance tests of each period took place on separate days of the same week at approximately the same time of day for each subject. We analyzed the data obtained from both tests at both time periods after exclusion of data from those women who had been unwilling or unable to perform all tests.

Before each test we measured body mass, inserted a catheter in an antecubital vein, performed a routine physical and obstetric examination, and monitored the FHR (HP 8040A FHR monitor, Hewlett Packard, Boeblingen, Germany) with the woman in semirecumbent position, to confirm the health of all individuals participating in the study.

The tests took place in an air-conditioned room kept at 21°C and 55% humidity. After 20 minutes of fetal monitoring at rest the subject was seated on an electrically braked cycle ergometer (Ergoline 900, Mijnhardt, Bunnik, The Netherlands) and connected by a face mask (Rudolph mask #7910 series, Hans Rudolph Inc, Kansas City,

MO) and a 1.5 m flexible outflow tube (ID 30 mm) to a gas flowmeter and O_2 and CO_2 analyzer (Oxycon-4, Mijnhardt). She was also connected to a heart rate monitor (Sport tester, Polar Electro, Kempele, Finland). Baseline measurements were taken during 5 minutes of sitting at rest; then the woman started to cycle.

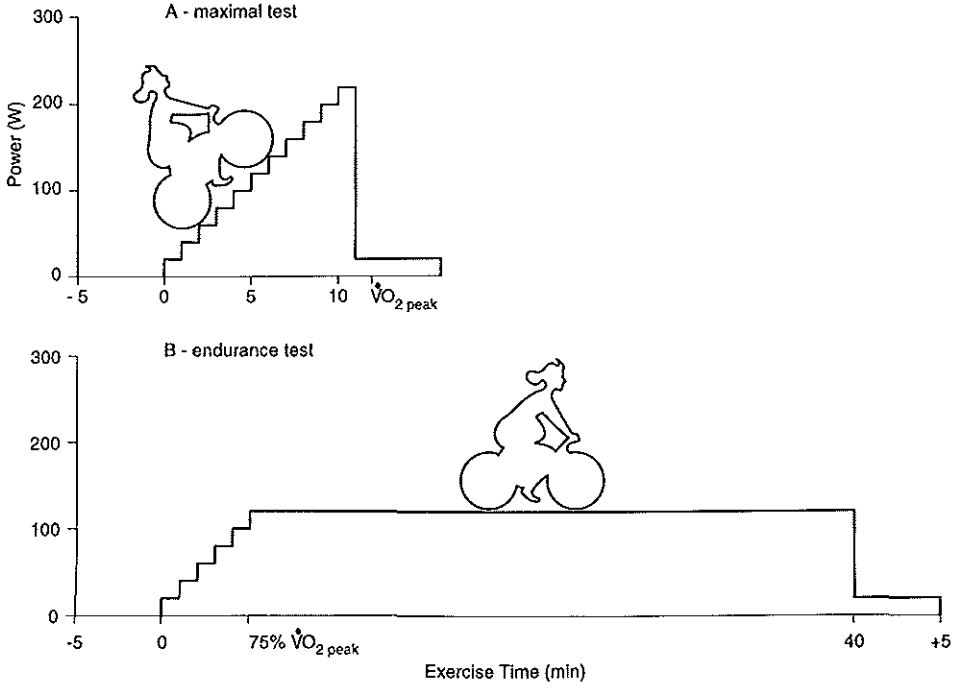

Fig. 4.1 Exercise protocol for the maximal (A) and the endurance (B) test.

Maximal testing

The exercise protocol is shown in Fig. 4.1A. During the maximal cycle test the initial power (20 W) was increased by 20 W per minute until the subject reached perceived maximal exertion. This was followed by 5 minutes of cooling down at 20 W. Thereafter the subject recovered for 20 minutes in the semirecumbent position while the FHR was being recorded.

Endurance testing

From the maximal test we determined the power, matching approximately 70% peak $\dot{V}O_2$, at which the woman would perform endurance exercise. The endurance test was performed on a separate day. The protocol for endurance testing is shown in Fig. 4.1B. After the control period at rest the initial power (20 W) was increased by 20 Watt per minute until the predetermined power, that matched 70-75% peak $\dot{V}O_2$, was reached. We asked the volunteers to perform at this level for 40 minutes, but several women reached perceived maximal exertion before this point. If exercise was maintained for less than 20 minutes, the data were considered not to represent true endurance activity and were discarded. The exercise was followed by 5 minutes of cooling down at 20 W. Thereafter the subject was returned to the semirecumbent position and recovered for 20 minutes while the FHR was being recorded.

Measurements and calculations

We continuously measured HR, gas flow and expiratory concentrations of O_2 and CO_2, and recorded time. All data were stored on a computer (model PCS 286, Olivetti, Ivrea, Italy). On-line 30-seconds average values of HR, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and respiratory exchange ratio (R) were calculated; 60-seconds changes in power and 30-seconds average maternal HR values were added off-line. From the 30-seconds average values we calculated mean values at 2.0-4.5 minutes of rest, at 10 (9.0-11.0) minutes of endurance exercise and 3.5-1.5 minutes before the end of the endurance test. From the maximal test we report the 30-seconds average peak values. We defined peak values of a variable as those values measured at the highest power. We defined $\dot{V}O_2$ plateau as an increase in $\dot{V}O_2$ of less than 5% in response to an increase in power.

Venous blood samples were obtained at rest, within 1.0 minute before the peak of the maximal test, at 10 (9-11) minutes of exercise at 70-75% peak $\dot{V}O_2$ and 4 (5-2) minutes before the end of the endurance test. We determined the concentrations of hemoglobin (EDTA tube; Sysmex NE-8000, Charles Goffin, Kobe, Japan), glucose (fluoride oxalate tube; EPOS, Merck, Hamburg, Germany), lactic acid (EDTA tube, kept on ice, analysis within one hour after sampling; ACA-analyzer, Du Pont, Wilmington, DE), and free fatty acids (EDTA tube, kept on ice, centrifuged within one hour after sampling, plasma stored at -20°; analysis by Bligh and Dyer method),

norepinephrine, epinephrine, and dopamine, (iced heparinized glutathione tube, centrifuged within one hour after sampling, plasma stored at -70°C; analysis by HPLC; fluorometric detection after liquid-liquid extraction and derivatization with the fluorogenic agent 1,2-diphenylethylenediamine, as described by Van der Hoorn et al.[48]

Statistical analysis

For each test period and each variable under consideration, we computed means ± SE. We used SPSS/PC + V 5.02 (SPSS, Chicago, IL) to perform Friedman two way analysis of variance and Wilcoxon signed-rank test to analyze differences between paired variables. A p-value of < 0.05 was taken as the level of significance.

4.3 Results

Of the 22 women recruited, one woman was hospitalized with threatened preterm labor before the first endurance test and two withdrew from the postpartum tests for personal reasons. Nineteen women completed all tests, but we discarded the data of three women: one because of deviation from the protocol and two in which endurance exercise was not maintained for at least 20 minutes. The data from the remaining 16 women were included in the analysis.

All 16 women, 12 nulliparous and four parous, remained healthy throughout the study period and all but one delivered a healthy infant. Apparently unrelated to the exercise, one baby was born alive with congenital anomalies. The mean age at delivery was 33.8±0.8 years, gestational age was 39.0±0.3 weeks, and birth weight was 3.30±0.15 kg. Fourteen women were lactating at the time of the postpartum test. Body mass during pregnancy (73.3±2.2 kg) was significantly higher than that post partum (66.8±2.2 kg). Peak $\dot{V}O_2$ per kilogram of body mass determined during postpartum cycling was taken as an index of physical fitness and varied between 26.5 and 51.2 ml O_2 per minute per kg, with a mean of 36.4 ml O_2 per minute per kg. There was no significant relationship between the level of physical fitness, parity, or lactation status and the difference between peak $\dot{V}O_2$ values in pregnancy and postpartum. Therefore, we report on the 16 volunteers as a uniform group.

Table 4.1. Effect of pregnancy on heart rate and ventilatory variables during maximal and endurance cycling (n=16).

	Power (W)	HR (beats/min)	$\dot{V}O_2$ (l/min)	$\dot{V}CO_2$ (l/min)	$\dot{V}E$ (l/min)
MAXIMAL TEST					
At rest					
pregnant	-	89±3*	0.32±0.01*	0.26±0.01*	11.86±0.47*
postpartum	-	76±2	0.27±0.01	0.21±0.01	8.82±0.37
At maximum					
pregnant	214±9	175±3	2.50±0.10	2.74±0.10	98.46±3.82*
postpartum	211±8	179±2	2.46±0.10	2.74±0.09	87.88±3.79
ENDURANCE TEST					
At rest					
pregnant	-	88±3*	0.32±0.02*	0.26±0.01*	11.96±0.54*
postpartum	-	78±3	0.27±0.01	0.22±0.01	8.96±0.35
At 10 min					
pregnant	124±8	162±3‡	1.88±0.07‡	1.77±0.07‡	64.76±3.03*‡
postpartum	124±8	164±3‡	1.83±0.08‡	1.72±0.07‡	55.81±2.59‡
At the end					
pregnant	124±8	173±3	1.87±0.07‡	1.68±0.07‡	69.97±3.53*‡
postpartum	124±8	176±3	1.92±0.08‡	1.69±0.07‡	61.62±3.31‡

Values are means ± SE; * $p < 0.05$; compared with postpartum control, ‡ $p < 0.05$ endurance exercise values compared with peak effort.

Table 4.2. Effect of pregnancy on concentrations of hemoglobin and metabolic variables during maximal and endurance cycling (n=16).

	Hemoglobin (mmol/l)	Glucose (mmol/l)	Lactic acid (mmol/l)	Free fatty acids (μmol/l)
MAXIMAL TEST				
At rest				
pregnant	7.29±0.07*	4.55±0.33	1.23±0.13	346±65
postpartum	8.16±0.12	4.12±0.15	1.21±0.13	291±79
At maximum				
pregnant	8.29±0.12*	3.90±0.12*	6.88±0.67	185±34
postpartum	8.85±0.10	4.42±0.17	8.41±0.80	147±36
ENDURANCE TEST				
At rest				
pregnant	7.14±0.10*	4.62±0.17	1.38±0.08	218±27
postpartum	8.06±0.13	4.42±0.19	1.36±0.11	228±33
At 10 min				
pregnant	8.09±0.13*	3.49±0.11*‡	4.54±0.50*‡	156±19
postpartum	8.78±0.13	3.95±0.13‡	5.73±0.55‡	171±32
At the end				
pregnant	8.26±0.10*	3.34±0.17*‡	4.51±0.50*‡	404±62‡
postpartum	8.82±0.10	4.38±0.26	5.54±0.67‡	445±71‡

Values are means ± SE; * $p < 0.05$ compared with postpartum control, ‡ $p < 0.05$ endurance exercise values compared with peak effort.

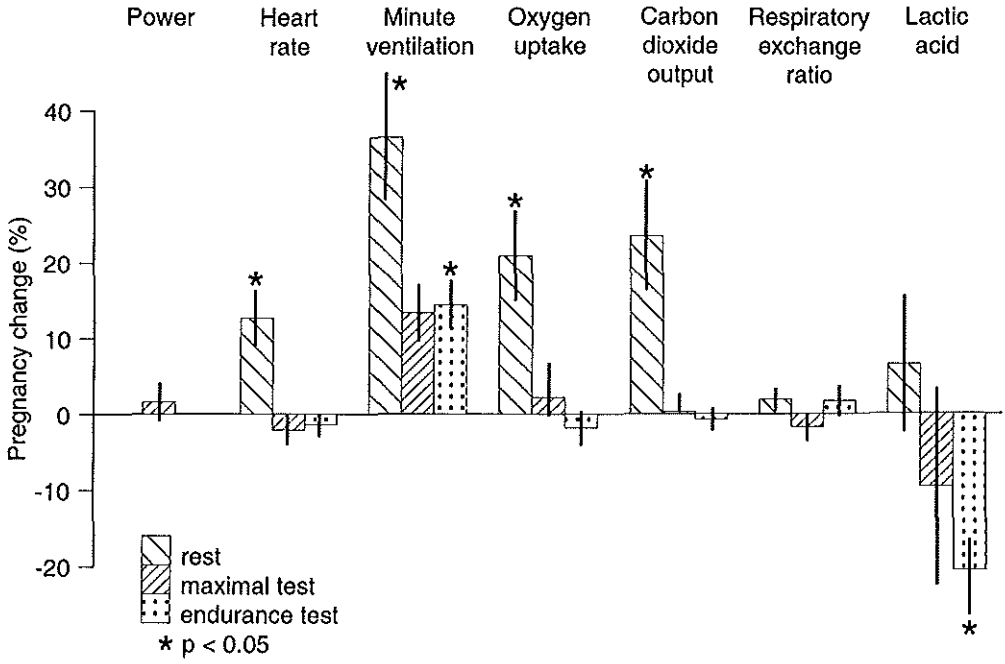

Fig. 4.2. Relative changes in selected variables during rest, maximal and endurance cycling as induced by pregnancy compared to postpartum controls.

Table 4.3. Effect of pregnancy on concentrations of catecholamines during maximal and endurance cycling (n=16).

	Norepinephrine (pg/ml)	Epinephrine (pg/ml)	Dopamine (pg/ml)
MAXIMAL TEST			
At rest			
pregnant	298±45	29±5	20±2
postpartum	258±28	29±6	20±3
At maximum			
pregnant	2262±358	217±45	64±9*
postpartum	2032±228	222±33	47±8
ENDURANCE TEST			
At rest			
pregnant	254±27	27±5	20±3
postpartum	244±33	27±4	18±2
At 10 min			
pregnant	1160±130‡	92±16‡	50±6
postpartum	1223±165‡	111±16‡	42±7
At the end			
pregnant	1554±236	170±32	95±17
postpartum	1989±293	237±38	109±21‡

Values are means ± SE; * $p < 0.05$ compared with postpartum control, ‡ $p < 0.05$ endurance exercise values compared with peak effort.

Values at rest.

The effects of pregnancy on the variables under consideration, at rest and during exercise, are summarized in Tables 4.1. to 4.3., the relative changes are shown in Fig. 4.2. Control values of all variables measured at rest before endurance exercise were not statistically different from the values determined prior to the maximal tests. Median control values at rest were significantly higher in pregnancy than postpartum, 13% for HR, 19% for $\dot{V}O_2$, 18% for $\dot{V}CO_2$ and 33% for $\dot{V}E$; R was not significantly increased in pregnancy (0.81±0.01) compared with postpartum (0.80±0.01). The pregnancy-induced increase in $\dot{V}E$ resulted from a 20% increase in tidal volume, and an 11% higher breathing rate. The hemoglobin concentration at rest was 11% lower in pregnancy than postpartum. Gestation did not affect the concentrations at rest of glucose, lactic acid, and free fatty acids, or of the hormones norepinephrine, epinephrine, and dopamine.

Maximal test

The imposed load was increased linearly with time. Peak effort in pregnancy (214±9 W) was not significantly different from that postpartum (211±8 W). The basal FHR was 138±6 beats per minute at rest, was increased to 147±6 beats per minute during the first 10 minutes of recovery, decreased with time, and returned to 142±7 beats per minute during the second 10 minutes of recovery from maximal cycling. None of the fetuses showed signs of hypoxic distress, i.e. loss of heart rate variability and late decelerations.

Peak values of HR, $\dot{V}O_2$, $\dot{V}CO_2$, and R were not statistically different between pregnant and postpartum women. Peak $\dot{V}E$, however, was significantly higher by 12% in pregnancy than postpartum as a result of a 5% higher tidal volume, and a 7% higher breathing rate. A $\dot{V}O_2$ plateau was reached in 69% of the tests.

The hemoglobin concentration increased more markedly during exercise in pregnancy (14%) than postpartum (8%), but the hemoglobin concentration at maximal cycling remained lower during gestation. The glucose concentration decreased significantly during exercise in pregnancy (-14%), but not postpartum (+7%); as a result the glucose concentration at maximal cycling was 12% lower in pregnancy than postpartum. The lactic acid concentration increased 6-fold during the maximal exercise test in pregnancy and 7-fold postpartum; as a result the lactic acid concentration at maximal effort was 18% lower during gestation than postpartum, a nonsignificant difference. Independent of pregnancy, the mean concentration of free fatty acids at maximal cycling was almost 50% lower than at rest, but the difference was not significant.

The levels of norepinephrine and epinephrine increased approximately 7- to 8-fold during the maximal exercise test and those of dopamine 2- to 3-fold. These changes resulted in equally high values at maximal effort of catecholamines in pregnancy and postpartum, and 36% higher concentrations of dopamine during gestation.

Endurance test

Of the 16 volunteers, eight completed the 40-minutes test both during pregnancy and postpartum. Of the eight other women, who reached subjective exhaustion between 20 and 40 minutes of exercise, three completed the 40-minutes test in pregnancy but not

postpartum, three vice versa, and two completed none of the two tests. The average exercise time was 37.6±1.0 minutes (range 30 - 40 minutes) during pregnancy and 35.8±1.7 minutes (range 23-40 minutes) postpartum; the average power was identical in both periods at 124±8 W. The goal of 70-75% peak $\dot{V}O_2$ during endurance testing differed by a mean of -1 to +3%. The basal FHR was 138±6 beats per minute at rest, was increased to 148±8 beats per minute during the first 10 minutes of recovery, decreased with time, and returned to normal at 141±8 beats per minute during the second 10 minutes of recovery from endurance exercise. The FHR pattern was not indicative of hypoxic distress in any of the fetuses.

Values of HR, $\dot{V}O_2$, and $\dot{V}CO_2$ during endurance exercise in pregnancy were not different from those obtained postpartum, but VE was higher by approximately 15% during gestation. HR reached 92% of peak HR after 10 minutes of endurance exercise; thereafter it gradually increased towards 99% peak HR. Oxygen uptake during exercise in pregnancy remained constant at 75% peak $\dot{V}O_2$ (range 62-91%) from 10 minutes onwards; postpartum it increased from 74% peak $\dot{V}O_2$ at 10 minutes to 78% peak $\dot{V}O_2$ near the end of endurance exercise. In contrast, $\dot{V}CO_2$ fell slightly from about 64% peak $\dot{V}CO_2$ at 10 minutes to approximately 62% peak $\dot{V}CO_2$ near the end of endurance exercise. As a result, R decreased significantly during the course of endurance exercise, from 0.94±0.01 (10 minutes) to 0.90±0.01 (end) in pregnancy, and from 0.95±0.01 (10 minutes) to 0.88±0.02 (end) postpartum. $\dot{V}E$ increased during endurance testing to about 65% peak $\dot{V}E$ at 10 minutes and to 70% peak VE near the end of endurance testing.

The hemoglobin concentration increased during the first 10 minutes of endurance exercise, and slightly further thereafter, to values similar to those observed with the maximal test. The glucose concentrations were lower at 10 minutes of endurance testing than at rest, with reductions of 24% in pregnancy, and 11% postpartum. With continued exercise in pregnancy the glucose concentrations remained low, whereas postpartum they returned to control levels by the end of the test. During the first 10 minutes of endurance exercise the lactic acid concentration increased markedly, about 3-fold in pregnancy, and 4-fold postpartum, and remained constant thereafter. As a result lactic acid concentrations during endurance exercise in pregnancy were significantly lower than postpartum, by about 19%, and both pregnancy and postpartum lactic acid values

were significantly lower during endurance testing than during maximal testing by about 35%. Plasma concentrations of free fatty acids were significantly lower at 10 minutes of exercise than at rest, and increased markedly thereafter to reach values near the end of endurance exercise that were twice those of control values, and 2- to 3-fold those observed with maximal testing, independent of pregnancy.

Within 10 minutes of endurance testing plasma concentrations of norepinephrine had increased approximately 5-fold, those of epinephrine 4-fold, and dopamine levels 2-fold above control values; at the end of endurance exercise norepinephrine, epinephrine, and dopamine concentrations had increased 6-, 6-, and 5-fold, respectively, in pregnant women, and 8-, 9-, and 6-fold in postpartum women. Except for higher dopamine concentrations with postpartum endurance testing, values near the end of endurance testing were not significantly different from those observed in the maximal test.

4.4 *Discussion*

Exercise studies in pregnant women have long been hampered by concerns about fetal well-being. For that reason, only limited information is available on the effects of strenuous prolonged exercise in pregnant women. Animal experiments showed that exercise for 40 minutes at 70% $\dot{V}O_{2max}$ to the point of exhaustion does not cause fetal hypoxemia or acidosis.[59] Also human studies indicate that rapidly progressive exercise in uncomplicated pregnancy is not associated with fetal distress,[61] that exercise at about 60% of peak HR does not cause changes in fetal behavioral state, as described in chapter 2 and that sustained recreational exercise induces only a mild increase in fetal heart rate.[22] On the basis of this data we felt that it was safe to undertake a study of strenuous endurance exercise in pregnant women. FHR monitoring after maximal and endurance testing showed a temporary increase in basal heart rate, most likely as a result of increased body temperature, but did not show signs of fetal distress in any of the cases. This observation supports our assumption that strenuous prolonged exercise is not harmful to the healthy fetus of a healthy mother.

The respiratory values at rest observed in this study are in agreement with those previously published[61] and the results in chapter 5. The higher resting values of HR, $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}E$ during pregnancy reflect the high maternal cardiac output, the

metabolic needs of the fetus, and the high levels of progesterone. Our results on maximal cycling are in accordance with earlier observations.[61,ch 5.2.] Pregnancy does not affect peak power, HR, $\dot{V}O_2$, or $\dot{V}CO_2$, but peak $\dot{V}E$ is significantly increased during gestation, most likely as a result of high progesterone levels.

We tested the hypothesis that pregnant and nonpregnant women are equally capable of performing endurance exercise. To allow for a reliable comparison between the pregnant and the nonpregnant state, despite marked differences in body mass, we studied the women during cycling. The test was strenuous, aimed at a duration of 40 minutes at 70% peak $\dot{V}O_2$. On average, however, the women performed at a somewhat higher intensity and for that reason some of them gave up before they completed the 40 minutes of exercise. This occurred equally often and at the same power in pregnancy and postpartum, while the mean exercise time was slightly, but not significantly, higher during gestation than postpartum. For that reason we conclude that women are indeed able to produce the same amount of energy and can perform strenuous endurance exercise equally well in late pregnancy as in the nonpregnant state.

During endurance testing HR gradually increased towards peak values. This may be explained by increases in core temperature and catecholamine concentrations, and by dehydration.[9] Despite the elevated HR at rest in pregnancy, the exercise response was not significantly different from that observed postpartum. Minute ventilation was consistently higher in gestation than postpartum, which may be attributed to the effect of progesterone, and continued to increase by about 9% between 10 and 40 minutes of exercise in pregnant as well as in nonpregnant women. Also $\dot{V}O_2$ and $\dot{V}CO_2$ responses to endurance exercise were independent of pregnancy.

Between 10 and 40 minutes of exercise $\dot{V}O_2$ remained constant at about 75% peak $\dot{V}O_2$, while $\dot{V}CO_2$ decreased by 5%. As a result, R gradualy decreased during the course of the exercise. The change in R, which was not different between pregnant and postpartum women, suggests a change in the use of substrates towards utilization of free fatty acids. The putative progressive utilization of free fatty acids during endurance exercise is supported by the observation of a simultaneous increase in plasma free fatty acid concentration[8] and in the levels of catecholamines that are known to increase free fatty acid mobilization.[8] We observed no differences in catecholamine concentrations between pregnant and postpartum women, which is in agreement with the absence of

differences in free fatty acid concentrations between the states. With no significant differences in energy expenditure, R, and concentrations of free fatty acids and catecholamines between the pregnant and nonpregnant state, it is surprising that glucose and lactic acid concentrations were found significantly reduced during endurance exercise in pregnancy. Our study does not allow to identify the cause of these differences between the states, but the fact that the glucose and lactate concentrations during exercise were lower during pregnancy than postpartum is in agreement with observations reported in the literature.[19,20] It seems unlikely that the reduced lactic acid concentration in pregnant women would indicate an increased level of physical fitness or a reduced anaerobic metabolism. For that reason, one may speculate that lactic acid could have been used preferentially as a substrate by the liver of the mother[93] as well as by the fetoplacental unit for which it serves as the second most important substrate.[11]

We conclude that pregnant women are equally capable as are postpartum women of performing a strenuous nonweightbearing endurance task. The physiologic responses to endurance exercise are largely independent of gestation, but pregnant women maintain a higher minute ventilation during endurance exercise and their plasma lactic acid concentration is lower than in the nonpregnant state. The latter observation may reflect increased lactate utilization by the maternal liver or by the fetoplacental unit during pregnancy.

CHAPTER 5

EXERCISE IN WATER DURING PREGNANCY

Pregnant women have traditionally been discouraged to participate in physically demanding exercise because it was thought that this exercise could harm the mother and the fetus. More recently, however, many physically active women have wished to continue sports and exercise during pregnancy, and research over the past twenty years has shown that in general it is safe to do so.

Swimming and diving are popular sports and swimming is often recommended to maintain a good physical condition during gestation. Yet, surprisingly little is known about the physiologic responses of the pregnant woman and her fetus to exercise in water. Most of our knowledge about exercise responses during pregnancy is derived from treadmill and cycle ergometer studies on land. Although it seems likely that the general mechanisms derived from these studies are valid also during water activities, it is possible that the water environment alters at least some physiologic responses.

In the following paragraphs we shall first briefly review what is known and what is not known about immersion, swimming, and diving in pregnancy. Second, we report the results of a study on peak ventilatory responses during cycling and swimming in pregnant and nonpregnant women.

5.1 *Review on swimming and diving by pregnant women*[4]

Immersion

Immersion causes physiologic changes as a result of the hydrostatic pressure and the temperature conductivity of the water. The mild increase in hydrostatic pressure during head-out immersion in approximately thermoneutral water of 34°C rapidly mobilizes interstitial fluid[36] and thereby increases the circulatory blood volume by about 9%.[86] As a result of the increased venous return of blood to the heart and the Frank-Starling mechanism, stroke volume and cardiac output are increased by about 30% without an increase in HR,[33] but mean arterial pressure falls by about 10%,[51,53] most likely because of reflex-relaxation of the vessel walls. In pregnancy, the circulation is hyperdynamic, with an approximate 40% increase in cardiac output through an increase in HR and stroke volume, and an equal reduction in systemic vascular resistance.[23,92] Despite the hyperdynamic circulation, the circulatory responses to immersion in pregnant and nonpregnant women are not much different with regard to HR[71,73,ch 5.2.] and mean arterial pressure.[71,73]

Immersion also affects ventilation and increases the work of breathing by 60%.[47] As a result of an increased intrathoracic blood volume and an upward displacement of the diaphragm by the hydrostatic pressure,[47] the forced vital capacity and the expiratory reserve volume are reduced by about 8% and 40%, respectively.[13] On the other hand, the inspiratory capacity increases, and both tidal volume[47] and maximal voluntary ventilation remain virtually unchanged.[13] Pregnancy increases inspiratory capacity and tidal volume without affecting maximal voluntary ventilation, and the ventilatory response to immersion is virtually unaltered.[13]

Water temperature has a marked influence on the physiologic responses to immersion. Below a thermoneutral temperature, increased heat production must compensate for conductive heat loss, or the body temperature will decrease. It has been

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[4] The main substance of this part of the chapter was published in: Frederik K Lotgering, Wilhelmina EM Spinnewijn and Henk CS Wallenburg. Swimming and diving by pregnant women. Fetal and Maternal Medicine Review. 1996;8:165-171

shown that immersion in water with a temperature of 33°C, not markedly affects $\dot{V}O_2$ or VE in either pregnant or nonpregnant women,[ch 5.2.] whereas $\dot{V}O_2$ in water of 30°C has been reported to increase by 50 to 100% in nonpregnant and pregnant women.[71] It has been calculated that pregnant women retain heat less well during immersion as a result of increased evaporative heat loss.[73] If pregnant women are subjected to water temperatures below thermoneutrality, they need to increase their metabolic rates more than nonpregnant women in order to maintain body temperature. Obviously, the physiologic responses associated with cooling will be more prominent at the lower water temperatures to which swimmers and divers are usually exposed.

Cycling in water

A group of investigators in Chapel Hill, NC, has studied exercise responses to cycling in water in pregnant and nonpregnant women.[13,51,70,71,73] This is not a common type of exercise in real life, and at least three methodological problems limit the value of the results. First, the water temperature in these experiments was 30°C, a few degrees below thermoneutrality. Second, the resistance to the moving legs is greater in water than in air, and most pregnant women were unable to pedal the ergometer for 20 minutes at an intensity higher than presumed 60% maximal capacity.[70] The authors themselves point out that water alters the HR response so that land-derived HR targets should not be used to define work rates in the water.[70] Third, all but one of the above studies,[51] lack comparative data on cycling in air.

During steady state cycling in water at a heart rate of about 130 beats per minute, blood pressure was increased by approximately 15% in pregnant and postpartum women.[71] Cardiac output during exercise in water showed higher values during the second half of pregnancy compared with postpartum values, but values at rest, known to be 40% increased during pregnancy,[23,92] were not reported. In a small group of pregnant women cycling in water and in air with comparable oxygen uptakes, a 13% reduction in HR and 11% reduction in blood pressure increment was found in water compared to air.[51] This suggests that total peripheral resistance during exercise is lower during pregnancy than after delivery,[71] as it is known to be the case at rest.

Levels of various hormones have been studied in pregnant and nonpregnant women cycling in water at 30°C. Concentrations of cortisol,[70] prolactin[52] and beta-

endorphin[72] at rest appeared to be elevated in pregnancy. Cycling in water did not affect the cortisol[70] or prolactin[52] concentrations, but a significant increase in beta-endorphin concentration was noted of similar magnitude in pregnant and nonpregnant women.[72] Because beta-endorphin concentrations do increase during hypothermia[24] the authors speculate that water temperature rather than exercise *per se* might have increased beta-endorphin concentration in their experiments.[72]

At the onset of cycling in water, skin temperature falls rather abrupt by approximately 0.6°C, which may be due to a reduction in skin blood flow or to greater convective heat loss by increased water movement.[73] Rectal temperature, however, gradually increases after 10 minutes of exercise at a maternal heart rate of about 130 beats per minute by 0.3°C in pregnancy and 0.1°C post partum. In contrast to a calculated reduction in heat retention in pregnant relative to postpartum women during immersion at rest, heat retention during exercise in water was calculated to be three times higher in pregnant than in postpartum women,[73] an adaptation which could serve as a protective mechanism for the fetus.

Swimming

The maximal oxygen uptake during swimming (swim peak $\dot{V}O_2$) is dependent on body size, training status, type of stroke, and water temperature.[43] Trained swimmers attain a swim peak $\dot{V}O_2$ that is about 6-7% lower than the maximal O_2 uptake that can be accomplished during running, and their swim peak $\dot{V}O_2$, is approximately similar to that during cycling. In untrained swimmers swim peak $\dot{V}O_2$ is approximately 20% lower than $\dot{V}O_{2max}$, and 10% lower than cycle peak $\dot{V}O_2$. At a given oxygen uptake, trained swimmers swim faster than untrained swimmers, as economy is increased as a result of reduced drag from a more streamlined stroke.[69] The highest peak $\dot{V}O_2$ is obtained using the breaststroke technique, which is a less efficient style than front crawl or even back crawl. Women may swim with greater economy than men, because of the increased buoyancy which results from their relatively higher fat mass.[69] The fact that the record for swimming the English channel was for some time held by a woman, seems to support the hydrodynamic advantage of women.[69] However, in 1994 a man improved the record.[68] In the absence of experimental data one may only speculate as to how the pregnancy-induced changes in body composition, most notably an increase in

water and fat, and in body dimensions might affect the economy of swimming by altering buoyancy and drag.

The physiologic responses to maximal swimming in pregnant women have been the subject of two studies.[74,ch 5.2.] In these studies breaststroke swimming was compared to cycling and a tethered swim ergometer[25] was used to provide increasing resistance to swimming. Breaststroke swimming requires more leg work and less arm work than other swimming techniques[41] and therefore provides the best possible comparison with cycling. In these studies swim peak $\dot{V}O_2$ was found to be lower than cycle peak $\dot{V}O_2$, both in pregnant and in nonpregnant women. This accords with the literature on untrained nonpregnant swimmers and probably reflects the somewhat smaller muscle mass involved in swimming. The authors of the first study found that the maximal swim $\dot{V}O_2$ was 17% lower during pregnancy than post partum and that this was associated with decreased ventilation.[74] This is in contrast to studies on land, which have consistently shown that cycle peak $\dot{V}O_2$ is unaffected by pregnancy.[61,74,ch 5.2.] Because the increase in resistance to swimming was not well defined, one cannot exclude the possibility that a difference in workrate may have contributed to the observed lower swim peak $\dot{V}O_2$ during pregnancy. The authors of the second study used a consistent progressive continuous swim protocol to allow optimal comparison to their cycle protocol.[ch 5.2.] In contrast to the first study, they did not observe a difference in swim peak $\dot{V}O_2$ between pregnant and nonpregnant women, nor higher swim peak ventilation in pregnancy. In addition, they found that pregnant women were able to sustain the same maximal resistance to swimming as nonpregnant women. Thus, pregnancy does not seem to affect either performance or aerobic capacity during swimming. The question if pregnancy affects the ability to perform strenuous endurance exercise has not been studied in the water, but it is unlikely that it will, because the endurance capacity of nonweightbearing exercise on land is unaffected by gestation as was shown in chapter 4.

In both studies discussed above,[74,ch 5.2.] the respiratory exchange ratio following swimming was about 6% lower than that following cycling, and 7-11% lower in pregnancy than post partum. This suggests a change in the use of substrates, favoring the utilization of free fatty acids in pregnant women during swimming, similar to the changes observed in nonpregnant subjects as a result of training. Hormonal changes that

might relate to the ventilatory or cardiovascular responses to maximal swimming in pregnancy have not been reported thus far, and cardiovascular changes in response to swimming in pregnancy have not been studied in detail. Maximal heart rate in swimming is similar to that in cycling and independent of pregnancy.[74,ch 5.2.] It has been calculated from the increase in hemoglobin concentration during swimming that the relative reduction in plasma volume during exercise is about the same in pregnant and postpartum women.[74] Blood pressure and cardiac output have not been studied in relation to swimming in pregnancy.

An unquantified increase in FHR was noted in 8 of 11 women following swimming for about 40 minutes,[84] a mean increase of approximately 5 beats per minute in FHR was reported following exercise in one maximal swim study[95] and no significant change from rest was found after exercise in another maximal swim study.[ch 5.2.] FHR increases with maternal body temperature and thus with the intensity and duration of exercise as well as with the water temperature. In two of 26 swim trials of one study, a FHR deceleration of less than 2 minutes duration was noted after swimming and the authors themselves question the importance of this finding.[95] The two other studies reported reactive and variable FHR patterns without decelerations after swimming, suggesting no impairment of fetal well-being[84,ch 5.2.] and neonatal outcome was uniformly good in all participants in the three studies. Results of Doppler ultrasound studies showed that umbilical and uterine blood flows are unaffected by a maximal interval swim test.[95] These data suggest that in a normal pregnancy a maximal swim trial is safe for the fetus. Whether this is also true for endurance swimming remains to be determined.

Diving

From a physiologic point of view diving and underwater swimming by breath-holding and with the use of selfcontained underwater breathing apparatus (SCUBA) is a fascinating exercise. A diver is exposed to increased surrounding pressure in water with a high heat capacity and conductivity, and he must breath. The classic Scolander diving response consists of apnea, bradycardia and peripheral vasoconstriction.[85] As described extensively in various reviews,[39,62] the massive peripheral vasoconstriction of the diving response in marine mammals is elicited through facial trigeminal receptors and arterial

chemoreceptors, and serves to redistribute blood flow in a state of bradycardia and low cardiac output induced by vagal activity. Blood flow to the brain is unaltered and flow to the heart, lungs and adrenal glands is relatively maintained, whereas blood flow to other parts of the body is markedly reduced. Through these adjustments the flow of oxygen is directed largely to the brain and heart, which allows mammals such as the Harbor seal to dive for approximately 20 minutes.

Diving animals such as seals have several other advantages over man. They are better adapted in terms of body composition, with a relatively small brain and heart, they have a relatively large (venous) blood volume and spleen, and a high muscle myoglobin concentration, they are capable of reducing their body temperature in anticipation of the dive, they are preadapted for fat utilization and the muscles can accumulate massive amounts of lactate while the arterial lactate concentration remains low during the dive. In addition, some marine animals are capable of metabolic and channel arrest, a most effective means of protecting tissues against hypoxia.[38]

Humans dive without these marvelous adaptations to the water environment and perform markedly less well. When the O_2 stores of a 70 kg man are about 1500 ml, moderately strenuous underwater swimming at a $\dot{V}O_2$ of approximately 1000 ml/minutes would result in an estimated time to unconsciousness of 80 seconds and to death from hypoxia in 4 minutes.[26] Korean Ama are professional breath-holding divers who dive to depths of 5-20 m for periods of 30-90 seconds, repeatedly for up to four hours each day, while surfacing for brief rest periods only.[49] In Ama, but not in occasional divers, the hematocrit increases by 10% when the spleen contracts and decreases in volume by 20%.[49] Although this demonstrates that man is capable of adaptation, the changes are of little quantitative importance in comparison with the seal. Humans may hyperventilate like the seal to increase their breath-holding time which reduces the CO_2 stores effectively but has little effect on O_2 stores of the body, and is therefore potentially dangerous.[26] Theoretically, also breath-holding diving carries the risk of decompression sickness,[81] but this is much more likely to occur in scuba diving.

Given the major physiologic changes and potential risks of breath-holding and scuba diving the question arises as to how this might affect pregnant individuals and their fetuses.

Fetal responses to normobaric breath-hold diving have been simulated in the

Weddell seal by submersion of the maternal head.[56] At the onset of the simulated dive, the fetal heart rate decreases rapidly, although it lags behind that of the mother, and it gradually decreases further to reach about 30% of its resting heart rate by the end of a 20 minutes dive. It recovers gradually and returns to baseline values within 10 minutes. During the dive placental blood flow is relative well maintained, as the result of a 6-fold increase in the fraction of maternal aortic flow.[56] Nonetheless, the fetal blood gas values change during the dive. PO_2 gradually decregases from a pre-dive value of 24 mm Hg to 9 mm Hg at the end of the dive; recovery is complete within 8 minutes. PCO_2 increases progressively during the dive to reach 85 mm Hg, while the fetal-maternal gradient remains constant at about 11 mm Hg. The fetal arterial pH decreases linearly with time to a value of 7.23 near the end of the dive, and decreases further to a pH of 7.15 in the post-dive period as a result of lactate washout. From this data, it appears that O_2 conservation mechanisms in the fetus tend to be similar to those in the mother.[56] Simulated diving in acutely instrumented animals is more stressful than free diving in chronically instrumented seals. In the free diving chronically instrumented Weddell seal the fetal heart rate was found to decrease gradually to only about 50% of its pre-dive value near the end of 37 minutes dive to 200 m.[40] Weddell seal fetuses are apparently unharmed by the marked changes in their blood gas values and heart rates. It is unknown to what extent this is true for the human fetus.

Scuba diving during pregnancy has been studied with retrospective questionnaires in humans[14] and has been simulated in experimental animals.[16,34,76,87] Questionnaire data showed no significant differences between women diving (n=109) and not diving (n=69) during pregnancy with regard to the incidence of perinatal death, low birth weight, vaginal bleeding or spontaneous abortion. However, a significantly higher frequency of birth defects was noticed in divers (6/109 = 5.5%) compared to non-divers. The defects consisted of skeletal malformations in two neonates, and a ventricular septal defect, a coarctation of the aorta, a pyloric stenosis and a hairy birthmark, in the four others. The two cases of skeletal malformations; one case of multiple hemivertebrae and one missing hand, occurred in two out of 20 women who dived deeper than 30 m, during the first trimester, and one cannot dismiss the possibility that they may have been caused by the diving. The four other women dived to a depth of less than 30 m in the first trimester. Animal experiments[76] provide

evidence that the fetus is unlikely to suffer from decompression sickness even after multiple exposures to pressures appropriate for sport diving. Therefore, the recommendation that pregnant scuba divers should double the times indicated in standard decompression tables,[14] is based on prudence rather than on evidence. This is even more true for the advise provided by British and American diving organizations that women who are pregnant or trying to become pregnant should not dive if they want to be certain that their pregnancy will not be affected by diving.[27]

5.2 *Peak ventilatory responses during cycling and swimming in pregnant and nonpregnant women*[5]

5.2.1 Introduction

When peak $\dot{V}O_2$ during cycling is known to be unaffected by pregnancy,[61,74,83] one would expect this would be true also for other types of nonweight-bearing exercise. Because water offers some relieve from gravitational force, one would expect this also applies to swimming. However, in the only published study on peak $\dot{V}O_2$ which compares swimming in pregnant and postpartum women,[74] swim peak $\dot{V}O_2$ was reported as 17% lower during gestation than post partum. The authors did not offer a compelling theoretical rationale for the lower swim peak $\dot{V}O_2$ observed during pregnancy but suggested that either the water environment could be responsible, or that pregnant women do not push themselves as hard during swimming as during cycling.

The purpose of our study was to test the hypothesis that, contrary to the unexpected finding in the literature,[74] swim peak $\dot{V}O_2$ is not reduced by pregnancy and is lower than cycle peak $\dot{V}O_2$ in pregnant as well as in nonpregnant women. We used a progressive, continuous exercise protocol for longitudinal comparison of the ventilatory responses to swimming and cycling in pregnant and postpartum women.

5.2.2 Methods

Subjects

From January 1994 to December 1994 we studied 11 healthy women at 30-35 weeks pregnancy and 8-12 weeks post partum. All women had uncomplicated singleton pregnancies. The physical fitness of the subjects who entered the study was variable and

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[5]The main substance of this part of the chapter was published in: WEM Spinnewijn, HCS Wallenburg, PC Struijk and FK Lotgering. Peak ventilatory responses during cycling and swimming in pregnant and nonpregnant women. J.Appl.Physiol. 1996;81:738-742

ranged from women with a sedentary lifestyle to recreational sportswomen. All volunteers were familiar with cycling and breaststroke swimming without participating in a physical conditioning program or specific training in either sport prior to or during the study period. The study was approved by the Hospital and University Ethics Committee and all women recruited gave their informed consent.

Study protocol

Each subject underwent a total of six rapidly progressive maximal tests: three cycle and three swim tests. The first set of tests, cycling and swimming, was performed to allow the subjects to become acquainted with the experimental circumstances; the results of these tests were discarded. The second set of tests was performed at 30 - 34 weeks of pregnancy and the third set of tests at 8 - 12 weeks post partum. The cycle and swim tests of each period took place on separate days of the same week, at approximately the same time of day for each subject. The order of the cycle and swim tests was assigned randomly.

Before each test we measured body mass, performed a routine physical and obstetric examination, and recorded the FHR with an FHR monitor (HP 8040A, Hewlett-Packard, Boeblingen, Germany), with the volunteer in the semirecumbent position, to confirm the health of all individuals participating in the study.

The cycle tests took place in an air-conditioned room kept at 21°C, on an electrically braked cycle ergometer (Ergoline 900, Mijnhardt, Bunnik, The Netherlands). We used a Sport tester (Polar electro, Kempele, Finland), with the electrodes placed on both sides of the thorax just below the breasts and the receiver around the wrist, to measure the HR continuously and to store the HR data as 30-seconds average values. After 20 minutes of rest the subject was seated on the cycle ergometer and connected by a rubber mouthpiece, attached to a two-way valve (Model 2700 series, Hans Rudolph, Kansas City, MO) with a flexible 0.6-m inflow and 2.5-m outflow tube (id 30 mm), to a gas flowmeter and O_2 and CO_2 analyser (Oxycon-4, Mijnhardt, Bunnik, The Netherlands). A noseclip prevented nasal breathing. Baseline measurements were taken during 5 minutes of sitting at rest, whereafter the volunteer started to cycle. The initial power (20 W) was increased by 20 Watt every minute until the subject reached perceived maximal exertion. Recovery values were taken during 5 minutes with

the volunteer sitting at rest on the cycle ergometer; whereafter she was returned to the semirecumbent position and a venous blood sample was taken, 5 to 6 minutes after the exercise, to determine the plasma lactic acid concentration. Thereafter the FHR was recorded for 20 minutes.

The swim tests took place in a 8.0 x 4.5-m pool with a water temperature of 33°C and an air temperature of 26°C. The pool had a movable platform, which allowed us to adjust the level of immersion of the upright subject. We used a tethered swim ergometer modified after Costill.[25] The ergometer consists of an adjustable weight connected by a pulley to a belt around the woman's waist. Because the HR signal was sometimes not picked up by the wrist receiver during swimming, a second receiver was attached to the swim suit. After 20 minutes of rest the volunteer took place on the platform of the pool, standing in air without immersion. Baseline measurements were taken during five minutes, as prior to the cycling test. The platform was then lowered and further measurements were taken during 5 minutes of head-out immersion standing in the water. After these 10 minutes of standing at rest, the subject started to swim with a breaststroke. The initial weight of 0.5 kg, connected to the woman's waist by the pulley, was increased by 0.5 kg every minute until the subject reached perceived maximal exertion and could no longer sustain the pull. She then recovered for five minutes standing in the water, after which the study protocol was identical to that following cycling.

Measurements and calculations

We continuously measured gas flow and expiratory O_2 and CO_2 concentrations (Oxycon-4, Mijnhardt, Bunnik, The Netherlands) and recorded time. All data were stored on a computer (PCS286, Olivetti, Ivrea, Italy). On-line 30-seconds average values were calculated of $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and R; 60-seconds changes in power and 30-seconds average maternal heart rate values were added off-line. The venous plasma lactic acid concentrations were determined from 4.5 ml blood samples, drawn anaerobically into EDTA tubes and kept on ice until analysis within one hour after sampling, with the use of the oxidation method (ACA-analyzer, Du Pont Compagny, Wilmington, DE). We defined peak values of a variable as those values measured at the highest power. We defined $\dot{V}O_2$ plateau as an increase in $\dot{V}O_2$ of less than 5% in

response to an increase in power.

Statistical analysis

From the 30-s average values we calculated mean values at 2.0 - 4.5 minutes of rest. For each test period and each variable under consideration, we computed means ± SE. We used SPSS/PC + V 5.02 (SPSS, Chicago, IL) to perform Friedman two way analysis of variamce and Wilcoxon signed-rank test to analyze differences between paired variables. A p-value of < 0.05 was taken as the level of significance.

5.2.3 Results

Of the 13 women recruited, two women found the test too physically demanding, indicated that they did not perform at their maximum, and had maximal heart rates of less than 140 beats per minute. The data of these two women were discarded; the remaining 11 women completed all tests. Gestational age at the time of the test was 33.1 ± 0.5 weeks; the postpartum test was performed 12.0 ± 0.4 weeks after delivery. The FHR patterns during recovery from both cycling and swimming were similar to those in the control period, without significant changes in basal FHR, loss of variability, or appearance of decelerations that might indicate fetal distress.

All 11 women, seven nulliparous and four parous, remained healthy throughout the study period and delivered healthy infants. Age at delivery was 32.5 ± 1.3 years, gestational age was 40.1 ± 0.3 weeks and birth weight was 3.45 ± 0.12 kg. Eight women were lactating at the time of the postpartum test. Body mass during pregnancy (74.9 ± 3.0 kg) was significantly different from postpartum values (67.6 ± 2.9 kg). Peak $\dot{V}O_2$ per kilogram of body mass determined during post partum cycling was taken as an index of physical fitness and varied between 28 and 57 ml O_2 per minute per kg, with a mean value of 34 ml O_2 per minute per kg. There was no significant relationship between the level of physical fitness, parity, or lactation status and the difference between peak $\dot{V}O_2$ values in pregnancy and post partum for either type of exercise. Therefore, we report on the 11 volunteers as a uniform group.

Control values at rest

The effects of pregnancy on control values are summarized in Table 5.1.

Table 5.1. Effect of pregnancy on control values at rest (n=11).

	HR (beats/min)	$\dot{V}O2$ (l/min)	$\dot{V}CO2$ (l/min)	$\dot{V}E$ (l/min)
Sitting				
pregnant	$90\pm3^{*}$	0.33 ± 0.01	0.27 ± 0.01	$11.95\pm0.42^{*}$
postpartum	81 ± 3	0.30 ± 0.02	0.24 ± 0.01	10.36 ± 0.34
Standing, in air				
pregnant	$99\pm3^{*\dagger}$	$0.37\pm0.02^{*\dagger}$	$0.30\pm0.02^{*}$	$13.54\pm0.68^{*\dagger}$
postpartum	83 ± 3	0.32 ± 0.01	0.25 ± 0.01	10.69 ± 0.46
Standing, in water				
pregnant	$82\pm3^{*\pi}$	$0.38\pm0.02^{*\dagger}$	$0.33\pm0.01^{\dagger}$	$14.06\pm0.63^{*\dagger}$
postpartum	$69\pm2^{\dagger\pi}$	$0.35\pm0.02^{\dagger}$	$0.31\pm0.01^{\pi}$	$11.99\pm0.54^{\dagger\pi}$

Values are means $\pm$ SE; * $p < 0.05$ compared with postpartum control; † $p < 0.05$ compared with sitting; $^{\pi}$ $p < 0.05$ compared with standing in air.

In all positions studied HR, $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}E$ at rest were higher in pregnancy than post partum. The increase in $\dot{V}E$ in pregnancy was caused by a significant rise in tidal volume (Vt), without a difference in breathing rate. Sitting on the cycle ergometer, the pregnancy-induced increase was 11% in HR, 10% for $\dot{V}O_2$, 13% for $\dot{V}CO_2$, and 15% for $\dot{V}E$, respectively, while R was not affected, with values of 0.81 ± 0.02 (pregnancy and postpartum).

Compared to sitting, standing in air was associated with somewhat higher values of HR, $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}E$ in both the pregnant and nonpregnant state, but the differences reached statistical significance only for HR (10%), $\dot{V}O_2$ (12%) and $\dot{V}E$ (13%) in the pregnant state. R values in standing position, 0.80 ± 0.01 and 0.81 ± 0.02 in pregnancy and postpartum, respectively, were not different from those in the sitting position.

Compared with standing in air, standing in water was associated with a significantly (17%) lower HR in the pregnant and the nonpregnant state. Values of $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}E$ were higher in the water than in air, but the increases were statistically significant only for $\dot{V}CO_2$ and for $\dot{V}E$ in the nonpregnant state. R values in the water,

0.86±0.02 and 0.91±0.04 in pregnancy and postpartum, respectively, were significantly higher than in air (+ 8%) in pregnancy as well as post partum (+ 12%).

Compared with sitting in air, standing in water was associated with significantly lower values of HR and higher values of $\dot{V}O_2$, $\dot{V}CO_2$ $\dot{V}E$ and R in both pregnant and postpartum women.

Peak aerobic exercise

The imposed load was increased linearly with time for both cycling and swimming, and the mean time to reach perceived maximal exertion (10 minutes) was not significantly different between periods or test types as shown in Table 5.2.

Table 5.2. Effect of pregnancy on peak responses during cycling and swimming (n=7 for HR, n=11 for all other variables).

	Exercise Time (min)	HR (beats/min)	$\dot{V}O2$ (l/min)	$\dot{V}CO2$ (l/min)	$\dot{V}E$ (l/min)
Cycling					
pregnant	10.05±0.61	171±7	2.36±0.12	2.76±0.16	92.55±5.34
postpartum	9.77±0.46	180±6	2.29±0.10	2.82±0.11	86.37±3.60
Swimming					
pregnant	9.77±0.45	169±6	2.11±0.11†	2.08±0.09*†	73.36±4.38†
postpartum	10.18±0.59	171±5	2.12±0.07†	2.32±0.09†	70.35±3.32†

Values are means ± SE; * $p < 0.05$ compared with postpartum control; † $p < 0.05$ compared with cycling values.

Cycling

HR and VO_2 showed a linear increase with power. Absolute peak values of HR, $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}E$ during cycling were not significantly different between pregnant and postpartum women. A $\dot{V}O_2$ plateau was found in 73% of the tests. In the absence of significant differences in peak $\dot{V}O_2$ and peak $\dot{V}CO_2$, also no significant difference in R was observed between test periods, with values of 1.18±0.03 and 1.24±0.02 in pregnancy and postpartum, respectively. Peak $\dot{V}E$ was increased slightly (7%) and nonsignificantly during gestation compared with post partum, as a result of a 10% higher Vt, with no difference in respiratory rate. The venous lactic acid concentration

during recovery was significantly lower in pregnancy (8.0±0.7 mmol/l) than post partum (9.6±0.6 mmol/l).

Swimming

Most participants found the low initial weight (0.5 kg) uncomfortable and reacted with an irregular stroke technique to remain floating. After about 3 minutes (1.5 kg) the strokes became more regular. This was reflected in HR and $\dot{V}O_2$, which were irregular during the first 3 minutes of swimming but increased linearly with time thereafter. No reliable HR signal was obtained in five of 22 swimming tests. Therefore only seven data pairs were available for comparison. As with cycling, peak values of HR, $\dot{V}O_2$ and $\dot{V}E$ during swimming were not significantly different between pregnant and postpartum women. A $\dot{V}O_2$ plateau was found in 73% of the tests. peak $\dot{V}O_2$ was not affected by gestation, but peak $\dot{V}CO_2$ during swimming was significantly lower during pregnancy than post partum (-10%). As a consequence, R was on average 11% lower during pregnancy (1.01±0.03) than post partum (1.14±0.03). Peak $\dot{V}E$ was slightly but not significantly increased (4%) during pregnancy compared with post partum as a result of 8% higher Vt, with no difference in respiratory rate. The venous lactic acid concentration during recovery from swimming was significantly lower in pregnancy (5.8±0.5 mmol/l) than post partum (8.4±1.0 mmol/l).

Swimming vs cycling

Exercise time and peak HR were not significantly different between exercise types. However, peak values of $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ were significantly lower during swimming than during cycling, in pregnancy and in the postpartum period. A typical example of the relationship between $\dot{V}O_2$ and $\dot{V}CO_2$ during cycling and swimming in pregnancy and post partum is shown in Fig. 5.1. Peak $\dot{V}O_2$ values were lower during swimming than during cycling by 11% in pregnancy and by 7% post partum, and peak values of $\dot{V}CO_2$ were lower by 25% in pregnancy and by 18% postpartum. As a result of the more pronounced reduction in peak $\dot{V}CO_2$ than in peak $\dot{V}O_2$ during swimming than during cycling, peak values of R were significantly lower during swimming, by 14% in pregnancy and by 6% post partum. Associated with the reduced peak $\dot{V}CO_2$ during swimming, peak $\dot{V}E$ was also markedly lower during swimming than during

cycling, by 21% in pregnancy and by 19% post partum. The lower peak $\dot{V}E$ during swimming resulted from a significantly lower Vt during swimming than during cycling, by 18% in pregnancy and by 16% post partum, with no difference in peak respiratory rate. The venous lactic acid concentration was significantly lower during recovery from swimming than from cycling by 27% in pregnancy and by a statistically not significant 12% post partum.

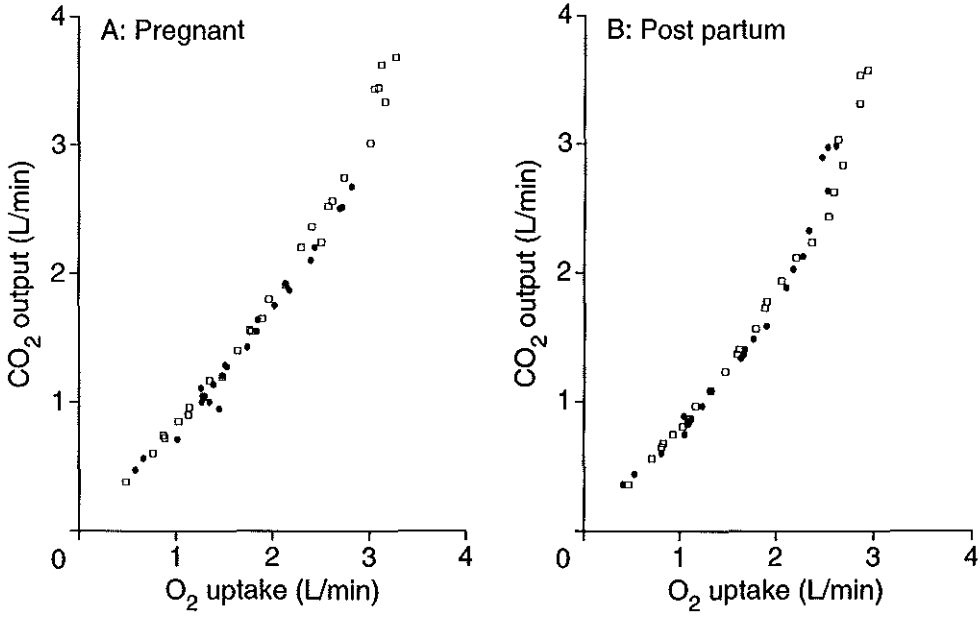

Fig. 5.1. Relationship between O_2 uptake and CO_2 output during cycling (□) and swimming (•) in pregnancy (*A*) and postpartum (*B*). Swim and cycle data are superimposed on each other. In pregnancy and postpartum a lower peak value is obtained in swimming than in cycling.

5.2.4 Discussion

$\dot{V}O_2$ during swimming is dependent on training, swimming technique and body dimensions.[43,44] The volunteers in our study had a variable level of physical fitness and were familiar with cycling and breaststroke swimming, without being competitive

cyclists or swimmers. We chose to study breaststroke swimming, because this technique requires more leg work and less arm work than other types of swimming[42] and for that reason allows the best possible comparison with cycling. We used tethered swimming because the experimental set up is relatively simple and because it allows a controlled, stepwise increase in power, analogous to the stepwise increasing cycle protocol. Body dimensions at 35 weeks gestation are different from those post partum, but it remains speculative if this affects the physiological responses to swimming. It has been suggested that the hydrostatic pressure could reduce tidal volume in pregnant women because the enlarging uterus could be forced towards the diaphragm and limit its contractility.[74] This seems unlikely, because immersion has been shown not to affect Vt, or even to tend to increase it.[13,43,47] We observed no significant change (+8%) in peak Vt during swimming in pregnancy compared with post partum.

The resting changes observed in our study were in accordance with those reported in the literature. During pregnancy resting values of HR, $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ were higher than post partum. This reflects the increased cardiac output, the metabolic needs of the fetus, and the high circulating levels of progesterone.[29,55,63,64,80] Standing values are higher than sitting values, due to circulatory changes and to the effort to maintain an upright position. These positional changes are more prominent in pregnant than in postpartum women.[55,61,88] The hydrostatic pressure during immersion increases stroke volume through an increase in venous return to the heart,[82] which reduces HR despite an increase in metabolism to compensate for heat loss.[73] We chose a water temperature of 33°C to avoid marked heat loss during immersion, which is more prominent in pregnant than in postpartum women[73] and which would have significantly increased metabolic heat production and $\dot{V}O_2$ at rest[70] before exercise. Given the rapidly progressive protocol, it seems unlikely that an increased body temperature would have negatively affected swim peak $\dot{V}O_2$ in our experiments.

Our results with regard to perceived maximal cycling are similar to those previously reported by our group in a comparable group of women.[61] During pregnancy there is no difference in peak $\dot{V}O_2$, compared with postpartum controls, but a slight tendency to lower peak values of HR and $\dot{V}CO_2$, and higher peak levels of $\dot{V}E$. To the best of our knowledge only one study has been reported on maximal swimming responses in pregnant compared to postpartum women.[74] The authors found that swim

peak $\dot{V}O_2$ was 17% lower during pregnancy than post partum, and that peak $\dot{V}O_2$ was lower during the swim than during the cycle trials, by 24% in pregnancy, and by 7% (nonsignificant) post partum. In contrast to their progressive continuous cycle protocol, they used an interval protocol to assess swimming responses. Resistance was increased on the basis of the volunteer's rating of perceived exertion and heart rate, but was otherwise unspecified and the exercise time to maximal effort was not reported. Thus one can not exclude the possibility that differences in power may have contributed to their observation of a lower peak $\dot{V}O_2$ during swimming, but not during cycling, in pregnancy. We studied tethered swimming responses with the use of a progressive continuous protocol, designed to achieve optimal comparison with the cycle protocol. Indeed, the average exercise time to perceived maximal exertion was not different between the swim and cycle trials, and neither between the pregnant and postpartum volunteers.

We found 9% lower values of peak $\dot{V}O_2$ values during swimming than during cycling. Although this might suggest that the volunteers simply pushed themselves less far during swimming than during cycling, this seems unlikely for several reasons. First, a $\dot{V}O_2$ plateau was reached equally often in the swim and in the cycle trials (73%). Furthermore, the observed difference was similar to that reported in untrained nonpregnant subjects. It probably reflects the more extensive use in swimming of arm relative to leg muscles.[6,42,66] Untrained swimmers may reach maximal voluntary exertion at a lower $\dot{V}O_{2max}$ during swimming than during cycling. The smaller overall energy expenditure[30,65] is illustrated by the fact the $\dot{V}CO_2$-$\dot{V}O_2$ plots of swim and cycle test data show identical patterns, except the peak is lower during the swim than during the cycle trial (Fig. 5.1). As a consequence, also the venous lactic acid concentrations were lower after the swim than after the cycle tests. More important, however, than the fact that swim peak $\dot{V}O_2$ is lower than cycle peak peak $\dot{V}O_2$, is the finding that swim peak $\dot{V}O_2$ appears to be independent of pregnancy.

Relative to peak $\dot{V}O_2$, peak $\dot{V}CO_2$ was lower during swimming than during cycling and, consequently, peak values of R were significantly lower during swimming. Because $\dot{V}CO_2$ is known to increase more steeply than does $\dot{V}O_2$ above the ventilatory threshold,[12, ch 3] this was to be expected when overall energy expenditure during the swim test was lower than during the cycle test.

Peak $\dot{V}E$ is lower during swimming than during cycling, both in absolute terms and relative to $\dot{V}CO_2$. The relative hypoventilation during swimming is attributed to more difficult mechanics as a result of hydrostatic pressure on the thorax.[10,45] However, despite the relative hypoventilation, the arterial O_2 pressure and saturation are unaffected in nonpregnant individuals.[10,45] This is probably true also for pregnant women.

CHAPTER 6

GENERAL CONCLUSIONS

In this chapter general conclusions are presented on the basis of the studies reported in the thesis against the background of the objectives formulated in Chapter 1.

Moderately strenuous exercise in healthy pregnant women at term does not cause changes in fetal heart rate pattern suggestive of fetal distress. As reported in Chapter 2, we did not observe a single case of fetal bradycardia during exercise or recovery in 26 term pregnant women. The changes in fetal movements that women may experience do not seem to reflect a consistent change in fetal behavioral pattern but rather a subjective impression that may be caused by distraction or by increased awareness of fetal quiescence as a normal state change. Our data confirm the subjective impression of exercising pregnant women that exercise increases uterine contractility. However, the results of this study should not be extrapolated to pregnant women who perform exercise of a different type or at a more strenuous level than that applied in the present study, and not to preterm women in whom the uterus may respond differently to various stimuli.

Ventilation is increased during pregnancy due to high circulating levels of progesterone, and results in a lower arterial PCO_2. In Chapter 3 we posed the question if ventilatory threshold changes in pregnancy. The traditional methods for assessment of the ventilatory threshold rely on visual inspection of graphical plots of ventilatory equivalents and on the determination of end-tidal gas concentrations. The large inter- and intra-observer variation led us to search for a mathematical method, which could be applied to the data from a longitudinal study of $\dot{V}E$, $\dot{V}O_2$ and $\dot{V}CO_2$ during rapidly

progressive cycle tests in pregnancy and postpartum. Through modification of the V-slope method we estimated the anaerobic threshold (AT) and respiratory compensation (RC) point for each test by nonlineair regression analysis in a three-dimensional space. The AT and RC points were found at exercise intensities of about 50% and 80% peak $\dot{V}O_2$, respectively, with no significant differences between the test periods. $\dot{V}E$ was significantly higher during pregnancy than during post partum at rest and throughout incremental exercise. A lower peak $\dot{V}CO_2$ relative to peak $\dot{V}O_2$ during pregnancy compared with post partum was reflected in a more shallow slope of $\dot{V}CO_2$ vs $\dot{V}O_2$ above the AT point. This suggests that during pregnancy the buffering of lactic acid is reduced, which may be explained by the increased utilization of lactate by the liver or by the fetoplacental unit.

Pregnant women perform rapidly progressive maximal nonweightbearing exercise equally well as in the nonpregnant state. This led us to test the hypothesis that women in late pregnancy and nonpregnant women are equally capable of performing nonweightbearing endurance exercise. The hypothesis is supported by the results of the study reported in Chapter 4. In that longitudinal study we found that time and power of endurance cycling exercise in pregnancy and post partum were similar. Also heart rate and respiratory responses were unaffected by gestation. Based on these results we conclude that pregnancy does not affect the capability or the physiologic mechanisms of women to perform strenuous endurance cycling. Because body weight increases during gestation it is obvious that weightbearing tasks, like running, will be performed less well during pregnancy. Strenuous prolonged exercise in late pregnancy appears to be not harmful to the healthy fetus, FHR patterns after maximal and endurance testing did not indicate fetal distress. Generally, the metabolic responses to endurance exercise were found to be largely independent of gestation, except for lactic acid and glucose concentration, which were significantly lower during pregnancy than postpartum.

Relatively little is known about the adaptations of pregnant women to exercise in water, especially compared to nonpregnant controls. In the study reported in Chapter 5 we compared peak ventilatory responses during cycling and swimming in pregnant women and post partum. We found that perceived maximal exertion is reached at a lower percent maximal $\dot{V}O_2$ in swimming than in cycling, and that the reduced energy expenditure is reflected in lower peak values of $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}E$. However, late

pregnancy appeared not to affect peak $\dot{V}O_2$ in cycling or swimming. Also, in healthy pregnant women strenuous swimming in thermoneutral water does not seem to harm the fetus. These conclusions may not apply when the swimming mother and her fetus are subjected to cold stress. The fetus is well protected, even during open water diving, and there is some evidence suggesting that scuba diving to a depth of 30 m is not associated with embryopathy. Further study is needed to provide insight in to questions as how the pregnant woman and her fetus adapt physiologically to a cold water environment, to strenuous endurance swimming, and to breath-holding and scuba diving.

SUMMARY

The studies presented in this thesis are concerned with various aspects of the physiology of the exercising pregnant woman and her fetus.

Chapter one presents the considerations that led to the objectives of the thesis. The gradual evolution from experimental studies on exercise physiology in the pregnant laboratory animal to clinical investigations in pregnant women is briefly reviewed. The fetal heart rate response is acknowledged as a reliable indicator of fetal wellbeing. Because practical and technical problems interfere with the recording of fetal heart rate and uterine activity in the exercising pregnant woman, little is known about the effects of maternal exercise on fetal condition and behavior, and on uterine activity.

Earlier studies indicated that peak $\dot{V}O_2$ remains unaffected during rapidly progressive cycle tests in pregnancy, but it is not clear if $\dot{V}O_2$ at submaximal levels of exercise, especially at the anaerobic threshold, remains equally unaffected. The anaerobic threshold determines the upper limit of the work rate that can be sustained for a long period of time. Above the anaerobic threshold blood lactate concentrations increase exponentially, and metabolic acidosis is associated with changes in gas exchange. A difference in anaerobic threshold during pregnancy compared to that in the nonpregnant state would imply a difference in endurance capacity of exercise above the anaerobic threshold.

Because water supports weight, exercise in water is attractive, in particular for pregnant women. However, knowledge of the physiologic effects of this environment on the exercising pregnant woman is limited and deserves further study.

Chapter two reports the physiologic responses of human fetal heart rate and uterine contractility to moderately strenuous maternal exercise. In 30 term women admitted for elective induction of labor fetal heart rate and intrauterine pressure were continuously measured with the use of internal electronic monitoring before, during, and after 20 minutes of maternal exercise at a heart rate of 140 beats per minute on a cycle ergometer. The fetal heart rate tracings were assessed by three observers and were classified using semiquantative scoring systems for fetal condition (Fischer) and behavior (Nijhuis), and the frequency and intensity of uterine contractions were determined.

Fetal outcome appeared to be good in all cases. There were no significant differences in Fischer scores and Nijhuis states between rest, exercise, and recovery periods. Uterine activity increased significantly during the exercise period, with a 5.5-fold increase in contraction frequency and a 4-fold increase in time-pressure integral compared to rest, with rapid recovery after the exercise. We conclude from these results that moderate exercise in healthy term pregnant women does not cause a change in fetal heart rate or behavioral patterns suggestive of fetal distress, but it does significantly increase uterine activity. We warn against extrapolation of these results to women with an unripe cervix.

Chapter three describes a mathematical model to determine the $\dot{V}CO_2$ - $\dot{V}O_2$ relationship, and to estimate the anaerobic threshold (AT) and the respiratory compensation (RC) point. The calculations are based on the test results of rapidly incremental cycle tests in 33 volunteers at 16, 25, and 35 weeks' gestation, and 8 weeks postpartum. AT and RC for each test were estimated through modification of the V-slope method, a nonlinear regression analysis in a three-dimensional space defined by the minute volume ($\dot{V}E$), O_2-uptake ($\dot{V}O_2$), and CO_2-output ($\dot{V}CO_2$). The AT and RC points were found at exercise intensities of approximately 50 and 80% peak $\dot{V}O_2$, respectively, with no significant differences between test periods. $\dot{V}E$ was significantly higher during pregnancy than postpartum, at rest and throughout incremental exercise. A lower peak $\dot{V}CO_2$ relative to the peak $\dot{V}O_2$ during pregnancy compared to postpartum was reflected by a more shallow slope of $\dot{V}CO_2$ versus $\dot{V}O_2$ above AT. These results suggest that in pregnancy the buffering of lactic acid is reduced, which may be

explained by increased utilization of lacate by the liver and by the fetoplacental unit.

Chapter four presents a prospective longitudinal study designed to determine to what extent endurance exercise responses in pregnancy differ from those post partum. In 16 women at approximately 32 weeks gestation and 10 weeks post partum, heart rate, respiratory responses, and plasma concentrations of substrates and catecholamines were determined at rest, during rapidly progressive maximal cycle testing, and during cycle exercise for about 35-40 minutes at 70%-75% peak $\dot{V}O_2$. Endurance capacity in terms of exercise time and power was 37.6±1.0 minutes and 124±8 W, respectively, and was found to be similar in pregnancy and post partum. Heart rate and respiratory responses near the end of endurance exercise appeared to be unaffected by gestation, with pregnancy values of 173±3 beats per minute (HR), 1.87±0.07 l/minutes ($\dot{V}O_2$), and 1.68±0.07 l/minutes ($\dot{V}CO_2$), except for VE which, at 70.0±3.5 l/minutes, was 14% higher than post partum. Plasma concentrations of free fatty acids (404±62 μmol/l), glucose (3.34±0.17 mmol/l), and lactic acid (4.51±0.50 mmol/l) near the end of endurance exercise were lower in pregnancy than post partum by 9, 24, and 24%, respectively. During endurance exercise in pregnancy plasma catecholamine concentrations - norepinephrine (1.554±0.236 μg/l), epinephrine (0.170±0.032 μg/l), dopamine (0.095±0.017 μg/l) - were not different from those determined post partum. It is concluded that pregnant and nonpregnant women are equally capable of performing strenuous endurance cycle exercise. The physiologic responses to endurance exercise are independent of gestation except for a lower plasma glucose and lactic acid concentration, which may reflect an increased utilization of these substrates during exercise in pregnancy. Fetal heart rates following endurance exercise were not suggestive of fetal distress.

Chapter five first presents a review of published knowledge and understanding of the effects of exercise in water on maternal and fetal adaptations, and it reports a study designed to asses whether or not pregnancy affects peak $\dot{V}O_2$ during rapidly progressive swimming compared to cycling.

In nonpregnant individuals a water environment markedly alters various physiologic responses such as ventilation and circulation. These effects appear to depend

to a large extent on the temperature and on the hydrostatic pressure of the surrounding water. In the single published study on peak $\dot{V}O_2$ that compared swimming in pregnant and postpartum women, swim peak $\dot{V}O_2$ was found to be reduced during gestation, but this was most likely due to methodologic errors.

Little is known about the effects of diving on the human fetus. From the available data it appears unlikely that diving in the first trimester of pregnancy to a depth of 30 m using a selfcontained underwater breathing apparatus (SCUBA) is associated with embryopathy. Further studies are needed to answer questions as to how the pregnant woman and her fetus adapt to a cold water environment, to strenuous endurance swimming, and to diving with or without equipment.

In a study designed to asses the effect of pregnancy on peak $\dot{V}O_2$ during rapidly incremental cycling and swimming, 11 women at 30-34 weeks gestation and 8-12 weeks post partum were studied during rapidly incremental cycling and swimming while HR, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and plasma lactic acid concentrations were measured. The water temperature was kept at 33°C. Peak HR appeared to be not significantly affected by the type of exercise or by pregnancy. Peak $\dot{V}O_2$ was 9% lower during swimming than during cycling but was not affected by pregnancy, with values for pregnancy cycling and swimming of 2.36 ± 0.12 and 2.11 ± 0.11 l/minutes, respectively. Peak $\dot{V}CO_2$ and peak $\dot{V}E$ were found significantly lower by 18-25% during swimming than during cycling, but only peak $\dot{V}CO_2$ during swimming appeared to be affected by pregnancy (-10%). Lactic acid concentrations in plasma were observed to be 12-17% lower after swimming than after cycling, and 17-31% lower during pregnancy than post partum. These results indicate that perceived maximal exertion is reached at a lower percentage peak $\dot{V}O_2$ in swimming than in cycling, and that the reduced energy expenditure is reflected by lower peak values of $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$.

Chapter six presents a general discussion of the studies reported in this thesis. No evidence was found that moderately strenuous exercise in healthy pregnant women causes changes in fetal heart rate patterns suggestive of fetal distress, but it does increase uterine activity in term pregnant women with a ripe cervix. The anaerobic threshold and respiratory compensation points during maximal cycle tests appear to be similar in various stages of pregnancy and post partum, but there is evidence of reduced

lactic acid buffering during pregnancy. The results of the studies indicate that women are able to perform strenuous nonweightbearing exercise equally well in pregnancy as in the nonpregnant state. Also, the metabolic responses to endurance exercise appear to be largely independent of gestation. Peak oxygen uptake during rapidly progressive maximal swimming in water close to thermoneutrality was found to be unaffected by pregnancy. Future research with regard to the exercise physiology of pregnant women should include the effects of strenuous endurance swimming and to various aspects of diving in pregnancy.

SAMENVATTING

De in dit proefschrift beschreven onderzoeken hebben, vanuit verschillende invalshoeken, betrekking op de inspanningsfysiologie van de zwangere vrouw en haar foetus.

Hoofdstuk een bespreekt de overwegingen die hebben geleid tot de in het proefschrift behandelde vraagstellingen. De geleidelijke verschuiving van experimenteel inspanningsonderzoek bij drachtige dieren in het laboratorium naar onderzoek van de fysiologische respons op inspanning bij de zwangere vrouw wordt kort besproken. De foetale hartfrequentie wordt beschouwd als een betrouwbare maat voor het welzijn van de foetus. Wegens praktische en technische problemen van registratie van de foetale hartactie en uterusactiviteit bij de zwangere vrouw tijdens fysieke inspanning zijn er weinig betrouwbare gegevens over de effecten van lichamelijke inspanning op de toestand en het gedrag van de foetus en op de uterusactiviteit.

Uit eerder onderzoek is gebleken dat tijdens de zwangerschap de piek $\dot{V}O_2$ tijdens fietsen niet wordt beïnvloed. Het is de vraag of dit ook geldt voor de $\dot{V}O_2$ op een submaximaal inspanningsniveau, vooral rond het niveau van de anaërobe drempel. De anaërobe drempel geeft het punt aan waarboven een prestatie wordt beperkt door de exponentiële toename van de concentratie van lactaat in bloed, de metabole verzuring en de daarmee gepaard gaande veranderingen in de gasuitwisseling. Als de anaërobe drempel tijdens de zwangerschap tengevolge van een veranderd metabolisme op een ander niveau zou liggen dan buiten de zwangerschap, zou dit consequenties hebben voor de inspanningscapaciteit boven de anaërobe drempel.

Door de opwaartse druk van water is bewegen in water prettig, vooral voor zwangere vrouwen. De kennis omtrent de fysiologische effecten van deze omgeving op de zich lichamelijk inspannende zwangere is beperkt en verdient nader onderzoek.

Hoofdstuk twee beschrijft de fysiologische respons van de foetale hartfrequentie en de uterus contractiliteit tijdens matig zware inspanning van de zwangere gedurende. Bij 30

à terme zwangeren die in aanmerking kwamen voor electieve inleiding van de baring, werden voor, tijdens en na een inspanningsproef van 20 minuten op een fietsergometer bij een maternale hartfrequentie van 140 slagen per minuut de foetale hartfrequentie en de uterusactiviteit door middel van inwendige elektronische registratie continu gemeten. De foetale harfrequentie registraties werden door drie beoordelaars geanalyseerd en met behulp van een semikwantitatief scoringsysteem ingedeeld naar foetale conditie (Fischer) en gedrag (Nijhuis). Tevens werden de frequentie en de intensiteit van de uteruscontracties bepaald.

Alle pasgeborenen waren gezond. De Fischer scores en de Nijhuis stadia vertoonden geen significante verschillen tussen rust, inspanning en herstel. De uterusactiviteit nam tijdens inspanning significant toe. In vergelijking met de activiteit in rust nam de frequentie van de contracties toe met een factor 5.5 tijdens inspanning, de tijd-uterusdrukintegraal met een factor 4. Na inspanning volgde een snel herstel tot de uitgangswaarden. Uit deze resultaten blijkt dat matig zware inspanning bij gezonde à terme zwangeren geen veranderingen veroorzaakt in de foetale hartfrequentie of het foetale gedragspatroon die wijzen op foetale nood. Inspanning veroorzaakt wel een significante toename van de uterusactiviteit. Wij wijzen erop dat het niet juist is deze resultaten te extrapoleren naar vrouwen met een onrijpe cervix

Hoofdstuk drie introduceert een mathematisch model om de verhouding tussen de $\dot{V}CO_2$ en de $\dot{V}O_2$ te bepalen en de anaërobe drempel (AT) en het respiratoire compensatie (RC) punt te berekenen. De berekeningen zijn gebaseerd op de resultaten van een onderzoek bij 33 zwangere vrouwen die bij 16-, 25- en 35 weken zwangerschapsduur en 8 weken post partum een snel oplopende maximaaltest uitvoerden op een fietsergometer. Voor elke test werden de AT en RC bepaald met behulp van een modificatie van de V-slope methode, een nonlineaire regressie analyse in een driedimensionale ruimte bepaald door het ademminuutvolume ($\dot{V}E$), de O_2-opname ($\dot{V}O_2$) en CO_2-afgifte ($\dot{V}CO_2$). De AT en RC punten werden gevonden bij, respectievelijk, 50% en 80% van de piek $\dot{V}O_2$. Er werden geen significante verschillen waargenomen tussen de perioden waarin werd getest. $\dot{V}E$ was significant hoger tijdens de zwangerschap in vergelijking met post partum,, zowel in rust als tijdens toenemende inspanning. De minder steile helling van de $\dot{V}CO_2$ ten opzichte van de $\dot{V}O_2$ boven het AT-punt wordt weerspiegeld in de lagere

piek $\dot{V}CO_2$ in de zwangerschap vergeleken met post partum. Deze resultaten wijzen op een verminderde buffering van lactaat tijdens de zwangerschap, welke wellicht kan worden verklaard door een toegenomen verbruik van lactaat door de lever en de foetoplacentaire eenheid.

Hoofdstuk vier beschrijft een longitudinaal prospectief onderzoek naar de invloed van zwangerschap op het leveren van duurinspanning. Bij 16 vrouwen werden rond de 32^e week van de zwangerschap en de 10^e week post partum metingen verricht van de hartfrequentie, de respiratoire respons en de plasma concentraties van substraten en catecholamines. De metingen vonden plaats op een fietsergometer in rust, tijdens een snel oplopende maximale fietstest en tijdens een duurtest van 35-40 minuten bij 70%-75% piek $\dot{V}O_2$. De duurcapaciteit, uitgedrukt in tijd en vermogen, bleek tijdens de zwangerschap niet verschillend te zijn van die post partum en bedroeg 37.6$\pm$1.0 minuten en 124$\pm$8 W. Ook de hartfrequentie en de respiratoire respons, met waarden van 173$\pm$3 slagen per minuut (HR), 1.87$\pm$0.07 l/minuut ($\dot{V}O_2$), en 1.68$\pm$0.07 l/minuut ($\dot{V}CO_2$) aan het einde van de duurtest, werden niet door de zwangerschap beïnvloed. Een uitzondering vormde de $\dot{V}E$, die met 70.0$\pm$3.5 l/minuut tijdens de zwangerschap 14% hoger lag dan post partum. De plasmaconcentraties van vrije vetzuren (404$\pm$62 μmol/l), glucose (3.34$\pm$0.17 mmol/l), en lactaat (4.51$\pm$0.50 mmol/l), waren aan het einde van de duurtest in de zwangerschap respectievelijk 9, 24, and 24% lager dan post partum. De catecholamineconcentraties in plasma tijdens de zwangerschap - noradrenaline (1.554$\pm$0.236 μg/l), adrenaline (0.170$\pm$0.032 μg/l) en dopamine (0.095$\pm$0.017 μg/l)- verschilden niet van die post partum. Hieruit kan worden geconcludeerd dat de fysiologische respons van zwangere vrouwen tijdens een duurprestatie op de fiets niet verschillend is van die bij niet-zwangere vrouwen. Een uitzondering hierop vormen de lagere waarden van plasmaglucose en lactaat tijdens de zwangerschap vergeleken met de waarden na de bevalling. De verlaagde waarden duiden mogelijk op een verhoogd gebruik van deze substraten tijdens inspanning in de zwangerschap. De foetale hartfrequentie na duurinspanning gaf geen aanwijzingen voor foetale nood.

Hoofdstuk vijf geeft eerst een overzicht van de huidige wetenschappelijke kennis en inzichten op het gebied van de fysiologische aanpassingen van de zwangere vrouw en de foetus in water, in rust en en tijdens inspanning. Vervolgens wordt een onderzoek beschreven naar het effect van zwangerschap op de piek $\dot{V}O_2$ tijdens snel oplopende zwem- en fietsbelasting.

Immersie in water veroorzaakt bij niet-zwangeren veranderingen van de fysiologische respons, onder andere voor wat betreft ademhaling en circulatie. Deze veranderingen zijn in belangrijke mate afhankelijk van de temperatuur en de hydrostatische druk van het omgevende water. In het enige bekende onderzoek waarin de piek $\dot{V}O_2$ tijdens zwemmen in de zwangerschap en post partum werd vergeleken, werd tijdens de zwangerschap een verlaagde piek $\dot{V}O_2$ gevonden, zeer waarschijnlijk ten gevolge van een onjuiste methodologie.

Er is weinig bekend over het effect van duiken op de zwangerschap. Uit de beschikbare gegevens lijkt het onwaarschijnlijk dat duiken met ademhalingsapparatuur (SCUBA) tot op 30 m diepte in het eerste trimester van de zwangerschap schadelijk is voor de ontwikkeling van het embryo. Nader onderzoek is nodig om inzicht te krijgen in de fysiologie van de aanpassing van de zwangere vrouw en haar foetus aan verblijf in koud water, aan duurinspanning in water, en aan duiken met of zonder apparatuur.

In een onderzoek naar het effect van zwangerschap op de piek $\dot{V}O_2$ tijdens snel oplopende zwem- en fietstesten werden bij 11 vrouwen met een zwangerschapsduur van 30-34 weken en daarna tussen 8-12 weken post partum, de HR, de $\dot{V}O_2$, de $\dot{V}CO_2$, $\dot{V}E$, en de plasma lactaat concentratie bepaald. De watertemperatuur was 33°C. De piek HR bleek niet te worden beïnvloed door het inspanningstype of de zwangerschap. De piek $\dot{V}O_2$ was tijdens zwemmen 9% lager dan tijdens fietsen, maar bleek niet te worden beïnvloed door zwangerschap; de waarden voor fietsen en zwemmen in de zwangerschap bedroegen respectievelijk 2.36 ± 0.12 en 2.11 ± 0.11 l/minuut. De piek $\dot{V}CO_2$ and piek VE waren met 18-25% significant lager tijdens zwemmen dan tijdens fietsen, maar alleen de piek $\dot{V}CO_2$ tijdens het zwemmen werd beïnvloed door zwangerschap (-10%). De lactaat concentraties in plasma lagen 12-17% lager na zwemmen in vergelijking tot na fietsen en 17-31% lager tijdens de zwangerschap dan post partum. Uit deze resultaten blijkt dat subjectief maximale inspanning tijdens zwemmen bij een lager percentage piek $\dot{V}O_2$ wordt ervaren dan tijdens fietsen. Dit komt

verder tot uiting in lagere piekwaarden van $\dot{V}O_2$, $\dot{V}CO_2$ en $\dot{V}E$.

Hoofdstuk zes geeft een algemene bespreking van de onderzoeken die worden beschreven in dit proefschrift. Er werden geen aanwijzingen gevonden dat matige inspanning bij gezonde zwangeren veranderingen veroorzaakt in het patroon van de foetale hartfrequentie passend bij foetale nood. Wel verhoogt inspanning aan het einde van de zwangerschap de uterusactiviteit, bij aanwezigheid van een rijpe cervix. In verschillende stadia van de zwangerschap en post partum bleken de anaërobe drempel en het respiratoire compensatie punt tijdens maximale fietstesten niet te veranderen, maar er werden wel aanwijzingen gevonden voor verminderde buffering van lactaat tijdens de zwangerschap. Zwangeren en niet-zwangeren blijken in staat te zijn tot dezelfde duurinspanning zonder gewichtsbelasting. Ook blijkt de metabole respons op duurinspanning grotendeels onafhankelijk te zijn van de zwangerschap. Tijdens snel toenemende inspanning bij zwemmen in water met een temperatuur dicht bij thermoneutraliteit bleek de piek $\dot{V}O_2$ niet door zwangerschap te worden beïnvloed. Toekomstig onderzoek op dit gebied zou zich onder meer moeten richtten op intensieve inspanning van langere duur bij zwemmen en op diverse aspecten van duiken tijdens de zwangerschap.

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APPENDIX

To determine a line with two breakpoints in a three-dimensional space, while assigning approximately equal weight to the variables, we converted the data of all individual tests to a scale of zero to one for VE, $\dot{V}O_2$, and $\dot{V}CO_2$.

Normalization procedure. The mean value at rest was set at zero, the peak value at one. The data of each step of incremental exercise were assigned a value between zero and one: for $\dot{V}O_2$, on the X-axis, as x_i = ($\dot{V}O_2$ incremental - $\dot{V}O_2$ rest) / ($\dot{V}O_2$ peak - $\dot{V}O_2$ rest), and similarly as y_i for $\dot{V}CO_2$, on the Y-axis, and as z_i for VE, on the Z-axis.

Finding the regression line.

If we assume that the AT point is $\begin{pmatrix} x_1 \\ y_1 \\ z_1 \end{pmatrix}$ and the RC point is $\begin{pmatrix} x_2 \\ y_2, \\ z_2 \end{pmatrix}$

we can describe the three segments (rest to AT, (AT to RC), and (RC to peak) as the vector equations:

$$1. \quad \begin{matrix} x \\ y \\ z \end{matrix} = a \begin{pmatrix} x_1 \\ y_1 \\ z_1 \end{pmatrix} \tag{A1}$$

$$2. \quad \begin{matrix} x \\ y \\ z \end{matrix} = \begin{matrix} x_1 \\ y_1 \\ z_1 \end{matrix} + b \begin{pmatrix} x_2 - x_1 \\ y_2 - y_2 \\ z_2 - z_2 \end{pmatrix} \tag{A2}$$

$$3. \quad \begin{matrix} x \\ y \\ z \end{matrix} = \begin{matrix} x_2 \\ y_2 \\ z_2 \end{matrix} + c \begin{pmatrix} 1 - x_2 \\ 1 - y_2 \\ 1 - z_2 \end{pmatrix} \tag{A3}$$

in which x_1, y_1, z_1 and x_2, y_2, z_2 can be determined by minimizing the sum of squared distances of the data points to the regression line, as

$$d\ (Q,l) = \frac{\| r * (q - p) \|}{\| r \|}$$

in which d (Q,l) is the distance of a point Q with a position vector q to line l, and p is the position vector and r is the direction vector of the line segment under consideration.

The following SPSS commands specify the three-segmented model in which join points AT and RC are to be estimated:

```
COMPUTE ZERO = 0
MODEL PROGRAM   Y1=0.4   Z1=0.3   Y2=0.6   Z2=0.5.
IF ( X <= 0.5 )
PRED1 = SQRT((Y1*Z-Z1*Y)**2 + (Z1*X-0.5*Z)**2 +
(0.5*Y-Y1*X)**2) /
      SQRT(0.5**2 + Y1**2 + Z1**2).

IF ( X > 0.5 AND X < 0.75 )
PRED1 = SQRT(((Y2-Y1)*(Z-Z1)-(Z2-Z1)*(Y-Y1))**2 +
          ((Z2-Z1)*(X-0.5)-(0.75-0.5)*(Z-Z1))**2 +
          ((0.75-0.5)*(Y-Y1)-(Y2-Y1)*(X-0.5))**2) /
      SQRT((0.75-0.5)**2 + (Y2-Y1)**2 + (Z2-Z1)**2).

IF ( X >= 0.75 )
PRED1 = SQRT(((1-Y2)*(Z-Z2)-(1-Z2)*(Y-Y2))**2 +
          ((1-Z2)*(X-0.75)-(1-0.75)*(Z-Z2))**2 +
          ((1-0.75)*(Y-Y2)-(1-Y2)*(X-0.75))**2) /
      SQRT((1-0.75)**2 + (1-Y2)**2 + (1-Z2)**2).

NLR ZERO WITH X Y Z / PRED=PRED1 / OUTFILE='ESTIM.SYS'.

MODEL PROGRAM   X1=0.5   Y1   Z1   X2=0.75   Y2   Z2  .

IF ( X <= X1)
PRED = SQRT((Y1*Z-Z1*Y)**2 + (Z1*X-X1*Z)**2 + (X1*Y-Y1*X)**2)/
     SQRT(X1**2 + Y1**2 + Z1**2).

IF ( X > X1 AND X < X2 )
PRED = SQRT(((Y2-Y1)*(Z-Z1)-(Z2-Z1)*(Y-Y1))**2 +
          ((Z2-Z1)*(X-X1)-(X2-X1)*(Z-Z1))**2 +
          ((X2-X1)*(Y-Y1)-(Y2-Y1)*(X-X1))**2) /
     SQRT((X2-X1)**2 + (Y2-Y1)**2 + (Z2-Z1)**2).

IF ( X >= X2)
PRED = SQRT(((1-Y2)*(Z-Z2)-(1-Z2)*(Y-Y2))**2 +
          ((1-Z2)*(X-X2)-(1-X2)*(Z-Z2))**2 +
          ((1-X2)*(Y-Y2)-(1-Y2)*(X-X2))**2) /
     SQRT((1-X2)**2 + (1-Y2)**2 + (1-Z2)**2).

NLR ZERO WITH X Y Z / FILE ='ESTIM.SYS' /  SAVE PRED .
```

The nonlinear regression routine can determine the parameters by minimizing the squared sum of residuals. As a first estimate of the necessary initial values, the routine was run as an intermediate step with $x_1=0.5$ and $x_2=0.75$ as constant values, and $y_1=0.4$, $z_1=0.3$, $y_2=0.6$, and $z_2=0.5$ as initial values. The estimates of x_1 to z_2, thus obtained, were used to run a subsequent routine to calculate the normalized values of the AT and RC points that were then converted back to conventional units and reported in the text.

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