CARDIORESPIRATORY FITNESS DURING PREGNANCY AND ITS RELATIONSHIP TO OUTCOME

by

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ABSTRACT

In order to determine the effects of aerobic fitness on pregnancy and the newborn 20 primigravid subjects were studied throughout their gestational period and immediately post-partum. The subjects were classified as trained (T=10) or untrained (UT=10) based on the heart rate response to submaximal cycle ergometry testing done in each trimester. Case room reports were reviewed after delivery. There was no difference between groups in the length of gestational period (T=40.75;UT=40.75 weeks) nor weight gained versus pre-pregnancy measures (T=13.92; UT=13.30 kgs). The first stage of labour was extended in the UT, 13hrs.58.8min. vs 11hrs.18.0min. UT had a longer second stage , 90.57 vs 70.0 mins. for T. Stage 3 was also prolonged in UT, 15.17 vs 7.43 mins. In both groups analgesia and/or anaesthesia was used equally. Two of the 10 T females had caesarean sections vs 3 of the 10 in the UT group. The mean apgar scores at 1 and 5 minutes were: T=7.70, 9.20; UT=7.90, 9.33, respectively. The birth weights of the T babies were marginally larger than the UT newborns (3733.00 vs 3679.97 gms). The T newborns were 8 males and 2 females, and the UT were 5 males and 5 females. All babies were healthy and without apparent abnormalities. There appears to be no positive or negative effects of maternal fitness on the newborn. The reduction in the active stage of labour in the T group may reflect their improved fitness levels.

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INTRODUCTION

Increased participation, by men and women in physical fitness programs of all varieties has been a characteristic of the 1980's. Exercise has been involved in inducing changes in all physiological systems and is responsible for positive changes in lifestyle modification. With the new emphasis on fitness, exercise programs have been expanded to include the pregnant woman. As exercise is perceived to promote health, then perhaps exercise during pregnancy might assure an ideal gestational period and a healthy fetus.

The lack of well-designed and controlled studies of humans exercising during pregnancy has health care professionals basing their advice to exercising pregnant women on anecdotal studies in humans or animal models. The physiological differences between humans and animals precludes the direct application of animal study results to humans.

The majority of animal studies have used sheep, guinea pigs, and rats as models for observing responses to exercise testing or training during gestation. Exercise testing healthy pregnant sheep on a treadmill has had no ill effects on the ewes or their fetuses (Orr et al., 1972; Curet et al., 1976), but exercising ewes at 'moderate to heavy sustained' levels (Longo et al., 1978), to exhaustion (Clapp, 1980), or with fetuses with impaired umbilical blood flow (Emmanoulides et al., 1972) produced transient decreases in fetal pO_2 responses during the exercise testing sessions. The decrease in uterine blood flow (UBF) in pregnant sheep was related to the level and duration of maternal exercise , but a relatively constant delivery of oxygen was maintained to the uterus (Lotgering et al., 1983a), and the preferrential shunting within the uterus maintained fetal and placental $\dot{V}O2$ at, or slightly above, the pre-exercise values during exercise (Lotgering et al., 1983b). The fetal pO_2 levels were within normal levels because total uterine oxygen consumption was maintained during exercise due to hemoconcentration and increased oxygen extraction. Studies with guinea pigs, rats, and mice have controlled physical activity prior to conception and during gestation. Training studies with pregnant guinea pigs found increasing the time and/or intensity of the exercise increased the abortion rate, shortened the gestation period, decreased the maternal weight gain, increased fetal mortality, decreased fetal weight gain, lowered newborn weight and increased newborn mortality in the trained groups (Gilbert et al., 1980).

Studies on exercising rats before and during pregnancy have found that increased mortality in the exercising groups' offspring may have been due to the testing protocol used, maternal cannibalism,or low birthweight (Wilson and Gisolfi,1980). The effects on the offspring of exercised pregnant rats involved cardiovascular and lipid metabolism changes (Parikova 1978, 1979), while others (Mottola et al.,

1983) found no developmental changes with mild aerobic exercise.

The human studies have focused on the physiological responses to exercise during pregnancy or the effect of exercise during pregnancy on the outcome of pregnancy. One of the first studies reported reduced uterine blood flow while cycling a bed cycle ergometer (Morris et al., 1956). The reduction in UBF may have been due to the testing protocol in that the supine position allowed the enlarged uterus to press on the inferior vena cava . No correlation was found between Physical Fitness Scores (PFS) and uteroplacental insufficiency determined from fetal heart rate(FHR) in women tested on a bicycle ergometer late in gestation (Pommerance et al., 1974a).

Early work using Physical Work Capacity(PWC) as an indication of physical fitness during pregnancy (Dahlstrom and Ihrman, 1960) found a constant value through gestation which decreased sharply a few days after delivery. A series of studies using PWC, (Erkkola 1975, 1976a, 1976b) found that this variable increased 10% with pregnancy and a further 17.6% with exercise training, and returned to pre-pregnancy values 2 weeks prior to delivery. Expressing the pregnant PWC values as a percentage of the non-pregnant PWC value, Erkkola (1976b) found that those women with greater than normal PWC values had significantly shorter spontaneous labours, heavier newborns and placentas .

Pommerance and co-workers(1974b) studied pregnant women who were classified as "fit" using their pre-pregnancy weights, cycle ergometer test responses, and compared them to the levels of physical fitness based on non-pregnant women using Astrand's nomogram for calculation of $\dot{V}O_2$ max from submaximal measurements (Astrand and Rodahl,1970). They found that the "fit" multiparas had shorter labour - stages were not specified.

Recent studies have found that aerobic fitness can be maintained through regular aerobic training during pregnancy (Sibley et al., 1981, Collings et al., 1983). Healthy primiparas and multiparas were aerobically trained for 10 to 12 weeks during gestation. All the pregnancies were problem-free and the newborns were healthy. Two case studies have been reported on exercising during pregnancy but data from these works are only applicable to the subjects analyzed. One study reported no problems with running during pregnancy and lactation where $\dot{V}O_2$ max increased 20% during pregnancy indicating an improvement in endurance performance (Dressendorfer,1979). The other work suggested that it was necessary to decrease running and its intensity during pregnancy due to the metabolic stress it caused (Hutchinson et al.,1981) . In the later study the increase in $\dot{V}O_2$ was proportional to weight gain and aerobic capacity was concluded to be constant throughout gestation.

There have been retrospective studies reported in the literature on exercise throughout pregnancy involving jogging, scuba diving, and a combination of cross-country skiing, aerobic dance and running. With all retrospective studies the accuracy of subject's recall of physical activity patterns introduces considerable error. Women reported decreasing their jogging as pregnancy progressed due to mechanical factors associated with gestation -uterine enlargement, changes in weight distribution, and the suggested decrease in circulatory reserve (Jarrett and Spellacy,1983). They had a lower abortion rate and incidence of maternal and fetal complications than normal which may have been due to their healthy condition, but fetal abnormalities were higher than expected (6% versus the norm of 2-4%). In women who dove during pregnancy, the frequency and depth of dives decreased as pregnancy progressed, and the incidence of the pre-selected birth complications was higher in women who dove (5.5% vs 0% ; Bolton,1980). Women who participated in a combination of endurance activities during gestation at or near pre-pregnancy levels gained less weight , delivered earlier, and had lighter newborns than those who stopped before the 28th week (Clapp and Dickstein,1984). Reasons for stopping activity were extreme fatigue, lower abdominal discomfort, musculoskeletal injury, fear of affecting the pregnancy, and lack of time .

Given the problems inherent to retrospective studies, this investigation was designed to determine the effects of exercise throughout gestation on the course of pregnancy and its outcome in healthy primiparas.

METHODS

Subjects: Twenty-six healthy caucasian, primigravid women (ages 25 - 35) with no medical contraindications to exercise volunteered to be studied prospectively through gestation. They were recruited through family practioners, prenatal education classes, pre and postnatal fitness classes, and the media: radio, newspaper advertisement and posters. The subjects were made aware of the testing protocol and any potential risks; informed consent was obtained from the subjects, and their physicians were advised of their participation in the study. Six of the subjects did not complete the study : 3 developed medical contraindications to exercise, 2 left the study due to lack of time and interest, and 1 changed geographic location. This was a descriptive study only; no attempt was made to modify their activity pattern. The subjects were asked to keep a log of their physical activity during gestation. This included the type of exercise, frequency, duration, and intensity of activity.

Fitness Assessment: The subjects entered the study at 10-14 weeks or 22-24 weeks gestation. They completed a questionnaire on their pre-pregnancy physical activity patterns, the consent form, and had their fitness assessed via heart rate response to a submaximal test on a Monark cycle ergometer. During the initial testing session (10-14 weeks or 22-24 weeks) and the subsequent sessions (22-24 weeks and 34-36 weeks), blood pressure , weight, and pre-test heart rates were recorded. The cycle ergometry test involved determining a steady heart rate response while cycling 50 rpm at each of the four designated workloads for 5 to 6 minutes : 25, 50, 75, 100 watts.The maternal heart rates were recorded during the cycling and the post-test period by direct ECG. The testing ended when the subject's heart rate reached 150 beats per minute (bpm) or the fourth workload was completed. Fetal heart rates were not assessed.

The subjects were classified as "fit" (n=10) if their heart rate was 130 bpm or less at 1.5 kpm in the second trimester session (22-24 weeks), or "unfit" (n=10) if their heart rate was greater than 130 bpm. The difference between the two groups in the heart rate response to the cycle ergometery was determined in the second and third trimesters using a student's t test with the confidence limit set at 0.05. With such a small sample size further statistical treatment was not deemed appropriate and the results treated as descriptive data only.

Postpartum data: The data was obtained from 6 hospitals - 5 in the Lower Mainland and 1 in Victoria . The patients' records were examined specifically for: length of labour, use of medication, surgical procedures, placental weight, newborn weight, apgar scores at 1 and 5 minutes, and sex of the newborn.

RESULTS

The age in each group was similiar (T=29.50years, UT=28.70years).The lengths of gestation (T=40.75 weeks,UT=40.75 weeks),and weight gained versus pre-pregnancy measures (T=13.92 kg., UT=13.30 kg) in the two groups were equivalent. The first stage of labour decreased in the T group (T=11 hrs 18.0 min., UT= 13 hrs 58.8 min.). The second stage of labour in the trained group was less than 80% of the untrained group (T=70.0min., UT=90.57min.). A difference was also seen in the third stage of labour between the two groups (T=7.43 min., UT=15.17 min.). Medication was administered to 8 of 10 trained and untrained subjects. There were 2 Caesarean sections in the trained group and 3 Caesarean sections in the untrained group. The maternal data with the means and ranges of the results are presented in TABLE 1.

The mean heart rates during the ergometry tests in the second and third trimesters indicated a significant difference between the two groups. In the second and third trimesters the mean heart rates were T=121.78 $\pm$ 9.4, UT=147.11 $\pm$ 5.3 bpm ($p < 0.05$); T= 133.22 $\pm$ 12.0, UT=147.33 $\pm$ 5.9 bpm ($p < 0.05$), respectively,therefore different levels of fitness existed between the subjects in each category throughout gestation. The T group increased its mean heart rate from the second trimester to the third trimester, T=121.78 $\pm$ 9.4 and 133.22 $\pm$ 12.02 bpm ($p<0.05$), whereas the UT group's mean heart rates did not change, UT= 147.11 $\pm$ 5.3 and 147.33 $\pm$

5.9 bpm. Based on these heart rate responses, using Astrand's nomogram (de Vries, 1968), the subjects were classified as being high, good, average, fair, or low based on their predicted maximum oxygen uptake (VO_2 max). The T group had more above average VO_2 max ratings, at 75 watts, in the second and third trimesters (T=5 High, 2 Good, UT=1 Good, 4 Average, 4 Fair; T=1 High, 4 Good, 3 Average, UT=8 Average, 1 Fair, respectively). The activity logbooks (T=9, UT=8) indicated similiar participation in aerobic activity , 3 > hours per weeks, in the second trimester (T=6;UT=6), but the T subjects were more active in the third trimester (T=5;UT=2). (See appendices C and D).

The newborns from the trained mothers were slightly heavier (T=3733.00 gms., UT=3679.97 gms), but their placental weights were lower (T=647.40 gm;UT=812.86 gm). The apgar scores at 1 minute and 5 minutes were similiar (T=7.70,UT=7.90;T=9.20,UT=9.33, respectively).There were more males in the trained newborns whereas the sexes were evenly distributed in the untrained newborns (T:M=8,F=2;UT:M=5,F=5). The newborn data is summarized in TABLE 2.

TABLE 1

Mean Maternal Data (range of values)

	T (n=10)	UT (n=10)
Age (years)	29.50 (25 - 33)	28.76 (25 - 31)
Gestational Period		
Length (wks)	40.75 (40.0 - 41.0)	40.75 (38.0 - 42.0)
Weight gain (kgs)	13.92 (8.64 - 20.90)	13.30 (11.82 - 16.36)
Labour		
Stage 1:	11hrs 18.0 min. (3hrs- 15hrs20min.n=9)	13hrs 58.8 min. (4hrs - 48hrs)
Stage 2:	70.0 min. (11min.- 110min.n=8)	90.57 min. (45min.- 150min.n=7)
Stage 3:	7.43 min. (4min.- 16min.n=7)	15.17 min. (3min.- 40 min.,n=10)
Placenta Weight		
(gm)	647.40 (482 - 820,n=5)	812.86 (700 - 1050,n=7)
Medication Administered	8/10	8/10
Caesarean Sections	2 1 Kjellands rotation and extraction	3

TABLE 2

Mean Newborn Data (range of values)

		T (n=10)	UT (n=10)
Apgar			
1 minute		7.70 (5 - 9)	7.90 (5 - 9, n=9)
5 minute		9.20 (8 - 10)	9.33 (9 - 10, n=9)
Weight (gms)		3733.00 (3200 - 4460)	3679.97 (2980 - 5320)
Sex	M	8	5
	F	2	5

DISCUSSION

The literature on the effects of exercise during pregnancy has reported changes in the length of gestation and maternal weight gain. The similiar lengths of gestation in these two groups confirms previous findings (Collings et al., 1983; Dibblee and Graham, 1983; Erkkola, 1976; Pommerance et al., 1974), but conflicts with those of Clapp and Dickstein (1984) who observed that women who exercised to term had shorter lengths of gestations. The maternal weight gain reported here was similiar in the two groups which differs with the greater body fat and weight in the unfit group in Dibblee and Graham's (1983) study, and lower maternal weight gain in women who exercised to term as reported by Clapp and Dickstein (1984).

It was possible to differentiate levels of cardiovascular fitness based on the heart rate response to submaximal exercise during gestation. The twenty primigravid subjects in this study were grouped, similiar to Dibblee and Graham(1983), who classified fitness levels in primigravid subjects by a step-test and Erkkola (1976) who used Physical Work Capacity (PWC) values. The majority of human training studies (Sibley et al.,1981; Collings et al., 1983; Erkkola 1976, 1976a) placed the subjects into an experimental (training) or a control (sedentary) group at the beginning of their investigation. This study investigated the effect of different fitness classifications on pregnancy and its outcome, whereas, the other studies analyzed the effect of their training programs on fitness levels, pregnancy, and outcome. Dibblee and Graham (1983) classified the subjects in the first trimester, but no differences between the T and UT groups existed by the third trimester. In this investigation we classified the subjects in the second trimester and found the groups to be distinct throughout the third trimester.

Exercising a cycle ergometer has been used as a means of studying pregnant subjects by those interested in specific physiological responses - respiratory (Pernoll et al., 1975; Edwards et al., 1981), cardiovascular (Ueland et al., 1969; Guzman and Caplan, 1970), hormonal (Rauramo et al.,1982), and for establishing (Pommerance et al., 1974) and/or monitoring (Collings et al., 1983; Erkkola 1976, 1976a) aerobic fitness levels during gestation. It is a safe procedure for determining aerobic fitness with no known adverse effects experienced by the subjects. Cycling the cycle ergometer ,a non-weight bearing activity, was not affected by the subjects' increasing weight, shifts in centre of gravity, and/or changes in body configuration during gestation -which would affect other testing protocols such as the step-test (Dibblee and Graham ,1983), walking (Sibley et al., 1981), or running on a treadmill (Dressendorfer 1978; Hutchinson et al., 1981). All these testing protocols classify the subjects based on one aspect of fitness - cardiovascular or aerobic fitness, and do not attempt to measure the subjects' anaerobic capacity, strength, or flexibility which combined with the aerobic component comprise "physical fitness". The subject's fitness levels in this study does not give any objective measurement of the condition of the muscle or ligamentous structures and, therefore does not allow speculation as to the effect of strong or tight pelvic floor muscles and their role in the delivery process.

The first stage of labour indicated differences in the two groups which disagrees with the findings reported in the literature (Sibley et al., 1981; Dibblee and Graham, 1983; Collings et al., 1983). This stage of labour is involuntary and would not be affected by the subject's aerobic fitness level. A major finding of this study is the reduction in the second stage of labour in the T group. This disagrees with other studies (Collings et al.,1983; Dibblee and Graham, 1983; Pommerance et al.,1974) where no difference in the length of labour had been observed in primigravid subjects. Zaharieva (1972) and Erdelyi (1960) however, found the second stage of labour to be shorter in elite athletes compared to non-athletes. This was disputed by Berg and co-workers (1984) who reported that high-performance (endurance and power-trained) athletes' lengths of labour (not separated into stages) were longer than those reported in a national perinatal study. The active stage of labour may have been reduced by the T subjects' higher level of aerobic fitness and, therefore, the subjects' ability to aid the birth process. Fatigue plays a major role in the process of labour and it has been demonstrated that increased aerobic fitness can postpone the onset of fatigue (Brooks and Fahey, 1984). Thus a fit woman is more capable of dealing with the physical ordeal of labour and this is reflected in the reduction of the second stage of labour by 20 percent. The third stage of labour was also quicker in the T group, but this variable is affected more by the medical management of this stage.

Two other variables of interest, medication administered and caesarean sections, could not be controlled or affected by the research design; the differences between the two groups were not apparent. Thus, in this population, fitness does not influence the need for analgesia or affect the incidence of surgical procedures required during delivery. This relationship between these variables and fitness supports the findings of Collings and co-workers (1983) and Dibblee and Graham (1983), but conflicts with Berg and co-workers(1984) who found a higher number of abdominal deliveries in athletes.

The placental weight has been reported in two studies (Erkkola 1976: Collings et al., 1983) where larger placentas were found in the T groups. The UT group's larger placental weights reported in this investigation is similar to animal study findings (Nelson et al., 1983) where increased maternal exercise decreased placenta weight. The sample size of the placentas weighed in the T group was smaller(n=5) compared to the UT group (n=7) . The relationship between decreased placenta weight and fetal weight, as reported by Nelson and co-workers (1983), was not observed in this study since there was no difference in the newborns' weights. Clapp and Dickstein (1984) found the T group newborns to be lighter which may have been related to the reported shorter gestation periods. Erkkola (1976) reported T offspring to be heavier due to the higher plasma volume, blood volume, and total hemaglobin in the T subjects.The one minute apgar scores of the T group and the UT group were equivalent, which conflicits with Dibblee and Graham (1983) and Collings and co-workers (1983) whose fit or exercised newborns' had higher 1 minute apgar scores. There were no differences in the 5 minute apgar scores which was a possible reflection on the healthy, normal pregnancies, and the absence of any long-term effects of maternal exercise on the fetus. There have been no reports in the literature of significant differences in 5 minute apgar scores.

The small sample size reported here limits the conclusions which can be made about exercise during pregnancy. The study was descriptive, no attempt was made to modify the subjects' physical activity patterns. This investigation was carried out at 6 hospitals with a different physician and caseroom nurse for each subject, therefore, a lack in consistency of recording apgar scores, and delivery procedures is apparent. The subjects were classified on the basis of cardiorespiratory fitness and no attempt was made to evaluate the other physiological components of fitness.

Exercise during pregnancy did not affect length of gestation,maternal weight gain, newborn weight, or the first stage of labour. It did decrease the second stage of labour in trained subjects. The 1 minute and 5 minute apgar scores were similiar indicating no long-term effects of exercise during pregnancy. In conclusion, participation in a physical fitness program resulted in no deterimental effects on the mother or fetus. In addition the second stage of labour was reduced in the fit mothers which may be related to their increased cardiorespiratory fitness and ability to postpone fatigue during the active stage of labour.

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APPENDIX A - REVIEW OF LITERATURE

Introduction

In the past exercise during pregnancy was restricted to stretch and relaxation exercises which were in preparation for labour and delivery. The recent interest in fitness presents a dilemma for physically active women of childbearing age: should she exercise during pregnancy? Popular literature cites women who were active throughout gestation with no or positive effects on pregnancy and its outcome. Reasons cited for encouraging regular exercise during pregnancy include controlling weight gain, decreased backache, constipation, and/or varicose veins, and increased energy levels for coping with daily life and the stresses of pregnancy (Jopke, 1983). However, there are few prospective clinical trials on the effects of exercise on pregnancy women and their fetuses. Animals studies have demonstrated some negative effects of exercise during pregnancy and the practicicng physician has little concrete data on which to base a decision when consulted by a woman who questions the merit of continuing an exercise program throughout her gestational period. This paper reviews the studies in the area in both animal and human populations in an effort to gain an overall picture of the current studies of research in the field.

Animal Studies

Animal studies provide models for the effects of exercise during pregnancy. The literature on exercising pregnant animals during gestation has offerred a variety of conclusions due to the experimental animals used, stages during gestation examined, and experimental protocols. The majority of animal studies have shown that in normal, healthy animals, exercise testing to exhaustion is well-tolerated (Orr et al., 1972; Curet et al., 1976; Mottola et al,, 1983) and that the effects on the fetus from acute exercise bouts is transient with no detrimental long-term effects. Some studies have found that training to exhaustion will cause shorter duration of gestation, lower birthweights, and increased fetal mortality (Gilbert et al., 1980; Wilson and Gisolfi, 1980) while others have found positive long-term effects of prenatal exercise (Parizikova, 1978 and 1979). Exercise before mating may affect pregnancy - weight gain, litter size - depending when training occurred in the females' life cycle. The general conclusion from the animal literature was that chronic maternal exercise, in moderation, had no long-term effects on the fetus, gestation length, or the mother.

Uterine Blood Flow

Fetal responses followed maternal responses during the pregnant ewe's exercise in work by Emmanoulides et al. (1972). At the end of maternal exercise, there was significant increase in fetal pH and decrease in fetal pCO_2 associated with maternal hyperventilation and alkalosis. Chronically-stressed fetuses had higher pCO_2 and lower pO_2 values, before and after maternal exercise, and an elevated heart rate after exercise for a longer period compared to normal fetuses. There was gross evidence of fetal malnutrition seen in the chronically-distressed fetuses (where one umbilical artery was tied) of exercising ewes and of non-exercising ewes. These fetuses were at an additional disadvantage during maternal exercise due to the reduced oxygen available. The redistribution of regional blood flow associated with the decreased circulatory reserve of pregnant animals, and the effects of the moderate maternal and fetal hemoglobin were causes for decreased oxygen. The study assumed that the fetal oxygen consumption and the pO_2 gradient (umbilical artery - umbilical vein) did not change during maternal exercise. The authors concluded that moderately severe maternal exercise may induce varying decreases of fetal pO_2 which were transient and well-tolerated by the fetuses with intact umbilical circulation where there was a rapid return of uterine blood flow (UBF) to pre-exercise levels during recovery.

Orr et al. (1972) found UBF in non-pregnant and pregnant sheep was not impaired by maternal exercise to exhaustion, if the animal was healthy. The increased heat produced during exercise was removed by increased carotid blood flow to the vascular plexus in the turbinate bones. In sheep, the vascular plexus is a heat exchanger. The iliac blood flow was increased to meet the increased oxygen requirements of the skeletal muscles. The significant increase in UBF in non-pregnant sheep was because the blood flow at rest was small and the increase in UBF during exercise was a greater percentage of the total UBF than in pregnant sheep. In pregnant sheep, the UBF would be near maximum values at rest and the increase with exercise would be a smaller percentage of total UBF when compared to the increase in exercising non-pregnant sheep. Orr disagreed with Emmanoulides' (1972) conclusions that a drop in fetal blood pO_2 indicated tissue hypoxia. Orr concluded that the absence of fetal acidosis indicated a lack of fetal hypoxia. Maternal exercise in Orr's work did not cause any hazardous effects on the fetus because UBF was not impaired.

Later, it was confirmed by Curet et al. (1976) that maternal exercise did not affect uterine blood flow, and there was no difference in response to exercise testing in trained and untrained pregnant ewes. Curet questionned whether a three week training period on the treadmill was sufficient to show a difference in stroke volume and heart rate during the exercise testing. Sheep responded to the exercise test by increasing cardiac output by increasing heart rate, which differs from humans who increase cardiac output by increasing stroke volume and heart rate.

Pygmy goats increase cardiac output like humans, by increasing their stroke volume. Dhinsda et al. (1978) found that the resting heart rate was significantly higher in late pregnancy and increased more with exercise compared to postpartum, as expected. The peripheral vascular resistance at rest was lower during pregnancy and was significantly lower with exercise, but not enough to account for the increased cardiac output with exercise. It was proposed that the increase in cardiac output resulted from the increased arterial blood pressure during exercise and its greater increase in pregnancy. The resting a-vO_2 difference and the amount it increased with exercise were the same during late pregnancy and postpartum. Pygmy goats were suggested as models for human blood flow distribution, tissue oxygen supply, and fetal oxgenation studies during maternal exercise.

In the last trimester of pregnancy, the nonplacental portions of the pygmy goat uterus suffered a major reduction in blood flow during exercise (Hohimer et al., 1982). The vasconstriction may have been due to exercise (walking on an inclined treadmill at 1.5 to 2.0 mph) or to concommitant hypocapnia or hyperthermia.

Longo and associates (1978) concluded that "moderate to heavy sustained" maternal exercise in pregnant sheep could result in significant fetal hypoxia and possibly cause intrauterine growth retardation. During maternal exercise, fetal descending aortic pO_2 fell and UBF dereased associated with a decrease in maternal arterial pCO_2 similiar to the earlier findings (Emmanoulides et al., 1972). There was a significant decrease in the weight of fetuses from exercising ewes compared to the controls. The exercising ewes had a lower placental diffusing capacity for carbon monoxide during rest, than the controls, and during exercise the diffusing capacity increased.

The rates of uterine and umbilical blood flow and fetal pO_2 decreased in pregnant sheep exercised to exhaustion (Clapp , 1980). During exercise there was a downward shift in the oxygen dissociation curve allowing normal oxygen uptake by uteroplacental tissue and the fetus. The exercise test was associated with significant increases in maternal temperature and respiratory alkalosis which led to a decrease in UBF. The increase in heart rate was related to the decrease in UBF. The pre-exercise condition of the ewes might have effected the UBF because the well-trained ewes had unchanged uterine blood flow values during short term exercise on the treadmill.

Recently, Lotgering and co-workers(1983a) found a decrease in pregnant ewe's UBF during exercise and a very fast return to normal values during recovery, similiar to findings with humans (Morris et al., 1956). The ewes were accustomed to the exercise stress by walking on the treadmill at various speeds for 10 minutes, for a week. Curet and associates (1976) had questionned whether three weeks were ample training time to find differences in sheeps' responses to exercise, but Lotgering felt that his acclimatization period was adequate and prevented excessive catecholamine release before or during the test. The sudden UBF changes during exercise suggested that the initial decrease in UBF was not due to hyperthermia or alkalosis (both occur in exercise and are associated with a decrease in UBF), but a neural or hormonal mechanism due to exercise. The further decrease in UBF was attributed to hyperthermia and alkalosis. It was concluded that the decrease in UBF maintained a relatively constant oxygen delivery to the uterus which was inversely related to the level and duration of maternal exercise.

In summary, the maternal exercise either had no effect on UBF (Orr et al., 1972; Curet et al., 1976;) or it decreased UBF (Emmanoulides et al., 1972) with a quick return to resting UBF levels during post-exercise recovery similiar to the effect in humans (Morris et al., 1956). The initial UBF decrease was proposed as a neural or hormonal mechanism due to exercise with the further decrease in UBF resulting from maternal hyperthermia and respiratory alkalosis (Lotgering et al., 1983a). The concern for the decrease in UBF was chronic fetal tissue hypoxia which could lead to intrauterine growth retardation (Longo et al., 1978). Oxygen availability to the fetus was assumed to be relatively constant during maternal exercise with the downward shift in the oxygen dissociation curve when uterine and umbilical blood flow was reduced (Clapp, 1980).

Exercising the pregnant animals to exhaustion did not affect the offsprings' development which could be expected since the majority of studies involved exercise testing, not training, during gestation. One study did find that offspring from exercised animals, at moderate to severe levels, had significantly lighter offspring compared to the controls, but the difference was not attributed just to reduced UBF. The majority of studies found that the offspring were normal at birth.

Effect on Offspring

Terada (1974) studied four groups of mice to see the effects of training, before and during gestation on fetal development. Half of the mice trained (T) before mating, and ran (TR) or were sedentary (TC) from the 9th to the 16th day of gestation, and the other half either ran after mating (CE), from the 9th to the 16th day, or were a control (CC). Training before mating was advantageous because the TR group had a lower fetal mortality rate compared to the CE group due to an unknown mechanism. There were no mortality differences between TC and CC fetuses. Terada proposed that the lower body weights of the trained mice at the start of gestation was due to a decreased caloric intake in their growing phase, when training occurred. Exercising during mid-pregnancy (9th to 16th day) was attributed to interferring with the body weight gains in the TR and CE mice. Fetal weights in the CE and TR litters were lower. The higher mortality and lower fetal weights in the CE group was caused by the decrease in water and food consumption, and the uterine environmental modifications (eg:decreased UBF).

Comparing maternal exercise to chronic hypoxia in guinea pigs Gilbert et al., (1979), found moderate exercise during gestation did not produce any changes in fetal body, organ or placental weights, whereas chronic hypoxia decreased fetal body and brain weights, and increased the ratio of brain, heart , and placenta weights to body weights when compared to control ratios. Previous work (Terada, 1974) found a decrease in fetal weight with maternal exercise, but the differences in the two studies were probably due to a higher level of exercise in the previous study. The first indication of stress produced by maternal exercise was a decrease in placental diffusing capacity, found with higher workloads and the changes in fetal body and organ weights found in other studies (Terada, 1974; Longo et al., 1978; Nelson et al., 1983). Based on weight, the authors felt the exercise level was well-tolerated by the exercising animals, but 15 of the 20 exercising animals aborted or delivered early (indicating the exercise level was severe). Exercising during pregnancy did not induce chronic hypoxia because the weights - fetal body, organ, placenta - of the exercising animals did not change as they did in the animals exposed to chronic hypoxia.

A subsequent study with guinea pigs, (Gilbert et al., 1980) used different levels of maternal exercise to determine the effects on placental and fetal weights, and diffusing capacity. Placental and kidney weights decreased as the exercise intensity increased and the ratio of brain and heart weight to body weight increased at higher levels of exercise. The brain and heart weights had no relationship to the exercise levels which may be due to "relative sparing" of these organs. The diffusing capacity decreased with more intense exercise levels supporting Gilbert's (1978) earlier hypothesis. The fetus, it was concluded, was compromised by a smaller placenta, therefore with less diffusing capacity per kilogram of fetal tissue which was supported later by Nelson et al., 1983.

Chandler and Bell (1981) suggested fetal oxygenation was compromised by the effects of reduced UBF and maternal alkalosis on placental transfer during exercise in pregnant ewes. Mild exercise caused the development of mild hypocapnea with no effects on fetal gases. Moderate exercise did not affect the uterine oxygen uptake significantly because an increase in the a-vO_2 difference across the uterus was about equal to the decrease in blood flow. The significant changes in maternal hemoblobin concentration, fetal and maternal blood gases were greater with moderate exercise than mild exercise. There were increases in maternal blood glucose and lactate levels, and fetal glucose and lactate concentrations after mild maternal exercise. Reasons for fetal hyperglycemia were 1) increased umbilical glucose uptake, 2) stimulation of glycogenolysis in fetal liver, and 3) reduced fetal glucose utilization due to reduced blood flow. The extraction of glucose increased significantly with exercise as the UBF decreased, but effects on placental oxygen transfer were balanced by the increase in maternal hemoconcentration.

Maternal respiratory alkalosis during moderate exercise could reduce placental transfer due to 1) a significant shift in the maternal oxyhemaglobin dissociation curve, therefore increasing the oxygen affinity of maternal arterial blood which is greater than fetal blood, or 2) fetal hypocapnia and alkalemia from maternal respiratory alkalosis could decrease umbilical blood flow.

In conclusion, the authors felt that short-term realistic levels of exercise caused significant disturbances in respiration and carbohydrate metabolism in fetal sheep, therefore, exercise close to term could affect fetal well-being and "birth vigour" more than gross effects on fetal growth. The lambs in the study were born within five (5) days of term and in the normal weight range.

Maternal exercise produced detrimental effects on fetal growth and development in guinea pigs (Nelson et al., 1983). Lower levels of exercise (15 or 30 minutes/day) decreased placental weight whereas diffusing capacity and higher levels of exercise (45 or 60 minutes/day) decreased fetal weights. Nelson 's findings agreed with Gilbert's earlier statement (1979) that increased workloads could be stressful, affecting diffusing capacity, and fetal weights. The diffusing capacity had a different response to exercise due to 1) a selection process whereby the fetuses with low placental diffusing capacities aborted before term, or 2) the appearance of some adaptive mechanisms not present at the lower levels. The mechanisms proposed for the changes in placental diffusing capacity during exercise were an alteration in placental morphology with a decrease in exchange surface, based on other work in their lab (Smith et al., 1983) and a decrease in fetal blood volume or decrease in maternal and fetal placental blood volume.

Parizkova (1978) used rats to study the effect of prenatal and postnatal exercise on cardiac microstructure. The pregnant rats either exercised (E) or did not exercise (C) during gestation, and their male offspring either exercised (EE,CE) or did not exercise (EC,CC). The highest density of cardiac muscle fiber was in the EE group and the lowest density was in the CC group. Parizkova suggested that with regular optimal workloads during pregnancy (E) the positive effects of exercise could be transferred to the fetus, and the effects of exercise on cardiac muscle would increase if the offspring (EE) continued regular optimal execise. The CE group increased their cardiac microstructure with postnatal exercise compared to the CC offspring, but the increased density was not the same as the EE group. There were two suggested reasons for the changes in the fetal development. First, the increase in placental blood flow during maternal exercise and change in distribution of blood to the placenta modified the fetal heart and its future development. Second, the changes were due to increased daily workloads which increased glycolysis, lipolysis, release of catecholamines, and blood levels of pyruvic acid, lactic acid, and free fatty acids, thereby increasing the fetus' exposure to these elements and modifying its development. Parizkova (1979) repeated the study with male and female offspring and found no differences in cardiac microstructure development between the sexes.

The effect of maternal exercise on selected factors of lipid metabolism - lipid and fatty acid concentration, serum cholesterol levels of free fatty acids (FFA), cholesterol and lipid synthesis was investigated by Parizkova and Petrasek (1978) in four groups of male and female offspring (35-108 days old) who exercised (E) or were sedentary (C). At 35 days, the E offspring had higher serum cholesterol levels of FFA's and cholesterol. The E females had a higher lipid and fatty acid concentration in their liver, whereas the E males had a lower or no change in lipid concentration compared to the other groups. Liposynthesis was lower in E females at 35 and 90 days, and it was varied in E males. The E males had a higher concentration of cholesterol, synthesis of fatty acids, and lower cholesterogenesis in the small intestine at 100 days. Pariskova and Petrasek proposed that lipid metabolism changed in the E offsprings' livers because repeated maternal aerobic exercise increased maternal FFA blood levels, therefore, leading to increased lipid metabolites in fetal blood which may have caused a higher concentration in the offsprings' livers. The increased serum levels of FFA and other lipid metabolites from the mother may have caused the increase lipid metabolism in the fetus during development regardless of physical activity postnatally. It was suggested that the females'increased liver lipid concentration may be evidence of a possible dimorphism of lipid metabolism due to the action of female sex hormones. The fat deposits in females have a variety of purposes - pregnancy and lactation- therefore, their lipid metabolism has an increased sensitivity to stimuli compared to the males, even during prenatal life. Exercise was the suggested stimuli that caused the increased lipid and fatty acid concentration .

Wilson and Gisolfi (1980) studied rats who exercised before and/or during pregnancy, and their offspring. rats who continued training through pregnancy (TT) had a higher oxidative enzyme (soleus cytochrome oxidase) activity than the other 3 groups. The rats who started training during pregnancy (NT-T) had a higher oxidative enzyme capacity than the group that stopped training during pregnancy (T-NT) and the control (C), whereas others (Corbett et al.,1979) found no differences in oxidative, glycolytic or contractile properties of skeletal muscle between offspring of rats trained during gestation and the control. The VO_2 max of the control groups was lower at the end of gestation than the TT group, but no difference in VO_2 max was found between the NT-T and C groups. There was no difference between pregnancy and non-pregnant rats when oxygen consumption was adjusted for weight gained during gestation, which agreed with human studies on energy needs of pregnant and non-pregnant women based on fat-free weight (Seitchik, 1972). The offspring of the T-T rats had the highest mortality in the first 28 days. The possible causes for increased mortality in the T-T rats were 1) unknown effects of shock avoidance techniques used to force rats to exercise, 2) maternal cannibalism which was observed but ,not controlled, and/or 3) low birth weight. The T-T rats gained less weight, but had the same litter size as the other groups. Wilson and Gisolfi found no significant differences in the weight of male offspring between 45 and 65 days, therefore their growth was not affected by maternal exercise.

Parizkova (1978) found male offspring of execising mothers to be lighter than the male offspring of non-exercising mothers.

There was no difference in capillary or cardiac muscle densities in male offspring in Wilson and Gisolfi's study (1980) whereas Parizkova's study (1978) found a greater capillary and muscle density in offspring of exercising mothers. Wilson and Gisolfi (1980) suggested the difference between the two studies was that they controlled the genetic influences, and the techniques used to preserve the tissue specimens.

Exercising during pregnancy had no effect on mean fetal weight or development of the diaphragm muscle in the rats studied (Mottola et al., 1983). Female rats were acclimatized to running on a treadmill before mating. After mating, the rats continued to run at a lower rate (PR) or were sedentary (PC). The training before mating did not alter the rats' normal development, based on weight gain, when compared to control rats, who were not acclimatized or mated. The PC rats had larger litter sizes which may have attributed to their greater weight gain during gestation. The difference in litter sizes between PC and PR rats was statistically insignificant.

The diaphragm muscle was examined because of its role in fetal breathing movements (FBM). As an indication of the fetus' condition FBM's were considered more sensitive than FHR, similiar to human fetal conditions (Marsal, 1979). It was proposed that changes in the diaphragm muscle would only occur under severe conditions with possible preferrential treatment similiar to the heart, brain, and liver. Mottola suggested examining a more sensitive muscle for signs of nutritional (chemical) deprivation during maternal exercise. Another reason offered for the lack of fetal changes with matermal exercise was the small sample size (n=5) which may have missed slight changes which would have been evident in a larger sample size. In conclusion, Mottola et al. (1983) found that mild aerobic exercise durint pregnancy did not cause any developmental changes.

Exposing pregnant sheep to air at increased atmospheric pressure during peak development of their fetuses (12 to 40 days) was a simulation of the effect of diving during gestation, Bolton-Klug (1983). Sheep were selected as a model for humans because their responses to hyperbaric exposure, and the size and number of offspring were comparable. The series of marginally-tolerated dives by the pregnant sheep did not affect the fetuses health. Bolton-Klug explained the lack of effects from the hyperbaric exposure as physiological alterations that were either reversed during development, or the effects occured infrequently to show in the small sample size (n=14), and/or were not found in gross anatomical examinations.
In Bolton-Klug's earlier work (Bolton, 1980) she advised diving to shallower depths or no diving at all about the time of conception or in the 1st trimester.

Lotgering et al.(1983b) examined temperature, uterine oxygen consumption, and blood gases in fetal sheep. The difference between fetal and maternal temperature, at rest, was 0.5 C and changed during maternal exercise. The higher fetal temperatures were related to the higher rate of metabolism and requirement to dissipate heat to the mother. There was a relatively slow response in fetal temperature during rapid temperature changes in the mother, due to the heat capacity of the amniotic fluid and the fetus, without changes in heat transfer efficiency across the placenta. There was no major increase in fetal metabolic rate. The decreased UBF during exercise demonstrated the occurrence of preferrential shunting within the uterus as placental VO_2 was maintained at or slightly above the control values. The oxygen requirements of the fetus and placenta in prolonged exercise were not met to the same extent as at rest. It was not possible to assess accurately to what extent fetal-placental oxygen requirements should have increased during exercise. The fetal oxygentation levels were within normal levels during maternal exercise because total uterine oxygen consumption was maintained during exercise due to hemoconcentration and increased oxygen extraction. Lotgering concluded that maternal exercise was not a major stressful or hypoxic event for the fetus.

The animal literature has had animals forced to exercise for specified periods of time during gestation. One study documented the effects of voluntary exercise of mice during gestation and lactation, on lactation (Karasawa et al., 1981). The daily activity of the mice - treadwheel rotations - was tallied during their growing phase. The mice were divided into the exercising group (cages with treadwheels) or the sedentary group after mating. Voluntary exercise gradually decreased as gesatation progressed and it decreased markedly before delivery. The activity level did not increase during lactation because suckling the young limited the females' free movement. Exercise during pregnancy did not affect lactation. It was not possible to study the effect of exercise during lactation on lactation because the females' activity levels were too low.

Jenkins and Ciconne (1980) used three groups of rats - control, forced and voluntary exercise - to study the effect of exercise during gestation on the offspring's brain nucleic acids. Rats who exercised voluntarily , on a treadwheel, did more total work than rats who were forced to exercise, on a treadmill. The exercise dose failed to alter brain nucleic acids, therefore the dose was not intense engough to produce a change or certain physiologic adaptations occured to protect brain nucleic acids of the offspring. The poorer motor performance of the treadmill run dams' offspring, on the Rotacone, indicated that maternal exercise did have some effects, possibly indicating selective involvement of cerebellum or vestibular system. The dams who exercised voluntarily had a lower weight gain possibly indicating their greater total work compared to those who were forced to exercise, therefore they could reduce their weight significantly without obvious effects on their offsprings' brain nucleic acids and/or motor performance.

In conclusion, the animal studies found that acute exercise bouts to exhaustion during prenancy of normal healthy animals with normal healthy fetuses did not have any long-term effects. Chronic exercise during pregnancy produced varied effects on the fetus , gestation and its outcome. There was some concern whether fetuses suffered from hypoxia in pregnant ewes exercised to exhaustion , but all offspring were normal at birth.

Rat offspring studies found that exercise during pregnancy may affect cardiac microstructure, capillary density, and lipid metabolism. One study found that exercise at a high intensity before and during pregnancy led to a higher offspring mortality which may have been due to experimental design or the effects of maternal exercise.

The animal studies provide a model from which to study humans exercising during pregnancy. Drawing direct conclusions from animal literature to humans must be done with caution since these studies had animals forced to exercise, and the duration of exercise with animals does not equate to the same time period in a human. In addition, differences in physiology between animals and humans could result in different effects on a human pregnancy.

The major difference with human studies is that the pregnant subject would exercise voluntarily, thereby eliminating the possible consequences of forced exercise and accompanying stress.

Human Studies

Human studies, most of which are anecdotal or too small for statistical analysis, have not shown the same results as many of the animal studies - shorter gestation, lower infant birthweight.

Physiological Changes during Pregnancy

The physiological changes with pregnancy are similiar to changes during submaximal aerobic exercise in an endurance trained individual- increased cardiac output, increased blood volume and red blood cell volume and decrease in peripheral resistance. The changes which would occur during training and pregnancy are usually noted in the literature in anecdotal or poorly contolled studies.

In pregnancy, major physiological changes involve the respiratory, cardiovascular systems, and the uterus to accommodate the growing fetus.

Respiratory Changes

Anatomical changes during pregnancy effect the respiratory system. The increasing size of the uterus presses up against the diaphragm, thereby decreasing the depth of the thorax, but this decrease is counterbalanced by the broadening of the thoracic cage. The respiratory changes in pregnancy include an increase in tidal volume(30%), inspiratory capacity (5%), and decrease in expiratory capacity (15%), residual volume(20%), leading to a decrease in funtional residual capacity (FRC),(18%). The higher levels of circulating progesterone, a known respiratory stimulant, are thought to stimulate the respiratory centres and lead to the increase in minute ventilation. The physiological shunt and dead space do not change usually during pregnancy. The hyperventilation of pregnancy decreases $PaCO_2$ (29-31mmHg) which increases pH to the 7.43 - 7.46 range leading to compensatory loss or sodium bicarbonate (21 mEq/liter).

At term, the oxygen uptake has significantly increased (32%), the pulmonary compliance increased, and resistance decreased. The closing volume (CV) - the lung volume at which the airways begin to close - does not change, but with the decrease in FRC, CV maybe greater than the FRC, therefore affecting tidal volume. Alveolar collapse may occur during tidal breathing increasing the possibility of hypoxemia, and could account for the high rate of dyspnea reported in pregnant women.

Cardiovascular Changes

The most dramatic changes in pregnancy are in blood volume (BV), cardiac output (Q), and the uterus. Blood volume increases (30-50%) from the end of the first trimester until the 30th week, plateaus for a short period before decreasing to pre-pregnancy values 2 weeks postpartum. The large increase in blood volume is to meet the metabolic needs of the fetus and to compensate for blood loss at delivery. The red blood cell volume increases during gestation, but not to the same extent as plasma, therefore a "dilutional anemia" results. The hematocrit (Hct) and hemaglobin concentration ([Hb]) are lowered in pregnancy (to values of 33-38% and 11-

12mg/100ml, respectively).

The cardiac output increases (30-50%) and like blood volume, peaks between 28-32 weeks, and declines to pre-pregnancy values the last few weeks of gestation. The decrease in Q at term depends on the position of the women when measurements occurred since smaller decreases are found in the lateral position versus supine or sitting. The cardiac output decreases as the size of the uterus increases and presses on the inferior vena cava. The increase in venous pressure in the lower extremities due to the pressure of the uterus on the inferior vena cava reaches a maximum at term and falls to pre-pregnancy values at delivery.

The decrease in total peripheral resistance is probably secondary to steroid hormones, especially estrogen acting on blood vessels (Gibbs, 1981).

Uterine blood flow increases ten-fold (10X) from the pre-pregnancy state to term. In the non-pregnant state the uterus is 30 to 60 gm, but becomes vessel-rich with pregnancy with 80% of the uterine blood flow to the placenta and 20% to the uterus muscle. UBF is not routinely measured in humans because of the invasive techniques involved, therefore, it is necessary to refer to older studies (Morris et al., 1956) or animals studies for data. Uterine blood vessels are thought to be maximally dilated at term with the blood flow being pressure dependent, therefore a lack of autoregulation (Gibbs, 1981).

Effects of Exercise and Pregnancy

Studies of exercise during pregnancy have reported responses of pregnant women to exercise stress testing at one instance or serially through gestation. The studies provide the expected responses of a healthy pregnant woman to a bicycle ergometer or treadmill submaximal test. A few studies have reported maximal responses to exercise testing. The variables of interest can be classified in to six main categories - respiratory responses, cardiovascular responses, thermal responses, work capacity, physical training effect on the outcome of pregnancy, and effects on the fetus of maternal exercise.

Respiratory

Pernoll et al. (1975a) studied pregnant women throughout pregnancy and postpartum to compare oxygen consumption during submaximal bicycle testing. They found that the oxygen consumption (VO_2), at rest and during exercise, increased gradually as the pregnancy progressed with VO_2 reaching peak values new term. At the peak, the VO_2 at rest was 33% above non-pregnant values, and during exercise it was significantly above non-pregnant values. The average VO_2 during the post-exercise periods increased throughout the pregnancy. Pernoll assumed the increased oxygen consumption was due to the cost of the exercise. Minimal work was involved in the movement and carrying of the extra weight gained during pregnancy since cycling is a non-weight bearing activity. Increased oxygen cost would also be attributed to 1) work of the muscles in hyperventilation during pregnancy and 2) increase in myocardial VO_2 due to increased Q. The increase in VO_2 during late pregnancy was greater than the estimated amount for respiratory and myocardial work. The authors could not explain why the effficiency of mild muscular work declined during pregnancy.

In a subsequent study on ventilation rates at rest and exercise, Pernoll and associates (1975b) found a significant increase in expiratory minute ventilation (VE), at rest and exercise, which was due to a significantly greater tidal volume (TV). The carbon dioxide production increased significantly at rest and with exercise during the second trimester (22-26 weeks). The respiratory quotient did not change significantly with exericse. The end tidal volume carbon dioxide concentration was lower at rest and exercise during pregnancy than postpartum, signifying relative alveolar hyperventilation both at rest and exercise during pregnancy.

Edwards et al.(1981) focused their work on rates of changes of VE, VO_2, VCO_2, before and after steady-state exercise, late in pregnancy (38 weeks) and postpartum (3 months). Pregnant subjects significantly higher VO_2, VE, and VCO_2 at rest and a significantly greater absolute increase in VE from rest to exercise (steady state) than postpartum subjects. In the first 90 seconds of the 6 minute bicycle test, VCO_2, and VE increased more rapidly in pregnancy than postpartum, but recovery rates for both conditions (pregnancy and postpartum) were the same. After sitting on the bicycle for 6 minute during the pre-exercise rest period, Edwards suggested that the sudden contraction of the lower extremity muscles of the legs would cause an increase of pressure on the blood vessels and venous return to the heart. The similiar recovery rates in pregnancy and postpartum maybe due to the slow refilling of the lower extremity veins. The increases in VE during pregnancy were greater, therefore, the same workload and efficiency would elicit a ventilatory response greater than in postpartum. The VE pattern during exercise was similiar to VCO_2 and VO_2 suggesting an unusual regulatory method where the blood flow carrying deoxyhemaglobin and CO_2 to the lungs regulated VE. Therefore, the accelerated ventilatory increment with exercise during pregnancy was due to increased CO_2 flow.

Cardiovascular

Some of the circulatory changes in pregnancy were the same as those from physical training- increased red blood cell volume,total blood volume, cardiac output, and fall in peripheral resistance (Ihrman, 1960;Gibbs, 1981).

Ihrman (1960) studied circulatory changes with physical training in pregnancy. Physical training was 35 minutes of exercise, where the heart rate was 140 bpm post-exercise twice a week for 10 weeks. Ihrman found no difference in pulse frequency between the trained and untrained pregnant women on the bicycle ergometer. There was a slight increase in cardiac output in the exercised group, but the other circulatory adjustments were not affected which led to the conclusion that pregnancy was characterized by a circulatory adjustment not influenced by heavy exercise, between the 20th and 30th week. The term "heavy" to desctibe the exercise intensity maybe questionable since it was not specified how long the heart rate was elevated. The program consisted of three bouts of very high intense activity for several minutes - 3 times per 35 minute session - perhaps the work was more anaerobic than aerobic. The schedule would not increase aerobic fitness in a non-pregnant woman unless the sessions were more frequent than 2 time per week (as in Sibley et al., 1981), longer duration, and/or increased intensity.

Mild exercise 100 kpm per minute) on the bicycle ergometer produced cardiovascular responses that were constant throughout pregnancy. During moderate exercise (200kpm per minute), there was a progressive decline in the cardiovascualr response (reserve) due to the peripheral pooling of blood and obstruction of venous return, as the uterus pressed against the inferior vena cava (Ueland , 1969). Cardiac output peaked 20-24 weeks and was maintained until the 32nd week when Q began to drop to non-pregnant levels between 38 - 40 weeks. The maternal heart rate increased slightly at the beginning of gestation and reached maximum values at 28 - 32 weeks. The early increase in cardiac output confirmed the accepted opinion that hemodynamic changes were not due to the metabolic and nutrition needs of the fetus, but perhaps estrogen could induce these changes proposed in an earlier work by Ueland and Parer (1966).

Guzman and Caplan (1970) followed cardiorespiratory responses to exercise in pregnant women from the first trimester to three months postpartum; they proposed that the physiological responses to exercise were the same in both states. The small increase in VO_2, at 29 weeks, was accounted for by the uterus and fetus, which led the authors to suggest that pregnant women had no decrease in muscular efficiency or no significant increase in metabolic demands during mild and moderate exercise on the bicycle ergometer. Adequate myocardial reserve was suggested from the increase in exercise cardiac output per unit increase of oxygen uptake, since the increase in Q was the same in pregnancy and postpartum. The higher demands on the pregnant woman's heart at the given workloads resulted in pregnant women reaching their maximum heart rates at lower workloads than postpartum. The cardiac output increase until the 20th week and was maintained until delivery, which differs from Ueland's findings on cardiac output which dropped to normal levels near term. Guzman and Caplan (1970) suggested the changes in cardiac output were due to ovarian and placental hormones, as did Ueland and Parer(1969), and not due to blood volume which reached peak values betwen 30 - 36 weeks. It was concluded that the hyperkinetic state of pregnancy in the first trimester did not change with the increasing uterus and fetus, but stayed in a stable state until delivery.

Pijpers et al.(1984) found an increase in maternal heart rate , and systolic and diastolic blood pressure during cycling a bed-type cycle ergometer , at 25 watts for 5 minutes, late in gestation (34-36 weeks) , an increase in heart rate would be expected when exercising compared to resting values.

Artal et al.(1981) found light exercise during pregnancy increased maternal heart rate, decreased the R time interval (a continuous cardiovascular technique, proposed by Koh et al.,1979), increased concentrations of glucagon, norepinephrine, and epinephrine, but these results were expected because blood redistribution is catecholamine-mediated. All values returned to baseline values 30 minutes after exercise stopped.
The norepinephrine which increased with exercise also stimulated the uterus with the possibility of triggering labour in women at risk of premature delivery. Artal had four subjects who experienced mild irregular uterine activity during the exercise testing, but the activity ceased when the

norepinephrine levels were decreased during the post-exercise period.

In a study of stress hormones and placental steriods in physical exercise during pregnancy (Rauramo et al., 1982) found increases of norepinephrine and epinephrine during submaximal workloads, greater than those reported previously by Artal et al., 1981.

The increase in plasma catecholamine levels were significantly correlated with the increased pulse rate, supporting the role of the catecholamines in blood re-distribution and hemodynamics. The catecholamine response did not change with pregnancy versus non-pregnancy. Serum levels of prolactin increased significantly 30 minutes after the exercise test and the levels were still elevated at 60 minutes post-test. The prolactin levels in the subjects were higher than non-pregnant prolactin levels.

The mean serum oesteriol concentrations were elevated significantly 30 minutes post-test, but were at baseline by 60 minutes post-test. Rauramo assummed that exercise did not change the rate of placental secretion of oesteriol but the increased serum levels were due to increased flow of uteroplacental blood into maternal circulation soon after exercise ceased. Morris et al.(1956) demonstrated uterine blood flows decreased during exercise, but blood flow was compensated when exercise stopped. The ablility of the fetus to cope with varying oxygen availability depended on efficient development of the placenta.

Pommerance et al. (1974a) found no correlation between Physical Fitness Scores (PFS) and uteroplacental insufficiency from FHR recordings. Subjects were tested, on a bicycle at 35-37 weeks gestation and maximum oxygen uptake were predicted from Astrans's protocol, giving the PFS. Five fetuses had "positive" tests between pre and psot-exercise FHR recording and four of these infants had problems in labour and delivery. Seven additional fetuses had indications of fetal distress and six of the seven had compromised unbilical circulation.

Temperature

Recently, Jones et al., (1985) followed the changes in maternal body temperature and heat storage (heat content/kg) in four women during aerobic exercise throughout gestation. They found that heat storage did not increase during exercise, and concluded that the subjects' thermal balance was maintained with advancing pregnancy due to their individual exercise prescriptions.

Work Tests

Early work by Dahlstrom and Ihrman (1960), on fitness assessment in pregnancy, utilized physical work capacity (PWC). PWC was a constant value throughout pregnancy, decreasing sharply a few days after delivery. A positive correlation between the PWC and the pregnant women's age was established and two possible reasons were 1) physically stronger women conceive more children, therefore they were over -represented in an older group of pregnant women, and 2) older women grew up during the second world war and had different activity patterns than younger women.

The PWC values were similiar in pregnant women with and without toxemia, therefore the cardiorespiratory system had adapted to the work test in hypotensive patients according to Soiva et al.(1969). There was no correlation between age and PWC, as in Dahlstrom and Ihrman (1960) or between birthweight and PWC. Soiva found a positive correlation between maternal weight and PWC which was contradicted by later works, who found PWC and weight were negatively correlated (Pommerance, et al.,1974b). The normal pregnant women had PWC values similiar to the control (non-pregnant women), but the toxemic women had the greatest PWC.
The expected ranking of PWCs, from lowest to highest , was toxemic, normal pregnancy, non-pregnant women of the same age. The controls were not matched for weight-based on the pregnant women's pre-pregnancy weights.Severe physical activity was a suggested contraindicator late in pregnancy especially for women with toxemia because of the great increase in systolic blood pressure. Soiva felt the sudden increases in blood pressure in toxemic patients could lead to

premature separation of the placenta. The myometrial flow was taken as adequate during resting because the FHR returned to normal levels 5 minutes post-exercise.

Erkkola (1976b) found PWC increased 10% spontaneously with pregnancy, and a further 17.6% with physical training, whereas Dalhstrom and Ihrman (1960) found PWC was constant in pregnancy. The training program was longer (26 vs 10 weeks), higher frequency 3 vs 2 times per week), and greater duration (60 minutes plus 10 minutes daily at home vs 35 minutes per session) in Erkkola's study compared to Dahlstrom and Irhman, therefore a greater change in physical fitness was possible. The PWC levels returned to pre-pregnancy levels about 2 weeks prior to delivery. Erkkola's work demonstrated that it was possible to improve PWC without harming the pregnancy . It concluded that training during pregnancy had little influence on heart rate and blood pressure, there was an insignificant decrease in the trained group.
The pregnant women's PWC's were expressed as a percentage of non-pregnant PWC's. Erkkola found that healthy pregnant women (n=51) whose PWC value was greater than normal had significantly shorter spontaneous labours, more newborns over 3500 grams, significantly heavier placentas, and fewer low values of relative placental weight. These high PWC pregnant women had higher plasma volume, higher blood volume, and total hemaglobin which led to their newborns greater weight. The increased plasma volume was associated with the larger placenta, improved circulation, and gas exchange which

benefited the fetus.

The duration of the pregnancy was not viewed an an important factor by Erkkola et al., (1976a). They found the duration of pregnancy was not related to the PWC, but due to the design of the experiment - testing at the 38th week eliminated premature deliveries, women who developmed medical problems were eliminated - and the high number of pregnancies (27%) which were terminated at 40 weeks, electively.

In a retrospective study, women who threatened premature labour had lower PWC's (Erkkola, 1976) . Increased bedrest was prescribed for women with threatened premature labour and this factor probably affected the PWC values which rapidly decreased with inactivity.

PWC and relative heart volume (RHV) were positively correlated by Erkkola and Makela (1976), confirming Erkkola's other work (1976b)on improving physical fitness during pregnancy. RHV was used as an indicator of improved physical fitness based on the relationship of heart volume increasing with physical training in non-pregnant subjects (Astrand and Rodahl, 1970). The authors cited one study (Klepzig and Frisch, 1965) that positively correlated RHV with physical fitness. The RHV values of the trained women were greater than the the control group I, who were tested throughout gestation, but simliar to the control group II , who were tested at 38 weeks. Control group II was slightly more fit than the control group I. The PWCs were significantly

greater in the trained group as was expected. No correlation was evident between RHV or PWC and length of gestation or birth weight.
Erkkola's work found PWC had no influence on duration of pregnancy, induced labour, time the baby's head was visible until delivery, apgar scores at 1 minute, development of toxemia or threatened premature labour.

Pommerance et al.(1974b) used Astrand's tables to calculate maximum aerobic capacity. The values obtained from a submaximal bicycle test were used to compare physical fitness levels of pregnant women at the same stage of pregnancy. The standard table was not accurate in predicting pregnant subjects maximum oxygen uptake because they were based on non-pregnant subjects. Pommerance assumed that the physical fitness scores (PFS) from the tests would represent an equal interval scale for use of parametric statistical tests of significance. The PFSs were inversely related with length of labour in multiparas. The length of pregnancy, and the apgar scores at 1 minute were not related to PFS which agreed with previous findings with PWC and pregnancy by Erkkola. Pommerance found no significant correlation of PFS with labour in primiparas, birth weight, newborn length or head circumference. The authors did not view the duration of gestation as an important outcome of pregnancy, similiar to the opinion later expressed by Erkkola (1976a). The testing of PFS between 35 and 37 eliminated premature deliveries and the subjects were eliminated whose pregnancies delivered after 42 weeks plus a day, or developed medical problems.

A highly significant negative correlation between Astrand's index and pre-pregnancy weight (Erkkola , 1975) confirmed Pommerance's work. The Astrand test supposedly avoids weight influence, but it was expected that an obese mother would be less fit than a normal weight mother. Erkkola presumed pregnancy had training effect on maternal circulation which conflicted with Ihrman's (1960) work for reasons mentioned previously. Erkkola used Borg's Perceived Exertion Rating scale (PER) to determine the subject's voluntary maximal test on a bicycle. A submaximal bicycle test is limited to a maternal heart rate of 150 bpm, therefore making it a less reliable test of physical condition than PER, according to Erkkola. When primigravidae women were tested 2 weeks before delivery their mean physical condition was simliar to non-pregnant women of the same age. Later studies by Erkkola (1976b) found that PWC increased 10% with pregnancy, therefore a PWC at 38 weeks would not reflect the non-pregnant PWC, but the PWC due to pregnancy. The predicted maximal VO_2 two days before and 10 days after delivery were nearly identical (Dressendorfer, 1978), but 4 months after delivery the values were higher. Dressendorfer's case study found that the maximal O_2 uptake and endurance performance could be improved during a normal pregnancy and lactation by physical training without harmful effects to the mother or newborn. A linear relationship between VO_2 max and running speed in non-pregnant states was established except 2 weeks postpartum when it appeared that the subject had lost her racing pace sense and had run too slow. Most women are not encouraged to exercise two to four weeks postpartum, the subject probably had not run since delivery and could not be expected to be in the same state of fitness as before delivery.

Dressendorfer's case study approach led to three major findings about running and pregnancy. First, the subject's pregnancies and milk production were not affected by training and its high caloric costs and fluid losses. Second, the treadmill tests, which elevated the subject's heart rate to 90% of her non-pregnant maximum heart rate, did not produce any serious effects on the pregnancy . Third, the decrease in the estimated maximum oxygen uptake during the first trimester of the second pregnancy, when mileage decreased, suggested a detraining effect. It would be unreasonable to assume that running has no contraindiations in pregnancy. Since Dressendorfer's report was a case study the conclusions can only be applied to the subject examined.

Hutchinson et al. (1981) used the case study method to examine the metabolic, respiratory, and circulatory responses to running during pregnancy. The metabolic stress, indicated by the percentage maximum VO_2 to perform the work test (a ten minute submaximal treadmill test), increased during pregnancy. The authors suggested that running speed should be decreased during pregnancy due to the increase stress seen from the substantial increases in oxygen uptake, heart rate, and ventilation as the pregnancy continued. The increase in oxygen uptake was proportional to the weight gain, but the increases in heart rate and respiratory exchange ratio were not proportional. The aerobic capacity was assumed to be constant throughout gestation, 61% of maximum at 3 months to 70% at 8 months.

Sibley et al .(1981) found pregnant women were able to maintain their initial physical fitness level over a 12 week period by participating in a 10 week individualized training (swimming) program. The control group became less efficient over the study period with oxygen consumption and work rate decreasing - 10% and 20.8%, respectively. All the subjects were tested within three weeks of delivery and were all able to reach 72% of their maximum oxygen consumption values on the treadmill (determined by a modified Blake Multistage Progressive Treadmill test) without undesirable effects. The fetal heart rates before and after the treadmill test were within clinical norms. The apgar scores of the trained mothers' infants were high with 6 of the 7 infants scoring 8 and 9 and one infant scoring 9 and 10 at one and five minutes, respectively. The apgar scores of the control group were not published, but it is assumed all infants were healthy.

Recently, Collings et al.(1983) compared pregnant women trained for an average of 13 weeks on a bicycle ergometer at submaximal workloads. The subjects were placed in the training (n=12) and control (n=8) groups initially by their choice and the remaining women were randomly assigned to the two groups when it became apparent the authors had an interested population. There were no differences in the predicted maximal VO_2 from the Astrand's protocol between the groups at the beginning of the study.

The submaximal bicycle test found an increase in the training group and decrease in the control group of absolute aerobic capacity and functional capacity. The statistical analysis (ANOVA with post hoc Scheffe's test) found the trained group's increase (18%) in absolute aerobic capacity (l/min) over the untrained group's decrease (4%) was significant as was the trained group's third trimester fundtional capacity (ml/kg/min) versus the untrained group's functional capacity. The fetal heart rate increased significantly, during exercise and post-exercise, over resting values. The increase in fetal heart rate was attributed to three possible factors: 1) the exercise woke the fetus and increased its heart rate, 2) placental transfer of maternal catecholamines or release of fetal catecholamines secondary to maternal exercise, and/or 3) increased maternal and fetal temperatures may influence fetal heart rate, since FHR has a positive correlation with fetal temperature.

Collings et al. (1983) found no correlation between training and outcome of pregnancy. The results of the study may have been affected by the small sample size and factors of labour which could not be controlled, such as maternal medication, inaccurate determination of the onset of labour, and fetal presentation. Maternal exercise had no effect on fetal growth.

Dibblee and Graham (1983) used the Canadain home Fitness Test (CHFT) to group 16 primigravids into "fit" and "unfit" groupings. The women were not involved in any organized physical activity. The "fit" group (CHFT scores >8) had a constant absolute VO_2 max (l/min) and the changes in aerobic fitness (VO_2 max in ml/kg/min) were due to changes in body mass with pregnancy. The "unfit" group (CHFT scores< 8) had an increasing absolute VO_2 max(l/min) during exercise which decreased during post-partum and a constant aerobic fitness (ml/kg/min) which was attributed to cardiopulmonary demands. The increased body mass provided a sufficient workload for the unfit group to increase their VO_2 max (l/min). Greater gains in body fat weight in the unfit group were attributed to lower levels of activity compared to the fit group. Dibble and Graham found the only significant difference between the groups was a higher apgar score at one minute in the fit group's newborns a finding similiar to Collings et al. (1983).

Effects on the fetus of maternal exercise

In the literature, the effect of maternal exercise on the fetus has been documented as part of studies on maternal responses to exercise testing or training, and these results have been discussed previously. The literature that focused on fetal responses only to maternal exercise will be reviewed.

One of the first studies to evaluate the effects of maternal exercise on fetal heart rate (FHR) was by Hon and Wohlgemuth (1961) who recorded FHRs before and after a three minute step test. The authors felt that maternal exercise may have decreased uterine blood flow (UBF), therefore putting a temporary additional load on the uteroplacental oxygen transfer mechanism which caused the abnormal FHR pattern found in 6 of the 26 subjects. The post-exercise FHR recordings were put into 3 categories: 1) no remarkable change (4/26), 2) minor changes (17/26), and 3) irregularities, tachycardia, bradycardia (6/26). In subjects that were tested weekly, the FHRs fluctuated between categories 1 and 2. The frequency of changing from one group to another was not possible to determine since the serially tested sample size was small(n=6).
Hon and Wohlgemuth's data was collected between the 33rd and 43rd weeks of gestation from low and high risk pregnancies. It would have been preferable to have FHR recordings from a specified week in gestation (Pernoll et al., 1977), pregnancies of the same risk level, and a larger sample tested serially. In high risk pregnancies the FHR may have reflected factors other than maternal exercise.

Pokorny and Rous (1967) studied how physical work during the last four weeks of pregnancy was expressed in fetal heart sounds. Three different fetal heart rate reactions to exercise were 1) no change, which was attributed to mothers who were well-adapted to the workload, 2) a gradual increase of the FHR to a maximum at the beginning of steady state exercise, but returning to baseline by the end of the exercise period, and 3) a continuous increase in FHR until the end of the exercise period after which the FHR and maternal heart rates fell below normal values. A larger sample was needed to determine whether there were three different reactions or variations of a single reaction to the maternal exercise. It was assumed a larger sample would indicate whether FHR was dependent on maternal heart rate, and/or her reaction to the workload.

Stembera and Hodr (1967) used an exercise test to see the differences in FHR of healthy and potentially distressed fetuses in normal and abnormal pregnancies. In potential hypoxia distressed fetuses, there was a greater influence of extreme variations in the FHR, more to tachycardia than bradycardia.

The stage of gestation should be considered when interpreting FHRs after maternal exercise (Pernoll et al., 1977). Early in gestation the post-exercise FHR decreased, but later in gestation the post-exercise FHR increased, illustrating that when FHRs is taken is important. Pernoll and associates found no differences in FHRs recorded monthly, from low risk pregnancies, and those with mild complications, after cycling on an ergometer for 6 minutes. Since the maternal cardiac output and uterine blood flow decreased near term due to inferior vena cava compression, pooling of the blood in the legs may cause, according to the authors, fetal hypoxemia, and therefore, tachycardia. Near term, tachycardia could be interpreted as a possible increase in fetal cardiovascular system responsiveness to autonomic stimuli, and placental "respiratory reserve" decreasing in relation to fetal requirements. Older fetuses would be more stressed due to the diversion of uterine blood flow.

Dressendorfer and Goodlin (1980) found that maternal exercise at 80% of maximum oxygen uptake did not produce fetal bradycardia or tachycardia. FHR was about 142 beats per minute (bpm) before a submaximal bicycle test and reached peak values of 146 bpm. These values were in the same range as Stembera and Hodr's which were 140 -180 bpm before and after testing. The pregnant women trained by swimming 3 times per week. They had above average cardiorespiratory fitness, were in excellent health, and were non-competitive swimmers.

FHR prior to maternal jogging was 140-150 bpm and after jogging (1.5 miles and climbing three flights of stairs) the FHR was 180 -204 bpm in Hauth et al. (1982). In this study, neither the speed or the jogging was not controlled and the fitness levels of the participants was not determined at the start of the test period. Hauth used a non-stress test (NST) to evaluate the potentially acute effects of maternal exercise on fetal well-being. The reactive NST (at least two fetal accelerations of at least 10 bpm in association with fetal movement) had similiar mean times before and after jogging, therefore the fetuses were not compromised during the exercise. Hauth proposed two conclusions from the results - 1) in humans the uterine blood flow remained adequate with maternal (non-exhaustive) exercise and/or 2) post-exercise fetal tachycardia represented fetal compensatory state, which was supported later by Artal et al.(1984).

Fetal bradyacardia was observed during maternal exercise in the third trimester (Dale et al., 1982; Artal et al., 1984). Dale and associates(1982) found that the fetal heart rates decreased for the first 3 to 3.5 minutes of maternal exercise, a treadmill test, and returned to the baseline rate prior to the subjects reaching 80% of their predicted maximum heart rates. Artal et al (1984) found fetal bradycardia continued through exercise ,a symptom-limited max VO_2 treadmill test. One of their subjects went into premature labour at 37.5 weeks, and agreed to an internal FHR monitoring during a submaximal cycle ergometer test up to 65% VO_2 max. The FHR increased slowly during exercise from 140 bpm pre to 150 bpm post-exercise. The difference in the FHR responses to exercise led the authors to question whether fetal bradycardia was a normal physiological responses to exercise and , if so, was the increase in FHR post-exercise a compensatory mechanism for brief periods of hypoxia during exercise. The recovery time, return to normal FHR, could be dependent on the status of the fetus. Artal and associates postulated that fetal response depended on gestational age, level of catecholamines released by mother and consequently by the fetus, maternal stress and level of fitness probably influenced the level of sympathetic activity.

Collings and Curet (1985) found no evidence of the relationship of gestational age on FHR (Artal et al., 1984) when they tested subjects serially from the 28th to 38th week to 70% of maximum aerobic capacity. In comparison to their previous work (Collings et al., 1983) the post-exercise heart rates were higher due to 1) different types of exercise 2) longer duration of exercise . There were no FHR >180bpm since the subjects exercise tests were set according to their maximum aerobic capacity intensity, others (Hauth et al., 1982) did not standardize the test, therefore unknown stress per subject. Pernoll (1977) used 6 minutes of mild cycling to illustrate that in normal and mildly abnormal pregnancies, that FHR decreased post-exercise <35 weeks gestation, but after 35 weeks it increased. Collings and Curet tried to monitor FHR while cycling and found too much "noise" from the movement of legs and trunk. They did count the FHR during exercise by using the amplification system of the FHR monitor and found FHR was normal with moderate tachycardia.

The Fetal Breathing Movements (FBM), in study by Marsal et al., (1979) were more sensitive to maternal challenge than FHR. The FBM increased with maternal dynamic work (submaximal bicycle test), did not change with static work and passive movements , and decreased with maternal hyperventilation and hyperoxygenation. The FHR did not change during the five maternal challenges. The changes in FBM paralleled the maternal pCO_2 levels supporting CO_2 as a major stimulator of breathing movements even in neonatal life.

There were no direct correlations betwen mild maternal exercise (walking at 2 mph) and fetal body (FB) or breathing movements (FBM) in a study by Platt et al. (1983). A relationship between maternal sympathetic activity and degree of fetal activity was confirmed. Fetuses with increased FM or FBM post-exercise had significantly higher mean pre and post-exercise epinephrine levels. The mean norepinephrine and epinephrine levels increased significantly from pre to post-exercise, but after 30 minutes post-exercise the epinephrine levels were still elevated. The five reasons suggested for the findings were 1) one level of stimulus may increase FM or FBM while a higher level would decrease it, 2) the fetus acts independently of the mother, therefore varied observations were due to individualized adaptation to changes in maternal environment, 3) reduction in UBF could be due to the increase in catecholamines which would affect FBM, 4) fetuses had four basic behaviour states related to fetal age, and wakefulness and these stages were organized at 36 weeks (testing was at a mean gestational age - 34.6 weeks), and 5) during periods of low heart rate variablility, shaking the fetus, as in maternal exercise, would significantly increase its reactivity. Platt concluded that mild exercise produced variable fetal biophysical responses. The fetal activity (measured as FM and FBM) seemed to be associated with an increased maternal sympathetic activity and independent of maternal activity. The changes in FM, FBM, and catecholamine levels due to exercise were reversible.

Jakobovits (1983) reported a significant negative relationship between maternal exercise - climbing 2 flights of stairs - and FBM in a majority of the subjects (24 out so 33) tested. The purpose of the study was to establish the validity of FBM as a test of fetal well-being. The FBM test was useful if post-exercise data was analyzed. The varied results of the study- 24/33 with negative correlation of maternal exercise to FBM, 6/33 positive correlation, and 3/33 with no correlation - could have been due to the fitness levels of the women before or during gestation, the rate at which women climbed the stairs , the risk level of the pregnancies, and the time during the test period (35-42 weeks) that the women were evaluated. Pernoll et al., (1977) previously suggested the time of testing was important in FHR and Marsal et al., (1979) supported FBM as more sensitive than FHR. Jakobivitis added that the week of gestation should be specified when comparing and analzying FBM, if they are to be read like FHRs.

Uterine activity did not change after maternal exercise; weight-bearing (running) and non-weight-bearing (cycling) during the last 8 weeks of gestation (Veille et al.,1985). Maternal heart rate was higher , as expected, post-exercise. Fetal heart rate increased during the first 15 minutes post-exercise, but returned to baseline in the next 15 minutes. The authors concluded that moderate exercise in highly motivated, trained pregnant subjects did not increase uterine activity post-exercise.

Retrospective Studies

Researchers have used questionnaires to study the effects of scuba diving, jogging, and a combination of endurance activities on the outcomes of pregnancy. A retrospective study has a built in drawback, the subject's ability to remember events required, especially if over a long period of time as 9 months gestation. A pre-selection of subjects occurs by the women who answer an advertisement for subjects.

Questionnaires were mailed to 208 women, who responded to advertisements in national diving magazines, and posters in diving shops (Bolton, 1980). The subjects had been pregnant within 5 years of completing a scuba course. The descriptive questionnaire compared the extent of diving to obstetric and fetal outcome . One hundred thirty-six of 208 women dove one or more pregnancies to an average depth of 42.6 feet; 24 of the 136 women dove deeper than 99 feet during the first trimester. The women who dove (D) during pregnancies had a significantly higher level of diver certification and approval of their physicians and families than women who did not dive (ND) during their pregnancies. The frequency and depths of the dives decreased as the pregnancy progressed. Bolton found no relation between the risk of the pregnancy and the frequency of the six pre-selected complications of pregnancy - neonatal deaths, stillbirth, low birth weight, vaginal bleeding during pregnancy, spontaneous abortion, and birth defects. There were significantly more birth defects in the D group than the ND group ,but the percentage of defects in the D group was within the norms of the general population. The results of the study may not be representative of the diving population because women who had complications or undesirable outcomes may not have answered the advertisements.

Bolton recommended that 1) every woman diver of childbearing age should be informed of the potential risks of diving during pregnancy, and 2) the physician should aquaint her with the problems and encourage her to decide before becoming pregnant whether to dive and what limitations regarding depth, duration, and character of dives should be made. It was felt that many women would dive against medical advice or before pregnancy was confirmed. The Undersea Medical Society officially discourages diving during pregnancy until further studies are available. Bolton's guidelines for diving during pregnancy were 1) limit dives to 60 feet and duration to one-half the limits of the U.S.Navy no-decompression tables, 2) avoid strenous dives, hypoventilation, and chilling, or 3) snorkelling under optimal conditions as an alternative to scuba diving.

If women suspected they were pregnant , Turner and Unsworth (1982) suggested no diving below 30 feet or no diving at all was preferred. They presented a case study of a pregnant woman who dove 20 times between her 40th and 50th days after her last menstrual cycle. Most of the dives were to sixty feet or less (as sugggested by Bolton, 1980), three dives were to 100 feet, and one dive to 110 feet. There was one problem dive where the ascent rate was described as "very rapid", but the remaining ascents were estimated at 60 feet per minute, The pregnancy was normal, but the newborn had a number of abnormalities - arthrogryposis, and some dysgenic features. Turner and Unsworth implied that the abnormalities were due to the diving. A similiar time course as thalidomide, where the upper limbs were affected about the 40th day and the lower limbs about the 45th day due to the drug affecting the migration of cells used to form the posterior root ganglia was a proposed reason for the abnormalities seen from diving between the 40th and 50th day. Arthrogryposis was supposedly due to a muscle disease or abnormalities of cells forming the anterior root ganglion. Theoretically, diving is a detrimental factor to the fetus either through the bubble formation affecting the placenta's function or the fetus' circulation, or a secondary effect through hypoxia because of its effect on placental function.

Jarrett and Spellacy (1983) found women decreased their jogging as their pregnancies progressed. Mechanical factors of pregnancy - uterine enlargement, changes in weight distribution, and the suggested decrease in circulatory reserve - were attributed to reduced activity levels which was simliar to findings with voluntary exercise in mice (Karasawa et al., 1981).

The abortion rate was lower than normal, but fetal abnormalities were higher than expected. A larger sample size was suggested to find whether the fetal abnormalities occurred more often in pregnant women who jogged. The rate of abnormalities, 4 out 67 subjects or 6% was above the norms for the general population, 2-4%. The anomalies reported were not all classic anomalies and generally not life-threatening (hypospadias, metatarus adductus, right arm phocomelia, cogenital hip dysplaxia, torticollis, facial asymmetry).The low incidence of maternal and fetal complications could probably be attributed to the healthy condition of the subjects. The author had some reservations about the data 1) women with poor outcomes may or may not have responded, and 2) recall accuracy was unknown. The general conclusion was women who were trained and continued to run during pregnancy did not adversely affect their pregnancies.

Subjects who continued to participate in a combination of endurance activities - running, aerobic dance, cross-country skiing - at or near pre-pregnancy intensities until term gained less weight, had lighter infants, and shorter lengths of gestation than subjects who ceased participation in these activities before their 28th week (Clapp and Dickstein, 1984). Participation in endurance exercise prior to pregnancy was no a significant determinant in pregnancy weight gain, as was the continued and sustained participation in endurance exercise. The lighter newborns for gestational age may have been related to the lower maternal weight gain of exercising subjects, but it was important to note that with the decrease in the variables mentioned , there was no increase in morbidity.

The retropsective studies with elite athletes (Erdelyi, 1962; Zaharieva 1972; Berg et al 1983) who trained prior to and/or during pregnancy have found results which are similiar to those mentioned previously. The second stage of labour was shorter in elite athletes compared to non-athletes (Erdelyi,1960;Zaharieva, 1972) which was attributed to their better trained musculature and nervous systems for physical strain, generally (Erdelyi, 1962).Berg et al. (1983) noted the second stage was longer in athletes than national perinatal results, no reason for the diffference was proposed. Zaharieva (1972) noted that the first stage was prolonged in the Olympic atheletes due to rigidity of the uterus, stronger muscle tone, and an unusual lack of flexibility in the soft parts of the birth tract. Erdelyi found athletes had a 50% reduction in the number of cesarian sections, but Berg et al. found an increase in abdominal deliveries and higher percentage of high-risk pregnancies in athletes compared . to national data and lower birthweight of newborn, therefore a possible influence of training history on course of pregnancy and labour.

Conclusion

The human studies suggest that normal healthy pregnant women can be physically active without affecting the outcome of their pregnancy or the fetus if the intensity level is not exhausting. A pregnant woman will reach her maximum heart rate at a lower workload than a non-pregnant woman, but a pregnant woman should be able to maintain her aerobic fitness level or increase her fitness levels compared to a non-exercising pregnant woman. The training period could be 10 or more weeks, but the frequency should be at least 3 times per week for a significant difference to show between the exercising and non-exercising pregnant women. The duration of pregnancy was not viewed by a number of the researchers as an important question, but their studies eliminated premature deliveries by testing late in gestation or eliminating subjects who delivered beyond the normal gestation period of 40 weeks.

Small sample sizes prevented finding significance, if one exists, in the apgar scores and birth weights of the infants of exercising and non-exercising women. Further studies on exercise during pregnancy should have a sample size large enough to show whether the fetus via fetal heart rate or fetal breathing movements, and/or the infant at birth via apgar scores are in any way affected by maternal exercise.

An ideal study of exercise during pregnancy would monitor physical activity and aerobic fitness levels from early in the pregnancy, such as the first trimester. Since most of the fetus' development occurs in the first trimester, maternal exercise should effect the fetus more during this trimester than the last trimester. The outcomes of exercise during pregnancy such as the apgar scores, birth weight, and other factors indicating how the fetus adapted to the mother's regular physical activity would also be of interest to the mothers and those involved in her prenatal care and activities. The sample sizes should be large enough to show any significant changes due to maternal exercise.

Animal studies have followed the offspring of exercising pregnant animals for differnces in growth and structure. In future, as more work is done in exercise during pregnancy, a follow-up on the infants to find long-term effects of the exercise during their fetal development should be undertaken.

In reviewing the literature on exercise during pregnancy, a number of the papers on human pregnancy have been published in the past 10 years and with this increased interest in the area, a number of questions pregnant women have about physical activity during pregnancy should be answered within the near future. There should be some standard workloads or values used in testing pregnant women to make the comparison of various physiological variables and physical activities possible among the studies published enabling definite conclusions to be drawn on the effects of the physical activity on the pregnancy and its outcome.

APPENDIX B

Raw Data

Subject — Mean Heart Rates(bpm) during Cycle Ergometer Test at four workloads (kg) (see note)

Subject	1st Trimester				2nd Trimester				3rd Trimester			
	0.5	1.0	1.5	2.0	0.5	1.0	1.5	2.0	0.5	1.0	1.5	2.0
A	-	-	-	-	100	106	125	131	109	122.5	138	144
B	n	(.66) 90	(1.3) 110	130	n	98	n	150	100	120	142	*
C	-	-	-	-	95	105	125	147.5	101	115	135	150
D	-	-	-	-	89	100	120	135	90	110	122	138
E	-	-	-	-	83	87.5	103	123	89	94	110	125
F	81	98	108	134	80	87	110	125	98	120	130	147
G	93	110	133	150	94	110	125	150	110	125	138	144
H	n	(.66) 105.5	(1.3) 127.5	146	n	(.66) 115	(1.3) 130	170	97.5	105	132	147.5
I	93	107	120	142.5	98	110	130	150	121	136.5	152	*
J	-	-	-	-	87	110	128	145	113	150	*	*
K	98	123	143	*	99	125	140	150	110	129	147	*
L	97.5	133	132.5	150	105	125	150	*	122	138	150	*
M	113	135	150	*	109	130	150	*	117	138	150	*
N	-	-	-	-	103	125	150	*	105	125	147	*
O	98	112	133	155	110	129	153	*	130	158	*	*
P	-	-	-	-	105	146	*	*	108	132	156	*
Q	-	-	-	-	125	142	150	*	115	125	150	*
R	102.5	125	138	150	93	110	138	150	95	110	138	150
S	-	-	-	-	95	110	143	*	105	114	138	150
T	-	-	-	-	113	125	150	*	110	135	150	*

Note: The workloads 0.05, 1.0, 1.5, 2.0 kg at 50rpm on the Monark cycle ergometer equal 150, 300, 450, 600 kgm/m, which are equivalent to 25, 50, 75, 100 watts respectively.

"-" no heart rate data because the subject started the study in the second trimester.

"*" no data at this workload because the test had been terminated at the previous workload when the subject's heart rate had approached or reached 150 bpm.

"n" due to changes in testing protocol - subject B was not evaluated at 0.5 kg in the 1st trimester, and 0.5 kg and 1.5 kg in the 2nd trimester, and subject H was not evaluated at 0.5 kg in the 1st adn 2nd trimesters.

APPENDIX C:PREDICTED MAXIMUM OXYGEN UPTAKES

Maximum oxygen uptake (VO2max) values were predicted from the heart rates during the submaximal cycle ergometry tests, at 75 and/or 100 watts in the second and third trimesters, using Astrand's nomogram (de Vries, 1968). The VO2max values were rated (low, fair , average, good, high) according to Astrand's table of norms for women 20-29, and 30-39.

The T group (subjects A-J) had predicted VO2 values greater than the UT group (subjects K-T) at each workload during the second and third trimesters.

FITNESS CLASSIFICATION

Second Trimester:

Group	Workload (watts)	High	Good	Average	Fair	Low
T	75	5	2			
	100	4	5	1		
UT	75		1	4	4	
	100		2	1	1	

Third Trimester:

Group	Workload (watts)	High	Good	Average	Fair	Low
T	75	1	4	3		
	100	3	4			
UT	75			8	1	
	100			1	1	

VO2(1/min) Rating(Low, Fair, Average, Good, High) at selected workloads (75 watts, 100 watts)

Second Trimester

Subject	Workload(watts) 75		100	
A	3.0	High	3.4	High
B	n	n	2.5	Good
C	3.0	High	2.6	Good
C	3.4	High	3.1	High
E	*		3.9	High
F	*		3.7	High
G	3.0	High	2.5	Good
H	2.5	Good	2.0	Average
I	2.7	Good	2.5	Good
J	2.8	High	2.7	Good
K	2.4	Good	2.5	Good
L	2.0	Average	**	**
M	2.0	Fair	2.0	Average
N	2.0	Fair	**	**
O	2.0	Average	**	**
P	**	**	**	**
Q	2.0	Fair	**	**
R	2.4	Average	2.5	Good
S	2.2	Average	*	**
T	2.0	Fair	2.7	Fair

Third trimester:

A	2.4	Average	2.7	Good
B	2.3	Average	**	**
C	2.6	Good	2.5	Good
D	3.2	High	3.0	High
E	*		3.7	High
F	2.7	Good	2.6	Good
G	2.4	Good	2.7	High
H	2.7	Good	2.6	Good
I	2.0	Average	**	**
J	**	**	**	**
K	2.1	Average	**	**
L	2.0	Average	**	**
M	2.0	Average	**	**
N	2.1	Average	**	**
O	**	**	**	**
P	1.9	Fair	**	**
Q	2.0	Average	**	**
R	2.4	Average	2.5	Average
S	2.4	Average	2.5	Good
T	2.0	Average	**	**

* The subject's heart rate at this workload was too low to predict a maximum oxygen uptake from Astrand's table and nomogram.

** The submaximal cycle ergometry test had been terminated at the previous workload when the subject's heart rate approached or reached the designated maximum of 150 bpm, therefore no predicted maximum oxygen uptake.

"n" Subject B was not tested at 75 watts in the second trimester, therefore no data for predicting maximum oxygen uptake.

APPENDIX D - ACTIVITY LOGBOOKS SUMMARIZED

First Trimester(if available)

Subject	* >3hr/wk	Activities Type	Duration	Freq/wk	Comments
A	started logbook in second trimester				
B	*	Aerobics Class	1hr	3	
C	*	Weight Training	90min	2-3	
D	started logbook in second trimester				
E	started logbook in second trimester				
F	*	Swimming		3-5	1-2.4km
G	started logbook in second trimester				
H	started logbook in second trimester				
I	no logbook				
J	started logbook in second trimester				
K	no logbook				
L	*	Swimming/tennis /or skating	1hr	1-2	
		Prenatal fitness	1hr	1-2	
M	started logbook in the second trimester				
N	started logbook in the second trimester				
O	no logbook				
P	*	Cycling		2-3	10miles
		Softball		2	
R	*	Aerobics classes	1hr	3	
S	started logbook in second trimester				
T	started logbook in second trimester				

Second Trimester

Subject	* >3hr/wk	Activities Type	Duration	Freq/wk	Comments
A	no activity recorded				
B	*	Aerobics Classes	1hr	2-3	
		Walking	30min	1	1-1/2miles
C	*	Weight training	90min	2-3	
		Walking	1-2hr	1	
D	*	Prenatal fitness	1hr	1	
		Walking	30-90min	1-2	
E	*	Aerobics Classes	1hr	1	
		Swimming		1-2	1km
		Windsurfing	1	4	

F	*	Swimming		4-5	1.5-4.8km
		Lifecycle	24-36min	1-3	
G	no activity recorded				
H	*	Prenatal fitness	90min	1	
		Swimming	20-30min	1-4	1km
		Walking	30min	1	
I	no logbook				
J	no activity recorded				
K	no logbook				
L	*	Prenatal fitness	1hr	3	
M	*	Walking	30-45min	3-4	
		Cycling	30min	irregular basis	
N	*	Prenatal fitness	1hr	2	
		Tennis	45-60min	3	
O	no logbook				
P	*	Badminton		1	
		Walking		2	
Q	*	Aerobics classes	40-60min	1-3	light-mod.
		Running	30min	2-4	
		Walking	45min	3-4	
R	*	Prenatal fitness	1hr	2-3	
		Walking	1hr	1-2	
S		Jazzercise	1hr	1-2	
		Tennis	30min	1	
T		Swimming	30min	1	
		Volleyball	15-20min	1	

<u>Third Trimester</u>

A	no activity recorded				
B	*	Aerobics classes	1hr	1	until term
		Prenatal fitness	1hr	2	until term
C	*	Prenatal fitness	1hr	1-2	to 37th wk
		Walking	1hr	1-2	
D		Prenatal fitness	1hr	1-2	to 37th wk
		Swimming		2-3	35-40th wk
E	*	Aerobics classes	1hr	3	to 37th wk
		Swimming	40-60min	1-2	to 34th wk
		Walking	60-90min	1	33-37th wk
		Windsurfing	1hr	1	to 29nd wk

		Cycling		1-2	20km 31-32nd wk
	(did not keep logbook beyond 37th week)				
F	*	Swimming		2-4	1.5-3.0km to term
		Lifecycle	48-60min	4	to term
G	no activity recorded				
H		Prenatal fitness	90min	1-2	to 37th wk
		Swimming	20-30min	1	to 36th wk
		Walking	30-90min	1-3	to term
I	no logbook				
J	*	Prenatal fitness	60min	1-2	to 38th wk
K	no logbook				
L	no activity recorded in third trimester				
M	-	Walking	30min	daily	to term
N	-	Prenatal fitness	1hr	2	to term
O	no logbook				
P	-	Walking	20-30min	1-2	24-36th wk
			20-40min	3	38-40th wk
Q	*	Aerobics classes	40min	1-3	25-33rd wk
		Walking	40min	3-4	to term
R	*	Prenatal classes	1hr	2-3	to term
		Walking	1hr	1-2	to term
S	-	Jazzercise	1hr	1-2	to 36th wk
		Tennis	30-45min	1-2	28-39th wk
		Walking	30-90min	irregular basis	
T	-	Prenatal fitness	30min	1	27-31st wk
		Aerobics classes	1hr	1	36-38th wk
		Volleyball	15-20min	1	27-31st wk 34-38th wk