Exercise in Pregnancy: Physiological Basis of Exercise Prescription for the Pregnant Woman

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A pregnant woman participated in cycling events in the 2000 Olympics. Recently there was concern about the participation of a pregnant woman in the Australian netball team. More and more women are anxious to pursue sports during their pregnancies and to maintain condition. For the clinician or sports physician caring for women who want to maintain a high-level of physical activity there is no simple exercise prescription. It is probable that continuing exercise by women who are already conditioned will not result in foetal compromise, unless there are hidden or unknown complications of pregnancy. Pregnant women should probably exercise within limits that do not cause severe discomfort and should, as pregnancy progresses, be prepared to moderate the intensity and duration of their exercise programs to avoid risks and injury. It is probably not advisable for women to begin high intensity exercise programs when pregnant, although moderate exercise is beneficial to both mother and baby. The type of activity that is undertaken has to be taken into consideration and in particular the adverse effects of supine activity in late gestation recognised.

Introduction

There can be no generalised prescription of types and levels of activities undertaken during pregnancy, except for those few sports which are contraindicated, because the risk of harm to the foetus or mother is almost inevitable (eg boxing, wrestling, scuba diving).

This review provides a conceptual basis to help in the definition of the risks and benefits of exercise in pregnancy. The effects of exercise are however complicated by the level of maternal conditioning and health prior to and during pregnancy, the presence (known and unknown) of other conditions that may affect foetal health and development, and the level and type of physical activity undertaken in pregnancy. Consideration could also have been given to those work-related activities associated with high levels of physical performance and endurance (e.g. firefighting, rescue services etc). This has not been discussed and it is not appropriate to extrapolate from studies that examine the effects of exercise in pregnancy to the potential risks/benefits of those work based activities that are physically demanding.

Ideally, exercise prescription in pregnancy should be based on the findings of clinical trials that have determined the effects of regular exercise on physical fitness, ease or difficulty of labour and delivery and on the course and outcome of pregnancy. In a recent review in the Cochrane database by Kramer (2001) only 5 trials involving 142 women were judged to be relevant. The trials were small and most were not of "high methodology quality". The 5 trials reported significant improvement in physical fitness in the exercise group but overall their results only allowed the exclusion of extremely large effects. There was a significant increase in the incidence of preterm labour, but this was biased by one study. In view of this lack of evidence-based information, the clinician who is going to assess and advise on the effects of exercise in pregnancy has to depend on his/her understanding of the changes that can occur in pregnancy, the potential effects of exercise on the mother and foetus and then develop an individual program for each woman, which should be conservative.

Maternal physiology

Ovulation and fertilisation

Assuming that a normal menstrual cycle is about 28 days long, then ovulation occurs at about 14 days and fertilisation within 24 hours. Implantation of the developing blastocyst into the uterine endometrium, which has been prepared through the combined actions of oestrogen and progesterone, in order to support early development, occurs some days later. At this very early stage of pregnancy, the corpus luteum, from which the developing ovum was shed and which is secreting oestrogen and progesterone, comes under the control of human chorionic gonadotropin secreted by the developing blastocyst. The blastocyst completely embeds in the lining of the uterus. The leading edge of its outer cell mass forms the placenta and the inner cell mass becomes the embryo. This occurs before a woman realises that she is pregnant.

Hormonal maintenance of pregnancy

Maintenance of pregnancy depends on the increasing production of the hormone progesterone. Blockade of progesterone causes abortion. As well, levels of the hormone oestrogen also increase progressively. Many of the changes in maternal physiology that occur during pregnancy depend on the secretion of oestrogen and progesterone, although other hormones such as relaxin, and human placental lactogen affect maternal physiology. Relaxin appears to be responsible for softening of ligaments, while placental lactogen induces insulin resistance and is, as far as the mother is concerned, "glucose sparing".

There are other changes in maternal endocrine systems not directly associated with reproduction or maintenance of pregnancy (for example, thyroid and adrenal function are both enhanced). At about 8 weeks gestation the foetus and placenta acting in conjunction take over oestrogen production, while the placenta becomes responsible for the production of progesterone.

Although many of the changes that occur in maternal physiology during pregnancy are related to the high levels of oestrogen and progesterone, some changes cannot be reproduced by the simple administration of excessive amounts of these hormones.

Metabolic demand of pregnancy

Under resting conditions $\dot{V}O_2$ increases with increasing gestation and is maximal at term (about 16-32 % above nonpregnant controls, Lotgering et al., 1985). When corrected for the high metabolic rate of the uterine contents, it has been determined that near term, maternal $\dot{V}O_2$ is only 4% higher, even though renal function, cardiac function and respiratory work are increased (see below).

Physical work capacity is more affected by pregnancy than $\dot{V}O_{2max}$. Studies on PWC_{170} (that is, absolute bicycling work load that elicits a heart rate of 170 bpm - determined at submaximal workloads) is unchanged during pregnancy (see Lotgering et al., 1985). However other studies (Cooper, 1991) suggest that this may not be the case for weight bearing exercise in late pregnancy.

Cardiovascular, renal and respiratory function in pregnancy

There are profound changes in cardiovascular, respiratory and renal physiology, and in the control of maternal fluid and electrolyte balance. These changes occur early in pregnancy and could affect physical performance. They occur before there is an increase in demand so that, under resting conditions, the increase in cardiac output is greater than the increase in oxygen demand. Coupled with the early increase in minute ventilation, there is a decrease in arteriovenous oxygen difference (Figure 1). Changes in renal function also occur early in pregnancy.

Blood volume increases gradually so that by term it is increased by nearly 50%. Plasma volume increases early in pregnancy and continues to rise until about 32 weeks gestation; the increase is about 30-60% (Scott, 1972). (Term is approximately 40 weeks.) There is considerable variation in the magnitude of this increase (see Lotgering et al., 1985). It has been postulated that this increase in blood volume is a result of the increased capacitance of the cardiovascular system. In particular there is an increase in compliance of the venous side of the circulation due to the smooth muscle relaxing effects of progesterone (Metcalfe et al., 1981). As well, there is a marked fall in peripheral vascular resistance that contributes to the increased capacitance of the cardiovascular system. For plasma volume to increase, there must be salt and water retention. In part this is due to the intense activation of the renin angiotensin aldosterone system that occurs in normal pregnancy. Both the levels of the enzyme renin, and its substrate angiotensinogen from which angiotensin is formed, are increased (Skinner et al., 1972). Angiotensin II is a potent stimulus for aldosterone. Both angiotensin and aldosterone stimulate salt retention.

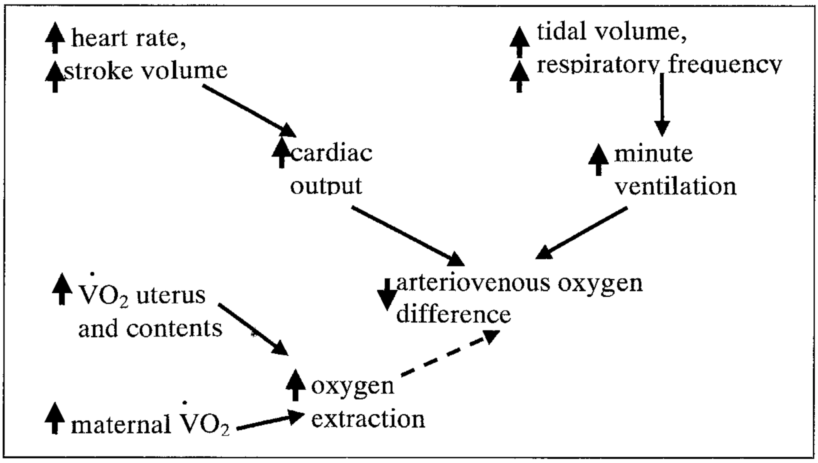

Figure 1: Effects of pregnancy induced changes in cardiovascular and respiratory function on arteriovenous oxygen difference. Solid arrows indicate effects of factors responsible for the fall in arteriovenous oxygen difference. Dashed arrows represent effects of factors that will increase arteriovenous oxygen differences.

As well as the increase in plasma volume there is also an increase in total red cell mass which reaches about 20% at term (Lund & Donovan, 1967). Since the increase in plasma volume and the increase in red cell mass are different, the former being 50% and the latter 20%, it follows that there is a physiological anaemia of pregnancy; that is, a decline in the oxygen carrying capacity per unit volume of blood. This acts as a stimulus to the production of erythropoietin (EPO). However renal blood flow is dramatically increased in early pregnancy so that there is a renal hyperaemia, thus stimulation of EPO production is delayed.

Cardiac output and its distribution change in pregnancy (see Lotgering et al., 1985). Cardiac output increases progressively throughout the whole of gestation. The increase is of the order of 40% and it peaks at about 20 to 30 weeks of pregnancy. More correctly, the rate of increase in cardiac output is reduced from this time on; as well, cardiac output becomes highly dependent on maternal posture. This is because, in the supine position, the gravid uterus compresses the inferior vena cava so reducing venous return.

Cardiac output is the product of heart rate and stroke volume. Most studies show that heart rate is increased early in pregnancy and continues to increase throughout pregnancy (Clapp et al., 1988). The mechanism behind this increase is unknown, although it is known that there is modulation of baroreflex mediated control of heart rate and renal sympathetic nerve activity in pregnancy, particularly through the central actions of the hormone, 16 α-hydroxyprogesterone.

There is also an increase in stroke volume, possibly due to an increase in left ventricular size rather than to a change in preload, afterload or contractility. The internal diameter of the left ventricle is enlarged during pregnancy with little or no increase in wall thickness (Rubler et al., 1977; Katz et al., 1978; Larkin et al., 1980). Since this response can be duplicated in guinea pigs by administration of oestrogen, it may be hormonally dependent (Morton et al., 1985). While preload may not be important in determining stroke volume when a woman is upright, it is important when she is supine as a reduction in preload is responsible for the fall in cardiac output of supine women in late pregnancy. The distribution of the cardiac output changes; most clinicians are aware that a very early sign of pregnancy is dilation of veins draining the breasts. Blood flow to the uterus is greatly increased (from <50mL/min in non pregnant women to >500mL/min in late pregnancy Sturgiss et al., 1994). About 90% of uterine blood flow (UBF) is supplied to the placenta in the late gestation. Placental blood flow determines nutrient and oxygen supply to the developing foetus.

There is also an increase in renal blood flow early in pregnancy. Changes in renal blood flow and other changes in renal function occur in animals which can be induced to go into a "pseudopregnant" state (see Lumbers, 2000). Thus they are not dependent on the presence of the conceptus. In addition to the increase in flow to the reproductive organs and the kidney, skin blood flow is increased. It is most likely that the increase in skin blood flow represents a physiological response to the increase in maternal metabolism and the metabolic activity of the conceptus (that is, it occurs in response to increased heat production). Therefore total peripheral resistance declines so that in early pregnancy arterial pressure falls. It then increases slightly to nonpregnant levels (see Lotgering et al., 1985)

Coupled with the increased capacity of the cardiovascular system, resulting from the vasodilation of peripheral vascular beds and increased capacitance on the venous side of the circulation there must be an increase in circulating blood volume. Thus, there is (as mentioned above) salt and water retention. As well there is a fall in plasma osmolality, so that the pregnant woman is slightly over hydrated. These changes in cardiovascular volume and function could enhance physical performance through the provision of a greater capacity to perfuse exercising muscles. This is particularly the case in early pregnancy when the competing demands of the conceptus are not excessive.

In addition, respiratory function is stimulated and respiratory frequency increases (caused by the hormone progesterone). This, coupled with increased alveolar ventilation results in an increased respiratory minute volume, lowered arterial PCO_2 and bicarbonate with a small increase in pH. Since total pulmonary resistance is only about 50% of nonpregnant values (due in part to increased compliance resulting from the softening of the rib cage), the increase in the work of breathing is small (see Lotgering et al., 1985). As Figure 1 shows, the increased capacity of the circulation, the increase in cardiac output and the changes in respiratory function all lead, in the resting state, to a reduced arteriovenous oxygen difference. This may offer, in early pregnancy, the potential for improved physical performance in those sports dependent upon maximum physical effort.

Effects of exercise on the mother

Cardiovascular

In a carefully controlled study Cooper (1991) showed that non weight bearing exercise (cycling) caused similar increases in cardiac output, stroke volume, oxygen consumption and ventilation to those occurring in the same women in the postpartum period. If, however, the exercise was weight bearing (e.g. treadmill exercise) the increase in $\dot{V}O_2$ was greater than could be accounted for by the increase in body weight. The effects of weight bearing exercise were compounded by the stage of pregnancy and the intensity of exercise. Thus when late gestation pregnant women worked on a treadmill at a rate that increased heart rate to 140 bpm this was a greater stress than the equivalent non weight bearing exercise.

During exercise, plasma volume decreases as a function of exercise intensity reaching a maximal reduction of 14% at approximately 60% $\dot{V}O_{2max}$. This occurs within 10 minutes (Greenleaf et al., 1977). The decrease in plasma volume following 5 min cycling exercise at 75W was greater after 29 weeks. (Pivarnik et al., 1990).

Maternal metabolism

Maternal plasma glucose levels fall with increasing gestation; Glucose turnover increases but not when normalised for body weight, suggesting that the increase is proportional to the increase in $\dot{V}O_2$ and the substrates used for combustion do not change. In exercising pregnant women, glucose levels fell with moderate short-term exercise, the decrease was greater at high work loads. It has to be suggested that this hypoglycaemia could affect mentation, although this does not appear to be documented.

Foetal development

The earliest stages of development are characterised both by growth and by differentiation. Formation of major organ systems occurs very early in pregnancy; in fact it begins before a woman may know she is pregnant. It is not the place of this text to go into a detailed description of embryogenesis; but it is important to realise that this highly complex, genetically controlled process is extremely sensitive to disruption and alteration. Even though this disruption may be transient and the initial effects minor, because they occur so early in development and because the human will grow 100 billion fold from conception to adult life, these very minor effects can be amplified and produce congenital abnormalities.

It is often not known what has caused a particular congenital malformation, although chemical agents, infectious agents and genetic factors can play a role. With respect to exercise in pregnancy, perhaps the factor most likely to be implicated in the aetiology of congenital defects is excess heat production. It is extremely unlikely that the redistribution of cardiac output away from the uteroplacental vascular bed would cause congenital malformations, simply because at the time at which dysmorphogenesis is most likely the metabolic demand of the total conceptus is small. Susceptibility to dysmorphogenesis and the production of major structural malformations is significantly reduced from about 12 weeks gestation onwards. By 12 weeks of pregnancy, the pregnant uterus is just palpable at the level of the symphysis pubis.

Once embryogenesis is complete, intrauterine development is characterised mainly by extremely rapid growth; at this time of life growth rate is maximal and many organs undergo substantial growth and differentiation up to and even after birth. Most renal nephrons are formed in the later stages of pregnancy and the brain continues to grow for the first two years of life. In late gestation, there is also maturation of organ systems in preparation for life after birth.

The questions most likely to be asked concerning exercise in pregnancy are whether or not exercise affects foetal growth, causes foetal distress or precipitates premature labour.

There are four potential adverse foetal outcomes from maternal exercise in pregnancy. These are;

1) risk of congenital malformations from hyperthermia
2) risk of physical injury to the foetus
3) effects of exercise in pregnancy on growth
4) risk of premature labour.

Regarding maternal health and well-being, aerobic exercise in pregnancy results in increased physical fitness (Kramer,2001). In other studies, swimming as a form of exercise in the second trimester did not improve physical fitness but was associated with an increased sense of wellbeing, improved appetite and more restful sleep patterns (Sibley et al.,1981). Exercise in pregnancy has been shown in experimental animals to reduce maternal insulin resistance (Lopez-Luna et al,1998) and it has been suggested that it has a role in management of gestational diabetes.

For the mother, potential adverse outcomes of exercise in pregnancy are few. There may be an increased risk of physical injury, bearing in mind that there is increased ligament laxity which may affect joint stability, and an increased risk of haemorrhage from the conceptus. To determine if an individual woman and her foetus are "at risk" when undertaking exercise the following need to be taken into account;

1) type of exercise
2) level of intensity and duration of the exercise
3) level of training before pregnancy
4) whether or not the pregnancy is complicated by any other factors which may place the foetus at risk. Table 1 shows conditions which are absolute or relative contraindications for exercise during pregnancy.

Cardiovascular	**Pregnancy related**	**Others**
Active myocardial disease	Uterine bleeding, ruptured membranes	Acute infections
Rheumatic heart disease	Foetal distress (suspected)	
Congestive cardiac failure	Intrauterine growth retardation	
Severe hypertension	Isoimmunisation	
Pulmonary embolism	'At risk' of preterm labour	
Thrombophlebitis	Inadequate antenatal care	

•There may be other clinically prescribed contraindications.

Table 1a: Some medical conditions that are an absolute containdication to exercise in pregnancy• (modified from Brukner & Khan, 2001).

Medical	**Pregnancy related**	**Lifestyle**
Diabetes mellitus	Multiple pregnancy	Sedentary lifestyle prior to pregnancy
Essential hypertension	3rd trimester breech presentation	Excess obesity
Thyroid disease	excess ligament laxity and joint mobility	
Anemia		

•Consultation with clinician re suitability of exercise and level of exercise required.

Table 1b: Medical conditions which require conservative management with respect to exercise.*

Risks arising from sport and exercise in pregnancy

Injury

As a general proposition it is not advisable for a woman to undertake, for the first time, new forms of physical activity and training in pregnancy.

The type of injury that an exercising woman may be exposed to depends on the type of activity undertaken. Thus contact sports such as boxing are contraindicated, skydiving and parachute jumping are also ill-advised and in late pregnancy, horseriding, and skiing are contraindicated. Scuba diving requiring decompression should be avoided throughout pregnancy because of the risk of the air embolism. High altitude trekking and mountaineering are also ill-advised (Niermeyer, 1999) because of the risk of foetal hypoxia (see below).

Late in pregnancy, exercises that involve lying in the supine position (which compromises venous return, see above) and exercises that increase lordosis and cause back pain should be avoided.

Heat and heat transfer

A high level of physical exercise is associated with increased heat production. The developing foetus has a higher temperature (about 0.5°C> than maternal, Bell, 1987). Thus, there is a gradient for heat transfer from foetus to mother. The foetus is dependent on the mother for loss of the heat it produces. If maternal heat production is high, and heat dissipation from the mother is slow (i.e. she is exercising in a warm and humid environment), then it is possible for the foeto-maternal heat gradient to be reversed so that heat flows from mother to foetus rather than in the reverse direction. In animal studies, during acute exercise (70%

$\dot{V}O_{2max}$ at 40°C) maternal temperatures returned to baseline at about 90-100 min but foetal temperatures were still elevated at this time (Lotgering, et al., 1983).

At rest, the mother loses about 60% of her body heat by radiation, about 30% by convection, and 5 to 10% by sweating. With moderate submaximal exercise or in studies where women self-select their level of activity, core temperature does not rise above 1.5°C (see McMurray et al., 1993).

Heat loss requires increased distribution of the cardiac output to the skin. Thus, with severe exercise in animals a high level of heat production is associated with diversion of uteroplacental flow not only to exercising muscles but also to sites responsible for heat loss (Bell et al., 1986). This may be responsible for delaying the return of foetal body temperature to normal (see above).

Thus there are two potential effects associated with high levels of maternal heat production. The first occurs very early in pregnancy and is related to potential teratogenic effects of raised body temperature. There is no evidence to suggest that transient increases in body temperature such as may occur following intense physical activity cause foetal malformation. There is evidence however, that sustained increases in body temperature, as may occur with infections, are associated with a slightly increased risk of congenital malformations. Studies performed in animals show that, if core temperature rises above 39°C, there is an increased risk of neural tube defects (Edwards, 1969; Kilham & Ferm, 1976). Therefore it is advisable in early pregnancy that either the intensity or duration of exercise is reduced or that activities are carried out in conditions in which there is rapid and efficient dissipation of maternal heat. For example, swimming allows rapid heat dissipation, provided the pool temperature is not excessive, as well as reducing stress on joints.

A second effect, more likely later in pregnancy, is the effect of redistribution of maternal cardiac output away from the uteroplacental unit to the skin and to exercising muscles, on foetal oxygen and metabolic supply and on the removal of the waste products of foetal metabolism. Studies in animals, in particular in the exercising pregnant ewe, have shown that high levels of aerobic exercise cause a reduction in uteroplacental bloodflow (UBF). For example in sheep, UBF fell 13% during 10 minutes of exercise at 70% $\dot{V}O_{2max}$, 17% at 100% $\dot{V}O_{2max}$ and 24% after 40 minutes at 70% $\dot{V}O_{2max}$. During prolonged submaximal treadmill exercise in sheep, uterine oxygen consumption was maintained however and blood flow was redistributed to the placental cotyledons at the expense of the myometrium(see Lotgering et al., 1985).

Uteroplacental blood flow and supply

If uteroplacental flow is compromised by exercise it may have acute or short-term effects perhaps resulting in foetal distress. If the activity is regular and intense then there may be long-term effects perhaps resulting in foetal growth retardation and possibly effects on the length of gestation resulting in an increased incidence of preterm labour.

There are problems with determining the degree to which these risks occur in human pregnancy, let alone determining the quantitative relationship (if any) between the intensity and/or duration of exercise and level of risk.

A major problem is the difficulty in obtaining detailed information on the effects of exercise on the human foetus. Monitoring of foetal heart rate (FHR) and its variability is all that can be easily accomplished and even with FHR monitoring it is not feasible to carry it out during exercise. Measurements of FHR not complicated by artefact can only be obtained before and immediately after exercise at a time when many of the effects of exercise are rapidly dissipating. Other methods for determining acute effects on the foetus of exercise are measurement of foetal movements and breathing activity (which provide indices of foetal well-being). Long-term effects can be assessed by measuring birth weight and body composition and the length of gestation.

It is difficult to extrapolate in a quantitative sense from animal studies because most experimental animals are quadrupeds and the degree to which utero-placental blood flow is maintained during severe exercise might be very different from the extent to which it is maintained in the upright bipedal woman. Nevertheless studies in animals do indicate the type of change that might occur in exercising pregnant women.

One study found that placental blood flow in late gestation exercising pregnant sheep fell to about 75% of control irrespective of the ambient temperature (Bell et al., 1986). In another study it was found that during pyrogen induced fever there was no diversion of flow away from the conceptus (Blatteis et al., 1988). When pregnant ewes were exercised at 70% VO_{2max}, foetal arterial PO_2 fell by 3mmHg and O_2 content fell by 1.5mL/100mL (resting value 5.8 mL/100mL) but returned to control at 20 minutes afterwards (Lotgering et al.,1985). These data suggest that acute severe exercise does not represent an hypoxic stress to the foetus.

There is a large literature on the effects of exercise on human FHR, so only a few studies will be mentioned. Effects are most evident in the third trimester when maintenance of supply to meet the ever increasing demands of the large foetal and placental mass is most likely to be compromised.

FHR is normally 120-160 bpm with a baseline variability of at least 5 bpm. It shows accelerations (>15 bpm for >15s) and decelerations (<15 bpm for >15s). FHR >160 bpm for >2 min is classified as a tachycardia; when FHR <120 bpm for >2min, it is classified as a bradycardia (Brenner et al., 1999). Bradycardia occurring as a result of medical complications has a poor outcome. However this cannot be extrapolated to effects of exercise on FHR. FHR generally increases after exercise and variability is reduced (MacPhail et al., 2000). The increase is to a maximum of 15±11 bpm (SD) at 60±12% maximal aerobic capacity but depends on gestation age, exercise intensity and duration (Clapp et al., 1993). There is no evidence that FHR responses to exercise are an index of the beneficial or adverse effects of exercise in pregnancy on foetal outcome, with the exception of the appearance of foetal bradycardia which might indicate foetal hypoxaemia (Manders et al., 1997).

Foetal movements and breathing activity are another index of foetal health. Active breathing foetuses are healthy foetuses. This is because these activities increase oxygen consumption. A simple example of the extent to which foetal movements and breathing increase oxygen consumption is seen by the rise in foetal arterial PO_2 that occurs when foetal sheep are paralysed (unpublished observation). In human pregnancies, maternal exercise to the level of 82% of maximal increase in heart rate (MIHR) for 30 min caused a reduction in body movements. This reduction was negatively correlated with the increase in %MIHR, but foetal breathing movements were increased unless %MIHR was >90%. In 2 other foetuses unexpected bradycardias occurred with exercise (Manders et al., 1997).

Overall, there is no evidence that foetuses are acutely compromised by moderate levels of exercise, provided there are no medical complications (see Table 1).

The third way in which the effects of conditioning and exercise over the long term after the outcome of pregnancy can be assessed is by measuring body mass at birth and determining the incidence of preterm labour. As pointed out in the introduction, there is still a paucity of information on this topic. Overall there is a suggestion (Bell et al., 1995, Clapp et al., 2000) that moderate exercise in fact improves foetal weight at birth, whereas severe exercise causes a reduction in birth weight (Bell et al., 1995, Clapp et al., 1990, 1995, 1996). The reduction in birth weight is due to a reduction in neonatal fat mass. In view of the overwhelming epidemic in the Western world of obesity, it is tempting to suggest that this may not be an adverse outcome of exercise in pregnancy. However this is speculation and there are strong arguments to the contrary (Barker, 2001). The cause of the reduction in birth weight is not clear. It could be the result of redistribution of the cardiac output so that uteroplacental flow is compromised for long periods throughout pregnancy. It is likely however that the reduction in body growth represents reduced nutrient supply due to maternal utilisation of carbohydrate. Studies in rats have shown that exercise reduces the deposition of glycogen in the foetal liver (Houghton et al., 2000).

It is not certain whether or not the length of pregnancy and the incidence of preterm labour is affected by a continuing high level of physical activity throughout gestation. Bell et al. (1995) and Kramer (2001) suggest that the incidence of preterm labour is increased but this evidence is weak. Uterine contraction frequency in late gestation women was increased 5.5 fold when they were exercised at a heart rate of 140 bpm for 20 minutes on a cycle ergometer (Spinnewijn et al.,1996). In other studies, length of gestation did not appear to have been affected by continuing physical activity (Kardel & Kase,1998; Sternfeld et al, 1995).

Therefore, for the clinician or sports physician caring for women who want to maintain a high-level of physical activity there is no simple exercise prescription. It is probable that continuing exercise by women who are already conditioned will not result in foetal compromise, unless there are hidden or unknown complications of pregnancy. Pregnant women should exercise within limits that do not cause discomfort or stress and should, as pregnancy progresses, be prepared to moderate the intensity and duration of their exercise programs to avoid risks and injury. Women should not begin high intensity exercise programs when pregnant, although moderate exercise is beneficial to both mother and baby. The type of activity that is undertaken has to be taken into consideration and in particular the adverse effects of supine activity in late gestation acknowledged.

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