Exercise and Diabetes — Dr Mac Robertson

Dr Mac Robertson MBChB, MFGP (SA)

Curriculum Vitae

Dr LI Robertson studied at Cape Town University where he received the MBChB in 1954. He did some post-graduate training at McCord Zulu Hospital (Durban), at St Monica's Home (Cape Town), received the MFGP(SA) in 1975 and has been in Private Family Practice in Durban since 1957. He has a wide interest in different fields of medicine, and at the moment still holds the following posts: Senior Medical Officer -Diabetes Dept (Addington Hospital), Medical Director - Institute of Human Sexuality. He also makes time to serve on several committees. He is an elected member of the SA Medical and Dental Council and gives time to many other committees serving the community. Dr Robertson has presented many papers at Medical Conferences, has published several scientific papers and contributed to two medical textbooks.

Summary

After the discovery of insulin, exercise was strongly promoted as a cornerstone in the treatment of diabetes. In the last two decades, our knowledge of the physiology and pathophysiology of exercise as well as our understanding of diabetes has increased so tremendously that GPs need to reconsider the prescription of exercise as a panacea for all diabetics. Many of these new concepts are analysed in this article and the practical implications for the GP and his patient summarised. Very useful, guidelines and recommendations are given throughout.

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Over the past decade we have witnessed an explosive increase in the popularity of exercise and training, which has become for the young, (and indeed many not so young), an integral part of their recreational and social activity.

As health-care professionals we welcome this, as there is no doubt about the importance of exercise in promoting physical and mental health, and, perhaps, even in preventing and helping to cure disease.¹

Due to its known blood-glucose

lowering effect, exercise has traditionally been recommended as an important component of diabetes management. Soon after the discovery of insulin, exercise was strongly promoted as a cornerstone in the treatment of diabetes.2 And for about half a century this dogmatic opinion appeared to be irrefutable. However, in the last two decades, our knowledge of the physiology and pathophysiology of exercise and training, 3,4 as well as our understanding of diabetes, has increased tremendously. Consequently, we have had to revise our opinion that physical activity is a panacea for all diabetic patients. 4,5,6,7

It is evident that the effect of physical exertion differs fundamentally in Type I and Type II diabetics. Further, the prescription of physical exercise as a means of improving metabolic control should now be regarded as obsolete. Even in Type II diabetics, the results from a number of elaborate investigations are disappointing with regard to the beneficial effects of physical activity on glucose tolerance. However, this must not lead to the conclusion that Type I and many Type II diabetic patients should not be advised to exercise. Indeed, diabetic patients should be encouraged to exercise for the same reasons as the non-diabetic population. Our task is to aim at teaching diabetic patients how to reduce or prevent any exerciseassociated complications. And this demands a knowledge and understanding of the current concepts of the physiological and pathophysiological mechanisms involved in the regulation of fuel

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homeostasis during exercise.

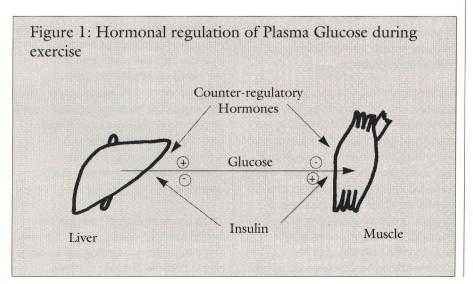
The successful performance of exercise requires a drastic increase of energy and oxygen supply to the working muscle whilst, at the same time, maintaining adequate energy and oxygen supply to the brain and other vital organs.

Effects of acute exercise in the non-diabetic (see Figure 1) During the first 5-10 minutes of exercise, muscle glycogen is the main source of energy, but as exercise continues, glucose and non-esterified fatty acids (NEFA) become increasingly important, and with prolonged exercise, NEFAs become the major fuel.

During the first hour of exercise blood-glucose levels remain virtually unchanged because hepatic glucose production rises to meet the needs of the exercising muscle. With strenuous exercise hepatic glucose

Table 1: Effects of short-term and prolonged exercise on hormones. Short-term Prolonged Insulin N/C or + Catecholamines N/C or + Growth Hormone Glucagon N/C or -Cortisol N/C = unchanged. +=increased. -=decreased.

production may even exceed the rate of glucose-utilisation and blood glucose may increase. 75% of hepatic glucose output comes from glycogenolysis, the rest from gluconeogenesis.



During prolonged exercise, a nondiabetic's insulin secretion declines and the release of counter-regulatory hormones (glucagon, cortisol, growth-hormone, adrenaline and nor-adrenaline) increases. However, despite these protective mechanisms, if exercise lasts for several hours, hepatic glucose production may not keep pace and blood glucose levels decline. Hypoglycaemia may follow 2 to 3 hours of continuous exercise without caloric intake.8 Again, this is in the *normal* subject. Furthermore, if the exercise is preceded by a large sucrose load causing hyperinsulinaemia when the exercise begins, then hypoglycaemia may develop as little as 30 minutes after exercise begins in the non-diabetic.9

Many endocrine changes occur during exercise. (See Table 1).

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Effects of physical training on glucose metabolism

Highly trained athletes have low fasting plasma insulin levels and reduced insulin response to a glucose challenge, suggesting not only hepatic, but *whole-body* sensitivity to insulin. Conversely, even a few days' immobilisation can markedly impair glucose tolerance and insulin sensitivity.¹⁰

Effects of physical training on lipid metabolism

Physical training renders lipid and lipoprotein profiles less atherogenic. Serum HDL cholesterol levels increase while total cholesterol levels remain unchanged or decline. Serum triglyceride levels may also decrease.

Effects of exercise on diabetics

Insulin-dependent diabetics
The metabolic and hormonal response to exercise in IDDM patients is determined by many factors, such as the intensity of the exercise, the patient's level of metabolic control, the type and dose of insulin injected before the exercise, the site of the insulin injection, and the timing of the previous insulin injection, and meal, relative to the exercise.

Accordingly, blood glucose concentrations can decline (most commonly) or increase, or stay unchanged. The major determinant is the availability of insulin, bearing in mind that insulin levels decline in non-diabetics during prolonged exercise.

Hyperinsulinaemia may occur for several reasons. First, short-acting insulin injected a few hours previously may exert its peak action during the exercise. This is exaggerated if the previously injected limb is exercised. During hypoinsulinaemia, the inhibitory effect of insulin on hepatic glucose production and its stimulatory effect on glucose uptake by the muscle are both reduced. In addition, the counter-regulatory response

Prescribing physical exercise as a means of improving metabolic control, is now obsolete

(catecholamines, glucagon, growth hormone and cortisol) to exercise is higher than normal in insulin deficiency, ¹² giving an overall result of hyperglycaemia. Increased lipid mobilisation and ketogenesis in the liver increase blood ketone body concentrations. Thus, the hypoinsulinaemic patient may become ketotic and hyperglycaemic following exercise.

Having said this, one should emphasise that many Type I patients successfully undertake the most strenuous sports like the Comrades Marathon, or Triathlons, provided that both diet and insulin dose are adjusted appropriately before and during exercise. After prolonged exercise, patients may have hypoglycaemic symptoms which may last into the next day due to persistently enhanced glucose uptake

by the exercised muscles to refill glycogen stores.

Non-insulin-dependent diabetics These patients characteristically have both hepatic and peripheral insulin resistance in the fasting state. During acute exercise peripheral glucose uptake rises more than hepatic glucose production and blood glucose levels tend to decline.13 However, at the same time plasma insulin levels fall so that the risk of exercise induced hypoglycaemia in NIDDM patients is small, even during prolonged exercise.14 If NIDDM patients perform strenuous, glycogen-depleting exercise, both peripheral and hepatic insulin sensitivity are increased and remain increased for 12-16 hours after exercise.15

Effects of physical training in diabetics

Insulin-dependent diabetics
Most IDDM patients are insulinresistant. Physical training in these
patients improves whole body insulin
sensitivity as it does in the nondiabetic but will only improve
metabolic control if the training
programme is accompanied by
blood-glucose monitoring and
appropriate changes in diet and
insulin in order to prevent
hypoglycaemia and reactive
hyperglycaemia during and after
exercise.

As in the non-diabetic we get the beneficial lipid changes as well as the same desirable psychological benefits, particularly in diabetic children,

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Table 2: General advice on exercise in diabetics

Total contra-indications:

Patients on insulin:

Scuba-diving, rock-climbing, single-handed sailing, motor-racing, (danger

Patients with proliferative retinopathy or maculopathy: Any strenuous exercise because of the danger of haemorrhage.

Observe caution in patients with:

Cardiovascular disease, especially NIDDMs. Autonomic neuropathy (risk of cardio-respiratory failure). Peripheral neuropathy (risk of injury to insensitive feet).

Generally, diabetics should remember:

- To exercise regularly, daily if possible.
- That strenuous exercise is unnecessary, walking is often sufficiently
- To tailor their exercise schedules to their own individual needs and fitness, and to increase exercise progressively.

including improved feelings of wellbeing, better acceptance (by peers and family), and greater self-esteem.

Non insulin-dependent diabetics Physical training also improves insulin-sensitivity in patients with NIDDM, and the improvement in glucose disposal lasts for months after the training programme ends. Glycaemic control improves, with HbA1 falling by 1-1,5% after six weeks of training.16 I find that participation in exercise programmes assists dietary compliance as well. In patients with high C-peptide reserve (where insulin resistance rather than insulin-depletion is responsible for their diabetes) oral glucose tolerance is also improved.

Then, physical training in NIDDM patients has been reported to produce anti-atherogenic blood lipid changes and to reduce other coronary risk factors (hypertension, obesity, coagulation abnormalities), but not in all studies. 17,18 This prophylactic value of exercise will be greater in younger patients without established atherosclerosis.

Exercise in the prevention of diabetes

Exercise cannot prevent the development of IDDM but regular exercise may prevent or delay the manifestation of NIDDM in patients with a strong genetic predisposition. Insulin-resistance, that important

pathogenetic factor in NIDDM, is significantly less in older athletes than in sedentary subjects, so that older athletes have a lower incidence of NIDDM.19

Recommendations for exercise in diabetics

I encourage my diabetics to participate in just about all sports, with few exceptions. The guidelines (see Table 2) must be tailored to the individual. Poor glycaemic control should be corrected, primarily with diet and insulin or oral agents, and exercise used as an adjunct. Encourage your diabetics to exercise regularly (and for the same reasons as you exercise or encourage your nondiabetics to exercise). Many top professional sportsmen have IDDM, but have learnt to judge their glycaemic fluctuations to varying levels of physical activity during the lead-up training period so that they seldom have unexpected hypoglycaemic episodes when the crunch is on. Thus, the diabetic

Diabetic patients should exercise for the same reasons as non-diabetic patients

training for the Comrades Marathon, who keeps close check on blood glucose levels during his shorter training runs and after, all the way up to the 56km runs just prior to the big day, is likely to complete the ultramarathon without marked bloodglucose variation.

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- * Monitor blood-glucose before, during, and after exercise.
- * Avoid hypoglycaemia during exercise by:
 - starting exercise 1 to 2 hours after a meal
 - taking 20 to 40g extra carbohydrate before and hourly during exercise
 - avoiding heavy exercise during peak insulin action
 - using non-exercise sites for insulin injection
 - reducing pre-exercise insulin doses by 30-50% if necessary.
- * After prolonged exercise, monitor blood-glucose and take extra carbohydrate to avoid delayed hypoglycaemia.

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