Guest Editorial

An apparent anomaly is that a medical curriculum can be considered complete even if it pays only passing reference to the largest organ in the human body, skeletal muscle which can comprise up to 25 kg in the average male. Skeletal muscle, it would seem, lacks the romantic charisma of that far more puny (less than 500g) organ, the heart. Yet the established, indeed revered specialities of cardiology and cardiac surgery and a thriving industry in cardiovascular medicines attest to the far greater importance humans and the medical profession attach to their hearts. Physiotherapists, neurologists and specialists in physical medicine are perhaps the sole medical practitioners for whom disorders of skeletal muscles are of more than passing interest.

But if much of medicine has yet to embrace the significance of such a weighty organ, powerful forces at its fringes will ultimately influence that indifference. Indeed the single most important factor influencing the Cinderella status of skeletal muscle in the medical curriculum has been the growth of the exercise sciences. It is perhaps obvious that those who would study the human in motion should begin to champion the study of the organ of movement.

But even amongst the exercise scientists there are still those who are reluctant to believe the importance of the skeletal muscles in exercise and sport. So a popular view is that exercise performance especially during high intensity exercise of short duration is limited by the capacity of the heart to provide an adequate oxygen supply to the large muscle mass that is activated during this type of exercise. If this is true, the main effect of physical training we are led to believe must be to develop so-called "cardiovascular fitness". Yet there are solid arguments that question the scientific validity of the pioneering studies on which this concept is based.[1]

Furthermore the factor that best identifies successful endurance athletes is the ability specifically of their skeletal muscle to resist the development of fatigue during prolonged exercise.[2] In these athletes it is their muscles, not their hearts, that determine their success.

Then perhaps most challenging is the new paradigm which suggests that the exercise tolerance of persons with advanced heart disease is limited by an associated myopathy (of chronic disease), rather than by the severely impaired function of their diseased hearts.[3] As a result, exercise training which improves skeletal muscle function, can enhance exercise capacity and reduce exercise-related symptoms even in persons with advanced heart disease, even heart failure.[4]

With such advances it is perhaps appropriate that the review section of the first issue of our re-launched Journal should focus attention on skeletal muscle and exercise. The aim has been to review various aspects of skeletal muscle function that will be of interest to the different disciplines that comprise the membership of the South African Sports Medicine Association.

In the article devoted to the basic sciences, Dr Kathryn Myburgh PhD, who has recently completed her post-doctoral studies in the muscle research laboratory of Dr Roger Cooke in San Francisco, carefully reviews the historical development, still less than 50 years old, of the biochemical and structural studies that have laid the foundation of the modern understanding of how muscles contract. She next discusses the different contractile properties including force production and resistance to fatigue of the separate muscle fibres and explains how the different (iso)forms of the contractile proteins can explain some of these functional differences. She concludes that training and other interventions alter not only muscle fibre size, their mitochondrial enzyme content and their substrate utilization patterns but also the mix of the different contractile protein isoforms comprising the different fibres. She wonders whether sporting success might be linked to specific molecular combinations in the different skeletal muscle fibres. Indeed one must ask whether one specific combination of skeletal muscle contractile protein isoforms explains the success of the sprinters of West African origin and another, different combination the exceptional fatigue resistance of the East African distance runners? Clearly if we are to honour our continent and its peoples, those questions must be tackled and answered by African scientists. Perhaps Dr Myburgh's article will have served its purpose if it were to encourage one young scientist to chose a career that will address these questions.

The dynamic disorders of skeletal muscle provide an intriguing insight into the interface between the basic and the clinical sciences. These extremely rare conditions which produce symptoms only during exercise are of interest to clinicians who must consider them in the differential diagnosis of conditions causing exercise-related symptoms. For the basic scientist, patients with these conditions provide a unique model for the study of the biochemical changes causing (premature) fatigue during exercise. The surprising finding is that fatigue in patients with McArdle's syndrome occurs, as expected, without changes in blood lactate levels or blood pH but, more interestingly, without a large reduction in muscle ATP concentrations.

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One explanation for this finding is that skeletal muscle function during exercise is regulated to prevent a reduction in muscle ATP content sufficiently large to produce muscle rigor. This postulate holds that under any condition in which the rate of muscle ATP production is approaching some maximum value, the rate of muscle ATP use is reduced by regulatory processes that reduce the force and frequency of muscle fibre contraction (thereby reducing the rate of ATP use). Thus in this model, fatigue of skeletal muscle during exercise is conceived as a regulatory process which links skeletal muscle contractile function to the rate of ATP production (perhaps through changes in the levels of metabolites of ATP breakdown including Pi and ADP) specifically to prevent the development of muscle rigor.

Another interesting finding is that patients with McArdle's syndrome exhibit an excessive rise in cardiac output, muscle blood flow and ventilation during exercise. This is because of a large fall in peripheral vascular resistance caused by excessive vasodilation in the active skeletal muscles. Metabolic interventions that reduce muscle ADP and Pi concentrations during exercise reduce the vasodilation, the skeletal muscle blood flow and ventilation. Interestingly the metabolic disorder common to all forms of muscle disease is that muscle ADP and Pi levels rise more rapidly than normal during exercise; conversely highly trained muscle is able to resist this change until very high intensities of exercise are reached.

Hence these studies provide intriguing evidence for metabolic factors in skeletal muscle that influence the cardiovascular and respiratory response to exercise and the onset of subjective symptoms of fatigue.

In the section dealing with clinical aspects of skeletal muscle, Dr Mike Lambert and Professor Steven Dennis review perhaps the most common skeletal muscle ailment in the exercising population, what is popular called muscle stiffness but which now enjoys the more elaborate official title of Delayed Onset Muscle Soreness (DOMS).

In this review we learn that DOMS is not due to lactate accumulation and retention in the previously active muscles. This is perhaps one of the commonest misconception in the general sporting population. Rather DOMS is due to tissue damage from which there is a delayed recovery; damage is especially likely after eccentric exercise, a finding well-known to runners in the "down" Comrades Marathon. Each step on the long downhill section from Kloof to the finish in Durban places a large eccentric load on the runner's quadriceps muscles. And the results are very apparent the following day. Readers of this article will now be able to give a detailed scientific explanation to help their runners through their post-race misery.

Practical information provided by these authros is that neither anti-inflammatory medications nor rest make any real difference to the rate of recovery from DOMS. But training reduces the amount of muscle stiffness symptoms during a subsequent bout of similar exercise. This effect may last for weeks to months.

The wish of the authors is that we have been able to convey some of the excitement and enthusiasm we feel in our studies of skeletal muscle in active persons and the relevance that such studies have for the exercise sciences and for clinical sports medicine.

Lastly we would hope that, perhaps, we have been able to convince you that, in exercise and sport, the heart of the matter is skeletal msucle.

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Professor TIM NOAKES
Guest Editor

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August, 1994. 40TH SAOA ANNUAL CONGRESS, PRETORIA. Guest speakers: Dr Allan E Gross, Toronto, Canada; Dr R W Bucholz, University of Texas, USA. Contact: Dr L van Wyk, 404 Muelmed Medical Centre, 577 Pretorius Street, Arcadia, Pretoria, 0083. Tel: (012) 341-7573.

October 3-4, 1994. WORKSHOP ON COMPUTER-BASED EXERCISE FOR TEACHING PHYSIOLOGY, AND:

October 5-7, 1994. 22ND ANNUAL CONGRESS ON THE PHYSIOLOGICAL SOCIETY OF SOUTHERN AFRICA, STELLENBOSCH. For further details please contact: Dr A W van Rijswijk, Department of Human and Animal Physiology, University of Stellenbosch, 7600.

October 5-7, 1994. SCIENTIFIC CONGRESS OF SOUTH AFRICAN ASSOCIATION FOR LABORATORY ANIMAL SCIENCE (SAALAS), JOHANNESBURG. The Transnet Conference Centre, Esselen Park near Jan Smuts Airport. Contact: Miss Monique de Villiers, National Institute for Virology, Private Bag X4, Sandringham, 2131. Tel: (011) 882-9910. Fax (011) 882-0596.

November 27-30, 1994. THE SOUTHERN AFRICA CARDIAC SOCIETY 19TH BIENNIAL CONGRESS. Education Building, University of Cape Town. Contact: Mrs Sally Elliott, Postgraduate Medical Centre, UCT Med School, Observatory, 7925. Tel: (021) 406-6381. Fax: (021) 448-6263.

March 22-24, 1995. SIXTH SOUTH AFRICAN SPORTS MEDICINE ASSOCIATION CONGRESS, ELANGENI HOTEL, DURBAN. Contact: Mrs Miriam Tennant, Sports Medicine, UCT Medical School, Observatory, 7925. Tel: (021) 406-6504. Fax: (021) 47-7669.

INTERNATIONAL

August 10-14, 1994. COMMONWEALTH GAMES 1994, INTERNATIONAL SCIENTIFIC CONGRESS, UNIVERSITY OF VICTORIA, BC, CANADA. Contact: Commonwealth Games International Science Congress, Conference Services, Division of University Extension, University of Victoria, PO Box 3030, Victoria BC, Canada V8W 3N6. Fax: 604 721 8774.

August 17-19, 1994. 7TH INTERNATIONAL CONGRESS ON OBESITY — SATELLITE SYMPOSIUM, QUEBEC, CANADA. Contact: Exercise and Obesity Satellite, c/-Angelo Tremblay, Physical Activity Sciences Laboratory, PEPS, University Laval, Ste-Foy, Quebec, Canada G1K 7P4. Tel: 418 656 7294. Fax: 418 656 3020.

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The Dynamic Disorders of Skeletal Muscle Metabolism with special reference to Exercise

TD Noakes MB ChB MD FACSM

Introduction

Most of the skeletal muscle disorders encountered in clinical medicine are of a sufficiently severe nature that they cause such weakness that participation in physical activity is not possible. These *degenerative or myopathic disorders* cause progressive muscle weakness and atrophy usually leading to death at a relatively young age.

However there are a group of skeletal muscle disorders which cause symptoms only during exercise. These are known as the dynamic muscle disorders and are compatible with an unaltered life expectancy. They are of special interest to sports scientists as the biochemical abnormalities present in these conditions allow insights into metabolic factors in skeletal muscle which influence cardiovascular function during exercise, and to sports physicians because these conditions need to be considered in the differential diagnosis of exercise-induced syndromes. In addition, the clinical syndromes of malignant hyperpyrexia, exercise-induced heatstroke and rhabdomyolysis which acute renal failure are probably inter-related and may be caused by one or more of the dynamic disorders of skeletal muscle metabolism, perhaps as yet undefined.

This review will emphasize especially the dynamic skeletal muscle disorders that cause symptoms during exercise. No attention is paid to the degenerative skeletal muscle disorders that cause symptoms at rest. These conditions are well described in conventional medical texts and are of no special interest to sports practitioners. It should be emphasized that the dynamic disorders of skeletal muscle metabolism are *exceedingly* uncommon. The intellectual interest they evoke is quite out of proportion to their clinical importance.

Clinical presentation

The dynamic disorders of skeletal muscle metabolism are characterized by the following features:

First, there are no symptoms at rest. In particular, muscle weakness, a characteristic feature of the atrophic and destructive myopathies, is absent.

Second, the diagnostic feature is the early onset of fatigue — easy fatigueability — during exercise, even when the exercise is of a mild intensity.

Third, the cardiorespiratory response to exercise is abnormal with an elevated heart rate, cardiac output and minute ventilation.

Fourth, there may be rhabdomyolysis with myoglobinuria. If severe, this may lead to acute renal failure.

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Classification

There are four subgroups in this category:

— Defects of carbohydrate metabolism, the classic form of which is McArdle's syndrome.
— Defects of lipid metabolism.
— Defects of mitochondrial function.
— Defects of adenine nucleotide metabolism.

MCARDLE'S SYNDROME

In this condition which has its onset early in life, either late childhood or early adulthood, the patient has severe exercise intolerance (McArdle, 1951). There is the onset of muscle pain and contractures during more vigorous exercise. The contractures are electrically silent unlike nocturnal cramps in which there is increased electrical activity. If the exercise is continued, the contractures may lead to ischaemic damage of the muscle leading to myoglobinuria with possible renal damage. In less severe cases, there may also be muscle swelling and soreness, followed after exercise by elevated serum enzyme activities and myoglobinuria, on occasion.

But if the exercise is continued at low intensity, the patient experiences a "second wind" in which the pain diminishes so that he or she is able to continue exercising albeit at a relative low exercise intensity. Factors that expedite the onset of the second wind include exercising in warm weather and the avoidance of a high carbohydrate meal shortly before exercise. Factors that aggravate the symptoms are obviously the reverse — exercising in the cold and eating a high carbohydrate meal shortly before exercise. Carbohydrate ingestion causes serum-free fatty acid concentrations to fall thereby limiting the availability of this alternate fuel for the exercising muscles. Factors which reduce skeletal muscle blood flow also delay or prevent the onset of the second wind.

The biochemical hallmark of the condition is the failure of the exercising muscles to produce appropriate amounts of lactate during vigorous or ischaemic exercise. McArdle (1951) recognized the similarity between this clinical picture and the development of con-

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tractures without lactate production by muscles poisoned with the glycolytic inhibitor iodoacetate. He concluded that patients with this syndrome must be suffering from an enzymatic defect in the glycogenolytic or glycolytic pathways. Indeed, the absence of one or more of the following enzymes have been described in patients with the symptom complex described by McArdle: glycogen phosphorylase, glycogen phosphorylase b kinase, phosphofructokinase, phosphoglycerate kinase, phosphoglycerate mutase and lactate dehydrogenase.

All these enzyme defects reduce the ability of the cell to produce ATP by oxygen-independent pathways. This is confirmed by the low levels of glycolytic intermediates and pyruvate measured by muscle biopsy during exercise in these patients (Kono et al 1984; Wahren et al 1973). In addition, there is an inability to produce pyruvate at a sufficiently fast rate to fuel oxidative metabolism (Lewis and Haller, 1986). Patients with glycogen phosphorylase deficiency are able to oxidize glucose and free fatty acids for energy whereas those with enzyme deficiencies below phosphorylase can oxidize only free fatty acids as interruption of the glycolytic pathway prevents the complete metabolism of glucose — > pyruvate — > acetyl CoA for further oxidation in the Krebs cycle. Thus whereas the provision of glucose by infusion will increase the exercise tolerance of patients with a pure phosphorylase deficiency, such infusions will have no effect on those with enzyme defects in the glycolytic pathway.

The result of this inability to supply adequate substrate to the Krebs cycle for oxidative metabolism is that, during exercise, there is an abnormally large decrease in muscle PCr concentrations and muscle pH and a large increase in muscle Pi concentrations.

When stimulated electrically, the muscles of patients with this syndrome also lose their action potentials, possibly suggesting that glycolytically-produced ATP may be required for maintaining the excitability of membranes especially the sarcolemma and T-Tubules.

Disturbances of oxygen transport and cardiac function in patients with McArdle's syndrome

Oxygen transport and utilization

McArdle's syndrome had been used as a model to study the metabolic factors in the active skeletal muscle that influence cardiovascular function (Lewis and Haller, 1986; Lewis et al 1991).

It is found that the maximum oxygen consumption ($VO_{2\,max}$) of patients with McArdle's syndrome is about 18 ml O_2 Kg^{-1} min^{-1} or roughly half that found in normal patients. The reduced $VO_{2\,max}$ is not due to reduced muscle mass and is thus distinct from the low $VO_{2\,max}$ observed in patients with primary muscle diseases such as muscular dystrophy in which there is muscle wasting.

The low $VO_{2\,max}$ of patients with McArdle's syndrome has been related to the abnormally slow rate of substrate flux through the Krebs cycle and the electron transport chain. The inability to fuel the Krebs cycle with pyruvate causes the Krebs cycle to become "run down". Thus the rate of formation of mitochondrial reducing equivalents (NADH and FADH2) will ultimately limit the rates of oxidative phosphorylation and therefore the $VO_{2\,max}$.

Although whole body VO_2 is normal at rest in these patients, at any given workload total body VO_2 is *greater* than normal. This is explained by an increased oxygen consumption of the cardiac and respiratory muscles due to an excessive tachycardia and elevation of systolic blood pressure and a greater than normal pulmonary ventilation. Respiratory exchange ratios during exercise are also abnormally low indicating an increased reliance on fat as the major metabolic fuel for exercise.

The $VO_{2\,max}$ of patients with McArdle's syndrome is increased by intravenous glucose or lactate infusions. Even then, blood lactate concentrations do not increase indicating that a major reason for the reduced work capacity of these patients is a lack of oxidizable substrate, especially pyruvate, to fuel the Krebs cycle. This also indicates that blood glucose cannot fully substitute for muscle glycogen as an oxidative substrate in short-term heavy exercise.

Similarly an elevation of serum-free fatty acid concentrations also increases $VO_{2\,max}$ by the order of 18-25%. Thus a limitation in the availability of free fatty acids also contributes to the low $VO_{2\,max}$ of these patients.

Cardiovascular function

Patients with McArdle's syndrome show an hyperkinetic circulatory response to exercise. This hyperkinetic circulatory response is not due to cardiac or vascular disease; cardiac muscle is spared in McArdle's disease because of the presence of cardiac specific glycogen phosphorylase isoenzymes. There is also no evidence for abnormal glycogen phosphorylase activity in vascular smooth muscle.

The elevated cardiac output during submaximal exercise is believed to result from excessive vasodilatation in the active muscles possibly related to the large fall in intramuscular PCr and the excessive rise in Pi concentrations. This hyperkinetic circulation is partially normalized during the infusion of free fatty acids or during exercise when serum-free fatty acid concentrations are elevated. This finding links mechanisms regulating systemic haemodynamics and local vasodilatation with the availability of oxidizable substate to the contracting muscles. Hence it is not only the availability of oxygen that controls peripheral vascular function.

Glucose infusion simultaneously decreases both the cardiac output and the extent of the decline in PCr and the rise in Pi concentrations during exercise in these patients. This indicates that there is probably a reciprocal relationship between skeletal muscle blood flow and cellular ATP/ADP.Pi ratios (the phosphorylation potential). This suggests that the crucial metabolic consequence of McArdle's disease is an abnormally large decline in this ratio in muscle during exercise.

The exact vasodilator substances acting in McArdle's syndrome are not known but increased concentrations of lactate and hydrogen ions, or a low arterial PO_2 or increased PCO_2 have all been excluded as none is dif-

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ferent from values measued in normal persons. Thus the likely vasodilators are phosphate, potassium and adenosine, the latter derived from AMP. Of these, intramuscular phosphate concentrations are known to increase steeply in these patients during exercise and there is also increased potassium release by muscle. Either or both could be important vasodilators in this disease. Alternatively, these metabolites may stimulate skeletal muscle (Group III and IV) afferents which then induce the excessive cardiovascular response during activity (Lewis et al 1991).

In contrast, during static exercise, patients with McArdle's syndrome do not show the expected increase in muscle sympathetic nerve activity. Thus, in normal muscle this reflex is probably activated by increased rates of glycogenolysis with a fall in muscle pH (Pryor et al 1990).

Patients with McArdle's syndrome also show an abnormal increase in ammonia production by muscle, further indicating increased AMP production with consequent deamination to ammonia and IMP. Glucose infusion also reduces the production of ammonia; thus a reduction of ammonia production may also be a factor reducing the abnormal cardiac output in these patients.

Biochemical features of fatigue in McArdle's syndrome

The outstanding observation in these patients is that despite the limited capacity of phosphorylase deficient muscle to resynthesize ATP, little or no decline in muscle ATP concentrations has been observed during fatigueing exercise in McArdle's patients. However there is an abnormally large accumulation of Pi and probably of ADP and AMP and these could contribute to fatigue by inhibition of intracellular processes involved in muscle contraction. Glucose infusion reduces the extent to which Pi concentrations rise and increases resistance to fatigue. This indicates a metabolic shift from the creatine kinase (Equation 1, below) and myokinase-AMP deaminase coupled reactions (Equation 2, below) in which there is net accumulation of phosphate, to those of oxidative phosphorylation and glycolysis in which both ADP and Pi are used to resynthesize ATP (Lewis and Haller, 1986).

Equation 1

The creatine kinase reaction

$$ATP \rightarrow ADP + Pi$$
$$ADP + PCr \rightarrow ATP + Cr$$
$$\text{net: } PCr \rightarrow Pi + Cr$$

Equation 2

The myokinase-AMP deaminase reaction

$$2ATP \rightarrow 2ADP + 2Pi$$
$$2ADP \rightarrow AMP + ATP$$
$$AMP \rightarrow NH_3 + IMP$$
$$\text{net: } ATP \rightarrow 2Pi + NH_3 + IMP$$

Thus the abnormally large rises in intramuscular Pi concentrations during exercise in these patients indicates their reliance on the creatine kinase and the myokinase-AMP deaminase coupled reactions to regenerate ATP (Equations 1 and 2) rather than on oxidative metabolism. The detrimental result is the accumulation of increased intracellular Pi concentrations which impair muscle contraction and cause fatigue.

There is also evidence that the rate of ATP turnover is reduced during fatigue in McArdle's patients indicating product inhibition of the rate of ATP hydrolysis. This mechanism is thought to explain exercise-induced fatigue also in healthy persons.

A decline in skeletal muscle excitation shown by a fall in the muscle action potential is also found in these patients. This could be the result of product inhibition of the membrane Na^+/K^+ ATPase reaction which can be relieved by the removal of phosphate via glycogenolysis in normal but not in phosphorylase deficient muscle.

Lewis and Haller (1986) conclude that events which limit ATP synthesis in McArdle's patients also limit ATP hydrolysis, thereby slowing ATP turnover and preventing muscle rigor from developing, analogous to the situation that develops in normal muscle during exhaustive, maximal exercise.

Thus the biochemical basis for fatigue in skeletal muscle in both normal and diseased muscle may be quite similar.

There are two other interesting features of this condition. First, patients with McArdle's syndrome show a "ventilation turnpoint" during exercise indicating that this phenomenon, also known as the "anaerobic threshold", has no relationship to lactate production by the skeletal muscles.

Second, patients with McArdle's syndrome also show a post-exercise oxygen debt (excess post-exercise oxygen consumption — EPOC). Traditional theories have suggested that the EPOC is due to an excess oxygen consumption required during the post-exercise recovery period to oxidize the lactate produced during the preceding exercise. The presence of this phenomenon in these patients indicates that this cannot be the correct explanation.

Lactate transporter defect

Fishbein (1986) has recently described the first case of an intriguing and possibly not uncommon muscle disorder in which there is an absence of the lactate transporter in both skeletal muscle and red blood cells. The lactate transporter is believed to be a membrane protein that co-transports a lactate anion and a proton externally without a requirement for ATP. The transporter is thought to be responsible for 90% of lactate efflux from these cells. Its activity is sensitive to the pH gradient across the cell membrane and increases markedly as the intracellular pH falls (Fishbein, 1986).

The patient described by Fishbein was a 26 year old military drill instructor who was investigated for chest pain and coincidentally found to have markedly elevated serum CK activities (up to 13,700 IU — normal up to 200 IU) on several occasions. Exercise tests revealed that the patient showed elevated blood lactate concentrations after exercise and was therefore not suffering from McArdle's syndrome. However the rate at which his blood lactate concentrations returned to the normal resting levels after exercise was profoundly delayed. Laboratory studies confirmed that the rate of lactate

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release from the patient's red blood cells was also severely abnormal.

Whilst the patient had no symptoms directly attributable to this condition, Fishbein suggests that extreme exercise in such patients could lead to rhabdomyolysis due to their inability to decrease intracellular acidosis sufficiently rapidly during and after exercise. The condition may also explain the frequently unexplained finding of chronically elevated serum CK activities and attacks of rhabdomyolysis in some apparently healthy individuals.

DEFECTS OF LIPID METABOLISM

The classic disorder of lipid metabolism is that due to the absence of the enzyme carnitine palmityltransferase (CPT) in skeletal muscle. This enzyme is essential for the transport of long chain fatty acids across the inner mitochondrial membrane for their further metabolism by beta-oxidation with the production of acetylcoA which enters the Krebs cycle. To date there have been approximately 25 subjects with this condition reported in the literature. Two have been females. The inheritance of the condition is autosomal recessive (Angelini et al 1981).

A related condition is muscle carnitine deficiency. Carnitine is also essential for the transport of fatty acids across the mitochondrial membrane. Carnitine is produced in the liver and transported to muscle via blood from where it is actively absorbed. Carnitine deficiency may therefore result from the failure of carnitine synthesis by the liver or be due to the failure of carnitine uptake by the muscles. Treatment with carnitine helps those patients who do not produce carnitine.

Muscle CPT exists in two different forms on the inner (CPT-II) and outer (CPT-I) mitochondrial membranes. It is a deficiency of CPT-II which causes this syndrome (Trevisan et al 1987).

Symptoms of CPT deficiency usually begin late in childhood or in early adulthood. As in McArdle's syndrome, there are no abnormalities at rest but fasting may cause serum CK activities to rise markedly (Carroll et al 1979).

Patients are usually able to perform vigorous exercise without complications and their $VO_{2\ max}$ values and cardiovascular function during exercise are normal (Lewis et al 1991). This finding indicates the importance of normal carbohydrate metabolism for maximum exercise and for the normal cardiovascular response to exercise.

However muscle tenderness, pain and swelling may occur after very prolonged exercise. Cramps may also be a feature of the condition (Bye and Kan, 1988). More generalized muscle swelling may occur when exercise follows a bout of prolonged fasting or after a period on a low carbohydrate, high fat diet. Muscle pain and swelling are accompanied by very marked elevations in serum enzyme activities and in myoglobinuria. There may also be acute renal failure.

Patients with this condition are dependent on carbohydrate to provide their energy requirements during exercise. Thus they burn carbohydrates more rapidly than usual during exercise and may develop rhabdomyolysis when muscle glycogen depletion occurs. Prevention of complications is therefore through the prescription of a high carbohydrate diet to maintain muscle and liver glycogen concentrations.

The condition can be diagnosed with muscle biopsy which confirms the absence of CPT activity. Lipid-filled vacuoles may be shown predominantly in Type 1 fibres; they are especially marked in the form of this condition which results from carnitine deficiency (Bye and Kan, 1988).

DEFECTS OF MITOCHONDRIAL FUNCTION

Syndromes associated with excessive lactate formation

In these patients there are deficiencies in one or more of the enzymes of the electron transport chain; in some patients no defect has yet been identified. The effect of these abnormalities is to reduce the capacity for oxidative ATP production and therefore to increase reliance on glycolytic ATP production with increased rates of lactate and pyruvate production and the associated severe metabolic acidosis.

The clinical features common to all these conditions are extreme exercise intolerance associated with early fatigue, muscle weakness and dyspnoea. The patients respond to acute exercise as if they were extremely unfit. Some patients have high resting blood lactate concentrations; however in all, there is a steep increase in blood lactate concentrations at very low levels of exercise.

In common with patients with McArdle's syndrome these patients also show very low $VO_{2\ max}$ values and an abnormal cardiovascular response to mild exercise including a disproportionate cardiac output, tachycardia and ventilation for the appropriate metabolic rate (Haller et al 1989). The metabolic basis for this is presumably the same as that in McArdle's syndrome, that is increased intramuscular Pi concentrations due to an abnormal reliance on the creatine kinase and myokinase-AMP deaminase coupled reactions to regenerate ATP in muscles which have an impaired capacity to produce ATP by oxidative metabolism.

The inheritance of these conditions is unique because the mitochondrial DNA, which codes for the 13 protein components of the electron transport chain, is transmitted exclusively by the mother (Jones and Round, 1990). This suggests that the mother may have an inordinate influence on the child's ability to produce ATP by oxidative pathways (and therefore to become a champion athlete?).

DEFECTS OF ADENINE NUCLEOTIDE METABOLISM

Myoadenylate deamine deficiency (MD)

The enzyme myoadenylate deaminase (MD) hydrolyzes the breakdown of AMP (produced by the myokinase reaction) to ammonia and IMP (Equation 2). Absence of this enzyme is one of the commonest of the known muscle enzyme defects and exists in two forms — the primary type which is inherited as a complete gene block in an autosomal recessive pattern and the secondary type which results from muscle damage due to other diseases (Fishbein, 1985).

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The enzyme defect is believed by some to explain cramping and exercise intolerance in some individuals, usually in adult to middle-age (Fishbein, 1985). Larger studies, however, have failed to shown any association between symptoms and this enzyme defect (Mercelis et al 1987). It may be that this is an enzyme defect still searching for a disease!

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Muscle Proteins and the Contractile Properties of Muscle Fibres

Kathryn H Myburgh PhD

Introduction

Muscles are machines which provide the power that is the basis for all vertebrate movement. The mechanical properties of an active muscle are dependent on many factors such as the shape and size of the whole muscle, its fibre type, fibre size and length, and the angle of the fibre-tendon attachment, as well as a variety of neural factors including cortical drive and motor unit recruitment patterns. However, the force originates at the molecular level from the interaction of the major muscle proteins, actin and myosin, in the presence of ATP and Ca^{++}. This review will outline some of the major discoveries which led to the theories of the mechanism of force production at this level in skeletal muscle, including the roles played by ATP and Ca^{++}, and the relationship between force and velocity. It will then discuss factors within a single muscle fibre which influence these contractile properties and which are of particular relevance to exercise scientists.

I. The mechanism of force production

Our current understanding of the mechanism of force production is based in large part on 3 discoveries made in the 1950's. At that time the band-like structure of muscle was already well described even at the level of the sarcomere, but the function of the bands was not known. Two important discoveries linking the structure of the sarcomere to its function were (i) that the pattern of the bands is caused by thick and thin filaments[1] and (ii) that in those bands where the two types of filaments overlap, they also interact at regular intervals by means of bridges.[2] The third discovery, which was made at the same time by two different investigators and was published in the same edition of the prestigious journal *Nature*, was that the filaments themselves do not shorten during contraction. Instead, they appear to slide between one another so that the amount by which they overlap increases during active shortening in a contracting muscle.[3,4] These results led to the formulation of the sliding filament theory of cross-bridge interaction during muscle contraction.[5] Huxley proposed that force was produced by each projection from the myosin thick filament as it attached to the actin filament and that the force was related to the kinetic energy available in the cross-bridge before attachment.[5] More than a decade later following many more mechanical experiments performed at faster time resolutions and employing very accurate, quick changes in tension[6] or length,[7] Huxley and Simmons[8] formulated a second proposal for the mechanism of force generation by the actomyosin cross-bridge. In the second model, it was proposed that force is generated shortly *after* attachment as the cross-bridge changes to a more stable configuration (see Fig. 1: 3a and 3b). This hypothesis requires that the attached cross-bridges exist in two or more configurations (see Fig. 1: 1, 3a and 3b) and that the head can rotate from a 90° angle to a 45° angle (Fig. 1: 3a to 3b). Indeed, a 45° orientation of the cross-bridges had already been shown in electron micrographs of rigor muscle,[9] and there was no reason to believe that the cross-bridge could not rotate through that orientation during a contractile cycle.

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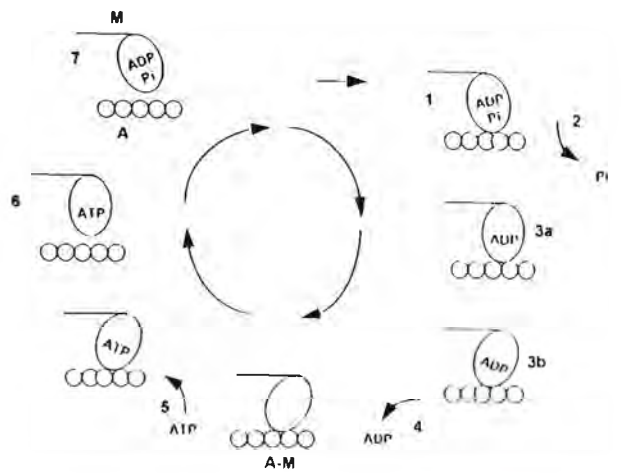

Figure 1. *A schematic representation of ATP hydrolysis, release of the products of ATP hydrolysis and actomyosin interaction during the contractile cycle. Abbreviations: A = actin; M = myosin; A-M = actomyosin complex; ATP = adenosine triphosphate; ADP = adenosine diphosphate; Pi = inorganic phosphate. Steps: 1 = cross-bridge attachment; 2 = phosphate release; 3a and 3b = configurational changes; 4 = ADP release; 5 = ATP binding; 6 = cross-bridge detachment; 7 = ATP hydrolysis.*

II. The role of ATP in the cross-bridge cycle

While physiologists and physicists were piecing together the structural and mechanical data, biochemists were actively investigating the kinetics of ATP hydrolysis by an ATPase enzyme which is part of the myosin molecule. It had already been discovered in 1939 that the major muscle protein complex, actin-myosin, (also termed actomyosin), catalysed the hydrolysis of ATP.[10] It was not until 1971 that a scheme was presented by Lymn and Taylor[11] which integrated the steps in the chemical splitting of ATP to ADP and Pi, with a cyclical attachment and detachment of actin and myosin.

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In short, ATP hydrolysis occurs before myosin attaches to actin (Fig. 1: 7), this is followed by attachment and formation of the actomyosin-ADP-Pi complex (Fig. 1: 1). After release of the hydrolysis products, Pi and ADP (Fig. 1: 2 and 4), actin and myosin remain associated (Fig. 1: A-M), probably with the cross-bridge head at an angle of 45°. Actin and myosin dissociate with the binding of ATP to myosin (Fig. 1: 5 and 6), and once again, ATP hydrolysis occurs in the detached state (Fig. 1: 7). It only became apparent later that Pi is released in a separate step before the release of ADP[12] and that the actomyosin-ADP-Pi complex (Fig. 1: 1) is relatively weak and produces less force than the subsequent strong-binding actomyosin-ADP complex (Fig. 1: 3b).[13]

III. The role of Ca^{++} in the cross-bridge cycle

Biochemical studies of actomyosin-ATP hydrolysis done in solution have highlighted different properties of the myosin-ATPase enzyme. For example, unlike whole muscle which requires both ATP and Ca^{++} to contract, a mixture of purified myosin and purified actin can hydrolyse ATP without the presence of Ca^{++} in the solution.[14] This indicated the presence of an additional protein, or proteins, that could inhibit the cross-bridge cycle unless calcium was present to remove that inhibitory action. These proteins were identified as tropomyosin and the troponin-complex[15,16] and became known as regulatory proteins. A very important early discovery which made it possible to define the role of Ca^{++} on actual force production in the cross-bridge cycle, was the development of the so-called "skinned" fibre. By incubating a muscle fibre in a solution containing 50% glycerol, the membrane is made permeable to ions, small molecules and even larger molecules.[17] The initiation of contraction in the skinned fibre is no longer dependent on membrane depolarisation, but is under control of the investigator, and is achieved by the addition of calcium to the solution bathing the fibre. It was later discovered that the level of force production, measured by a force transducer to which one end of the skinned fibre is attached, could be varied by varying the concentration of free calcium.[18] The characteristic relationship between force and calcium ion concentration (see Fig. 2) shows that force is negligible at pCa 6 ($=10^{-6}$ M Ca^{++}) and maximal around pCa 4 ($=10^{-4}$ M Ca^{++}). The sigmoidal shape of the curve indicates that few cross-bridges attach initially, but that there is a large cooperative action between adjacent myosin-binding sites on actin, so that force increases steeply from approximately pCa 5.5.

IV. The force-velocity relationship

When a skinned fibre is attached to a force transducer on one side and a very rapid servo-controlled motor arm on the other, it is possible to vary the tension in the fibre by varing the position of the motor arm and hence the length of the fibre. The shortening velocity can be determined at specific fractions of the maximum tension by several load clamps in which the tension in the fibre is released to specified levels of submaximal tension and the velocity of tension redevelopment is measured. The data can be fitted to the Hill equation[19] and the maximal velocity of shortening (Vmax) is calculated

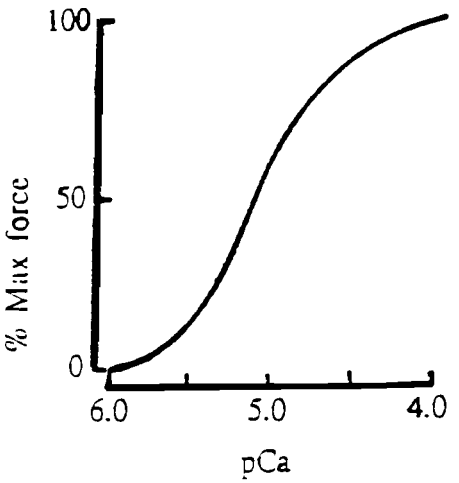

Figure 2. *The characteristic relationship between force and calcium ion concentration. Abbreviation: pCa = negative log of calcium concentration in moles e.g. pCa 6 = 10^{-6} M Ca^{++}.*

from an extrapolation of the data to 0 load.[20] A characteristic hyperbolic curve is obtained (see Fig. 3) showing how the velocity of contraction is maximal when the load (P) is equal to 0, and slows down as P increases until the velocity is 0 at maximal load (Po). At this point, no shortening occurs and Po is analogous to maximum isometric force.

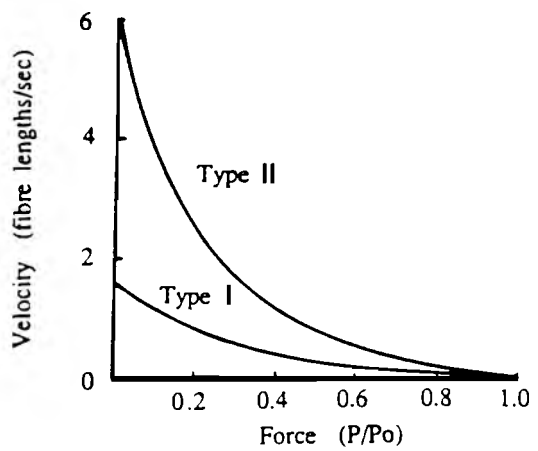

Figure 3. *The characteristic relationship between force and velocity of contraction. Abbreviations: P = force; Po = maximum force.*

V. The effect of muscle fibre type on the mechanics of contraction

V.i The characteristics of the basic fibre types

The basic characteristics of the contractile cycle described above apply to all muscles with the banded sarcomere structure. But, in 1873 the French physiologist,

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Ranvier, noted that red and white skeletal muscles have different contractile properties: red muscles contract slowly, while white muscles contract and relax rapidly.[21] This apparently simple observation set the stage for numerous physiological, biochemical and histological studies which aimed to define the properties of different mucles and muscle fibre types.

In mammals, whole muscles are generally composed of various fibre types. This heterogeneity complicates research studies, but allows for a great deal of physiological variation. Fortunately, in smaller mammals such as mice, rats and cats, the soleus is composed almost entirely of red muscle fibres and the extensor digitorum longus of white fibres. This made it quite simple for electrophysiological studies to determine clear differences betweent those muscle groups in the properties of a single twitch contraction and led to general acceptance of the terms slow-twitch and fast-twitch fibres.[22] At around the same time biochemical studies showed that the myosin from fast-twitch fibres hydrolysed ATP much faster than did the myosin from slow-twitch muscle[23] and histochemists were able to differentiate between different myosin ATPase types (isozymes) due to their differing susceptibility to losing their enzymatic activity at different pH's.[24] (See Table I). Another approach was to differentiate between the fibre types by determining either their resistance to fatigue during imposed stimulation,[25] the number of mitochondria present and the oxidative enzyme capacity,[26,27] or the glycogen content.[28] In these ways it was made possible to draw up a table of *basic* fibre type classifications (see Table I).

Interestingly, it was found that all the muscle fibres activated by the same motor unit fit into the same histochemically determined classification of fibre type.[25] However, the following question arises: can the contractile characteristics of each fibre type be ascribed to a combination of the neural activation process and the metabolic characteristics, or do the contractile proteins themselves also exert an independent influence? The single skinned muscle fibre is the perfect experimental model to answer this question since, as mentioned before, the activation is controlled by the investigator. The following paragraphs will consider whether fibre type affects peak isometric force production, velocity of contraction and the force-pCa curve irrespective of neural control.

TABLE I
Basic Skeletal muscle fibre type classifications

Skeletal Muscle Fibre Types			
Nomenclature	Type I slow oxidative SO	IIA fast oxidative-glycolytic FOG	IIB fast glycolytic FG
Characteristics:			
Colour	red	red	white
Twitch	slow	fast	fast
ATPase activity	low	high	high
lost at pH	9.4-10.4	4.4-4.6	4.4
Glycogen content	low	high	high
Fatigue resistance	high	high	high
Oxidative capacity	high	high	low

V.ii Contractile properties of the basic fibre types

Early experiments on a large range of muscle types and sizes showed that the maximal force was related to the cross-sectional area of the muscle.[29] Fibre diameters of single muscles fibres can vary up to 10-fold. Therefore, measurements of force or tension must be corrected for cross-sectional area, before differences inherent in the muscle proteins can be discovered. Early reports found that there were no differences in the maximal force (Po) when expressed relative to the cross-sectional area of slow- or fast-twitch muscles[30] or fibres dissected from purely slow-, or purely fast-twitch muscles[31] (see Fig. 3: both Type I and II have the same Po). In skinned fibres the data on maximal values of force per unit of cross-sectional area in different fibre types are not consistent and range from indicating no significant difference,[32] to higher values in slow-twitch fibres,[33] or higher values in fast-twitch fibres.[34] Data from human biopsy samples were in agreement with the latter findings.[35] These discrepancies are most likely due to difficulties in the measurement of the cross-sectional areas of single fibres. The fibre diameter is not always consistent along the length of a fibre which has been attached to a force transducer and a small error in the measurement of diameter is magnified in the calculation of cross-sectional area. However, further technical advances have reopened this question and it is currently under investigation in several laboratories.

Although the issue of maximal force production by different fibre types is not resolved, it is currently accepted that there are most likely no differences between fibre types of healthy muscle and this observation is explained by assuming that the number of cross-bridges per unit area of muscle is consistent[36] and that the force per cross-bridge is the same. As mentioned in section I, cross-bridges can be in either a weak, low force (actomyosin-ADP-Pi) (see Fig. 1: 1) or a strong, high force (actomyosin-ADP) state (see Fig. 1: 3b). The relative proportion of each state could therefore also influence force production. Recent mechanical data suggest that during a maximal isometric contraction in a previously rested muscle, the majority of the cross-bridges are in the strong-binding state.[37] But when muscle is fatigued and the intracellular Pi concentration is high, more cross-bridges are in the weak-binding state and force can be reduced by as much as 30%.[20]

Although peak isometric force is apparently similar in both fibre types, the force developed at submaximal Ca^{++} concentrations is different for fast- and slow-twitch fibres of the rat.[33,38] Slow-twitch fibres could produce low levels of force at significantly lower concentrations of Ca^{++} than fast-twitch fibres. However, a steeper slope of the force-pCa curve in fast-twitch fibres indicate that once the Ca^{++} concentration threshold for force development has been reached the fast-twitch fibres develop peak force at a faster rate. These observations can be attributed to differences in the calcium sensitivity of different isoforms of the regulating pro-

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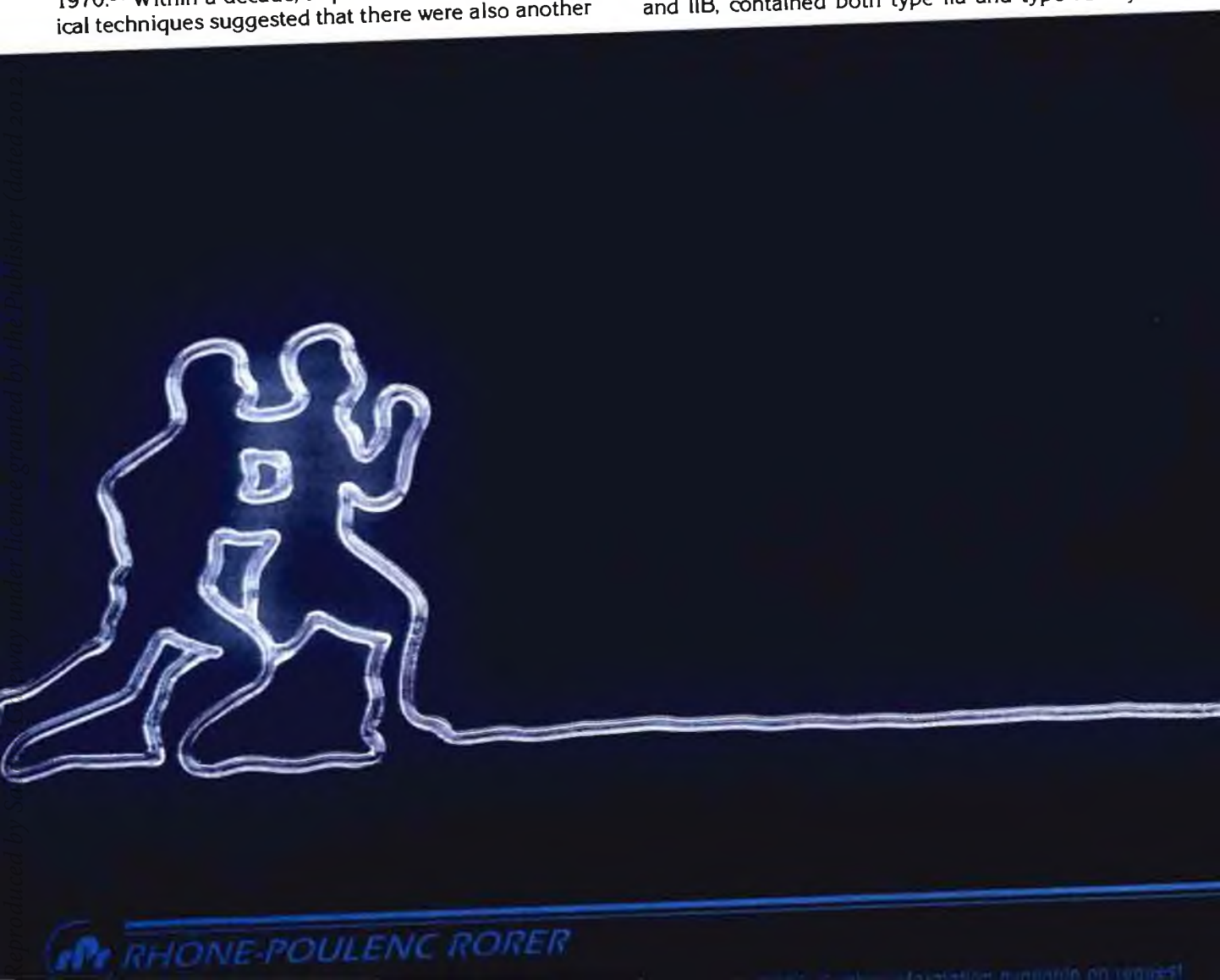
tein, troponin.[38,39] It is interesting to note that the myosin and troponin isoforms change after complete inactivity and weightlessness induced by hind limb suspension in a rat model and revert to faster isoforms.[40] This indicates that even adult muscle proteins are continuously regulated to adapt to environmental changes.

As mentioned before, the broad division of fibres into fast- and slow-twitch types is based on their speed of contraction. Maximal shortening velocity (Vmax) can be as much as 6-fold higher in fast glycolytic fibres compared with slow oxidative fibres, while the fast, oxidative fibres have a Vmax only 3-fold higher (see Fig. 3).[33] In addition, the shape of the force-velocity curve is more concave for slow-twitch fibres than fast-twitch fibres indicating that even at a specific fraction of the maximal force, the velocity is slower in the slow-twitch fibres.[34] Since the fibres are already fully activated, these differences are unrelated to Ca^{++} concentration or Ca^{++} sensitivity and can therefore be ascribed to the contractile proteins themselves.

V.iii Further division of fibre type based on contractile protein subunits

Even though the classification of fibre types in Table I is not simple, for a number of reasons there are not sufficient categories to take into account all the differences that are now apparent between muscle fibres. For instance, a type IIC fibre, which can be placed between type IIA and IIB in characteristics was identified in 1970.[24] Within a decade, improvements in histochemical techniques suggested that there were also another two distinct subtypes of fast-twitch fibres named IIAC and IIAB.[41] Also, comparisons of biochemical and histochemical classification has shown that types IIA and IIB fibres do not always correspond to the FOG and FG classification,[42] further suggesting that the basic fibre classification scheme in Table I is too simplistic.

This diversity is perhaps not surprising, when one considers that the myosin molecule is a large, complex molecule with several subunits, any of which may occur in slightly different forms (isoforms). Two heavy polypeptide chains form the thick filament or backbone of myosin and also extend into the neck region and the globular heads which together form the cross-bridges between the myosin backbone and the actin filament.[43] The portions of the two heavy chains which make up the head region contain the myosin ATPase enzyme subunit. The two heads each also contain two different light polypeptide chains, so that the entire myosin molecule has 6 subunits.[43] Staron and Pette have done several elegant experiments which combined histochemical ATPase staining followed by microdissection of each of the various fibre types into small pieces which were then analysed biochemically by gel electrophoresis for the presence of different myosin heavy chain isoforms.[44,45] These studies showed that there are three different myosin heavy chain isoforms which are named type I, type IIa and type IIb since they correlate with histochemically identified type I, type IIA and type IIB fibres. Type IIAB fibres, which show a continuum of ATPase staining intensities between Type IIA and IIB, contained both type IIa and type IIb myosin



heavy chains.[45] The presence of both types of fast myosin heavy chains has also been shown by several other groups in rat and human muscle.[46,47] It has recently suggested that the type IIAB fibres can be broken down into type IIAb (containing predominantly type IIa heavy chains), type IIAB (containing 50-50 type IIa and b heavy chains) or type IIaB (containing predominantly type IIb heavy chains).[48]

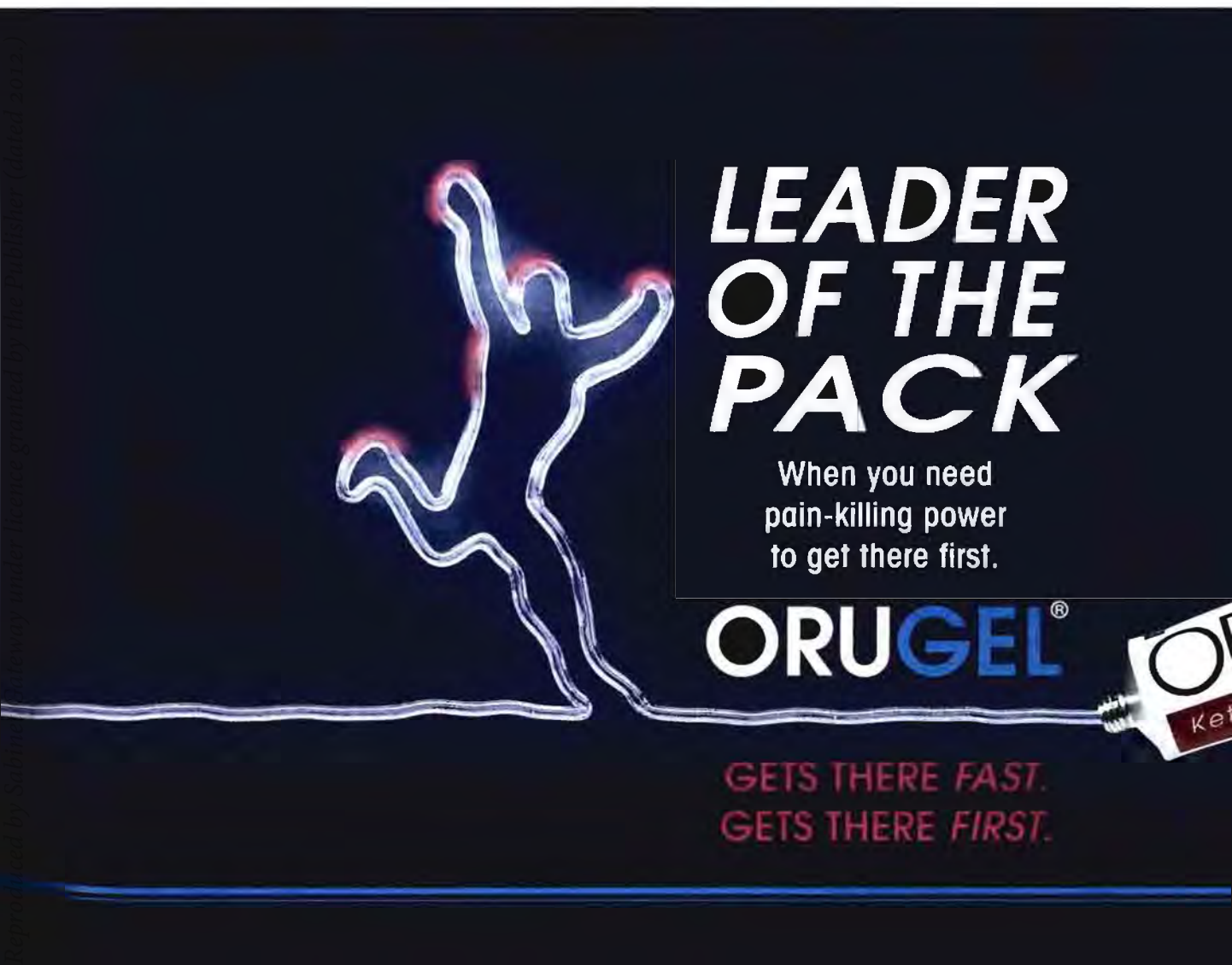
For the exercise scientist an important question arises at this point: are these more subtle histochemical and biochemical differences really *functionally* significant?

V.iv Relationships between contractile properties and contractile protein subunits

Reiser et al[49] tested the relationship between myosin heavy chain composition and maximal shortening velocity in rabbit soleus. The majority of the soleus fibres contained only the slow heavy chain isoforms and contracted with a low Vmax. A few fibres which had significantly higher Vmax values had differing proportions of slow and fast myosin heavy chain isoforms, and the greater the proportion of the fast isoform, the higher the Vmax values obtained. This study did not subdivide the fast myosin heavy chain into the IIa and IIb isoforms. However, recently a rat study which combined mechanical measurements with immunocytochemical staining with 6 different myosin heavy chain antibodies, identified type I, type IIA and type IIB fibres as well as another fibre type which they named type IIX (see Table II).[34] As expected, type I fibres had significantly slower maximal shortening velocity than any of the fast fibre types. Based on average values, type IIB were the fastest and significantly faster than either type IIX or type IIA. The range of Vmax values for type IIA, IIB and IIX fibres overlapped considerably, and the authors suggested that differences in the isoforms of the myosin light chain subunits could be responsible for additional regulation of the maximal velocity of contraction. Indeed, Greaser et al[39] had previously shown in rabbit plantaris (mixed fibre type composition), that myosin light chain composition significantly influenced the Vmax of the fast fibres. In short, all the slow velocity fibres contained only slow isoforms of light chain; the fibres in the group with highest velocities contained only fast isoforms of both heavy and light chains; the group with intermediate velocities had various combinations of fast and slow isoforms of both heavy and light chains.

V.v Contractile protein subunits in human skeletal muscle

Another important issue from the point of view of the exercise physiologist, is whether the findings in animal studies are relevant to human muscle. As noted earlier, rat muscle contains an extra distinctive fast fibre, namely the type IIX,[34] which is not present in either rabbit or human muscle. But histochemical and biochemical studies on human muscle biopsy samples have shown heterogeneity within the broad classifications of fibre types. Biopsies obtained from the vastus lateralis of healthy men showed that the slow-twitch fibres contained only slow myosin heavy chains, but light chains of both the slow and fast isoforms, although the slow



isoforms predominated (see Table II).[50] Furthermore, the histologically classified fast type IIA fibres (shown in the rat to have intermediate maximal shortening velocities[34]) contained predominantly a different light chain isoform than did the fast type IIB fibres (as was also found in the rat). These histochemical and biochemical results are also very similar to those found in the study of the rabbit plantaris, a muscle of mixed fibre type.[39] Although Wada et al[50] did not do mechanical measurements on their human muscle fibres, one would expect that their contractile properties would also demonstrate the continuum of shortening velocities correlating with the biochemical composition of the myosin molecule subunits which was shown in the animal studies.

TABLE II

Heterogeneity of skeletal muscle typed by histological methods or by biochemical analysis of contractile protein composition

Skeletal Muscle Fibre Divisions		
Histological typing:	Slow-twitch: I, IC	Fast-twitch: IIA, IIAC, IIC, IIAB, IIX* IIB
Biochemical typing:		
— myosin heavy chain (MHC):	I	IIa, IIAb, IIaB, IIX*, IIB
— myosin light chain (MLC):	mostly: 1s, 2s also: 1f-3f	mostly: 1f, 2f, 3f also: 1s, 2s

* *rat muscle only*

VI. Current research issues

It is therefore apparent that muscle contractile protein composition is even more heterogeneous than was previously believed. The cause of this heterogeneity is almost certainly a combination of both genetic and environmental factors. The relative importance of each of these mechanisms is a current issue in the field of skeletal muscle research, and there is no doubt that interesting information will soon become available. Another issue currently under investigation is the effect of training on muscle histology and muscle contractile protein isoforms. A recent human study reported the composition of a biopsy of the vastus lateralis of a highly strength-trained woman.[52] In addition to the usual type I, IIA, and IIB fibres, there was a distinctly different set of C-fibres which comprised 15% of the total number of fibres. The biochemical data revealed that even the C-fibres had sub-types (IC, IIC and IIAC) which could be distinguished by the ratios between the different isoforms of myosin heavy and light chains which they contained. The authors suggested that these fibres were either specifically adapted and existed as a distinct population of fibres in highly trained muscle, or they were in the process of being transformed from fast to slow fibre types. In a rat study, where the plantaris was overloaded by surgical removal of the medial gastrocnemius and the soleus, Diffee et al[52] showed that the myosin heavy chain composition changed from predominantly fast to about 25% slow isoforms. The effects of more dynamic exercise on contractile protein isoform composition has not yet been investigated.

VII. Implications for sport

Variability in athletic potential between individuals can, at least in part, be traced to heterogeneity of the contractile proteins themselves. The adaptations of skeletal muscle to exercise training include more than hypertrophy, improved resistance to fatigue and changes in enzyme activity and substrate utilisation. It is as yet unknown exactly how different types and quantities of training influence the contractile protein composition and to what extent their mechanics can be changed. However, these issues provide a challenge to researchers in this area.

Conclusion

The diversity of mammalian skeletal muscle is due in part to both obvious as well as more subtle molecular differences in the contractile proteins. These are characterisable histochemically and biochemically and there is abundant evidence that these differences influence the mechanics of contraction of the individual muscle fibres. It is appropriate to mention again that we have not even considered differences which exist in the architecture of the whole muscle or the activation of the motor units. We have also paid little attention to the enzymatic and substrate content of the muscle fibres, except as these directly influence their histochemical classification. Nevertheless, this paper has described the molecular basis of muscle contractile protein heterogeneity and its functional consequences (see Table III for summary). Given all these complexities, it is not surprising that quantitative differences exist in the muscular capacity of athletes, particularly elite athletes, and we suggest that these differences can be traced to the contractile proteins themselves. In addition, with this information at hand, it is now possible to investigate more systematically the adaptation of muscle proteins to environmental influences such as increased or altered exercise training regimens.

TABLE III:

A summary of mechanical and biochemical properties of skeletal muscle fibres

- Maximal force production is related to cross-sectional area in intact muscle as well as in single fibres in vitro
- Maximal shortening velocity can be as much as 6-fold higher in fast glycolytic fibres compared with slow oxidative fibres
- Myosin is a large molecule with 6 subunits, all of which may occur in different isoforms
- The myosin heavy chain isoform regulates maximal velocity of contraction
- The myosin light chain isoform is responsible for additional regulation of the maximal velocity of contraction
- Myosin isoforms change after inactivity indicating that adult muscle fibres adapt to environmental influences

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Delayed-Onset-Muscle-Soreness

MI Lambert and SC Dennis

Introduction

Muscle soreness that occurs during intense exercise is most often caused by muscular compression of capillaries and hence an inadequate blood flow within the contracting muscle. The resulting accumulation of protons, phosphate ions and nucleotides arising from inadequate removal of the products of energy production, all stimulate pain receptors in the muscle (Jones and Round 1990). Once the exercise is terminated, this acute muscle soreness subsides rapidly. Teleologically, it may be argued that such pain occurs as a "back-up" to the rapid fatigue that prevents the muscles from becoming irreparably damaged by critically depleting the energy stores that are vital for muscle relaxation.

Another form of muscle soreness occurs several hours after the end of exercise. This condition is characterised by discomfort or pain in skeletal muscle with even modest muscular exertion. The pain is first evident several hours after the cessation of exercise, peaks 24-72 hours later and subsides after 5 to 7 days. The pain is elicted when the muscle is pressed, contracted or stretched. The pain, in contrast to the acute pain that occurs during exercise, is known as delayed-onset-muscle-soreness (DOMS). Although DOMS can be caused by stretching a muscle and by isometric and concentric muscle contractions, it occurs most commonly after eccentric contractions when muscles are used in a breaking motion (Ebberling and Clarkson, 1989).

Theories of DOMS

Several theories have been proposed to explain DOMS. The two most common theories are the metabolite accumulation theory and the muscle damage theory.

(1) Metabolite accumulation theory for DOMS

Although no longer supported by any scientific evidence, the metabolite accumulation theory is still often used as an explanation for DOMS by many coaches. This theory erroneously relates DOMS to the increased blood and muscle lactate levels that occur during high intensity exercise. However, neither muscle nor blood lactate concentrations remain elevated for more than 15-30 minutes post-exercise and return to pre-exercise concentrations within 60 minutes (Oyono-Enguelle et al, 1989). Furthermore, DOMS is more severe after eccentric exercise, even though there is less blood lactate accumulation with this type of exercise. In addition, severe DOMS may occur after stretching a muscle, despite the fact that blood lactate levels do not increase. A final point is that McArdle's patients, who lack the enzyme glycogen phosphorylase and are therefore unable to break down glycogen to lactate during exercise, do not show an increase in blood lactate concentrations while exercising, and yet may still experience severe DOMS.

(2) Tissue damage theory for DOMS

The more scientifically plausible theory is that DOMS is caused by tissue damage. The following observations support of this theory:

(a) Muscles with DOMS have microscopically identifiable disruptions of myofibrils and streaming of the Z-lines, a common non-specific finding in many muscular diseases (Friden, 1984).

(b) DOMS causes an increased leakage into the blood of intramuscular enzymes such as creatine kinase (CK) and lactate dehydrogenase, (LDH) (Clarkson et al, 1992).

(c) DOMS is associated with the excretion of high concentrations of 3-methylhistidine in the urine which is a non-metabolizable amino acid found in the actin protein of the muscle thin filaments.

(d) After exercise causing DOMS there are high concentrations of C-reactive protein in the blood which indicate tissue damage (Strachan et al, 1984), and

(e) Following a marathon race, runner's leg muscles have intra- and extra-cellular oedema with endothelial injury, and dilation and disruption of the T-tubules. There is also focal disruption of the mitochondria (Warhol et al 1985). In a rat experiment Armstrong et al (1983) showed that monocytes, macrophages, satellite cells and fibroblasts were present in the muscle 1 to 2 days after severe exercise which also suggest severe muscle damage. Furthermore, Warhol et al (1985) showed that 12 weeks after a marathon, many muscle fibres had central nuclei and satellite cells indicating that the previously damaged fibres had regenerated

Why Does Eccentric Exercise Cause DOMS?

The contrast between eccentric and concentric muscle contractions in terms of causing DOMS was shown clearly by Asmussen (1956) in an experiment in which subjects raised a weight with one arm, and lowered it with the other. The subjects claimed that raising the weight (concentric contraction) often exhausted the muscle, whereas lowering the weight (eccentric contraction) felt easier. Nevertheless, the muscles involved in the eccentric contractions were more painful several days later. This can be explained because eccentric mus-

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cle contractions lowering a load have less electromyographic activity (EMG) compared to concentric contractions raising the same load (Newham et al, 1983). A lower EMG activity indicates that less fibres are recruited to move the same weight and so during eccentric contractions each fibre undergoes more strain and hence is more prone to mechanical disruption (Lieber and Friden, 1988).

Muscle damage and pain during DOMS

Mechanical disruption of the muscle fibre affects the structural and functional proteins and the connective tissue network (Lieber and Friden, 1988), causing a decrease in maximal force production (Clarkson and Tremblay, 1988) which may remain impaired for 3 weeks (Newham et al, 1987). Within a few hours there is a significant elevation in circulating neutrophils (Smith et al, 1989) which infiltrate the site of injury (Kuipers et al, 1983). The pain in the muscles at this stage is possibly due to a disruption of the connective tissue and to the damage in the muscle itself (Jones et al, 1987).

During exercise which causes DOMS it appears that the weak or more susceptible fibres are damaged to a greater extent (Newham et al, 1987). If the damaged fibres are not repaired within a day the entire fibre or a segment of the fibre degenerates as macrophages infiltrate and remove the cellular debris. At this stage there is an increase in serum prostaglandin E_2 (PGE_2) concentrations. (Bansil et al, 1985; Smith, 1991). PGE_2 decreases the threshold of the pain receptors in the muscle (Smith, 1991). The delayed muscular pain may in part be due to inflammation that occurs several hours after the activity (Smith, 1991). During this period the size of the damaged muscle may increase by 5-10% (Jones and Round, 1990). Most of this swelling can be attributed to oedema. This produces a significant increase in intramuscular pressure at rest (Newham and Jones, 1985). Small movements or palpitations may further increase the intramuscular pressure, which provides a stimulus for the pain receptors already sensitized by the PGE_2. This pain, however, is not reduced by anti-inflammatory drugs (Donnelly et al, 1988; Jones and Round, 1990). The potential benefits of the delayed pain are not clear. Whereas it can be argued that acute pain has a protective role by preventing further muscle contractions that may cause damage, DOMS does not fall into this category because of its delayed response.

Physiological consequences of DOMS

(a) Accumulation of calcium in the sarcoplasm

One to two days after exercise causing muscle damage, the affected muscles shorten, and remain shortened for about 5 days (Jones et al, 1987; Cleak and Eston, 1992). This excessive contraction at rest is thought to occur as a result of an influx of Ca^{++} into the sarcoplasm, either through the Ca^{++} channels or through physical disruption of the cell membrane (Cullen and Fulthorpe, 1975). It is also possible that the damaged connective tissue network is involved in causing the muscle to shorten (Jones et al, 1987). The stiffness and shortening develop before the delayed pain which suggests that they are not causally related.

Continuously higher than normal resting levels of Ca^{++} in the sarcoplasm are sequestered by the mitochondria. Ca^{++} uptake into mitochondria occurs at the "expense" of ATP production and in extreme cases compromises the extent to which cytoplasmic Ca^{++} concentrations can be controlled. Uncontrolled rises in cytoplasmic Ca^{++} concentrations activate a Ca^{++}-dependent proteolytic enzyme which preferentially degrades Z-discs, troponin and tropomyosin. A Ca^{++}-activated phospholipase also destroys cell and other membranes (Jones and Round, 1990). It is not known whether phospholipase activity promotes CK release or whether the release of CK occurs earlier as a result of the mechanical damage.

(b) Elevated plasma CK activity

The mechanism causing increased plasma CK activity is not entirely understood. It appears to be exercise dependent since downhill running causes plasma CK activity to increase 3-6 hours after the bout, peaking 18-24 hours later, whereas local eccentric exercise causes plasma CK activity to increase after 48 hours and to peak at about 7 days post-exercise (Clarkson and Tremblay, 1988). The delayed appearance of CK activity in the plasma is thought to be due to the slow increase in membrane permeability (Newham et al, 1987). It has also been suggested that the delayed CK response might also be due to the diffusion and mixing within the lymphatic system that clears the CK from the extracellular space (Jones and Round, 1990).

Because there is large variability in plasma CK activity increases following exercise, which is not dependent on age, fitness or degree of soreness (Noakes, 1987), elevated blood CK activity cannot be used as a clinical test to quantify the degree of muscle damage,

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especially as the time course of the elevation of plasma CK activity is dissociated from both pain and muscle function impairment (Newham et al, 1987).

Training response to DOMS

The muscles may adapt to DOMS for several weeks after a single bout of eccentric exercise. This is suggested by the lower plasma CK activity in response to a subsequent exercise bout, even though the pain rating is similar. There are 3 possibilities to explain this phenomenon (Newham et al, 1987):

(1) there is a change in motor unit recruitment pattern,
(2) there is some intrinsic adaptation in the muscle fibre, or
(3) fibres nearing the end of their growth, or a small part of a weakened fibre are destroyed at the first exposure.

At this stage it seems that the latter 2 points are the more likely explanations because the EMG pattern does not change with training, suggesting that muscle recruitment is unaltered. There is also evidence for connective tissue involvement in the adaptation to DOMS. One possible explanation may be that the first exposure to unfamiliar exercise causes mainly muscle tissue disruption, but that as the muscle becomes trained the DOMS is attributed more to connective tissue disruption.

Treatment for DOMS

Presently the treatment for DOMS can only be prescribed based on empirical observations. There is debate whether to rest completely until the pain subsides or whether to continue exercising, albeit at a low intensity. Sherman et al (1984) did not find a difference in the recovery rate after a marathon between a group of runners who rested compared to a group who continued to train. Also, since both modes of treatment are commonly practised by sports participants, it can be assumed that neither method is overtly superior to the other.

Although static stretching exercises have been proposed as a mode of treatment for DOMS, (De Vries, 1966) this theory has not been tested adequately. Intuitively it appears that if the muscles are damaged in the acute phase of DOMS, then stretching the muscle may be counter-productive.

Summary

Exercise, particularly exercise involving eccentric muscle contractions, causes delayed muscle pain with peak soreness occurring about 24 hours after the activity and continuing for up to a week. Muscle contractile function may be impaired for several weeks after the damage. There is much evidence to show that muscle fibres with delayed-onset-muscle-soreness are damaged. These fibres do, however, repair and become more resistant to damage in subsequent bouts of exercise. Although elevated plasma creatine kinase activity is associated with muscle damage, it cannot be used to quantitatively assess the amount of muscle damage due to a large intra-subject variation and the variation due to the type of exercise activity. Anti-inflammatory medication does not reduce the severity of DOMS. There is no difference in recovery time when subjects have complete rest compared to when they exercise moderately.

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Advances in Sports Equipment Technology that Enhance Performance

Chester R Kyle Ph D

Introduction

Modern equipment has revolutionized many sports, leading to major improvements in performance, radical changes in technique, an increase in the sport's popularity or safety, or in some cases a complete change in the structure of the sport. It is the purpose of this presentation to show the importance of equipment technology in modern Olympic sports and the competitive advantage that can be gained by equipment improvement.

Whenever there is an athletic competition, there is an automatic conflict between those who make the contest rules and those who try to interpret the rules to their own advantage. In sports that use high technology, it is even more complex. The rule makers would like to ensure a fair contest between athletes and not have their sport degenerate into a competition to see who can design the best equipment. On the other hand, manufacturers and designers will try to find ways of stretching or interpreting the rules so that equipment improvements might give their athletes an "unfair" advantage. For this reason, athletic governing bodies frequently rule against equipment improvement and in effect "freeze" the sport in a time capsule. An example is the racing bicycle which remained basically unchanged for about 100 years, (it has undergone rapid change recently).

Obviously, a certain amount of standardization in equipment is necessary. Logically a contest should be between athletes and not between equipment designers. However, many times there are good reasons for allowing equipment innovation. Changes will often improve athletic performance and make a competition safer. Better equipment can give the athlete a longer competitive life. For example modern materials in running shoes absorb shock and help prevent injury. In combination with resilient track and field surfaces, there is no doubt that they also improve performance.

In the United States, sporting goods manufacture and sale is a 30 billion dollar per year business. There is a great deal of competition among manufacturers to have winning athletes use their equipment and there is a great deal of pressure upon the sports rules committees by the equipment manufacturers to permit innovation in order to stimulate sales of new equipment in the huge sporting goods market. There is also pressure from the athletes and coaches to permit use of the best equipment available. These forces have much the same effect as lobbying in government. With pressure of this sort on rules committees, there will always be some degree of continual change in sports equipment.

This cycle of change creates problems of major proportions. A new piece of equipment previously undefined within the rules gives an athlete an obvious advantage. The sport's rules committee passes a regulation to control the new innovation. Designers find a loophole in the new regulations and design equipment to evade the rule which leads to new revisions and rules. This adversarial ballet continues on and on. It can be a distinct disadvantage to a country that does not have the technology to produce its own sports equipment. Such countries must rely upon the open market which may be several generations behind.

However, in general, designing equipment to the very limit and even beyond the rules has a healthy, stimulating and beneficial effect on sports. Without such activity, for example, skis would still be made of wood with leather straps and the pleasure of skiing with confidence and skill would be denied to millions of skiers.

High tech sports

A 1984 report of the USOC Sports Equipment and Technology Committee classifies the Olympic Sports as either High, Medium or Low Technology. See Table 1.[1]

In each of the high tech sports, the outcome of the event depends heavily upon the type of equipment used. Equipment design changes can often result in major improvements in athletic performance. An average athlete could conceivably defeat a world class performer by using illegal equipment. For instance, in archery, modern compound bows and long radius sights are forbidden since they would radically improve scores compared to the more traditional bows. If equipment designers are seeking to get measurable results, they would do well to concentrate on the high tech sports. Medium tech sports, depend somewhat on equipment, however athletic skill far outweighs equipment technology. In low tech sports, any equipment is so standardized that it has almost no influence on the outcome of the event. Track and field is a sport that spans all three categories.

EQUIPMENT CHANGES THAT HAVE REVOLUTIONIZED SPORTS: TRACK AND FIELD

The Pole Vault

Until the 1950's, pole vaulters used bamboo poles and vaulted into a sawdust pit and the runways were of cinders or dirt. Modern pits are now made of deep absorbant foam and the runways are of a shock absorbant synthetic material that gives better traction. It would be inconceivable for a modern vaulter to jump as high as 6 metres and land in a sawdust pit without injury. The

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TABLE 1

Olympic Sports where equipment technology influences performance

High Tech	ARCHERY	BIATHLON	BOBSLEDDING
	CANOEING/KAYAKING	CYCLING	LUGE
	MODERN PENTATHLON	ROWING	SHOOTING
	SKIING	YACHTING	
Medium Tech	FENCING	FIELD HOCKEY	ICE HOCKEY
	FIGURE SKATING	ROLLER SKATING	SPEEDSKATING
	TENNIS		
Low Tech	BASEBALL	BASKETBALL	BOXING
	DIVING	EQUESTIAN	GYMNASTICS
	JUDO	FOOTBALL	SOFTBALL
	SWIMMING	SYNCHRO. SWIMMING	TABLE TENNIS
	TEAM HANDBALL	VOLLEYBALL	WATER POLE
	WEIGHTLIFTING	WRESTLING	
HIGH, MEDIUM AND LOW TECH, COMBINED		TRACK AND FIELD	

most spectacular improvement, however, has been in the poles. Using a bamboo pole, Cornelius Warmerdam held the record for nearly 18 years at 4.72 meters, from 1942 to 1961. A slight improvement came with the introduction of steel and aluminium poles in the 1950's although they did not improve the record significantly.

In the early 1950's, Herb Jencks, a manufacturer of fiberglass fishing poles in Southern California made a new model deep sea tuna pole for commercial fishermen. The pole was about 3 meters long and 3 cm in diameter. Jenck's son was a 45 kilo Junior High-school polevaulter. As a lark, he and some of his friends took one of the fishing poles, used wooden plugs for pole tips and tried polevaulting with the tuna pole. They promptly bettered their best height by several inches compared to the bamboo or steel poles they had been using. Jencks began building poles for local high school vaulters and later for all comers as they became more popular. Figure 1 shows what happened to the pole vault record after introduction of the fiberglass pole. The record went from 4.7 meters to over 5.2 meters in just four years, from 1961 to 1965, after remaining stagnant for the previous 18 years. At present the record is about 6.1 meters.

The reason composite poles are superior is that energy may be stored in the pole when it flexes, allowing the vaulter to hit the vault box much faster without undue shock.[2] The pole bends to one side, so that the vaulter may swing straight through. At the top of the swing, the stored energy in the pole is returned, catapulting the vaulter over the bar. Poles are wrapped so that they have a soft side and will bend preferentially in one direction — this side is marked on each pole. Modern poles are made of carbon and glass laminate. The pole used by the present World record holder Serge Bubka weighs only 2.6 kg and is 5.25 meters long (about .75m shorter than his record height)! Poles are specifically designed for the weight and skill of the vaulter. Bubka's pole is manufactured by Gill Sports of Urbana, Illinois.

Probably the most famous incident involving a composite pole was when US Vaulter Bob Seagren had his carbon pole outlawed at the last minute by officials at the 1972 Olympics. They ruled that the pole had not

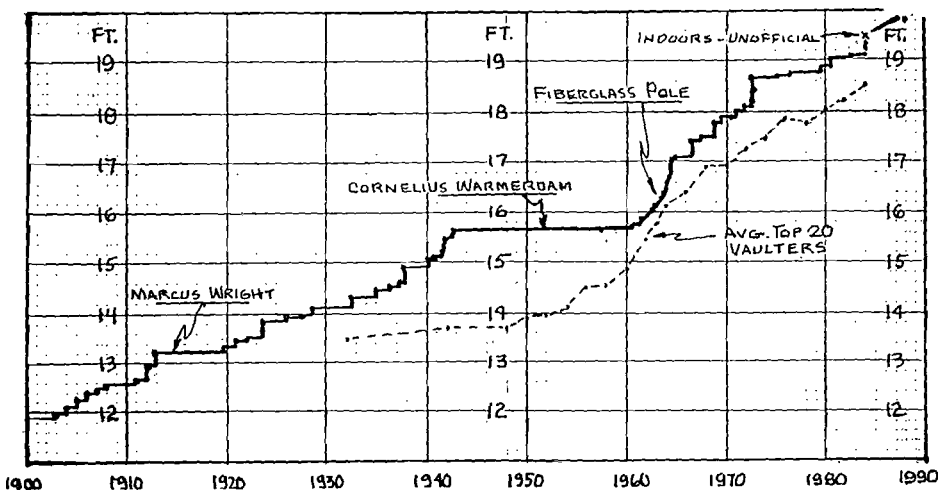

Figure 1 WORLD POLE VAULT RECORDS 1900-1984.

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Figure 2a&b: *Composite poles bend and store energy on the upswing, and release this energy when the vaulter is at the top, allowing pole vaulters to hit the box at higher speeds without undue shock. The pole bends to the side to let the vaulter swing through. Since the introduction of the fibreglass and carbon poles in 1961, the pole vault record has increased over 1.5 metres.*

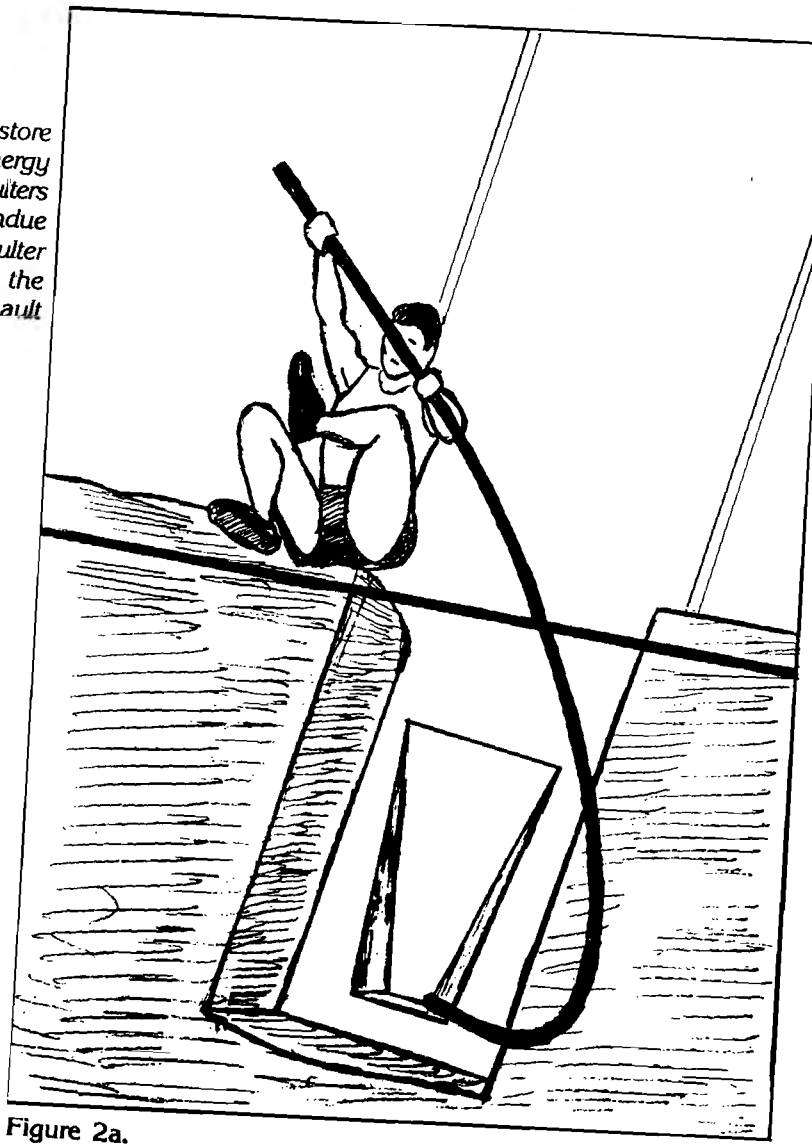

Figure 2a.

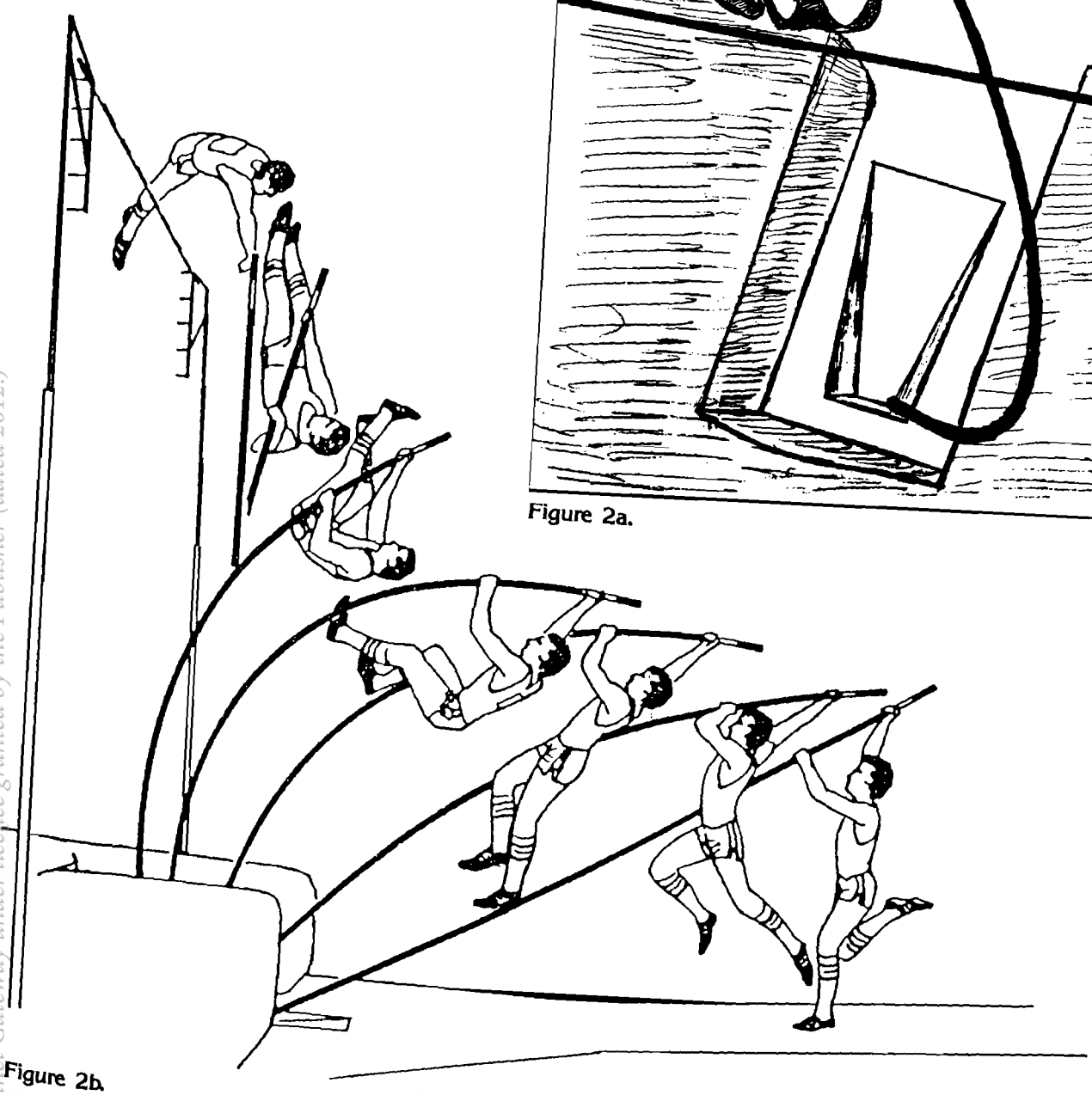

Figure 2b.

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been on the market for sufficient time to permit everyone to have the chance to purchase one. He was forced to vault with a borrowed pole and won the silver medal although he was the World record holder. At present a vaulter may use any pole design as long as it is commercially available at least six months before the Olympic games.

One unforeseen side-effect of high technology in the polevault is that poles are so expensive (about $300-$450 US) that their cost eliminates many of the developing countries from competing in the event. A dent or mar in the surface can cause a pole to shatter. Extreme care must be taken to protect the pole or a good vaulter can go through several poles in a season.

The introduction of the foam pit is often overlooked when considering pole vault and high jump records. This made it safe for jumpers to fall from great heights safely even if they landed incorrectly. Also runway surfaces have been specially designed to give the right spring and traction to match the stride frequency of the jumpers. Modern shoes give better traction and resilience. The combination is a very high-tech system which permitted the vault record to increase more in 30 years than it had in the previous 65 years since the beginning of the sport.

The high jump record also increased 15% in the same time period. Part of this was no doubt due to changes in technique and training, but the major improvement was due to equipment technology. Aside from improvements in shoes and the take off surface, the Fosbury flop would be impossible without the foam pit. Jumpers could only land a few times in sawdust without spending the rest of their life in a wheel chair.

The javelin, discus and hammer

Like the pole vault, the javelin record didn't change much during the 1930's and 1940's. In the late 40's, a Stanford javelin thrower, Bud Held, was discouraged at the high cost of hollow wooden Swedish javelins. The wooden javelins of the time were very fragile and bad luck might cause several to break in a week. They were also hard or impossible to repair. He asked his brother Dick Held, an electrical engineer and contractor, to build a javelin for him that wouldn't break. Dick made one of aluminium that was an immediate success. Using the new javelins, Bud won the NCAA championship three times and the AAU meet several times. When he improved his distance by about 7 meters, the Held javelin attracted international attention. Within a few months, Dick Held was designing and building javelins full-time. He is still in business at Oregon Track Equipment, Eugene, Oregon and has designed the javelins that have set most of the recent World records.

The brilliant innovation introduced by Held came when he realized that with javelin flight as with any other flight, aerodynamics was critical. He built his javelins to take advantage of aerodynamic lift like a wing and he streamlined their shape so that they were far more efficient. Unlike conventional javelins, the Held javelin barely landed point down if thrown properly. It kept a positive angle of attack during its flight. If thrown in a vacuum for maximum distance, a modern javelin would not travel as far, about 18% less than in air. The Held javelins were termed "unstable" since they did not align themselves with the flight path like an arrow, but continually changed their angle of attack during flight.

By 1986, the javelin World record was 104,8 meters, a distance so far that javelins were sometimes landing on the track, an obvious danger to the competitors. See Figure 3. A new javelin design was mandated by the governing body that changed the center of gravity and shape of the javelin so that it would not have as much aerodynamic lift. The World record immediately fell to 84.7 meters — it has since risen to 90.98 meters, about 15 meters short of the old record. Among other things, the regulations specify the dimensions and weight distribution of the new javelin and that they must be made of a single material. Javelins have been ruled illegal several times in the past three years for violating these rules. One recent World record was voided because it was determined the competitor had used an aluminium javelin stiffened with composite materials which changed the launch and flight characteristics.

One recent development in javelin technology is the work of Mont Hubbard of the University of California

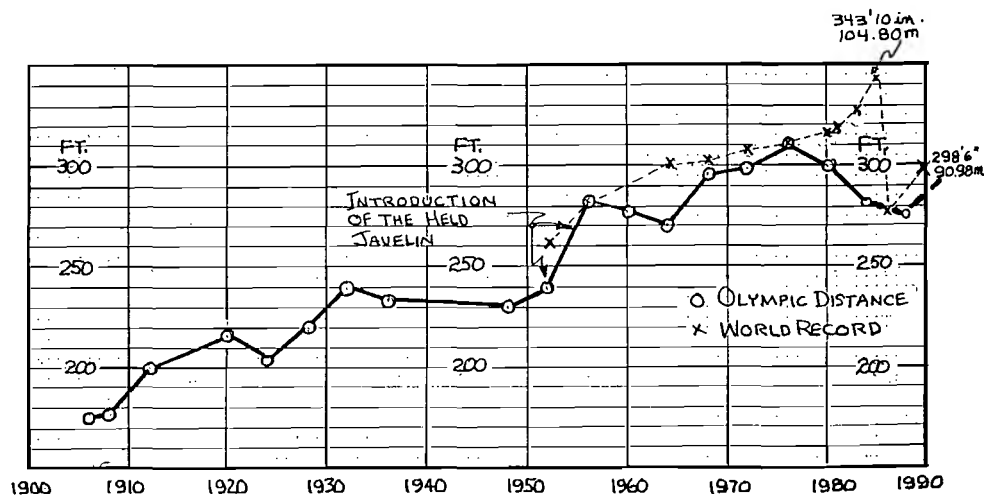

Figure 3 JAVELIN: OLYMPIC WINNING DISTANCES AND WORLD RECORDS.

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at Davis.[3,4,5] Using an interactive computer and a high speed digitized video camera (200 frames per second), Hubbard has built a device that will measure the launch characteristics of the javelin including velocity, launch angle, angle of attack and angular velocity. Using computer simulation, Hubbard has been able to successfully predict the optimum launch conditions that will produce maximum distance. The javelin thrower is able to instantaneously compare his actual throw with the ideal throw and to correct technique to achieve longer distances. World class javelin throwers can launch the javelin at about 30 meters per second. Hubbard, using wind tunnel data, has shown that at this velocity, the old Held javelin should be thrown with a 5.9 degree initial angle of attack, a launch angle of about 30 degrees and a slight negative pitching angular rotation of 9.2 degrees per second. The maximum distance is very sensitive to errors in pitch angular velocity (twist). If an error of a few degrees per second is made, the javelin could fall 20 meters short even when thrown with the same velocity. In other words, the "lucky" throw was a real factor with the old javelin. An analysis of former World record holder Tom Petranoff showed that his record launch conditions with the old javelin almost identically matched Hubbard's predictions.

For the new javelin, the release conditions are quite different.[6] Although the launch angle is the same at 30 degrees, the ideal pitch rotation is nose upward at three degrees per second while the angle of attack is a negative two degrees. The new javelin is much less sensitive, so the lucky throw is not such a factor in elite competition. Probably the next major improvement in the javelin will be to increase its stiffness. Vibrations decrease the efficiency of the throw.

This brings up a point that when new equipment and techniques are introduced, careful analysis by biomechanicists and scientists can help improve performance still further. Close ties between athletes, coaches, trainers and sports scientists has become essential for success.

Like the javelin, the discus also has the potential of flying much further in air than in a vacuum due to its airfoil shape (about 11% further). When the wind blows from the right direction, the discus throwers are ecstatic since a proper wind lifts the discus and carries it further. The stronger the wind the better. Contrary to logic, it is a quartering head wind that is best; it corrects the tendency of the discus to tip over and follow a curved path. Figure 4 shows the progress of discus Olympic records. Unlike the pole vault and javelin, the shape of the discus has been fixed rigidly for a long time and distance improvements have mostly been due to better technique and training. This is shown by the uniform rise in the distance versus time curve in Figure 4. One major change has been made recently; the discus weight has been concentrated almost entirely in the rim, making the discus more stable in flight and easier to throw at higher velocities. The body is made of Kevlar or sheet aluminium, which weighs very little as compared to the rim. This high-tech discus is expensive and is only used in competition since it can be easily damaged.

Even with the hammer, aerodynamics can play a part. In the early 1980's the East Germans manufactured a hollow bronze hammer ball filled with mercury. Since the ball had a smaller diameter, it had less air resistance and flew further. The rules committee solved this problem by specifying a minimum diameter for the ball, thus making the material irrelevant.

Fast running tracks, tuned tracks and shoes

Igor Garrow, a US inventor, has recently obtained a patent on a tuned running shoe that he claims will conserve 70% of the impact energy now lost when the foot contacts the ground. The shoe utilizes light efficient carbon springs in the heel and sole to absorb the shock of impact and later returns the energy when the foot leaves the ground. In theory this should work, however the spring would have to be tuned to the stride frequency, weight and running technique of each athlete to be effective without altering the runner's motion patterns

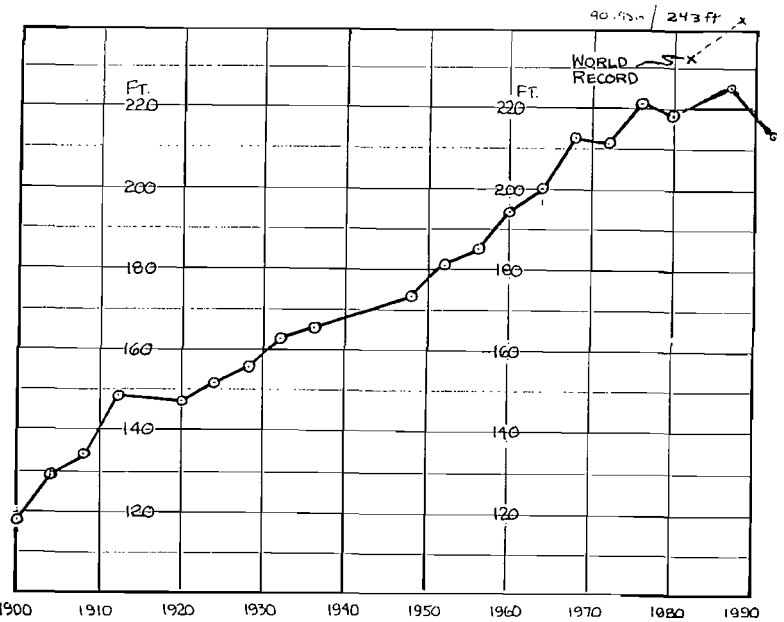

Figure 4 DISCUS: OLYMPIC WINNING DISTANCES 1900-1990.

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and hindering performance.

In 1977, a group of Harvard scientists were asked to help design a new indoor track; in their studies, they predict that if the compliance (springiness) of the track were tuned to the runner, that running times should improve from 2 to 3% when compared to a hard track surface. In observations of comparative running times on the Harvard track and at other tracks, this was proven. One of the last real frontiers to be explored in events involving running or jumping, is to theoretically and experimentally design the surface to suit the event (i.e. the long jump, triple jump, pole vault, high jump, and track running events). Each event and distance would thus use a different surface, and even have variations in material at different locations (such as the long jump take off area). This of course would be time consuming and expensive, however the results should be worth the effort.

OTHER SPORTS Rowing, Canoeing, Kayaking

In 1982 the US Olympic Committee created the Elite Athlete Program to improve the chances of US athletes in the Olympic Games by trying to develop better equipment, training techniques, coaching and support. One of the outgrowths of this program was the design of new rowing shells, canoes and kayaks. Ted Van Dusen, president of the Composite Engineering Inc. of Concord, Massachusetts who is an Ocean Engineer and a former US elite rower, has designed and built a fleet of carbon/Kevlar boats for the US teams.[9]

Using shapes derived from computer models and refined in tow tank tests, Van Dusen's field experiments and competition results have proven the value of his boats. US rowing, canoe and kayak teams have medaled in both the 1988 and 1992 Olympics. Besides improved shape to lower wave and friction drag, Van Dusen has developed light weight composite wing-shaped paddles and oars which are more efficient in producing propulsion. Composite oars and paddles, weigh about half what traditional wooden ones do. Another of Van Dusen's innovations is the aerodynamic rigger for rowing shells. Instead of a conventional round tubing framework, the riggers are airfoil shaped, making them both lighter and stronger than conventional riggers. In addition they have a much lower air drag. Although air drag is only 12 percent of the total drag of a rowing shell, races are often won by fractions of seconds and any improvement could be the winning margin. One interesting sidelight of Van Dusen's computer studies showed that for maximum speed, the length and shape of a boat should be matched to the weight of the crew. Van Dusen has therefore built special boats for the women's teams since their weight is much less than a typical men's crew.

Simulators

One rapidly growing field is the design of sports simulators to give the athlete a chance for a realistic work out in a laboratory environment. With the advent of fast computers, with crisp graphics displays, athletes are able to gauge their performance in real time and thus use bio-feedback to improve.

Several new devices have recently been built in the United States. A stationary paired oar rowing simulator that displays force and power during a rowing cycle for both rowers, allows athletes, coaches and trainers to more accurately gauge training status and ability.

At the US Olympic training center, an instrumented swim flume allows swimmers to be video taped in place for analysis of their technique. A light nylon line fed from an instrumented reel, is attached to the swimmer and gives instantaneous velocity. Pressure pads on the swimmer's palms provide a measure of the thrust force per stroke. Biomechanical studies are under way that should identify efficient swimming motions and allow swimmers to refine their technique.

A new table tennis robot has recently been demonstrated that can fire balls to any point on the table with variable velocities up to 160 kph and with adjustable spin up to 6 000 RPM. It can be programmed to simulate the serve and returns of any player from average to an elite World champion. The inventor of the robot, Waquidi Falicoff of the Logic Handle of Ashland, Oregon, has improved his standing in US table tennis by 200 places since he started training with the simulator. It allows a player to practise against World class competition at any time.

Probably the most spectacular of the current simulators is a project of Mont Hubbard of the University of California, Davis. Mont and his students have obtained the digital coordinates of several of the World Cup and Olympic bobsled runs. The coordinates are used as input into a real time computer model of bobsled dynamics.[10]

Using an actual bobsled, they have fitted hydraulic actuators that tilt the sled to simulate motion, allowing the driver to practise sled runs down the track of their choice without leaving the laboratory. They can follow any arbitrary path they choose. A Silicon Graphics computer and display monitor allows the driver to steer using the standard bobsled steering linkage and pull handles. At the finish, the computer gives both the time and top speed of the run and plots a red line along the course showing the path as well as a green line showing the fastest run. By repeating the program, the driver can compare each trial with the fastest path and try different strategies to improve the time. The colour computer display is amazingly convincing, it includes shadowing, a horizon that tilts as the sled travels up and down the banks and realistic motion as indicated by the texture and shadows speeding by the sled. Since actual ice time is very expensive, the bobsled simulator allows the driver to become familiar with a course before actually making live runs.

The object of any simulator is to reproduce reality accurately enough to make any training experience on the simulator as valuable as the real thing. In this age of electronics it is a fertile field for development. Probably the easiest sport to simulate is cycling. An electronically controlled ergometer can realistically simulate any course in the world. A powerful fan is required to provide equivalent cooling for the rider. Performance on a cycling ergometer has been shown to correlate well

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with actual road or track performance.[11]

Athletic clothing

Well designed athletic clothing can improve performance in ways that are not always obvious. Dr Susan Watkins along with her Textiles and Apparels Department students at Cornell University have designed an award winning protective uniform for female hockey players.[12] Previously women were forced to wear uniforms made for male players, and the protection was dangerously inadequate since nothing fitted properly. Designed specifically for the female form, the new uniform is lighter, more flexible, the padding remains in place in competition and the special protective padding is placed where needed. The lighter, well fitting, and more flexible clothing allows the players to accelerate faster and to move with increased fluidity, therefore improving their game. The freedom from bruises and injury, assured by the efficient padding, allows the athletes to play with more confidence. The same approach taken by Dr Watkins could be applied to field hockey or any other contact sport where protective clothing is used. Light weight, flexibility and better protection should be the goals of such a design program.

In cycling, speed skating, skiing, the bobsled, the luge, and even in track and field, clothing is important for another reason — aerodynamic drag. Loose fitting clothing has a much higher wind resistance at the high speeds of these sports. Tight fitting, wrinkle-free suits that cover the entire body like a second skin have been used to substantially lower the wind resistance. Wind tunnel tests indicate that even in running, wind resistance is significant, see Table 2.[13,14] The tests in Table 2 were performed on a manikin, fixed in a running posture.[15] By changing the clothing or hair fashion, large differences in wind resistance have been recorded. A mathematical model shows that even a 2% reduction in wind resistance can have dramatic results on running performance, see Table 3. The lead generated by a 2% reduction in air drag varies from 0.1 meters in the 100 meter dash to 35 meters in the marathon.

In 1987, I designed a prototype skin suit for the US Olympic track team that included a hood to cover up the runners hair. This was worn by Roger Kingdom in winning the 1988 Olympic gold medal in the 110 meter hurdles. However in the 4×100m relay, the second US Team was allowed to race in the preliminaries and failed to qualify when one of the runners passed the baton over the line. The team complained that the hood muffled the audible commands. However the relay team had not tried the suits before the Olympics. This underscores the importance of thoroughly practising with any equipment you intend to use before competition. Because of the bad experience, the hooded suits have not been used since, however, skin suits are now common in track and field.

Even in swimming, clothing can lower drag. The US team has been experimenting with slick full body suits for male swimmers. In theory they could have a lower drag than human skin.

Another important factor in clothing is heat reflectivity. Larry Bergland of the Comell John B Pierce Foundation has shown that reflective clothing improves heat dissipation from an athlete exercising in direct sunshine, as compared to bare skin.[14] At high noon, the body can absorb as much as 100 watts in radiant energy from the sun. This added heat load must be carried away through sweating, thus adding to dehydration. Reflective clothing can decrease this water loss, keep the body cooler and therefore lower the heart rate and increase endurance. The best clothing Bergland tested had an aluminized reflective coating, but lighter colours proved to be better than dark. Bergland highly recommended that the New Zealand All-Blacks change their colours. Joking aside, in hot climates, special heat reflective clothing that efficiently wicks moisture away from the skin can definitely improve performance in endurance events.

TABLE II

Clothing wind tunnel tests

TYPE OF CLOTHING	Drag (g)	Change in wind resistance (%)
Hair Tests		
No Hair	1.50	0.0
Long Smooth Hair	1.52	+1.5%
Short Curly Hair	1.556	+3.9%
Medium Wavy Hair	1.56	+4.2%
Clothing Tests		
Tight Lycra Track Suit	1.50	0.0
Medium Track Suit	1.53	+2.1%
Large Track Suit	1.57	+5.1%

Tests at California Institute of Technology, CR Kyle, 1986.

TABLE III

Advantage in running due to a 2% decrease in wind resistance

Distance (Metres)	Lead (Meters)	Time Difference (Seconds)
100	0.1	0.01
200	0.3	0.03
400	0.9	0.10
800	1.3	0.17
1 500	2.0	0.29
5 000	5.8	0.90
10 000	9.2	1.55
Marathon (42 195 m)	35.4	6.44

Kyle CR, Athletic Clothing, Scientific American, March 1986, 254: 3: 104-110.

Cycling

Historically, European manufacturers have always provided bicycles for the US Cycling teams because European racing equipment was superior. In 1984, for the first time, the US Olympic Cycling Team used their own cycling equipment with spectacular results. The last Olympic cycling medal the US had won was a bronze in the road race in 1912. In 1984, they won nine, including two gold medals.

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Starting in 1982, under the USOC Elite Athlete Program, at the request of Dr Edmund Burke, then Technical Director of the USCF, I organized a group of 20 people to design and test cycling equipment. The group included Dr Paul MacCready, Paul Van Valkenburgh, Don Guichard, Dr Allan Abbott, Jack Lambie, Jim Gordon and Bruce Longson. We designed and built a completely integrated aerodynamic cycling system including clothing, helmets, shoe covers, disk wheels, an aero tubing funny bike, aero shifters, disk sprockets, aero cranks, and clip on pedals. The wheel size of the bikes varied with the event. In the team pursuit we used two 24 inch wheels so that the teams could draft closer for lower air drag. We performed hundreds of full scale wind tunnel tests at Texas A&M and Cal Tech that verified that our system had a much lower air drag than conventional racing bicycles. The US Olympic bike frames were made by Huffy based on our prototypes. The other equipment was manufactured by our group. Our tests showed that the equipment was seconds to minutes faster in the Olympic cycling events. Experience proved us right.[17,18]

Figure 5. *Wind tunnel testing of bicycles lead to the development of the time trial funny bike in 1984. The photo shows the Texas A&M wind tunnel, and the bicycle that was used to set the World Amateur Hour record (John Frey USA, 49.946 km; Oct, 10, 1991).*

Since 1984, every important time trial record in the world has been broken using variations of the "funny bike". In 1986 and 1987 Don Guichard and I built a monocoque carbon funny bike for the Dupont Company which was intended for use by the US team in the 1988 Olympics. It had the lowest air drag of any bike we ever measured in the wind tunnel. However when Ed Burke took our plans to the Technical Commission of the UCI, they rejected the bike as illegal and immediately passed a set of rules restricting tubing size and mandating a triangular frame. This assured that our Dupont bike could not be used. We abandoned the project.

In 1991 the UCI unaccountably changed their racing regulations, making monocoque composite bikes legal. Mike Burrows of England along with Lotus Ltd, designed and built a bike very similar to ours, (but much improved) on which Chris Boardman set a new World Record in the 4 000 meter pursuit, winning the event by several seconds. Burrows had been working on composite monocoque bikes since 1982 and neatly took advantage of the rule revision with brilliant results. As I mentioned in the beginning of this paper, rule changes continually occur, usually as a result of political pressure of one sort or another. When changes do happen, it pays to rapidly take advantage of them as Burrows did.

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Die Effek van 'n Stresweerbaarheidsprogram op Valskermspringers

Dawie Scheepers en Justus Potgieter

Summary

A stress management programme for skydivers was developed from available literature. An Experimental group (n=18) and Control group (n=18) were used to evaluate an eight-week programme. Pre- and post-experimental psychological and physiological data proved the programme to be effective according to a variety of criteria.

Inleiding

Stres kan 'n beduidende invloed op sportprestasie uitoefen en in 'n sportsoort soos valskermspring kan dit een van die belangrikste sielkundige probleme wees waarmee die deelnemer te doen kry. Talle valskermspringers met goeie potensiaal tree vroeg uit, dikwels omdat hulle nie geleer het om stres te hanteer nie. Daar is ook valskermspringers wat as gevolg van stres nooit werklik doeltreffend kan konsentreer op hul spronge en formasies nie.

Fenz en Jones (1972) se navorsing dui daarop dat stres wat in valskermspring ervaar word, nie net oplos en verdwyn nie, maar deur die springer beheer moet word. Fenz (1973) haal enkele studies aan wat daarop dui dat die kwessie van streshantering in valskermspring vaag en indirek aangespreek word. Verder wys Brewer en Shillinglaw (1992) daarop dat die literatuur wat handel oor sportsielkundige dienste gewoonlik verwys na intervensies op 'n individuele basis en dat daar 'n behoefte is aan navorsing oor die geskiktheid van sielkundige ondersteuning in groepsverband. Die huidige ondersoek het in hierdie behoefte probeer voldoen. Die primêre probleem wat hierdie ondersoek aangespreek het, was die daarstelling en evaluering van 'n stresweerbaarheidsprogram spesifiek vir 'n groep valskermspringers, maar wat ook vir ander sportsoorte van nut kan wees.

'n Belangrike faktor by die ontwerp van 'n sielkundige program is die praktiese toepasbaarheid daarvan op die teikengroep. Die opsteller van so 'n program behoort dus benewens sy sielkundige bevoegdheid ook genoegsame praktiese ervaring in valskermspring te hê. Die hoofnavorser het aan hierdie vereistes voldoen.

Streekproef

Ses-en-dertig aktiewe valskermspringers in die Westelike Provinsie en Boland is by hierdie eksperiment betrek. Die ervaringsvlakke van die springers het gewissel van drie tot 1 720 spronge. Hierdie groep is in 'n eksperimentele groep (n=18) en 'n kontrolegroep (n=18) verdeel. Om praktiese redes is die verdeling gedoen op die basis van klublidmaatskap. Slegs die eksperimentele groep het tussen die twee evaluasiesessies 'n stresweeerbaarheidsprogram gevolg.

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Die stresweerbaarheidsprogram

Die intervensieprogram het oor 'n periode van agt weke gestrek en word kortliks bespreek.

Tydens die eerste sessie word psigometriese evaluering gedoen. Toetslinge voltooi die *IPAT-Angsskaal* asook 'n biografiese vraelys. Die *Sport Competion Anxiety Test (SCAT)* (Martens, 1977) 'n sportspesifieke meetinstrument wat uit 15 stellings bestaan en trek-angs binne sportsituasies bepaal, is ook voltooi. Beide groepe is opgelei om hul harttempo te monitor. Toe hierdie studie uitgevoer is was daar nie tegnologiese middels, soos byvoorbeeld polshorlosies, geredelik beskikbaar om harttempo op 'n gerieflike en ekonomiese manier te monitor nie. Harttempo is geneem op die oggend van hul sprong, tydens die opstygfase en wanneer die vliegtuig sy hoogste hoogte bereik.

In sessie twee tree die intervensieprogram in werking met 'n bespreking van rasionele emotiewe terapie (RET) (Ellis, 1974). Die grondslag van hierdie benadering is dat sielkundige probleme grootliks die gevolg is van irrasionele denke. Die mens kan homself van baie ongelukkigheid bevry as hy leer om irrasionele denke te elimineer en logies en rasioneel te dink. Die basis van hierdie sielkundige intervensie is dus om die valskermspringer te leer om rasioneel te dink. Die RET-sessies is spesifiek van toepassing op valskermspring gemaak.

In hierdie sessie maak die springers ook kennis met progressiewe ontspanning. Die tradisionele tegniek word effens gewysig om aan te pas by die beknopte ruimte binne die vliegtuig. Aan die einde van die sessie, asook na afloop van elk van die daaropvolgende sessies, kry die springers tuiswerkopdragte.

Sessie drie word weer eens ingelei met RET. Die inhoud van die vorige sessie word hersien waarna die springers se tuiswerk oor RET en progressiewe ontspanning gekontroleer en bespreek word. Irrasionele gedagtes wat algemeen voorkom word bespreek. Die springers word daarop gewys dat irrasionele opvattings wat emosionele probleme veroorsaak deur vorige ervarings (kondisionering) maar veral as gevolg van sosiale leer ontstaan. Die belangrikste kategorieë waaronder hierdie irrasionele gedagtes manifesteer sluit onder andere in absolutistiese denke, katastrofering, negatiewe selfbeoordeling, ongeldige gevolgtrekkings, swart/wit denke, en "ek-kan-dit-nie-verdra-nie" denke. Die sessie word,

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soos die vorige sessie, afgesluit met 'n volledige oefening van progressiewe ontspanning en 'n RET-tuiswerkopdrag asook 'n opdrag om progressiewe ontspanning met behulp van 'n geskrewe uiteensetting van die prosedure daagliks tuis te oefen.

Sessie vier begin met hersiening van vorige werk. Daar word seker gemaak dat die springers die kriteria van rasionele opvattings ken en dat almal 'n paar voorbeelde met betrekking tot die ABC-analise van emosies neergeskryf het. Die sessie word afgesluit met progressiewe ontspanning, maar sonder die spanningsfase. Die doel van hierdie ontspanningsprosedure is om die springer meer suggereerbaar te maak vir die daaropvolgende beeldingsoefening.

In sessie vyf word RET bespreek waarna progressiewe ontspanning en outosuggestie as ontspanningstegnieke geoefen word.

Sessie ses word met RET begin waarna progressiewe ontspanning, outosuggestie-ontspanning en beelding geoefen word. Realistiese beelding van die valskermspring in die bepaalde formasies wat gedoen gaan word, word geoefen. In hierdie stadium word springers gehelp om in rasionele terme aan valskermspring te dink sonder om byvoorbeeld te verabsoluteer of te katastrofeer. Dit is belangrik dat hulle hierdie prosedures so veel moontlik oefen aangesien dit is hoe hulle sal moet dink wanneer hulle spring. Die springers word aanbeveel om of progressiewe ontspanning, outosuggestie-ontspanning of 'n kombinasie van die twee tegnieke te gebruik wanneer die vliegtuig besig is om tot op hoogte te klim. Die springer moet tydens hierdie fase kan ontspan terwyl hy die sprong wat voorlê in detail beeld.

Sessie sewe word gewy aan ontspanning en beelding. Daarna word die hele proses van rasionele denke hersien in die vorm van 'n lesing en 'n groepbespreking.

Tydens sessie agt word psigometriese evaluering (*SCAT* en *IPAT*) gedoen asook bepaling van harttempo. Springers word ook gevra om die afgelope stresweerbaarheidsprogram te evalueer.

Resultate

In die voortoets is met die uitsondering van die SCAT-tellings geen verskille tussen die eksperimentele en kontrolegroep gevind nie. Op grond van die feit dat daar wel een veranderlike was waar die twee groepe van mekaar verskil het, is daar in die statistiese verwerking van die data 'n ontleding gedoen van die relatiewe verskille tussen die voor- en na-toetse binne die twee groepe. Die mate waarin die veranderlikes binne een groep toe- of afgeneem het, is vergelyk met die hoeveelheid verandering wat binne die ander groep plaasgevind het.

Die resultate (sien Tabel 1) dui daarop dat beide die psigometriese en fisiologiese veranderlikes by die eksperimentele groep 'n verbetering getoon het, terwyl die kontrolegroep se tellings konstant gebly of selfs versleg het.

In die geval van die IPAT-veranderlike (angs) het die kontrolegroep se telling toegeneem terwyl die eksperimentele groep se telling nie beduidend verander het nie. Die toename in die angstelling van die kontrolegroep kan nie verklaar word nie, maar het moontlik te make met 'n verhoging in stres in die alledaagse lewe van die toetslinge.

Die gemiddelde SCAT-telling (kompetisie-angs) van die eksperimentele groep het 'n beduidende afname getoon na afloop van die stresweerbaarheidsprogram. Die kontrolegroep se telling, daarenteen, het redelik konstant gebly.

Wat die harttempo betref, het die eksperimentele groep beduidende afnames getoon by al drie metings (oggend van sprong, opstygfase en wanneer die vliegtuig sy hoogste punt bereik het). Die kontrolegroep se gemiddelde tellings het konstant gebly, maar in die geval van die opstygfase het dit selfs verhoog.

As gevolg van 'n gebrek aan narvorsingspublikasies hieroor kon hierdie bevindinge ongelukkig nie met ander navorsingsresultate vergelyk word nie.

Subjektiewe evaluering deur proefpersone

Springers is gevra of hulle steeds deel van die program sou wou gewees het as hulle vooraf geweet het wat die program behels. Van die agtien proefpersone het elf aangedui dat hulle "beslis" daaraan sou deelgeneem het, terwyl die res (7) aangedui het dat hulle wel aan die program sou deelgeneem het. Verder het ses persone aangedui dat die program "beslis nodig" was, terwyl nege persone van mening was dit dit "nodig" was. Drie persone was onseker. Sewe toetslinge het aangedui dat hulle "geweldig baie" baat daarby gevind het,

TABEL 1

'n Vergelyking van Voor- en Natoets-Gemiddeldes

	Eksperimentele Groep (n=18)			Kontrolegroep (n=18)		
	Voortoets	Natoets	*t*	Voortoets	Natoets	*t*
IPAT	31.28	27.39	1.84	26.67	30.89	−6.31***
SCAT	22.28	19.78	3.10**	18.44	18.50	−0.08
HARTTEMPO						
Oggend	79.33	76.44	2.82*	80.39	82.00	−1.71
Opstyg	96.61	90.56	4.64***	93.00	95.33	−2.17*
Hoog	101.50	96.78	3.19**	102.11	104.89	−1.83

* $p < .05$
** $p < .001$
*** $p < .0001$

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tien persone "redelik baie" en een persoon het "gemiddeld" baat gevind.

Die respondente is versoek om aan te dui by watter aspekte van die program hulle die meeste baat gevind het. Vyftien persone het aangedui dat die rasioneel-emotiewe terapie vir hulle die nuttigste was. Twee persone het progressiewe ontspanning as die waardevolste aspek beskou. Agt persone het outosuggestie as die swakste gedeelte van die program geëvalueer terwyl sewe persone beelding as die aspek beskou het waarby hulle die minste gebaat het. Drie persone het aangedui dat die progressiewe ontspanning die minste nut gehad het. Die lae waarde wat aan progressiewe ontspanning toegeken is, is verrassend aangesien hierdie tegniek in talle programme van hierdie aard buite die sportarena ingesluit word.

Hoewel die response van die toetslinge uiteraard subjektief is en nie statisties bevestig kan word nie, is dit nogtans belangrik om na hul subjektiewe ervarings te kyk. Die meeste proefpersone (10) wat die program gevolg het, het aangedui dat hulle na afloop van die program stres beter kon kanteer as voor die aanvang van die program. Daarteenoor het die meeste toetslinge (11) in die kontrolegroep aangedui dat hulle dieselfde mate van stres as tevore ervaar het. Daar het dus nie 'n verbetering ingetree met betrekking tot hul subjektiewe vermoë om stres te hanteer nie. Dit impliseer egter nie dat hierdie groep persone tekortkominge met betrekking tot streshantering gehad het nie.

Slot

Op grond van die die objektiewe data en subjektiewe beoordeling van die proefpersone kan daar aanvaar word dat hierdie stresweerbaarheidsprogram suksesvol was in terme van streshantering vir die groep valskermspringers. Hoewel dit nie impliseer dat hul prestasies noodwendig verbeter het nie, onderstreep dit nogtans die nut van sportsielkundige ondersteuning by die voorbereiding van sportlui.

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