SOUTH AFRICAN JOURNAL OF

SPORTS MEDICINE
SPORTGENEESKUNDE

JOURNAL OF THE S.A. SPORTS MEDICINE ASSOCIATION
TYDSKRIF VAN DIE S.A. SPORTGENEESKUNDE-VERENIGING

VOLUME 6 NUMBER 2 APRIL/MAY 1991

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PUBLISHED BY THE SOUTH AFRICAN
SPORTS MEDICINE ASSOCIATION
HATFIELD FORUM WEST
1067 ARCADIA STREET
HATFIELD
PRETORIA, 0083

The journal of the SA Sports Medicine Association is published by Medpharm Publications, 3rd Floor Noodhulpliga Centre, 204B HF Verwoerd Drive, Randburg 2194. Tel: (011) 787-4981/9. The views expressed in this publication are those of the authors and not necessary those of the publishers.

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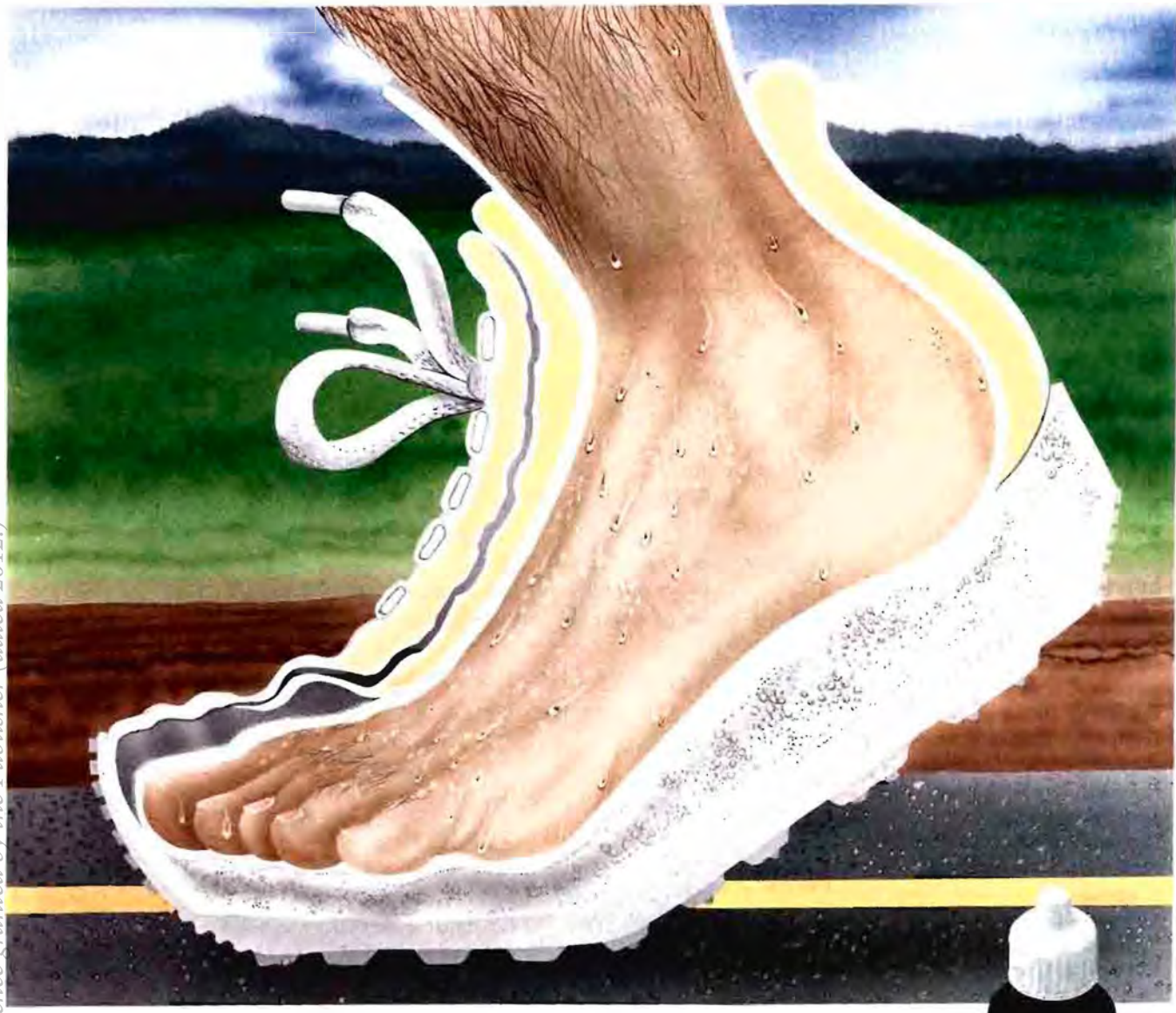

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PROGRESS IN SPORTS MEDICINE

Since I first became interested in Sports Medicine 30 years ago, many changes have taken place.

In my main field of interest, i.e. traumatology the changes have been remarkable. Take the torn meniscus for example. As a registrar we were taught to do a total meniscectomy as the only proper method of treating a torn meniscus. Numerous papers since then have shown that this form of treatment greatly increased the incidence of osteoarth-ritis and has led to many an old sportsman being crippled and even requiring knee replacement. The present technique of arthroscopic partial meniscectomy has greatly reduced the degree of osteoarthritis and more recently the repair of peripheral tears of menisci has resulted in the restoration of normal articular archi-tecture.

In the old days a patient was immobilised following this procedure for two to three weeks and was kept in hospital for at least 10 days. Today the usual treatment is early mobilisation and a day in hospital.

The operation is even being done on an outpatient basis. Many sportsmen are back at sport within 10 days.

Rehabilitation in those days consisted of straight leg raising exercises and then active mobilisation following cast removal. Now early rehabilitation using isokinetic and other techniques only allows return to activity when 90% of strength has been achieved. This has led to a remarkable reduction in reinjury.

The anterior cruciate ligament injuries in the past were often neglected because of the poor results of primary repair. This neglect led to meniscal injury and progressive ar-ticular cartilage degeneration leading to early osteoarthritis. Modern techniques with isometric positioning of the reconstructed ligament has greatly improved the results in cases of chronic instability. The use of artificial ligaments has not been clarified but in chronic instability may be a valuable adjunct in salvage cases, i.e. cases where all other techniques have failed. Added to this, the understanding of initial degeneration in transplanted tendon used in the reconstruction has made the scientific rehabilitation of these knees far safer.

Assessment of tendon strength has also helped in replacing of the damaged anterior cruciate ligament.

Isometric placement has been improved by the use of suitable jigs and other similar instruments, a great improvement on the "eye-balling" technique of yesteryear. In acute disruptions of the anterior cruciate ligament, besides adequate repair, augmentation with a tendon or artificial ligament have greatly improved results.

In collateral ligament injuries of the knee immobilization in plaster casts has greatly restored rehabilitation and return to active sport. Modern techniques using early physiotherapy and strenuous strength-exercise programmes as well as support with side-splint braces in the more severe cases have resulted in early, improved healing and return to sport.

A similar situation has occurred in ankle ligament injuries where splinting with such splints as an "aircast" brace have allowed the player more rapid mobilisation but in many cases have protec-ted the player during play without radically impeding his ability. In these days of professional sport these improvements have saved players large amounts of money.

A similar situation can be found in all aspects of sports medicine. However in many fields we still have a long way to go. In traumatology the answers to such problems as "shin splints", and osteitis pubis in runners and other sportsmen are lacking.

The prevention and treatment of degenerate joint disease in the older sportsman is an example of another area of difficulty.

I hope I'm around in 30 years time to see the future improvements in sports medicine.

Dr Clive Noble MBBCh, FCS (SA)
Editor-in-Chief

TENDINITIS

Clive Noble

Tendinitis is a "scrapheap" diagnosis in sportsmen. It is a diagnosis in many ways which is similar to "having a virus" in general medicine, sometimes correct but very often wrong. Tendinitis certainly does occur, in fact it is the commonest overload injury in the older athlete. As its name implies, tendinitis affects tendons. Any tendon in the body may be involved. The "itis" part of its name is only partially correct. Inflammation is a minor part of the pathology and its function is most likely reparative. Cellular trauma is the major pathology. This, in the chronic case, causes secondary cellular degeneration which in turn causes inflammatory cell repair. This tendon trauma is caused by cellular overloading which may be the result of a singular large overload resulting in rupture or repetitive minor overloading resulting in microtearing and secondary degeneration. It is far more common in the older athlete where the tendon progressively weakens with age. In youth tendinitis is extremely rare as here the growth areas or apophyses are the weakest link in the musculo-skeletal chain. Thus in the 9-12 age group traumatic calcaneal apophysitis (Severs Disease) is the most common site of overloading. In the 13-15 age group traumatic upper tibial apophysitis becomes more common. This is usually called Osgood Schlatters disease named after the men who described it independently in 1903.

In the 16-17 age group the pelvis apophyses become the weakest area. Here, a number of sites are involved depending on the overloaded muscular group. Usually by the age of 18 most of the growth points are fused to the main body of the bone but in the iliac crest region they may stay open as late as 22 years of age. After the age of 18, muscle is more likely to be overloaded causing muscle fibre rupture which may be either partial or complete.

From the age of 30 , tendon weakness causes the tendon to become the forerunner in the injury stakes.

The affected muscles are usually those crossing two joints such as the hamstring and quadriceps. Muscle rupture on overloading diminishes steadily after the age of 30 years with the exception of the calf muscles which may rupture well into middle age.

From the age of 30, tendon weakness causes the tendon to become the forerunner in the injury stakes. Thus tendon injury is a problem of the ageing athlete.

Tendon consists mainly of collagen and elastin fibres. In the relaxed position it is wavy in appearance but on loading can stretch to approximately 4% of its total length. On further loading rupturing of the tendon fibres occurs. Microscopically collagen fibres have a helical pattern and are joined together by cross linkages. The cross linkages are not well developed giving the tendon more plasticity. As ageing occurs these cross linkages are better developed resulting in more rigidity of tendon tissue. On overloading the cross linkages are the first to rupture and then the collagen fibres themselves.

Elastin fibres rupture last. If the overload is severe, all fibres will tear resulting in rupturing of the tendon itself. If overloading is not as great, microscopic tearing of the fibres will occur. Repair in the microtorn tendon is poor largely due to the relative avascularity and low basal metabolic rate of tendon tissue. This most likely accounts for the long duration symptoms in the area of greatest avascularity, eg. +/- 5 cm above the heel in tendo-achilles tendinitis. The other site which is extremely common is the osseo-tendinous junction, eg. tennis elbow. This is most likely a functionally weak area

and is more liable to overloading. With ageing tendon also undergoes a number of biomechanical changes and a significant reduction in water content. It is interesting to note that similar changes occur in inactive tendon. It would appear that ageing and inactivity are inter-related and it also accounts for the significant injury rate occurring in the ageing athlete who commences activity after a long period of sedentary behaviour.

SYMPTOMS

Microtearing

Pain is the most common symptom which occurs usually on loading the tendon. In the early stages pain may occur at the beginning of activity but once the tendon inversion "warms up" pain often disappears. In the more severe cases no loading is possible owing to the severity of the pain, eg. a player has to give up tennis.

Stiffness

If the limb has been kept in one position for a long period of time, movement may be difficult owing to pain and stiffness but usually on movement the pain rapidly disappears. In chronic cases actual limitation of joint movement may also be found. When pain occurs it will often radiate into the muscle which is loading the tendon, eg. down the forearm in tennis elbow and occasionally even up the arm.

Signs

Two basic signs are important:

- Tenderness - is present on palpation and may be extremely severe.
- Production of pain on stressing the muscle attached to the tendon. This stress test is positive in all cases of tendinitis, eg. in tennis elbow this may be achieved by asking the patient to extend his elbow fully and to strongly dorsiflex his wrist. Then by applying flexor pressure to the extended wrist, pain will occur in the region of the elbow. The severity of this pain is to a large extent dependent on the severity of the degeneration of tendinitis.

In mild cases the patient will complain of pain but will be able to withstand normal forces on the elbow. In cases with moderate pain the wrist will gradually give way owing to the severity of the pain. In severe cases, immediately the patient will be unable to withstand the pressure exerted on the wrist. If rupturing has occurred a defect of variable extent may be found in the tendon. In a total rupture marked weakness of the muscle will be found and in the achilles tendon rupture, compression of the calf muscle will not result in the normal movement of the foot in the relaxed position.

In recent ruptures it may be difficult to palpate the defect owing to the amount of swelling and bleeding and in chronic ruptures this may also be difficult owing to the scar tissue formation which has occurred between the ruptured area. However, marked weakness will still be the order of the day.

DIFFERENTIAL DIAGNOSIS

Pain may occur in tendons as a result of friction between the tendon and its surrounding tissue. In some areas where there is a distinct sheath, this is termed tenosynovitis. In other areas where there is no distinct sheath, paratendinitis occurs. The exact relationship between tenosynovitis/paratendinitis and tendon degeneration is not clear. In runners for example, paratendinitis is common without any form of tendinitis being present either macro or microscopically. In some areas, eg. bicipital groove of the humerus, tendon degeneration is often accompanied by a tenosynovitis. This may even lead to rupturing of the tendon possibly due to the damage of its blood supply.

TREATMENT

Tendinitis is an extremely difficult condition ot treat. Thus prevention is most important. Loading must be progressive with overloading either acutely or chronically being avoided at all costs. Strengthening exercises may well be beneficial in avoiding these injuries. Biomechanical abnormalities should be corrected eg. orthotics of anti-pronation shoes should be used in cases of achilles tendinitis and tibialis posterior tendinitis. Orthotics also appear to be helpful in cases of patella tendinitis or if there is excessive patellar mobility in association with

pronation.

When injuries occur the principles are to treat the cause and to treat the result. Thus the cause is by and large overloading, a reduction of the loading is extremely important. Jarring appears to be a major feature and therefore in severe cases all jarring should be avoided, eg. a runner should change to cycling while waiting for healing to take place. In the less severe cases reduction of loading factors i.e. hill running and street running, should be done. Loading may also be reduced by the use of biomechanical devices which take pressure off the damaged tendon while allowing the player to continue with sport, eg. tennis elbow guard which acts as a counterforce brace taking some of the load off the tendon during loading activities such as tennis. In the severe cases, however, these do not appear to have any great value. Elevation of the heel in the case of achilles tendinitis appears to similarly reduce the loading.

Treatment of the results is extremely difficult as inflammation plays a minor role in tendinitis. Non-steroidal anti-inflammatories do, however, appear to be effective especially in the minor cases. This would most likely be due to the analgesic properties.

Physiotherapy also appears to be of value in non-severe cases. The use of steroidal anti-inflammatories injected locally is controversial. It has been clearly shown that injection of cortisone into tendon can cause degeneration of tendon. It is thus important to avoid injections into the tendon but should cortisone be used, bathing the tendon appears to be beneficial. This can be assured by feeling minimal pressure when pushing on the plunger during injection into the region of the tendon. Should it be difficult to push in the plunger this usually means that the needle is in the tendon and therefore would be stopped immediately. A maximum of three injections should be given.

Surgery should only be considered in cases that have been symptomatic for more than six months, if the symptoms are of sufficient severity to warrant surgery.

In cases of rupture however, surgery still gives the best results. In the chronic case, surgery usually consists of decompression of the tendon by removing the paratendon and then multiple cuts in the lines of the fibres of the tendon to increase its blood supply, or in the case of osseo-tendinous injury, a release of the tendon from the bone provided no significant displacement occurs to that bleeding may also allow for more adequate scar tissue formation to occur with further adherence of the tendon. In general, however, most of these cases may take as long as four months before healing finally occurs. In less severe cases simple reduction of activity is usually enough to allow healing to ultimately occur. □

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DISLOCATION OF THE CERVICAL SPINE IN A RUGBY PLAYER DUE TO "CRASHING" OF THE SCRUM

AT Scher

INTRODUCTION

The phase of the game responsible for the highest number of serious rugby injuries to the cervical spine and spinal cord is the scrum. In South Africa,[1] New Zealand[2] and the United Kingdom,[3] scrumming is responsible for approximately 40% of these injuries. In a paper published in 1982[4] I reported on players injured due to "crashing" of the rugby scrum. Deliberate crashing of the two packs of forwards is an illegal manoeuvre and should not be allowed to occur.

The case history of a school boy who sustained injury as a result of this manoeuvre is presented to emphasize the dangers of the practice and illustrate the mechanism of injury.

CASE HISTORY

A 16 year old school boy playing in a position of hooker was in the process of binding with his two props prior to a scrum. The other pack of forwards having already formed up rushed at them, catching them unawares. The hooker's head was caught in an awkward position and forcibly flexed. Immediately afterwards, he developed weakness of the arms and legs and play was stopped. He was rushed to hospital, where examination revealed a quadriparesis at the C6 level with weakness of the arms and legs. X-rays of the cervical spine demonstrated anterior dislocation of C6 and C7 with bilateral locking of facets. He was immediately taken to theatre where open reduction of the dislocation together with wiring and bone grafting was performed. This operation was done within 2 hours of injury. Postoperatively he began to regain muscle power and sensation and made a good recovery, having no residual defect 6 months after the injury.

DISCUSSION

The mechanism of injury in this case as in other cases reported, is flexion force. Most cervical spinal injuries sustained in the scrum are due to flexion force, whether as the result of collapse of the scrum or as in this case, crashing of the scrum.

Figure 1: Anterior dislocation of C6 and C7 with bilateral locking of facets, similar to the injury described in the case history (the original X-ray is unobtainable).

The force or "impact weight" exerted as the packs meet is considerable and, if the front rows are not correctly positioned, a dangerous situation may arise. Hodge[5] has shown experimentally that the force exerted on impact is approximately 80% of the maximum generated during the scrummage. Using an 8-man pack with a combined mass of 727 kg the force recorded on impact was 746 kg.

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Deliberate crashing in an attempt to intimidate or unsettle the opposing pack is a contravention of the rules, as clearly stated in The Laws of the Game of Rugby Football.[6] Law 20 deals with the scrum and reads: "It is dangerous play for a front row to form down some distance from its opponents and rush against them".

PREVENTION

Prevention of the above injuries depends on educating players to an awareness of the dangers of crashing, especially as serious cervical spinal injury is more prevalent in inexperienced players. Vigorous enforcement of the rule quoted is of obvious importance. The referee should ensure that the ball is not put into the scrum until it is properly formed, with the front row players bound.

It is encouraging to note that Footnote 5, appended to Law 20 which previously read[7] *"The referee has no authority to permit delay in putting in the ball because a player has not succeeded in getting his head down in the scrummage"*, has been changed to Footnote 10 in the most recent edition of the Laws of the game of Rugby Football.[6] This Footnote now reads, *"In the interest of safety, the referee has authority to permit a delay in putting in the ball if a player in the front row has not succeeded in getting his head down in the scrummage, but otherwise he should ensure that there is no delay"*.

A specific amendment of the rules which would remove this hazardous practice has been suggested.[8] This is the adoption of the "staggered scrum" in which the front rows of each team first bind together and pack down. Only then are the locks and loose forwards allowed to join the scrum. The impact force is thus removed and the total impetus is less than that generated in conventional rugby scrum. □

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IRON STATUS AND ATHLETIC PERFORMANCE

Mieke Faber

FUNCTIONS OF IRON

Iron is an essential component of the oxygen transportation compounds haemoglobin, myoglobin, and the cytochromes (Williams, 1984) as well as various enzymes (Steinbaugh, 1984). An iron deficiency can lead to impaired haemoglobin synthesis and a reduction in oxygen transport (O'Neil *et al*, 1986).

IRON DEFICIENCY ANEMIA

Iron deficiency may be explained by decreased iron absorption and increased iron loss (O'Neil *et al*, 1986). Iron can be lost through sweat losses, intestinal losses and haematuria. Other causes for iron deficiency are increased demands for increased total body haemoglobin and poor dietary intake (ADA Report, 1987).

Iron deficiency can impair physical performance, particularly of an aerobic nature (Williams, 1984). Iron depletion without anemia can also affect physical performance (Steinbaugh, 1984). Such impairment can be manifested as a reduction in total exercise time, increased heart rate, decreased oxygen uptake, increased blood lactate concentrations and decreased work tolerance (O'Neil *et al*, 1986).

Low serum iron, haematocrit and haemoglobin levels have been reported among athletes (O'Neil *et al*, 1986) as well as low iron stores (ADA Report, 1987). Women are more likely to become iron deficient as compared to men because of a lower energy intake and because of iron losses due to menstruation (Williams, 1984). Deficient iron stores result in weakness, fatigue, pallor, dyspnea on exercise, palpitations and prolonged restoration of cardiorespiratory function to pre-exercise level (O'Neil *et al*, 1986).

It has been recommended that female athletes take a multivitamin with iron to insure an adequate intake. It is extremely unlikely for this amount of iron to cause any side effects (Vitousek, 1979).

The American Dietetic Association recommended that if an individuals iron intake remains low after careful monitoring of the diet, a moderate iron supplement, containing the 18 mg RDA should be taken (ADA Report, 1987). Since ascorbic acid enhances iron absorption, an iron supplement should be taken in conjunction with a source of ascorbic acid (O'Neil *et al*, 1986). It is however recommended that plasma ferritin levels be checked before supplementation is prescribed (ADA Report, 1987). Iron supplementation to individuals with iron deficiency anaemia will increase performance capacity (Williams, 1984). However, iron supplementation has no beneficial effect for an athlete with normal haemoglobin levels (Williams, 1984). Large doses of iron supplementation are of no value to athletes, unless there is a measurable iron deficiency. Large iron supplements may cause nausea, gastric upset, constipation and accumulation of iron in the tissues (Vitousek, 1979). Excessive iron intake can also inhibit the absorption of zinc due to their similar physiochemical characteristics (McDonald and Keen, 1988).

SPORTS ANEMIA

Sports anemia or dilutional anemia results from an in-

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crease in red blood cell mass and plasma volume. Causes for sports anemia are adaptive response to maximise oxygen transport, haematuria, haemolysis, iron loss through sweating, inhibition of erythropoiesis, increase in blood volume, decreased iron absorption (O'Neil *et al*, 1986), increased erythrocyte osmotic fragility, causing reduced red blood cell survival time and a possible shift in the oxygen dissociation curve (ADA Reports, 1987). Sports anemia is both normocytic and normachromic (ADA Report, 1987). While some believe that marginal iron intakes may play a role in the development in sports anemia (ADA Report, 1987), others believe that a deficient iron intake is not the cause since the haemoglobin levels does not respond to iron supplementation (Vitousek, 1979). Sport anemia is transitory (O'Neil *et al*, 1986). Endurance performance is not impaired by sports anemia (O'Neil *et al*, 1986). No specific treatment is required and iron supplementation is unnecessary (O'Neil et al, 1986).

FOOD SOURCES OF IRON

Organ meats, especially liver, are by far the best sources of iron. Other sources include meats, egg yolk, whole wheat, seafood, green leafy vegetables, nuts and legumes (Williams, 1973). □

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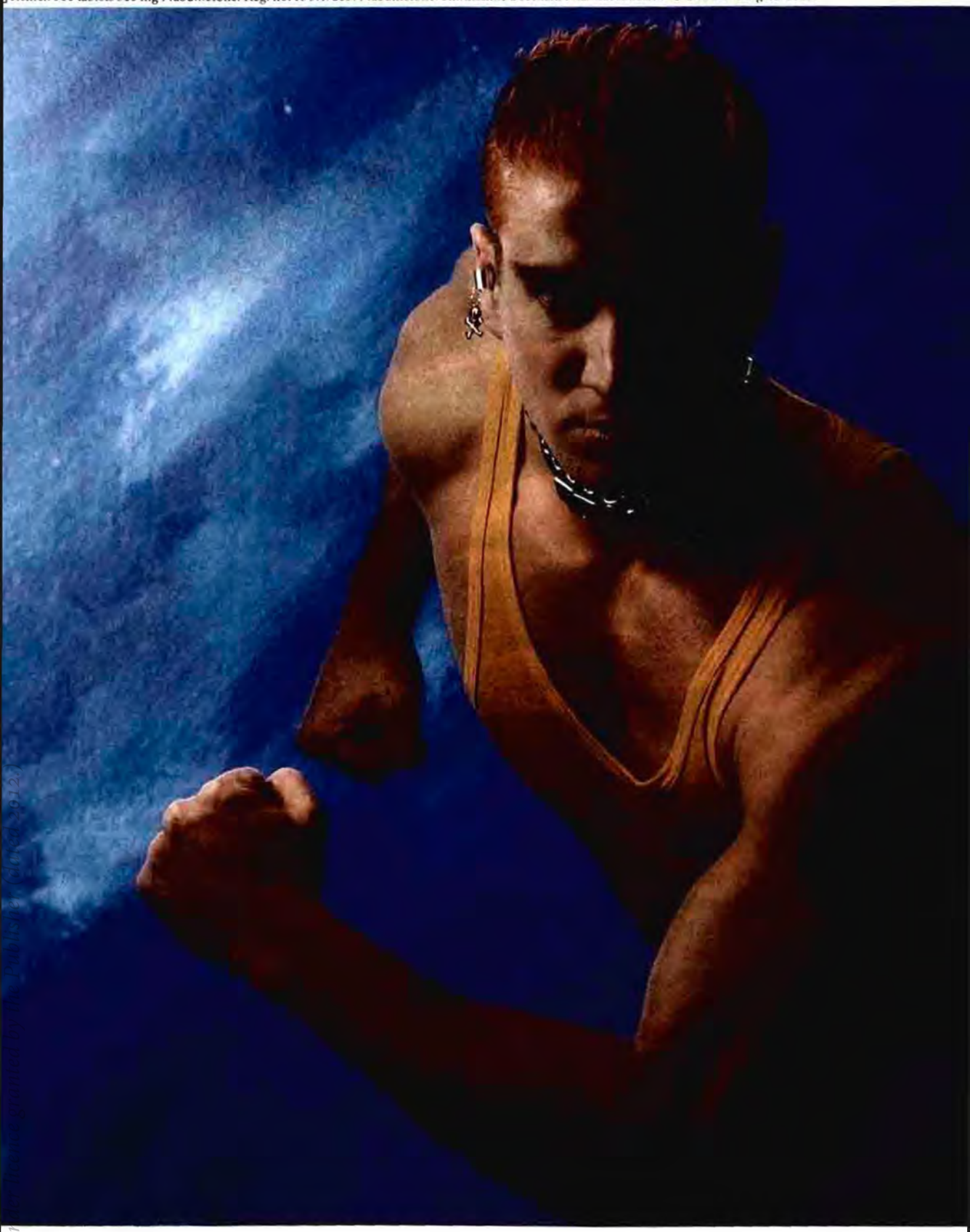

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THE PHYSIOTHERAPY MANAGEMENT OF CHRONIC MUSCLE TEARS OF THE CALF IN LONG DISTANCE RUNNERS

The Comrades Marothon is just around the corner and it is a good time to discuss the common condition of calf muscle tears.

This injury is well documented by Prof Noakes in his book "Lore of Running". Usually a runner will give a history of having increased his training of both distance and speed. Amazingly common is the fact that the veteran runner will confess he is wearing shoes which have lost heel height due to age. Surely the micro trauma which develops in the calf muscle, must be aggrevatred by this loss of heel height as the foot strikes the ground? The onset of pain is gradual. The first stage is Grade 1 where the pain is felt after the run and disappears druing rest. Grade 2 pain is felt at the beginning of the run (as well as after the run) but disappears as the muscles warm-up. By the time the pain has reached Grade 3, i.e. pain during the entire duration of the run, the runner is no longer able to train. He either stops running for a short time hoping that the pain will disappear with rest, or he seeks help. Rest, however, does not cure this problem. The muscle fibres run perpendicular and parallel to each other. If a micro tear develops and is continually traumatized, it will increase in size and heal with scar tissue. Unfortunately as the runner continues to run, the tissue continues to become inflamed, more scar tissue is formed and the muscle fibres stick together. The muscle no longer functions biomechanically.

On examination the physiotherapist, palpating with her fingers, will find an exquisitely tender area in the muscle belly, surrounded by an area of protective muscle spasm. The treatment for this injury is transverse or cross frictions. The physiotherapist massages the muscle fibres at right angles to their length, in order to separate the muscle fibres, and achieves the result of a small malleable scar which is no longer a problem. Electrical modalities such as laser, interferential, ultrasound and pulsed shortwave diathermy are included in the treatment in order

to decrease inflammation and promote healing. Transverse frictions are only given every second day. They are extremely painful for the patient but are the key to the success of the treatment.

Physiotherapists advise runners on their shoes and future training methods. If necessary the runner is referred to an orthotist for further advice.

It is felt that one of the reasons that the long distance runner has a tendency to re-injure the original injury, is that there is a weakness in the muscle group. If the physiotherapist has access to an isokinetic testing machine, the runner is not discharged until the muscles have been tested for strength and endurance comparing left and right leg, as well as comparing the muscle balance between the plantarflexors (primarily gastrocnemius and soleus) and the dorsiflexors (primarily tibialis anterior). When the weakness has been identified, an exercise regime is given to the runner. This programme should include isokinetic and isotonic exercises for rapid results.

Finally the runner is taught the correct stretching methods. Many runners feel that to stretch is to waste valuable running time. Stretching, especially as one gets older and loses some of one's youthful natural elasticity, is essential. The gastrocnemius passes over the knee joint and the ankle joint. to stretch it, the knee is hyperextended and the foot is dorsiflexed. Soleus on the other hand only passes over the ankle joint. To stretch it, the knee is flexed and the foot is dorsiflexed. Stretching should take place after a 10 minute warm-up run. Each muscle is slowly stretched for 30 seconds. As the inverse stretch reflex is activated, the muscle can be stretched slightly more for another 30 seconds.

At the end of the run, the runner is encouraged to warm-down slowly and not to just stop running. In this way muscle soreness is avoided and muscles are being prepared for the next run. □

SASMA NEWS

In this issue the position statement of the South African Sports Medicine Association is published. This opinion is underscribed by the Medical Association of South Africa, and is a milestone in the activities of the Sports Medicine Association insofar that it expresses the concern of the health implications of the use and abuse of potentially harmful medications in the quest for better performance in sport.

Research done in South Africa has shown a dramatic increase in the illegal use of anabolic androgenic steroids by South African sportsmen.[1] It may well be argued whether the authorities are doing enough to combat this evil, because world champion athletes called for strong action against offending athletes: "We consider doping to be the most shameful abuse of the Olympic ideal: we call for the life ban of offending athletes, we call for the life ban of coaches and the so-called doctors who administer this evil". (Sebastian Coe, Olympic Congress at Baden-Baden, 1986).

Tydens die 4de Sportgeneeskunde kongres in Sun City, was daar baie vrugbare samesprekings gevoer insake die gebruik van opkikkers in sport. Gesien in die lig van Suid-Afrika se hertoetrede tot wêreldsport, is dit belangrik dat daar 'n eenvormige beleid in ons land geformuleer moet word insake die hantering van hierdie netelige probleem. Alle sportliggame moet saamstaan om 'n verenigde front te vorm om hierdie euwel te bestry tot voordeel van sport en die gesondheid en welsyn van ons sportlui. Geneeshere, aptekers en afrigters moet ingelig wees insake die nadelige effekte wat die gebruik van opkikkers het op die moraal en ideaal van sport. Persone wat anaboliese steroïede voorsien aan sportlui, moet genadeloos gestraf word terwyl atlete wat hulle skuldig maak aan hierdie misdryf die maksimum vonnis opgelê moet word in 'n poging om ons sport te vrywaar van hierdie euwel.

Die Suid-Afrikaanse Sportgeneeskunde Vereniging doen 'n beroep op die sportgemeenskap om kragte saam te snoer om ons huis in orde te kry sodat ons toegerus is op alle terreine om die uitdagings te aanvaar wat internasionale kompetisies aan ons sportlui bied.

TYPES OF MUSCLE CONTRACTION AS BIOMECHANICAL CONCEPTS

MC Siff

Keywords: *Muscle contraction, isotonic, isometric, isokinetic, biomechanics.*

ABSTRACT

The terms isotonic, isometric and isokinetic commonly used to describe different types of muscle contraction are biomechanically inappropriate in most exercise situations. The less impressive terms 'static' and 'dynamic' offer more accurate superordinate categories. The implications for isokinetic physiotherapy machines and rehabilitation are discussed.

INTRODUCTION

Several terms used in exercise science to refer to the different types of muscle contraction are not applied with the level of correctness they warrant. Among the most casual employed are those beginning with the prefix *iso-*, namely *isokinetic, isotonic* and *isometric*. Historically, they were invented to describe types of contraction in which some property remains the same: velocity, tension or length. The consistency of these logical words led to their rapid acceptance, with little criticism of their precise meaning and scope ever taking place.

It is the purpose of this article to examine and redefine these terms with particular reference to their biomechanical origins, so that their scope may be appreciated more thoroughly and they may be used with greater precision in describing muscular activity. In doing so, the advisability of continuing to use these terms is considered.

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ISOKINETIC TERMINOLOGY

The term 'isokinetic' is encountered in two contexts: *firstly,* exercise physiology textbooks sometimes regard it as a specific type of muscle contraction, and *secondly*, so-called isokinetic rehabilitation machines are often used by physiotherapitsts.

Use of this term is dubious in both contexts, for reasons which become clear on examining the fundamental biomechanics describing this concept. Isokinetics (*Iso* - 'same', *kinetics* - 'velocity') literally means movement which occurs at *constant velocity.*

If *isokinetics* is defined to be a specific form of muscle contraction, along with static (*isometric*), and dynamic (*concentric* and *eccentric*) contraction, then it must be asked how isokinetic contraction, also a dynamic activity, differs from concentric and eccentric contraction.

Concentric refers to muscle contraction which brings the origin and insertion of a muscle closer, or when muscle action produces a force which overcomes the load being acted upon. For this reason, Soviet scientists use the term *'overcoming'* contraction or movement. The term *eccentric* refers to contraction which increases the distance between

the origin and insertion of a muscle, or, in other words, when the force of contraction succumbs to the load. The Soviets call this '*concessive*' contraction. Both of these types of contraction can occur at varying or constant velocity, so that it is difficult to classify isokinetic contraction as a totally discrete form of muscle contraction.

This means that there can be *isokinetic concentric* action and *isokinetic eccentric* action. Isokinetic muscle contraction is not a discrete entity. Isokinetic action is merely a particular case of dynamic action. The word 'action' has intentionally been chosen here, since distinction must be made between the external *action* and the internal *contraction* of the muscle. What the textbooks invariably refer to is isokinetic action, or constant angular velocity, of the moving limb, and not the biomechanical process within the muscle.

It is impossible in the clinical or training setting to measure the contraction velocity of a muscle deep within the body, so that most laboratory analysis of muscle contraction has been on biopsied muscle. Even under these circumstances, contraction is usually elicited by electrical stimulation under experimental conditions, so that it is generally invalid to discuss *isokinetic contraction* of muscle during natural movement.

ISOKINETIC MACHINES

Application of the term 'isokinetic' to specific physiotherapy machines is beset with similar problems. By definition, *it is biomechanically impossible to design a purely isokinetic machine.* Since *isokinetic* means 'constant velocity', then these machines must constrain the patient to move a limb at constant velocity from beginning to end of range of the movement.

This is impossible, since the patient has to start his limb from rest and push against the machine until it can constrain the motion to approximately constant *angular* velocity. Now, if any object moves from rest to any velocity, Newton's first two Laws of Motion decree that a force and an acceleration must be involved - and the existence of any acceleration means that there can be no constant velocity. When the patient reaches the end of the range of movement, limb and machine arm stop mementarily, then change direction. The presence of any deceleration or change of direction heralds the absence of constant velocity.

This implies that isokinetic action is not possible during initiation or termination of any movement. Thus, all isokinetic machines are able to produce approximately isokinetic action *only over the middle of the range of movement.* Consequently, these machines cannot offer realistic evaluation or therapy in the transition zones near the extremes of the movement range, regions where injuries commonly occur.

Therefore, *there is no such entity as a true isokinetic machine.* So-called isokinetic machines actually generate resistance which is a function of the applied muscle force. It would be more accurate for manufacturers to refer to their 'isokinetic' devices as *semi-isokinetic* machines. It is unlikely that this will happen, so it is the responsibility of physiotherapists to appreciate the limitation of these machines when they use them therapeutically.

One of the very few occasions when isokinetic action takes place is during isometric contraction. In this case, the velocity of limb movement is constant and equal to zero. Approximately isokinetic action also occurs during mid-range movement phases in swimming and aquarobics, with water resistance (which is proportional to the square of velocity of movement) serving to limit any increase in velocity.

ISOTONIC TERMINOLOGY

The term *isotonic*, which means 'same tone', should be limited or avoided under most circumstances, since it is virtually impossible for muscle tension to remain constant while joint movement occurs over any extended range. Constancy is possible only over a very small range under very slow or *quasi-isometric* conditions of movement. Naturally, constant tone also exists when a muscle is at rest, a state referred to as *resting tonus.* Whenever movement occurs, muscle tension increases or decreases, particularly if acceleration is involved or one of the stretch reflexes is activated.

The West Germans and Soviets prefer to use the term *auxotonic*, which refers to muscle contraction involving changes in muscle tension and length.[2] Other authors prefer to

use the term *allodynamic*, from the Greek 'allos' meaning 'other' or 'not the same'.[1]

The term 'dynamic' is sufficiently precise to describe the type of contraction in question and should be used whenever the word *isotonic* is intended to mean any form of dynamic, or non-isometric, contraction. The term *isotonic* should be reserved for the highly limited, short movement range situations where muscle tension definitely remains constant.

ISOMETRIC CONTRACTION

Even the concept of isometrics is not as simple as it appears. *Isometric* literally means 'same length', a state which occurs only in a muscle at rest. Actually, it is not muscle length, but joint angle which remains constant.

Contraction means 'shortening', so that isometric contraction, like all other forms of muscle contraction, involves sliding of actin and myosin muscle filaments relative to one another. Isometric contraction may be defined more accurately by reserving it to mean muscle contraction which occurs when there is no external movement or change in joint angle (or distance between origin and insertion). The term *isometric* may be replaced by the simple word 'static', without sacrificing any scientific rigour.

Furthermore, all isometric contractions are not the same. Distinction may be drawn between *rapid initiation* and *slow initiation* isometric contractions, where the former refers to isometric contraction occurring when a muscle contracts rapidly against an imposed load, and the latter refers to contraction produced gradually against a load. Similarly, it is useful to recognise *rapid termination* and *slow termination* isometric contractions, which take into account the rate at which the load is withdrawn. Recruitment of muscle tissue and the various stretch reflexes is different in all of these cases, so it is important to be aware of these differences when isometric rehabilitation or training is being considered.

CONCLUSION

If the term isokinetic, isotonic and isometric are to be used to describe specific types of muscle activity, then it is essential that the scope and limitations of each term are appreciated, particularly since casual usage by exercise professionals, gymnasium users and equipment manufacturers has led to human movement and physical conditioning being explained and analysed in a biomechanically inaccurate manner. Although the term *static* and *dynamic* may lack the more authoritative tone of the *iso*-prefixed words, they offer the average exercise scientist and fitness professional adequate re- placements for *isometric* and *isotonic*, respectively. Isokinetic activity may then be regarded as a special case of dynamic activity in which the resistance is a function of the applied muscle force. □

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SPARE YOUR JOINTS

Sport trauma is a common event. Painful joints and muscles and tendons must surely be regarded as an "early warning signal" that all is not well in the musculoskeletal or locomotor systems.

The pain, and the often associated tenderness and swelling, are intended by Nature to slow us down, to urge us to take recuperative measures, to "go quiet" - for a sufficient period to allow for the re- establishment of full physiological and functional integrity.

POWERFUL PAIN RELIEVERS AVAILABLE

Powerful painkillers are available, and the indiscriminate use of such analgesics is a real concern in sport medicine. Why? Because such analgesics may mask the warning signals so effectively, that excessive activity is resumed before healing has had sufficient time to occur.

NON-STEROIDAL ANTI-INFLAMMATORY ANALGESICS

The non-steroidal anti-inflammatory analgesics (NSAIAs) are very widely used to provide pain relief in the event of sport injuries. These medicines interfere with the biochemical and cellular components of the inflammatory response to trauma. The damage is the stimulus that evokes the inflammation and pain, and these NSAIAs interfere with the chemical mediators of the inflammation and the pain.

INFLAMMATION - GOOD AND BAD

While it is true that the inflammatory response - if too enthusiastic, may itself promote damage, yet it must be regarded as part of the healing process. Thus, suppression of the inflammatory response is likely to suppress or delay healing, and because the pain relief if often very notable, physical activity is recommenced prematurely.

JOINTS PARTICULARLY AT RISK

While the repercussions of such premature physical activity may not be too significant for soft tissue injury, it is considered to be highly significant for joints - where permanent and recurrent damage may be produced.

THE MESSAGES SHOULD BE CLEAR

- Pain, swelling, tenderness, inflammation in the musculoskeletal system is a signal to "go slow".
- NSAIAs are often excellent pain relievers, that also suppress components of the inflammatory response.
- NSAIAs can provide an entirely false sense of recovery, and so permit premature return to full physical activity.
- Joints in particular, need time to recover from injury, otherwise cumulative permanent damage may accrue.
- Circumspect use of these NSAIAs by well informed persons hold few dangers in terms of damage to joints and soft tissues of locomotion.
- Be sensible - be kind to your joints.

Note:

The Non-Steroidal Anti-Inflammatory Analgesics (NSAIAs), constitute all those medicines which function in a manner akin to aspirin to reduce pain, fever and inflammation.

NSAIAs currently available in RSA include preparations that contain any one of the following:

aspirin, azapropazone, bumadi-zone, diclo-fenac, diflunisal, feno-profen, fenbufen, flurbiprofen, ibuprofen, indometchacin, keto- profen, meclofenamate, mefenamic acid, nabumetone, naproxen, oxaprozin, oxyphenbutazone, phenylbutazone, piroxicam, sulindac, tenoxicam, tiaprofenic acid, tolmetin.□

ABSTRACTS FROM THE CLINICAL JOURNAL OF SPORTS MEDICINE

The official Journal of the Canadian Academy of Sports Medicine

Injury Profiles in Wheelchair Athletes: Results of a Retrospective Survey

DAR McCormack, DC Reid, RD Steadward and DG Syrotuik Department of Physical Education and Sports Studies, Department of Athletics, and the Glen Sather Sports Medicine Clinic, University of Alberta, Edmonton, Alberta, Canada

Little is known about the nature and mechanism of sports injuries among wheelchair athletes. The purpose of this study was to develop an injury profile for this group. A total of 90 wheelchair athletes were surveyed retrospectively by means of a questionnaire. Three hundred and forty-six injuries were reported in 18 different sports, 107 (30,9%) of which occurred in basketball, 106 (30,6%) in track, and 42 (12,1%) in road racing. Eighty percent of the athletes were competitive, with 60% competing at a national level. Most of the athletes trained 6-10 h/week. Injuries to the soft tissue of the upper extremities were most common, with the hand involved 21,3% and the shoulder 16,7% of the time. Blisters and abrasions accounted for 47,7% of the injuries. All of the athletes surveyed sustained injuries, yet little protective gear was worn except for gloves (60%). In view of the potentially serious long-term complications of some of these injuries, and the corresponding impact of the ability of these athletes to carry out their functions of daily living, prompt diagnosis and treatment are mandatory. Despite this, less than one-third (30,8%) of all wheelchair athletes sought medical assistance for their sports injuries. **Key Words:** Wheelchair sports - Disabled sports - Spinal cord injury. *Clin J Sport Med* 1991; **1(1)**: 35-40.

Muscle RQ and Lactate Accumulation from Analysis of the V_{CO2}-V_{O2} Relationship During Exercise

William L Beaver and Karlman Wasserman, Division of Respiratory and Critical Care Physiology and Medicine, Department of Medicine, Harbor-UCLA Medical Centre, UCLA School of Medicine, Torrance, California, USA.

We analyzed the V_{CO2}-V_{O2} relationship derived from 1 min incremental (15 W/min) exercise tests of 10 normal subjects. The curve was quite linear below the anaerobic threshold (AT). We deduce that the slope must equal the respiratory quotient (RQ) of the exercising muscles, with a mean value for these subjects of 0,97 +/- 0,06, indicating that the metabolic substrate is essentially glycogen. Beyond the AT, respiratory CO_2 output rises at a faster a rate above as compared to below the AT, reflecting the rate of HCO_3 - buffering of lactic acid. Projecting the straight line of V_{CO2} versus V_{O2} below the AT into the region beyond the AT provides an estimate of the V_{CO2} due to continuing aerobic metabolism. The difference between the actual V_{CO2} and the aerobically produced V_{CO2} (excess V_{CO2}) describes the rate of CO_2 generated from HCO_3 - buffering of lactic acid. The integrated excess CO_2, corrected for any hyperventilation, provides a measure of the quantity of HCO_3 - depletion and thus lactate accumulation. Since our measurements are non-steady state, a dynamic simulation model of total lactate accumulation and arterial lactate concentration, based on excess CO_2 output and compartmental blood flows and volumes, was developed and found to predict experimental results of lactate concentration increase. Thus, the excess CO_2 output can be a useful measure of lactate accumulation and, with the developed model, serve to describe

the rise in arterial lactate concentration during a progressively increasing work rate test. **Key Words**: CO_2 production - Excess CO_2 - Lactate accumulation - Lactate concentration model - Lactate threshold - Muscle "RQ". *Clin J Sport Med* 1991; **1(1)**: 27-34.

Clinical Evaluation of Shoulder Instability

Richard J Hawkins, MD, and Nicholas GH Mohtadi, MD, Department of Orthopaedic Surgery, University of Eastern Ontario and University Hospital, London, Ontario, Canada; and University of Calgary Sport Medicine Centre, Calgary, Alberta.

The shoulder is the most unstable joint in the body. Patients with shoulder instability may present in a variety of ways, ranging from subtle complaints of pain including the "dead arm syndrome", to those with subjective instability, apprehension, or even those who can voluntarily demonstrate a dislocation. Instability can be classified according to the onset (traumatic, atraumatic, overuse), the direction (anterior, posterior, multidirectional), the timing or frequency (acute or recurrent), the degree of instability (subluxation or dislocation), and whether it occurs voluntarily or not. The physical examination should be directed toward ruling out other problems and determining whether generalized ligamentous laxity is present. Specifically, the stability assessment includes apprehension tests, the relocation test, or Fowler's sign and an estimation of glenohumeral translation. The use of a local anaesthetic injection into the subacromial space can be very helpful in guiding the physical examination. An accurate diagnosis of shoulder instability can usually be made on a clinical basis by utilizing the information outlined in this report. **Key Words**: Apprehension tests - Glenohumeral translation - Relocation tests. *Clin J Sports Med* 1991; **1(1)**: 59-64.

Isokinetic evaluation of quadriceps and hamstring symmetry following anterior cruciate ligament reconstruction.

Harter RA, Osternig LR, Standifer LW. *Arch Phys Med Rehabil* 1990; **71**: 465-8.

Isokinetic muscle parameters are commonly measured after anterior cruciate ligament (ACL) reconstruction to determine the dynamic status of the knee as well as monitor progress in rehabilitation. In this study, the symmetry of the quadriceps and hamstrings musculature in postsurgical and contralateral normal limbs of subjects who had undergone one of two types of ACL reconstruction was evaluated. In addition, subjects were evaluated for differences on selected isokinetic parameters between types of surgery and lengths of postoperative periods. Forty six subjects aged 18 to 49 years (mean, 23.7 years) postsurgical and normal contralateral limbs were divided into groups according to type of autogenous intraarticular ACL substitute and length of postoperative period. From the results of paired tests and analyses of variance it was evident that significant asymmetries between limbs for all measures of quadriceps and hamstrings musculature strength and endurance existed ($p < 0.001$) irrespective of the type of reconstruction technique. Average surgical knee deficits in hamstrings endurance were significantly less for the long-term (41 to 101 months) follow-up group (1.9%) than for the intermediate (24 to 40 months) group (12.1%). It would seem that extended periods of time are required to approximate hamstrings endurance symmetry after ACL reconstruction. In these subjects, assymmetries between postsurgical and contralateral normal limbs may reflect either incomplete rehabilitation or an inability to regain full isokinetic strength and endurance following ACL reconstruction.

MASA TAKES STAND AGAINST DISPENSING OF ANABOLIC STEROIDS

MASA has come out strongly against the dispensing of anabolic steroids, warning the profession it considers making these drugs available to the public for non-medical reasons in a serious light.

At present the biggest dilemma faced by doctors is that there is little indisputable evidence concerning the effect of high dosage or extended usage of anabolic steroids on athletes' health, as research with high dosages of these substances is not permissible.

However, the South African Sports Medicine Association (SASMA), a special interest group of MASA, is of the opinion - based on a comprehensive literature survey and a careful analysis of the claims concerning the psysiological, physical and psychological effects and adverse effects of anabolic-androgenic steroids - that their potential medical use is limited and can be replaced with safer and more effective drugs.

SASMA believes that the medical supervision of sportsmen should primarily be directed to clinical diagnosis and treatment of patients to restore normal health and function rather than to scientific methods or to enhancement of performance.

The Association has formulated a Position Statement on the use and abuse of anabolic steroids, which has been adopted by MASA as official policy on the issue and includes the following recommendations:

- Serious and continuing efforts should be made to educate athletes, coaches, trainers, physicians, parents, physical educators and the general public regarding the limited benefits and potential risks of steroid use as documented by scientific research.

- All sports governing bodies (including the body building association) must uniformly reject the use of anabolic-androgenic steroids on the basis of ethics and the ideals of fair play of competition and because their use gives possible competitive advantages harmful to the user.

- A national drug testing programme should be instituted by all sport governing bodies to be a deterrent to steroid usage and testing should equally encompass all levels of sportsmen. For this reason an accredited laboratory should be appointed to conduct the testing and the testing procedures should be standarised throughout the country.

- Consideration should be given to re-evaluating the medical use and indication for the anabolic-androgenic steroids, by the Medicines Control Council in view of rescheduling or even banning these drugs for medical or non-medical reasons.

There is currently no method for predicting which individuals are more likely to develop adverse effects, some of which are potentially hazardous.

- Doctors should not be allowed to prescribe these drugs in non-pharmacological doses to obviously healthy athletes with the sole intention of increasing in an artifical and unfair manner their performance in competition.

- Pharmacists, veterinarians, pharmacological companies and other suppliers should be strongly discouraged and prohibited to supply any sportsman or sports club with anabolic steroids without a valid prescription. Should instances come to light where athletes were illegally supplied with these potentially harmful drugs, the necessary disciplinary steps should be taken.

- Doping control in South Africa for all national sporting bodies should be coordinated and standardised by an independent body with medical, scientific and sport representation to comply with international standards and the rules of the International Olympic Committee Medical Commission. □

South African Sports Medicine Association

Suid-Afrikaanse Sportgeneeskunde-Vereniging

A Specialist group of the MASA (incorporated association not for gain)
'n Spesiale groep van die MVSA (ingelyfde vereniging sonder winsbejag)

PO Box 13206
CLUBVIEW
0014
May 1991

Dear Doctor,

The characteristic that distinguishes a professional from an educated person is the professional person's desire and responsibility to stay abreast of the developments of his or her field of expertise. The halflife of our professional knowledge is about 3 years, and for this reason it is vitally important to continuously refresh and supplement our knowledge.

Sports Medicine, the official mouthpiece of the SA Sports Medicine Association, has over the years become a treasured source of knowledge for the health care professional in the sports medicine arena. The journal features not only original research papers and articles by leading specialists in sports medicine, but also current news and relevant abstracts.

The publishing of **Sports Medicine** has been taken over by Medpharm Publications. The journal will be published quarterly namely, February, May, August and November.

Due to the present rate of inflation SASMA are no longer able to distribute **Sports Medicine** free of charge and have reluctantly introduced a subscription fee of R20,00 per annum (Members of SASMA will continue to receive the publication free of charge). In addition to managing costs, this will enable the editorial board maintain a high quality editorial content and render a more effective service to you, the health care professional.

Your subscription to **Sports Medicine** is an investment in your future and the future of the health care profession. We count on your continued support.

Regards,

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NOTE

* Full membership: Medical practitioner who is a member of MASA

** Associate membership: Members of supplementary Health Service professions, registered with the SAMDC and who are members of their own professional associations

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