

# Editorial

## The future of the professional practice of Sports Medicine in South Africa



I am frequently asked by young physicians considering a full-time career in Sports Medicine to comment on the future of postgraduate training, professional practice, career opportunities and financial sustainability of Sports Medicine in South Africa. In this editorial I would like to comment on these areas and to suggest how Sports Medicine may possibly evolve as a profession in the next decade.

I am writing this editorial while attending the European Federation of Sports Medicine meeting in Innsbruck, Austria, as a member of the Executive Committee of the International Federation of Sports Medicine (FIMS). During the opening address of this meeting the current President of the European Federation of Sports Medicine, Professor Norbert Bachl, indicated that one of the aims of the European Federation is to standardise the postgraduate training and registration of specialists in Sports Medicine across the European Union. The global recognition of Sports Medicine as a specialty has also been identified and recognised for a number of years by the FIMS Education Commission on which I served from 1992 to 1998. A number of countries already recognise Sports Medicine as a specialty — the question is what is the position in South Africa?

At the SASMA Conference in 1996 the first tentative step in the direction of advancing specialist Sports Medicine training and registration was taken. At this meeting a steering Committee of the 'South African College of Sports Physicians' was formed. This Committee has been working to establish contact with the Health Professions Council of South Africa, and the Colleges of Medicine of South Africa (CMSA). A draft proposal for a new College of Exercise and Sports Medicine is presently being finalised, and will be submitted to the CMSA for consideration. This proposal was discussed at the second general meeting of the 'South African College of Exercise and Sports Medicine' in Midrand a few weeks ago. This proposal is based on similar documents that are under discussion by professional bodies in the UK, Australia, and New Zealand. Although Sports Medicine still has a road to walk before a full specialist curriculum, examination procedures and full registration of specialist sports physicians are finalised, I predict that this process could well be complete in South Africa within the next 5 years.

In this interim period I advise prospective sports physicians to use every opportunity to advance their professional training by completing existing postgraduate academic train-

ing programmes, joining the professional national and international organisations, and registering with the group that formed the Interim South African College of Exercise and Sports Medicine. Once the curriculum and examination procedures of a newly formed College are finalised, it is very likely (and I support this fully) that every prospective specialist will have to complete a Fellowship examination. I am not in favour of any 'grandparent' clause that will allow Fellowship registration without a final examination.

It is clear that only those who are truly dedicated to the profession and who are willing to sacrifice time and effort to complete their training will reap the benefits of professional recognition. This recognition is essential for the continued growth and development of true professional sports physicians.

**Martin P Schwellnus**

*Editor-in-Chief*

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# Exercise-induced leg pain: a review of 34 cases of chronic exertional compartment syndrome

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## Abstract

**Objective.** To review the presentation of chronic raised intracompartmental pressure as a cause of exercise-induced claudication in the young fit athletic community.

**Design.** Retrospective review of the records of patients diagnosed as having chronic exertional compartment syndrome over a 2-year period.

**Setting.** A tertiary referral centre at the University of Natal Medical School.

**Results.** Thirty-four patients met the diagnostic criteria for chronic exertional compartment syndrome based on direct measurement of intracompartmental pressure. All patients presented with exercise-induced pain that was relieved by rest. Thirteen patients had associated motor deficit and 13 had sensory deficit. The pain was bilateral in 29 patients (79%). The pathophysiology, diagnosis and treatment of the condition are discussed. The condition appears to be under-diagnosed and should be considered in patients with claudicant-type pain that does not respond to conventional physiotherapeutic measures.

## Introduction

Exercise-induced limb pain in the fit athletic population is relatively uncommon, and many of these cases are referred for physiotherapeutic management without an accurate diagnosis having been made. This places an onus on the physiotherapist to reassess the patient's complaints, make the diagnosis and initiate the appropriate treatment. The causes of exercise-associated limb pain are well documented and include delayed onset muscle soreness, referred pain, pain of bone origin, pain arising from the muscular tendinous interface (shin splints), muscle and/or tendon injury, pain of vascular origin and nerve entrapment.

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It is our experience, based on referral patterns, that a minority of patients with exercise-associated leg pain do not respond to physiotherapy or the provision of orthotics. A reason for this is that the initial diagnosis and subsequent treatment may have been incorrect. The most common error that we have observed is the failure to recognise vascular pathology as the cause of limb pain. While intermittent claudication is well recognised in the middle aged and elderly population, it is not always considered when dealing with relatively fit and healthy active people. Claudication is usually ascribed to inadequate arterial inflow to a limb, secondary to vascular pathology such as atheromatous disease, but similar symptoms can occur when raised intracompartmental muscle pressure occludes the nutrient microcirculation of the working muscle. Chronic exertional compartment syndrome is well documented but appears to be poorly recognised. Diagnosis requires invasive, direct intracompartmental pressure (ICP) measurement.

This paper reviews our experience of 34 patients diagnosed with chronic exertional compartment syndrome, and discusses the pathophysiology, clinical presentation, diagnosis and management options.

## Methods

The records of patients referred to the exercise laboratory at the University of Natal Medical School for evaluation of exercise-associated lower leg pain over the last 2 years were reviewed. The files of those patients for whom a diagnosis of chronic exertional compartment syndrome had been made form the basis of this report. The exercise laboratory serves as a referral centre for sports medicine patients who are not responding adequately to treatment. Referrals come from all sectors of the medical and paramedical professions in Durban and surrounding areas.

## Results

Thirty-four patients met the diagnostic criteria for chronic compartment syndrome and 4 patients had a diagnosis of raised ICP but not chronic compartment syndrome. There were 24 male patients, giving a male to female ratio of 2.4:1. The average age at presentation was  $28.3 \pm 7.1$  years, with an average duration of symptoms of  $27.2 \pm 35.0$  months. Types of exercise that induced pain were running (20 patients) power and competitive walking (9 patients), rugby (3 patients), hockey (1 patient) and duathlon (1 patient).

All patients presented with muscle pain that was brought on by exercise and relieved by rest. After exercise the affected compartment was tense and the leg slightly swollen. Peripheral pulses were present and of normal volume before and after exercise. No patients had tibial bone pain. Other

**TABLE I. Chronic exertional compartment syndrome patients with additional presenting and associated features**

Additional feature	Associated features					
	Sensory N change	Motor change	Morning stiffness	Night pain	Tibial margin pain	Muscle hernia
Sensory change	13	-	6	2	1	0
Motor change	13	6	-	0	1	0
Morning stiffness	3	2	0	-	0	0
Night pain	3	1	1	0	-	0
Tibial margin pain	1	0	0	0	1	-
Muscle hernia	4	0	2	0	0	0

findings on history and examination are presented in Table I.

The anterior compartment was the primary site of pain in 24 patients (71%), with the pain starting in the deep posterior compartment in 9 patients (27%) and in the lateral compartment in 1 patient. More than one compartment was involved in 12 patients (35%). Twenty-seven patients (79%) noted that the pain was bilateral, but worse on one side.

The ICP pressures were measured in 54 compartments in 34 patients. The mean values obtained before and after exercise are shown in Table II.

**TABLE II. ICP measured at rest before exercise, and 1, 3 and 5 minutes after treadmill run to pain**

	Pre-exercise	1 minute	3 minutes	5 minutes
Mean (mmHg)	21.6 ± 5.4	52.7 ± 16.3	44.0 ± 11.3	38.4 ± 11.1
Range (mmHg)	13 - 38	33 - 86	28 - 72	20 - 68
N	54	12	33	47

All patients had been treated by at least one physiotherapist, and the average number of medical health professionals consulted for the condition before referral was 3.6 (range 2 - 7 people).

## Discussion

The problem of raised compartmental pressure is not new, having been recognised by Hippocrates.<sup>10</sup> Richard von Volkmann described some of the causes and the sequelae in his classic paper entitled 'Ischaemic muscular paralyses and contracture', written in 1881.<sup>20</sup> Almost 100 years later, Matsen defined a compartment syndrome as '... a condition in which increased pressure within a limited space compromises the circulation and function of tissues in that space'.<sup>14</sup> While acute compartment syndrome is well known, chronic exertional compartment syndrome is a relatively new diagnosis.<sup>15,21</sup> Chronic compartment syndromes have been reported in all the compartments of the leg,<sup>22</sup> thigh,<sup>18,26</sup> forearm,<sup>27,34</sup> hand<sup>28</sup> and in the paraspinal muscles.<sup>29</sup>

Diagnosis and subsequent management of chronic compartment syndrome is dependent on a careful history, thorough appropriate examination, ICP measurement, and when necessary additional vascular investigation. To appreciate the signs and symptoms of the condition it is necessary to

understand the pathophysiology of chronic compartment syndrome.

## Pathophysiology

Raised ICP occurs naturally during muscle activity and plays a beneficial role in working the muscle pumps that augment venous return during exercise. In some individuals the venous pressures are sufficiently elevated during and immediately after exercise to impair the nutrient microcirculation of the muscle causing ischaemic, claudicant-type pain. This pain usually subsides on stopping the muscular activity as the nutrient blood supply meets the reduced metabolic demands of the muscle and the ICP falls.

The problem is at the level of the microcirculation. Cellular nutrition is dependent on adequate provision of nutrients by the microcirculation, and as with any fluid flow, this is pressure dependent. Blood flow is usually expressed in terms of Poiseuille's law of flow,  $F = \Delta P / R$ , where  $\Delta P$  is the pressure difference between two ends of a tube and  $R$  is the resistance to flow. In the supine resting individual, the pressure at the arterial end of a capillary bed is 30 - 35 mmHg and at the venous outflow, 10 - 15 mmHg. The pressure in the venules must be lower than that of the venous end of the capillary bed, and the pressure in the veins must be still lower in order for there to be a pressure difference and for flow to occur from one segment to the next.<sup>31</sup>

Any increase in interstitial fluid pressure or ICP will tend to compress the low-pressure thin-walled venous system. In order to resist this increase in transluminal pressure and remain patent, the pressure within the venous vessels must increase. Increasing the venous pressure reduces the arteriovenous pressure gradient in the capillary bed, reducing blood flow. Eventually the pressure gradient is insufficient to drive plasma and cells through the capillaries and stasis occurs, affecting the provision of oxygen and nutrients to the cells.<sup>2</sup>

The pressure at the arterial end of the capillary is a function of the mean arterial pressure (MAP) within the systemic circulation, and is therefore likely to increase slightly during dynamic exercise as the MAP rises in response to an increase in systolic pressure. In terms of Starling's hypothesis, an increase in hydrostatic pressure will tend to drive more water out of the arterial end of the capillary, while an increase in venous pressure will tend to reduce the water return from the interstitial fluid at the venous end of the bed, thus increasing the interstitial fluid pressure. The volume of the muscle compartment will also be increased by cellular oedema secondary to the production of osmotically active metabolites of anaerobic metabolism and muscle fibre microtrauma. Lymphatic drainage is the natural defence mechanism to reduce raised ICP. Rising ICP increases lymphatic drainage to a maximum rate after which it remains constant despite increasing ICP. Further increases in ICP tend to deform the lymph channels, ultimately collapsing them.

Why some people are susceptible to the problem of exercise-induced raised ICP is unclear. Possible causes are an increase in the muscle volume associated with exercise-induced hypertrophy and abnormality of the fascia. Fascia excised from patients with chronic compartment syndrome

have been shown to have reduced elasticity and altered structure.<sup>33</sup> Whether this is a cause or effect is not known.

A more complete description of the pathophysiology of raised ICP is presented in a recent review.<sup>12</sup>

## Presentation

Exercise-induced pain is the most common presentation. It often begins as a feeling of tightness, which goes on to develop into an aching pain. Occasionally it is described as a sharp pain. The site of the pain is dependent on the compartment involved. It is common for the pain to begin distally in a compartment and to ascend and affect the whole compartment or more than one compartment. In many instances (79%) the pain was bilateral but more severe in one limb. This is in keeping with the findings of other series.<sup>9</sup> Pain is also dependent on the intensity, duration, and nature of the exercise. Patients may report that the pain comes on after a certain distance has been completed. In our patients this ranged from 200 m to 32 km.

The distance at which the pain commences is not fixed and may vary with the intensity at which the exercise is undertaken. Some patients report that the pain may not occur if they participate at a lower intensity, or it may be exacerbated if they go out too fast in a competitive run. There appears also to be an additive effect in that the pain may come on sooner in the next run after a high-intensity session. The probable explanation of this is that the resting ICP is higher at the commencement of the second run and the pressure at which nutrient flow is occluded is reached sooner.

The nature of the exercise also affects the pain. Several patients have indicated that they do not get the pain if they play squash, whereas the pain may be severe after as little as 1 - 2 km of road running. This may be explained by the intermittent nature of activity in squash, with frequent relative recovery periods. High-intensity cycling is known to evoke lower ICPs in the anterior compartment than treadmill running at the same intensity.<sup>3</sup>

It is unusual to be able to run through the pain of chronic exertional compartment syndrome. The 2 patients who were able to do this achieved it by stopping briefly until the pain subsided and then continuing at a lower intensity. A feature of the condition is the rapid relief from the pain when exercise is stopped. In the majority of patients (74%) the pain was relieved within 1 - 10 minutes of stopping. In 2 patients the pain continued for hours and even days, depending on the intensity of the event inducing the pain. It was difficult to determine whether the prolonged pain was that of ischaemia or whether it was a feeling of muscle tightness.

At the time of stopping exercise because of the pain, all patients noted that the muscle felt hard. Twelve people (35%) noted lumps on their legs associated with the pain and muscle hardness that disappeared over a couple of hours. When examined after a treadmill run to the point of unbearable pain, these 'lumps' were found to be muscle hernias in 4 patients (12%) and venous engorgement was noted at the perforators in all 12 patients. Muscle herniation is well documented in chronic exertional compartment syndrome and has been reported as occurring in 39 - 46% of patients in

other series.<sup>8,19,21</sup> Venous engorgement is to be expected as the venous drainage of the leg is from superficial to deep. If the pressure in a compartment is raised the pressure in the superficial veins will have to rise to a higher pressure for flow to occur. As veins are thin-walled and distensible, this increase in pressure is seen as venous engorgement.

The nutrient blood flow is impaired at different pressures in different tissues. Nerve appears to be most susceptible to the effects of raised ICP, with axoplasmic flow reduced at pressures between 20 and 30 mmHg.<sup>11,25</sup> Patients seldom report the sensory changes that they note in their feet, as their focus appears to be on the pain. On close questioning, paraesthesia or pins and needles was present in 13 people (38%), and lasted longer than the pain. The distribution of the sensory deficit is dependent on the compartment involved.

Motor changes are subtle. Five patients (15%) noted feeling loss of control of the foot or development of a limping style when they had the pain, and 13 (38%) reported that their foot strike made a noise when they had the pain. This sound is often noted by running companions. We have termed this 'foot slap' and believe that it is an early manifestation of motor dysfunction.

Night pain is uncommon, as is morning stiffness in the affected muscle, but both may occur. Bone tenderness to percussion and pain along the tibial margin are not usual features of raised ICP.

As the problem revolves around the pressure in the nutrient vessels, which is ultimately derived from the systemic blood pressure, patients should be questioned about their blood pressure, which should be measured. Three patients were found to be hypotensive, one because of over medication with antihypertensives. Similarly, fluid retention can exacerbate the problem.

## Examination

A thorough examination is essential to exclude other pathology, such as referred pain, pain of bone origin, pain from the bone-muscle interface and muscle or tendon injury. All pulses from the femorals to the pedal pulses should be palpated and bruits over the femorals should be sought. Absence or weakness of a pulse should alert the examiner to the possibility of underlying vascular pathology, which may present in a similar manner to raised ICP. The absence or diminution of pedal pulses after a run to the point of unbearable pain is most likely due to a popliteal artery entrapment. Peripheral pulses are not affected by the pressures associated with chronic compartment syndrome.

Fascial defects and venous engorgement may alert the examiner to the possibility of raised ICP.<sup>21</sup> The limb girths should be measured at the level of the pain and compared with the unaffected side. Muscle tenderness and muscle firmness should be noted and motor and sensory function should be tested.

In rare instances it has been reported that chronic compartment syndrome has converted into an acute compartment syndrome after heavy exertion.<sup>9</sup> In this situation the pain worsens after exercise. This is a surgical emergency and requires urgent decompression if significant morbidity and even mortality are to be avoided.

## Diagnosis

The possibility of chronic compartment syndrome as the cause of leg pain is usually reached on the patient's history and by exclusion of other pathology during examination. The cornerstone of diagnosis is direct measurement of muscle pressures. This is performed by manometry using a pressure transducer; commercial hand-held fluid-filled manometry devices are available. Other techniques include slit and wick catheters and constant infusion pumps.<sup>17,24</sup> All involve the insertion of a needle into the affected muscle and reading of the pressure. The commercial needle used is a large 18-gauge needle with an end and side port.<sup>16</sup> It is important when using these needles to ensure that both ports are sub-fascial when taking the reading. Laboratory evidence supports the use of smaller single-ported needles in children and small compartments.<sup>13</sup>

Direct ICP measurements are made in the involved compartments with the patient at rest. In the case of chronic compartment syndrome of the legs the patient is then exercised on a treadmill to the point of unbearable pain. The compartment pressures are then measured at fixed time intervals thereafter. We have taken measurements at 1, 3 or 5 minutes post exercise.

Various diagnostic criteria have been suggested.<sup>5,19</sup> We have followed the criteria suggested by Pedowitz *et al.*<sup>19</sup> as being diagnostic of chronic compartment syndrome, that is the presence of one or more of the following: a resting pressure equal to or more than 15 mmHg, a 1 minute post-exercise pressure greater than 30 mmHg, or a 5 minute post-exercise pressure of more than 20 mmHg. As it is often difficult to transfer the patient from the treadmill to an examination couch, reinsert the needle in the compartment, and take a measurement with confidence, all within 1 minute of stopping exercise, we have included a 3 minute post-exercise pressure of greater than 30 mmHg as a further criterion.

Other criteria based on compartment pressure flux during dynamic exercise and the rate of decay of pressure after exercise have been used to diagnose chronic compartment syndrome.<sup>23,30</sup> The difficulties associated with measuring compartment pressure during exercise has led to the general acceptance of pre- and post-exercise pressure measurement as the method of choice.

In those patients who have stopped exercising because of the pain we have found that the pressures may be elevated, but not sufficiently to meet the diagnostic criteria. Our approach has been to advise the patient to return to exercise and to repeat the measurements when the patient has been symptomatic for at least three consecutive exercise bouts.

In some patients there may remain an element of doubt as to whether there is concomitant vascular inflow pathology or popliteal artery entrapment. In these patients we have routinely performed a stress Doppler test and when appropriate, angiography. Ten patients underwent stress Doppler studies and 2 patients had angiography.

X-ray examination and bone scans are of no benefit, save to exclude bone pathology. Nuclear magnetic resonance imaging has been proposed as a diagnostic investigation but its role at present appears limited.<sup>1,7</sup> Thallium single photon emission computed tomography (SPECT) imaging may

prove to be useful in localising ischaemic compartments, but requires further investigation.<sup>32</sup>

## Treatment

To date, the definitive treatment of chronic compartment syndrome is surgical decompression by fasciotomy. While 5 weeks of massage and stretching has been shown to increase the work done before the onset of exercise-induced pain, it does not prevent the pain from occurring and had no effect on the 3 minute post-exercise compartment pressure.<sup>4</sup>

There is, however, a role for trial of conservative treatment in some patients. In 3 patients the pain resolved when their hypotension was corrected. Diuretics relieved the pain in 1 patient whose pain was linked to fluid overload during her menstrual cycle. In the less competitive athlete, the symptoms can be avoided or reduced by decreasing the intensity at which the exercise is performed. Changing activity to one that does not elicit pain can be useful for those who do not wish to suffer the scars of fasciotomy.

Twenty-four patients have undergone fasciotomy, performed by several surgeons using different techniques. There have been no reports of failure to resolve the pain. To date, there has been one recurrence, after a 1-year asymptomatic period during which the patient ran several marathons. The mean follow-up time is presently too short to report the outcome of surgery, which will be reviewed after 5 years.

It should be remembered that chronic exertional limb pain may present at sites other than the lower leg. Not included in this series are patients who have presented with pain in their forearms following windsurfing, kayaking and motorbike racing, and pain in the thenar muscle of the hand associated with writing.

Chronic exertional compartment syndrome appears to be an underdiagnosed cause of exercise-induced limb pain. The diagnosis may be suspected after careful history and examination, but it requires direct ICP measurement to be confirmed. The definitive treatment is decompressive fasciotomy, but conservative avoidance measures may be useful in certain cases.

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## SPORTS MEDICINE TITLES

### The Injured Athlete, 3/e

Thoroughly revised and updated, with a new editor and additional contributors, the third edition of **The Injured Athlete** remains the standard-setting guide to the prevention, diagnosis, and treatment of athletic injuries and rehabilitation of injured athletes. It provides encyclopedic coverage of the full range of athletic injuries and includes complete descriptions of pre-participation examinations, prescriptions for training programs, instructions for fitting protective equipment, and guidelines for drug testing. Illustrations and photographs — many of them new to this edition — complement the text throughout, showing injury mechanisms, disrupted anatomy, diagnostic methods, and treatments.

The third edition features new chapters on the back, principles of therapeutic exercise, therapeutic modalities, and preparation for athletic participation. Coverage of head, neck, and maxillofacial injuries has been greatly expanded. Chapters on the shoulder, the torso, hip, and thigh, the knee, the leg, ankle, and foot, and medical concerns of the athlete have been completely rewritten.

**David H Perrin, Dec 1999, hb, 512 pp, 442 illus, R699**

### Sports Neurology

The updated and expanded second edition of this volume is a comprehensive guide to the recognition, evaluation and treatment of neurologic illnesses in athletes. This edition features expanded coverage of cervical, thoracic, and lumbar spine injuries and new chapters on transient quadriparalysis, degenerative and movement disorders, and multiple sclerosis and exercise. Other topics addressed include sports neuropsychology, functional anatomy and biomechanics of the spine, electromyographic analysis of functional activity, and Special Olympics.

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# Vascular pathology: a cause of lower limb claudication in young sportspeople

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## Abstract

**Objective.** This paper reviews a personal experience of the diagnosis and management of patients with vasculopathy presenting as sport-provoked lower limb pain.

**Design.** Retrospective review of patients presenting to the tertiary referral services of the Durban Metropolitan Vascular Service and the exercise laboratory at the University of Natal Medical School over a 5-year period.

**Results.** The records of 23 patients with vasculopathy were reviewed. All presented with exercise-induced lower limb pain. The primary site of pathology was the aorta in 10 patients, the iliac arteries in 3 patients and the femoropopliteal segment in 10 patients. All were managed with conventional surgical techniques. Three patients presented late with popliteal artery occlusion and had a poor result. All the other patients have returned to their sport.

**Conclusions.** Vasculopathy is an unusual cause of exercise-induced claudication in apparently fit and healthy teenagers and young adults. It is often overlooked in the differential diagnosis of leg pain and failure to diagnose and implement prompt appropriate treatment may result in significant morbidity.

cause of the above problem. The usual history up to the time of diagnosis of arterial insufficiency in this group of patients is one of multiple referrals until the diagnosis is suspected and appropriately investigated, or the complication of acute arterial insufficiency due to thrombosis intervenes. This report presents our personal experience with a group of such patients; our purpose is to increase awareness of the possibility of this diagnosis in young people presenting with exercise-induced lower extremity pain.

## Patients and methods

The Metropolitan Vascular Service of the University of Natal maintains a prospective computerised database of all patients referred for investigation and treatment. Records were selected of patients under the age of 35 years with claudication-like pain of the lower extremities induced by sport. Over the last 5 years, 23 records were found. There were 18 male patients ranging in age from 14 to 35 years.

Table I shows the types of sport in which the patients participated. The majority of patients were road runners, with rugby and soccer being the next most frequent sports involved.

TABLE I. Primary sports evoking claudication in 23 patients

Sport	N
Road running	9
Rugby	5
Soccer	5
Cycling	1
Surfing	1
Karate	1
Hockey	1

Table II gives a summary of the anatomical location and the pathology involving the arteries. In 10 patients the major pathology involved the aorta.

Four patients had coarctation involving the descending thoracic aorta in its classical site just below the origin of the left subclavian artery. In every case this took the form of a

## Introduction

'Young' is an arbitrary definition, but in the under-35 age group lower extremity exercise-induced pain due to arterial insufficiency is an unsuspected and indeed uncommon

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**TABLE II. Anatomical location and pathology of arterial lesions in 23 patients**

Site and pathology	N
Aorta	10
Coarctation	4
Abdominal aneurysm and iliac stenosis	1
Hypoplastic distal aorta	5
Iliac artery	3
Post-traumatic arteriovenous fistula	1
Post-traumatic stenosis	1
Fibromuscular dysplasia	1
Femoro-popliteal	10
Atheroma	2
Entrapment	5
Adventitial cysts	3

fibrous stenosis. All were male patients, ranging in age from 18 to 24 years.

Five patients had the classical hypoplastic distal aorta, in which the aorta 'funnels down' just proximal to its bifurcation. Four were female patients ranging in age from 28 to 35 years. In 2 of these patients there was a superimposed atherosclerotic plaque at the site of narrowing. One male patient aged 35 years had an atherosclerosis-associated abdominal aortic aneurysm with associated common iliac stenotic lesions.

Three patients had isolated external iliac artery involvement. Two followed previous penetrating arterial trauma. In one instance the patient presented 6 years later with an arteriovenous fistula. One other patient developed stenosis at the site of previous arterial repair for a gunshot wound. Stenosis due to fibromuscular dysplasia was found in one 15-year-old male patient, causing him to claudicate during soccer matches.

In the femoro-popliteal area 2 patients had atheromatous lesions in the distal superficial femoral artery. Both were men in their early thirties. Five male patients had the classical popliteal artery entrapment syndrome, while 3 other male patients had popliteal artery stenosis due to adventitial cysts.

## Clinical presentation and diagnosis

Clinical presentation is summarised in Table III. It will be

**TABLE III. Clinical presentation in 23 patients with vasculopathy**

Feature of presentation	N
Claudication	23
Bruit	10
Acute popliteal artery occlusion	3
Hypertension	3
Limb swelling	1
Pulse deficit — resting	5
Pulse deficit — post exercise	15

seen that all patients had claudication at some stage in their clinical course. In almost half, a bruit could be heard in relation to the stenotic area, particularly in relation to aortic and iliac occlusions, in which case the bruit was heard over the femoral arteries. Five patients had an obvious pulse deficit at rest. A further 15 patients developed a clinically detectable pulse deficit following exercise, such as climbing a flight of stairs. Three of the 4 patients with aortic coarctation had significant hypertension. Three patients presented with an acute occlusion of the popliteal artery and presented with critical ischaemia of the foot. The patient with the iliac arteriovenous fistula presented with significant limb swelling due to associated venous hypertension.

The cornerstone of investigation is segmental Doppler pressure studies both before and after treadmill exercise. In every patient who presented with claudication there was a significant pressure drop post exercise. Only 5 patients had a resting ankle brachial pressure index of less than 1, and in all of these patients the pulse deficit was clinically detectable.

## Treatment

The treatment is outlined in Table IV. It has been possible to treat 2 of the 4 patients with coarctation by means of patch aortoplasty. The remaining 2 required the classical interposition graft replacement. The hypoplastic aorta was managed with a 'Y'-shaped patch graft placed over the distal aorta and both common iliac artery origins. In 2 patients a local endarterectomy was also performed. The iliac lesions were treated by replacement grafts using prosthetic material. Both atheromatous distal femoral lesions were treated with simple patch angioplasty, while the popliteal lesions were treated by means of entrapment release or removal of the cyst with appropriate arterial reconstruction using either a patch angioplasty or an interposition replacement graft involving the autogenous saphenous vein.

The 3 patients with acute popliteal occlusion presented 1 week, 10 days and 3 weeks after occlusion respectively. Due to adherent thrombus it was not possible, in any of these patients, to disobliterate the distal vascular tree by means of a combination of balloon embolectomy and regional thrombolytic infusion.

**Table IV. Surgical procedures required for treatment of vasculopathy in 23 patients**

Site and pathology	Surgical procedures	N
Aorta (N = 10)		
Coarctation	Graft replacement	2
	Patch angioplasty	2
Abdominal aneurysm and iliac stenosis	Aortobifemoral bypass graft	1
Hypoplastic distal aorta	Patch angioplasty ± endarterectomy	5
Iliac artery (N = 3)	Replacement graft	
Femoro-popliteal (N = 10)		
Atheroma	Femoral patch angioplasty	2
Entrapment	Entrapment release + patch angioplasty	3
	Entrapment release + interposition graft	2
Adventitial cysts	Cyst evacuation + patch angioplasty	1
	Cyst evacuation + interposition graft	2

## Results

Over follow-up periods ranging from 3 to 60 months, the patients undergoing aortic and iliac reconstructive procedures were all found to have returned to their sports and to have remained symptom free.

Results of femoral and popliteal reconstructive procedures have not been as good as those for aortic and iliac reconstruction. One patient with a femoral patch angioplasty has re-occluded and claudicates quite severely. He has been forced to stop running. The other has since suffered a myocardial infarct and has unreconstructable coronary artery disease. Both patients were found to be hypercholesterolaemic. Of the 3 patients who presented with acute popliteal artery occlusion, 2 with adventitial cysts and 1 with popliteal artery entrapment, all claudicate severely and have had to give up their sports. The 4 remaining patients are doing well and have resumed full activity.

## Case reports

### Case report 1: Adult aortic coarctation

A male patient aged 19 years had been an outstanding sportsman at school, excelling as a sprinter and rugby wing. However, he could only participate in the 100 m and was never competitive over any distance longer than this. He had, in fact, never been able to complete the school cross country course. He stated that while playing rugby his legs felt extremely heavy by the end of the first half. He was, however, of sufficient standard to represent the provincial schools' team. At school he was always regarded as being lazy with regard to training.

On moving into the under-21 ranks and undergoing more intense training in the provincial squad, he developed severe cramping pain in the quadriceps and calves, very suggestive of claudication. At this juncture he was referred to the exercise laboratory at the Natal Medical School.

**Examination.** He was found to have an athletic habitus with a well developed upper body. His legs were, however, relatively underdeveloped. His blood pressure was 140/85 mmHg at rest, which is possibly a little higher than one would expect in this age group. The feet were well perfused, all peripheral pulses were easily palpable and there was no radio-femoral pulse delay. On exercise, namely running up a flight of stairs, there appeared to be a diminution in his femoral pulses and a soft bruit could be heard over the lower aorta, which disappeared with rest.

**Investigation.** His Doppler ankle brachial pressure index was greater than 1 at rest. Following running on the treadmill to the point of claudication, the ankle brachial index dropped bilaterally to 0.7, with the femoral velocity wave form showing gross reduction in velocity. At this stage the femoral bruit was very obvious. On the strength of these findings he was submitted to aortography.

**Aortogram.** This showed a narrowing at the classical coarctation level in the proximal descending thoracic aorta (isthmus), with post-stenotic dilatation (Fig. 1). As he wished to continue with a promising rugby career he was prepared to submit to surgery with its attendant risks.

**Operation.** The aorta was exposed by means of a pos-



Fig. 1. A digital subtraction angiogram of the aortic arch and thoracic aorta showing stenosis (arrowed) and post-stenotic dilatation.

terolateral thoracotomy. The aorta was noted to be narrowed over a very short segment (approximately 1 cm). A longitudinal aortotomy opened the area and a localised 'membrane' of fibrous tissue that reduced the cross-section diameter of the aorta by 50% was found.

This was excised by sharp dissection, in similar fashion to an endarterectomy, and the defect was closed with a prosthetic patch, as an aortoplasty. The postoperative course was uneventful and within 2 months the patient returned to full training, totally symptom free. He has subsequently played representative rugby at junior provincial level.

### Case report 2: Popliteal adventitial cyst

This case involves a 14-year-old male patient who was an 'A' team rugby player and a keen surfer. He had had pain in his right calf following strenuous exercise for some time, which had deteriorated over a period of 6 months before diagnosis. This had been treated symptomatically by the family physician. Pulse status was never established.

During a rugby practice he developed acute pain in his calf and his leg gave way. His foot was noted to be painful and numb. That evening he reported to the family practitioner who diagnosed a 'pulled muscle' and again gave symptomatic treatment. The leg remained painful over the course of the following week. It was worse at night and was relieved by

hanging the foot over the edge of the bed. After 10 days there was minimal improvement and he reported to a physiotherapist who perceived that the foot was possibly ischaemic. The patient was then referred for further investigation.

**Examination.** He was noted to have an athletic habitus. The right foot was significantly cooler and paler than the left. All pulses were present on the left, while on the right no pulses could be felt distal to the femoral. The Doppler ankle brachial pressure index on the left was greater than 1 and on the right it was 0.4. A clinical diagnosis of probable popliteal artery entrapment syndrome with popliteal artery thrombosis was made. He was admitted to hospital and an angiogram was performed. The angiogram confirmed occlusion of the distal popliteal artery with extensive collaterallisation around the knee and into the calf, but there was no major vessel runoff below the knee.

**Treatment.** It was decided to explore the popliteal artery and attempt disobliteration. A direct posterior approach was made and an adventitial popliteal cyst was noted, with thrombosis of the popliteal artery. A longitudinal arteriotomy was performed. The vessel was found to be virtually occluded by the cyst impinging on the lumen with superimposed organising thrombus. A 2 cm segment was excised together with the cyst, and a balloon embolectomy catheter was passed distally (Fig. 2). It was not possible to catheterise the anterior tibial artery, but the catheter passed approximately 7 cm down the posterior tibial artery, with retrieval of some organising thrombus. A perfusion catheter was then passed distally and 150 000 units of streptokinase were infused over a 30-minute period. Following this there was some back bleeding. Control angiography showed patency of a tenuous posterior tibial artery with obvious residual clot. It was felt at that stage that further interference might aggravate the situation and a short reverse saphenous interposition graft was used to reconstruct the distal popliteal artery.

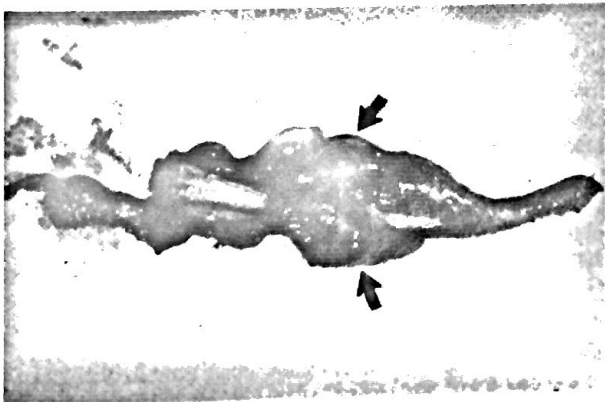


Fig. 2. Specimen of popliteal artery with a large adventitial cyst (arrowed).

**Postoperative course.** Following this procedure the foot warmed up considerably, but no distal pulses were palpable and the rest pain disappeared. There was a reasonable biphasic Doppler signal over the posterior tibial artery and

the Doppler ankle brachial index improved to 0.8. The patient then commenced an active exercise programme, and the current status, now 2 years since the procedure, is that he has been able to resume surfing but is not able to run more than 400 m without severe claudication. He is, however, able to walk quite comfortably for a considerable distance.

## Discussion

This paper does not aim to provide a detailed discourse of the different pathologies, but rather to make the point that although unusual in young sportspeople, claudication due to arterial insufficiency must be considered as a possible diagnosis in those presenting with exercise-induced pain or discomfort. In this particular series all patients gave the classical history of claudication, namely cramping pain or discomfort in a major muscle group provoked by exercise and relieved with rest. In addition, of the 23 patients, 5 (22%) had an obvious pulse deficit at rest and a further 15 patients developed an obvious pulse deficit on simple exercise, such as running up a flight of stairs. Therefore in 20 patients (87%) this was a consulting room diagnosis. All 23 patients had obvious derangement on Doppler and stress Doppler testing. A common factor in all patients was repeated and multiple referrals before the diagnosis was considered, hence the major problem is a lack of awareness of the possibility of vasculopathy.

Coarctation of the aorta is usually diagnosed in infancy, but milder forms may persist into adulthood.<sup>2</sup> It is often unmasked by the unexpected finding of hypertension or the development of claudication when the patient is subjected to unaccustomed exercise. In the past the classical history was that of claudication developing during military training in school leavers. In many cases this was the first time that the blood pressure had ever been taken, and enforced exercise on the parade ground that could not be avoided induced claudication. The case report presented is slightly unusual in that the patient was not significantly hypertensive.

The hypoplastic aorta syndrome is classically seen in young female patients.<sup>1,3</sup> The pathology is thought to result from embryonic overfusion of the dorsal aortae during development, resulting in significant narrowing of the distal aorta. Symptoms classically develop in the early thirties due to superimposed atheromatous plaque in the area. This is probably a consequence of the altered haemodynamics.

Fibromuscular dysplasia is also classically seen in young women.<sup>9,10</sup> It is a process of intimomedial fibrosis of uncertain aetiology. Arteries affected include the carotid, renal and iliac vessels; the visceral arteries can be affected, although this is less common. The syndrome is rare in males and very rare in Negroes. The patient in this series was in fact a 17-year-old black boy, which makes this an exceptionally rare presentation.<sup>6</sup>

Although well documented, we have not seen the iliac syndrome of cyclists. In this condition, associated with large volumes of high-intensity training and competition, unilateral, eccentric intimal hyperplasia of the external iliac artery occurs and presents as buttock and thigh claudication, with ensuing numbness of the leg. Treatment involves shortening of the artery and endarterectomy.<sup>4</sup>

Popliteal adventitial cysts are also rare.<sup>5,8</sup> These are thought to represent synovial rests in the adventitia of the artery. They gradually increase in size and are filled with gelatinous material. The presentation is similar to that of the popliteal artery entrapment syndrome.<sup>7</sup>

With regard to therapy, the aortic pathologies have all done well from the standpoint of rehabilitation and return to full sporting activity. The popliteal syndromes will also do well if diagnosed and treated early. However, once complications occur, such as thrombosis as in the case report presented, the outlook is not a happy one. The report presented is typical of neglected pathology.

Precocious atherosclerosis occurring in young people also has a guarded prognosis. Under these circumstances it is extremely important to seek and attempt to control the underlying risk factors. These patients frequently have a familial history and some form of hyperlipidaemia. It is important to control smoking and gain pharmacological control of the hyperlipidaemia. Exercise is an important part of the treatment.

Whatever the underlying vasculopathy may be, it is extremely important to consider and exclude an underlying vasculopathy as a cause in all young patients actively partic-

ipating in sport who develop exercise-induced lower limb pain. The importance of this cannot be over-emphasised as neglected and complicated pathology may result in severe disability and even limb loss.

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# Injuries among adolescent soccer players during an interprovincial tournament in South Africa

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## Abstract

**Objective.** To describe the pattern of injuries among soccer players taking part in the interprovincial Under-20 soccer tournament in Cape Town in 1997.

**Design.** The injuries recorded were those that occurred during the 76 matches played at the tournament. Four hundred and five players participated from all the nine provinces.

**Results.** During a total of 76 matches, 74 (19%) of the players were injured. Types of injury included muscle strain (34.6%), bruises (25.6%), ligament strain (15.4%), and joint swelling (14.1%). The ankle was the commonest site of injury.

**Discussion.** The injury rate during the tournament was high (74 injuries in 2 224 player hours, which equals 332 injuries in 10 000 player hours). However, if proper preventive measures are employed the injury rate among adolescent soccer players may decrease.

## Introduction

Few systematic epidemiological studies have been conducted on sports injuries in South Africa; even fewer studies have addressed sports injuries in youth, or reported participation rates against which the incidence of injury can be assessed. Recent information obtained from the Department of Sports and Recreation indicated that there is no national system for the collection of data on sports injuries in South Africa. Internationally, there has been a growing drive to promote safe and successful competition by documenting the epidemiology of injuries sustained at large sporting events.<sup>2</sup>

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Soccer is a team sport commonly played in South Africa and around the world. Ekstrand and Gillquist<sup>1</sup> have identified soccer as one of the most common team sports played by children worldwide. Soccer has also been identified as the sport causing the most sports injuries among children.<sup>3,4,7</sup> These studies identified the most common site of injury to be the lower extremity (whole lower limb), of which the thigh and hip area had the highest injury rate, closely followed by the ankle and knee areas. Other sites of injury included the shoulder, head and face, arm, and back. The most common types of injuries were bruises, muscle strains and ligament sprains.

The results of a study<sup>3</sup> conducted on soccer players aged 12 - 19 years in the USA indicated that for 480 soccer games, equivalent to 74 900 player hours, 179 injuries were reported. This gave an average of 23.8 injuries per 10 000 player hours. A study done in Denmark<sup>6</sup> on young soccer players aged 12 - 18 years and involving 3 soccer clubs over a period of 1 year, reported that the average incidence was 37 injuries per 10 000 player hours. The latter study indicated the most common injury sites to be the knee and the ankle. Another study conducted in the USA<sup>8</sup> reported the injury rate of soccer players to be 7.7 per 100 players, with 160 injuries per 10 000 player hours.

Literature giving a similar description of soccer injuries in South Africa is not available. Therefore the purpose of the current study is to describe the pattern of injuries in adolescent soccer players at a major youth soccer tournament in South Africa, and to estimate the incidence of injury among players.

## Methodology

This study reports injury data gathered at an interprovincial U20 soccer tournament held in Cape Town from 14 to 19 July 1997. Representatives from all the provinces in South Africa were present at the tournament. Twenty-six teams were registered from Gauteng, KwaZulu-Natal, Eastern Province, Free State, North Western Province, Northern Cape, Mpumalanga, Northern Province and Western Province.

A total of 405 players (aged 17 - 19 years) participated in the tournament. Each team consisted of approximately 15 players. A total of 75 matches was played in the first 4 days, while the final match was played on the fifth day. Each match lasted 80 minutes, and overall there was 101.3 hours of match time. The teams were divided into 4 groups. Each group played 15 matches, except for group 4 which played 28 matches. Groups 1 - 3 each contained 6 teams, and each

team played 5 matches. There were 8 teams in group 4, and each team played 7 matches. The winner from each section played in the semifinals.

An injury was defined as an incident occurring during a match and causing a player to seek medical attention. At the start of the tournament, coaches, managers, parents and players were informed of the medical facilities available at the venues of the soccer matches and about the information that would be collected at the medical tents. Injuries that occurred on the field and that were treated at the side of the field were not included in the study. The information gathered included age, type of injury, location of injury, previous injuries and treatment intervention at the time. This information was gathered as players reported for treatment at the medical tent, and injuries were treated by physiotherapists.

## Results

The results clearly reflect the prevalence, site and type of injuries that occurred at the tournament. Seventy-four players (18.3%) were injured during the tournament. Of those injured, 8.1% ( $N = 6$ ) sustained injuries that required withdrawal from the tournament, 27% ( $N = 20$ ) sustained injuries in their first match, and 20.3% ( $N = 15$ ) entered the tournament with previous injuries or had sustained similar injuries before the tournament (Table I).

**TABLE I. Prevalence of reported injuries ( $N = 74$ )**

Day	Injured ( $N$ )	Previous injuries	Withdrawal from tournament
1	20	5	1
2	12	4	-
3	11	1	2
4	19	3	1
5	12	2	2

Some of the injured players reported more than one type (Table II) or site of injury (Table III). The total number of all injuries sustained was 78. The commonest types of injuries recorded were muscle strains (34.6%), bruises (25.6%), ligament sprains (15.4%) and joint swelling or inflammation (14.1%). Two players sustained a fracture.

**TABLE II. Number of injuries ( $N = 78$ )**

Type of injury	Injuries ( $N$ )	%
Muscle strain	27	34.6
Bruises	20	25.6
Ligament strain	12	15.4
Joint swelling/inflammation	11	14.1
Others (haematoma, fracture, etc.)	8	10.3

There was a total of 83 injury sites. The most frequently reported injury sites were the ankle (26.6% of injured), knee (21.7%) and thigh (21.7%). Other injury sites included the groin, leg and back. Most injured sites (86.4%) involved the lower extremity (Table III).

**TABLE III. Sites of injury ( $N = 83$ )**

Location of injury	Injury sites ( $N$ )	%
Ankle	22	26.6
Knee	18	21.7
Thigh	18	21.7
Groin	8	9.6
Leg	6	7.2
Back	3	3.6
Other (shoulder, rib, wrist)	8	9.6

There was a total of 190 physiotherapy treatment sessions, which included treatment given at time of injury and during follow-up sessions (Table IV). The most common treatment modalities included ice, ultrasound, compression, strapping, massage, joint mobilisations and soft-tissue stretch. The most commonly used modality was ice (31.6%), followed by therapeutic massage and ultrasound.

**TABLE IV. Physiotherapy treatments given during the tournament**

Modality used	Times used ( $N$ )	%
Ice	60	31.6
Ultrasound	35	18.4
Massage	30	15.8
Compression	20	10.5
Strapping	20	10.5
Joint mobilisations	10	5.3
Soft-tissue stretch	15	7.9
Total	190	100

From the data collected it was estimated that 1 out of 5 players was injured (74 injured players: 405 players), and an average of 1 player was injured per game (74 injured players: 76 games). It was also estimated that there was an injury rate of 332/10 000 player hours. In addition the study revealed that there were 0.2 injuries per player, and 1.05 injuries per injured player.

## Discussion

This study set out to describe the pattern of injuries in soccer players who took part in the interprovincial U20 tournament in Cape Town in 1997. Throughout the tournament 78 injuries were reported in 76 matches. Compared with the study by Kibler<sup>8</sup> in the USA in which the injury rate was 179 injuries in 480 matches, it seems that the injury rate during the period of the current study was high. Possible reasons for this could be the differences in the duration of the tournaments and the levels of competition. The study in the USA involved a weekend tournament, whereas the tournament in South Africa was held over one week with games played on a daily basis. The level of competition in the South African study was high, which meant an increase in the intensity of training and conditioning programmes as well as the number of games per week. These factors dramatically increased risk of injury in young soccer players.

The incidence of injury was high enough to justify the need for physiotherapy services at major tournaments. Knowing that medical services were available at the venue

of the tournament could have encouraged the players to report the injuries sustained. The availability of medical services also made the prompt withdrawal of seriously injured players possible.

The most common types and sites of injuries identified in this study are similar to those found in other studies<sup>3,5,6</sup> which indicated that the ankle, knee and thigh were the most common sites of injury, while the most common types of injury were muscle strains and bruises. The high incident rate of injuries reported during the tournament emphasises the need for preventive measures. During the tournament approximately 20% of the injured players had sustained similar injuries before the tournament. Soccer coaches should be made aware of their responsibility in terms of preventing some of these injuries. They should encourage proper warm-up exercises and stretches prior to matches or training sessions. The coaches should prevent previously injured players from playing until their injuries are adequately healed. The study conducted by Ekstrand and Gillquist<sup>1</sup> highlighted the need for players to be supple in order to reduce injury risk. They also emphasised the need for coaches and players to ensure that injuries have healed before players return to the game. Other factors such as protective equipment and the condition of the playing ground should also be included as ways to prevent or reduce the risk of injury.

Though the information collected during this tournament cannot be generalised, the data will contribute to a data bank on sports injuries in South Africa. The researchers are collecting similar data from other soccer tournaments and from other sporting codes.

## Conclusion

The interprovincial U20 soccer tournament held in Cape Town in 1997 had a high injury rate. However, if proper preventive measures are employed the injury rate among adolescent soccer players may decrease.

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# Modifiable health risk factors among white male executives in the Zululand area

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## Abstract

The objective of this study was to draw up a profile of modifiable health risk factors among male executives in the Zululand area. Data for 176 subjects with a mean age of 40 years, were collected between 1989 and 1996 during fitness and health assessments offered to companies for their employees at executive level. All assessments were performed by the author personally and a standard procedure was used. The following parameters were tested: body composition, blood pressure, total cholesterol, physical working capacity and aerobic capacity.

The majority of subjects (54.7%) were in the obese (fat percentage > 20%) or high-risk group. Body mass index for 30.1% of subjects was greater than 27.8 kg/m<sup>2</sup>, which put them in the high-risk group. Mean resting systolic blood pressure was 128 ± 12.5 mmHg and mean diastolic blood pressure was 88 ± 9.8 mmHg. Hypertension (blood pressure > 140/90 mmHg) was prevalent in 17.1% of subjects. Mean cholesterol for the group was 5.68 ± 1.23 mmol/l. Normal cholesterol (< 5.2 mmol/l) was displayed by 35.9% of the subjects, while 21.7% were at moderate risk (cholesterol level of 5.3 - 6.0 mmol/l) and 42.4% were at high risk, with a cholesterol level of > 6.0 mmol/l. The mean physical working capacity<sub>170</sub> for the subjects of this study was 204 ± 51 Watt (2.45 W/kg), with a maximum of 340 W and a minimum of 70 W. The mean aerobic capacity for the subjects was 3.54 ± 0.85 l/min, with a maximum of 6.0 l/min and a minimum of 1.9 l/min. Relative values were 43.59 ± 10.71 ml/kg/min, with a maximum value of 78.7 ml/kg/min and a minimum value of 23.31 ml/kg/min. These results are useful for comparison and for establishing a norm for this particular region.

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## Introduction

Coronary heart disease (CHD) has been identified as a major national health problem because of its relatively high prevalence among white South African males. CHD is the leading cause of death in the white population of Durban.<sup>12</sup> It is therefore of paramount importance to train health providers, especially those involved in preventive health, in identifying and implementing effective ways to reduce risk factors associated with the development of CHD. The public should also be educated to take part in preventive measures. Multiple factors are responsible for the development of CHD in an individual. In order to control the disease it is important to establish the risk factors present in an individual and to introduce management and intervention strategies. Although no conclusive proof exists that all recommended interventions eliminate CHD risk, the magnitude of the problem does not permit indefinite temporising while awaiting such proof.<sup>4</sup> The major modifiable risk factors for CHD are cigarette smoking, hypercholesterolaemia, physical inactivity, diabetes mellitus, hypertension, stress, and obesity.<sup>10</sup>

In the last few decades physical health and fitness of employees in the South African corporate sector have received increasing attention. Employers have started to realise that it is in their best interests to have a fit and healthy workforce. This is especially true in the case of executives, as the financial loss is great when such an employee is unable to function optimally, or is absent from work owing to illness such as CHD. It is now increasingly recognised that physical fitness can play an important role in preventing conditions that could lead to absenteeism and reduction in a person's productivity. In South Africa it is slowly becoming popular for major companies to introduce health and fitness assessments and intervention programmes for their employees. The identification of preventable health risk factors constitutes an important component of these assessments. Many companies in cities have established their own health and fitness centres or have encouraged and subsidised their employees to join local health and fitness centres. However, the same enthusiasm is not found in smaller urban environments, such as occur in the Zululand area. Apart from isolated initiatives, there is a general lack of responsibility among employers as regards preventive corporate health and fitness programmes. It seems that management of health risk factors utilising non-medicinal measures, such as exercise, has been particularly neglected.

This paper aims to encourage health and fitness awareness by highlighting the prevalence of CHD risk factors prevalent in Zululand. Since 1989 the Department of Human Movement Science at the University of Zululand has offered a physical health and fitness assessment and management programme to the corporate sector in the Zululand area. Apart from the



obvious benefit to the individuals in terms of their personal state of health and fitness and how to improve these, the data were collected in order to draw up a health and fitness profile of executives in the area. Such a profile could prove invaluable in educating decision makers as to the need for corporate contributions to health and fitness programmes for employees. This paper reports on the incidence of some modifiable risk factors for CHD.

## Methods and procedures

Data are reported for 176 white male subjects (Table I), and the assessments were done by the author. Subjects were all from middle to senior management positions in the corporate sector in Zululand. Fifty-eight per cent of the men were of English origin, and 42% were Afrikaans. Intergroup correlations did not show any significant differences between the two groups, and for the purposes of this paper the results of the two groups were pooled to represent the white male executives of the area at the time of testing. The following medicines were taken by some subjects at the time of testing: diabetes medication (5.4%), antihypertensive medication (10.8%), medication to control heart rhythm (2.7%), diuretics (5.4%) and hypercholesterolaemia medication (2.7%).

TABLE I. Anthropometric measurements (N = 176)

Parameter	$\bar{x}$	SD	Max	Min
Body mass (kg)	82.4	13.8	145	52.6
Height (cm)	176.4	8.3	195	125
Age (yr)	40	9.9	66	25

SD = standard deviation.

Subjects were fully informed about the assessment procedures and understood that they were free to withdraw at any time. All assessments were performed strictly according to a standard protocol.

Fat percentages were calculated from the skinfolds taken with a Lange caliper by means of the method described by Pollock *et al.*<sup>9</sup>

Blood pressure was recorded at rest (3 times) and during the cycle ergometer test by means of the computerised sphygmomanometer of the Ergoline 900 cycle ergometer. This device uses a microphone to assess the sound of blood passing beyond the cuff and has proved, during repeated comparison with the conventional stethoscope method, to be very accurate even during intensive exercise.

Total cholesterol was measured by means of a Reflotron analyser (Boehringer Mannheim) using whole blood extracted from the finger. Subjects were asked to fast for at least 3 hours before the test. The Reflotron was calibrated regularly and validated using results from the pathological laboratory in Empangeni.

Physical working capacity (PWC<sub>170</sub>) and predicted maximum oxygen consumption (VO<sub>2max</sub>) were determined with subjects cycling on the Ergoline 900 electro-mechanically braked cycle ergometer (Mijnhardt) starting at 25 Watts (W) for 3 minutes, thereafter increasing by 25 W per minute until voluntary exhaustion. The heart rate was recorded at the end of each minute using an Exercentry heart rate monitor, backed up by ear pulse recording of the Ergoline. The PWC<sub>170</sub> and pre-

dicted VO<sub>2max</sub> were calculated using the methods described by Astrand and Rodahl.<sup>3</sup>

## Results and discussion

### Body composition

Although not a primary risk factor for CHD, obesity is now recognised as an independent risk factor.<sup>9</sup> Distribution of fat on the body further increases the risk, with android obesity having a higher risk than gynoid obesity. Although not measured in the present study, it is relatively certain that the male subjects tested and found to be obese were mainly android obese.

The mean fat percentage for the 176 subjects was 21.73% (Table II). This compares with the mean 50th percentile of 21.4% for 18 - 65-year-old males reported by the Young Men's Christian Association (YMCA) physical fitness test battery.<sup>6</sup> When the skinfolds are compared with the physical fitness levels of South African adults aged 18 - 55 years,<sup>2</sup> some interesting observations are noticed. When the average skinfold values in the present study are compared with the percentiles of Andrews,<sup>2</sup> they fall on: triceps (35th), subscapula (60th), supra iliac (30th), biceps (45th) and abdomen (80th). This seems to support the observation that the population represented in this study tends towards android obesity.

TABLE II. Average body composition values of the subjects (N = 176)

Parameter	$\bar{x}$	SD	Max	Min
Fat (%)	21.73	9.27	66	4.2
Lean body mass (kg)	63.6	8	89.7	28.1
Fat mass (kg)	18.73	10.48	66.7	2.66

SD = standard deviation.

It is, however, apparent that obesity should be defined clearly before conclusions as to risk for CHD can be made. Further analysis of the data of this study (Fig. 1) shows that 20.4% of subjects had a fat percentage lower than 15%, 24.9% between 15 and 20%, 23.8% between 20.1 and 25%, and 30.9% greater than 25%. McArdle *et al.*<sup>9</sup> and Robergs and Roberts<sup>10</sup> regard male obesity as a body fat percentage of more than 20%. If this is accepted it would place the majority (54.7%) of the subjects tested in the obese and therefore high-risk group for CHD.

When using height and weight to assess obesity and risk for CHD, the body mass index (BMI) is probably the best method to use.<sup>5</sup> The mean BMI in this study was 26.5 kg/m<sup>2</sup>. This is slightly below the figure of 27.8 kg/m<sup>2</sup> stated to be the value where significant increase in risk begins.<sup>1</sup> However, 30.1% of the subjects in this study had a BMI equal to or higher than 27.8 kg/m<sup>2</sup>, which signifies an increased risk of CHD. In comparison, Seedat and Mayet<sup>11</sup> found that 44.2% of white males in Durban had a BMI  $\geq$  25 kg/m<sup>2</sup>, and 13.2% had a BMI  $\geq$  30 kg/m<sup>2</sup>.

### Blood pressure during rest and exercise

Hypertension is the most common cardiovascular disease in human populations. Although the World Health Organisation defines normal blood pressure as less than 140/90 mmHg, and

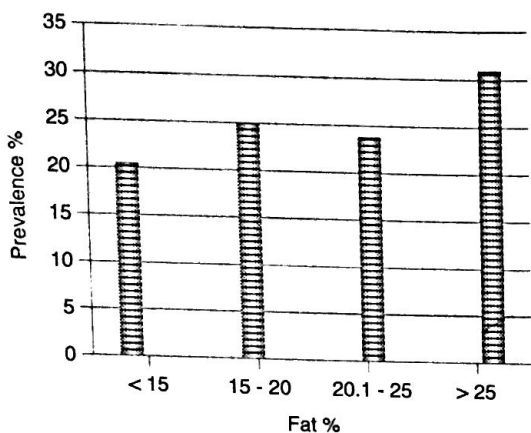


Fig. 1. Percentage of subjects in the fat % groups (N = 176).

blood pressure above 160/95 mmHg as hypertension, it is suggested that blood pressures of greater than 140/90 mmHg be regarded as hypertension with increased risk of cardiovascular disease. Using this norm the prevalence of hypertension in people from most Western industrialised countries is 15 - 20%.<sup>7</sup> This corresponds with the present study where hypertension (> 140/90 mmHg) was found in 17.1% of the subjects. This is much lower than the 37.8% prevalence found in white males in Durban by Seedat and Mayet.<sup>11</sup>

Further analysis of the data reveals the following (Fig. 2): using the norms prescribed by the American College of Sports Medicine,<sup>1</sup> 80.6% of the subjects tested displayed normal systolic blood pressure (< 140 mmHg), 11.8% were borderline to mildly hypertensive (140 - 149 mmHg), 4.7% were mild to moderately hypertensive (150 - 159 mmHg), 2.4% were moderate to severely hypertensive (160 - 170 mmHg), and 0.6% had uncontrolled systolic blood pressure (> 170 mmHg). As regards diastolic blood pressure, 55.3% of subjects were normal (< 90 mmHg), 28.2% were borderline to mildly hypertensive (90 - 95 mmHg), 8.8% were mild to moderately hypertensive (96 - 100 mmHg), 5.9% were moderate to severely hypertensive (101 - 110 mmHg), and 1.8% had uncontrolled diastolic blood pressure (> 110 mmHg).

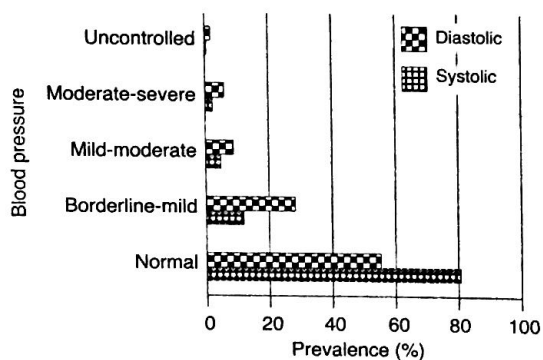


Fig. 2. Incidence of systolic and diastolic hypertension (N = 176).

Mean resting systolic blood pressure was  $128 \pm 12.5$  mmHg, with a maximum of 178 mmHg and a minimum of 95 mmHg. Mean resting diastolic blood pressure was  $88 \pm 9.8$  mmHg, with a maximum of 126 mmHg and a minimum of 65 mmHg. During graded exercise mean systolic blood pressure for the subjects tested increased with increasing workload, and mean diastolic blood pressure decreased slightly with increased workload (Fig. 3). This tendency was also reflected by Shaver.<sup>14</sup>

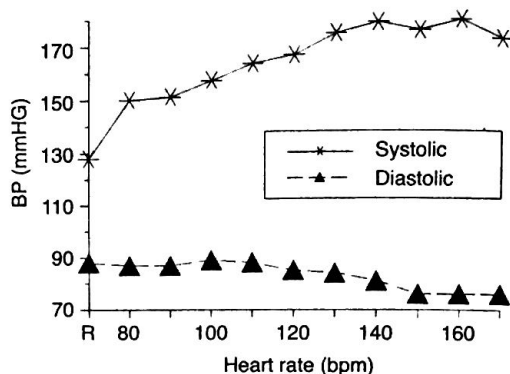


Fig. 3. Blood pressure during rest and graded exercise (N = 176).

### Total cholesterol

Abnormalities in blood lipid levels appear to be at the base of the atherosclerotic process, and thereby to constitute a real risk for CHD.<sup>15,16</sup> In the present study (see Fig. 4) mean total cholesterol was 5.7 mmol/l (with a maximum of 9.04 mmol/l and a minimum of 2.59 mmol/l). According to the American College of Sports Medicine<sup>1</sup> this figure would represent a high risk for cardiac disease. According to the norms proposed by the Study Group of the European Atherosclerosis Society,<sup>17</sup> 35.9% of the subjects had a normal cholesterol (< 5.2 mmol/l), 21.7% were at moderate risk (cholesterol level of 5.3 - 6.0 mmol/l) and 42.4% were at high risk with a cholesterol level of > 6.0 mmol/l.

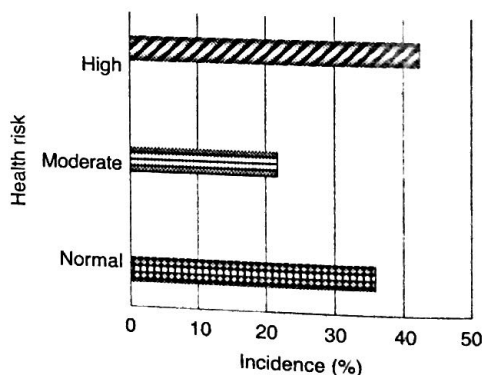


Fig. 4. Total cholesterol displayed at low risk (< 5.2 mmol/l), moderate risk (5.3 - 6.0 mmol/l) and high risk (> 6.0 mmol/l) (N = 173).

Using the norms of the American Heart Association,<sup>10</sup> 33.0% of the subjects had a desirable cholesterol level (< 5.2 mmol/l), 28.9% had borderline to high cholesterol (5.2 - 6.1 mmol/l), and 38.1% had high cholesterol (> 6.1 mmol/l). Seedat and Mayet<sup>11</sup> found a 34.4% prevalence in cholesterol levels  $\geq$  6.5 mmol/l among white males in Durban.

### PWC<sub>170</sub> and VO<sub>2max</sub>

Physical inactivity is widely recognised to be a major risk factor for CHD.<sup>10</sup> The degree of physical inactivity is difficult if not impossible to measure accurately; under normal circumstances it can only be measured as a subjective rating. In the physical activity questionnaire, 65% of subjects indicated that they exercised regularly. One question asked if they participated in moderate to strenuous exercise, and the answers were as follows: none (18.9%), 1 day per week (8.1%), 2 days per week (13.5%), 3 days per week (16.2%), 4 days per week (18.9%), 5 days per week (13.6%) and 6 days per week (10.8%). Most subjects (91.9%) indicated that they could walk briskly for 5 km, and 56.8% indicated that they could jog 4 km continuously. The above might be an overestimation of the individual's actual abilities.

Each subject's physical condition was tested by means of the PWC<sub>170</sub> and indirect VO<sub>2max</sub>. These tests rely on heart rate which is affected by the state of fitness of the subject. Therefore, if a specific population scores higher for PWC<sub>170</sub> and indirectly measured VO<sub>2max</sub> than the general population norm, then one of the factors responsible for this could be a higher state of fitness.

The mean PWC<sub>170</sub> for the subjects in this study was 204  $\pm$  51 W (2.45 W/kg), with a maximum of 340 W and a minimum of 70 W (Table III). These figures are slightly higher than those reported by Andrews,<sup>2</sup> who found a mean of 2.29 W/kg for South African males. The mean of 204 W corresponds with the 203 W reported in the YMCA physical fitness test battery as being the 50th percentile for males aged 18 - 65 years.<sup>6</sup>

The mean aerobic capacity for the subjects in this study was 3.54  $\pm$  0.85 l/min, with a maximum of 6.0 l/min and a minimum of 1.9 l/min (Table III). Relative values were 43.59  $\pm$  10.71 ml/kg/min, with a maximum value of 78.7 ml/kg/min and a minimum value of 23.31 ml/kg/min. The mean value of this study compares well with the 44 ml/kg/min reported by McArdle *et al.*<sup>6</sup> and the 45 ml/kg/min reported by Sharkey<sup>13</sup> for sedentary untrained adult males. It is, however, much higher than the mean 50th percentile of 38 ml/kg/min reported in the YMCA physical fitness test battery for 18 - 65-year old males.<sup>6</sup>

**TABLE III. Measured values for physical working capacity and oxygen consumption (N = 176)**

Variable	$\bar{x}$	SD	Max	Min
PWC <sub>170</sub> (W)	204 (2.45 W/kg)	51	340	70
VO <sub>2max</sub> (l/min)	3.54	0.85	6	1.9
VO <sub>2max</sub> (ml/kg/min)	43.59	10.71	78.7	23.31

SD = standard deviation.

### Smoking habits

Smoking is widely recognised to be a major risk factor for CHD. In the present study 16.2% of subjects smoked, with 2.7% smoking more than 40 cigarettes per day, 8.1% smoking 10 - 19 a day, and 5.4% smoking 1 - 9 cigarettes per day. Seedat and Mayet<sup>11</sup> found 19.7% of white males in Durban to be smoking more than 10 cigarettes per day.

### Conclusion

The above results indicate that adult males from the Zululand area are susceptible to avoidable health risk factors. The majority of subjects tend to be android obese, which further intensifies the risk for CHD. The incidence of hypertension is equivalent to Western norms. The majority of subjects were at risk due to unacceptably high blood lipid levels. Fitness levels did not differ significantly with those reported in the literature for other South Africans of the same age.

It is quite clear that the white male population from the Zululand area displays significant risk factors for CHD. However, these risk factors are avoidable and intervention strategies are strongly advised. It is hoped that this article will contribute toward educating the population, and that it will assist those concerned in taking corrective action.

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# Exercise-induced acute compartment syndrome

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## Introduction

Exercise-induced limb pain is usually benign, does not constitute a medical emergency, and resolves with appropriate treatment and/or rest. The exception to this rule is limb pain of vascular origin that requires prompt, accurate diagnosis and that may warrant urgent surgical intervention, when appropriate, to prevent morbidity and even mortality.

Ischaemic limb pain in active, relatively fit people is uncommon and may easily be overlooked in a differential diagnosis that includes delayed onset muscle soreness, referred pain, pain of bony origin, pain derived from the muscular tendinous interface ('shin splints'), muscular or tendinous injury, and nerve entrapment. The vascular causes of limb pain in apparently healthy athletic individuals encompass the full spectrum of vascular pathology including: (i) arterial pathology, ranging from previously undiagnosed congenital abnormalities such as coarctation of the aorta, to aortic aneurysm, acute thrombosis and embolism, popliteal artery entrapment, and degenerative conditions including mucoid degeneration and atheromatous disease; (ii) venous pathology resulting in raised venous pressure; and (iii) raised intracompartmental pressure (ICP) within the muscle compartments. Of these uncommon causes of exercise-induced limb pain, raised ICP occurs most frequently in the exercising population.<sup>9</sup>

Raised ICP occurs naturally during muscle activity and plays a beneficial role in working the muscle pumps that augment venous return during exercise. In some individuals the muscle pressures are sufficiently elevated during exercise to impair the nutrient microcirculation of the muscle causing ischaemic, claudicant-type pain. This pain usually subsides on stopping the muscular activity as the nutrient blood supply meets the reduced metabolic demands of the muscle and the ICP falls. When confirmed by direct ICP measurement

this scenario is diagnosed as chronic exertional compartment syndrome.

On rare occasions, the ICP remains elevated for a sufficient period after exercise such that the associated ischaemia results in sensory and motor impairment and thrombosis of the occluded microcirculation with ensuing muscle necrosis. Matsen<sup>24</sup> has defined this situation as a compartment syndrome, which is '... a condition in which increased pressure within a limited space compromises the circulation and function of the tissues in that space'. The key words are pressure, circulation and function. The syndrome results from allowing ICP to remain elevated for too long. Ideally a diagnosis of raised ICP should be made and appropriate treatment instituted before the functional impairment associated with the syndrome is present.

The sequelae of the syndrome include delayed rehabilitation with associated limb swelling and subjective sensory and motor deficit as seen in the post tourniquet syndrome, objective sensory and motor deficit, Volkmann's ischaemic contracture, rhabdomyolysis and myonephropathic syndrome. Amputation following delayed diagnosis is not uncommon.

This paper presents an unusual case of atraumatic exercise-induced compartment syndrome, reviews the literature on exercise-induced acute compartment syndrome and discusses the diagnosis and management of this condition.

## Case report

A 22-year-old male student participated in a basketball game on a Sunday evening. He played the full game and noted no injury. After the game the lower anterolateral aspect of his right leg felt 'tight'. During the night the 'tightness' became pain. In the morning he sought treatment. The general practitioner who saw him diagnosed a muscle injury, prescribed anti-inflammatory medication and referred him to physiotherapy where he was treated with ice and electrotherapy. The pain worsened during that day and when he returned to the physiotherapist on the Tuesday he was in severe pain. The sensory, motor and pulse status of the leg was not recorded. He was referred back to the general practitioner who could only see him on the following day, Wednesday, by which time he had a foot drop. Urgent referral to an orthopaedic surgeon was made for diagnosis of the cause of the foot drop.

At that point the muscles of the anterior compartment were described as 'woody hard', there was sensory deficit over the distribution of the peroneal nerve, foot drop, and the

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dorsalis pedis pulse was not palpable. A diagnosis of compartment syndrome was made and the patient was referred to a surgeon with an interest in this condition for urgent fasciotomy. In view of the urgency and the unavailability of the necessary equipment, direct ICP studies were not performed. The patient was taken directly to theatre and a four compartment fasciotomy was performed. At operation the anterior compartment did not respond to stimulation, areas of necrosis were present, and generalised thrombosis of small vessels was noted. Necrotic tissue was excised and the wound left open. A back slab was applied to maintain the ankle in a neutral position. Forty-eight hours later at wound inspection in theatre under general anaesthetic, the remaining muscle in the anterior compartment was found to be necrotic and was excised *in toto*. Wound closure was achieved over the following 5 days and the patient was discharged with a foot drop splint.

## Method

### Literature review

A Medline search was undertaken for the period 1966 - 1998, based on the terms 'compartment syndromes' and 'anterior compartment syndrome'. This produced 1 618 references, which were further filtered using the key words 'exercise', 'exertion', 'athletic injury', 'acute', 'spontaneous' or 'traumatic'. Where there were subsets to any of these key words, all subsets were included in the filter. This produced 353 references. These were then reviewed and 56 papers (in English) reporting acute or spontaneous compartment syndromes relating to associated exercise were identified and studied. Six of these papers were reviews and two were comments on other papers, leaving 48 papers reporting cases of acute exertional compartment syndrome.

### Discussion

Acute exertional compartment syndrome is well documented in the literature; 112 patients with 124 compartment syndromes have been reported since 1912 when Dr Edward Wilson, the medical officer on Scott's Antarctic mission, recorded the signs and symptoms of the condition as he observed his own developing compartment syndrome.<sup>30</sup> The American literature recognises Vogt as the first to describe the condition in 1945, when he reported it in the military setting as 'march gangrene' following prolonged unusual exertion.<sup>30</sup> The literature prior to 1975 was summarised by Reneman, who describes the 52 patients with exercise-associated compartment syndrome reported up to that date.<sup>30</sup> The remaining 47 papers since then describe an additional 70 patients with 75 compartment syndromes.

While exertion is common to the compartment syndromes described in these patients, there are three identifiable subsets of situations associated with the compartment syndromes. These include compartment syndromes that appear to be solely exercise-induced (91 patients), those in which muscle contusion during participation in a sporting event appears to have been the precipitating event (15 patients), and those in which the compartment syndrome appears to be secondary to a muscle tear (6 patients).

Of interest is the distribution of the compartment syndromes. Early papers report on the anterior compartment of the leg (71 patients), with subsequent papers describing the condition in the anterior and lateral compartments (3 patients), lateral compartment (4 patients), superficial and deep posterior compartments (6 patients), and multiple compartments (3 patients). Acute compartment syndromes are not confined to the distal leg. Two acute compartment syndromes of the foot have been reported<sup>6,28</sup> and 23 patients have had compartment syndromes of the thigh.<sup>4,11,16,31,32,38,41</sup> Acute exertional thigh compartment syndromes are more commonly associated with contusions (11 patients) or muscle tears (8 patients). Two cases of paraspinal muscle compartment syndrome have been documented,<sup>8,10</sup> as have 4 cases involving muscles of the forearm<sup>1,5,13,15</sup> and one of the upper arm.<sup>14</sup>

In 10 cases the acute compartment syndrome appears to have been preceded by a history of chronic compartment syndrome.<sup>7,29,30</sup>

Acute exertional compartment syndrome is not confined to the athletic community. Sweeney *et al.*<sup>36</sup> reported a 79-year-old patient in a geriatric ward who developed an anterior compartment syndrome following intensive walking exercise as part of an active functional rehabilitation programme.

All the reports of acute exertional compartment syndrome have consistently stressed the need for vigilance when managing a case of limb pain in a healthy exercising person. The predominant presentation has been that of pain of increasing severity, which may start towards the end of exercise,<sup>30</sup> or which may start as late as 24 hours after exercise.<sup>34</sup> The pain is followed by sensory dysfunction and ultimately motor weakness. Many of the reported cases have involved delayed diagnosis with resultant morbidity, and in one instance, death.<sup>2,12,25,30,35</sup> In Reneman's review<sup>30</sup> of 52 patients prior to 1975, only 5 patients completely recovered after fasciotomy, suggesting that diagnosis had been delayed in the majority of cases.

Compartment syndrome has been described in many clinical situations other than exercise. It may be caused by either an increase in volume within a compartment, raising the pressure, or externally applied pressure compressing a compartment.

Clinicians must be aware of the many scenarios in which raised compartmental pressure may occur, as the diagnosis rests largely on clinical suspicion. The causes and pathophysiology have been reviewed in detail recently.<sup>19</sup>

### Diagnosis

Recognition of the development of raised compartmental pressure depends greatly on suspicion and clinical evaluation. Objective information about compartmental pressures can be gained from direct pressure monitoring, but as noted above, compartmental pressure is only one half of the equation, and elevation of compartmental pressure is not in itself diagnostic of impaired cellular nutrition.

It is important to realise that the aim is to recognise and treat raised ICP before irreversible cell damage occurs. Clinical suspicion should be heightened by the following clinical signs.

1. Pain that appears to be out of proportion to the stimulus, or pain that worsens.

2. Pain on passive stretching of the muscles in the compartment. Occasionally it may be difficult to distinguish pain of deep origin from discomfort at skin level.

3. Paraesthesia, which results from disordered cell membrane function in nerves running through the affected compartment. Anaesthesia will result if the pathology is allowed to proceed. Both pain and anaesthesia can only be assessed if peripheral nerve injury can be excluded.

4. Pressure within the compartment which can be appreciated clinically and is invariably associated with swelling and tenseness.

5. Pulses are present. Absence of pulses is indicative of direct arterial trauma, or of prolonged delay in making the diagnosis of raised compartmental pressure, with ICP rising to levels approximating mean arterial pressure, and occluding the main axial arteries. It must be remembered that capillary flow ceases at pressures well below mean arterial blood pressure, and it is upon capillary flow that cells depend for their survival. There may be perfectly normal flow in the major arteries and veins, with no blood able to percolate the tissues. A compartment syndrome with an absent arterial pulse and no arterial trauma has a poor prognosis and usually results in Volkmann's contracture or amputation.

6. Paralysis is frequently due to a combination of pain on muscle contraction, poor neuronal function and muscle cell death. A concomitant nerve injury should be excluded.

Eliciting these signs requires patient co-operation and is subjective. Direct pressure measurements can be made using needle manometry, a simple fluid-filled column,<sup>40</sup> a hand-held micro-processor,<sup>3</sup> or an *ad hoc* arrangement such as an arterial pressure monitor,<sup>22</sup> an Ivac pump,<sup>37</sup> or by placement of a wick<sup>27</sup> or slit catheter.<sup>33</sup>

It is our practice to measure ICP in the region of maximal pain using needle manometry. When measuring the deep posterior compartment of the leg we routinely attempt to measure the pressure in the tibialis posterior compartment. As falsely elevated readings may occur if the needle is sited in tendon or against bone, we take three readings, repositioning the needle without withdrawing it from the compartment. In children we have noted that with the large double-ported 18-gauge needle the side port is not always subfascial, and the resultant pressure reading is low. This is a problem in small compartments in small children, which has led us to evaluate the use of small single-ported needles in this situation.<sup>23</sup>

Pulse oximetry does not correlate with compartment pressures and should never be used to assess limb viability.<sup>18</sup>

## Management

As raised ICP threatens the viability of cells, and often the entire limb, this represents a true management emergency. The treatment is restoration of microcirculatory bloodflow, if necessary, by surgical decompression. Physiotherapeutic modalities play no role in the management of the condition and serve only to delay surgery.

The level of ICP at which surgery should be undertaken is

controversial. We feel that no absolute number should be quoted, as to do so takes no cognisance of the blood pressure that varies not only with the clinical status but also with the age of the patient. In adults we have offered surgery to patients whose compartmental pressure has risen to within 20 mmHg of the diastolic pressure,<sup>19</sup> while McQueen and Court-Brown<sup>26</sup> have suggested that fasciotomy be performed if the ICP rises to within 30 mmHg of diastolic pressure. In children we have performed fasciotomies when the ICP rises to within 30 mmHg of the mean arterial pressure.<sup>20,21</sup> As duration of pressure increase is an important variable, continuous or frequent intermittent ICP monitoring of patients at risk allows a trend to be identified that may predict appropriate management.

The non-surgical treatment of compartment pressure changes hinges on cell protection rather than direct relief of pressure. In patients in whom the diagnosis is being considered, we have followed a protocol of 'first aid to hypoxic cells'. This includes: (i) ensuring that the patient is normotensive, as hypotension reduces perfusion pressure and facilitates tissue injury; (ii) removing any circumferential constricting bandages as they increase ICP; (iii) maintaining the limb at heart height as elevation reduces the arterio-venous pressure gradient; and (iv) supplemental oxygen administration to ensure optimal saturation.

## Surgery

Fasciotomy should be open, wide and should decompress all the affected compartments. In selected athletes with chronic exercise-associated compartment syndrome there may be a place for 'closed' or limited skin incision fasciotomy.

Much of the morbidity associated with fasciotomy relates to wound closure, and long-term problems with venous pump function following fasciotomy appear to be uncommon.

## Conclusion

Acute compartment syndrome following exercise is rare but must be considered and excluded as a cause of post-exertional muscle pain. Clinical recognition of increasing or elevated ICP is unreliable in most patients. Invasive monitoring of an evolving situation is essential. When critical levels are approximated, urgent steps to restore capillary flow and maximise oxygenation of the tissues are necessary. Both sides of the pressure gradient may be manipulated. Restoration of systemic pressure may, therefore, be as important as and complementary to reduction in tissue pressure.

Surgical decompression must be open, wide and fully decompress the compartment. An increasing body of experience calls for inclusion of the fifth compartment of the leg, that of the tibialis posterior. Clinical failure may in part be due to microcompartment syndromes that occur simultaneously within the affected limb. Such microcompartments include the neural sheath.<sup>17</sup>

The morbidity of fasciotomy is significant although generally preferable to amputation, and pharmacological manipulation of the local response to injury holds out the prospect of non-operative management for the future.

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# Position Statement: Bicycle Helmets International Sports Medicine Federation (FIMS)

The following Statement was approved by the FIMS Executive Committee and has been reproduced without alteration.

Bicycles are owned by 1 in 10 people and are involved in more accidents per kilometer than any other vehicle excluding motorcycles.<sup>1</sup> In a study by Begg *et al.*, 57 of 848 cyclists reported 62 (6.7%) accidents with 40 (4.7%) injuries.<sup>2</sup> In an earlier paper by Kilburz *et al.*, accidents, defined in a broader sense, were reported by 46% of 492 cyclists with 9% requiring hospitalization and 23% having lost days from work.<sup>3</sup> In total, the bicycle injury rate is 163 per population of 100 000. Forty-two of these are head injuries, the most serious bicycle related injury.<sup>1,4</sup>

Head injuries in children are most frequently caused by bicycles and, according to various authors, result in 70 to 90% of bicycle related deaths<sup>5,6</sup> and 50% of significant injuries.<sup>9</sup> Only 20% of bicycle injuries involve the head, but these account for 70% of bicycle related hospitalizations.<sup>5,7,13</sup> The frequency of bicycle accidents is greatest in the 13 to 16 year old age group and 40% of bicycle deaths occur between the ages of 3 and 14.<sup>14,15</sup> Ninety percent of the fatalities involve a motor vehicle; 50% take place at an intersection.<sup>11</sup>

Helmet use plays a significant role in reducing the severity of trauma to the head, particularly when those approved by SNELL (Snell Memorial Foundation), CSA (Canadian Standards Association), or ASI (American National Standards Institute) are worn.<sup>5</sup> A study by Benz *et al.* showed that in a 1.5 meter fall, forces acting on the head were reduced fivefold from 547 - 1 078 g to 122 207 g with the use of a helmet.<sup>10</sup> Another report states that, at 15 kilometers per hour, the energy absorbed by the head is lessened by 90% if a helmet is worn.<sup>5</sup> Helmet use has been shown to reduce the Injury Severity Score (ISS) from 18 to 3.8; serious head injury from 47 to 5.2%; mortality from 60 to 0.9%; the occurrence of head injury by 85 to 95%;<sup>1,13,14</sup> brain injury by 88%;<sup>13</sup> skull fractures from 11 to 1%; soft tissue facial injuries from 18 to 5%.<sup>5,15</sup>

While literature is inconsistent, reports by various authorities clearly reflect the positive impact of helmet use on prevention. These describe decreases in injury rates ranging from 39 to 90%.<sup>1,11,16,17</sup> As well, reductions of 86% in loss of consciousness,<sup>17</sup> 40% in fatalities, 20% in total injuries,<sup>18</sup> and a protection factor of 3.25 (11% versus 4% head injuries)<sup>10</sup> are reported.

These estimates support observations that a bicycle helmet was not worn by any child who sustained a head injury<sup>5,9</sup> or died<sup>5</sup> as a result of a head injury. Cooke *et al.* report that, in Western Australia, helmets reduced deaths by eight-fold.<sup>20</sup> It appears that a cyclist travelling on a hard surface at 25 kilometers per hour will be protected from head injury by a standard helmet.<sup>21</sup> The overall effect of bicycle helmets has been compared to that of a car seat belt.<sup>22,23</sup> Consequently, various authorities recommend regular use of helmets by cyclists of all ages.<sup>5,24,25</sup>

Compliance with bicycle helmet use can be improved through education and legislation. Increases ranging from 8 to 13%<sup>26</sup> and 5 to 62%<sup>27</sup> have been attributed to campaigns

in education and the media. Education has been most successful in elementary schools, resulting in a 70% compliance rate.<sup>28</sup> Other factors affecting helmet use include subsidization of helmet costs<sup>29,31,32</sup> and cultural influences.<sup>33</sup> The history of previous injury did not affect compliance.<sup>30</sup> Several references in recent literature describing the various aspects of public campaigns and education report that 20 to 73% of the populations studied eventually used helmets.<sup>26-29,31-37</sup> Two such campaigns resulted in limited, 8 to 43%,<sup>26</sup> or no effect on compliance.<sup>29,33</sup> Legislation has been the strongest influence, credited with increases ranging from 47% to 90%.<sup>28,31,38</sup>

## Summary and conclusions

Bicycle helmets reduce the incidence of head, face, and brain injuries. Both educational campaigns and legislation have improved child, adolescent, and adult compliance, but legislation is more effective. It is important that bicycle helmet education and legislation be promoted by all health care providers.

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