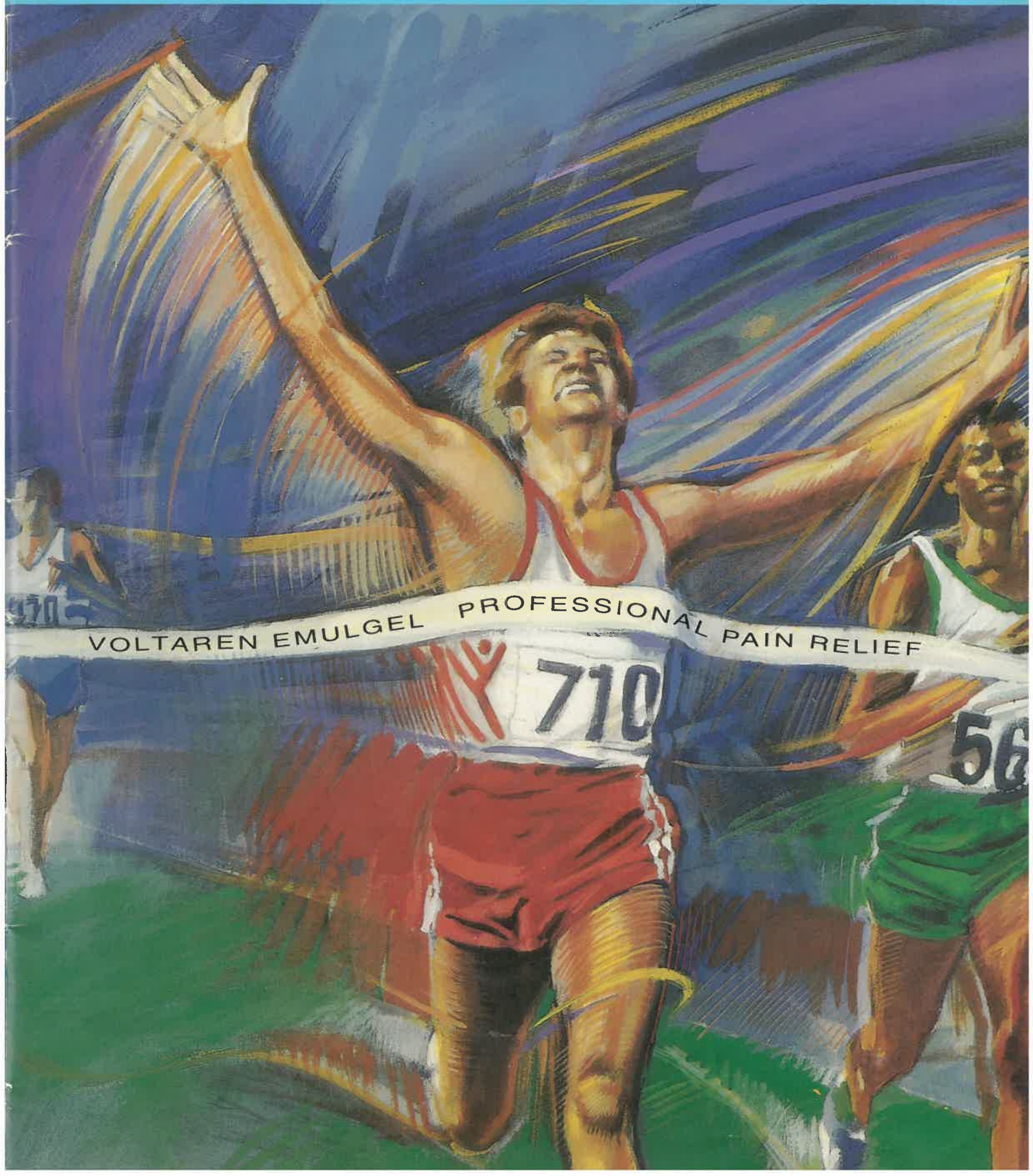
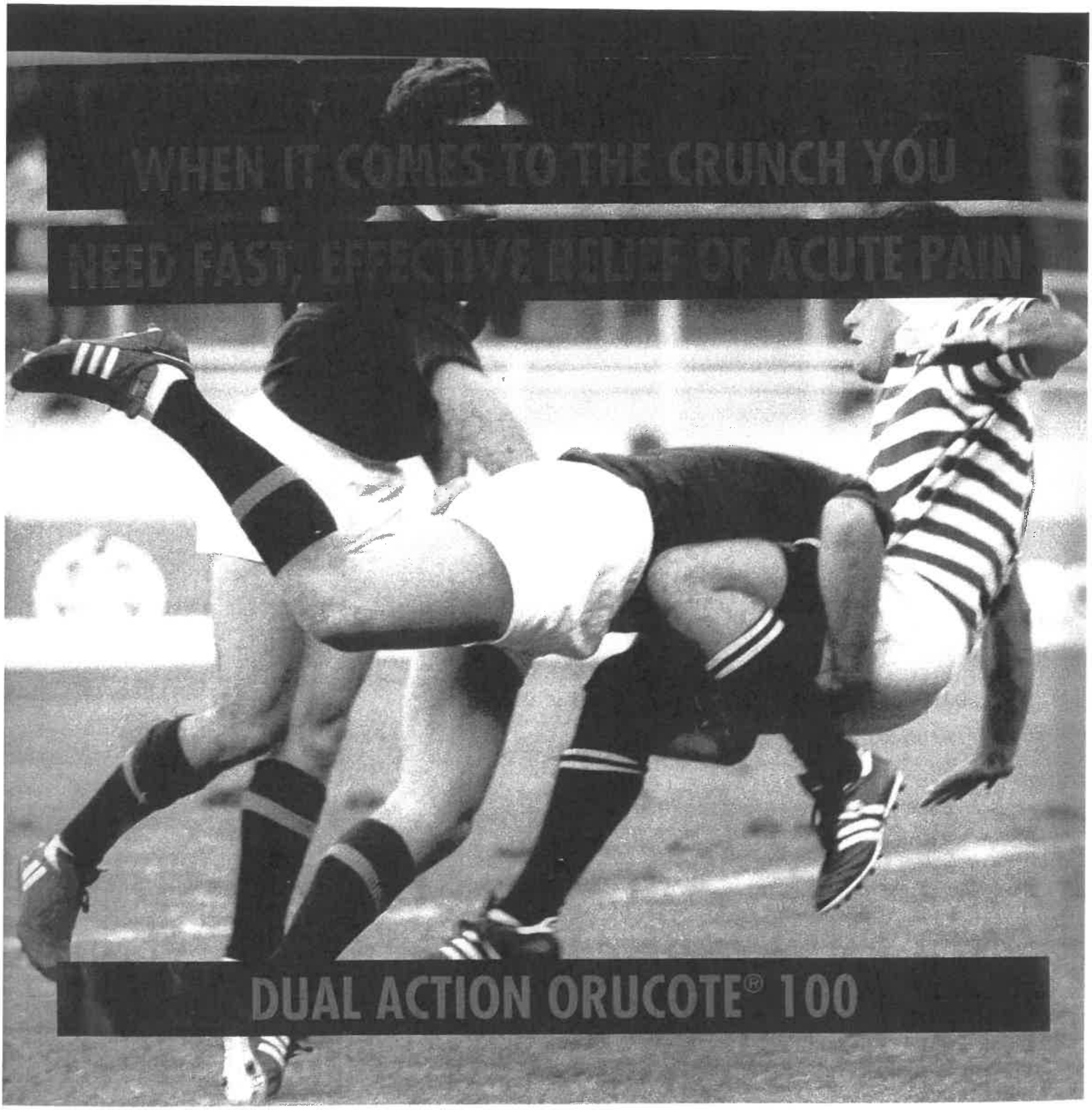
SOUTH AFRICAN JOURNAL OF

SPORTS MEDICINE

Vol 2 No 1 March 1995





"The discovery of a central component of post-injury pain hypersensitivity.. indicates that therapy directed only at the peripheral changes may be inadequate to prevent pain."

Woolf C J. The role of NSAIDs in the management of postinjury pain, 1992 [1]

Rely on dual action **ORUCOTE® 100** for rapid **peripheral** [2,3,4] **and central** [2,5,6,7] relief of acute pain.

DUAL ACTING
ORUCOTE® 100
DUAL ACTING PAIN RELIEF

1. Woolf CJ. The role of NSAIDs in the management of postinjury pain. Postinjury Pain Symposium Series, Oxford, 31st March 1992, Chairman Prof Dr K Brune. 2. Netter P and coll. Eur J Clin Pharm 1985, 29 (3): 319. 3. Kantor TC. Pharmacotherapy, 1986, 6,3: 97. 4. Huber-Bruning et al. Scand J Rheuma 1988:15(10). 5. Boulu P, Bastien C. Electrophysical and biochemical evidence for a central analgesic action of ketoprofen. Rev Rheum Mal Ostearlic, 1989, 56 (3): 249. 6. Dubourdio C, Dray F. Central analgesic action of ketoprofen on two main types of mediators of central nociception. 3rd Interscience World Conference on Inflammation, Antirheumatics, Analgesics and Immunomodulators, 1989, 15-18 March. 262. 7. Willer JC, De Broucker T et al. Central analgesic effect of ketoprofen on humans: Electrophysiological evidence for a supraspinal mechanism in a double blind and cross-over study. Pain, 1989, 38 (1): 1-7.

Ketoprofen 100 mg

GUEST EDITORIAL

Is exercise beneficial or harmful to the gastrointestinal tract?

It is well recognized that exercise has benefits for the musculoskeletal, cardiovascular, pulmonary and renal systems. However, risks and benefits of exercise for the gastrointestinal tract have not been well documented. This edition of the South African Journal of Sports Medicine draws attention to the relationship between exercise and the gastrointestinal tract (GIT). The frequency of lower gastrointestinal symptoms during exercise varies from 25% to 60%, and that of upper gastrointestinal symptoms from 6% to 58%. Previously published articles describing the frequency of gastrointestinal symptoms during exercise have only focussed on one sport at a time.

Utilizing novel methodology Schwartz, Schwellnus and Koorts documented the true incidence of GIT symptoms as expressed by symptoms/athlete/1 000 exercise hours, in six different endurance sports in South Africa. Their findings show that lower gastrointestinal symptoms in endurance exercise are more common than upper gastrointestinal symptoms; that females have more symptoms than males; that younger (novice) athletes are more likely to have symptoms than experienced athletes; and that symptoms are more likely to occur during hard exercise and competition than in training. These authors also discuss a possible new mechanism for the development of GIT distress in endurance sports. It appears that athletes who have had a previous history of abdominal or gynaecological surgery are more likely to develop gastrointestinal distress during exercise. Furthermore, drug use appears to be a common method to control gastrointestinal symptoms as indicated in this study by Schwartz et el.

The article by Garisch and Schwartz, on the Management of GIT Distress During Exercise, provides information for first aiders, coaches, trainers and doctors, to assist athletes who present to them with mild or severe forms of gastrointestinal distress during exercise.

What and how much to eat or drink during exercise has confused some of us runners who have been jogging since the early 1970's. We have seen the pendulum of advice swing from one extreme to the other. One decade it was salt, the next decade water and then only recently, the need to ingest carbohydrate during prolonged exercise. Dennis, Hawley and Noakes in their article on the Limits to the Replacement of Fluid, Electrolyte and Energy during Prolonged Exercise provide an interesting historical perspective of how much fluid to ingest during exercise. The first evidence of severe hypoglycaemia during athletic performance was documented in the 1924 Boston Marathon. Six runners finished the race in a confused and disorientated state. These same six runners were told to ingest carbohydrates during their 1925 Boston Marathon run. Not only did these athletes feel better after the race, but also, their performance was enhanced. The authors of this article give the athletic population insight and understanding of the importance of fluid and energy requirements during exercise. Much of the confusion surrounding this subject is clarified by their article.

This March 1995 edition of the South African Journal of Sports Medicine coincides with the 6th South African Sports Medicine Association Çongress (The Pursuit of Excellence) in Durban from March 22-24, 1995. For those of you who attend, the Editorial Board wishes you a fruitful and successful Congress.

Dr Peter Schwartz

CONTENTS

THE EDITOR
THE SOUTH AFRICAN JOURNAL OF SPORTS MEDICINE
PO Box 38567, Pinelands 7430

PRODUCTION
Andrew Thomas

PUBLISHING
Glenbarr Publishers cc
Private Bag X14
Dunkeld 2196
Tel: (011) 442-9759
Fax: (011) 880-7898

ADVERTISING
Marika de Waal/Andrew Thomas

REPRODUCTION
Output Reproduction

PRINTING
Hortors

Cover sponsored by Ciba-Geigy

Limits to the replacement of fluid, electrolytes and energy during prolonged exercise

Steven C. Dennis PhD
John A. Hawley PhD, FACSM
Timothy D Noakes MB ChB, MD, FACSM

Introduction

The first studies showing that carbohydrate (CHO) ingestion during exercise lasting more than 90 min enhanced performance by preventing hypoglycaemia were conducted in the 1920's and 1930's.[1,2,3] However, these findings were largely ignored by the athletic community, as too were the early industrial and military investigations showing the importance of adequate fluid replacement during prolonged exercise in the heat.[4,5]

One of the first references to fluid replacement during prolonged exercise was in the 1953 International Amateur Athletic Federation (IAAF) Handbook controlling marathon (42 km) foot races (Figure 1.). The handbook stated that "refreshments shall (only) be provided by the organisers of the race after 15 km. No refreshments may be carried or taken by a competitor other than that provided by the organisers." As water was the only drink available to runners, it was clear that the IAAF had little knowledge of the benefits of CHO ingestion during prolonged exercise.

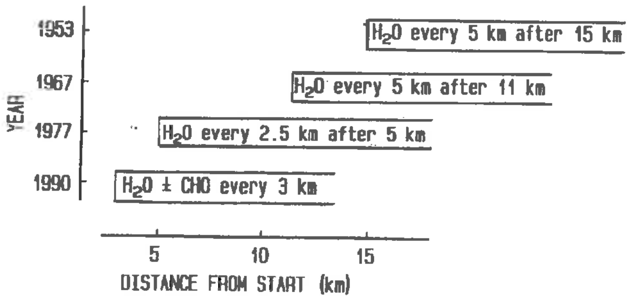


Figure 1: *Changes in the International Amateur Athletics Federation rules on drinking in long distance running races over the past 40 years.*

Over the next 20 years, water rather than CHO ingestion was promoted during exercise, in part because of studies showing that runners who were the most dehydrated after distance races had the highest post-race rectal temperatures.[6] This observation led to the belief that rises in rectal temperature and dehydration were causally related, so that water, in large volumes, should be ingested to prevent "heat-exhaustion". However, this hypothesis was only partially correct. The major determinant of the rise in rectal temperature during exercise is the metabolic rate, which determines sweat rate and hence the level of dehydration. Levels of dehydration have rather small, independent effects on rises in core temperature during exercise, which are described later.

Nevertheless the notion that fluid replacement alone was of primary importance for optimising performance during prolonged exercise was promoted to such an extent that CHO ingestion was discouraged because it was believed to slow the rate of gastric emptying and hence the rate at which fluid could be replaced during exercise.[7] Indeed, in the 'state-of-the-art' proceedings of the 1976 New York Academy of Sciences conference "The Marathon: Physiological, Medical, Epidemiological, and Psychological Studies", there was not a single reference to CHO ingestion during prolonged exercise.[8]

The question of whether replacement or CHO ingestion should be emphasised during prolonged exercise was not fully resolved until the mid to late 1980's, when American commercial interests revived scientific research into the value of CHO ingestion during exercise. Not surprising, those studies confirmed the 50 year old findings in the field[2,3] and showed that the ingestion of CHO solutions also enhanced exercise endurance in the laboratory.[9,10] Hence, the IAAF currently advocates the consumption of CHO-electrolyte beverages in all races of 10km and longer (Figure 1).

Fluid changes during prolonged exercise

The exact amount of fluid, electrolytes and CHO that should be consumed to replace sweat and energy losses during exercise, however, remain to be established. The losses of Na^+ and Cl^- ions in sweat depend on an individual's level of fitness and heat acclimation: despite a 12% increase in overall sweat rate after heat acclimation, Na^+ losses in sweat decrease by almost 60%.[11]

Estimated sweat rates in runners racing over varying distances are shown in Table 1. These data show that while the sweat rates of runners in races lasting > 2 hr are ~ 1 l/hr, their rates of fluid intake are usually < 0.6 l/hr.[12] Such discrepancies result in body mass losses of 2-3 kg and this loss of weight appears to be independent of race duration.

Address for correspondence:
Professor Steven C. Dennis
MRC/UCT Bioenergetics of Exercise Research Unit
Department of Physiology
University of Cape Town Medical School
Observatory 7925
South Africa

TABLE 1: Rates of fluid loss and fluid ingestion during running races over various distances

Race distance (km)	Fluid intake (l/hr)	Sweat rate (l/hr)	Body mass loss (kg)
32	0.2	1.4	2.4
42*	0.4 ± 0.2	1.1 ± 0.1	2.4 ± 0.3
56	0.5	0.9	2.0
67	0.4	0.8	2.4
90	0.5	0.9	3.5

Data are from Noakes.[12] 42 km marathon are the means ± standard deviation of the average values from 7 studies on male subjects. Female sweat rates were lower than the males over 42 km (0.6 vs. 1.1 l/hr) and over 67 km (0.5 vs. 0.8 l/hr).

One explanation for the failure of most athletes to meet their fluid requirement during exercise is that they develop symptoms of abdominal 'fullness' when they attempt to drink fluid at high rates.[13,14] Distension of the bowel reduces the desire to drink until the ingested fluid has been absorbed. Feelings of abdominal 'fullness' are probably due to limited rates of ingested fluid absorption. Based on urine output, the maximum rates of ingested water absorption are probably ~ 0.8 l/hr.

Alternatively, humans may dehydrate during exercise because they lose up to 60 mEq of Na^+ for each litre of sweat[15] (Figure 2). As a result, the equivalent of ~ 10 g of NaCl is lost over three hours of exercise and serum osmolality does not rise sufficiently to induce an adequate dipsogenic drive. Thirst therefore ceases prematurely when serum osmolality is returned to isotonicity by the ingestion of either plain water or isotonic CHO +

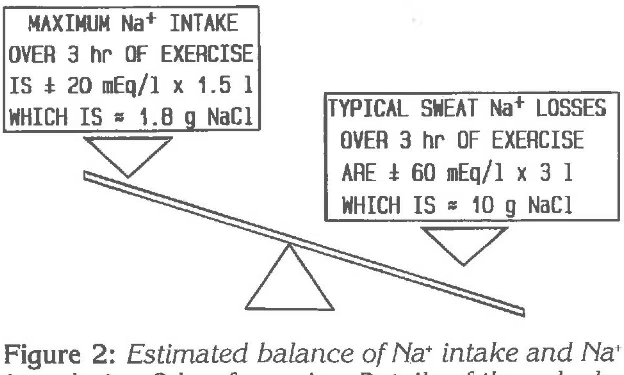
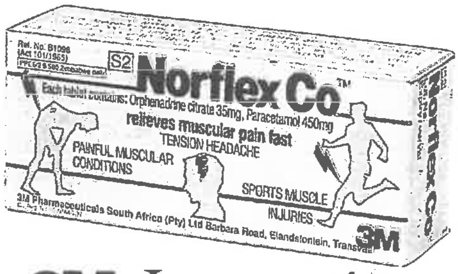
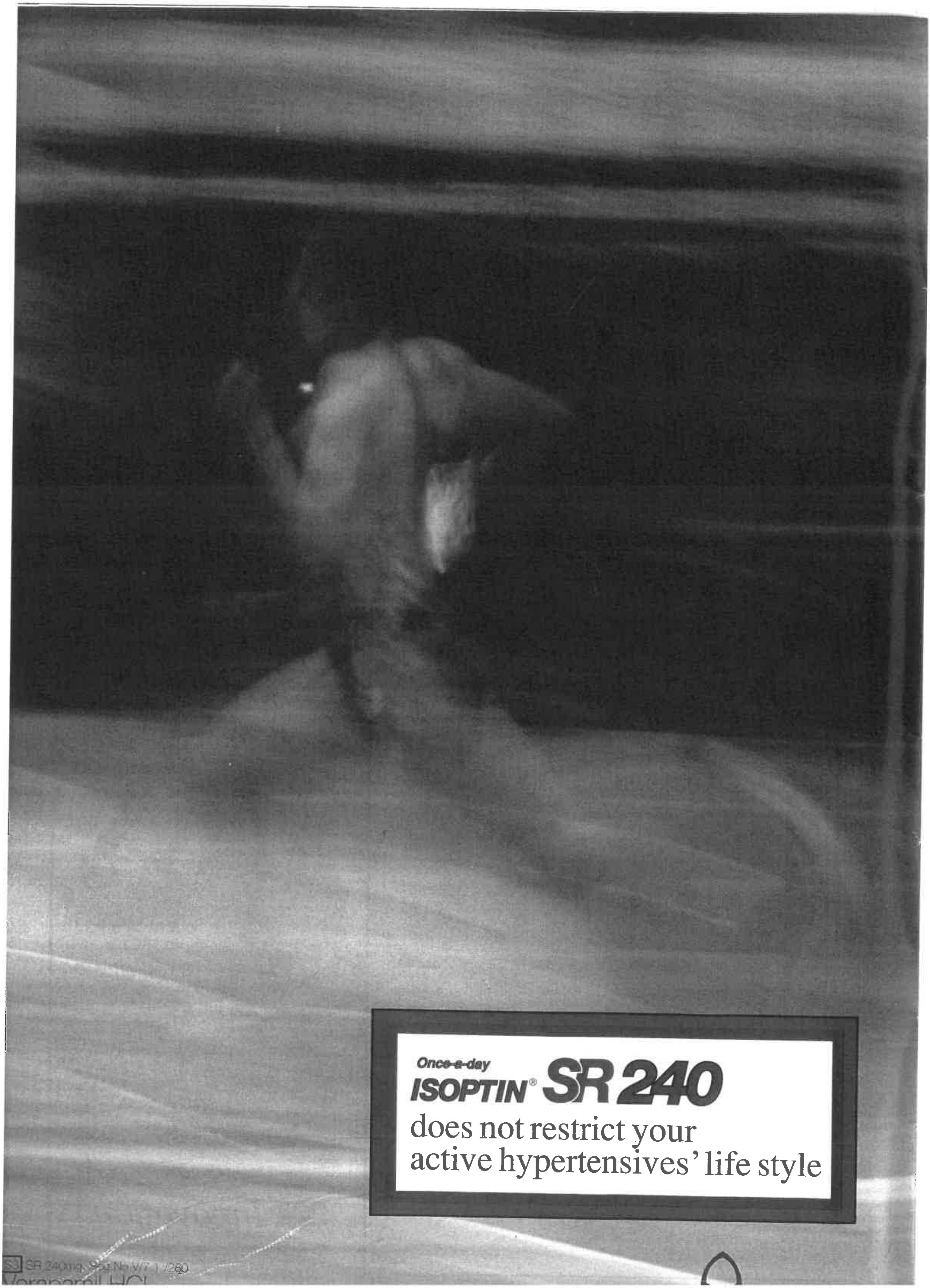


Figure 2: *Estimated balance of Na^+ intake and Na^+ loss during 3 hr of exercise. Details of the calculations are given in the text.*

NaCl solutions.[16] A complete restoration of the fluid and electrolyte losses in three hours of exercise can only be achieved by the consumption of ~10g of NaCl in food over a 24 hr period.

Drinking isotonic (280 mOsm) CHO + NaCl solutions will not adequately replace sweat Na^+ losses. For the maintenance plasma and interstitial Na^+ ion concentrations and hence volumes, an athlete would have to ingest one litre of a CHO solution containing 60 mmol/l of NaCl every hour.




Once-a-day
ISOPTIN® SR 240
does not restrict your
active hypertensives' life style

Such a solution would not end up in the intestine; it would end up on the side of the road! NaCl is only palatable up to concentrations of 20 mmol/l and most athletes can only drink ~ 0.5 l/hr. Hence, whether the replacement of 10 of the ~ 60 mEq of Na^+ lost in sweat per hr significantly helps to maintain plasma volume during prolonged exercise is open to debate.

There is also a question as to whether adding NaCl to CHO containing drinks increases rates of fluid absorption. While isotonic CHO + NaCl solutions have been shown to be absorbed more rapidly than water[17] or isotonic NaCl,[18] the effect of NaCl on the absorption of CHO solutions has not been reported. The omission of such an obvious study suggests that NaCl probably has little influence on the absorption of CHO solutions.

Certainly there is no physiological rationale for adding NaCl to drinks to promote fluid absorption. As ingested solutions empty from the stomach into the duodenum, the gastric HCl secretions are neutralised by the pancreatic secretion of high (140 mmol/l) concentrations of $NaHCO_3$ into the chyme and the products of that neutralisation are CO_2 and NaCl (Figure 3). This addition of isotonic NaCl to the chyme 'guards' against major water and electrolyte losses from the vascular compartment into the bowel, that would otherwise occur through the 'leaky' junctions between the columnar epithelial (absorptive) cells of the duodenojejunum. Hence, any ingested solution eventually contains sufficient NaCl for absorption.

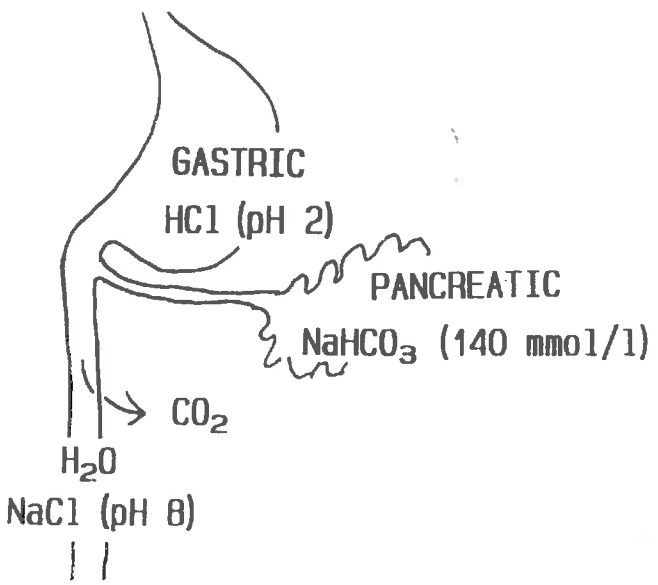


Figure 3: *Neutralisation of gastric HCl secretions by pancreatic $HaHCO_3$ production. This figure shows that manufacturers of 'sports drinks' do not need to add NaCl to their products. That task can be left to the stomach and the pancreas.*

Effects of 'voluntary' dehydration during exercise

Absorption of fluid from the intestine mainly helps to attenuate the progressive falls in plasma volume after ~ 1 hr of exercise. During the first hr of exercise, changes in plasma volume are unaffected by drinking.[19] In the first 5-10 min of exercise, rises in blood pressure 'push' fluid from the plasma into the interstitium and plasma volume falls by ~ 8% at 55% of maximum oxygen uptake (VO_2 max)[20] to ~ 18% at 90% of VO_2 max.[19] Thereafter, plasma volume remains relatively constant until the loss of fluid from the vascular compartment to sweat is no longer replaced by fluid movements out of the interstitium.

If no fluid is ingested during prolonged exercise, serum Na^+ concentration, osmolality and anti diuretic hormone activity all increase.[21] With severe dehydration, rises in serum Na^+ concentration and osmolality correlate with the increase in oesophageal temperature and may be a stimulus for the reduction in skin blood flow and sweating that develops at advanced levels of dehydration.[22] An important goal of fluid ingestion during exercise may therefore be to prevent rises in serum osmolality and thereby maintain sufficient skin blood flow for maximum evaporative and convective heat losses.

With sufficient fluid ingestion, rises in rectal or oesophageal temperatures are attenuated. However, the magnitude of this effect is not great. Most studies indicate that a 2-4 l fluid loss increases rectal temperature by < 1°C, whereas the rise in metabolic rate associated with high-intensity exercise can increase the rectal temperature by 3-4°C.[22,23,24]

Fluid deficits that develop during exercise also proportionately reduce stroke volume and increase heart rate. Falls in stroke volumes are prevented when the rates of fluid ingestion are sufficient to maintain euhydration,[22] but heart rates continue to rise as catecholamine concentrations increase during exercise, even when dehydration is prevented.[25]

Perhaps the greatest effect of fluid ingestion is on the perception of effort during prolonged exercise, especially in the heat. Improvements in ratings of perceived exertion with fluid ingestion correlate better with increases in endurance than any other physiological variable currently measured.[22] We have recently shown that fluid replacement during a 60 min cycle at 70% of VO_2 max significantly improves the perception of effort and time to exhaustion during a subsequent cycle at 90% of VO_2 max at ambient temperatures of 32°C, even with very modest (1.1-1.3l; 1.8% of starting body mass) levels of dehydration.[19]

In contrast, there is probably no advantage in trying to replace fluid losses during intense exercise in a thermoneutral environment. Our recent data show that high rates of fluid ingestion (>1.5 l/hr) impaired performance in a 40km cycling time trial lasting ~ 60 min.[26] At these high (~ 85% of VO_2 max) exercise intensities, sweat rates were ~ 1.7 l/hr and attempts to replace those fluid losses simply filled the small intestine with ~ 1.0l of water and resulted in a feeling of abdominal discomfort. Only 0.1-0.2l of the water delivered to the intestine from the stomach was absorbed during the exercise bout.

Carbohydrate ingestion during exercise

Water absorption into the body largely results from the intestinal re-absorption of NaCl and the co-transport of glucose, galactose or amino acids with Na^{+} ions. As these solutes are actively absorbed, an iso-osmotic equivalent of water simply follows to keep the osmotic pressure of the small intestine the same as that of the plasma. With the exception of fructose which is only slowly absorbed in humans, the ingestion of any non-hypertonic CHO solution should increase rates of intestinal Na^{+} re-absorption and fluid uptake. Further the appearance of hexose and some triose molecules from CHO digestion in the systemic circulation will provide fuel for prolonged exercise.[27]

Originally, the rate of ingested CHO oxidation was assumed to be limited by gastric emptying, and therefore affected by factors such as the drink osmolality and caloric content.[28] More recent studies, however, have shown that the volume of the drink is a more important determinant of its rate of gastric emptying than its osmolality or caloric content. When CHO solutions with vastly different osmolalities are repeatedly ingested in sufficient volumes during exercise, their rates of gastric emptying are quite similar.[27] Typically, we feed subjects 400 ml at the start of exercise and, thereafter, 100 ml every 10-15 min. With such an ingestion protocol, the residual gastric fluid volume remains constant at 300-400 ml and the delivery of a 15 g/100ml glucose solution into the intestine at a rate of 10 ml (1.5 g of CHO/min) exceeds the peak rates of ingested glucose oxidation (Figure 4).[29] Irrespective of the ingestion regimen, rates of ingested glucose oxidation only rise to ~ 1 g/min after 60-90 min of exercise at intensities of >50% of VO_2max.[27] Hence, the rate of oxidation of glucose ingested repeatedly during exercise is not limited by gastric emptying, as originally proposed.[7]

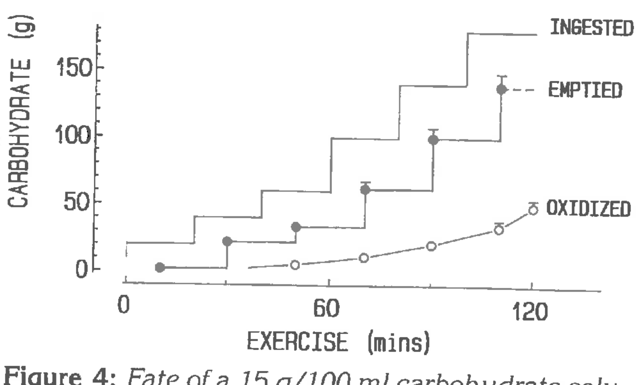


Figure 4: *Fate of a 15 g/100 ml carbohydrate solution repeatedly ingested during 120 min of cycling at 70% of VO_2 max. Note that far more carbohydrate is emptied from the stomach than is oxidised. An excessive accumulation of glucose in the bowel may impair fluid absorption.*

Instead, these and other data suggest that the oxidation of ingested CHO after 60-90 min of exercise is limited either by its rate of appearance in the systemic blood supply or by the rate of muscle glucose oxidation. Comparisons of the oxidation rates of ingested sucrose, maltose, varying chain length glucose-polymers and even complex starches during exercise have shown that ingested CHO oxidation is not limited by digestion.[27] With the exception of fructose, which is absorbed 50-60% more slowly than glucose,[30,31] all of the above ingested CHO's were oxidised at peak rates of ~ 1 g/min after 60-90 min of exercise.[27]

After ~ 1 hr of exercise, ingested CHO oxidation is also not limited by its rate of appearance in the systemic blood supply. We recently showed that an intravenous glucose infusion, which could theoretically supply an unlimited rate of U-^{14}C labelled glucose into the systemic circulation, did not elicit > 1 g/min rates of glucose oxidation in cyclists riding for 2 hr at 70% of VO_2max than when they ingested the U-^{14}C glucose.[32] The only difference was that the higher plasma insulin concentrations with glucose ingestion reduced the final contribution to energy production from fat oxidation from 51±10% to 18±4% and thereby increased muscle glycogen oxidation.

Instead, the eventual rates of ingested CHO oxidation are more likely to be regulated by the prevailing concentrations of plasma glucose (5 mmol/l) and insulin (10 mU/l) during exercise. When we intravenously infused U-^{14}C glucose into six cyclists during 2 hr of moderate-intensity exercise to increase their plasma glucose and insulin concentrations from 5 to 10 mmol/l and from 10 to 25 mU/l, respectively, their peak rates of plasma glucose oxidation rose from 1.0 to 1.8 g/min.[33] Thus, the maximum rates of ingested CHO oxidation during exercise are probably limited by the rate at which physiological concentrations of glucose can be oxidised by the working muscles.

Fat ingestion during exercise

Because muscle glucose utilisation is limited, it is still important for endurance athletes to maximise their starting working muscle glycogen stores by 'CHO-loading'. Ideally, it would also be desirable to slow muscle glycogen depletion by promoting the utilisation of fat. Unfortunately, most fat is ingested as long-chain (C_{16-22}) triacyglycerols and their rates of digestion and absorption via the lymphatic system are too slow to be of any advantage to the athlete during exercise. Lipaemia only develops several hours after a meal containing fat and, when it does occur, the circulating triacyglycerols 'packaged' in chylomicrons are mainly stored rather than directly oxidised.

In contrast, ingested medium-chain (~C_{8-10}) triacylglycerols are metabolised as rapidly as glucose.[34] In this case, the products of pancreatic lipase activity are not re-assembled into triacylglycerols for incorporation into chylomicrons for transport via the lymph. Because medium-chain fatty acids are more water-soluble than long-chain fatty acids, they diffuse across the enterocytes and enter the systemic blood supply via the hepatic portal system. Once in the blood supply, medium-chain fatty acids diffuse into muscle mitochondria independently of the transport mechanism(s), that limit the rates of long-chain fatty acid oxidation.

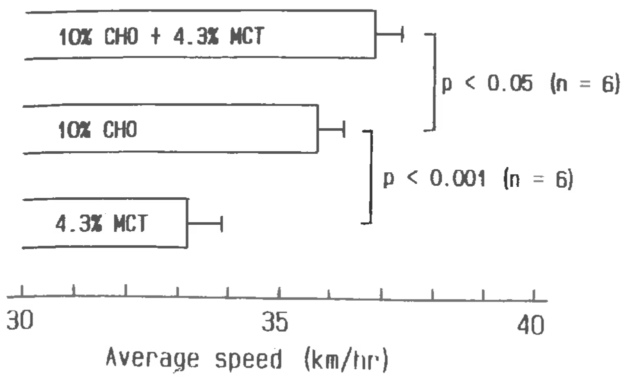


Figure 5: *40 km cycling performances after 2 hours of exercise at 60% of VO_2 max. These data show that the addition of medium chain triacylglycerols (MCT) to carbohydrate (CHO) solutions ingested during exercise improve athletic performance.*

Hence, medium-chain triacylglycerols can be rapidly oxidised and are currently attracting commercial interest.

Our studies of adding 4.5% medium-chain triacylglycerols to 10% glucose solutions ingested by six cyclists during a 2 hr ride at 60% of VO_2 max followed by a stimulated 40km time-trial showed marked improvements in their athletic performance (Figure 5).[35] Times to complete the 40km rides were consistently decreased by ~ 2 min.

Optimal fluid replacement during exercise

Based on the above information, the advantages of adding electrolytes to drinks consumed during exercise are probably not as great as their manufacturers would have us believe. However, there is no doubt that some form of CHO (other than fructose) should be ingested during prolonged (> 90 min), moderate intensity exercise. Under these circumstances, liver glycogen depletion and hypoglycaemia can limit endurance.[9]

Whether an athlete should ingest a large amount of CHO at the onset of exercise, however, is open to question. As only 20 g of ingested CHO is utilised in the first hour of exercise,[27] drinking more than that amount of CHO may attenuate the fall in insulin concentration and thereby delay fat mobilisation.[33] A resultant increased reliance on the limited working muscle glycogen stores early in exercise could lead to a more rapid onset of muscle fatigue.

High concentrations of CHO accumulating in the small intestine may also impair fluid absorption. While isotonic glucose polymer drinks enter the stomach with an osmotic pressure of 280 mOsm, the products of their digestion can become very hypertonic if they accumulate in the bowel. For instance, a 10 g/100 ml concentration of unabsorbed glucose in the intestine would produce an osmotic pressure of 555 mOsm. That osmotic pressure, together with the osmotic pressure generated by the $NaHCO_3$ secreted into the chyme, would be expected to 'pull' water into the bowel.

In the first 60-75 min of exercise, athletes should therefore probably consume 100 ml every 10 min of a dilute (3-5 g/100 ml) CHO solution. Only after 90 min of exercise should the ingested CHO concentration perhaps be increased to ~ 10 g/100 ml to match the peak (~1 g/min) rates of blood glucose oxidation.

Because the provision of medium-chain free fatty acids slows the depletion of muscle glycogen and provides a readily oxidisable alternative fuel source to CHO,[35] it might also be advisable for endurance-athletes to ingest medium chain triacylglycerols. We would recommend that they begin ingesting 100 ml every 10 min of a ~ 4.5 g/100 ml solution of medium-chain triacylglycerols 60-90 min before exercise and, thereafter, add the same amount of medium-chain triacylglycerols to the CHO solutions ingested during exercise. With this drinking pattern, medium chain free fatty acids would be available from the start of exercise and the stomach would be filled to tolerable (~300 ml) limits to promote gastric emptying.

REFERENCES

1. *Dill DB, Edwards HT, Talbott JH. Studies in muscular activity: VII Factors limiting the capacity for work. J Physiol (London) 1932; 77: 49-62.*
2. *Levine SA, Gordon B, Derick C L. Some changes in the chemical constituents of the blood following a marathon race. JAMA 1924; 82: 1778-1779.*
3. *Gordon B, Kohn LA, Levine SA, Matton M, Schriver W, Whiting WB. Sugar content of the blood in runners following a marathon race, JAMA 1925; 185: 508-509.*
4. *Ladell WSS. The effects of water and salt intake upon the performance of men working in hot and humid environments. J Physiol 1955; 127: 11-46.*
5. *Talbott JH, Dill DB, Edwards HT, Stumme EH, Consolazio WV. The ill effects of heat upon workmen, J Hygiene Toxicol 1937; 19: 258-274.*
6. *Wyndham CH, Strydom NB. The danger of an inadequate water intake during marathon running, SA Med J 1969; 43: 893-896.*
7. *Costill DL, Saltin B. Factors limiting gastric emptying during rest and exercise, J Appl Physiol 1974; 37: 679-683.*
8. *Milvy P. (Ed) Ann NY Acad Sci. The Marathon: Physiological, Medical, Epidemiological, and Psychological Studies. Volume 301, The New York Academy of Sciences, New York, 1977.*
9. *Coyle EF, Hagberg JM, Hurley BF, Martin WH, Ehsani AA, Holloszy JO. Carbohydrate feeding during prolonged exercise can delay fatigue, J Appl Physiol 1983; 55: 230-235.*
10. *Coyle EF, Coggan AR, Hemmert MK, Ivy JL. Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. J Appl Physiol 1986; 61: 165-172.*
11. *Kirby CR, Convertino VA. Plasma aldosterone and sweat sodium concentrations after exercise and heat acclimation. J Appl Physiol 1986; 61: 967-970.*
12. *Noakes TD. Fluid replacement during exercise. In Holloszy JO (Ed). Exercise and Sport Sciences Reviews, Volume 21. Williams and Wilkins, Baltimore, pp. 297-330, 1993.*
13. *Brouns, F., Saris, W.H.M., and Rehrer, N.J., Abdominal complaints and gastrointestinal function during long-lasting exercise, International Journal of Sports Medicine 1987; 8; 175-189.*
14. *Brouns, F., Beckers, E., Knopfli, B., Villiger, B., and Saris, W., Rehydration during exercise: effect of electrolyte supplementation on selective blood parameters, Med Sci Sports Exerc, 1991; 23, S84.*
15. *Gisolfi CV, Summers R, Schedl H. Intestinal absorbtion of fluids during rest and exercise. In: Gisolfi CV, Lamb DR, eds. Perspectives in Exercise Science and Sports Medicine, Volume 3. Fluid Homeostasis During Exercise. Benchmark Press Inc, Carmel, Indiana, 1990: pp. 129-180.*

(Continued on page 15)

Clinical approach to the diagnosis and management of gastrointestinal symptoms in endurance athletes

PA Schwartz, MB ChB, BSc Sports Medicine (Hons)
JAM Garisch

Introduction

During the last decade there has been a proliferation of all types of enduro-sports amongst adults and children both in the recreational or competitive field. These enduro-sports include aerobic dance workouts, canoeing, running, the triathlon, cycling and swimming.[1]

There has been a recent upsurge of interest in the research of the physiological effects of running with prolific metabolic, renal, cardiovascular and pulmonary responses to exercise have been well researched.[2] However, alterations of gastrointestinal (GIT) function during prolonged endurance exercise, in particular the underlying mechanisms and aetiology of GIT distress has largely been ignored. This paucity of knowledge underlines the need for further research in this neglected field.[3,4]

The fact that physical activity can influence GIT function has been recognized as far back as 1832. Bell and Roche stated in the appendix of Broussai's "Treatise on physiology", that ingestion is impaired by the exercise of the body meals.[5] Although this observation had no scientific or experimental backing, it has since been recognized that changes in bowel habits can occur when sedentary individuals embark on an exercise programme.[6]

The reason why most athletes seek medical advice is for musculoskeletal injuries. Injuries are usually severe enough to cause pain which prevents optimum performance. However, most GIT symptoms are usually mild and do not cause severe discomfort. Athletes are therefore often reluctant to obtain medical advice.

The severity of GIT distress experienced during exercise can range from mild to severe. Mild complaints of the upper GIT are dyspepsia, nausea, vomiting, loss of appetite and flatulence. The common lower GIT complaints are abdominal cramping, urge to defeacate and diarrhoea.[10,11] In 1969 GIT bleeding (haematochezia) during exercise was first reported in the Runner's World Magazine.[12] In 1982 the first case of a documented death due to massive haemorrhagic gastritis was reported in a 28 year old runner while jogging.[13] Severe GIT distress has also recently received attention in South Africa. The female winner of the 1990 Comrades Marathon, had profuse vomiting at the end of her 96 km run. Bruce Fordyce, one of the greatest ultramarathon runners and most successful consecutive winner in the history of the Comrade Marathon, failed to obtain a gold medal during the 1991 event, due to numerous "pit stops" secondary to diarrhea.

Dr P Schwartz
403 No 1 Summerstrand
Port Elizabeth
6001
Tel: 27-41-33 1209
Fax: 27-41-33 1209

Coaches, trainers, doctors and first aiders at enduro-competitions must be made aware of these complaints so that they are able to assist athletes who present with the severe forms of GIT distress.

Gastrointestinal tract function at rest has been well studied, but little data are available on the effects of acute exercise and training on the GIT.[1] In particular, little information is available on the exact mechanisms associated with upper and lower GIT distress during prolonged exercise.

The aim of this article is to briefly review the risk factors and postulated mechanisms for GIT distress in endurance athletes. The clinical approach and management of common gastrointestinal symptoms in endurance athletes will also be discussed.

Risk factors for GIT symptoms in endurance athletes

In the last decade a number of surveys have been conducted to document the frequency of GIT symptoms during or after exercise in several different sports.[10,11,14-16] The true incidence of GIT symptoms (symptoms/athlete/time) has only recently been studied and the results are published in another article in the journal. The results from previous studies are mostly surveys of the frequency of symptoms occurrence in a population.

The frequency of occurrence of GIT symptoms in athletes depends on a number of factors. These include the site of origin of the symptoms (upper or lower GIT), type of symptom, gender, exercise intensity, type of sport, age and training.

Site of origin of the symptom

It has consistently been shown that symptoms of the lower gastrointestinal tract (abdominal cramps, urge to have a bowel movement and diarrhoea) are more frequently reported by athletes than upper gastrointestinal symptoms (dyspepsia, nausea and vomiting).[4,10,11,14] The frequency of lower gastrointestinal symptoms varies from 25% to 60%[4,11] and that of upper gastrointestinal symptoms from 6% to 58%.[4,10,16]

Type of symptom

In the upper GIT the most common symptoms are nausea and dyspepsia, while in the lower GIT the urge for a bowel movement and abdominal cramps are common.

Gender

The frequency of gastrointestinal symptoms appears to be more common in females (40%) than males (22%). Both upper and lower GIT symptoms are reported more frequently in females (31% – upper, 50% – lower) than males (15% – upper, 30% – lower).[10,14]

Exercise intensity

Symptoms appear to be more common during "hard" runs (high intensity) 24% than during "easy" runs (low intensity) 8%.[3,10]

Type of sport

A greater prevalence of GIT symptoms in triathletes has been reported in the running leg compared to the cycling or swimming leg.[15] It is suggested that mechanical jarring associated with running is associated with GIT symptoms. In sports such as cycling, skating or skiing, which are characterized by more gliding movement, a lower frequency of GIT symptoms is reported.[17] The difference in the incidence of GIT symptoms in endurance sports is well documented in another article in this journal.

Age

GIT symptoms during exercise are reported more frequently in younger than older athletes.[10,14]

Training

It has been observed that runners are more likely to develop symptoms after either an episode of particularly severe exercise to which they are not accustomed or during a period of rapid increased training mileage. Once the increased training has been maintained for a few weeks, symptoms appear to decrease.[3]

Mechanisms of GIT symptoms in endurance sports

The precise aetiology and pathology of gastrointestinal distress during endurance exercise is not clear. Several researchers have given attention to this topic but there are no conclusive findings.[18]

A number of mechanisms have been proposed in the literature to explain the aetiology and pathology of gastrointestinal distress in endurance exercise. A detailed discussion of all the mechanisms is beyond the scope of this article. A brief list of the postulated mechanisms for GIT distress in endurance athletes is as follows:

- A decrease in blood flow to the viscera during exercise which is related to exercise intensity
- Mechanical movement of the abdominal viscera during exercise
- Hormonal changes associated with exercise intensity affecting gastric motility
- Psychomotor stress associated with competition
- Other mechanisms such as diet, drugs, and infections

Clinical approach to GIT problems in endurance athletes

The endurance athlete achieves a level of competitive ability through a training and conditioning process which involves all the physical, mental and psychological factors which normally would function to protect the body against sustained endeavor at this level. It is therefore possible that health-related problems will be "unmasked" and manifest as symptoms which relate to physical effort and in particular the digestive system.

The clinician should therefore adhere to the time-honoured principles of a detailed history which includes a rigorous family history, birth and childhood details with particular reference to feeding problems, allergies and developmental milestones.

Particular attention should be paid to surgical history with reference to pelvic and abdominal surgery, especially in the female athletes, whereby severe functional symptoms may ultimately result under the conditions of endurance performance.

A medical history should include details about various vitamin, mineral and trace element supplements as self-medication and inadvertent "overdosing" may add to the "stress related symptoms". A detailed dietary history should include the "daily menu" for training periods as well as the pre-competition diet.

The general physical examination may frequently provide a useful clue to an underlying problem or disorder which has not become clinically manifest:

- A tinge of scleral discoloration may point to one of the congenital disorders of bilirubin metabolism so often associated with "effort fatigue syndrome" such as Gilbert's syndrome.
- Discoloration or coating of the tongue may point to bile reflux, chronic constipation or irritable bowel syndrome.
- Examination of the finger nails may demonstrate the classical changes of low grade iron deficiency of liver disease in the absence of other manifestations.

In sports where the body is relatively stable or in the horizontal position like cycling and swimming, gastrointestinal symptoms seem to be less frequent and troublesome than in sports which involve upright position and mechanical pounding (marathon running) according to the postulated mechanical mechanism.[15,17,25,27]

Education and enlightenment to this possible effect should be told to the athlete. For example, in the triathlon, the athlete would be more troubled with gastrointestinal symptoms during the running leg as compared to the cycling and swimming legs.[15]

Experienced athletes seem to have less symptoms than novice endurance athletes. A number of investigators have found a higher incidence of gastrointestinal symptoms in younger athletes compared to older athletes; that faster times due to increased intensity; and that "harder" runs compared to "easier" runs were associated with more symptoms. Also, females experience more gastrointestinal symptoms than males, of which lower gastrointestinal symptoms were more common and troublesome than upper gastrointestinal symptoms.[10]

Training over a period of time allows for gastrointestinal adaptation to take place. This is evident from studies demonstrating that physiological changes that occur after training are associated with decreased gastrointestinal symptoms during exercise, a smaller increase in sympathetic activity and a smaller decrease in parasympathetic activity which would affect gastric emptying, hormonal secretion and gastric motility.

Relative GIT ischaemia due to splanchic vasoconstriction during exercise has been a proposed mechanism.[19-22] However, when combined with dehydration, there seems to be an increase of gastrointestinal symptoms.[23] A combination of decreased blood flow, decreased blood volume, a rising core body temperature associated with decreased sweat response at high levels of dehydration could disrupt gastrointestinal secretions, absorptions and effect gastric emptying. The athlete should be advised on fluid requirements during exercise based on a number of factors. These include the type, intensity and duration of exercise. The amount of fluid (osmolality), the drinking pattern (being most important), temperature of fluid must be discussed with the athlete. Furthermore, the ambient temperature of the day will influence the volume and frequency of fluid intakes.

There is much confusion amongst the athletic population, coaches, trainers and physicians on the pre-competitive meal and the amount to drink and consume during an endurance event. In general it would be wise not to eat 3-5 hours prior to exercise. The composition of this meal should be devoid of protein and fat which could delay gastric emptying. From a practical point of view it should be realized that carbohydrate and water needs vary under different conditions. Humans performing mild exercise in the heat have a greater need for fluid replacement than for carbohydrate supplementation. On the other hand, an endurance athlete exercising in a cool environment may experience only moderate levels of dehydration, but could benefit significantly from carbohydrate feeding. For this reason, the composition of glucose-water solution administered during prolonged exercise should be determined on the basis of individual needs and potential benefits.

The rate of liver glycogen utilization to that of muscle glycogen utilization is influenced by exercise intensity. In brief, muscle glycogen utilization is increased at exercise intensity of 85% VO2 max or greater. Muscle glycogen depletion would occur within approximately 2 hours of exercise. An example of an intensity of 85% VO2 max is the standard marathon. In contrast, at exercise intensities of less than 70% VO2 max, such as the Comrades Marathon, liver glycogen is more likely to be depleted before muscle glycogen with the consequence development of hypoglycaemia. Present evidence suggests that optimum carbohydrate source to prevent hypoglycaemia during ultramarathon running would seem to be a 15% glucose polymer or starch solution.

As fluid requirements during exercise are about 500 ml/hr, the key in developing the optimum replacement fluid for exercise is to develop a solution that will provide maximal carbohydrate replacement at a gastric emptying rate of 500 ml/hr.

A 10-15% glucose polymer or starch solution is the greatest concentration that achieves this. When drunk during exercise a 10% polymer or starch solution would, at a gastric emptying rate of 500 ml/hr provide 50g of carbohydrate per hour, or about 35% of the total carbohydrate utilized at ultra-marathon record pace, or nearly all of the liver glycogen utilization.

Furthermore, if the fluid content is too hypertonic this could delay gastric emptying by causing nausea and upper abdominal distension due to water flux into the stomach. This distension could lead to an unwillingness to drink during an event, which could compound dehydration. The consumption of simple carbohydrates and sugars within several hours before exercise should be discouraged because of rebound hypoglycaemia due to insulin release from the pancreas.

Athletes are known to consume high fibre and roughage diets. Excessively high fibre diets may cause difficulties because of the resultant increase in stool volume and excessive fluid loads. These athletes should consult a dietician on the composition of their meals.

Athletes should attempt to defeacate at a relative set time daily by taking advantage of the morning gastrocolic reflex.

Athletes should stimulate peristalsis and defecation before an event in whatever fashion they find helpful, such as drinking coffee or tea, performing a light morning warm up or workout.

There is a high incidence of musculo-skeletal over-use and overload injuries in endurance athletes especially the lower limbs as in marathon runners. These athletes should avoid taking non-steroidal anti-inflammatory drugs (NSAID) and aspirin 12-24 hours before competing, as these drugs aggravate certain gastrointestinal symptoms of dyspepsia and gastrointestinal bleeding. The effects of certain drugs acting on the gastrointestinal tract during prolonged endurance exercise are unknown. The use of drugs should only be used as a last resort.

Athletes, coaches, and physicians must be made aware of the rules and regulations governing the use of medications according to those laid down by the International Olympic Committee. A number of simple drugs are allowable, but certain drugs that have been used for "runner's diarrhoea" eg. codeine phosphate, are banned.

Psychomotor stress prior to competition is a possible mechanism of increased gastric motility and hormonal secretions causing gastrointestinal distress. Athletes should undergo mental preparedness of the event prior to competition. Certain athletes might need psychological intervention and support to alleviate pre-competition stress. Referral to a sport psychologist is recommended.

For endurance athletes with chronic refractory problems lactose intolerance should be ruled out either through an elimination diet or a lactose tolerance test. Milk intolerance may cause specific problems, in particular "runner's trots" and irritable bowel syndrome in susceptible persons. The same findings have been described in persons unable to absorb gluten, a protein found in all grain foods except maize and rice. This condition, known as coeliac disease or gluten enteropathy, is completely cured by avoiding all gluten-containing foods. So the paradox is that the staple foods of life – bread and milk – may be just the foods that some endurance athletes would do best to avoid.

Management of common GIT symptoms in endurance athletes

The principles of management of the following common GIT symptoms in endurance athletes will be discussed: dyspepsia, nausea, vomiting, loss of appetite, abdominal cramps, urge for a bowel movement and/or diarrhoea, and gastrointestinal bleeding.

Dyspepsia

a. Recency of meals:
Recency of meals aggravate dyspepsia. The last pre-competition meal should be 3-4 hours before the event. It should consist of easily absorbable carbohydrates eg, fruit and fruit juices, cereals, pasta, bread. It should be low/devoid in protein and fat which could delay gastric emptying.

b. Dietary fibre:
Roughage and high fibre diets should be discouraged in those athletes with the symptoms of dyspepsia.

c. Antacid use:
Simple surface antacids before a competition or during rest periods in an event can be prescribed. However, large doses of antacids may cause cramping in cases. After having excluded organic gastrointestinal disease (special investigations and referral to a gastroenterologist) the empirical use of H2-Receptor antagonists the evening before an event, and 4 hours prior to the event can be considered. However, it must be stated that the effects of H2 blockers on exercise tolerance are not known.

d. Other pathology
It is very important to remember that chest pain due to ischaemic heart disease could be confused with dyspepsia secondary to diffuse oesophageal spasm. As many as 18-76% of patients with angina pectoris and normal coronary angiograms have oesophageal dysfunction, including abnormal gastro-oesophageal reflux and oesophageal motility disorders. Further investigations are necessary and include a progressive exercise treadmill test with electro-cardiac monitoring, barium swallow and follow through, endoscopy and oesophageal sphincter pressure studies.

Nausea and vomiting

a. Recency of meals:
Recency of meals can aggravate nausea and vomiting. A change in the eating time of the precompetitive meal to at least 3 to 4 hours prior to exercise is advisable.

b. Nature of diet:
The composition of food and fluid entering the stomach has to be considered. The correct balance between the amount, type, volume and temperature of food and fluid entering the stomach must be determined. It is advisable that athletes discuss their diet with a dietician or a sports physician.

c. Associated symptoms:
Associated symptoms such as unexplained nausea, vomiting and chest pain must be investigated further. Stress electrocardiography, oesophageal pH monitoring and pressure studies during exercise testing on a treadmill can differentiate those athletes or individuals who develop angina-like chest pain secondary to oesophageal spasm as opposed to the angina secondary to poor coronary perfusion of ischaemic heart disease.

d. Drugs:
Athletes must be advised to avoid taking aspirin and non-steroidal anti-inflammatory drugs as these can cause nausea and vomiting.

e. Pharmacological management:
Once organic disease has been excluded and all possible causes have been identified and treated, can the use of drugs are motility modifying agents (prokinetics), secretory modifying agents or centrally acting agents be considered (Table 1).

Loss of appetite

It is well known that athletes have a loss of appetite after severe prolonged exercise. It appears to be more common in younger athletes and female athletes. There is usually a positive relationship with the symptom and the intensity of the exercise. Furthermore, as gastrointestinal adaptation to endurance training occurs, the untrained athlete with the symptom can usually be reassured that, as training progresses, the symptoms will improve.

It has been shown that with intense physical activity carried out repeatedly every hour, the appetite of the person tested decreased sharply so that the next meal was postponed until late evening. However, athletes should be made aware that the desire to drink also decreases or can be suppressed as exhaustion sets in.

In summary, the sports physician should therefore educate the athlete to decrease exercise inten-

TABLE 1: Drugs acting on the gastrointestinal system

1. Surface active or locally acting preparations eg. antacids
2. Motility modifying agents
   - prokinetics (Maxolon, Motilium)
   - anti-diarrheals (Immodium, Lomotil)
3. Secretory modifying agents
   - specific receptor antagonists (Tagamet, Zantac)
   - anti-cholinergic agents (Buscopan, Avafortan)
4. Centrally acting agents
   - specific central action (Valoid, Stemetil)
   - dominant central and secondary peripheral activity (Codeine phosphate)

sity and duration until the adaptive gastrointestinal training effect has occurred.

Again it must be emphasized that organic disease must be excluded. Further investigation is advised if the symptoms do not decrease after altering exercise intensity.

Abdominal cramps

The causes of abdominal cramps during exercise are related to recency of meals, composition of food and exercise intensity. Consequently, faster runners and novice runners experience more abdominal cramps than the more experienced older athletes. The basis of the abdominal cramps can be related to the pharmacological concentrations of gastrointestinal hormones.[28]

Athletes can be advised either exercise only 3-4 hours after meals, or to have a bowel action prior to exercise. If this is not successful and provided organic disease has been excluded, the use of medication can be considered. The more commonly used drugs would be the motility modifying agents or anticholinergic agents (Table 1).

Urge for a bowel movement and/or diarrhoea

These two symptoms represent the most common problem of gastrointestinal distress in endurance athletes.[3,4,10,11,14-16] Many athletes suffer from "Traveller's diarrhoea". The aetiology is one of viral or bacterial infective origin. This is not the same as "runner's diarrhoea" which should be treated with hydration and medication.

The clinical characteristics of "Runner's diarrhoea" (RD) are:[16]

- passage of semi-formed or watery stools
- can be associated with abdominal pain below umbilicus
- rectal urgency
- multiple stools
- large volume stools
- abdominal pain and rectal urgency relieved by defecation
- diarrheal cramps after completion of exercise
- faecal incontinence with soilage during exercise
- "irritable bowel syndrome" when not exercising

Advice to athletes with "runner's diarrhoea" would include the following:

- to eat a pre-competition meal 4 hours prior to exercise
- to exercise in a fasted state
- to decrease fibre in diet
- practice relaxation therapy to reduce stress
- encourage a bowel movement prior to an athletic event by drinking coffee or tea
- gentle warm up prior to exercise
- encourage the gastro-colic evacuation reflex when first getting up in the morning.

Before resorting to drug therapy, it would be wise to exclude lactose intolerance and to exclude any other bowel pathology such as Crohn's Disease, ulcerative colitis, diverticular disease or colonic cancer. The appropriate investigations (blood, urine, stool, x rays, colonoscopy, biopsy and histology) should be done by a gastroenterologist who preferably has an interest in Sports Medicine.

The most common medication that can be used to treat this symptom are the motility modifying agents and secretory modifying agents (Table 1).

Gastrointestinal bleeding

Numerous anecdotal reports indicate that the passage of bloody stools (haematochezia) occurs occasionally.[12] There is a possible association between GIT bleeding and the development of "runner's anaemia".

Athletes should be evaluated with screening methods such as the Haemoccult cards. Approximately six cards should be sufficient, using two for light training and the normal daily regime, two for periods of heavy training, and two for post-race testing.

If the bleeding occurs only after completion in a person under 35 years of age, no further evaluation is necessary unless the bleeding is massive, causes persistent anaemia or other symptoms, or the athlete requests further evaluation. If the bleeding occurs chronically, even during light training then a thorough evaluation is necessary for athletes in all age groups.

Those older than 35 to 40 years should probably have roentgenologic and/or endoscopic evaluation if their haemoccults are positive during heavy training, even if they do not bleed when they are less active. A rectal examination and flexible sigmoidoscopy or colonoscopy should be performed if they have not been done within the previous year in athletes over the age of 40. Reports of small amounts of bright red blood with a negative haemoccult and a positive rectal examination for haemorrhoids might obviate the need for intensive further investigation in borderline cases.

Other specific gastrointestinal diseases

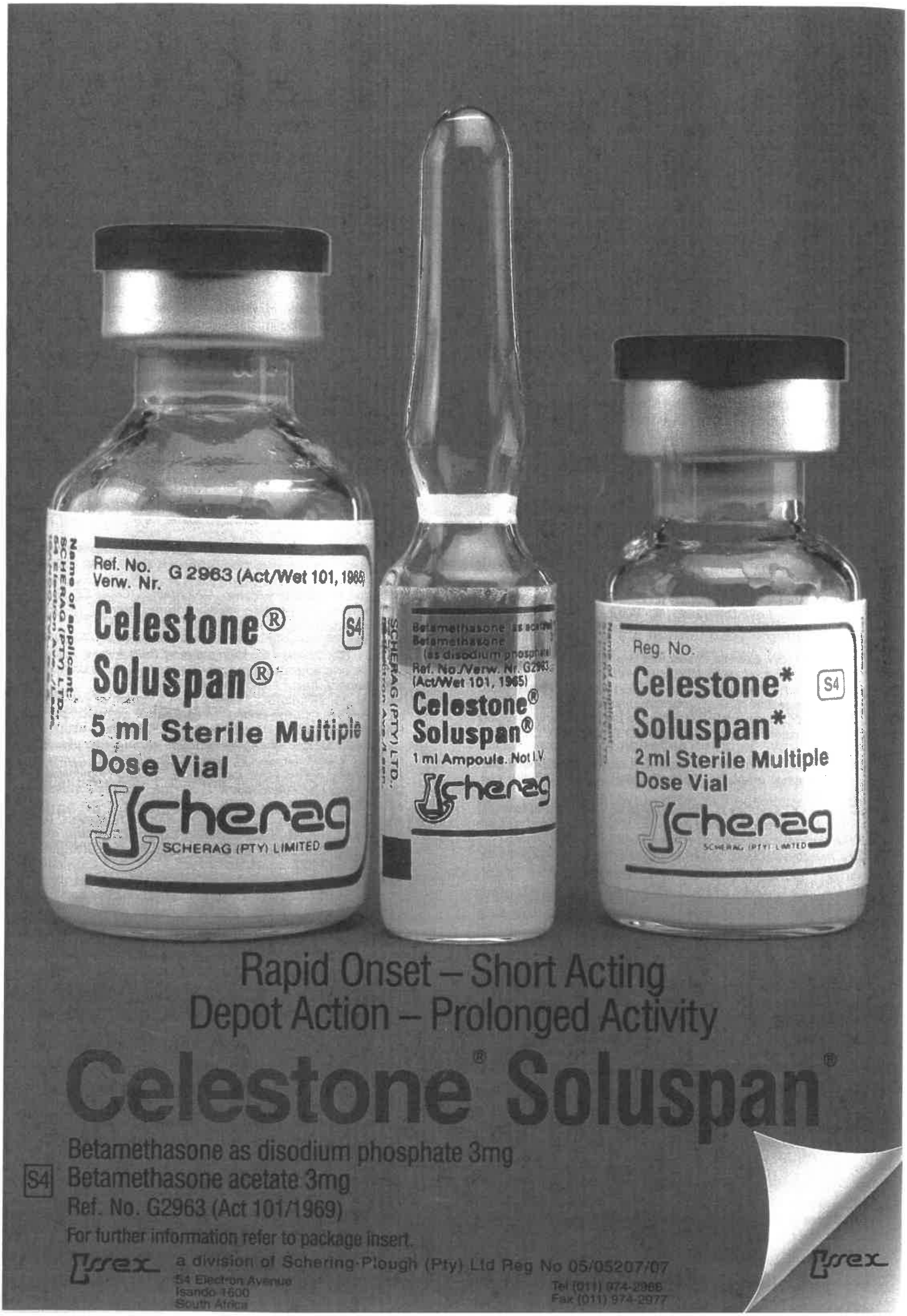
There is evidence that patients with duodenal ulcers who undertake mild exercise have an increased secretion of acid and an increased volume of stomach fluid after exercise which would make these athletes more susceptible to progressive ulcer pathology. The possible mechanism is an increased autonomic nervous system response. This raises the question of whether strenuous exercise in patients with active duodenal ulcer should be discouraged.

REFERENCES

1. Browns F, Saris WHM, Rehrer NJ: Abdominal complaints and gastrointestinal function during long-lasting exercise. Int J Sports Med 1987; 8: 175-189.
2. Astrand P, Rodahl K: Textbook of Work Physiology. Physiological Bases of Exercise (3rd Ed), New York City, McGraw-Hill Book Co 1986.
3. Forgoros RN: "Runners Trots". Gastrointestinal disturbances in runners. JAMA 1980; 243 (17): 1743-1744.
4. Sullivan SN: The gastrointestinal symptoms of running. N Engl J Med 1981; 304 (15): 915.
5. Broussais FJV: Treatise on physiology applied to pathology. #rd Amer Ed., Philadelphia, 1832: 638.
6. Cordain L, Latin RW, Behnke JJ: The effects of an aerobic running program on bowel transit time. J Sports Med 1986; 26: 101-104.
7. Cannack J, Read NW, Cann PA, Greenwood B, Holgate AM: Effect of prolonged exercise on the passage of a solid meal through the stomach and small intestine. GUT 1982; 23: 957-961.
8. Burkitt D, Walker AP, Dainter NS: Dietary fiber and disease. J Am Med Assoc 1974; 229: 1068-1072.
9. Vena JE, Graham S, Zielezny M et al: Lifetime occupational exercise and colon cancer. Am J Epidemiol 1988; 22 (2): 71-74.
10. Riddoch C, Trinick R: Gastrointestinal disturbances in marathon runners. Br J Sports Med 1988; 98: 644-646.
11. Worobetz LJ, Gerrard DF: Gastrointestinal symptoms during exercise in enduro-athletes: Prevalence and speculations on the aetiology. NZ Med J 1985; 98: 644-646.
12. Runner's World, May 1969; 72:
13. Thompson PD, Funk EJ, Carleton RA et al: Incidence of death during jogging in Rhode Island from 1975 through 1980. JAMA 1982; 274: 2535-2536.
14. Keeffe EB, Low DK, Goss JR, Wayne R: Gastrointestinal symptoms of marathon runners. West J Med 1984; 141 (4): 481-484.
15. Sullivan SN: Exercise-associated symptoms in triathletes. Phys Sportsmed 1987; 15 (9): 101-110.
16. Prieve WM, Priebe J: Abstracts. Runner's Diarrhoea (RD). Prevalence and clinical symptomatology, 1984.
17. Rehrer NJ: Biomechanical vibration of the abdominal region during running and bicycling. PhD Thesis 1990; Chapter 4: 77-84.
18. Larson DC, Fisher R: Management of exercise-induced gastrointestinal problems. Phys Sportsmed 1987; 15 (9): 112-126.
19. Wade OL, Combes B, Chilos AW et al: The effect of exercise on the splanchnic blood flow and splanchnic blood volume in normal men. Clin Sci 1956; 15: 457-463.
20. Clausen JP: Effect of physical training on cardiovascular adjustments to exercise in man. Physiol Rev 1977; 57 (4): 779-815.
21. Jager K, Bollinger A, Valli C, Amman R: Measurement of mesenteric blood flow by duplex-scanning. J Vasc Surg 1986; 57 (4): 462-469.
22. Heer M, Repond F, Harry A, Sulser H, Kehl D, Jager K: Acute ischaemic colitis in female long distance runner. GIT 1987; 28: 896-899.
23. Rehrer NJ: Fluid intake and gastrointestinal problems in runners competing in a 25 km race and a marathon. 1990; Chapter 8: 153-163.
24. Rehrer NJ, Beckers RJ, Browns F, Ten Hoor F, Saris WHM: Effects of dehydration on gastric emptying and gastrointestinal distress while running. Med Sci Sports Exerc 1990; 22: (in press).
25. Porter AMW: Case report. Marathon running and caecal slap syndrome. Brit J Sports Med 1982; 16: 178.
26. Dawson DJ, Khan AW, Shreve DR: Psoas muscle hypertrophy: Mechanical causes for "joggers trots"? Letter: Brit Med J 1985; 291: 787-788.
27. Blacklock NJ: Bladder trauma in the long distance runner: "10 000 meters haematuria". Brit J Urol 1977; 49: 129-132.
28. Sullivan SN, Champion MC, Christofides ND, Adrian TE, Bloom SR: The gastrointestinal regulatory peptide responses in long-distance runners. Phys Sportsmed 1984; 12 (7): 77-82.

(Continued from page 9)

16. Nose, H., Mack, G.W., Shi, X., and Nadel, E.R. Role of osmolality and plasma volume during rehydration in humans. J Appl Physiol 1988; 65: 325-331.
17. Gisolfi CV, Spranger KJ, Summers RW, Schedl HP, Bleiler TL. Effects of cycle exercise on intestinal absorption in humans. J Appl Physiol 1991; 71: 2518-2527.
18. Gisolfi CV, Summers RW, Schedl HP, Bleiler TL. Intestinal water absorption from select carbohydrate solutions in humans. J Appl Physiol 1992; 73: 2142-2150.
19. Walsh RM, Noakes TD, Hawley JA, Dennis SC. Impaired high-intensity cycling performance time at low levels of dehydration. Int J Sports Med 1994; 15: 392-398.
20. Coyle EF, Hamilton M. Fluid replacement during exercise: effects on Physiological homeostasis and performance. In: Gisolfi CV, Lamb DR, eds, Perspectives in Exercise Science and Sports Medicine, Volume 3. Fluid Homeostasis During Exercise. Benchmark Press Inc, Carmel, Indiana, 1990 pp. 281-308.
21. Brandenberger G, Candas VM, Follenius M, Kahn KM. The influence of the initial state of hydration on endocrine responses to exercise in the heat. Eur J Appl Physiol 1989; 58: 674-679.
22. Montain SJ, Coyle EF. Fluid ingestion during exercise increases skin blood flow independent of increases in blood volume. J Appl Physiol 1992; 73: 903-910.
23. Barr SI, Costill DL, Fink WJ. Fluid replacement during prolonged exercise: effects of water, saline, or no fluid. Med Sci Sports Exerc 1991; 23: 811-817.
24. Gisolfi CV, Copping JR. Thermal effects of prolonged treadmill exercise in the heat, Med Sci Sports Exerc 1974; 6: 108-113.
25. Hamilton M, Gonzalez-Alonso J, Montain S, Coyle EF. Fluid replacement and glucose infusion during exercise prevent cardiovascular drift. J. Appl Physiol 1991; 71: 871-877.
26. Robinson TA, Hawley JA, Palmer GS, Wilson GR, Gray DA, Noakes TD, Dennis SC. High rates of fluid intake do not improve one hour cycling performance in a thermoneutral environment. Eur J Appl Physiol 1994; in press.
27. Hawley JA, Dennis SC, Noakes TD. Oxidation of carbohydrate ingested during prolonged endurance exercise. Sports Med 1992; 14: 27-42.
28. Costill DL. Gastric emptying of fluids during exercise. In: Gisolfi CV and Lamb DR, eds, Perspectives in Exercise Science and Sports Medicine, Volume 3. Fluid Homeostasis During Exercise. Benchmark Press Inc., Carmel, Indiana 1990: pp. 97-127.
29. Hawley JA, Dennis SC, Nowitz A, Brouns F, Noakes TD. Exogenous carbohydrate oxidation from maltose and glucose ingested during prolonged exercise. Eur J Appl Physiol 1992; 64: 523-527.
30. Massicotte D, Peronnet F, Allah C, Hillaire-Marcel C, Ledoux M, Brisson G. Metabolic response to [13C] glucose and [^{13}c] fructose ingestion during exercise. J Appl Physiol 1986; 61: 1180-1184.
31. Massicotte D, Peronnet F, Brisson G,Bakkouch K, Marcel CH. Oxidation of glucose polymer during exercise: comparison with glucose or fructose. J Appl Physiol 1989; 66: 179-183.
32. Hawley JA, Bosch AN, Weltan SM, Dennis SC, Noakes TD. Effects of glucose ingestion or glucose infusion on fuel substrate kinetics during prolonged exercise. Eur J Appl Physiol 1994; 68: 381-389.
33. Hawley JA, Bosch AN, Weltan SM, Dennis SC, Noakes TD. Glucose kinetics during prolonged exercise when hyperglycaemic, Pflugers Archives 1994; 426: 378-386.
34. Decombaz J, Arnaud MJ, Milon H, Moesch H, Philippossian G, Thelin AL and Howard H. Energy metabolism of medium chain triglycerides versus carbohydrate during exercise. Eur J Appl Physiol 1983, 52: 9-14.
35. Van Zyl C, Lambert EV, Noakes TD, Dennis SC. Effects of medium-chain triglyceride ingestion on carbohydrate metabolism and cycling performance. In Conference Proceedings of the Ninth International Biochemistry of Exercise Meeting 1994: pp.30.


Ref. No. Verw. Nr. G 2963 (Act/Wet 101, 1965)
Celestone® Soluspan®
S4
5 ml Sterile Multiple Dose Vial
Scherag
SCHERAG (PTY) LIMITED
Ref. No./Verw. Nr. G2963 (Act/Wet 101, 1965)
Celestone® Soluspan®
1 ml Ampoule. Not I.V.
Scherag
Reg. No.
Celestone* Soluspan*
S4
2 ml Sterile Multiple Dose Vial
Scherag
Rapid Onset – Short Acting
Depot Action – Prolonged Activity
Celestone® Soluspan®
Betamethasone as disodium phosphate 3mg
S4
Betamethasone acetate 3mg
Ref. No. G2963 (Act 101/1969)
For further information refer to package insert.
Essex a division of Schering-Plough (Pty) Ltd Reg No 05/05207/07
54 Electron Avenue
Isando 1600
South Africa
Tel (011) 974-2966
Fax (011) 974-2977
Essex

The incidence and risk factors of gastrointestinal symptoms in six endurance sports

Peter A Schwartz, MBBCh, BSc (Med) (Hons) Sports Medicine*
Martin P Schwellnus, MBBCh, MSc (Med) Sports Science, MD, FACSM*
Albertus S Koorts, MSc, PhD #

Abstract

Gastrointestinal (GIT) symptoms appear to be very common in endurance athletes. Previous studies have only documented frequency rates of GIT symptoms during sports events, and usually only in a single sport. Differences in methodology between studies make comparisons on the incidence and risk factors of GIT symptoms in different sports difficult. The aims of this study were to document the true incidence (symptoms/1 000 hours of participation) and risk factors of gastrointestinal symptoms in six endurance sports. Data on the incidence and risk factors pertaining to six common GIT symptoms were obtained on 1 158 athletes in six endurance sports: (canoeing (n = 205), aerobic dancing (n = 109), running (n = 321), triathlons (n = 68), cycling (n = 226) and swimming (n = 224). The significant (p < 0.05) findings of this study were that there was a higher incidence of lower GIT symptoms than upper GIT symptoms; younger athletes have a higher incidence of symptoms; female athletes have a higher incidence than male athletes; that the urge for a bowel movement was the most common lower GIT symptom; that nausea was the most common upper GIT symptom; and that symptoms were more likely to occur during competition than in training. Furthermore, experienced athletes had a lower incidence of symptoms than novice athletes, and that GIT symptoms were more common in vertical sports (running) compared to gliding sports (cycling and swimming).

Introduction

During the last ten years there has been an upsurge of interest in the physiology of endurance sports such as aerobic dance workouts, canoeing, running, the triathlon, cycling and swimming.[1] The metabolic, renal, cardiovascular and pulmonary responses to endurance exercise have been well researched.[2] However, alterations of gastrointestinal (GIT) physiology during prolonged endurance exercise, in particular the incidence and aetiology of GIT distress has largely been ignored.

There are several anecdotal reports in the lay press and the medical literature of endurance athletes who experience GIT symptoms during exercise. The severity of these symptoms can range from mild (not affecting training or performance) to severe (limiting training or performance). Common complaints of the upper GIT include heartburn, nausea, vomiting, loss of appetite and flatulence. The common lower GIT complaints are abdominal cramps, the urge for a bowel movement and diarrhoea.[28-30] Recently, GIT bleeding after exercise has been the focus of attention. Severe rectal bleeding (haematochezia) during exercise was first reported in the Runner's World Magazine in 1969.[31] In 1982 the first case of documented death due to massive haemorrhagic gastritis was reported in a 28 year old runner.[32]

These observations have prompted researchers to study the frequency of gastrointestinal symptoms during or after exercise in several different sports.[29,30,33,35] The studies that have been conducted to date have a number of limitations including poor sample selection and documentation of the frequency of symptoms rather than a true incidence. Furthermore, differences in the methodology between studies makes comparisons on the risk of developing GIT symptoms between groups and different sports difficult. Nevertheless, data obtained from these surveys appear to have identified a number of trends.

It appears that the frequency of occurrence of GIT symptoms in athletes differs and depends on factors such as the site of origin of the symptoms (upper or lower GIT), the type of symptom, the gender of the athlete, exercise intensity, type of sport, and training.[1,28-30] In most surveys, it has been shown that symptoms of the lower GIT are more common (25% to 60% of respondents) than upper GIT symptoms (6% to 58% of respondents). The most common upper GIT symptom appears to be nausea (16%) followed by heartburn (13%). In the lower GIT, the most common symptom appears to be the urge for a bowel movement (36%) followed by abdominal cramps (30%).[29] The frequency of symptoms appears to be more common

Address for correspondence:
Dr M P Schwellnus
Sports Medicine
MRC/UCT Bioenergetics of Exercise Research Unit
Department of Physiology
University of Cape Town Medical School
Observatory, 7925
*South Africa**
Telephone: 27-21 406 6504
Facsimile: 27-21-47 7669
Department of Mathematical Statistics,
University of Port Elizabeth, South Africa #

in female (40%) compared to male (22%) athletes.[29,33] Symptoms appear to be more frequent during "hard" runs (higher intensity) (24%) compared to "easy" runs (lower intensity) (8%).[27,29] In one study in triathletes, a greater frequency of GIT symptoms has been reported in the running leg compared to the cycling or swimming leg.[34] Data from these surveys also appear to indicate that in sports such as cycling, skating or skiing, which are characterized by gliding movements, a lower frequency of GIT symptoms is reported.[11]

Gastrointestinal symptoms during exercise have also been reported more frequently in younger than older athletes. The reason for this has been attributed to a higher intensity of exercise in younger people. It has also been reported that symptoms of GIT distress decrease with training. In other words, GIT "adaptation" might take place with training in endurance athletes.[29,33] It has been observed that runners are more likely to develop symptoms after either an episode of high intensity exercise to which they are not accustomed or during a period of rapid increased training volume. Once the increased training has been maintained for a few weeks, symptoms appear to decrease.[27]

As already mentioned, the data from different surveys reported in the literature have to be interpreted with caution. Comparisons between the occurrence of GIT symptoms in different sports, genders or age groups can only be made if the true incidence of symptoms is recorded in the groups. Accordingly, the main aim of this present study was to document the incidence (symptoms/athlete/1 000 hours of physical activity) of GIT symptoms in endurance athletes in six different endurance sports in South Africa. The study is novel in that the true incidence of GIT symptoms in six endurance sports (aerobic dancing, canoeing, road running, triathlons, cycling and swimming) have been surveyed at the same time.

Methods

Subjects

A survey by self-completed questionnaire was conducted on 2 534 athletes from the six endurance sports. Athletes from each sport were randomly selected from those that entered for a major event of that particular sport in the period 1990-1991 in South Africa. At the event, a questionnaire was handed to a randomly selected group of athletes in each sport. The athletes were asked to complete the questionnaire and return it in a self-addressed envelope to the principal investigator for analysis. Incentive schemes, in the form of prizes, were used to improve the response rate. The overall response rate to the questionnaire was 46% (n = 1 158).

The response rate for each individual sport and the event during which subjects were identified is indicated in Table 1. Of the respondents, 884 (76,3%) were males, 268 (23,1%) females and 6 (0,6%) did not give their sex. Of the 1 157 athletes only one athlete did not indicate the sport he/she was competing in. Incorrectly completed questionnaires were considered as non-responders and therefore omitted from the analysis.

TABLE 1: The response rate and events for each of the endurance sports

Sport	Event	Rate(%)
Aerobics (n = 208)	Dance classes	52
Canoeists (n = 418)	84 km race	50
Runners (n = 554)	42 km race	58
Triathletes (n = 105)	Std Triathlon	60
Cyclists (n = 750)	105 km tour	30
Swimmers (n = 501)	1 mile race	45

Questionnaire

A questionnaire was compiled in two of the official languages of South Africa (English and Afrikaans). Each endurance athlete chose the language of his/her own choice. The questionnaire was divided into four subsections. These included an introduction letter, personal characteristics of the enduro-athlete, questions relating to a number of possible abdominal complaints whilst training or competing in their particular sport, and a detailed analysis of the athlete's training profile. In addition, the questionnaire included questions about previous abdominal surgery or any major medical abdominal complaint that the athlete may have had, as well as information on the use of medication to control GIT symptoms during exercise. In the questionnaire, the term "surgery" referred to previous abdominal surgery (gynecological, abdominal or urological) and a "major medical abdominal complaint" referred to a previous major medical illness (peptic ulcer disease, ulcerative colitis or Crohn's Disease).

Specific information was obtained on possible symptoms relating to either the upper gastrointestinal tract (heartburn, nausea, vomiting or loss of appetite) or the lower gastrointestinal tract (abdominal cramps, urge to defecate or diarrhoea). Two questions relating to the urinary system (urge to pass urine and darkened urine) were also included in the questionnaire. The reason for this was that some athletes are under the impression that urinary symptoms are part of the abdominal system.

In athletes who complained of a specific abdominal symptom the frequency of the symptoms in the last ten successive training/competitive sessions was obtained.

Statistical analysis

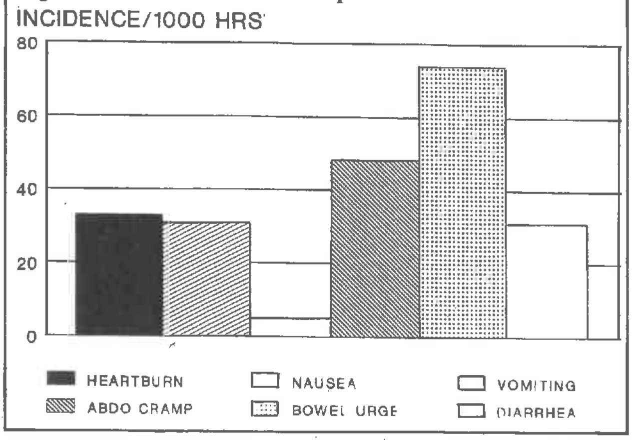
All the statistical analysis in this study were conducted at the Department of Statistics of the University of Port Elizabeth using the statistical package BMDP (1990 PC-version). The Pearson Chi Square test was used to analyze for differences in the frequency of occurrence of GIT symptoms between groups (sport, gender, age) and between different GIT symptoms within groups. The level of statistical significance was established at $p < 0.05$.

Results

Incidence of GIT symptoms in all endurance sports

The incidence of upper and lower GIT symptoms in the athletes from all six endurance sports is de-

Figure 1: **All Endurance Sports.**



picted in Figure 1. The GIT symptom with the highest incidence (occurrence/1 000 participant hours) was the urge to defecate (74), followed by abdominal cramping (48) and heartburn (32). There was a significantly higher incidence of lower GIT symptoms (abdominal cramps, urge for bowel movement and diarrhoea) compared to upper GIT symptoms (heartburn, nausea and vomiting) for the total population (Figure 1).

Incidence of GIT symptoms in individual endurance sports

Aerobic dancing

The incidence of GIT symptoms in aerobic dancers (all, males and females) is depicted in Figure 2. The GIT symptom in all aerobic dancers with the highest incidence (occurrence/1 000 participant hours) was abdominal cramps (65), followed by nausea (60), and the urge to defecate (38). In general, GIT symptoms were more common in female compared to male aerobic dancers, and lower GIT symptoms were more common than upper GIT symptoms.

Canoeing

The incidence of GIT symptoms in canoeists (all, males and females) is depicted in Figure 3. The GIT symptom in all canoeists with the highest incidence (occurrence/1 000 participant hours) was the urge to defecate (114), followed by heartburn (76), diarrhoea (65) and abdominal cramps (61). In general, GIT symptoms were more common in female compared to male canoeists, and with the exception of heartburn, lower GIT symptoms were more common than upper GIT symptoms.

Road runners

The incidence of GIT symptoms in road runners (all, males and females) is depicted in Figure 4. The GIT symptom in all road runners with the highest incidence (occurrence/1 000 participant hours) was the urge to defecate (120), followed by abdominal cramps (55), diarrhoea (42), and heartburn (31). Again, GIT symptoms were more common in female compared to male road runners, and lower GIT symptoms were much more common than upper GIT symptoms.

Triathletes

The incidence of GIT symptoms in triathletes (all, males and females) is depicted in Figure 5. The GIT symptom in all triathletes with the highest inci-

Figure 2: **Aerobics.**

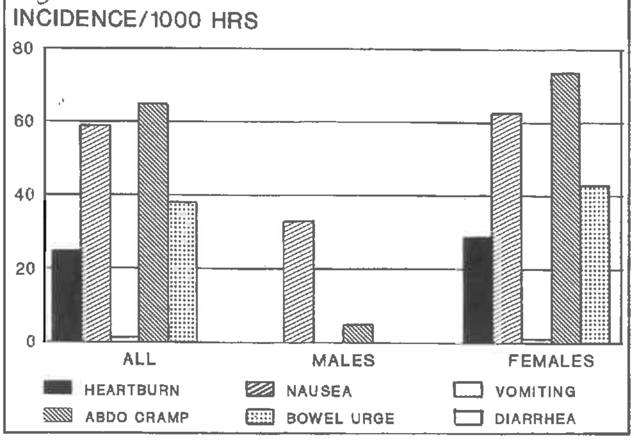


Figure 3: **Canoeists.**

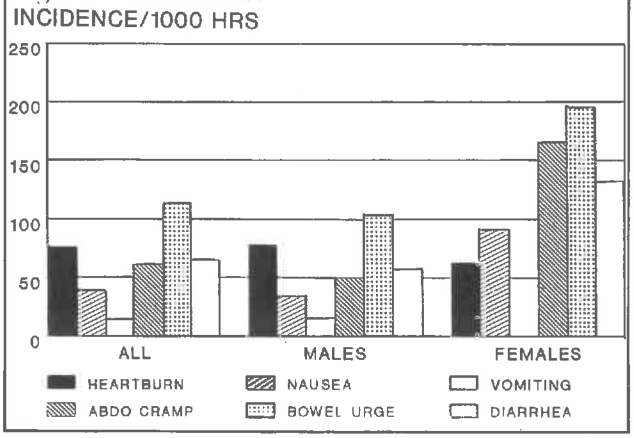


Figure 4: **Runners.**

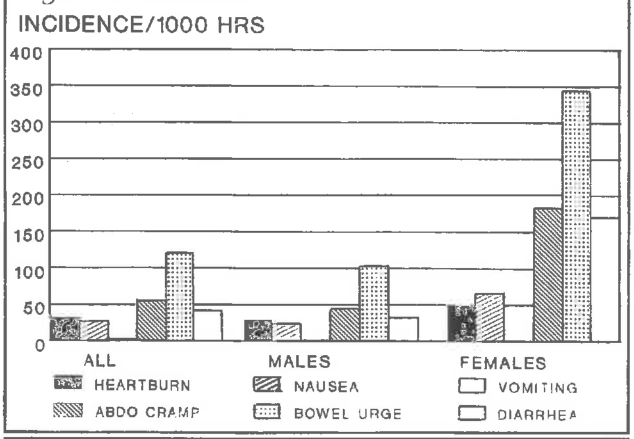
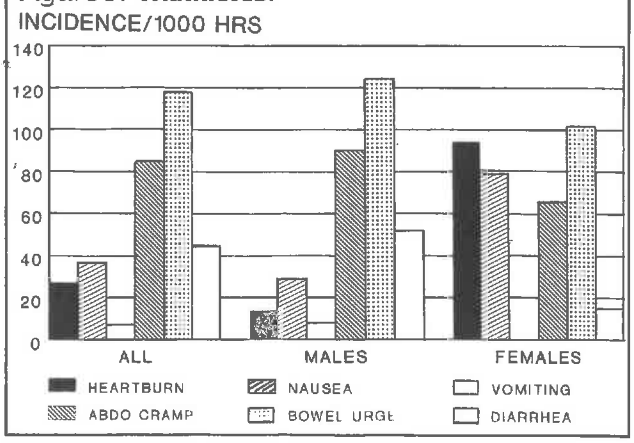


Figure 5: **Triathletes.**



dence (occurrence/1 000 participant hours) was the urge to defecate (118), followed by abdominal cramps (85), diarrhoea (45), and nausea (37). Upper GIT symptoms were more common in female compared to male triathletes, whereas lower GIT symptoms were more common in male triathletes.

Cyclists
The incidence of GIT symptoms in cyclists (all, males and females) is depicted in Figure 6. In general, GIT symptoms were less common in cycling than in other sports. The GIT symptom in all cyclists with the highest incidence (occurrence/1 000 participant hours) was abdominal cramping (31), followed by the urge to defecate (29), nausea (24) and heartburn (21). Female cyclists tended to have GIT symptoms more frequently than male cyclists. There appears to be no difference in the incidence of upper and lower GIT symptoms in cyclists.

Swimming
The incidence of GIT symptoms in swimmers (all, males and females) is depicted in Figure 7. In general, GIT symptoms were uncommon in swimmers. The GIT symptom in all swimmers with the highest incidence (occurrence/1 000 participant hours) was abdominal cramping (23), followed by nausea (22), the urge to defecate (18), and heartburn (12). Except for nausea, female and male swimmers appear to have the similar incidences of GIT symptoms. As for cyclists, there appears to be no difference in the incidence of upper and lower GIT symptoms in swimmers.

Risk of GIT symptoms in endurance sports
Table 2 summarizes the risk of developing GIT symptoms in the six endurance sports that were studied. Sports associated with a high risk (incidence of GIT symptoms > 100 occurrences/1 000 participant hours) are canoeing, road running and triathlons. In all these sports the symptom associated with this high risk is the urge to defecate. Abdominal cramps occur with a medium risk (incidence of GIT symptoms of 50-100 occurrences/ 1 000 participant hours) in aerobic dances, canoeing, road running and triathlons. Heartburn and diarrhoea occur with a medium risk in canoeists. All the other sports have a relatively low risk (GIT symptom risk of < 50 occurrences/1 000 participant hours) of developing GIT symptoms in endurance sports.

Incidence of GIT symptoms in different age groups
The incidence (occurrence/1 000 participant hours) of upper GIT symptoms in males and females of different age groups in all the sports that were surveyed is depicted in Figure 8.

There was a significantly lower incidence in upper GIT symptoms in older compared to younger age groups for both males and females (p = 0.0463). The incidence (occurrence/1 000 participant hours) of lower GIT symptoms in males and females of different age groups is depicted in

Figure 6: **Cyclists.**

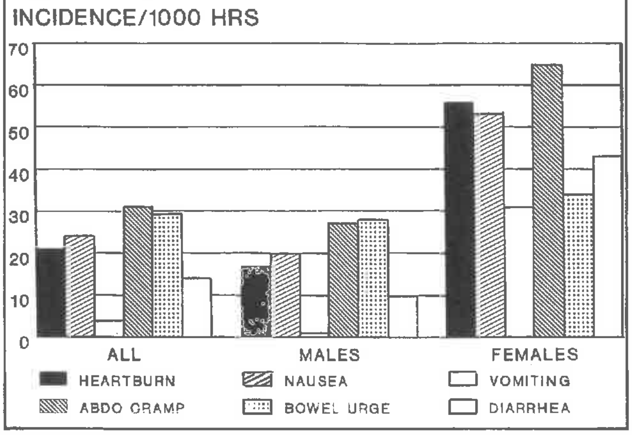


Figure 7: **Swimmers.**

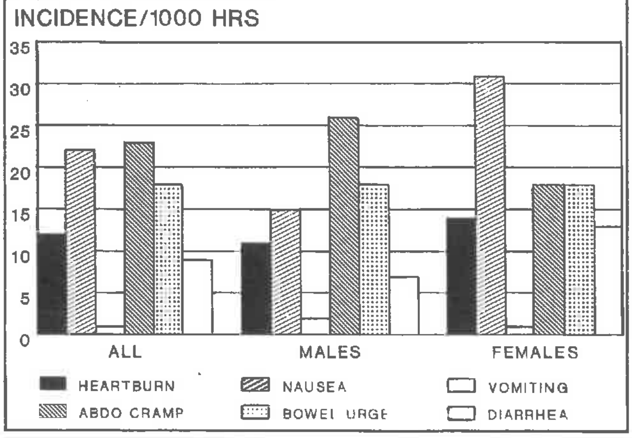


Figure 8: **Upper GIT Symptoms.**

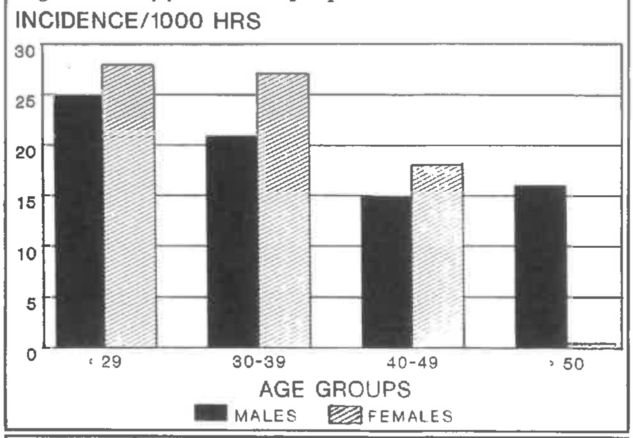
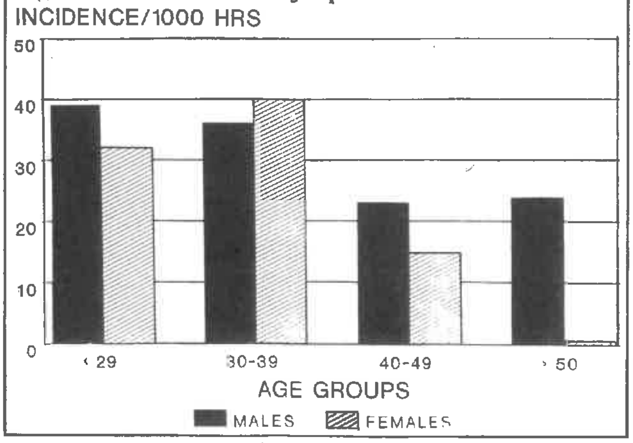


Figure 9: **Lower GIT Symptoms.**



- For muscular pain
- Effective pain relief
- Site specific
- Non-staining
- Non-greasy

Reparil®-Gel

Relieves muscular pain.

MADAUS PHARMACEUTICALS (PTY) LTD
Reg. No.: 77/00174/07 2nd Road (cnr 16th Road), Randjespark, Midrand 1685

S1 Reparil®-Gel 100 g contains: Aescin 1,0 g; Diethylamine salicylate 5,0 g. Reg. No. G/13.9/2367

Figure 9. There was a significantly lower incidence in lower GIT symptoms in older compared to younger age groups for both males and females (p = 0.0004).

Incidence of GIT symptoms in male and female endurance athletes

For the total population the incidence of lower gastrointestinal symptoms in females was significantly higher than in males (Figures 2-7). For each of the sports a similar trend was found in that females had a higher incidence of lower gastrointestinal symptoms than males (Figures 2-7).

For the upper gastrointestinal tract, females had a higher incidence of heartburn and nausea in each sport. However, in most sports, with the exception of cycling, males had a higher incidence of vomiting than females.

TABLE 2: Risk of GIT symptoms in endurance sports

Risk*	Sport	Symptoms
High (> 100)	Canoeing	Urge to defecate
	Road running	Urge to defecate
	Triathletes	Urge to defecate
Medium (50-100)	Aerobic dance	Abdominal cramps, nausea
	Canoeing	Heartburn, diarrhoea, abdominal cramps
	Road running	Abdominal cramps
	Triathletes	Abdominal cramps
Low (< 50)	Aerobic dance	Urge to defecate
	Road running	Diarrhoea, Heartburn
	Triathletes	Diarrhoea, nausea
	Cycling	Abdominal cramps, Urge to defecate, nausea
	Swimming	Abdominal cramps, nausea, Urge to defecate

*: High risk (Risk of symptom > 100 occurrences/1 000 participant hours)
Medium risk (Risk of symptoms between 50-100 occurrences/1 000 participant hours)
Low risk (Risk of symptoms < 50 occurrences/1 000 participant hours)

Incidence of GIT symptoms in athletes with previous abdominal surgery or previous major medical abdominal complaint

The prevalence of previous abdominal surgery or a previous major medical abdominal complaint in the entire population was 14%. There was a significant association between previous abdominal surgery and the development of an upper GIT symptoms (p = 0.0471) but not lower GIT symptoms (p = 0.855) during endurance exercise for the whole population.

The use of medication by endurance athletes to control GIT symptoms

In the total population of endurance athletes, 17% indicated that they use medication to control GIT symptoms during training. The symptoms for which medication was used by the total population of athletes during training and competitions is indicated in Table 3. The most common symptom for which GIT medication was used by endurance athletes during training was heartburn (33% of users), followed by abdominal cramps (20% of users), the urge to defecate (18% of users), and diarrhoea (16% of users). During competition, the most common symptom for which GIT medication was used was the urge to defecate (36% of users), followed by diarrhoea (28% of users), and abdominal cramps (14% of users).

TABLE 3: The frequency of use (% of users) of GIT medication for different symptoms in the total population of endurance athletes

Symptom	Training	Competition
Heartburn	33%	10%
Nausea	7%	6%
Vomiting	1%	3%
Loss of appetite	5%	3%
Abdominal cramps	20%	14%
Urge to defecate	18%	36%
Diarrhoea	16%	28%

The frequency of GIT medication use by endurance athletes during competition in each of the six sports that were surveyed is indicated in Table 4. The use of medication (as a % of users in each sport) to control heartburn was common in canoeists (29%), road runners (27%), and cyclists (29%). For other upper GIT symptoms, canoeists commonly used medication to control nausea (20%) and vomiting (33%). GIT medication was commonly used to control the urge to defecate in canoeists (39%), road runners (33%), and triathletes (24%). The use of medication to control diarrhoea was common in canoeists (61%), cyclists (40%), road runners (35%), and triathletes (19%).

The types of GIT medication that were used by athletes are depicted in Table 5. Anti-diarrheals followed by antacids were the most common medications used by endurance athletes in this study.

TABLE 4: The frequency (% of users) of medication use by endurance athletes to control GIT symptoms during competition in six different sports

	Heartburn	Nausea	Vomiting	Abdominal cramps	Urge to defecate	Diarrhoea
Aerobic dancing	11%	5%	–	10%	8%	–
Canoeing	29%	20%	33%	12%	39%	61%
Road running	27%	5%	7%	21%	33%	35%
Triathlon	9%	6%	12%	4%	24%	19%
Cycling	20%	–	–	7%	12%	40%
Swimming	7%	–	–	–	–	–

TABLE 5: The types of GIT medication used by endurance athletes (expressed as a % of the athletes using medication)	
Medication	**% of use by athletes**
Loperomide HCl	36
Diphenoxylate HCl	10
Rennies*	10
Gaviscon*	4
Hyoscine butylbromide	4
Gelucil*	3
Ranitidine HCl	2
Mucaine*	2
Cyclizine HCl	2
Others	24

*: Generic names for combination antacids.

Discussion

This study is the first to document the true incidence of GIT symptoms in athletes participating in six common endurance sports. The main findings of the study are that i) there is a high incidence of GIT symptoms in endurance athletes, ii) the incidence of lower GIT symptoms is higher than that of upper GIT symptoms, iii) the incidence and profile of GIT symptoms differs between the six endurance sports that were surveyed, iv) the incidence of upper and lower GIT symptoms is more common in younger athletes, v) females have a higher incidence of GIT symptoms compared to males, vi) previous abdominal surgery is associated with upper GIT symptoms in endurance athletes, and vii) medication is used frequently to control GIT symptoms during competition.

Previous surveys have only documented the prevalence and frequency of GIT symptoms in one sport at a time.[29,33-35] In this study, the documentation of the true incidence of GIT symptoms allowed for a comparison to be made between the risk of developing GIT symptoms in different endurance sports. Based on the result of this study, endurance sports could be classified as high, medium or low risk for the development of different GIT symptoms (Table 2). These data confirm the hypothesis that certain sports, such as road running and triathlons, have a particular high risk for the development of lower GIT symptoms. Road running and the running leg of the triathlon are characterized by repetitive vertical oscillating movements. The mechanical movement of the bowel during these activities has been suggested as one of the mechanisms for the development of lower GIT symptoms in athletes.[10-13] Although the risk of GIT symptoms in triathletes has not been documented for each individual leg of the competition in our study, it has previously been reported that triathletes have noted that GIT symptoms are more frequent during the running leg of the competition.[34]

The relatively high incidence of GIT symptoms in canoeists was surprising. Canoeing is a gliding sport and the mechanical movement of the small and large intestines can not be postulated as a mechanism for the high incidence of GIT symptoms. It has however previously been reported that canoeists are exposed to water borne gastro-intestinal infections. Although this was not specifically documented in our study, this may account for the increased incidence of GIT symptoms experienced by this group of athletes.

The results from this study confirm the observations of other researchers that, in endurance sports, lower GIT symptoms are more common than upper GIT symptoms.[28-30] However, a novel finding is that this apparently holds true only for sports characterized by repetitive vertical oscillating movements (road running, triathlons, aerobic dancing) but not for gliding sports (swimming, cycling). This supports the hypothesis that the predominant mechanism responsible for the development of lower GIT symptoms is related to mechanical movement of the intestines. However, evidence to support this "mechanical theory" is still at best indirect.[10-13]

The results of this study also confirm the observations by other researchers that the incidence of GIT symptoms is age and gender related, with the groups at higher risk being younger and female.[29,33] The mechanisms for the gender and age related difference in risk of GIT symptoms are not known.

A further novel finding in this study was the documentation of an increased risk of GIT, in particular upper GIT, symptoms in athletes with a history of previous abdominal surgery. The precise mechanism for this is not clear and requires further investigation. However, young athletes undergoing abdominal surgery should perhaps be aware of this potential side effect.

There has been a lot of anecdotal evidence that the use of medication to control GIT symptoms in endurance athletes is common. This study is the first to document the frequency of use of medication to control symptoms. The nature of the medication used appears to be a reflection on the type of symptoms commonly experienced. It is however important to note that, despite the frequent use of medication by endurance athletes, the potential negative effects of GIT medication on performance and health during exercise has not been documented. This is an area that requires urgent investigation.

In summary, this study has shown that GIT symptoms are common in certain endurance sports, and that the younger female athlete is at particular risk to develop GIT symptoms. Furthermore, athletes with a previous history of abdominal surgery are more likely to develop upper GIT symptoms during exercise. Finally, although drug use appears to be a common method to control GIT symptoms, there are no data available for the prescribing physician to indicate whether these medications are safe and do not affect athletic performance.

REFERENCES

1. *Browns F, Saris WHM, Rehrer NJ: Abdominal complaints and gastrointestinal function during long-lasting exercise. Int J Sports Med 1987; 8: 175-189.*
2. *Astrand P, Rodahl K: Textbook of Work Physiology. Physiological Bases of Exercise (3rd Ed), New York City, McGraw-Hill Book Co 1986.*

3. Larson DC, Fisher R: Management of exercise-induced gastrointestinal problems. Phys Sportsmed 1987; 15 (9): 112-126.
4. Wade OL, Combes B, Chilos AW et al: The effect of exercise on the splanchnic blood flow and splanchnic blood volume in normal men. Clin Sci 1956; 15: 457-463.
5. Clausen JP: Effect of physical training on cardiovascular adjustments to exercise in man. Physiol Rev 1977; 57 (4): 779-815.
6. Jager K, Bollinger A, Valli C, Amman R: Measurement of mesenteric blood flow by duplex-scanning. J Vasc Surg 1986; 57 (4): 462-469.
7. Heer M, Repond F, Harry A, Sulser H, Kehl D, Jager K: Acute ischaemic colitis in female long distance runner. GUT 1987; 28: 896-899.
8. Rehrer NJ: Fluid intake and gastrointestinal problems in runners competing in a 25 km race and a marathon. 1990; Chapter 8: 153-163.
9. Rehrer NJ, Beckers RJ, Browns F, ten Hoor F, Saris WHM: Effects of dehydration on gastric emptying and gastrointestinal distress while running. Med Sci Sports Exerc 1990; 22: (in press).
10. Porter AMW: Case report. Marathon running and caecal slap syndrome. Brit J Sports Med 1982; 16: 178.
11. Rehrer NJ: Biomechanical vibration of the abdominal region during running and bicycling. PhD Thesis 1990; Chapter 4: 77-84.
12. Dawson DJ, Khan AW, Shreve DR: Psoas muscle hypertrophy: Mechanical causes for "joggers trots"? Letter: Brit Med J 1985; 291: 787-788.
13. Blacklock NJ: Bladder trauma in the long distance runner: "10 000 meters haematuria". Brit J Urol 1977; 49: 129-132.
14. Pruett TL, Wilding ME, Gamble WG: Caecal volvulus: A different twist for the serious runner. Letter N Engl J Med 1985; 312 (19): 1262-1263.
15. Sullivan SN, Champion MC, Christofides ND, Adrian TE, Bloom SR: The gastrointestinal regulatory peptide responses in long-distance runners. Phys Sportsmed 1984; 12 (7): 77-82.
16. Rehrer N: Gastrointestinal symptoms and physiological changes as a result of ultraendurance running. Chapter 9, Thesis PhD, 1990.
17. Riddoch C: Exercise induced gastrointestinal symptoms – hormonal involvement. PhD Thesis, The Queen's University of Belfast, 1990.
18. Harber VJ, Sutton JR: Endorphins and exercise. Sports Med 1984; 1: 154-174.
19. Neely J: The effect of analgesic drugs on gastrointestinal motility in man. Br J Surg 1969; 56: 925-929.
20. Demers LM, Harrison TS, Halbert DR, Santen RJ: Effect of prolonged exercise on plasma prostaglandin levels. Sports Med 1984; 12: 77-82.
21. Upjohn HL: Nutrition and athletes. JAMA 1953; 151: 818-819.
22. Eichner ER: Gastrointestinal bleeding in athletes. Phys Sportsmed 1989; 17 (5): 128-140.
23. Sullivan N: The effect of running on the gastrointestinal tract. J Clin Gastrointestinal 1984; 6: 461-465.
24. Gabelli VJ, Durfour AP, Levin MA, McCabe KJ, Hebermin PW: Relationship of microbial indications to health effects at marine bathing beaches. Am J Public Health 1979; 69: 690-692.
25. Koopmans JS, Eckert EA, Greenber HB, Strohn BL, Isaacson RE, Monly AS: Norwald virus enteric illness acquired by swimming exposure. Am J Epidemiol 1982; 115: 173-177.
26. Jenkins DJA: Carbohydrate Dietary Fiber. In: Modern Nutrition in Health and Disease (7th ed). Eds: Shils NE, Young VR: Lea and Fabinger, Philadelphia.
27. Forgoros RN: "Runners Trots". Gastrointestinal disturbances in runners. JAMA 1980; 243 (17): 1743-1744.
28. Sullivan SN: The gastrointestinal symptoms of running. N Engl J Med 1981; 304 (15): 915.
29. Riddoch C, Trinick R: Gastrointestinal disturbances in marathon runners. Br J Sports Med 1988; 98: 644-646.
30. Halvorsen F, Ritland S: Gastrointestinal problems related to endurance event training. Sports Med 1992; 14(3): 157-163.
31. Runner's World, May 1969; 72:
32. Thompson PD, Funk EJ, Carleton RA et al: Incidence of death during jogging in Rhode Island from 1975 through 1980. JAMA 1982; 274: 2535-2536.
33. Keeffe EB, Low DK, Goss JR, Wayne R: Gastrointestinal symptoms of marathon runners. West J Med 1984; 141 (4): 481-484.
34. Sullivan SN: Exercise-associated symptoms in triathletes. Phys Sportsmed 1987; 15 (9): 101-110.
35. Prieve WM, Priebe J: Abstracts. Runner's Diarrhoea (RD). Prevalence and clinical symptomatology, 1984. □

ISOSTAR LONG ENERGY –
NEW ENERGY DRINK FOR ENDURANCE SPORT

Sports nutrition has taken on a new dimension with the launching of a revolutionary fast-acting, long-lasting isotonic energy drink – ISOSTAR LONG ENERGY – specially developed with complex soluble carbohydrates for endurance sport.

Scientifically formulated in Switzerland by the manufacturers of Europe's No 1 isotonic sports drink, ISOSTAR LONG ENERGY is the new endurance fuel to enhance performance, conserve energy reserves and ensure rapid recovery after exercise to maximise performance potential.

ISOSTAR is trusted by top international sportsmen and women to combat dehydration, replace lost energy, enhance performance and revitalise the body. The isotonic formulation of the new ISOSTAR LONG ENERGY ensures rapid absorption and optimal utilisation of three complex carbohydrates, vitamins and electrolytes. Energy reserves are conserved through this rapid absorption during exercise resulting in delayed fatigue and improved performance. Athletes are able to recover more quickly as the energy reserves in their muscles are restored after exercise and this in turn maximises the recovery of the performance potential. Another unique property of new ISOSTAR LONG ENERGY is its antioxidant content which helps to prevent the build up of toxic

oxidation products.

It should be taken before and during intensive exercise. For endurance events, 150-300 ml should be taken beforehand and 100-200 ml is recommended every 15-20 minutes during the race.

For further information about ISOSTAR products, contact Mike Brown of Pharmaceutical Enterprises (Pty) Ltd, Tel: (021) 531-1341 or Fax: (021) 531-2692.

The effect of Protein Supplementation on Muscle Strength and Body Composition in Body-builders

M.I. Lambert, E.V. Lambert, L. Bock, and T.D. Noakes

Summary

In spite of little scientific evidence, there is a belief among bodybuilders and other sportspersons that a diet high in protein promotes the development of muscle hypertrophy and strength. To examine whether this is indeed true we randomly assigned 22 body-builders, who trained 6 hours per week and who ingested less than 2.0g protein.kg^{-1}.day^{-1}, to either an experimental group (n = 12) or a control group (n = 10). In a double-blind study design, the experimental group received an additional 75g protein per day for 8 weeks which increased their daily intake to 2.7g protein.kg^{-1}.day^{-1}. The control group received an isoenergetic carbohydrate supplement. Before starting the supplementation and again 8 weeks later, the following were measured in subjects; body composition, 24-hour urinary urea and creatinine excretion and tests of muscle function. Urinary urea and creatinine excretion were also measured every 2 weeks. Although body mass, relaxed arm, contracted arm and chest girth increased significantly ($p < 0.05$) and equally in both groups, none of the other anthropometric variables changed significantly in either group during the study. In both groups there was a significant increase in the bench press and squat exercise strength, but isokinetic torque of the forearm extensor and flexor and knee flexor and extensor muscles did not change. Daily urea nitrogen excretion was higher in the experimental group for the duration of the study. We conclude that body-builders who consume 175-200% of the protein RDA, and who train about 6 hours per week, show no additional gains in muscle size or strength if they increase the protein content in their diets by 75g.

Introduction

It is commonly believed that a diet high in protein promotes muscle hypertrophy and increases strength.[1,2] As a result sportspersons, particularly resistance-trained athletes, often have habitual diets which exceed the recommended daily allowance (RDA) for protein[3] by 250%.[2,4] In addition, they often boost their habitual protein intake by ingesting protein supplements.[2] Indeed, a lucrative commercial market exists for these supplements.

Although many studies have examined the effect of a high protein intake on muscle size and strength the results are equivocal.[5,6,7] In a recent study,[7] Lemon et al., (1992) found that novice bodybuilders needed about twice as much protein to remain in a positive nitrogen balance compared to sedentary men. However, the development of muscle strength and hypertrophy did not increase when this level of protein intake was increased to about 300% of the recommended intake (2.6 g protein.kg^{-1}.day^{-1}).[7] This experiment was conducted over 4 weeks. A question arising from this study was whether the findings would have differed had the supplementation period been longer.

Therefore, the aim of the present study was to determine the effects of an 8 week programme of protein supplementation on muscle strength and muscle mass in a group of amateur body-builders. A double-blind experimental design was used and care was taken to ensure that the two groups were carefully matched for age, training volume, body composition, and energy, protein and carbohydrate intake.

Methods

Subjects

Amateur, male body-builders who reported not using anabolic steroids, volunteered to participate in this study. Subjects were selected based on their reported resistance training and their daily protein intake. They were excluded from the study if their protein intake exceeded 2.0g protein.kg^{-1}.day^{-1} or if they had been training for less than 2 years. All subjects reported having reached a "plateau" in muscle and strength development.

Twenty-two subjects were selected and randomly assigned to either the experimental or control group after they had given written informed consent. All procedures were approved by the Ethics and Research Committee of the Faculty of Medicine of the University of Cape Town.

Experimental design and supplementation

The experimental group received a protein powder which, when mixed with 750ml skim milk, provid-

Address for correspondence
M.I. Lambert, PhD.
MRC/UCT Bioenergetics of Exercise Research Unit, Department of Physiology,
University of Cape Town Medical School,
Cape Town, 7925,
South Africa.
Telephone 021-4066535. Fax 021-477669.

ed 75g of protein per day (Health and Performance Products International, Johannesburg, South Africa).

The control group received an isoenergetic carbohydrate powder which was indistinguishable from the supplement that the experimental group received. They were instructed to mix their powder with 750ml skim milk in an identical manner to the experimental subjects. This supplied an extra 24g of protein daily.

The supplements for both groups were coded so that neither the researchers nor the subjects knew their identity. Specific instructions were given to each subject to ensure accurate supplement ingestion. Subjects were instructed to continue to ingest either the supplement or placebo for the 8 weeks of the study.

Prior to beginning the supplementation, the following were measured on all subjects; body composition, 24-hour urinary urea and creatinine excretion and various tests of muscle function. Each of these tests was repeated after the 8-week period, except for urinary urea and creatinine excretion which were measured every 2 weeks.

Experimental procedure

Subjects were asked to record all the food they ingested for 7 days. They were requested to either weigh their food or use standard household measures to describe portion sizes accurately. Diatery composition was analyzed using the Floro Diet Data Program (Nederlandse Unilever Bedrijven, Rotterdam).

Body composition was assessed using an anthropometric technique which fractionated the body composition into muscle,[8] fat and lean body mass.[9] In our laboratory this technique of body composition assessment has been shown to relate well to assessments of body composition using densitometry and bioelectrical impedance ($r = 0.86$ and $r = 0.87$ respectively) (unpublished).

Chest girth and relaxed and contracted arm girth measurements were also recorded. The chest measurement was recorded at the level of the nipples after the subject had exhaled naturally. The arm girth measurements were recorded on the subject's dominant side, at the mid-point between the acromion process and the olecranon.

Subjects were given bottles in which they collected urine for a complete 24 hour period. The subjects were instructed to empty their bladders upon rising on the day of collection. Thereafter all urine voided, up to and including the sample voided upon rising the next day, was collected in the bottle. Samples of urine were frozen (-20°C) after the total volume for the 24 hour period was measured. On completion of the study urinary urea and creatinine concentrations were measured in these samples (Astra 8, Beckman Instruments, Brea, California).

Muscle function was assessed by determining the single maximum repetition for a bench press and squat exercise. Subjects performed this test after a day of light training using the same weight training technique they normally used in training. This technique, and the warm up procedure, were kept constant for the tests done both at the beginning and the end of the study.

Peak torque for forearm flexion and extension and knee flexion and extension were assessed on a Cybex isokinetic dynamometer (Lumex, Ronkonkama, NY) at a speed of $90^\circ.s^{-1}$. The subjects were thoroughly familiarized with the dynamometer before the assessment.

Each subject kept a diary in which he recorded daily training details and a daily rating of perceived exertion while training. The scale ranged from 1 ("very unpleasant") to 6 ("fantastic"). Subjects were requested to conform to their regular weight training programme and eating habits for the duration of the study. Subjects were interviewed every 2 weeks to discuss whether changes had been made to training and eating patterns. Supplement to last the next 2 weeks was given to each subject at this interview.

Statistical methods

All data are presented as means and standard deviations. A two-way analysis of variance with repeated measures was used to determine differences between groups and differences in the measurements within groups which occurred over time. Differences were accepted as being significant when $p < 0.05$. A Scheffe's post-hoc test was used to determine differences between groups when the overall F ratio was significant.

Results

Pre-trial

Subject characteristics

The age of the experimental subjects (26.9 ± 7.5 years) was similar to the control subjects (24.9 ± 6.7 years).

The anthropometric characteristics of both groups are presented in Table 1. There were no differences between groups for any of these variables.

TABLE 1: **Anthropometric characteristics of the experimental (n = 12) and control groups (n = 10) at the start and end of the 8 week study.**

	Experimental		Control	
	Pre	Post	Pre	Post
Mass (kg)	75.3 ± 10.7	76.2* ± 10.4	79.1 ± 9.7	79.5* 9.8
Fat %	11.5 ± 3.9	11.7 ± 3.9	11.7 ± 4.2	12.0 ± 3.8
Muscle Mass (kg)	42.3 ± 7.0	43.0 ± 7.1	46.6 ± 6.8	47.1 ± 6.2
Relaxed arm Girth (cm)	32.8 ± 2.8	33.3* ± 2.9	34.0 ± 2.1	34.5* ± 2.2
Contracted arm Girth (cm)	36.5 ± 3.0	37.2* ± 3.0	37.8 ± 2.2	38.4* ± 2.2
Chest girth (cm)	98.7 ± 8.3	100.3* ± 6.9	101.5 ± 6.8	102.9* ± 8.1

(p < 0.05 pre vs post)

(Values are expresed as means ± SD).

There was also no difference in the amount of resistance training done each week between the experimental group (5.7 ± 2.0 h.wk^{-1}) or the control group (6.2 ± 3.4 h.wk^{-1}). At the beginning of the study the index for the rating of perceived exertion was 3.6 ± 0.5 and 4.1 ± 0.9 for the experimental and control groups respectively.

The reported energy intake and nutrient composition of the pre-trial diets are shown in Table 2. There was no difference in the percent contribution of any macro-nutrient group to the energy intake of the groups.

Post-trial

The training was similar between groups and did not change throughout the study. During the last week the experimental group trained 5.1 ± 2.2h and control group 5.4 ± 2.5h. The rating of perceived exertion did not change throughout the study. During the last week of training the rating of perceived exertion was 4.1 ± 0.9 and 4.3 ± 1.1 (experimental and control).

We tried to repeat the 7-day diet record at the end of the study, but were unable to analyze the results because of poor subject compliance. However, every 2 weeks we had detailed discussions with each subject about his eating patterns. Based on these discussions there was no reason to believe that any subject made significant changes to his eating patterns during the study. In addition, all subjects reported that they had ingested all the supplement they were given for that period.

The small increase in body mass in both groups was statistically significant ($p < 0.05$) (Table 1). In addition, relaxed arm girth, contracted arm girth and chest girth increased significantly in both groups ($p < 0.05$) (Table 1).

TABLE 2: **Reported nutritional intake before protein supplementation for the experimental (n = 12) and control groups (n = 10).**

	Experimental	Control
Energy intake (kJ)	12 209 ± 2 742	11 180 ± 1 638
Carbohydrate (g)	330 ± 111	287 ± 80
Fat (g)	109 ± 29	109 ± 19
Protein (g.kg^{-1}.day^{-1})	1.6 ± 0.4	1.4 ± 0.2

Values are expressed as the mean ± SD.

Protein intake and urinary urea excretion

The experimental group increased protein intake by 63% to approximately 2.7 g protein.kg^{-1}. day^{-1} after the start of the study. Subjects in the control group ingested an additional 24g protein in the skim milk (21% increase), increasing their daily protein intake to 1.7 ± 0.2g protein.kg^{-1}.day^{-1}, but which was nevertheless significantly lower than the protein intake of the experimental group ($p < 0.0001$).

Daily urinary urea nitrogen excretion did not change in the control group, but was higher in the experimental group for the duration of the study ($p < 0.5$; Table 3). This pattern remained the same when the urinary urea excretion was divided by the creatinine concentration to normalize for urine output (figure 1).

TABLE 3: **Urinary urea nitrogen excretion (g.24hr^{-1}) for the experimental (n = 12) and control groups (n = 10) for the duration of the 8 week study.**

	Experimental	Control
0 weeks	11.6 ± 4.6	11.3 ± 2.9
2 weeks	18.2 ± 3.7*	12.5 ± 2.5
4 weeks	19.8 ± 5.0*	12.5 ± 5.6
6 weeks	18.5 ± 5.2*	14.1 ± 6.0
8 weeks	16.4 ± 4.0*	11.5 ± 3.6

* $p < 0.05$ experimental vs control, and experimental 2, 4, 6, 8 week vs experimental 0 weeks.

Values are expressed as the mean ± SD.

Muscle function

The maximal strength results are shown in Table 4. Both groups had a significant but similar increase in the bench press (7% and 6%; experimental and control) and squat (12% vs 9%; experimental and control) exercises over the 8 week period ($p < 0.05$).

Peak torque for forearm extension and flexion and knee extension and flexion did not change as a result of training or supplementation (Table 5).

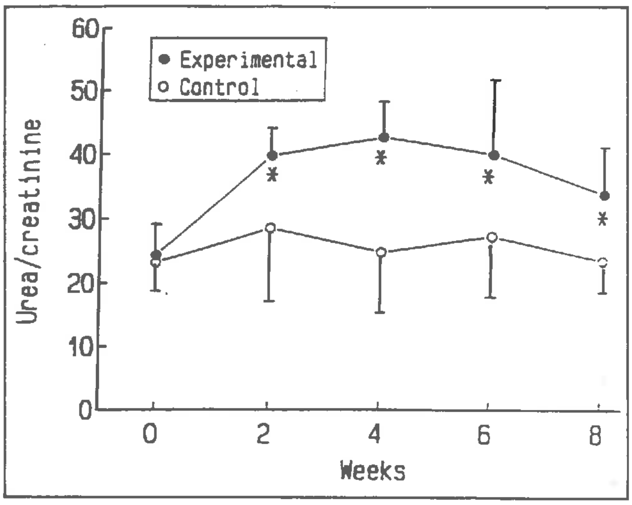


Figure 1: *Urinary urea/creatinine ratios for the experimental and control groups over 8 weeks (* p < 0.05).*

TABLE 4: Maximal voluntary strength for the experimental (n = 12) and control groups (n = 10) before and after 8 weeks of training and protein supplementation.

	Experimental		Control	
	Pre	Post	Pre	Post
Bench Press (kg)	98.1 ± 15.4	105.2* ± 13.0	105.4 ± 19.3	111.5* ± 23.3
Squat (kg)	131.0 ± 37.1	146.4* ± 31.9	138.5 ± 37.7	151.3* ± 35.5

* p < 0.05, pre vs post

(Values are expresed as means ± SD).

Discussion

The popular lay belief that resistance-trained athletes need to consume more protein than the RDA to remain in a positive nitrogen balance has been supported by several scientific studies.[7,10,11] However, the belief that muscle size and strength may be increased by ingesting protein in levels exceeding the RDA has yet to be supported by firm scientific evidence. A pitfall of the studies that have examined this question is that they have either lacked adequate dietary and exercise control[6] or were of short duration (4 weeks).[7]

Accordingly, in our study we placed special emphasis on the matching of the two groups for those factors such as age, body composition, dietary intake and training (Table 1 and 2), all which may influence the rate of adaptation to resistance training. Furthermore, the research protocol was double-blind and the energy intake of the two groups was kept the same by increasing the carbohydrate intake of the control group to match the increased energy of the protein supplemented group. This is important because differences in dietary energy content may affect nitrogen balance[12,13] which may also influence the rate of adaptation to resistance training.[14] Therefore, we believe that any differences between the groups at the end of the study could have been attributed to the differences in protein intake.

TABLE 5: Peak torque (N.m) for forearm extension and flexion and knee extension and flexion measured on the Cybex isokinetic dynamometer (at 90o.S-1) for the experimental (n = 12) and control groups (n = 10) before and after 8 weeks of training and protein supplementation.

	Experimental		Control	
	Pre	Post	Pre	Post
Isokinetic torque (N.m)				
Forearm Flexion	72.0 ± 15.9	77.4 ± 16.3	76.9 ± 17.1	82.8 ± 11.1
Forearm Extension	69.6 ± 13.3	75.7 ± 10.8	80.7 ± 14.9	76.7 ± 11.5
Knee Extension	265.3 ± 44.6	262.5 ± 53.8	301.0 ± 17.8	289.9 ± 25.2
Knee Flexion	141.5 ± 17.8	144.7 ± 25.2	180.9 ± 37.0	165.3 ± 17.8

(Values are expresed as means ± SD).

Therefore, this study shows that an 8 week period of resistance training resulted in a gain in strength, body mass and girth measurements in body-builders who were consuming about twice the protein RDA.[3] This level of protein ingestion has been shown to be comparable to the amount of protein habitually ingested by body-builders.[2,4]

The main finding of the present study, however, is that no further change in muscle strength or body composition resulted after increasing the protein intake to 3-fold greater than the RDA. In addition, it was shown in our study that increasing the protein content in the diet of bodybuilders, already ingesting twice the RDA for protein, did not modify the perception of fatigue during training and therefore did not offer any advantage to the body-builders by allowing them to train harder.

The second important finding of this study was that at least 60% of the increased nitrogen intake was excreted as urinary urea nitrogen (Figure 1

(Continued on page 35)

The efficacy of low power laser irradiation in the treatment of Chronic Tendonitis in Athletes. A controlled clinical trial.

LM Jankelowitz, BSc (Physio), BSc (Med) (Hons) Exercise Science.
MP Schwellnus, MB BCh, MSc (Med) Sports Science, MD, FACSM.
TD Noakes, MB ChB, MD, FACSM.

Abstract

The purpose of this study was to investigate the efficacy of low power infrared laser irradiation in the treatment of chronic tendonitis (Achilles, semimembranosis, patella) in athletes. Twelve athletes presenting with chronic tendonitis were matched for injury site and severity and randomly allocated to either an experimental or a control group. Patients were instructed to avoid exercise for the fourteen day duration of the study and both groups received the same standard physiotherapy treatment. Low power laser irradiation was administered to both groups in a double blind fashion using either an active or a sham laser. On days 1 and 14, patients performed a functional treadmill running test during which they reported pain on a scale of 0-10, for each minute run. Area under the pain vs time curve, average pain per meter and average pain per minute were calculated. Daily 24 hour pain recall scores at rest, walking and overall pain were also recorded. Pain scores decreased significantly ($p < 0.5$) for both groups during the 14 days of treatment, but no differences were observed between the two groups. Isokinetic muscle strength tests were conducted on days 1 and 14 and the percentage deficit between the injured and uninjured muscles in peak torque was calculated. No significant improvements ($p < 0.05$) were found. The results of this study indicate that irradiation did not alter the pain reduction during running in patients already receiving conventional physiotherapy treatment for chronic tendonitis.

Key Words
laser, tendonitis, athletes.

Introduction

Laser is an acronym originating from Light Amplification by Stimulated Emission of Radiation.[5,15] The therapeutic effect of electromagnetic radiation in the visible spectrum (sunlight) was recognized from as early as 400 BC.[24] Since the late 1960's, low power energy laser treatment has been advocated as a new, safe and effective treatment for a multitude of neurological, musculoskeletal and soft tissue conditions.[3] However, despite 20 years of investigation and clinical use, the clinical effectiveness of low power laser is still not well documented.[3]

Low power laser irradiation has been fund to be of value in the treatment of pain, swelling and haematomas.[7,11] The proposed mechanisms for pain reduction are reduction of prostaglandin E levels,[12,14,24] increasing gate control,[9] changing nerve conduction thresholds[27] and the stimulation of central descending pain inhibitory systems.[25] It has been proposed that swelling is reduced by changes in cell membrane and capillary membrane permeability.[27] Haematomas are more effectively treated by low power irradiation because of increasing vasodilation, mechanical action and by causing an anti-aggregating effect on platelets.[11] Wound healing is also enhanced by low power laser irradiation as a result of increased cellular activity and collagen synthesis,[1,4,8,16,17] enhanced immune cell activity and by phagocytosis of debris.[17]

Published literature on the therapeutic use of laser in chronic sports injuries is difficult to interpret and only a few well controlled trials have been published.[7,13] In general, these clinical trials lack details of treatment, double-blind controls, well described statistical analysis and adequate follow up data.[7] Despite this lack of adequate data, physical therapists use low power laser irradiation in the treatment of chronic sports injuries.

The purpose of this study was to investigate the effects of adding low power laser irradiation to a standard physiotherapy treatment regime for chronic tendon injuries in athletes. The design of this study is novel because it uses a double blind controlled clinical trial design and a sensitive method of determining clinical outcome.[22] The goal of this study was to provide scientific information to the therapist using low power laser irradiation in the treatment of sports injuries.

Address for correspondence
Dr MP Schwellnus, MRC/UCT Bioenergetics of Exercise Research Unit, Department of Physiology, University of Cape Town Medical School.
Observatory, 7925.
South Africa.
Telephone: 021-406-6504. Fax 021-47-7669.

Methods

Subjects over the age of sixteen presenting with chronic Achilles, semimembranosis or patella tendonitis were recruited from the Sports Injury Clinic of the University of Cape Town Sports Centre over a period of 8 months (March 1990 – October 1990).

The diagnosis of chronic tendonitis was made by a medical practitioner using the following diagnostic criteria:

- symptoms for more than 14 days
- pain in the area of the tendon during activity[20,21]
- local tenderness over the tendon during pinching or palpation[20,21]
- thickening of the tendon
- crepitus[21]
- pain on repetitive resisted movement.

Patients were excluded from the study if they had: received medication or had physiotherapy within the previous 7 days; a history of previous disorders such as fractures or surgery in the area of the tendon, a medical history of rheumatoid arthritis, sero-negative arthritis or Reiter's syndrome, or any clinical evidence of abnormal blood supply to the area.

The severity of the condition was assessed by grading the pain experienced during running as follows:[19]

Grade 1: Pain experienced only after running and therefore not affecting performance.
Grade 2: Pain experienced during running and not affecting performance.
Grade 3: Pain experienced during running and severe enough to affect performance.
Grade 4: Pain so severe that it prevents running.

Patients presenting with Grade 1 and 4 pain were not included in the study.

Written informed consent was obtained from all the patients. The study was approved by the Ethics and Research Committee of the Faculty of Medicine of the University of Cape Town. A running history was obtained from all the patients which included: years of running, duration of symptoms, weekly training distance and average training speed.

Sixteen patients were initially entered into the trial but four subjects were subsequently excluded. The reasons for exclusion were either not complying with home instructions or not presenting themselves for treatment on the correct days. The final number of patients included in the trial was, therefore, twelve.

Procedures

Subjects were matched for injury site and severity and were randomly assigned to either the experimental group (E) or the control group (C). Six pairs were therefore studied. The tendons that were involved in the six pairs were Achilles tendon (3), semimembranosus (2) and patellar tendon (1).

Both Group E and C received six sessions of standard physiotherapy treatment on days 1, 4, 6, 8, 11 and 13 of a 14 day treatment trial. The standard physiotherapy included: rest, deep transverse frictions (DTF), stretching, and icing.

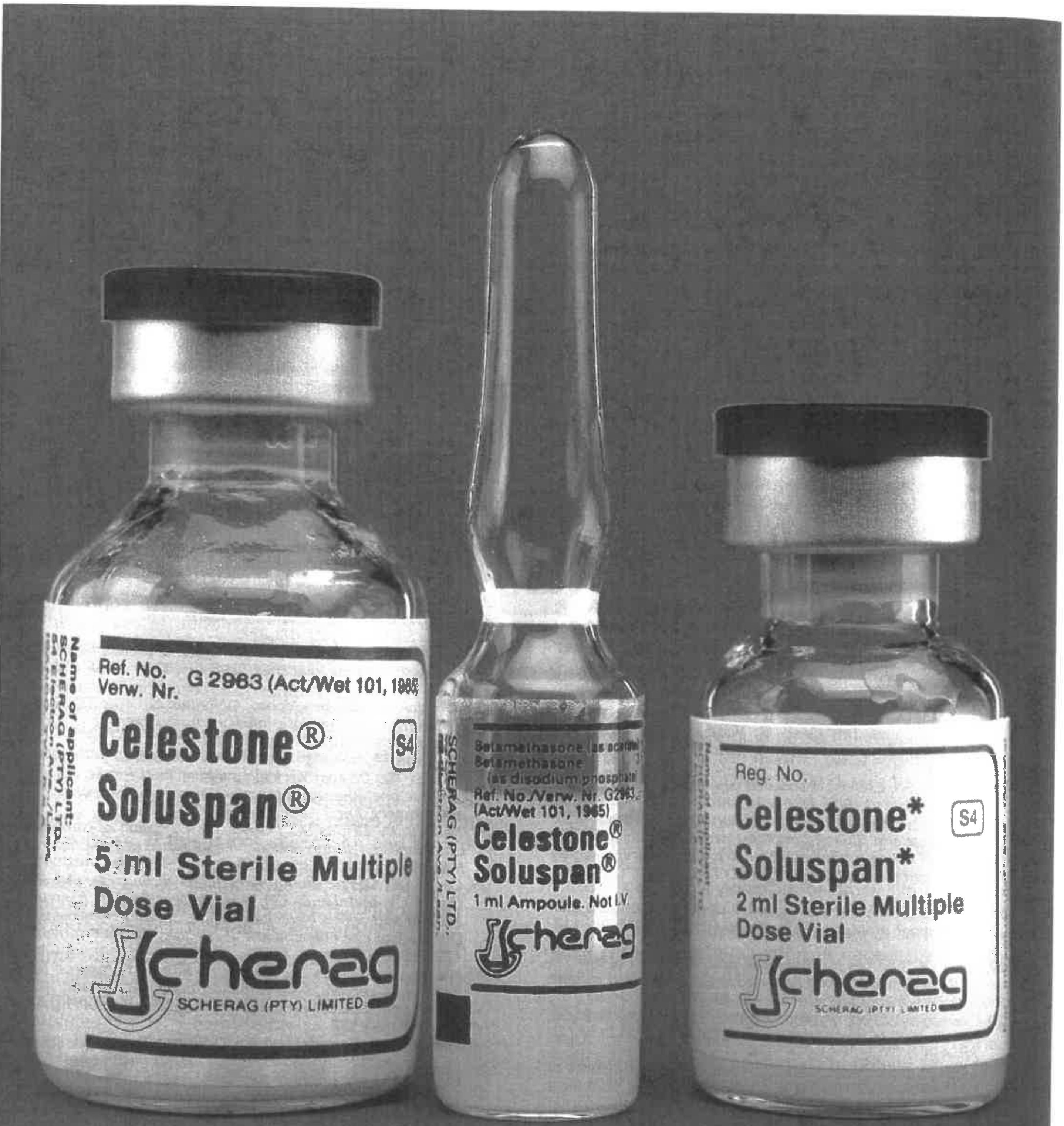
All the patients were instructed to rest during the treatment period. This meant that they refrained from participating in all sporting activities for the duration of the study. Deep transverse frictions were applied to the most tender region of the tendon for 10 minutes. The same therapist administered the DTF treatment to all the patients. The pressure was light for the first 3 minutes and then progressively increased but remained within the pain tolerance of the patients. The tendon was kept in an accessible position and frictions were applied at right angles to the tendon.[25,26] The DTF treatment was followed by a static 30 second stretch.[10] Patients stretched into discomfort but not into pain and were requested to repeat the stretch each morning and evening at home. Crushed ice in a damp towel was applied to the area for a period of 15 minutes following treatment.

At the end of the standard physiotherapy treatment session, laser treatment by the same physiotherapist was administered to both groups in a double blind placebo controlled fashion on days 1, 4, 6, 8, 11 and 13 of the study. Group E received active laser treatment while Group C received treatment from a sham laser. Two Mesolaser IRO1 generators were supplied by Medico Electronics (Belgium) and Medical Distributors (South Africa). One of these machines was altered by the technical staff of the local distributors so that no irradiation took place when the laser was switched on. This machine was coded as the sham laser. The active machine was calibrated every six weeks for the duration of the study. The codes for the active and sham lasers were only revealed once the statistical analysis was complete.

Prior to the laser treatment both the laser head and the area of the injured tendon were cleaned with isopropyl alcohol. The treatment area was then divided into blocks of 1 cm^2 and laser irradiation was then applied in a "grid" technique.[12] Patients received 40 seconds of laser irradiation per 1 cm^2 at a peak power of 30 Watts and a frequency of 5 000 Hz. The infrared beam applied to the tendon had a wavelength of 904 nm and a pulse-width of 200 n secs. The infrared laser was chosen because it has a greater depth of penetration than the HeNe (Helium-Neon) laser. Research indicates that the infrared laser would be more useful in the treatment of tendon injuries.[7,12,23,24,27,28] The laser was kept in a stationary position on the surface of the skin for each 40 second bout. The laser probe was then lifted and transferred to the adjacent 1 cm^2 block for the next 40 seconds. In this fashion, 4J of energy was delivered to each cm^2 of the tender area.

The efficacy of the treatment was assessed by a functional treadmill test, a conventional daily recall on a pain scale and an isokinetic muscle strength test.

The functional treadmill test[22] was performed on Day 1 and Day 14. The subjects were dressed in running shorts and vests and wore the same running shoes for both tests. A Powerjog treadmill


Ref. No.
Verw. Nr. G 2963 (Act/Wet 101, 1965)
Celestone®
Soluspan®
S4
5 ml Sterile Multiple
Dose Vial
Scherag
SCHERAG (PTY) LIMITED
Ref. No./Verw. Nr. G2963
(Act/Wet 101, 1965)
Celestone®
Soluspan®
1 ml Ampoule. Not I.V.
Scherag
Reg. No.
Celestone*
Soluspan*
S4
2 ml Sterile Multiple
Dose Vial
Scherag
SCHERAG (PTY) LIMITED

(Sport Engineering Ltd, Birmingham, UK) which had been calibrated prior to the onset of the study was used. The gradient was set at 0° and the subjects started with a one minute warm-up. The test was then started and a running speed was chosen which corresponded to the subject's normal training speed. The identical running speeds were used during the second test on Day 14. During the test, patients were asked to report the degree of pain they experienced each minute while running. The pain scale was a visual analogue scale ranging from 0 to 10 where 0 represented no pain and 10 unbearable pain. Subjects were free to stop the test at any time. The speed and distance run was recorded every minute. The test was discontinued if the patients experienced pain equivalent to 8 on the pain scale, or after 30 minutes of running.

The pain grading, which was reported every minute, was plotted against time (min) for each subject on Day 1 and Day 14.

The following parameters were calculated from the pain vs time graph.

- Total pain experienced which was calculated as the area under the pain vs time curve for that test.
- Maximum pain, which is the time run multiplied by 10 (the maximum pain that can be recorded).
- Percentage maximum pain which is the total pain experienced divided by the maximum pain (expressed as a percentage).
- Pain per minute which is the total pain experienced divided by the duration (min) of the test.
- Pain per kilometer run which is the total pain experienced divided by the distance (km) run during the test.

The percentage maximum pain, pain per minute and pain per kilometer were compared between the two groups and over the 14 day period.

On the first visit the patient was familiarized with the pain scale that was used for both the daily pain recall and the treadmill running test. Patients were instructed to record their pain experienced at rest, during walking and overall pain at the end of every day on a daily pain report form. The changes in reported pain over the 14 days were then compared between the two groups.

The Isokinetic muscle strength test was performed on Day 1 and Day 14 on a Cybex II isokinetic dynamometer (Cybex, Division of Lumex Inc, New York, USA). The ankle plantar and dorsiflexion torque was measured at a speed of 30°/sec in patients presenting with Achilles tendonitis. The knee flexion and extension torque was measured at a speed of 60°/sec in patients presenting with patella or semimembranosis tendonitis. Patients were strapped to the chair in a standard fashion[6] and were asked to perform five warm-up repetitions at the designated speed. This was followed by five maximum repetitions through the full range of movement. Peak torque (Nm) was measured for the muscle on the injured and the uninjured side. The deficit (%) between the injured and the corresponding uninjured muscle was taken as the percentage peak torque deficit. The percentage peak torque deficit (%) was then compared between the two treatment groups before and after the treatment period.

Data Analysis

Statistical analysis was performed on all the dependent variables using a two-way analysis of variance (Groups by time). The independent student t-test was used to determine differences in the physical characteristics and running history variables between the groups. The level of statistical significance was established at $p<0.05$.

Results

The physical characteristics and running history of the subjects in the two groups are listed in Table 1.

Functional treadmill test: The results (Table 2) of pain during the treadmill running test are depicted in Figures 1, 2 and 3. Although there was a significant decrease in all three variables over the treatment period, the changes in percentage maximum pain, pain experienced per kilometer run and pain per minute were not significantly different (ANOVA test) between the groups.

Daily pain recall: The mean daily pain scores recorded for rest, walking and overall pain over the treatment period in each group showed no significant difference between the two groups over the 14 days. However, there was a significant reduction in pain during rest, walking and overall pain for the two groups over the 14 days. The patterns of decrease in pain was similar for pain at rest, during walking and overall pain. The pattern for overall pain is shown in Figure 4.

TABLE 1: Physical characteristics and training history in the experimental (E) and control (C) group

	E Group	C Group
Age (years)	28.0 ± 8.0	26.0 ± 10.0
Weight (kg)	66.5 ± 9.9	65.8 ± 11.1
Years of running (years)	5.0 ± 5.0	9.0 ± 3.0
Running distance/ weeks (km)	40.0 ± 21.0	51.0 ± 40.0
Training speed (min/km)	4.9 ± 0.4	4.25 ± 0.6
Duration of symptoms (months)	5.9 ± 9.0	28.6 ± 35.6
Injury grade at presentation	3.0 ± 0.9	3.0 ± 0.9

No significant differences ($p < 0.05$) were observed between the groups using the independent t-test. Values are means ± SD.

Isokinetic muscle strength test: The results of the isokinetic muscle strength testing are listed in Table 3. The percentage change in peak torque deficit between the injured and uninjured limbs was not significantly different between the groups or over the treatment period.

TABLE 2: A comparison of parameters obtained during the treadmill running test for the experimental (E) and control (C) groups

	E Group	C Group
Percentage maximum pain (%)		
Before	24.6 ± 10.7	23.3 ± 17.8
After	6.0 ± 8.9*	7.5 ± 8.5*
Treadmill pain per kilometer (units/kilometer)		
Before	13.6 ± 4.5	14.7 ± 13.3
After	4.8 ± 4.4*	4.7 ± 5.4*
Treadmill pain per minute (units/min)		
Before	2.5 ± 1.1	2.3 ± 1.8
After	0.9 ± 0.9*	0.8 ± 0.9*

No significant differences ($p < 0.05$) were observed between the E and C groups. Values are means ± SD.

* Indicates significant difference before and after the 14 days of treatment ($p < 0.05$).

TABLE 3: Percentage change in peak torque deficit for the experimental (E) and control (C) groups

	E Group	C Group
Before	8.0 ± 13.6	1.3 ± 11.8
After	-1.5 ± 8.7	9.0 ± 12.2

No significant differences ($p < 0.05$) were observed between the groups or for the groups over the 14 day treatment period. Values are means ± SD.

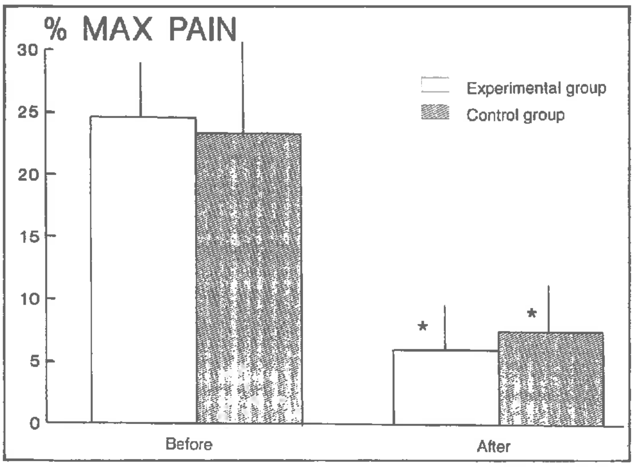


Figure 1. *The percentage maximum pain experienced during running in each group before and after treatment for 14 days. Values are mean ± SEM.*
**: indicates significant differences ($p < 0.05$) before and after treatment.*

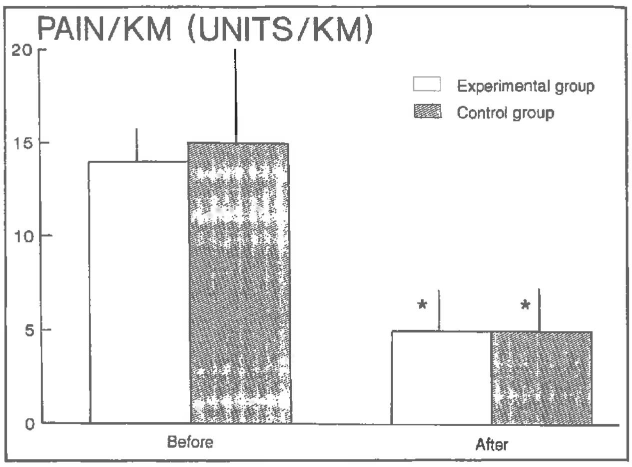


Figure 2. *The pain experienced per kilometer, during running by each group before and after treatment for 14 days. Values are mean ± SEM.*
**: indicates significant differences ($p < 0.05$) before and after treatment.*

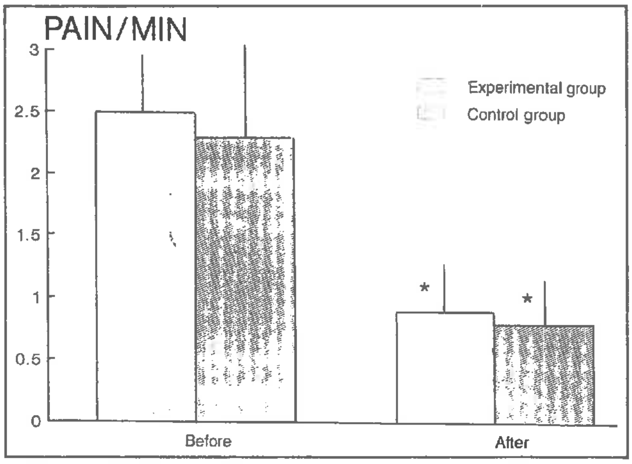


Figure 3. *The pain experienced, per minute, during running by each group before and after treatment for 14 days. Values are mean ± SEM.*
**: indicates significant differences ($p < 0.05$) before and after treatment.*

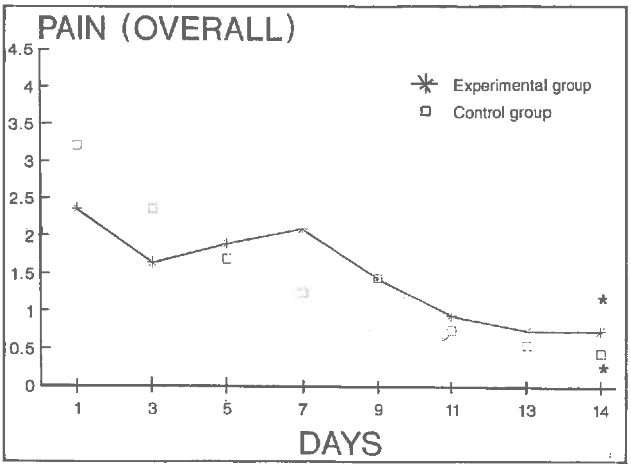


Figure 4. *The mean daily overall pain recorded in each group over 14 days of treatment. Values are mean ± SEM.*
**: indicates significant difference ($p < 0.05$) over 14 days of treatment.*

Discussion

The principle finding of this study was that rest, DTF, ice and stretching improve pain during running in athletes with chronic tendonitis. The addition of low power laser irradiation did not result in a further reduction of pain.

In this study, a functional treadmill running test showed significant decreases in pain for both groups over the 14 days of treatment. An additional finding was that percentage peak torque deficit did not change in either group with treatment.

The failure of the treatment to bring about changes in peak torque deficit can be attributed to the fact that injured tendons are not necessarily associated with weak muscles; in fact in this study the muscles on the injured side were often stronger than the uninjured side. Furthermore, treatment would not necessarily have resulted in an improvement in muscle strength particularly because strength training was not specifically included in this treatment regime. The duration of treatment (14 days) was also too short to result in dramatic increases in muscle strength.

The failure to demonstrate any additional clinical improvement with low power laser irradiation to the treatment programme in these athletes could be attributed to any one of the following:

- the methods of determining pain reduction were not sensitive enough;
- low power laser irradiation does not provide any therapeutic benefit;
- an incorrect laser treatment regime was used;
- standard physiotherapy negated any additional effects of laser treatment;
- different pain grades at the onset of the study.

A subjective method of determining clinical improvement is not always ideal, but the functional treadmill running test used in this study is a direct measure of pain during the specific activity that elicited pain. This test has been shown to be a sensitive indicator of therapeutic outcome in clinical trials.[22] It is therefore not likely that this test was not sensitive enough to highlight small differences in clinical improvement between the two groups.

A definite protocol for effective laser treatment in chronic injuries has not yet been established. The laser treatment regime used in this study was based on protocols in the existing literature. Laser irradiation at higher intensities or for longer durations may have influenced the clinical outcome.

The possibility that low power laser irradiation does not offer any benefit in the treatment of chronic injuries must be considered. Although previous researchers have described benefits in the use of low power laser irradiation in the treatment of chronic injuries[2,13,18] this study can not substantiate these observations.

The pain reduction that our subjects experienced may have been the result of receiving adequate standard physiotherapy. A group receiving no treatment was not studied and it can therefore not be concluded that the standard physiotherapy was responsible for the improvement in pain. The standard treatment may have negated or masked the effects of adding low power laser irradiation.

In summary rest, DTF, ice and stretching significantly reduce pain during running in athletes with chronic tendon injuries. The addition of low power laser irradiation in the dose and frequency as described in this study did not influence this reduction of pain.

Acknowledgements

The authors wish to acknowledge the support of Medico Electronics (Belgium) and Medical Distributors (South Africa). We also wish to thank the staff of 2 Military Hospital, Wynberg as well as all our subjects for their co-operation.

REFERENCES

1. *Abergel RP, Meeker CA, Lam TS, Dwyer RM, Lesavoy MA, Uitto J: Control of connective tissue metabolism by lasers: Recent developments and future prospects. Unpublished from Division of Dermatology, Department of Medicine, UCLA School of Medicine.*
2. *Airaksinen O, Rantanen P: Laser in physical medicine. A review. Scandinavian Journal of Acupuncture and Electrotherapy 2:101-104, 1987.*
3. *Basford JR: Low-energy laser therapy: Controversies and new research findings. Lasers Surg Med 9:1-5, 1989.*
4. *Bosatra M, Jucci A, Olliaro P, Quacci D, Sacchi S: In vitro fibroblast and dermis fibroblast activation by laser irradiation at low energy. Dermatologica 168:157-162, 1984.*
5. *Caspers KH: Stimulation therapy with laser beams. Physikalische Medizin und Rehabilitation 18(9):426-445, 1977.*
6. *Cybex Manual: isolated-joint testing and exercise. Cybex, Ronkomkana, New York, 1982.*
7. *de Bie RA, Steenbruggen RA, Bouter LM: Effect of laser therapy on ankle sprains. Nederlandse Tydschrift Fysiotherapie 99:4-7, 1989.*
8. *Diamantopoulos C: Low power lasers – theory and practice in physiotherapy. Omega Universal Technologies Limited, London, January 1987.*
9. *Guyton AC: Somatic Sensations. In: Textbook of Medical Physiology, 6th Edit II, Chap 50, pp 616-617. Ed. WB Saunders Company, USA, 1981.*
10. *Kibler WB, Chandler TJ, Uhl T, Maddax RE: A musculoskeletal approach to the pre-participaton physical examination – preventing injury and improving performance. Am J Sports Med 17(4):525-531, 1989.*
11. *Kiyoizumi T: Low level diode laser treatment for haematomas under grafted skin and its photobiological mechanisms. Keio J Med 37:415-428, 1988.*
12. *Liggins C: Light amplification by stimulated emission of radiation. Contents of a workshop on Laser Therapy, Dept Physiotherapy, Univ of Durban Westville, South Africa, 1988.*
13. *Lundberg T, Haker E, Thomas M: Effect of laser versus placebo in tennis elbow. Scand J Rehab Med 19:135-138, 1987.*
14. *Macfarlane TS, Callander R: Inflammation, Infection and Healing 29-84. Pathology Illustrated, 2nd ED: Churchill Livingstone, London, 1986.*
15. *Mesolaser IRO1 and Frequency Scanning manual. Medico Electronic, Belgium (Medical Distributors Pty Ltd), Woodstock, South Africa, 1985.*
16. *Mester E, Mester AF, Mester A: The biomedical effects of laser application. Lasers Surg Med 5:31-39, 1985.*
17. *Mester E, Spiry T, Szende B, Tota JG: Effect of laser rays on wound healing. Am J Surg 122:532-535, 1971.*
18. *Mitsou A, Emmanouilidis O, Roumeliotis D, Piskopakis N: Super luminous diodes and laser diode applications in*

SPORTS SCIENCE INSTITUTE OF SOUTH AFRICA

Applications are invited from interested persons or practices who may wish to set up a practice at the Sports Science Institute of South Africa at Newlands, Cape Town.

The Sports Science Institute of South Africa is an Incorporated Association not for Gain which, inter alia, will offer sports medical services to sportspersons as well as development, wellness and corporate fitness/health programmes.

The University of Cape Town's Bio-Energetics of Exercise Research Unit, headed by Professor Tim Noakes, will occupy space in the building and the Institute will work closely with the University in promoting teaching and research in sports medicine.

Facilities for sports medicine related activities include the following:

- Sports Medicine Physicians
- Physiotherapists
- Orthotists/Prosthetists
- Sports Psychologists
- Optometrists
- Orthopaedic Surgeons
- Dieticians
- Radiologists

The above will operate either on sessional bases or will establish private practices at the Institute and lease space in the building.

It is envisaged that the successful applicants will:

(i) have an established track record in providing services to sportspersons;
(ii) be prepared to assist in the training of post graduate students in sports physiotherapy, sports medicine, sports science and biokinetics;
(iii) be involved in research in sports medicine related fields;
(iv) be prepared to assist in lecturing to post graduate students.

Rentals are to be negotiated and will be based on current market rates and will take into account the use by the practices of other facilities at the Institute (eg., rehabilitation gymnasium, swimming pool, gymnasium, exercise area and equipment).

The Sports Science Institute of South Africa will commence its activities during July 1995.

All interested persons are invited to forward their curriculum vitae and proposals in writing to Mr A Donovan, c/o Sports Science Institute of South Africa, PO Box 2593 Clareinch, 7740 or fax number (021) 472735. Applications to reach this office by 26 April 1995.

recovery time reduction on athletes injuries. Presented at the Congreso Internacional Sobre Las Aplicaciones Medicas Y Quirurgicas De Los Laser, Barcelona, 8,9 y 10 de Noviembre, 1985.
19. Noakes TD: Running injuries, 368. In: Lore of Running, Cape Town: Oxford University Press, 1985.
20. Noble C: The Pfizer Manual of Sports Injuries. A Medical Tribune Publication, South Africa, 1:21-23, 1988.
21. Petersen L, Renstrom P: Sports injuries and their prevention and treatment, 41-42. 1st Ed, (ed) Juta and Company Ltd., South Africa, 1986.
22. Schwellnus MP, Theusissen L, Noakes TD, Reinach SG: Anti-inflammatory and combined anti-inflammatory/analgesic medication in the early management of iliotibial band friction syndrome: A clinical trial. South African Med J 79(10): 602-606, 1991.
23. Seitz LM, Kleinhart JA: Low-power laser: its application in Physical Therapy, 217-238. In: Michlovitz SL and Wolf SL (eds). Contemporary perspectives in Rehabilitation: Thermal agents in Rehabilitation. Philadelphia: Davis, 1986.
24. Thiel H: Low power laser therapy – An introduction and a review of some biological effects. The American Chiropractor 8:14-77, 1987.
25. Walker J: Relief from chronic pain by low-power laser irradiation. Neuroscience Letters 43:339-344, 1983.
26. Winter B: Transverse frictions. South African Physiotherapy J March: 5-7, 1968.
27. Woolley-Hart A: A handbook for low-power lasers and their medical application. New Medicine Series 19-36: 1987.
28. Woolley-Hart A: Soft laser – light devices. Inter Medica 24-26, 1987. □

(Continued from page 28)

and Table 3), which is similar to the findings of Lemon et al (1992).[7] The urinary urea nitrogen excretion remained elevated for the duration of the study (Figure 1 and Table 3). Based on the nitrogen balance data from the study of Lemon et al., (1992)[7] we can assume that each subject in the high protein group retained about 7g nitrogen per day. Had this nitrogen been directed at the myofibrillar protein pool, the subjects in the high protein group would have had an increase of about 12kg in muscle mass (assuming muscle consists of about 20% protein and protein consists of 16% nitrogen). Clearly this was not the case (table 1), which suggests that the additional protein was metabolized, as also found by Zaragoza et al., (1987)[15] and that protein synthesis does not increase.[11]

In summary, the findings of this study add to the findings of Lemon et al (1992)[11] and show that there is no advantage for bodybuilders to supplement their diets with protein if they are already consuming about twice the RDA for protein.

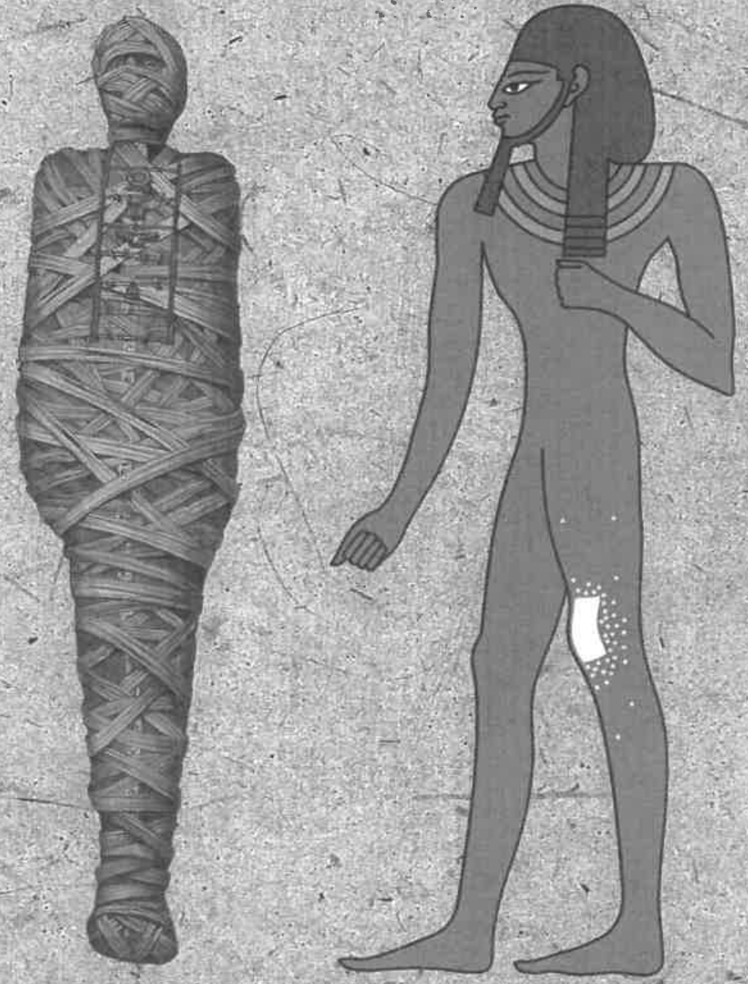
Acknowledgements
The Renal Unit, Groote Schuure Hospital, Cape Town, assisted with the analyses of urinary creatinine and urea concentrations.

This work was supported by the South African Medical Research Council and the Harry Crossley and Nellie Atkinson Staff Research Funds of the University of Cape Town.

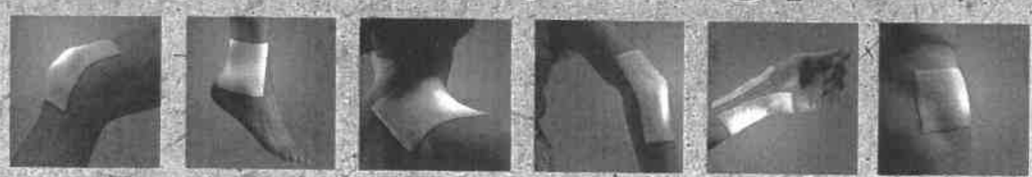
REFERENCES

1. Grandjean, A.C., Hursch, L.M., Majure, W.C. & Hanley, D.F. Nutrition knowledge and practices of college athletes. Med. Sci. Sports Exerc. 1981; 13: 82.
2. Faber, M. & Benade, A.J.S. Nutrient intake and dietary supplementation in body-builders. S. Afr. Med. J. 1987; 72: 831-834.
3. Food and Nutrition Board. Recommended Daily Allowances. Washington, DC: National Academy, 1989; 10: 52-77.
4. Lemon, P.W.R. Protein and amino acid needs of the strength athlete. Int. J. Sports Nutr. 1991; 1: 127-145.
5. Celejowa, I. & Homa, M. Food intake, nitrogen and energy balance in Polish weight-lifters during a training camp. Nutr. Metab. 1970; 12: 259-274.
6. Dragan, G.I., Vasiliu, A. & Georgescu, E. Effects of increased supply of protein on elite weight-lifters. In Milk Proteins' 84. eds T.E. Galesloot & B.J. Tinbergen. 1985:99-103, Wageningen, Netherlands: Pudoc.
7. Lemon, P.W.R., Tarnopolsky, M.A., MacDougal, J.D. & Atkinson, S.A. Protein requirements and muscle mass/strength changes during intensive training in novice bodybuilders. J. Appl. Physiol. 1992; 73: 767-775.
8. Martin, A.D., Spenst, L.F., Drinkwater, D.T. & Clarys. J.P. Anthropometric estimation of muscle mass in men. Med. Sci. Sports Exerc. 1990; 22: 729-733.
9. Durnin, J.V.G.A. & Womersley, J. Body fat assessed from the total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. Br. J. Nutr. 1974; 32: 77-97.
10. Tarnopolsky, M.A. MacDougall, J.D. & Atkinson, S.A. Influence of protein intake and training status on nitrogen balance and lean body mass. J. Appl. Physiol. 1988; 64: 187-193.
11. Tarnopolsky, M.A., Atkinson, S.A., MacDougall, J.D., Chesley, A., Phillips, S. & Schwarcz. H.P. Evaluation of protein requirements for trained strength athletes. J. Appl. Physiol. 1992; 73: 1986-1995.
12. Butterfield, G.E. Whole-body protein utilization in humans. Med Sci. Sports Exerc. 1987; 19: S157-S165.
13. Chiang, A-N. & Huang, P-C. Excess energy and nitrogen balance at protein intakes above the requirement level in young men. Am. J. Clin. Nutr. 1988; 48: 1015-1022.
14. Walberg, J.L., Leidy, M.K., Sturgill, D.J. Hinkle, D.E. Ritchey, S.J. & Sebolt, D.R. Macronutrient content of a hypoenergy diet affects nitrogen retention and muscle function in weight lifters. Int.; J. Sports Med. 1988; 9: 261-266.
15. Zaragoza, R., Renau-Piqueras, J., Portoles, M., Hernandez-Yago, J., Jorda, A. & Grisolia, S. Rats fed prolonged high protein diets show an increase in nitrogen metabolism and liver megamitochondria. Arch. Biochem. Biophys. 1987; 258: 426-435. □

Why punish the whole body for one painful inflammatory site?



Focus your NSAID power with TransAct LAT, the new locally acting patch



- TransAct LAT is effective
- TransAct LAT is locally-acting
- TransAct LAT is very well accepted by patients
- TransAct LAT has an excellent tolerability profile

NEW
TransAct LAT
Focus your NSAID power
Flurbiprofen 40mg

S3 TransAct LAT. Each patch