Proceedings of a Workshop on

SPORTING INJURIES IN HORSES AND MAN: A COMPARATIVE APPROACH

23rd – 25th September 2004
Lexington, USA

Editors: E. J. L. Soulsby and J. F. Wade

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EDITORS' FOREWORD

The focus of this meeting is to promote a sharing of expertise between the disciplines whose primary aim is to enhance the sporting performance and protect the health of the human or animal athlete and particularly to relate new developments in the biomedical science to the prevention and treatment of sporting injuries.

From the perspective of injuries that occur during training or competition, the human being has been studied more than any other animal species. However, due to the economic importance of the horse more veterinary scientific effort has been devoted to this species than any other.

Many of the diseases and injuries that occur to horses during training and competition have direct parallels with those seen in man. For example, tendon injuries, stress fractures, osteochondrosis, osteoarthritis, ulceration of the gastro-intestinal tract, pulmonary haemorrhage, myxomatous valve disease and sudden cardiac death. For medical studies, animal models are often developed to study these conditions whereas, by contrast, equine conditions are more commonly studied in the target species.

This workshop will review the conditions that occur to the different body systems under various forms of exercise in horse and man; and explore whether the similarities and differences can be studied to mutual advantage. A number of questions could be considered:

- Is the horse a good animal model for man due to its longevity, its 5cism and the fact that certain injuries share similar patho-physiology?

- Can man be used as an 'animal' model for conditions seen in the horse?

- What can we gain from a comparative approach that will potentially benefit both the horse and man?

Our objectives, within the context of this Workshop, are to:

- Review each designated area highlighting the types of injury most frequently seen in equine and human athletes and our knowledge of the pathophysiology;

- Identify conditions in the horse which have parallels in man;

- Determine whether there are good animal or human models that can be applied to the horse and vice versa;

- Explore the potential for comparative studies of sporting injuries in horse and man;

- Investigate ways to reduce the severity and or frequency of sporting injuries in the horse

Those of us working in the field of equine research are extremely fortunate to have the ongoing support of the Havemeyer Foundation. This organisation has become renowned for organising small workshop meetings on fairly esoteric topics, almost always with a comparative element. One of its main aims in doing so is to promote international collaboration at a multi-disciplinary and often multi-species level. We are indebted to the Foundation's President, Mr Gene Pranzo, for his enthusiasm in this task and for the help and encouragement he provides to so many of the equine science community.

Lord Soulsby of Swaffham Prior
Workshop Chairman

Dr David Marlin
Workshop Organiser

HAVEMEYER SCIENTIFIC WORKSHOPS

1981   **First International Workshop on Lymphocyte Alloantigens of the Horse**
October - New York City, USA
Organiser: Dr D. F. Antczak

1982   **Second International Workshop on Lymphocyte Alloantigens of the Horse**
October - Cornell University, Ithaca, New York, USA
Organiser: Dr D. F. Antczak

1983   **Third International Workshop on Lymphocyte Alloantigens of the Horse**
April - New Bolton Center, University of Pennsylvania, USA
Organiser: Dr D. F. Antczak

1984   **First International Symposium on Equine Embryo Transfer**
October - Cornell University, Ithaca, New York, USA
Organisers: Drs D. F. Antczak and W. R. Allen

1985   **Fourth International Workshop on Lymphocyte Alloantigens of the Horse**
October - University of Kentucky, USA
Organisers: Drs D. F. Antczak and E. Bailey

1986   **Workshop on *Corynebacterium equi* Pneumonia of Foals**
July - University of Guelph, Canada
Organiser: Dr J. F. Prescott

1987   **Fifth International Workshop on Lymphocyte Alloantigens of the Horse**
October - Louisiana State University, USA
Organisers: Drs D. F. Antczak and J. McClure

1989   **Second International Symposium on Equine Embryo Transfer**
February - Banff, Alberta, Canada
Organisers: Drs D. F. Antczak and W. R. Allen

1990   **International Workshop on Equine Sarcoids**
April - Interlaken, Switzerland
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1992   **Workshop on Equine Neonatal Medicine**
January - Naples, Florida
Organisers: Drs D. F. Antczak and P. D. Rossdale

Third International Symposium on Equine Embryo Transfer
February - Buenos Aires, Argentina
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1995   **Equine Perinatology**
July - Cambridge, England
Organiser: Dr P. D. Rossdale

Second International Equine Leucocyte Antigen Workshop
July - Lake Tahoe, California, USA
Organisers: Drs D. F. Antczak, P. Lunn and M. Holmes

First International Workshop on Equine Gene Mapping
October - Lexington, Kentucky, USA
Organisers: Drs D. F. Antczak and E. Bailey

Erection and Ejaculation in the Human Male and Stallion: A Comparative Study
October - Mount Joy, Pennsylvania, USA
Organiser: Dr S. M. McDonnell

Bone Remodelling Workshop
October - Corcord, Massachusetts, USA
Organiser: Dr H. Seeherman

1997   **Second International Workshop on Equine Gene Mapping**
October - San Diego, California, USA
Organisers: Drs D. F. Antczak and E. Bailey

Maternal Recognition of Pregnancy in the Mare
January - Dominican Republic
Organisers: Drs W. R. Allen and T. A. E. Stout

Uterine Clearance
March - Gainesville, Florida, USA
Organiser: Dr M. M. LeBlanc

Trophoblast Differentiation
September - Edinburgh, Scotland
Organisers: Drs D. F. Antczak and F. Stewart

1998   **Third International Genome Workshop**
January - San Diego, California, USA
Organisers: Drs D. F. Antczak and E. Bailey

Third International Workshop on Perinatology: Genesis and Post Natal Consequences of Abnormal Intrauterine Developments: Comparative Aspects
February - Sydney, Australia
Organiser: Dr P. D. Rossdale

Horse Genomics and the Genetic Factors Affecting Race Horse Performance
March - Banbury Center, Cold Spring Harbor, New York, USA
Organisers: Drs D. F. Antczak, E. Bailey and J. Witkowski

Allergic Diseases of the Horse
April - Lipica, Slovenia
Organisers: Drs D. F. Antczak, S. Lazary and E. Marti

Equine Placentitis Workshop
October - Lexington, Kentucky, USA
Organisers: Drs D. F. Antczak, W. R. Allen and W. Zent

Septicemia II Workshop
November - Boston, Massachusetts, USA
Organiser: Dr M. R. Paradis

1999   **Equine Genome Project**
January - San Diego, California, USA
Organisers: Drs D. F. Antczak and E. Bailey

Third International Equine Genome Workshop
June - Uppsala, Sweden
Organisers: Drs D. F. Antczak, E. Bailey and K. Sandberg

Fourth International Meeting of OIE and WHO Experts on Control of Equine Influenza
August - Miami, Florida, USA
Organiser: Dr J. Mumford

European Equine Gamete Workshop
September - Lopuszna, Poland
Organisers: Drs W. R. Allen and M. Tischner

Fetomaternal Control of Pregnancy
November - Barbados, West Indies
Organisers: Drs T. Stout and W. R. Allen

2000   **Equine Genome Project**
January - San Diego, California, USA
Organisers: Drs D. F. Antczak and E. Bailey

Uterine Infections in Mares and Women: A Comparative Study
March - Naples, Florida, USA
Organiser: Dr M. M. LeBlanc

5th International Symposium on Equine Embryo Transfer
July - Saari, Finland
Organiser: Dr T. Katila

2001   **USDA International Plant & Animal Genome Conference**
January - San Diego, California

Equine Immunology in 2001
January - Santa Fe, New Mexico
Organiser: Dr D. P. Lunn

Asthma and Allergies II
April - Hungary
Organisers: S. Lazary and E. Marti

From Elephants to Aids
June - Port Douglas, Australia
Organiser: Professor W. R. Allen

International Equine Gene Mapping
July - Brisbane, Australia
Organiser: K. Bell

Second Meeting of the European Gamete Group (EEGG)
September - Loosdrecht, The Netherlands
Organiser: Dr T. A. E. Stout

Foal Septicemia III
October - Tufts University European Center, Talloires, France
Organiser: M. R. Paradis

Infectious Disease Programme for the Equine Industry and Veterinary Practitioners
October - Marilyn duPont Scott Medical Center, Morvan Park, Virginia, USA
Organisers: Drs J. A. Mumford and F. Fregin

From Epididymis to Embryo
October - Fairmont Hotel, New Orleans, USA
Organiser: Dr L. H-A. Morris

2002   **USDA International Plant & Animal Genome Conference**
January - San Diego, California

Comparative Neonatology/Perinatology
January - Palm Springs, California
Organiser: P. Sibbons

Stallion Behavior IV
June - Reykjavik, Iceland
Organisers: S. McDonell and D. Miller

Rhodococcus Equi II
July - Pullman, Washington
Organiser: J. Prescott

Equine Orthopaedic Infection
August - Dublin, Ireland
Organiser: E. Santschi

Inflammatory Airway Disease
September - Boston, USA
Organiser: Dr E. Robinson

2003   **USDA International Plant and Animal Genome Conference**
January - San Diego, California

Embryonic and Fetal Nutrition
May - Ravello, Italy
Organiser: S. Wilsher

Genomics and the Equine Immunity System
June - Ithaca, New York
Organiser: D. F. Antczak

Fifth International Gene Mapping Workshop
August - Kreuger Park, South Africa
Organiser: E. Baily and E. Vandyke

Equine Recurrent Laryngeal Neuropathy
September - Stratford-upon-Avon, UK
Organisers: P. Dixon and E. Robinson

Transporting Gametes and Embryos
October - Brewster, Massachusetts
Organiser: E. Squires

Third Meeting of the European Gamete Group (EEGG)
October - Pardubice, Czech Republic
Organisers: J. Müller and Z. Müller

Nosocomial Infections and Biosecurity in Equine Hospitals
October - Lexington, USA
Organisers: F. Bain and J. Taub-Dargatz

2004   **USDA International Plant and Animal Genome Conference**
January - San Diego, California

Equine Viral Herpes Virus Workshop
June/July - Tuscany, Italy
Organiser: P. Lunn

6th International Symposium on Equine Embryo Transfer
August - Rio de Janiero, Brazil
Organiser: M. Alvarenga

Sporting Injuries in Horses and Man: A Comparative Approach
September - Lexington, USA
Organiser: E. J. L. Soulsby

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SESSION I:

Epidemiology

SPORTS INJURIES: EPIDEMIOLOGICAL STUDIES IN RACEHORSES

J. L. N. Wood[*], K. L. P. Verheyen, E. R. Ely and J. Price[*]

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Musculoskeletal problems are far and away the greatest cause of Thoroughbred racehorses not being entered for training, not being trained when in training yards or being lost from the training population altogether. Musculoskeletal injuries are the main cause of racehorses requiring euthanasia (Jeffcott *et al.* 1982) and, of these injuries, fractures are the main reason (Johnson *et al.* 1994; Parkin *et al.* 2004). Musculoskeletal injuries are also responsible for 80% of all clinical events noted after racing, be their outcome fatal or otherwise (Williams *et al.* 2001). Various authors have studied reasons for fatal injury during racing (Mohammed *et al.* 1991; Peloso *et al.* 1994; Bailey *et al.* 1998; Parkin *et al.* 2004) and most have reported that risk increases with the age of the horse and the firmness of the racing surface. In addition, a number have described how risk of catastrophic fracture often increases with the intensity of high speed training in the 30 or 60 day period prior to the fracture (Estberg *et al.* 1996).

Numerous epidemiological studies of factors associated with different types or definitions of injury after racing have been undertaken around the world over the last 10 years, but fewer have considered specific injuries sustained during a racehorse's career in a training yard. An average horse in the UK only races on 6 days each year (unpublished observations) and so spends the vast majority of its time away from the racecourse.

Our research interests have related to identification of modifiable risk factors for injury, especially fracture and tendon breakdown. As well as identifying exercise-related factors that may influence the risk of injury, we have taken particular interest in determining any effects of age at the start of racing or training.

Most Thoroughbred racehorses, like human athletes, are trained from an early age. Training starts at the end of their second year of life for racing on the flat. Flat races are usually a mile or less in distance, occasionally as long as 2 miles. Those horses bred to race over jumps have not traditionally been trained until the end of their fourth year (Jeffcott *et al.* 1982), being introduced to racing in 2 mile 'National Hunt flat races'. They have usually raced in 3 such races when aged 4 or 5 years. However, despite horses being more skeletally mature, the death rate in NHF racing is around 4 times higher than in younger horses racing in normal 'flat' races (McKee 1995). This paradox has never been explained, although studies in humans and experimental animals have shown that the immature skeleton is more sensitive to the effects of exercise. This led us to hypothesise that age at the onset of training influences long term resistance to fracture as horses which start training when immature are less likely to fracture because their bones are more sensitive to the osteogenic effects of mechanical loading.

RACECOURSE STUDIES

A large scale retrospective epidemiological study of fatal injuries in British horseracing was undertaken, based on the 719,000 race starts made in Britain in the 1990s. It was designed to determine the effects of both age at the time of the race, as well as at the time of the start of the racing career. Results from race starts made by relatively immature horses racing in flat races were

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compared to those from more mature horses being introduced to racing in National Hunt flat races.

As reported previously (Wood *et al.* 2001), the incidence risk of fatal injury was 0.9/1,000 starts (95% confidence interval (95%CI): 0.8–1.0) in flat races, whereas it was 3.8/1,000 starts in National Hunt flat races. Detailed statistical analysis of the flat dataset showed that risk increased significantly in horses starting racing when older, with those starting at 4 years being at around twice the risk of those starting when 2-years-old. Other factors associated with the risk of fatal injury included the firmness of the racing surface, or the 'going', which was officially recorded following a subjective assessment by racing officials, the racing frequency, the distance of the race and the quality of the horse, assessed by the official rating given to that horse by the racing authorities on the basis of its previous performance. The risk was highest on the firmest turf surface and progressively decreased with softness. Horses with the highest ratings were at lowest risk and, in general, the risk of fatal injury increased with length of race, although the degree of this increase varied markedly with the racing frequency. The risk in longer races was particularly increased for horses racing for the first time. For horses that had raced recently, the overall risk was lower and there was a much smaller increased risk associated with longer races.

The modelling predicted the overall fatality in National Hunt flat races in the same time period to be 4.0 deaths/1,000 starts. This prediction was much increased compared to that for normal flat racing in particular because of the longer race distance at the time of the first race ever and because of the much increased average age at first race appearance. As a result of these findings and other experience, including that of training horses to race over jumps in France, a series of NHF races open to 3-year-olds held over 1.5 miles has subsequently been started.

TRAINING YARD STUDIES

We have undertaken detailed prospective studies of equine injuries, particularly to bone, in a selection of flat training yards in England over a 2-year period, from 1998 to 2000 (Verheyen and Wood 2004). Thirteen UK racehorse trainers participated in a prospective study, providing 12,893 months of data on 1,178 horses in their care for 2 years, including their daily exercise regimes and fracture incidents. The incidence of non-traumatic fracture was 1.15/100 horse months (95% CI = 0.98, 1.35), with 78% of fractures occurring during training. A wide variety of fracture types and bones were involved, although at least 57% were stress fractures, and pelvic and tibial stress injuries accounted for 28% of fractures diagnosed. The substantial proportion of fractures that occurred during training emphasised the importance of studying these injuries away from the racecourse and the large number of stress injuries suggested that training regimes for young Thoroughbreds could be improved to create a more robust skeleton, able to withstand injury. More detailed analyses of this large dataset have started to identify specific training patterns that are associated with a higher risk of both fracture and sore shins, or dorsometacarpal disease (Verheyen *et al.* 2003; Verheyen *et al.* 2005).

Data from a pilot study of fracture and tendon injury in older jump racehorses (Ely *et al.* 2004) were used to plan a large-scale study that is currently underway in collaboration with scientists at the Royal Veterinary College. A main objective of this work is to determine whether horses that have come out of flat training (and hence that were in training since the end of their second year) have a lower incidence of fracture compared to horses that were bred for racing over jumps and that were not trained until the end of their third or fourth year of life. These results become available in 2006-2007.

Studying injuries in racehorses in training can provide a scientific basis for the design of safer training regimes and can also provide unique insight into the impact of different training surfaces, but their statistical analyses must be conducted with considerable care to ensure that effects of confounding variables in this complex environment are minimised and conclusions are robust. These studies are essential to provide hypotheses suitable for testing in basic research as well as testing different hypotheses in their own right.

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SESSION 2:

Tendon

HORSES FOR COURSES

A. E. Goodship

The Royal Veterinary College and Institute of Orthopaedics and Musculoskeletal Sciences, University College London, UK

Athletes have evolved in the animal kingdom through processes of long and short term adaptation to environmental demands. For both horses and man the evolutionary adaptations for enhanced performance as a requisite for survival have been developed further for elite competitive performance. The ability of biological systems to adapt to the functional demands of training for particular athletic activities has led to the ongoing drive to optimise these systems for competitive advantage.

Horses are precocial animals showing the ability to run shortly after birth. As a prey species there has been an evolutionary adaptation toward high speed locomotion. To this end the locomotor system has developed to accommodate the need for optimisation in terms of both mechanical advantage and energy utilisation. In athletic competition there are winners and losers, the extreme demands of competition and the rigours of training can result in injuries, as a consequence of demand exceeding the capacity of the tissues and structures of the biological systems (Goodship *et al.* 1994). The ability to minimise these injuries and improve performance requires an integrated appreciation of the pathophysiology of many different biological systems and their interaction with the external environment.

Tendon and ligament injuries are common in both equine and human athletes. Each species is able to act as a model for the other in understanding the pathophysiology of the injuries and in development of strategies to reduce injury. The prevalence of these injuries in the racehorse, an equine athlete trained for speed, is high (Williams *et al.* 2001) and localised to specific tendons and ligaments, the superficial digital flexor tendon being the most frequently affected. With modern methods of diagnostic imaging it has been possible to identify lesions in these specific tendons prior to overt clinical injury (Pickersgill 2000). Tendon 'bows' are devastating injuries and despite many methods of clinical management no one treatment has yet proved successful. Thus, once injured the general prospects for the animal to continue as an athlete are poor and recurrent injury is common. This results in significant welfare and financial problems for the equine industry.

The ability to prevent or reduce the incidence of tendon injury requires a greater understanding of the pathophysiology of tendons. Tendons are complex structures and specific tendons respond differently to increased levels of exercise. In addition, the response to exercise and susceptibility to injury may be age and exercise related. The superficial digital flexor tendon (SDFT), the most commonly injured tendon, supports and flexes the fetlock joint, but also acts as an elastic energy store, contributing to locomotor efficiency. For maximum energy storage the SDFT is stretched to its limits at the gallop; any small decrease in tendon strength will increase injury risk. Extensor tendons with a positional role rarely show over-strain injury.

In general, both tendons and ligaments have a complex structural hierarchy, the gross tendon comprising a series of structural sub-units. The fascicles within the gross tendon are separated by inter-fascicular fibrous connective tissue containing blood vessels and fibroblast like cells which produce growth factors that influence the intra-fascicular tenocytes. The collagen fibres within the fascicles have planar waveform morphology; this 'crimp' results in a non-linear load/deformation behaviour of the tendon. There are morphologically different sub-populations of tendon cells within the fascicles; these may have different roles in relation to cell signalling and

maintenance of the tendon matrix (Stanley *et al.* 2004). Tenocytes have cell processes similar to osteocytes and the cells communicate through gap junctions. The connexin proteins that form these gap junctions have different distributions in young and old horses and also differ between positional and energy storing tendons. This suggests different cellular communication systems are evident in different tendons.

At the ultra-structural level in developing tendons the collagen fibril diameters are initially uniformly distributed. During development the fibrils form a bimodal diameter distribution and there is an associated increase in strength with increased fibril diameter. Fibrillogenesis is modulated by the proteoglycans and other non-collagenous proteins within the matrix. There are tendon specific profiles of collagen fibril diameter distribution in the adult horse. The SDFT and suspensory ligament have a high proportion of small diameter fibrils, compared to the common digital extensor tendon and deep digital extensor. These different fibril diameter profiles are related to the functional differences in these tendons.

The molecular composition of tendon matrix comprises a high proportion of water, approximately 65%. The major constituent of the dry matter is the protein fibrillar collagen as type I collagen and in normal tendons only a small proportion of type III collagen. The non-collagenous proteins present, such as cartilage oligomeric matrix protein (COMP) and fibromodulin, may also influence the structural organisation and fibrillar morphology of collagen.

COMP is found in a number of load bearing fibrous tissue structures. In the equine SDFT the levels of COMP increase during development to a peak at 2 years of age in Thoroughbreds, after which levels decline to a low residual level by 5 years of age. Thus COMP is evident during the period in which the SDFT increases in cross-sectional area to it mature size at 2 years of age. In 2-year-old Thoroughbreds there is s significant correlation between COMP tissue levels and material properties of both stiffness and strength (Smith *et al.* 2002). This may suggest that COMP plays a role in the structural optimisation of the tendon matrix in young horses. The levels of COMP in tendons which have a different functional role, such as the common digital extensor tendon (CDET), in which the tendon acts as a positional tendon, are low throughout development and in the adult.

The general structural and material characteristics of tendon are related to the functional behaviour of the tendon. When loads are applied to a tendon there is a characteristic load deformation and stress/strain plot. With progressive loading to failure there is an initial non-linear phase with large extensions for increments of load, followed by a linear elastic phase, yield, plastic deformation and failure. In older horses the collagen crimps in the central core of the SDFT have different characteristics to the peripheral fibres. This results in higher stresses per unit deformation and earlier failure (Wilmink *et al.* 1992). The pattern of site specific failure of the core of this tendon is also seen in clinical lesions. In non-destructive cyclical loading tendon exhibits both pre-conditioning in the first few cycles of loading together with hysteresis representing energy loss as heat. Wilson and Goodship (1993) showed significant rises in SDFT core temperature during short periods of canter. Birch *et al.* (1997) demonstrated that cells from the SDFT core were able to tolerate these hypothermic conditions and that this phenomenon has now been found to be present in fetal horses.

Functional adaptation is a key component of training in all biological systems. In skeletal structures such as muscles and bone the response to changes in mechanical loading is well documented and cellular mechanisms are being unravelled. However, in tendons generally, and energy storing tendons specifically the response to increased exercise is not clear. Several training studies involving long and short periods of controlled conditioning in adult and growing Thoroughbreds have shown that energy storing tendons do not appear to undergo functional hypertrophy, but when adult horses are trained the SDFT may show changes such as fibrillar degradation and changes in the molecular composition of matrix, such as reductions in COMP levels, that resemble changes normally seen with ageing.

In young horses exercise appears to accelerate the rate of growth related increase in cross-sectional area in both extensor and flexor tendons, thus exercise during growth may represent a strategy for modification of tendon characteristics.

Batson *et al.* (2003) have observed differences in the material properties of equine positional and energy storing tendons. In the neonate the material properties of functionally different tendons are the same but after approximately 3 days the positional

tendons show a significant increase in modulus and the adult pattern of differential moduli between energy storing SDFT and positional CDET is evident. Training horses during growth can facilitate the rate of development of the SDFT, without inducing the changes seen in adults, providing a scientific if controversial, rationale for racing young horses.

On the basis of these observations it could be hypothesised that changes in energy storing tendons occur as a function of the number of loading cycles. These are increased in relation to age and exercise.

From analysis of tendons from a large number of Thoroughbreds there is a significant correlation between matrix morphology, molecular composition and material properties. Understanding the cellular mechanisms that control these matrix characteristics will provide input for the development of more appropriate training strategies in both equine and human athletes, and in addition novel approaches to the management of tendon injury. This information can also be used in development of tissue engineered constructs for tendon replacement in veterinary and human orthopaedic surgery.

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DIAGNOSIS AND MANAGEMENT OF TENDINOSIS AND TENDON RUPTURES IN HUMANS

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Tendons are the ropes that attach muscle to bone and transmit the forces enabling us to move. With repetitive lower extremity loading in sports requiring running and jumping, injuries to the patellar and Achilles' tendons are very common. The cause is not inflammation, rather repetitive loading, degeneration and reduced vascularity. Histologically, tendon biopsies show few inflammatory cells and disorganised collagen with vascular compromise. In the skeletally immature with open epiphyseal plates, tendon injuries are very uncommon and the weakest link is through the apophysis or off-shoot of bone and cartilage where the tendon attaches. Treatment is with stretching and strengthening and avoiding jumping activities until pain subsides. Within this age group, a fracture can occur through the tibial tubercle if there is an awkward landing such as in dunking a basketball. Reduction in internal fixation of the fracture is performed. Complete ruptures of the quadriceps, patellar, and Achilles' tendon occur in older individuals (age 40–60 years), with risk factors of less condition, activity which has not been recently performed, and reduced vascular supply to the tendon itself. Younger individuals with an overdeveloped quadriceps can sustain patellar tendon rupture typically in a deceleration, landing from a jump mechanism. Underlying medical conditions can also pre-dispose to tendon ruptures, as presented in this renal failure patient who sustained bilateral quadriceps tendon ruptures.

The classic positional tendon in the human foot and ankle is the posterior tibialis tendon which fails with repetitive loading and attrition and not by single event. Comparison to the horse, superficial digital flexor tendons supporting the fetlock seems logical. If repair of posterior tibialis injuries is not successful, then tendon transfers or mid-foot fusions are surgical options.

In the upper extremity, the rotator cuff–4tendons inserting on the greater tuberosity – has components of positioning the shoulder in space and energy absorption as in throwing overhead. The treatment of rotator cuff tears has advanced significantly with arthroscopic techniques and suture anchors.

The diagnosis of tendon injuries is by detailed history and thorough physical exam. If the diagnosis is not clear-cut, MRI scan or ultrasound may provide helpful information. Treatment of overuse tendon problems is by correction of underlying anatomic problems and assessment of the biomechanics of sport. Complete tendon rupture is an unpredictable event, but often, when the individual is doing an eccentric muscle loading activity without forethought, it may not be humanly possible. Future studies comparing diagnosis and treatment of tendon disorders in humans and horses, immature and mature, would greatly benefit the 2- and 4-legged athletes.

SESSION 3:

Physiology

PHYSIOLOGY OF HUMAN RUNNING: FROM MOTORS TO FUEL PUMPS

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THE ELASTIC HUMAN

In order for a human to run quickly and efficiently for a given period of time a number of physiological, biochemical and biomechanical processes must be optimised. Human running is accomplished by performing a series of bounces as gravitational energy is stored in our 'leg springs' during the leg shortening, or impact, phase and is released during the leg lengthening, or propulsion, phase (Fig 1). Approximately 0.6 J of energy are stored and released per kilogram per bounce in the foot and calf (Ker *et al.* 1987), compared with about 1.1 J per kg in a 0.5 ton horse (Minetti *et al.* 1999). The total energy stored and released in the whole leg represents a substantial portion (about half in humans) of the energy required to propel the body into the next step. Because the highly elastic (ie high energy return) tendons are most responsible for this spring-like behaviour, and their properties change in response to loading, it is reasonable that some portion of training should target the tendon. The tendon stiffness that is optimum for performance depends on the force transmitted through the tendon and on the tendon lengthening velocity during the run (eg it differs for different tendons and between sprint and long-distance running). However, it is known that chronic endurance or strength training increases tendon stiffness, while flexibility training reduces it. The effects of plyometrics training have not been measured directly in humans, although in rats there is evidence that it reduces stiffness and hysteresis (ie reduces the energy lost from the tendon). Thus, training a human runner may require some portion of these training modalities in order to optimise tendon stiffness, maximise energy return and therefore increase running efficiency.

OPTIMUM MUSCLE CONTROL

The remaining energy required for running must come from muscle contraction. It has been held traditionally that muscles lengthen, or work eccentrically, during the impact phase of running and shorten, or work concentrically, during the propulsive phase. Recent evidence from human research, and experiments on animals, shows

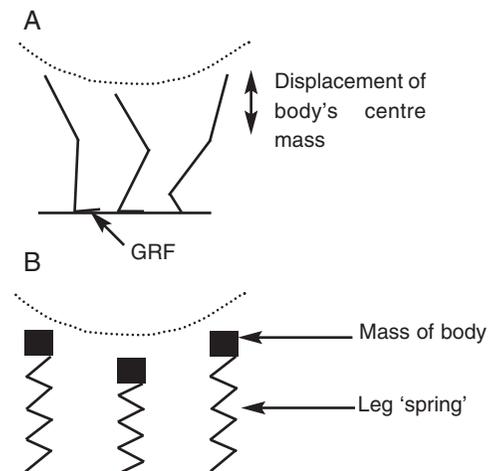*Fig 1: During running, the leg shortens during the impact phase as a result of the ground reaction force (GRF; A), and then lengthens during the propulsion phase as a result of the work performed by tendons (re-use of stored elastic energy) and muscles. Thus, the leg essentially functions as a spring (B) with about half of the work required to continue the spring bouncing being done by the muscles.*
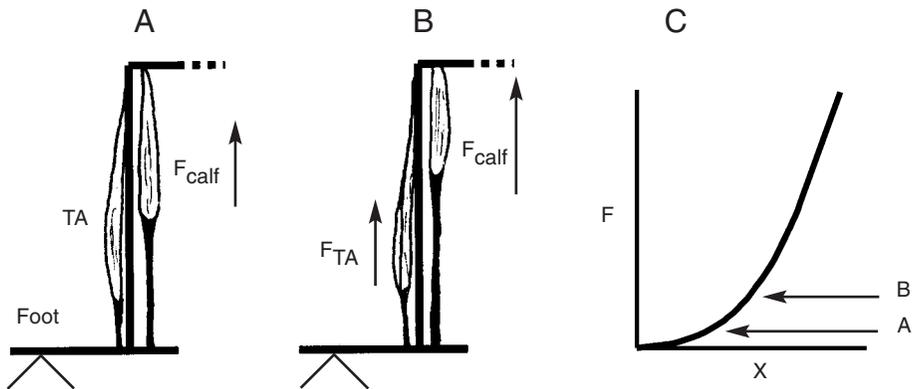
Fig 2: In order to produce the appropriate ankle plantarflexion moment during ground contact (A), the calf produces a force (F_{calf}), which stretches the calf tendon (Achilles'). However, by simultaneously generating force in the tibialis anterior muscle (F_{TA}) an opposing moment is created that allows the calf to generate even more force while maintaining the appropriate ankle angle (B). Since the Achilles' tendon is stretched further, it works at a higher point on its force-length curve (C), which allows greater energy to be stored during the impact phase. Importantly, the calf muscles can work quasi-isometrically during the propulsion phase and the metabolic cost of work is reduced.

however that muscles contract quasi-isometrically during the propulsive phase of many stretch-shorten-type movements (eg Kurokawa *et al.* 2003), or during high-speed movements performed without a counter-movement (Kurokawa *et al.* 2001). This makes sense when one considers the work of Hill (1938), who showed that concentrically-contracting muscle uses more energy than isometrically-contracting muscle, with the disparity increasing as muscle force or length change (or velocity) increased. As muscle power increases, the relative cost of performing work by concentric muscle action increases, and the benefit of using stored energy becomes greater.

As most human tendons work in the toe region of their force elongation curve during walking and jogging, slight muscle contraction to stretch the tendon and increase the joint's angle prior to foot-ground contact stores some energy in the tendon while resistance is low, and prepares the joint for foot-ground contact (Fig 2). By simultaneously contracting the antagonist muscle group the muscles of the limb shorten further and the tendon is stretched further to store more energy. This also increases the stiffness of the tendon (according to its length-force curve) so it can more effectively store energy during high-force elongation that occurs at foot-ground contact. During foot-ground contact then, the already-shortened muscle works quasi-isometrically, although there may be a further brief period of shortening immediately at foot

contact, while the tendon lengthens (and the leg shortens under the load). In this case, more energy is stored in the tendon and used in the propulsive phase, while relatively little work is performed by the muscle. Thus, optimisation of force production necessarily involves some muscular co-contraction. This should be taken into account when human runners are trained, as well as when the results of electrophysiological assessments of muscle activation during running are examined.

MAINTENANCE OF MUSCLE WORK

Regardless of the efficiency of the muscle-tendon complex, running performance is ultimately dictated by the ability to maintain the rate of muscle work. This requires the maintenance of optimum pH in the muscle and blood and the provision of ATP for operation of the contractile machinery. With respect to pH maintenance, higher-intensity training at, or above, lactate threshold improves H^+ buffering capacity more than longer training sessions at lower intensities (Weston *et al.* 1997; see Table 1). Also, the supply of oxygen to working tissue is improved with exercise (described below), and increases in total haemoglobin in response to very prolonged training or altitude exposure can improve buffering significantly (haemoglobin being a stronger buffer than bicarbonate ions or other proteins). More importantly, endurance training improves the rate of flux of H^+ from the muscle

TABLE 1: Summary of adaptations to exercise training

Adaptation	Training types influencing adaptation	Type of improvement
Tendon stiffness	Resistance, endurance (flexibility)	Increased (decreased) stiffness and natural frequency
Tendon hysteresis	Flexibility. Plyometrics (?)	Improved re-use of stored elastic energy
Muscle activation timing	Specific practice, drills.	Optimum control of muscle contraction sequences
ATP, PCr and glycogen storage	Resistance, sprint/anaerobic, endurance	Increased storage
Slow-twitch fibre proportion	Endurance	Transition from fast- to slow-twitch fibres
Capillary density	Endurance, longer anaerobic	Increased capillarisation
Blood shunting	Endurance, long anaerobic	Improved redistribution of blood during exercise
O_2 delivery	Endurance	Improved O_2 delivery
O_2 extraction	Endurance, long anaerobic	Improved O_2 extraction at muscle
Heart muscle contractile force	Endurance, anaerobic, resistance	Increased muscle contractility/force
Inspiratory muscle force/endurance	Endurance, anaerobic, inspiratory muscle training	Improved endurance of inspiratory muscles

into the blood, and thus favourably affects muscle pH. With respect to ATP provision, both endurance and sprint/resistance training result in increased ATP, PCr and glycogen stores in muscle, although prolonged endurance exercise is probably most beneficial for glycogen increases. However, in ultra-distance-type endurance exercise it is necessary to continually provide energy substrates for the production of ATP, which would also be improved with an increased perfusion of muscle.

Approximately 200–500 capillaries deliver blood to each square millimeter of working muscle (about 5–7 per fibre), with endurance-trained athletes having around a 40% higher capillary density than untrained individuals (Andersen and Henrickson 1977). Also, 4–7 ml of blood perfuses each 100 g of muscle per minute at rest with this increasing to about 75 ml at maximal exertion (Rowell *et al.* 1986). Most of this increased blood flow is directed to oxidative muscle tissue such that parts of the active quadriceps, for example, receive 300–400 ml per 100 g per min (eg Richardson *et al.* 1995). Indeed, some 80–85% of the total cardiac output is directed to working muscles during running. A greater ability to shunt blood to working muscle along with the increased capillarisation that

accompanies long anaerobic and aerobic training, allows a marked increase in muscle blood flow and a subsequent increase in oxygen and substrate delivery, as well as H^+ and heat removal, after prolonged training. Thus, local blood flow changes account for a large part of the total increase in muscle work capacity. It is this adaptation that probably triggers the accumulation of mitochondria in muscle, fibre-type-specific hypertrophy and possibly a shift in predominant fibre type toward the slow-twitch fibre.

In order to support the greater blood flow, cardiac output must increase. Endurance training causes significant increases in both left ventricle volume and wall thickness. This allows a greater stroke volume and greater contraction force (or, perhaps more importantly, the chance for the heart to work at a lesser load relative to maximum) during exercise. Indeed, sedentary individuals probably increase their cardiac output about 4-fold from resting levels to 20–22 l min^{-1} with a stroke volume of about 110 ml, while elite endurance athletes might increase their cardiac output by 7–8 times to 35–40 ml min^{-1} with a stroke volume of up to 220 ml.

The greater total blood flow also allows more oxygen to be obtained from the lungs. The ability for the lungs to breathe enough air to supply

oxygen is not considered a barrier to exercise performance since there exists a breathing reserve during maximal exercise; minute ventilation is only 60–85% of maximum voluntary ventilation. However, this view might be somewhat narrow as there is some evidence in elite endurance athletes that saturation of blood may not be complete at maximum exercise levels, although the reasons for this are unclear and it is not known how to influence this with training. Furthermore, significant fatigue of the diaphragm has been shown to cause a redistribution of blood flow away from the limbs at rest (Sheel *et al.* 2001). If this occurs as the respiratory muscles fatigue during exercise, the redistribution would compromise muscle work capacity. Indeed it is well known that inspiratory muscle resistive training positively influences both maximal inspiratory force and endurance performance (Romer *et al.* 2002).

CONSIDERATIONAS IN PHYSIOLOGICAL TRAINING

The efficient performance of running involves a complex interaction of physiological, biochemical and biomechanical processes. Each process can be targeted by specific training, with resistance, plyometrics, flexibility, anaerobic, aerobic and interval training all required. The difficulty for the athlete, coach or scientist is to programme the appropriate training with the appropriate volume, intensity and timing; so called periodisation. It is well known that adaptations of specific processes are compromised when training is performed concurrently, ie, training 2 processes on the same day or in the same session (Docherty and Sporer 2000), so informed planning is required. Ultimately, the task is to determine what the appropriate requirements are for a particular runner, examine what his/her current fitness parameters are, then target training to optimise these parameters. Such process should result in performance improvements, and reduce the risk of muscle-tendon, skeletal, cardiovascular, and heat-and fatigue-related injury.

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SESSION 4:

Muscle

EXERCISE ASSOCIATED MUSCLE INJURY IN HORSES

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Exercise associated muscle injury in equines is likely to occur more frequently than is recognised, and may be confused with other conditions that cause lameness or unwillingness to perform. Massive demands of the muscular system in equine performance athletes may lead to muscle strain or tears, or may provoke exertional rhabdomyolysis, especially in the unprepared animal.

THE MUSCULAR SYSTEM OF THE HORSE

Horses are capable of higher speeds than comparably athletic species including greyhounds and human sprinters. In part this ability is due to their greater muscle mass, which comprises approximately 45% of bodyweight in most breeds, and up to 55% of bodyweight in Thoroughbreds. Horses have a limited number of slow-twitch muscle fibres, usually a maximum of 40% even in endurance trained animals, whereas human endurance athletes may have up to 75% slow-twitch fibres. Thoroughbred horses can attain speeds in excess of 65 km/h. Other adaptations of the muscular system that contribute to this ability include the proximal placement of the locomotor muscles of the limbs which decreases the energy required to swing the limb forward, and the strategic insertion of muscle fibres into their tendinous junctions at an angle to the direction of the force in many muscles which maximises power output by the musculature.

POST EXERCISE MUSCLE SORENESS AND MUSCLE STRAIN

Several factors may contribute to the occurrence of post exercise muscle soreness or muscle strain, including insufficient training, inadequate warm-up procedures, pre-existing lameness, exercising to fatigue, and the performance of novel activities to which the horse is not accustomed. Commonly affected muscle groups are the lumbar and gluteal muscle groups in jumpers, harness horses and dressage horses. In the forelimb the biceps brachii, brachiocephalicus, pectoral muscles and musculotendinous insertion of the superficial digital flexor are common sites of injury. The adductor, gluteal, gastrocnemius and semimembranosus/tendinosus muscles can be affected in the hindlimb. Signs of muscle strain are often mild and include subtle lameness, pain on palpation of the affected muscle group and unwillingness to engage the hindquarters appropriately (Valberg 1996). In horses used for barrel racing, reining and similar activities the semitendinosus muscle is frequently damaged. The affected muscle may be warm, painful and swollen with acute injuries. With recurrent injury, fibrosis and ossification can result in a muscle that is hard on palpation, and mechanical lameness represented by a shortened anterior phase of the stride. Adhesions to surrounding musculature (biceps femoris, semimembranosus) can occur. Radiographs and/or ultrasonography may detect ossification of muscle tissue. Chronic inflammation or trauma of the biceps brachii and bicipital bursa can lead to a similar syndrome in the forelimb, and affected animals have a shortened stride and land on the toe, which may lead to a mistaken diagnosis of navicular disease Turner 1987. Circulatory disturbances can also lead to perturbations of muscle function during exercise. Aorto-iliac thrombosis can result in transient lameness or weakness of the hindlimbs, which often recurs during hard exertion.

The diagnosis of muscle strain can be difficult since clinical signs are often subtle, and localising

the source of the pain may prove challenging. Physical examination should focus particularly on muscle symmetry, response to palpation and whether heat or swelling is detectable in the musculature. Serum muscle enzyme activity (CK, AST) may be normal to mildly increased. Ultrasonography and thermography can be performed to detect inflammation and damage using the contralateral limb for comparison.

Treatment of muscle strain requires rest, anti-inflammatory treatment and physiotherapy. Horses should undergo a gradual return to exercise with a thorough gentle warm-up routine. In most cases uncomplicated acute muscle strain has a good prognosis. Chronic recurrent injuries carry a poorer prognosis. Tenectomy may be indicated in severe cases of fibrotic myopathy, but is not a useful modality to treat calcification of the biceps brachii tendon (Turner 1987).

Muscle repair following injury depends on the severity and nature of the injury. Damage to the basement membrane from severe trauma usually results in scar tissue formation, whereas exertional rhabdomyolysis usually spares the basement membrane and permits complete muscle regeneration in most cases.

EXERTIONAL RHABDOMYOLYSIS

Exertional rhabdomyolysis (ER) is a syndrome of exercise-associated muscle cell necrosis, and is the most prominent exercise-associated muscle disorder in equines. The condition may be sporadic or chronic in nature, and affects a wide variety of equine athletes, including racing thoroughbreds, endurance horses, polo ponies and Quarter horses.

Clinical signs and diagnosis

Stiffness, excessive sweating and reluctance to continue exercise are common signs, and commence shortly after the onset of exercise, or may be observed once exercise ceases. Palpation of the lumbar and gluteal musculature frequently reveals firm and painful muscles, and in severe cases horses may refuse to move or become recumbent, with concurrent myoglobinuria. In endurance horses there may also be indications of profound electrolyte derangements, including synchronous diaphragmatic flutter. The severity of episodes of ER varies extensively between individuals and to some degree within the same

individual. A diagnosis of ER requires establishing that serum CK and AST activity are elevated when clinical signs of muscle stiffness are present. Elevations in CK and AST activity can also be present in asymptomatic horses (Valberg *et al.* 1997).

Differential diagnoses that should be considered include colic, acute pleuritis, laminitis, aorto-iliac thrombosis, tetanus, lactation tetany, ionophore toxicity and exhaustion.

A etiology and pathophysiology of exertional rhabdomyolysis

The long-standing myth that ER is attributable to lactic acidosis occurring during exercise following rest and consumption of a high carbohydrate diet has been dispelled. Most horses develop clinical signs of ER during submaximal exercise, and there is no correlation between muscle and plasma lactate concentrations and the occurrence of ER. Sporadic episodes of rhabdomyolysis can occur in horses that are exerted beyond their current level of conditioning or in those that undergo exhaustive exercise. In some cases horses may have a higher incidence of ER following respiratory infections, and therefore should be rested if they display a fever and symptoms of respiratory disease. In the last 15 years it has become apparent that recurrent episodes of ER are frequently the result of heritable abnormalities of muscle function.

Polysaccharide Storage Myopathy (PSSM) predominantly affects Quarter horses, and has also been identified in other breeds including Paints, Appaloosas, and Morgan horses. Horses afflicted with PSSM may show moderate to severe signs of ER shortly after exercise commences, and frequently have a triggering factor evident in their history, such as a period of unaccustomed stall confinement prior to the episode of ER. Serum CK and AST activity are frequently increased, and often remain so even after weeks of stall rest. PSSM is characterised by high muscle glycogen concentrations (up to 4-fold normal) and the presence of abnormal polysaccharide complexes in 1–40% of skeletal muscle fibres. Affected horses also have enhanced sensitivity of muscle tissue to insulin, resulting in an increased rate of blood glucose clearance. PSSM can be managed successfully by enforcing a daily exercise routine combined with a drastic reduction in dietary carbohydrate (<10% daily digestible energy

intake) (Valberg e*t al.* 1997; Mckenzie *et al.* 2002).

Recurrent Exertional Rhabdomyolysis (RER) commonly afflicts Thoroughbred horses, and possibly also Standardbred and Arabian horses. In Thoroughbreds, RER represents a heritable defect in intracellular calcium regulation resulting in excessive muscular contraction and necrosis with exercise. There is age and gender predilection with 2-year-old fillies in race training at the track being heavily represented (MacLeay *et al.* 1999). Clinical episodes of rhabdomyolysis are often stress-induced, tend to occur during training rather than racing, and may become more frequent as training progresses. Elevations in muscle enzyme activity tend to be intermittent, and unlike PSSM, there are no characteristic findings on muscle biopsy other than evidence of muscle cell necrosis and regeneration. RER can be managed by substituting dietary carbohydrate with fat, undertaking measures to reduce stress and anxiety, introducing a regular exercise routine, and in appropriate cases, by the administration of dantrolene sodium (McKenzie *et al.* 2002; McKenzie *et al.* 2003*;* McKenzie *et al.* 2004.

Diagnosis of ER

Horses afflicted with chronic ER may be diagnosed through exercise response testing, when serum CK/AST activity is measured 4–6 h following 15 min of submaximal exercise. Muscle biopsy of the middle gluteal using a Bergstrom needle, or open surgical biopsy of the semitendinosus/membranosus can be performed, and is particularly useful in the diagnosis of PSSM, although affected horses may not show characteristic amylase resistant polysaccharide inclusions until they surpass one year of age. Where it is possible that nutritional factors or electrolyte derangements may be contributing to episodes of ER, urinary fractional excretion of electrolytes can be calculated to determine potential deficiencies and blood selenium and plasma vitamin E concentrations should be determined (Valverg 1996; McKenzie *et al.* 2002.

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MUSCLE INJURY IN THE HUMAN ATHLETE

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INTRODUCTION

Skeletal muscle (SM) accounts for 40–45% of total body weight in humans. The main role of SM is to provide locomotion through its tendinous attachments to the axial skeleton. Secondarily, SM acts to protect the deeper underlying bones, soft tissues and organs. Muscular development is a primary response to repetitive exercise. While providing the means for athletic performance, failure or injury to skeletal muscle is the most common reason for athletes to be sidelined from participation.

RESPONSE TO CONDITIONING

Skeletal muscle, like most human tissues, adapts and changes with repetitive loading. Exercise training results in changes to functional capacity, structure, and volume of skeletal muscle. On a cellular level conditioning of muscles leads to an increase in the ratio of capillaries to muscle fibre, a rise in myoglobin content, and a more efficient production of ATP by muscle cell mitochondria. Exercise trained muscles are therefore better equipped to extract oxygen for use during physical activity (Holloszy and Coyle 1984). Certain types of exercises tend to affect SM differently. For example, endurance of SM is increased primarily through low-intensity, high repetition exercises. This type of activity involves contractions of moderate force of large muscle groups for relatively long time periods. Jogging is a classic endurance building exercise. Alternatively, high-tension, low repetition training exercises such as weight-lifting produce gains in volume (muscle fibre size) and overall strength of SM.

INJURIES TO SKELETAL MUSCLE

In athletics, SM is susceptible to injury from a variety of mechanisms. Extrinsic forces such as those that occur from collisions with obstacles, other athletes, or equipment often result in muscle injury. SM is also injured when its inherent strength is exceeded by tensile forces.

Contusion

Muscular contusion results from blunt force trauma as energy is transferred to skeletal muscle from another object. (like those that occur from player to player contact in collision sports). As a result the muscle sustains partial rupture or disruption of the muscle fibres. Haematoma formation occurs secondary to compromise of the local capillary network at the site of injury. Vascular leakage into a closed space results in blood collection as well as subsequent loss of motion and pain. The quadriceps musculature is a common site for such an injury. Severity of the injury is judged based on the loss of function of the involved muscle. Mild injuries have little in the way of functional impairment. Moderate injuries are indicated by ≥25% loss of motion, and severe injuries result in >50% loss of motion of the affected area. (Ex: Quadriceps contusion; Mild — knee flexion normal, Moderate — knee flexion to >90 degrees, Severe – <90 degrees of knee flexion obtainable).

Severe muscle contusions can have devastating complications. Large haematoma formation within the confines of investing muscle fascia can produce an acute compartment syndrome compromising both vascular and neurologic structures and risking viability of the

extremity. Rapid diagnosis (elevated compartment pressures on manometry) and emergency fasciotomy are necessary to restore blood flow to ischaemic tissues and preserve function.

A relatively late potential complication of a muscular haematoma is the development of heterotopic bone within the muscle, so called myositis ossificans (MO). Bone formation can be visualised on radiographs around 2–4 weeks post injury. MO is more common with severe contusions and may result in restricted motion of affected area. Because aggressive stretching and the use of therapeutic ultrasound can potentially increase the risk of MO, these are to be avoided in the treatment of muscular contusions.

Strain

Sports that require quick bursts of speed such as football, basketball, track and soccer report the highest incidence rates for muscular strains. Certain muscles are also at higher risk of strain injury. Muscles that cross 2 joints, such as the hamstrings and gastrocnemius, are notorious for their propensity to be strained. Muscles can suffer a strain injury in an acute or chronic manner. Acute strains occur when a muscle experiences a single excessive force. Chronic strains result from repetitive overuse of a muscle or an occurrence of sustained over activity. In both acute and chronic strains the underlying problem is eccentric overload of the muscle-tendon unit.

The majority of muscular strains occur at some point along the myotendinous junction. This is thought to be the weakest area of the muscle-tendon unit (Garrett *et al.* 1988). Strain injuries resemble that of contusions in that haemorrhage, oedema, and inflammation are likely at the site of injury. Additionally, grading of a strain injury follows the same scheme, with injuries being classified as mild (minimal strength or motion loss), moderate (clear loss of strength or motion), or severe (total lack of muscle function) (Kujala *et al.* 1997). Healing of muscular strains involves the formation of muscular fibrosis at the place of injury. This area of healing scar tissue and resulting underlying muscle imbalance may be to blame for the presence of persistent symptoms and significant rate of recurrent injury (Croisier *et al.* 2002).

The RICE (Rest, Ice, Compression, Elevation) principles of treatment are applicable for both strains and contusions. These general techniques help limit pain, haemorrhage, and oedema. Therapeutic exercise with progression from isometric to isotonic to isokinetic activities may be followed by eccentric strength training in the late phase of rehabilitation. Return to full athletic activities is granted when the athlete possesses full strength and range of motion of the affected muscle.

Delayed onset muscle soreness

Delayed onset muscle soreness (DOMS) is characterised by the development of muscular pain after unaccustomed vigorous exercise. Symptoms may occur hours or even days after onset of inciting activity. Heavy bouts of eccentric exercise can cause muscle fibre injury leading to an inflammatory response and subsequent symptoms associated with DOMS. Symptoms are usually self limiting and resolve in 2–3 days. Application of ice and rest may help to reduce symptoms and prevent further injury.

Chronic exertional compartment syndrome

Chronic exertional compartment syndrome (CECS) is a condition that typically involves the lower extremities. It is characterised by reproducible pain (and occasionally parasthesias) brought on by physical activity. In the exercising individual, increased blood flow to muscles in the constrained fascial compartments of the lower leg can result in elevated compartment pressure causing muscle ischemia, swelling and pain. CECS is commonly encountered in runners who often are able to recall exact distances when symptoms commence. Symptoms usually resolve gradually after cessation of activity.

Diagnosis of CECS is can usually be made based on history alone. Measurement of intra-compartment pressures by manometry during rest and with exertion can be used to confirm the diagnosis. Conservative treatment is often unsatisfactory with most cases requiring fasciotomy for resolution of exertional symptoms.

IMAGING TECHNIQUES IN MUSCLE INJURY

Plain radiographs may be helpful in diagnosing or confirming muscular injury. They are particularly useful in identifying avulsion fractures of the musculo-tendon complex from its origin. Soft tissue swelling may also be visualised. The

formation and extent of ectopic bone as occurs with MO following muscular haematoma can also be gauged via plain radiography.

Magnetic resonance imaging (MRI), however, has become the favoured imaging technique for evaluating muscular injuries. MRI is superior to plain radiographs for identifying the site and extent of muscular injury as well as determining associated injuries. For these reasons MRI may also be helpful in the prognosis recovery time. One such study evaluated days lost from competition in Australian Rules football players with hamstring injuries and corresponding MRI findings. In this study the percentage of abnormal muscle area and volume of muscle affected as visualised on MRI showed fairly strong correlation with lost time from athletic participation (Slavotinek *et al.* 2002).

PREVENTION OF MUSCULAR INJURY

'Warming up' and stretching exercises are 2 techniques often employed by athletes to help reduce the risk of muscular injury. These prophylactic measures are thought to decrease muscle tension and increase elasticity thereby reducing chances of injury. Unfortunately, the scientific literature fails to provide sufficient evidence to conclude that either of these activities alone reduces the risk of injury (Thacker *et al.* 2004). There is, however, some evidence to suggest that a conditioning programme employing stretching, warm up, in combination with strength, proprioceptive, or plyometric training can enhance performance and reduce certain types of injury (Thacker *et al.* 2004). In addition, specific exercises involving eccentric muscle training have been shown to increase eccentric muscle strength (Mjolsnes *et al.* 2003). This type of training may prove to be beneficial in preventing muscular injury in the future.

SUMMARY

Muscular injury in athletes is a common entity and a significant source of lost time from athletic activity. Rehabilitation from such injuries may be fraught with prolonged duration of symptoms and re-injury. For the athlete there is a struggle between allowing sufficient healing and returning to activity. Balancing these competing forces and choosing an appropriate time frame for resumption of sport is often difficult. Additional research evaluating mechanisms of injury, rehabilitation, and prevention of muscular injuries is needed to help in this decision process.

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SESSION 5:

Biomechanics (different surfaces)

MECHANICAL FACTORS IN MUSCULOSKELETAL INJURY

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Many racehorse injuries are attributable to the loads imposed on the distal limb tissues by high speed exercise. In most cases these are the result of repeated loading over time rather than a single catastrophic event. As a horse goes faster the amount of energy dissipated at foot impact, the loading rate on the bones and tendons, and the peak force experienced by the bones and tendons all increase. These all have the potential to cause damage to bone, tendon and cartilage which will accumulate as a function of the number of loading cycles, the stimulus magnitude and the loading rate. Considering each in turn:

IMPACT

The foot of a galloping horse hits the ground at about 5ms^{-1}. This energy is dissipated by the foot sliding through and penetrating into the surface and by muscles within the limb. The forces at impact are lower than those experienced in the rest of the stride but the load is applied very quickly which appears to be important in eliciting musculoskeletal injury. The impact 'shock' also causes the leg to vibrate at about 30 Hz, somewhat like plucking a violin string. This vibration is predominantly in a horizontal direction and is due to the large amount of elastic tendon tissue contained in the equine limb and the pogo stick like lever system. Vibration is apparent in both ground reaction force and the kinematic data recorded from both front and hind limbs but the vibration amplitude and duration is greater in the front limb. The vibration may, in itself, cause damage through increasing loading rate and the number of loading cycles experienced by the tendons. It will also have a potent remodelling stimulus on bone and perhaps other tissues.

It is possible, using appropriate computer models, to simulate the vibration and investigate the surface properties that are critical in damping leg vibration. This topic is discussed in more detail in Wilson *et al.* (2001). The magnitude of the vibration is dependent on several factors: Surfaces that allow the foot to slip at impact (or on hard surfaces a shoe that slips at contact) will dissipate the impact energy and reduce the impulse that causes the vibration. Surfaces with good damping characteristics will absorb the vibration energy rapidly. This feature is different from the stiffness of a surface and it is possible to have a surface that is relatively stiff (firm) but with good damping characteristics. The other important damping system is the muscles. Active muscles will absorb energy during very small amplitude high frequency oscillations. This phenomenon is also demonstrated in mathematical models of individual cross bridges (the basic contractile element of muscle). These models predict that muscles are excellent at absorbing the energy associated with oscillation around 50–200 Hz. The digital flexor muscles have extremely short fibres and a large physiological cross sectional area which means that they can develop high forces but only over a length change of a few millimetres. This arrangement makes then ideal for absorbing the energy associated with the small amplitude vibration.

PEAK FORCE

At top speed a race horse's front foot is only on the ground for about 80 ms or 19% of the stride and the limb will experience a peak force of about 2.5 body weights. This load is applied in only 40 ms. The load on the hind limb is considerably lower at

about 1.5 body weights. This difference in peak load may explain, in part, why the hind limb suffers a lower incidence of tendon and ligament injuries. The tissues of the distal limb function within a narrow safety margin, this is partly the result of minimising distal limb mass but also a pre-requisite of having tendons that function as elastic energy stores (because energy stored is a direct function of the elongation of a tendon).

The force acting through the leg is multiplied by the lever system of the distal limb to impose much higher forces on the bones and tendons of the distal limb. These forces approach the mechanical capacity of these tissues and a small increase in peak load will result in a substantial reduction in the number of cycles to failure for the tissue. The relative load distribution between the individual flexor tendons will also vary by, for example, the angle of the foot to the ground (due to either shoe design or the way the foot penetrates into the surface). For instance, elevation of the heel results in a transfer of load from the deep digital flexor tendon to the superficial digital flexor tendon and the suspensory ligament.

NUMBER OF LOADING CYCLES

Large volumes of high speed exercise may exceed the fatigue life of the high stress tendons and bones. The fatigue life of these tissues (in number of cycles to failure) depends on the peak strain (deformation) experienced and a small increment in strain will result in a substantial reduction in the fatigue life of a tissue. The volume of high speed exercise undertaken by a horse is therefore critical. High speed exercise is also the most potent stimulus for eliciting hypertrophy so there is a trade off between providing this stimulus and exceeding the mechanical capacity of these tissues. A galloping horse will impose about 220 loading cycles per mile (strides) on its bones and tendons at fast gallop and about 360 strides per mile at canter. The fatigue life of these structures is limited and estimates of as little as 10,000 cycles (strides) to failure have been made (these are however approximate and could vary widely between individuals. The relationship between speed and number of cycles to failure is of interest in the development of training programmes that elicit hypertrophy and hence a greater mechanical capacity (greater strength is not necessarily the goal). The timecourse of these adaptations may be as long as 6 months and there is the potential for a

training programme to accumulate damage (due to the exercise volume) before such protective responses occur.

CONCLUSION

The musculoskeletal tissues cannot be considered as simple engineering structures since they will respond to the loads placed upon them. This response is critical and the interface between the mechanical environment for the tissues and how they respond to that environment is critical in understanding whether mechanical stimuli of training result in adaptive hypertrophy and stronger tissues or other potentially deleterious responses. It is therefore possible to use experimental measurement and computer modelling to evaluate the load experienced by the musculoskeletal system of the racehorse and make predictions about the time course and mechanisms of injury. Integration of these data with knowledge of the time course of bone and tendon adaptation is however crucial.

ACKNOWLEDGEMENTS

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THE INFLUENCE OF RUNNING SURFACE PROPERTIES ON LOWER LIMB INJURY IN HUMAN ATHLETES

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INTRODUCTION

Injuries sustained during locomotion in human athletes are very common. Indeed, epidemiological studies of runners of varying ability have estimated that as many as 70% can succumb to an overuse injury in any one-year period (Hreljac 2004). These injuries have a significant impact on athlete development and medical/rehabilitation resources, so it is vital that the factors affecting injury incidence are determined so effective preventative strategies can be developed. There is some evidence that injury incidence is increased when ground reaction forces (GRF's) at foot-ground contact, or the rate at which the GRF's are applied, are increased (for review see Hreljac *et al.* 2000). Such research is consistent with the theory that running-related injuries are linked to the high impact loads applied when the foot strikes the ground. Nonetheless, much recent evidence has shown that injury rates are associated with anatomical and technique constraints either exclusive of (Van Mechelen *et al.* 1992; Crossley *et al.* 1999; McCrory *et al.* 1999) or in conjunction with (Hreljac *et al.* 2000; Derrick 2004) the magnitude and rate of GRF application. Certainly, factors such as leg touchdown angle, thigh lateral rotation, foot supination, and the magnitude and rate of GRF application are positively correlated, while hamstring flexibility and hip range of motion are negatively related, to injury incidence. Thus, injury incidence is affected by the interaction of numerous factors.

EFFECT OF SURFACE FRICTION

One factor that affects both the movement technique and magnitude/rate of GRF application is the surface on which the movement occurs. With respect to technical aspects of movement and injury, surface friction (or more correctly the friction coefficient of the surface plus the footwear or foot) is a major factor affecting injury when running is not performed in a straight line. While it might appear intuitive that surfaces of low friction would promote slipping and therefore injury, injury rates are usually lower on these surfaces (Nigg and Segesser 1988). This is because the sliding of the foot allows energy dissipation, and consequently a lower velocity of the foot or player, at the point where the friction is great enough to brake the foot. With a lower velocity at braking, there is a smaller velocity change and therefore smaller change in momentum. Given that the contact between the foot and the surface acts as a pivot point about which the foot can roll, having a smaller momentum when the foot stops will minimise the likelihood of the foot rolling and tissues around the ankle being stressed excessively (Fig 1). Also, the rate of application of the GRF is slower when the foot slides compared to when braking occurs instantaneously, again placing the tissues of the leg under less stress during landing. Thus, surfaces of lesser friction are probably better for injury prevention in sports where rapid direction changes are necessary.

EFFECTS OF SURFACE STIFFNESS

There is little debate that the repetitive applications of high impact forces to the lower limb will probably result in injury to bone or soft tissues (Derrick *et al.* 2004). In order to minimise this likelihood, one can either apply fewer impacts, or reduce their magnitude. While it
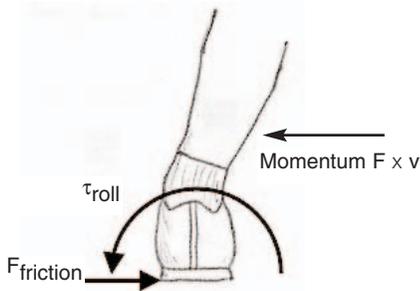
Fig 1: On surfaces of high friction, where there is little or no sliding of the foot before the force of friction ($F_{friction}$) stops it, there is a greater moment of force (or torque causing foot roll, τ_{roll}) at the ankle joint for a given momentum of the foot before braking. On surfaces of lower friction, sliding of the foot reduces its momentum since energy is lost from the system as heat and sound. With the reduced momentum, there is a lesser rotational moment and therefore less chance of injury to the ankle and other structures of the lower limb.
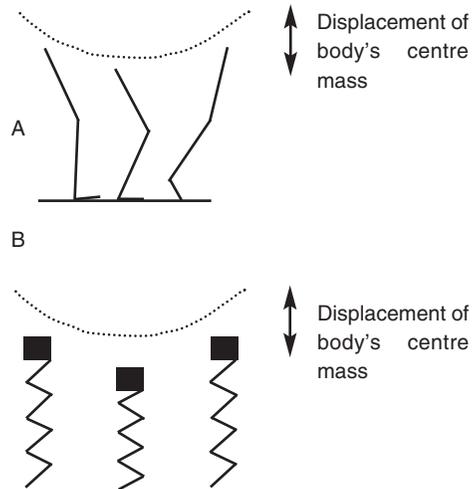

Fig 2: During ground contact (A), the leg first shortens as a result of the ground reaction force opposing the fall of the runner's body during the impact phase, and then lengthens during the propulsion phase. Thus, the leg acts like a spring (B). While some studies have not reported changes in leg kinematics when running on surfaces of different stiffness, there is good evidence that the leg is held straighter at foot-ground contact when we run on stiffer surfaces (Derrick, 2004). Since the centre of mass of the body tends to fall to the same height above ground regardless of the surface stiffness (Ferris et al. 1998), the overall leg shortening is greater on stiffer surfaces while the ground reaction force remains constant. Thus, the leg spring is more compliant when running on stiffer surfaces.

would seem intuitive that running on more compliant (less stiff) surfaces would increase the time over which impact forces are applied and therefore reduce the peak impact force, humans show very consistent GRF magnitudes when running on surfaces, or in shoes, of varying stiffness.

ALTERATION IN LEG KINEMATICS/ STIFFNESS

It is a well-known phenomenon that if a human jumps in place on an increasingly stiffer surface they will lower their own leg stiffness in order for the total stiffness of the surface-body system to remain relatively constant (Farley *et al.* 1998). This is done by allowing a greater descent of the body's centre of mass during the contact phase. A similar phenomenon has been shown when humans run on surfaces (or in shoes) of increasing stiffness (Ferris *et al.* 1998; Derrick *et al.* 2004). Thus, changing the stiffness of the surface does not seem not to dramatically alter GRF magnitude. However, while some research has not shown changes in running kinematics with changes in surface stiffness, there is much evidence to show that runners tend to contact the ground with greater hip and knee angles (and with different rear foot-to-forefoot angles) when running on stiffer surfaces. This provides a more rigid leg at contact, with segments aligned so that connective tissues cannot absorb force during joint rotation, and causes an increase in the rate of application of

the GRF, which could increase the risk of injury (Dixon *et al.* 2000). Because the leg angles at mid-stance are more consistent across surfaces, the overall stiffness of the 'leg spring' is decreased, a consequence of which is that the muscular work would necessarily increase. Such changes increase muscle fatigue, which in turn has been shown to alter muscle activation patterns (causing unusual muscle stress or greater co-contraction) and kinematics and can then also affect loading rates and peak GRF's (Christina *et al.* 2001). So while GRF magnitude does not seem to increase when runners run on stiffer surfaces, it is likely that the technique alterations that maintain GRF magnitude result in increased loading rates and greater muscle fatigue, both of which are likely to contribute to a higher incidence of injury.

COPING WITH VIBRATION

When a runner's foot contacts the ground, large forces are transmitted to the body in the first 30 ms

or so (impact peak). This impact causes vibration to be transmitted through the body, and it is well-known that high frequency vibration within the limb causes fatigue damage to both bone (Carter 1984) and soft tissues such as tendons and ligaments (Wang *et al.* 1995). Given that the soft tissue packages, constituting largely the muscles and their connective tissues and associated fat, have natural oscillation frequencies ranging from about 10–60 Hz, and that impact forces generally have a major frequency component of about 10–20 Hz, one might expect that the soft tissue packages would oscillate significantly given that the impact and tissue natural frequencies are somewhat similar (ie they are in resonance). Despite this, the tissue oscillation is effectively stopped after 2 oscillations, so the human body shows an exceptional ability to dampen vibration during running.

Recent research has provided substantial evidence that humans alter the natural frequency of their soft tissue packages by changing muscle activation (Wakeling *et al.* 2002). More importantly, this change in muscle activity seems to dramatically affect the damping characteristics of the tissues, which is possibly a more important effect than the change in natural oscillation frequency (Wakeling *et al.* 2002, 2003). While changes in muscle activation probably protect the body reasonably effectively from harmful vibrations, the resulting muscle fatigue could conceivably contribute to an increased incidence of running injury, although more research is required to substantiate this. An optimum foot-surface composition would thus be achieved when the major frequency of the impact force is sufficiently different from the natural frequency of the soft tissue packages, so that vibration is minimised without significant alterations in muscle activation. Since there is a high inter-individual variability with respect to the optimum stiffness of a surface in this regard, it is probably more practical to increase the damping of the surface.

CONCLUSION

Although biomechanical and anatomical factors significantly affect injury incidence in human athletes, properties of the running surface can have a marked effect. While in sports requiring changes in running direction running performance might be greater on surfaces of high friction, there is an increased prevalence of injury. When running in a straight line, humans show considerable ability to minimise GRF's and tissue vibration, although the changes in muscle activation, and possibly running mechanics, needed to achieve this may leave the runner susceptible to injury when training volumes are high. Running surfaces can be produced with their natural frequency characteristics differing from those of a runner's soft tissues, however the high inter-individual variability amongst runners makes a 'global' surface difficult to construct (this might also not be ideal from a performance perspective). Importantly, surfaces with high damping probably reduce the high-frequency components of the impact forces and thus reduce the need for muscle activation changes. This is associated with a lower metabolic cost, and therefore with increases in running performance, and is probably important as an injury prevention strategy. Further prospective studies examining injury rates on different surfaces are however required to understand fully the influence of surface properties on overuse injuries.

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SESSION 6:

Thermoregulations

THERMOREGULATORY DEMANDS OF EXERCISE AND EXERTION-ASSOCIATED HEAT ILLNESS IN EQUINE ATHLETES

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The demand for horses to compete in hot environments has focused attention on strategies to prevent potentially life-threatening hyperthermia. The mass-specific maximal oxygen uptake of horses is at least 2-fold higher than in man and therefore, at a given workload, the metabolic heat load is considerably higher. Further, relative to body mass, the surface area available for dissipation of heat in horses is approximately 50% that in man (Hodgson *et al.* 1993; Hodgson *et al.* 1994). On the other hand, the horse's efficient thermoregulatory mechanisms provide for effective transfer of heat from contracting skeletal muscle to the environment. Also, the horse can selectively cool the brain during exercise or heat exposure via cooling of venous blood within the cavernous sinus during respiration (McConaghy *et al.* 1995).

The primary physiological mechanisms driving heat loss during exercise are an increased proportion of cardiac output directed toward the cutaneous circulation and an increased rate of sweat secretion. Sweating and cutaneous evaporation are the most important heat dissipatory mechanisms in horses, accounting for 65–70% of heat loss during prolonged exercise (Hodgson *et al.* 1993; Hodgson *et al.* 1994). Sweating rates of 20–55 g/m²/min have been measured on the necks and backs of exercising horses; for a 500 kg horse these rates correspond to sweat fluid losses of 6–15 litres/h. When expressed in terms of sweating rate per unit area of skin, these rates are 2- to 3-fold greater than those reported for human subjects (McCutcheon and Geor 2000; McCutcheon and Geor 2004).

Unsurprisingly, the thermal responses to exercise are affected by ambient conditions. Under conditions of high heat (>30°C) and humidity evaporative heat loss is severely limited with resultant increases in the rate of heat storage and degree of hyperthermia. In horses, the rate of heat storage when exercising in hot, humid conditions can be more than twice the rate occurring during exercise at the same intensity in cool, dry conditions (Geor *et al.* 1995; Kohn *et al.*1999a). Increased demands for respiratory heat loss are reflected by an increase in respiratory rate during and after exercise (Geor *et al.* 1995; McConaghy *et al.* 2002; McCutcheon and Geor 2004). Further, dehydration associated with profuse sweat fluid losses can further compromise heat transfer and exacerbate hyperthermia (McCutcheon and Geor 2004). An important consequence of this impairment of heat dissipation during exercise in the heat is a decrease in the time to attainment of a critical upper limit in core body temperature that results in development of fatigue (Gonzalez-Alonso *et al.* 1999; Nybo and Nielsen 2001). Moreover, exercise in such conditions increases the risk of developing heat-related illnesses in horses (McCutcheon and Geor 2004). Several factors may contribute to this decrease in performance when exercise is undertaken in hot versus cool conditions, including the effects of hyperthermia on brain and muscle function, compromise of muscle blood flow (McConaghy *et al.* 2002) and reduced aerobic power (Art and Lekeux 1995).

In trained human subjects, exhaustion during exercise in the heat corresponds to a core temperature of about 40°C (Gonzalez-Alonso *et al.* 1999; Nybo and Nielsen 2001). The onset of fatigue at this critical upper limit may represent a mechanism to avoid heat stroke. Measurements of central blood (pulmonary artery) temperature in horses during heavy exercise have demonstrated that fatigue occurs as blood temperature approaches 42.5–43°C. As such, horses may have greater thermal tolerance than man, perhaps in part because the selective brain cooling mechanism maintains hypothalamic temperature approximately 1°C lower than central blood

temperature during exercise (McConaghy *et al.* 1995).

Clinical experience has indicated that poor physical conditioning, prolonged exercise (eg endurance races; speed and endurance test of a 3-day event) in hot environments, lack of heat acclimatisation, and dehydration are factors that may increase the risk of exertional heat illnesses in horses. Horses with a history of anhydrosis are obviously at higher risk for development of exercise-associated heat illnesses.

A concerted international research effort in the 1990's identified several strategies for ensuring the welfare of horses competing in the heat, with emphasis on the 3-day event competition. Both physical conditioning in cool ambient conditions (McCutcheon and Geor 2000; McCutcheon and Geor 2004) and a 10–14 day period of active heat acclimatisation will enhance thermoregulatory ability during exercise in hot conditions (Marlin *et al.* 1999; Geor *et al.* 2000). Adaptations associated with exercise training include decreases in heat production and storage, improved cardiovascular stability during exercise in the heat, a lowering of the threshold temperature for sweating onset, increased sweating sensitivity, and a decrease in sweat sodium concentration (McCutcheon and Geor 2000; McCutcheon and Geor 2004). Following heat acclimation, there is a decrease in resting core temperature and improved thermal tolerance during intense exercise in the heat (Marlin *et al.* 1999; Geor *et al.* 2000). However, it is important to emphasise that these improvements in thermoregulatory ability are modest and largely negated when exercise is performed in uncompensable heat stress conditions (high heat and humidity). Thus, additional strategies are needed to prevent development of excessive hyperthermia during competitions. These include the use of aggressive cooling methods, such as the repeated application of cool water over the body during rest stops and after completion of exercise (Kohn *et al.* 1999b), close clinical monitoring to ensure prompt recognition and treatment of heat stress, and a decrease in event distance and/or speed requirements at high environmental heat loads (Wet Bulb Globe Temperature Index >28–30) (Schroter *et al.* 1996).

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THERMAL STRESS DURING EXERCISE

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INTRODUCTION

While humans can tolerate a large range of environmental temperatures and allow their skin temperature to vary widely, the temperature of the deep tissues must be maintained within only a few degrees of the normal resting level of about 37°C. Therefore, the rate of heat gain by the body must be more-or-less balanced by the rate of heat loss. When this is not the case, body temperature will change. An elevation of body temperature to 41°C or a decrease to 35°C may start to be a cause for concern. Furthermore, both these temperatures are capable of being reached by healthy humans during exercise.

Exercise markedly increases the metabolic rate of an individual. The resting metabolic rate is about 60W but elite athletes can sustain rates of heat production in the order of 1,200W for a little over 2 h, which is the time it takes to complete a marathon. Therefore, to prevent a catastrophic rise in body temperature, the rate of heat loss from the body must be increased to match the increased rate of heat production. Indeed, as exercise does increase heat production, because the majority of exercise situations are not in extreme cold environments and humans can frequently choose what clothing to wear, overheating and heat injuries are more of a concern than hypothermia or cold injuries in terms of sporting injuries. However, both extremes of heat illness may occur in sporting situations.

In order to prevent a catastrophic rise in body temperature during exercise, the rate of heat loss from the body must be increased to match the increased rate of heat production. In the situation of a 65 kg marathon runner, for example, taking the heat capacity of human tissue to be 3.47 kJ/°C/kg a rate of heat production of 1,200W

would cause body temperature to rise by 1°C approximately every 3 min, and the runner would exceed the upper limit of the tolerable core temperature within the first 10–15 min of the race. This clearly does not happen, and core temperature seldom rises above about 40–41°C indicating that the capacity for heat dissipation is high. Evaporation of sweat from the skin surface accounts for the bulk of this heat dissipation in most sporting situations. High rates of evaporation require high rates of sweat secretion onto the skin surface, and the price to be paid for the maintenance of core temperature is a progressive loss of water and electrolytes in sweat.

SWEATING

Sweat rates exceeding 2 l/h can be maintained for many hours by trained and acclimated individuals exercising in warm, humid conditions. This is demonstrated by the body mass losses in marathon runners, which can range from about 1–6% (0.7–4.2 kg of body mass for a 70 kg man) at low (10ºC) ambient temperatures to more than 8% (5.6 kg) in warmer conditions (Maughan and Shirreffs, 1998). With exercise in a warm environment, 30–40% of total body water may be turned over in a single day, but a deficit of even half of that amount will result in serious disability or even death (Adolph *et al.* 1947). When sweating takes place, the free exchange of water among body fluid compartments ensures that the water content of sweat is derived from all compartments, with the distribution being influenced by sweat rate, sweat composition and total water and electrolyte loss.

In healthy humans, sweat is hypotonic with respect to the body fluids. Sodium is the primary cation lost in sweat, with typical concentrations of about 10–80 mmol/l, compared with about 4–8

TABLE 1: Potential effects of heat stress
Heat stress, may cause
Fluid and electrolyte imbalance
Skin blood flow alterations
Thoracic blood volume changes
Cardiac filling alterations
Stroke volume changes
Rapid pulse
Headache
Nausea and vomiting
Dizziness
Cramps
Shortness of breath
Dependent oedema
Orthostatic hypotension

TABLE 2: Risk factors for heat illness
Risk factors for heat illness
Pre-pubescent age
Obesity
Lack of fitness
Dehydration
Lack of heat acclimatisation
Prior history of heat illness
Sleep deprivation
Some medications
Stimulants
Alcohol consumption
Sweat gland dysfunction
Sunburn

mmol/l for potassium (Maughan and Shirreffs 1998). Given the higher sodium loss and the distribution of these cations between the body water compartments, the primary water loss is likely to be from the extracellular space.

EXERCISE IN A WARM ENVIRONMENT

It is well documented that even small body water deficits incurred before (Armstrong *et al.* 1985; Sawka 1992) or during (Cheuvront *et al.* 2003) exercise can significantly impair aerobic exercise performance, especially in the heat (Sawka 1992; Cheuvront *et al.* 2003). This, therefore, is an issue during exercise in a warm environment when high sweat rates can very easily and quickly lead to significant dehydration developing. Armstrong *et al.* (1985) studied participants in track races over 1,500, 5,000 and 10,000 m after reducing body mass by 2% using a diuretic. The time to complete these races was increased by 0.16, 1.31 and 2.62 min (3.4, 6.7 and 6.7%), respectively, relative to their finishing time when euhydrated. The plasma volume decrease that accompanies dehydration may be of particular importance in influencing work capacity; blood flow to the muscles must be maintained at a high level during exercise to supply oxygen and substrates, but a high blood flow to the skin is also necessary to transfer heat to the body surface where it can be dissipated. Hypohydration is associated with higher cardiovascular strain and impaired thermoregulation and with loss of the protection conferred by acclimation (Sawka 1992).

Furthermore, exercise capacity and exercise performance are generally reduced in a warm environment in comparison to the same exercise in a cooler environment (Galloway and Maughan 1997; Parkin *et al.* 1999). Accompanying these performance effects, rating of perceived exertion, sweat rate, heart rate and core temperature are higher in the warm environment. It appears that the majority of this effect is due to the effect of dehydration as described above, but Gonzalez-Alonso *et al.* (1997) have reported that moderate hyperthermia without dehydration has an impact on the physiological responses to exercise; heart rate is increased and stroke volume slightly reduced.

It has been proposed that exercise is stopped when the core temperature reaches a set and consistent value (Nielsen *et al.* 1993). This has also been shown in studies in which core temperature is slightly reduced prior to exercise by water immersion.

The mechanisms where by exercise capacity is reduced have been widely discussed. The current research indicates that dehydration, because of effects on the circulation, makes hyperthermia more difficult to cope with (Gonzalez-Alonso *et al.* 1997) and that hyperthermia per se, rather than the altered metabolism it causes, is the main factor in early fatigue (Gonzalez-Alonso *et al.* 1999). However, the precise mechanism whereby early fatigue occurs with exercise in the heat has not been established. A series of studies by Nybo and Nielsen (2003) have demonstrated that hyperthermia results in impaired maximal muscle activation, altered brain activity and increased perceived exertion. Whether or not neurobiological mechanisms are behind these findings is unclear. However, that fact that there is increased prolactin release after exercise in the heat provides indirect evidence (Pitsiladis *et al.*

2002). It is also known that serotonin and dopamine are involved in thermoregulation.

HEAT STRESS AND THE HEAT ILLNESSES

Heat stress has been reported to cause a number of effects in humans as summarised in Table 1. In addition, a number of factors have been identified as risk factors for heat illness (Table 2).

There are a number of classifications of heat illness in the published literature.

Heat cramps

Heat cramps present as painful muscle spasms of the arms, legs, abdomen, truck or all of these. They are often seen in situations of poor heat acclimatisation or with excessive sweat sodium losses.

Heat syncope

Heat syncope is fainting and loss of consciousness. It often occurs with prolonged standing in the heat but individuals usually recover very quickly upon taking a supine position. Indeed in humans it is frequently prevented when pre- or near syncopal symptoms are identified and a seated or supine position adopted.

Heat exhaustion

The signs and symptoms of heat exhaustion are fatigue and an inability to continue exercise, mild confusion, nausea, vomiting, syncope, 'chills' of head and neck and an increased core temperature (up to 40.5ºC).

Heat stroke

Heat stroke is the most severe of the heat illnesses. Its signs and symptoms are pronounced mental status changes, fatigue, nausea, vomiting, syncope, an increased core temperature (above 40.5ºC), hypotension, tachycardia, possible sweating cessation, potentially leading on to coma, acute renal failure and death.

Heat stroke fatalities

Clear and extensive data on the extent of the occurance of heat illness in sporting situations is not generally widely available. However, one example where good records are available are heat stroke fatalities in American Football. Over the past 10 years, from 1993 to 2003, the National Center for Sports Injuries (Muller and Diehl 2003) has reported more than 20 heat stroke fatalities in the US associated with American Football. Indeed over the period of 1959 to 2003, there has been on average 2 deaths due to heat stroke per year.

REDUCING THE RISK OF HEAT INJURIES

In recent years a number of groups and organisations have addressed the issue of reducing the risk of sporting heat injuries. One detailed publication by the National Athletic Trainers Association (Binkley *et al.* 2002) includes a prevention plan (Table 3).

SUMMARY AND CONCLUSIONS

Humans are inefficient 'machines' and as such produce a lot of heat during exercise. If the exercise takes place in a warm environment this can add an additional heat load to the individual. There is a substantial body of evidence demonstrating that endurance exercise performance is reduced in the heat and this seems to be via a number of physiological mechanisms.

TABLE 3: Summary of the National Athletic Trainers Association prevention plan for exertional heat illnesses

Medical care givers - available and knowledgeable about heat illness
Pre-participation medical
Heat acclimatisation
Education of coaches/athletes to recognise heat illness
Education on hydration issues
Encourage appropriate rest and nutrition
Have guidelines in place
Check environmental conditions
Plan rest breaks in training and after meals
Provide appropriate fluids
Weigh high-risk athletes
Minimise clothing when appropriate
Minimise/modify warm-up
Use shaded areas, and fans if possible
Have thermometers, cooling baths etc available
Inform local hospital of mass participation events
Check hydration status at weigh-in for weight category athletes

Although heat illness can be common, and severe heat illness is life threatening, its threat can and should be minimised.

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SESSION 7:

Bone

SPORTS INJURIES: THE EQUINE SKELETON

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Musculoskeletal injuries are the major cause of wastage in equine athletes; in a recent study of 2 and 3-year-old Thoroughbreds in flat race training in the UK 'we' found them to be responsible for approximately 50% of total days lost. In contrast, less than 10% of days lost from training were the consequence of medical conditions. In these young Thoroughbreds, fractures, suspected fractures, dorsal metacarpal disease and joint disease (particularly of the carpus and metacarophalyngeal joint) are the major cause of days lost. These are overuse injuries that occur because bone is insufficiently robust to withstand the loads engendered by training and racing. This implies that many training regimens fail to 'induce' the adaptive responses in bone necessary to match the bones' strength to the loads applied to them. Establishing the characteristics of what distinguishes an osteogenic from a potentially harmful training regimen remains a major challenge.

It is well established that bone's ability to withstand injury is achieved as a result of an adaptive response in which load-bearing regulates bone cell function to achieve and maintain an appropriately robust skeletal mass and architecture (for review see Lanyon 1992). Like any other structure, when a bone is loaded it changes its shape. The extent of this deformation can be measured on the bone's surface *in vivo*. Several studies, many undertaken several years ago, showed that the peak strains in adult cortical bone during loading are similar in different species and at different locations. For example, in the galloping adult horse surface strains recorded have ranged from 3,000 to 3,317 micro strain (Rubin 1984; Nunamaker *et al.* 1990). This led to the hypothesis that bones have a 'target strain environment' (Lanyon 1992) and that in order to

maintain this an increased strain related stimulus (eg high speed exercise) will increase bones mass and architecture whereas a decreased strain related stimulus leads to bone resorption and decreased mass.

If a bone is exposed to high and/or unusual strain distributions before its mass and architecture has had sufficient time to adapt appropriately, microdamage will result. It is fatigue damage and bone cells' response in trying to repair it that underlies the pathogenesis of most equine bone injuries. Microdamage stimulates repair, the initial process of which is the removel of damaged tissue by remodelling. This decreases bone stiffness and strength still further. However, provided the bone is not loaded excessively at this stage the repair process will be completed. However, if the horse continues to be trained hard then microdamage will accumulate faster than it can be repaired and this leads to stress facture (Riggs 2002). Stress fractures typically occur in the absence of any traumatic event and at consistent locations (scapula, humerus, third metacarpal, first phalanx, pelvis, tibia etc). Verheyen and Wood (2004) showed recently that the majority of these injuries occur while horses are in training rather than during racing. Catastrophic fractures then occur if overloading continues preventing repair of this fatigue damage. Post mortem studies have shown that previous pathology can often be identified at the site of catastrophic fractures, lending support to the idea that most fractures represent accumulated microdamage (Stover *et al.* 1992; Riggs 2002). Fortunately the incidence of catastrophic fractures has decreased in recent years because improved diagnostic techniques such as nuclear scintigraphy mean that stress fractures are identified more frequently. Notwithstanding, a significant

proportion of fractures have a fatal outcome and this continues to raise public concern regarding the welfare of equine sporting activities.

Dorsal metacarpal disease (DMD or 'sore shins') is a less severe manifestation of fatigue damage that is very common in 2-year-old racehorses in their first year of training. These horses are the equivalent age to human teenagers. Clinically, DMD is associated with heat, pain, swelling and tenderness over the surface of the third metacarpal bone (MCIII) and animals are often bilaterally lame. David Nunamaker conducted a series of *in vitro* and experiments some years ago which contributed a great deal to understanding of this condition's pathogenesis (for review see Nunamaker 2001). Essentially, DMD reflects an adaptive response involving the rapid formation of plexiform bone on the dorsal surface MCIII in response to the high compressive strains engendered by high speed exercise. Two-year-old racehorses are particularly vulnerable because surface strains engendered by high speed exercise range from -4,400 to 5,5670 (Nunamaker *et al.* 1990). However, because it is a training-related disease DMD it is not restricted to 2-year-olds; it does occur in 3 and 4-year-olds when they first start training.

Exercise induced damage to subchondral bone also plays an important role in the pathogenesis of joint disease and injuries at this site are now recognised to be an important cause of joint pain in horses (Arthur *et al.* 2003). As complex organs, the normal function of joints depends on well balanced interactions between subchondral bone, articular cartilage, synovial membrane, the joint capsule and ligaments. A number of studies have shown that exercise leads to altered density in equine subchondral bone and this can potentially alter the mechanical and/or biochemical environment of overlying articular cartilage (for review see Riggs 2004). Sclerosis of the subchondral bone in MCIII and the third carpal bone may also underlie the pathogenesis of fatigue fracture at these sites.

Clearly a strategy for reducing the incidence of these injuries requires identification of those training regimens which stimulate injury-resistant bone compared to those which may have detrimental effects. In humans it has been shown that sporting activities that engender 'error' strains (eg weight lifting) are osteogenic whereas exercise that involves repeated cycles of relatively low strain (eg endurance sports) are not (Heinonen

1993). In humans subjects it is possible to monitor bone's response to different training regimens *in vivo* using dual energy X-ray absorbiometry (DEXA) or peripheral quantitative computerised tomography (pQCT). Unfortunately, there are no methods currently available for measuring bone mineral density (BMD) reliably in the standing horse, although he authors are about to test a peripheral DEXA device for measuring BMD in the third metacarpal. We are therefore some way from being able to identify training regimens that are 'good' for equine bone. However, progress is being made because this question has been the focus of a number of research studies in recent years. Most of this work is done in racehorses because they provide a large, accessible study population. What is becoming clear is that there is a critical balance between the amount of training undertaken at high speed (gallop, 'work' or 'breezing'), that engenders higher strains and strain rates, and training at slower canter speeds.

David Nunamaker has hypothesised that changes in bone's intertial properties as it adapts to training decrease surface strains and that to engender these changes bone should be exposed during training to strains of the magnitude and direction that it 'sees' during racing. In an intervention study he found that increasing the number of episodes of high speed training produced large changes in modelling, remodelling and inertial properties of MCIII (Nunamaker *et al.* 1990). In a later epidemiological study of horses in training Boston and Nunamaker (2000) found that short periods of high speed work reduced the risk of developing sore shins. An approach that we have taken is to use biochemical markers, which are instantaneous measures of bone cell activity (Price *et al.* 2003), to monitor bone's response to training. This has shown that of all the different components of a racehorse's training regimen fast work appears to have the most significant effect on bone cell activity (unpublished observations). This is consistent with what has been found in experimental studies and in humans.

Age is likely to be a important factor when considering how bone responds to exercise; studies in humans and experimental animals have shown that 'young bones' are more responsive. As discussed previously, surface strains are higher in skeletally immature horses and a number of experimental studies have shown that training of young horses will engender significant osteogenic responses. In a study of treadmill trained 2-year-

old Thoroughbreds it was found that exercise would induce changes in bone marker concentrations very rapidly and this was associated with a significant change in BMD after only 4 months of training (Jackson *et al.* 2003). Bone marker measurements have also revealed that the effect of exercise on bone cell function decreases as animals get older (unpublished observations).

Another strategy for reducing the incidence of injury is to develop methods for identifying horses at increased risk of injury *before* clinical signs develop. The measurement of BMD by DEXA or pQCT is used to predict fracture risk in women and men with osteoporosis. Biochemical markers have also been used to predict fracture in humans, although we have found that they appear to have little value for identifying racehorses that go on to fracture. However, they may be of some value for identifying animals that will develop DMD (Jackson *et al.* 2005). In future it may also be possible to use genetic markers to identify horses a risk of skeletal injury, an approach that has been used in humans to identify individuals at risk of developing osteoporotic fracture.

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BONE INJURIES IN CHILDREN

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Bone injuries in children/adolescent athletes are different to those in adults because of the presence of the growth plate and the inherent 'plasticity' of pediatric bone. Distinct types of fracture that occur in children include bowing ('plastic'), greenstick, torus and Salter-Harris type physeal injuries.

Incomplete fractures, which do not completely cross or break the bone shaft, can be very subtle and involve soft tissue injury (eg swelling, loss of fat planes and deformity); elevation of the posterior fat pad of the elbow (lucency posterior to the distal humerus), for example, is highly suspicious for fracture. In light of this (even if the exact fracture line cannot be definitively demonstrated), the arm should be immobilised and presumptively treated as a fracture. Repeat films can be obtained in 7–10 days to confirm the injury (presence of healing periosteal reaction).

An example of incomplete fracture is the bowing or 'plastic' fracture; a greenstick fracture penetrates only one side of the cortex in addition to the bowing deformity. Its name arises from the analogy of moist spring tree branches that readily bend but do not break. A torus or buckle fracture is a cortical fracture with buckling deformity of the bone. The most common site is the distal forearm (falling on an outstretched arm). Torus fractures are notoriously subtle and difficult to diagnose. Radiographs demonstrate a cortical 'step-off', a sudden curve to the bony cortex and the trabecular 'wrinkle': an irregular trabecular bony pattern.

In growing children, the physeal region (growth plate) is the weakest and most easily injured part of the bone, particularly with shearing forces. Up to 20% of all pediatric fractures relate to the physis and are often sports-related. The most common sites are the wrist and ankle. Physeal injuries are more common in boys than girls (male growth plate persists for longer than in females). These physeal injuries are classified as Salter-Harris type fractures. One example is the 'little Leaguer's elbow' whereby avulsion of the medial epicondyle is associated with pitching too hard, too early in life. The snapping wrist motion of throwing a curveball is the main culprit as the flexor carpi ulnaris tendon pulls off the medial epicondyle. Conventional plain radiograph of the elbow can demonstrate the avulsion with widening of the distance between the epiphysis and the medial humeral condyle. The injury may require surgical pinning and relocation of the epicondyle to ensure proper growth. Occasionally, the avulsed epicondylar fragment becomes a loose body and is entrapped within the joint space itself. An epidemic of this injury in the 1970s and 1980s led to coaches and officials trying to stop adolescent pitchers from throwing curveballs until they reach high school.

In general, the prognosis of growth plate injuries is quite good. Salter-Harris Types I, II and III (injury does not cross the growth plate itself) have excellent prognosis and usually heal with no long term sequelae. However Types IV, V and VI (fracture line crosses or crushes the growth plate) can disrupt the physeal plate with resulting slowed, asymmetric or interrupted growth leading to limb shortening, deformity or angulation.

Most of the above injures are readily seen on conventional radiographs. An exception is the stress fracture - a reactive change in bone to repeated stresses. These microfractures are often present with no visible fracture line. 'Fatigue fracture' occurs in stressed bones with normal metabolism/physiology. In contrast, abnormal bone (rickets, (osteoporosis, genetic bone disease) leads to 'insufficiency fracture' from the weakened state of the bone and injuries can occur with minor trauma).

Stress fractures are commonly seen in adolescent athletes. Common sites include tibia,

femoral neck and foot injuries (such as the base of the metatarsals, navicular and calcaneous). Early in the course of a stress fracture, conventional radiographs are usually not helpful. Delayed radiographs might show subtle sclerosis or periosteal reaction (healing). Far more helpful in the early diagnosis is stress fracture is a bone scan. Nuclear Medicine Bone scan utilises radiopharmaceutical agents to elicit early, positive 'hot spots' in the areas of 'stressed' bone.

The spine is also vulnerable in adolescent athletes. Persistent back pain in a child can be due to a traumatic lesion, even though the child may not recall a specific incident. In particular, adolescent athletes can sustain stress spine injuries from overuse, particularly repetitive trauma. Football, soccer and gymnastics are 3 sports that can stress the pediatric spine via repetitive motion, often causing a focal injury to the *pars interarticularis* (spondylolysis); 90% of pars defects occur at the L5 level with 8% at the L4 level. The exact aetiology is unknown but is presumed to relate to repetitive 'fatigue-like' fracture rather than a single traumatic event. Congenital factors may also play a role as individuals with a hypoplastic pars may be more prone to stress injury. Classic radiographic finding is a lucency through the *pars interarticularis* as seen on oblique radiographs. Lateral radiographs can also indicate the presence of spondylolisthesis or slippage of a vertebral body in patients with bilateral pars defects/injuries.

Spondylolysis, however, can sometimes be difficult to detect on plain radiographs or even on conventional planar bone scans. SPECT (Single photon emitted computed tomography) bone scan in conjunction with conventional bone scans can increase the sensitivity of radionuclide imaging of the pediatric spine. In addition, the advent of multi-detector SPECT cameras equipped with ultra-high resolution collimators has led to an improved image quality with increased detection of subtle spinal lesions such as injuries involving the spinous process or vertebral endplate.

The pediatric bony spine is also more mobile than in adults with normal ligamentous laxity that 'tightens up' as we become adults. The unusually elastic biomechanics of the pediatric spine can allow deformation of the musculoskeletal structures beyond physiological extremes, resulting in direct spinal cord trauma followed by spontaneous reduction of the bony spine. The result is SCIWORA (Spinal Cord Injury Without Radiographic Abnormality). This pediatric phenomenon of closed spinal cord trauma often with significant neurologic sequelae (but without demonstrable bony spine injury) was first described by Pang and Wilberger in 1982.

Although apparently a common aetiology in the child with a spinal cord injury, SCIWORA is rare in adults. The infantile/child bony spinal column is remarkably elastic, stretching up to 5 cm prior to rupture. The spinal cord, however, is much more vulnerable, rupturing with only 5–6 mm of traction. Over 8 years of age, the pediatric spine rapidly progresses to the stiffer and stronger adult spine that greatly reduces the incidence of hypermobility-related spinal cord injuries. But, SCIWORA can still be seen in adolescent athletes, particularly high acceleration injuries that can be seen in football and equestrian accidents.

These patients present with varying degrees of neurological deficit, ranging from transient paresthesias to total paralysis. Corticosteroids have a proven value in spinal cord injury and should be used in patients with persistent neurological deficits, preferably administered within 6 h of the injury. Although conventional radiographs and even CT scans offer no diagnostic help in SCIWORA, MRI can often reveal the direct spinal cord injury on sagittal and axial images. MRI can also be prognostic; a normal appearing MRI of the spine suggests excellent recovery of function independent of presenting symptoms. Conversely, major contusion, haemorrhage or spinal cord transection are associated with permanent cord injury.

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SESSION 8:

Heart

HEART DISEASE IN THE EQUINE ATHLETE

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Although horses are often considered to be the premier athlete amongst mammals, VO_2 relative to body mass of elite racehorses (~200 mls/kg/min) pales into insignificance compared to that of the Etruscan shrew (400 mls/kg/min), or the Pronghorn antelope (300 mls/kg/min). Nevertheless the cardiovascular system of the Thoroughbred has evolved to allow it consume more oxygen per kilogram than most other large mammals. The superiority of the Thoroughbred cardiovascular system rests in a proportionately larger heart and spleen per unit body mass than other large mammals (Poole and Erikson 2004). The equine cardiovascular system is hugely compliant with a heart rate range from 20–240 beats per minute and a splenic red cell reserve able to double packed cell volume and oxygen delivery during maximal exercise (McKeever *et al.* 1993).

ARE THERE DOWNSIDES TO BREEDING AN ANIMAL WITH THE ULTIMATE AEROBIC CARDIOVASCULAR SYSTEM?

It seems increasingly likely that 2,000 years of selective breeding for superior athletic ability has resulted in changes to the equine cardiovascular system that, in addition to endowing improved aerobic capacity, are also responsible for some current problems, including exercise-induced pulmonary haemorrhage, audible murmurs of atrioventricular (AV) valve regurgitation and paroxysmal and sustained atrial fibrillation; the commonest cardiovascular cause of poor performance in the Thoroughbred.

ATRIOVENTRICULAR VALVE REGURGITATION

The prevalence of audible murmurs of AV valve regurgitation varies between 54 (tricuspid valve)

and 21% (mitral valve) in mature National Hunt steeplechasers (Young and Wood 2001). These proportions increase with training in both flat (Young and Wood 1998) and National Hunt horses (Young and Wood 2001). The epidemiological data support work in human athletes suggesting that training-induced eccentric hypertrophy is important in the pathogenesis of atrioventricular valve regurgitation (Pollak *et al.* 1988). Douglas *et al.* (1988) observed an increased prevalence of mitral and tricuspid regurgitation and altered ventricular inflow patterns in human athletes using pulsed Doppler echocardiographic techniques. They concluded that regurgitation at multiple heart valves was common in human athletes and that it did not imply structural valvular abnormalities. It seems likely that a similar, if not more extreme, situation also applies to the athletic horse. It is possible that the very high pulmonary arterial pressures generated during exercise that are responsible for exercise-induced pulmonary haemorrhage (Erickson *et al.* 1990), may also be influential in the development of tricuspid valve incompetence in athletic horses of all breeds and types (Patteson and Cripps 1993; Young 2003).

Although uncommon, severe AV valve disease can progress to cause heart failure in equine athletes. Usually, such failure results from catastrophic mitral regurgitation, often in conjunction with secondary atrial fibrillation. Progression of tricuspid valve disease to cause cardiac failure is extremely rare.

AORTIC INSUFFICIENCY

Aortic insufficiency is most common in middle aged to older horses and is the least common of the murmurs seen in performance horses, although the condition may have a negative impact on performance in some groups of racehorses.

The condition is progressive and is associated with nodular thickening of the valve leaflets. Usually it is detected initially as a low-grade murmur (Grade 1–2/6) in horses >10 years old, and progresses very slowly. In older horses, it rarely affects performance as work expectations usually decrease with age. Aortic insufficiency causes volume overload of the left ventricle resulting in dilatation. Pleasure horses can perform adequately even with severe aortic insufficiency and marked volume overload. However, a dilated ventricle works against increased afterload and has an increased oxygen requirement. In aortic insufficiency, coronary perfusion decreases due to reduced diastolic aortic pressure. As a result, oxygen delivery to the myocardium is compromised. Increased oxygen demand and reduced coronary reserve provide the substrates for ventricular arrhythmias to develop during periods of high cardiac work. Ventricular tachyarrhythmias and fibrillation that result in sudden death rather than poor performance, may be a consequence in older-middle aged horses affected with severe aortic valve regurgitation.

HEART MURMURS AND ATHLETIC PERFORMANCE

In our recent epidemiological study, associations between an audible murmur, or regurgitation detected by colour Doppler echocardiography, and race performance were explored using univariable analyses, followed by the development of multivariable models of the association between performance outcomes and regurgitation for each type of racehorse (chaser/hurdler or bumper).

In race-fit steeplechasers, there was no consistent strong overall association between murmur grade and performance measure, although significant (≥2.5/6) mitral valve murmurs were associated with lower Timeform ratings and mitral regurgitation by colour flow Doppler echocardiography was significantly associated with lower win to run ratios. In race-fit hurdlers, the effects of regurgitation appeared weaker than in steeplechasers; only aortic valve regurgitation was associated with reduced performance. In 145 race-fit flat race horses who ran enough times to attain an official rating, their ratings were not associated with presence or severity of valve regurgitation, or of audible heart murmurs. However, because of a much lower prevalence of valvular regurgitation in the flat dataset, the power of the study was limited. To date we have not demonstrated any effect of tricuspid valve regurgitation on performance in racehorses, despite its very high prevalence.

ATRIAL FIBRILLATION

Atrial fibrillation (AF) is the commonest cardiovascular cause of poor performance in the equine athlete (Young 2003). Affected animals usually present with a history of poor performance at maximal exertion (during finishing or sprinting in racehorses, galloping or hill work in event horses and hunters). Horses engaged in less aerobically challenging activities such as dressage or show-jumping, may be affected with sustained atrial fibrillation, yet still compete at International levels. Although horses can compensate for their sub-optimal diastolic filling and reduced cardiac efficiency by increasing their heart rate at all levels of exercise thus maintaining forward cardiac output, they nevertheless attain a higher than normal, maximum heart rate at a lower exercise intensity and fatigue sooner (Buntenkotter and Deegen 1976; Maier-Bock and Ehrlein 1978). During exercise when AV nodal conduction is facilitated, heart rates in excess of 300 beats per min are not uncommonly encountered in affected horses. Based on our epidemiological studies on UK training yards, the approximate incidence of the sustained form of the dysrhythmia is 1% in National Hunt Thoroughbreds. When large numbers of horses from a mixed population were examined, the prevalence of AF varied between 2.5 and 2.4% (Else and Holmes 1971) and increased with age; an observation that probably explains the difference between the two groups of horses. Else and Holmes (1971) also observed that draught and heavy horses were over-represented in their affected horses, providing further evidence of the importance of left atrial mass in sustaining the re-entrant rhythm. If the abnormal rhythm develops suddenly, during fast work, there is an acute decrease in cardiac output, and affected horses may pull up suddenly, sometimes with ataxia and distress. Immediate thoracic auscultation reveals a rapid chaotic rhythm. Obvious performance decrements are not invariably the case, however, as affected horses may appear to work normally at sub-maximal intensity. The rhythm, once initiated may be sustained, but short-lived paroxysmal atrial fibrillation also occurs during exercise (Holmes *et al.* 1986). Paroxysmal atrial fibrillation resolves in the minutes, hours or days following

exercise and as in humans may be difficult to detect. As a result, atrial fibrillation remains a possible cause for fading during racing or competition when horses are subsequently presented in normal sinus rhythm. The prevalence of paroxysmal atrial fibrillation in horses during racing, training and competition is difficult to assess, since riders or trainers are usually unaware of the condition, unless performance is obviously affected. Both atrial flutter and true atrial fibrillation occur in the horse. The difference in electrocardiographic appearance probably reflects the number of wavelets circulating in the atria. It seems likely that large atrial mass and changing autonomic influence during exercise provide the anatomical and physiological substrates for the re-entrant rhythm to develop and be sustained.

In the majority of horses affected with sustained and paroxysmal atrial fibrillation, there is no evidence of significant underlying cardiac disease. In such cases, when the rhythm is sustained, conversion to normal sinus rhythm using the Vaughan Williams type 1A antiarrhythmic agent, quinidine can usually be achieved. If conversion is successful, prognosis for the horse's return to its previous athletic performance is good. Unlike humans and cats, horses with sustained atrial fibrillation are not at risk of atrial thrombus development and all the attendant risks. In fact, in the absence of primary heart disease lone atrial fibrillation usually carries a good prognosis, even if conversion to normal sinus rhythm is not attempted, or it fails.

EXERCISE-RELATED SUDDEN CARDIAC DEATH

Although equine exercise-related sudden death is relatively rare, estimated from Jockey Club data to be one death per 4,000 race-starts under rules in the UK, the relative risk of sudden death during exercise for a racehorse during competition is considerably higher than the one in 200,000 for comparable human athletes. As exercise-related sudden death accounts for almost 21% of point-to-point fatalities in the UK compared to only 10% of racecourse fatalities, it seems likely that the one death per 4,000 competitive exercise events may underestimate the true prevalence of the condition in the general equine population. The relatively high frequency of sudden death during exercise in horses compared to people is somewhat surprising as there is a much lower overall prevalence of significant cardiovascular disease in

Thoroughbred horses compared to a mixed population of humans. Whilst hypertrophic cardiomyopathy and a variety of other congenital cardiac or soft tissue disorders account for the majority of sudden cardiac death cases (SCD) in comparable human athletes, none of these disease entities are believed to occur in horses. Studies in the 1980s demonstrated that when sudden death occurred during exercise, pulmonary or other internal haemorrhage was the most common lesion found (Brown *et al.* 1998; Gunson *et al.* 1988; Platt 1982). However, a substantial proportion (30–68%) of the sudden death cases had no significant lesions, leading to the suggestion that functional disorders of the heart resulted in acute cardiac failure during exertion and SCD. The reasons for arrhythmogenesis during exercise can be readily speculated upon, but there is sparse data to support the mechanisms that have been proposed. Workers in Japan performed detailed examination of the cardiac conduction system in 5 racehorses that died suddenly during exercise, and demonstrated areas of fibrosis or vascular degeneration close to the sinoatrial node, atrioventricular node and bundle branches (Kiryu *et al.* 1987). In one of these horses, electrocardiography (ECG) revealed ventricular fibrillation in the terminal period after collapse (Kiryu *et al.* 1999). The same degenerative changes were lacking in horses that were examined post mortem with no history of cardiac disease. As a result of these findings, it was suggested that coronary vascular lesions might have caused ischaemia and fibrosis of the His/Purkinje system thus providing a substrate for arrhythmogenesis. Abnormal automaticity of His/Purkinje and myocardial cells is most likely to occur during periods of high sympathetic nervous system stimulation and periods of increased cardiac work and myocardial oxygen demand, such as occur during fast exercise. Fibrosis, ischaemia and rapidly changing autonomic influences also alter myocyte refractory period and cause physiological and physical block to normal cardiac impulse conduction. Variability of myocyte refractory periods, alterations in regional impulse conduction and the presence of circulating catecholamines and/or electrolyte and acid-base derangements provide the physiological substrates for rapid re-entry rhythms. These factors explain the increased likelihood of atrial and ventricular fibrillation developing during exercise in horses, and certainly ventricular ectopy

is not uncommonly observed during the early recovery period after strenuous exercise.

IS THERE AN UPSIDE TO SELECTIVE BREEDING FOR PERFORMANCE?

In contrast to the situation in other companion animal species, selecting horses for performance has left the species almost entirely free of the inherited and acquired degenerative cardiac diseases. The cardiomyopathies and the congenital and acquired myxomatous and dysplastic valve abnormalities recognised relatively commonly in humans and that occur with depressing frequency in many breeds of cat or dog are rarely encountered by the equine practitioner. Inherited dilated cardiomyopathy has not yet been reported in the horse, and only after accidental poisoning of equine feed with ionophores, does the equine ventricle take on the dilated hypocontractile appearance that is so prevalent amongst Dobermann Pinschers and many other large dog breeds. The yard cat, or the horse's rider are both much more likely to suffer premature-onset diastolic heart failure or sudden death due to inherited hypertrophic cardiomyopathy or coronary artery disease, than the horse munching hay in his stable. So in conclusion, breeding the horse for speed has, for the most part, been good for his heart. In fact if only development of the equine lung had kept up with that of the heart and skeletal muscle, then the horse might truly have become invincible.

ACKNOWLEDGEMENTS

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ATHLETICS AND THE HEART

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To appreciate the potential effects of sport upon the human heart, one must consider the many ways in which the heart interfaces with sporting activities. This paper will cover direct cardiac injuries, the more complex issue of sudden death during athletic activity and the adaptation of the normal heart to athletic training.

Direct cardiac injuries usually result from a blunt trauma to the chest overlying the heart. When the inciting injury is small, a local shock wave is transmitted through the chest wall to injure the heart muscle (Maron *et al.* 1995; Link *et al.* 1998). Initially this may produce ventricular fibrillation or ventricular stand-still, resulting in death. These cases are rarely resuscitated from this arrhythmogenic shock, but those who are will have ECG changes consistent with myocardial injury. Occasionally, the heart is 'trapped' in the chest between the sternum in front and the vertebral bodies posteriorly, resulting in rupture of one of the walls of the heart.

The sudden death of an individual during athletic activity can occur in what appear to be healthy, fit young men in the prime of their performance career (Benson *et al.* 1983). In the USA, 1 in 200,000 participants die each academic year. The trigger for sudden death is intense activity (Liberthson 1996) and a profile has emerged of the individuals who die suddenly during sports activity (Table 1). Sudden death is most frequently seen in high-intensity sports such as basketball, American football and track athletics (Maron *et al.* 1996a).

Screening for fitness for sport in the USA is not standardised and, generally, no specific cardiac screening is performed; in fact, 40% of the states do not require screening of athletes for high school athletics at all (Maron *et al.* 1996b). While a thorough history and physical examination would highlight some of the causes of sudden death during sport, a detailed cardiac evaluation, including an echocardiogram, is not cost effective, considering that there are 5 million high school athletes and 500,000 collegiate athletes in the USA.

The heart is a muscle and responds to repetitive training with hypertrophy. In addition, as it becomes more efficient at generating cardiac output, the heart rate slows and the left ventricle dilates. Thus, athletes have a thick, dilated heart with a slow rate. This was initially felt to be a beneficial adaptation and was termed the 'athletic heart' (Raskoff *et al.* 1976).

Physical examination of individuals with the 'athletic heart' reflects these adaptations (Huston *et al.* 1985). They have a strong, slow pulse. The pulse pressure (difference between systolic and diastolic blood pressures) is increased, because there is increased stroke volume, raising the systolic pressure, and prolonged diastole, decreasing the diastolic pressure. The apical impulse is laterally displaced, reflecting the larger cardiac size. A systolic ejection murmur is frequently heard and a diastolic filling sound (S3) is often appreciated. The echocardiogram shows an increased diastolic dimension (>5.4 cm) in the adult and the left ventricular posterior wall in diastole is thick (>14 mm) (Maron 1986). Some

TABLE 1: Athlete

Participates in organised sport
Regular competition
Increased premium on excellence and achievement
Requires vigorous and intense traning in a systematic fashion

TABLE 2: Cardiovascular findings in young competitive athletes with sudden death

Hypertrophic cardiomyopathy	36%
Unexplained increase in cardiac mass	10%
Coronary artery anomalies	24%
Ruptured aortic aneurysm	5%
Aortic valve stenosis	4%
Myocarditis	3%
Myocardial scarring	3%
Dilated cardiomyopathy	3%
ARVD	3%
Mitral Valve Prolapse	2%
Atherosclerotic coronary artery disease	2%
Other congenital heart diseases	1.5%
Long QT syndrome	0.5%
Sarcoidosis	0.5%
Sikle cell trait	0.5%
'Normal'heart	2%

athletes have marked ventricular re-polarisation abnormalities on ECG which, in some cases, persist long after athletic training is discontinued (Serra-Grima *et al.* 2000). Further long term evaluation of athletes is necessary to determine whether this cardiac adaptation, beneficial in the short-term, has any serious effects in later, more sedentary life.

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SESSION 9:

Lung

INJURY TO THE EQUINE RESPIRATORY SYSTEM ASSOCIATED WITH EXERCISE

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BACKGROUND

Of the animal species considered to be 'athletic', the horse has the greatest bodymass. Other animals can run faster, jump higher or demonstrate greater endurance but the horse stands out for its ability to achieve similar levels of performance whilst carrying a human, usually amounting to around an additional 15% of its own bodymass. As a large, athletic quadruped, the horse faces a range of size related constraints and specific problems. These are particularly manifest in the structure and function of the respiratory system and its response to exercise.

The horse has the capacity to increase its minute ventilation from ~50 l/min at rest up to 2,000 l/min during maximal exercise. In the horse the conducting pathway from the external environment to the gas exchange surface is long and ~60% of ventilation at rest comprises deadspace ventilation. Even during maximal exercise the deadspace ventilation may still amount to ~40% of total ventilation. The horse is also an obligate nasal breather and the upper airway contributes around two thirds of the total resistance to airflow at rest, during both inspiration and expiration. During intense exercise ~90% of inspiratory resistance is due to the upper airway, falling to around half on expiration.

In order to overcome the limitation of a stiff chest and high airway resistance and to minimise the energetic costs of ventilation, the horse adopts a strategy of 1:1 respiratory-locomotory coupling during the canter and gallop gaits. As a result of its physical size, the horse has a stiff chest in order to constrain and support the viscera. The thorax is also large, reflecting the relatively large size of the heart (~1% of bodymass in athletic breeds of horse) and lungs (~30 l total capacity in a 500 kg

horse). However, the large lung capacity combined with the large dorso-ventral and caudo-dorsal dimensions of the lung require high pulmonary vascular pressures to ensure adequate perfusion during exercise (mean pressure ~80–140 mmHg). Combined with large swings in intrapleural pressure (~140 cm H_2O), the stresses placed on the walls of the alveolar-capillary membrane, which in the horse are around 1/100th the thickness of a human hair, are extremely high.

It is clear that the respiratory system limits exercise capacity and performance in the trained, fit horse in events where a moderate to high proportion of energy is derived from aerobic metabolism and the exercise intensity is high (above ~75% VO_{2max}). Whilst the skeletal muscles and cardiovascular system demonstrate a clear adaptation to training, the respiratory system of the horse does not. Unloading the respiratory system by supplying oxygen in helium increases maximum oxygen uptake as does increasing the inspired oxygen fraction above 0.21. These observations indicate that the capacity for the cardiovascular system to deliver oxygen, and therefore the muscles to use it, are limited by the capacity and function of the respiratory system.

The horse experiences 3 primary dysfunctions related to the respiratory system during exercise:

- Dynamic upper airway obstruction, the most common manifestations of which are laryngeal hemiplegia and dorsal displacement of the soft palate. Whilst these conditions become apparent during exercise and have a marked effect on exercise capacity and/or performance, it is still unclear what role exercise plays, if any, in the pathogenesis of these conditions.

- Whilst exercise-induced asthma or exercise-

induced bronchoconstriction are well recognised conditions affecting human athletes, they are not overtly recognised and documented in horses. However, this is an area of growing interest and airway epithelial injury has been demonstrated in horses following exercise whilst breathing cold (+4°C) air (Davis *et al.* 2002).

- The most common injury to the respiratory system associated with exercise is exercise-induced pulmonary haemorrhage (EIPH) which appears to be ubiquitous in horses exercising at moderate speeds (~50% VO_{2max}) and above.

Other common conditions that affect the respiratory tract, such as inflammatory airway disease (IAD) and recurrent airway obstruction (RAO; 'heaves'; a condition similar to human asthma) may also limit exercise capacity and performance through their adverse effects on gas exchange. These disorders may be exacerbated by exercise and may also predispose the horse to or exacerbate other conditions such as IAD and EIPH.

EXERCISE INDUCED PULMONARY HAEMORRHAGE (EIPH)

EIPH continues to frustrate the veterinary and scientific professions, as well as horse owners and trainers. However, the last decade has seen significant advances in our understanding of the condition and its effects on pulmonary function and performance. Whilst it has been almost universally considered that severe haemorrhage (ie epistaxis) has a negative effect on performance, the significance of the 'normal' grades of EIPH on lung function and performance encountered following moderate to intense exercise have been unclear. A major advance in research into EIPH has been the application of bronchoalveolar (BAL) lavage to quantify the severity of haemorrhage (Meyer *et al.* 1998). An increasing weight of evidence from experimental and epidemiological studies into EIPH points to this condition being multifactorial, with the most important factors determining the level of bleeding within an individual being pulmonary vascular pressures, airway inflammation and locomotory trauma.

Studies using BAL to quantify haemorrhage have shown that even though blood may not be present in the trachea and visible on examination around 30–60 min following intense exercise, even a light canter may increase the number of free red blood cells (RBC) from less than 10 RBC/ul BAL at rest to easily in excess of 200 RBC/ul BAL post exercise. This level of haemorrhage will not result in blood being visible in the trachea. From our experience, the RBC count in BAL must exceed around 10,000 RBC/ul before the lowest grade of EIPH (ie Grade 1 out of 5) is visible in the trachea on post exercise endoscopic examination. Further evidence that only light-moderate exercise is required to cause mild EIPH comes from studies in Japan. Oikawa (1999) identified EIPH in the lungs of 2-year-old Thoroughbreds that had been trained at speeds of less than 9 m/s (20 mph) – the equivalent of a slow canter.

It is well recognised that there is considerable variation in the severity and indeed the occurrence (ie whether a horse has blood in the trachea or not) of EIPH when assessed post exercise. This has been apparent from epidemiological studies of racing Thoroughbreds where the prevalence of EIPH positive horses has ranged from 40–75%. There is evidence of an age effect, with the chance of finding blood in the trachea post exercise increasing from 2 to 4 years of age. For example, in a study in 223 Thoroughbreds racing on the flat in the UK, 40% of 2-year-olds, 65% of 3-year-olds and 82% of 4-year-old (or older) horses had blood in the trachea following racing (Roberts and Marlin, unpublished data). A recent study from the USA provided important information relating to the prevalence of endoscopic EIPH post racing (Birks *et al.* 2002). These authors reported that for a single endoscopic examination, ~75% of horses had blood in the trachea after racing. This increased to ~95% when horses were examined on 2 occasions and when horses were examined on 3 occasions, all horses had visible blood on at least one of the examinations.

A number of different theories have been proposed to explain the occurrence of EIPH, however few, if any, have been able to explain the initial site of occurrence and pattern of progression of haemorrhage through the lung. The most widely accepted theory at present is that of pulmonary capillary stress failure due to high transmural pressures (pressures or stresses acting on the pulmonary capillaries). Pulmonary capillary transmural pressure is determined by pulmonary capillary pressure and airway pressure.

The horse has high pulmonary vascular pressures during intense exercise. When the high pulmonary vascular pressures (exceeding 100 mmHg) distending the blood vessels are opposed by high positive airway pressures, such as occur during expiration, the transmural pressure (and by implication, wall stress) will be low. However, when the distending internal vascular pressure is associated with a large negative airway pressure (as occurs during inspiration), the transmural pressure and wall stress will be high. Studies *in vitro* have demonstrated that significant disruption of the pulmonary capillaries occurs at pressures of approximately 80 mmHg. *In vivo* it has also been shown in one study that there is probably a threshold mean pulmonary artery pressure of around 80–95 mmHg, above which significant haemorrhage is more likely to occur (Meyer *et al.* 1998; Langsetmo *et al.* 2000). On the basis of this theory, any factor or disease that would increase pulmonary vascular pressures (eg hypervolaemia) or increase the magnitude of the negative pressures in the lung during inspiration (eg dynamic upper airway obstruction) would be expected to increase the severity of EIPH. Interestingly, it has been shown that neither experimentally induced laryngeal hemiplegia nor dorsal displacement of the soft palate increased pulmonary capillary transmural pressure (Jackson *et al.* 1997). The limitation of the pulmonary capillary stress failure theory is that it does not in itself explain the site or pattern of progression of EIPH.

More recently a new theory for EIPH has been proposed based on locomotory forces (Schroter *et al.* 1998; Schroter *et al.* 1999). This theory attempts to explain the site of initiation in the tips of the dorso-caudal lung, the nature of the damage and the pattern of progression. The theory is based on the fact that during galloping, the absence of any bone attachment of the forelegs to the spine causes the shoulder to compress the cranial rib cage (see Colborne *et al.* 2004). The compression occurs largely during the stance phase (when the limb is planted on the ground and the body swings over the limb). The shoulder is moved in a dorsal and cranial direction into the chest. The compression of the chest initiates a pressure wave of compression and expansion which spreads outwards. However, due to the shape of the lung and reflections off the chest wall, the wave of expansion and compression becomes focussed and amplified in the dorso-caudal lung. The alternate

expansion and compression at the microscopic level in adjacent areas of lung tissue creates shear stress and capillary disruption. The notion that haemorrhage could occur in the lung in this way is consistent with that due to blunt trauma to the front of the chest or head which commonly results in lung or brain haemorrhage in car accident victims and boxers. In both cases the haemorrhage occurs at the opposite side of the body to that which is initially struck. The theory predicts that haemorrhage would be more severe on hard track surfaces.

Recent data from our own laboratory (Newton *et al.* 2005) based on over 200,000 UK race starts showed that the risk of epistaxis was increased for both hurdle and chase race types compared to both flat and National Hunt flat races. There was also a trend for increasing risk of epistaxis with increasing ground hardness ('going') and accumulated years spent racing. There was also increased risk of epistaxis associated with faster winning speeds in flat races, with male gender in hurdle races and carrying weight >150 lbs in chases races. A highly statistically significant trend for increasing risk of epistaxis associated with poorer finishing position was also demonstrated.

A number of other factors, such as concurrent airway inflammation, have been suggested to exacerbate EIPH. A recent epidemiological study in 7 training yards in the UK over 3 years has shown that risk for EIPH was 3 times higher in horses with mild inflammation and 10 times higher in horses with moderate to severe airway inflammation, as assessed by tracheal wash (Newton and Wood, 2002).

TREATMENT OF EIPH

The fact that the precise aetiology of EIPH is still far from clear and that it may well be multi-factorial or exacerbated by other co-existing disease processes or by inherited factors is reflected in the wide variety of approaches used to treat or manage EIPH. However, to date the number of treatments shown, under close scientific scrutiny in properly conducted trials, to have any efficacy in terms of reducing the severity of EIPH remains small. It should be considered that the goal of abolishing EIPH in an individual horse asked to exercise intensely is unrealistic. All horses will have EIPH to some extent, even if only detectable on the basis of BAL or identification of

haemosiderophages in TW or BAL. However, treatment to reduce a horse consistently experiencing EIPH at Grade 4 or 5 to Grade 2–3 may well be achievable.

The mainstay of treatment for EIPH for many years has been furosemide (Lasix). In North America and some other racing jurisdictions, racing after furosemide treatment is permitted. However, in many other countries, whilst training horses on furosemide is permitted, its use during racing is banned. There is now a wealth of evidence to show that furosemide reduces pulmonary vascular pressures both at rest and during exercise when administered in doses ranging from 250–500 mg 1–4 h prior to exercise. Based on post race surveys at racetracks it has been shown that furosemide does reduce the severity of bleeding (based on the amount of blood in trachea visualised endoscopically), but that in a significant proportion of horses there is no clear reduction in EIPH. The failure to record reductions in the severity of EIPH could point to a relative insensitivity of endoscopic grading in relation to the true severity of haemorrhage or to the fact that in some horses, the major underlying and precipitating cause of EIPH is not related to high pulmonary vascular pressures.

More recently a number of studies have been conducted using RBC counts in BAL to quantify the severity of EIPH in treadmill studies on horses treated with and without furosemide. Lester *et al.* (1999) studied the effect of 250 mg of furosemide given either iv or by nebulisation 30 or 240 min before track exercise and compared the RBC counts in BAL obtained from the right lung 30 min following exercise with that in a control run (no treatment). Furosemide given iv 30 min before exercise reduced mean pulmonary artery pressure during exercise by approximately 11% and BAL RBC count by 61%. The other 3 treatments (250mg iv 4 h before exercise; 250 mg nebulised 30 min before exercise; 250 mg nebulised 240 min before exercise) produced a much smaller decrease in both mean pulmonary artery pressure (<5%) and only around a 25% decrease in BAL RBC count with all 3 treatments. The reason why 250 mg furosemide iv in this study only produced a decrease in mean pulmonary artery pressure around half that seen by other workers is unclear.

Kindig *et al.* (2001a) demonstrated an average reduction in RBC count in BAL of 90% when horses were treated with 1 mg/kg furosemide iv

4 h prior to exercise at 95% of maximal oxygen uptake. This intensity is perhaps slightly below that which many horses would experience in racing. A more recent study by Geor *et al.* (2001) showed a reduction in RBC count in BAL of 66% at an intensity of 120% VO_{2max} following administration of furosemide at 0.5 mg/kg bwt (235–278 mg) iv 4 h before treadmill exercise. In the latter study furosemide had the greatest effect on those horses that exhibited the most EIPH in the control run.

Inhaled nitric oxide (NO; 80 ppm), a potent smooth muscle dilator, has been shown previously to decrease pulmonary vascular pressures during exercise in the horse from 98 to 84 mmHg (Mills *et al.* 1996). Infusion of nitroglycerin (an NO donor) at a dose of 20 ug/kg/min has been shown to decrease pulmonary vascular pressures at rest but to have no effect on pressures during maximal exercise (Manohar and Goetz 1999) and both control and treated horses showed blood in the trachea after exercise. Oral nitroglycerin administered to horses at a dose of 22.5 mg however had no effect of pulmonary vascular pressures. The substrate for nitric oxide synthase (NOS), L-arginine at a dose of 200 mg/kg iv has also been reported not to reduce pulmonary vascular pressures during moderate intensity exercise. However, L-arginine at this dose can reverse the increase in pulmonary vascular pressures caused by the administration of L-NAME, a NOS antagonist (Mills *et al.* 1996). A more recent study has shown that inhaled NO (80 ppm) produced a small but consistent reduction in pulmonary vascular pressures, but in fact the RBC count in BAL was doubled with NO inhalation compared with the control run (Kindig *et al.* 2001b).

Whilst reduction in pulmonary vascular pressures by circulatory volume reduction with furosemide appears to be effective in reducing the severity of EIPH, reduction in pressure using vasoactive drugs may well increase the severity of EIPH. This points to the fact that the pre-capillary arterioles may be constricted in order to protect the pulmonary capillaries and thus be the cause of the high pulmonary vascular pressures seen in the horse. This would tend to suggest that treatment with vasodilators for EIPH is contra-indicated.

On the basis that a large proportion of the resistance to breathing occurs in the upper airways, and particularly in the nasal passages, nasal dilator strips have recently been developed

for horses (FLAIR, CNS Inc, Minneapolis, Minnesota, USA). There is no doubt that the soft tissue overlying the nasal incisive notch is poorly supported and can be observed to be drawn inwards during inspiration, effectively narrowing the nasal passages. This would have the effect of increasing the airway component of transmural pressure during inspiration and place greater stress on the blood gas barrier. In fact, preliminary findings in one study have shown that the FLAIR strip does decrease both upper airway resistance and tracheal pressure in horses during treadmill exercise (Holcombe *et al.* 2002). In addition, the FLAIR strip has also been shown to decrease oxygen consumption during exercise (Geor *et al.* 2001), presumably due to decreased work of breathing.

In 2 recent treadmill studies, the FLAIR strip was shown to reduce the number of RBC in BAL by an average of 44% (Kindig *et al.* 2001a) and by 74% (Geor *et al.* 2001). In the latter study, the greatest reduction in haemorrhage was seen in those horses exhibiting the higher volumes of bleeding in the control runs (no nasal strip). One recent study failed to demonstrate any change in the incidence of EIPH (that is scored as blood present or absent in the trachea following exercise; Goetz *et al.* 2001). However, given that the FLAIR strip in other studies has been shown to attenuate rather than abolish EIPH this 'negative' finding is of limited consequence. It is also important to emphasise that correct placement of a nasal strip is essential. The tendency for many users appears to be to place the strip too high on the nose. For this reason a template is included with the FLAIR strip to facilitate correct placement and should be used.

In the UK and many other racing jurisdictions the use of nasal strips is currently prohibited during racing but allowed during training. This is in contrast to North America were its use in racing is widespread. The efficacy demonstrated in the recent study by Geor *et al.* (2001) (74% reduction in BAL RBC count) approaches that of furosemide on the same horses (80%). The FLAIR strip and furosemide in combination reduced the average BAL RBC count by 87%. Thus, the use of nasal dilator strips based on these 2 treadmill studies, although on a limited number of horses, suggest that such devices merit strong consideration to use with or as an alternative to treatment with furosemide. After a brief spell of popularity in the UK following their introduction,

the use of nasal strips appears to have declined dramatically.

Water restriction is not an uncommon practice in many countries that have racing, in the belief that the dehydration may alter 'blood pressure' and thus prevent or reduce the severity of EIPH. To the author's knowledge, there is no information in the scientific literature that demonstrates any efficacy of water deprivation against EIPH. Prolonged water deprivation and dehydration cannot be controlled in the same way as with diuretics such as furosemide and any benefit from a reduction in severity of EIPH might well be offset by a reduction in performance due to prolonged dehydration.

The greater the severity of EIPH the greater the likely implication for health and welfare. In addition, moderate to severe EIPH is commonly thought to be a contributing factor in poor performance. This has been shown to be true for epistaxis (Kim *et al.* 1998) However, despite considerable anecdotal evidence, only Mason *et al.* (1983) found that severe endoscopic EIPH was less common in placed than in unplaced horses. A study from our laboratory (Roberts and Marlin, unpublished data) showed that the incidence of endoscopic EIPH in a group of 166 horses examined immediately post race in the UK (flat and jump racing) because of poor performance was no different to that of controls (horses performing to expectation). In addition, there was no relationship between EIPH incidence or severity and finishing position in either the control or poor performance group.

The failure to show any adverse relationship between increasing severity of endoscopic EIPH and performance in the majority of studies that have been conducted may have been due to poor experimental design and bias. A recent study conducted by Hinchcliff and colleagues in Australia on over 700 horses clearly demonstrated that increasing severity of EIPH was associated with finishing further behind the winning horse (K. Hinchcliff personal communication).

To conclude, EIPH is almost certainly multifactorial. All horses bleed to some degree each time they race. Increasing evidence suggests that EIPH has adverse effects on pulmonary function and performance in intense competition, such as racing. The only effective managements at present that have stood up to the rigours of scientific investigation appear to be diuretics and nasal dilator strips. If ground conditions and weight carried can be shown to influence EIPH,

then these potentially offer further routes to manage this condition.

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HUMAN LUNG INJURY IN SPORTS

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Serious lung injuries in sports are uncommon events. The range of aetiologies spans the gamut of sports. From high altitude pulmonary oedema associated with mountain climbing to near drowning secondary to scuba diving, the method of pulmonary injury encompasses human endeavour. Pulmonary barotrauma (from scuba diving or high altitude exposure) is probably the most common life threatening circumstance, though percentages are difficult to obtain.

Exercise-induced pulmonary dysfunction is the most common form of sport-related lung injury. Studies have shown a decline of approximately 10% in pulmonary diffusion capacity (DLCO) within hours of various forms of exercise, eg rowing, treadmill running, arm cranking, or marathon running (Nielsen *et al.* 1995). Of interest, the decline in DLCO is unrelated to the same mechanisms involved in adult respiratory distress syndrome.

At the alveolar level, the diffusion capacity of the lung for gas exchange decreases to below pre-exercise values in the hours following a bout of intense exercise or during prolonged or extreme exertion. The controversial mechanisms proposed for the decline in lung function are: (Nielsen *et al.* 1995) increase in interstitial lung water, (Johns *et al.* 2004) redistribution of central blood volume to peripheral muscles causing a reduction in pulmonary capillary blood volume, (Wagers *et al.* 2004) reorientation of red blood cells within the pulmonary blood capillaries, and (Gilbert and McFadden 1992) a decline in alveolar membrane diffusion capacity. In worst-case scenarios, there is a complete breakdown of alveolar and pulmonary capillary integrity (Nielsen *et al.* 1995).

Airway hyper-responsiveness with exercise is a well-documented event impairing athletic capability. The mechanisms provoking bronchospasm may be related to inhaled air temperature, inhaled antigens, hyperpnea, underlying airway inflammation, or combinations of the above (2.3). Studies in mice and in humans have suggested that leakage of diverse plasma proteins such as fibrinogen and thrombin promote bronchial reactivity while aerosolised fibrinolytic tissue-type plasminogen activator diminished airway hyper-responsiveness (Wagers *et al.* 2004).

Thermally-induced bronchospasm is an important environmental component of athletics. It is suggested that the rate and magnitude of airway rewarming during the respiratory cycle can trigger bronchial narrowing during exercise hyperpnea. This response can be blocked by inhaled norepinephrine, which decreases bronchial rewarming during exhalation via diminution of bronchial blood flow (Wagers *et al.* 2004).

Haemorrhagic pulmonary oedema is not well documented in humans. It was noted in 2 athletes running an extreme marathon in South Africa (McKechnie *et al.* 1979). The strength of the alveolar-pulmonary capillary interface (APCI) appears to be a component in protection from exercise-induced pulmonary haemorrhage. Various disease states can lead to deterioration of the APCI and accumulation of plasma proteins in the alveolus and a decline in pulmonary efficiency (West and Mathieu Costello 1995). Stress failure of pulmonary capillaries, decrease in alveolar efficiency, and bronchial reactivity during exercise are major contributors to impairment of exercise induced pulmonary dysfunction.

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SESSION 10:

Long distance

CHALLENGES OF LONG DISTANCE EXERCISE IN HORSES

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Long distance exercise provides a number of unique challenges to both equine and human athletes. An important difference between short-term, high intensity (ie sprinting) and prolonged, low intensity endurance exercise is the thermoregulatory challenge imposed by the heat load produced by ongoing metabolic activity of muscle. In both species, this challenge is largely met by heat dissipation through continued evaporation of sweat. However, differences in sweat composition lead to a comparatively greater loss of body electrolytes with each litre of sweat produced by athletic horses. This physiological difference may be important in the apparently greater risk to equine endurance athletes for development of 'metabolic' sporting injuries, ranging from premature fatigue to multiple organ failure and death. Further risk factors for 'metabolic failure' by competitive equine athletes are that the rider externally imposes the level of work effort and that horses may be forced to compete under ambient conditions that are minimally conducive to dissipation of metabolic heat.

A second difference between short-term high intensity exercise and endurance exercise is fuel utilisation. In both human and equine endurance athletes, fat is the primary fuel utilised but as exercise continues glycogen reserves are progressively depleted. The syndrome of 'hitting the wall' described by human athletes is generally thought to be caused, in part, by depletion of carbohydrate reserves. A similar syndrome has not been clearly recognised in equine endurance athletes and, despite considerable glycogen depletion, the need or benefits for supplemental carbohydrate ingestion by horses competing in endurance events is unclear.

Although the 'exhausted horse syndrome' is widely recognised at endurance events, the pathophysiology of 'metabolic failure' and the array of associated clinical problems that develop during or after exercise is more poorly understood. Traditionally, dehydration consequent to prolonged sweating has been implicated as the most important risk factor for 'metabolic failure'. However, recent reports of human endurance exercise, and limited data in equine endurance athletes, suggests that the magnitude of body fluid loss, typically reflected by weight loss, may be less important than previously thought. Rather, factors leading to a decrease in effective circulating volume, including maintenance of sympathetic tone and tissue perfusion, may be more important in the development of fatigue and possible organ dysfunction. This latter statement is supported by multiple anecdotal descriptions of marked clinical improvement after exhausted athletes are treated with simple manipulations (ie a few minutes of rest in the supine position with the pelvic limbs elevated for people) or administration of volumes of intravenous fluids that are less than would be required to replace sweat fluid losses. In addition to apparent loss of effective circulating volume and tissue perfusion, duration of exercise and decreased perfusion appears to be an important factor determining whether transient tissue hypoperfusion will be largely reversible or may result in either immediate or delayed organ failure. In horses, organ dysfunction and failure may appear clinically to affect one organ system primarily (eg gastrointestinal tract, kidneys, or central nervous system) but can also be manifested as multiple organ failure leading to death despite aggressive supportive care. Perhaps the most important, but as of yet unanswered, question is what is (are) the key factor(s) that differentiate whether a moderate degree of tissue hypoperfusion will lead to

reversible organ dysfunction or irreversible organ failure. Further, it is unclear why some metabolic disorders, typically muscle cramping and rhabdomyolysis, may occur relatively early in the course of endurance exercise while other equine athletes can successfully complete the endurance competition with similar, dramatic elevations in circulating muscle enzyme activities.

Although 'metabolic failure' often has more dramatic clinical consequences, equine endurance athletes more commonly fail to finish endurance competitions due to development of musculoskeletal disorders. Occasionally, musculoskeletal failures are also dramatic (ie when a horse crashes over a solid cross country fence or when an endurance horse falls or slides down a steep slope leading to a fracture) but, fortunately, these events are rare. In fact, lameness is the most common cause for elimination of equine athletes from endurance competition. Anecdotal reports describe tendon and ligament strain as the most important musculoskeletal problem limiting completion by equine endurance athletes, as compared to a greater prevalence of fetlock and carpal fractures in sprinting horses. However, less serious injuries, including heel bulb

abrasions during prolonged exercise in soft or sandy soil and foot damage sustained during loss of a shoe, are also important causes of elimination of endurance horses. During the longer rides (ie 100-mile and multi-day rides), skin irritation under the saddle and girth may also lead to elimination from competition.

With the current trend toward higher speeds (eg up to 20 km/h) in the elite international equine endurance competitions, orthopedic injuries typically limited to horses subjected to higher ground reaction forces (eg racehorses and jumping horses) are now being recognised in equine endurance athletes. For example, veterinarians officiating at these elite competitions are starting to observe horses that fracture the short pastern bone (second phalanx) during such competitions.

At present, epidemiological data that would provide a detailed overview of the types and prevalence of both 'metabolic' and musculoskeletal problems that affect equine endurance athletes are lacking. Clearly, this type of data is essential for improvements in both training and officiating during competition in order to decrease the occurrence of both types of problems.

HUMAN CAPABILITY AND VULNERABILITY IN MASS MARATHON RUNNING

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Modern mass marathon events show that many modern men and women have to create unnecessary physical challenges over which they can triumph in order to gain a sense of achievement. The challenge of the modern marathon race, of running 26.2 miles (42.2 kilometres) on a hard surface, exposes runners to considerable physiological stresses as well as repetitive trauma from what a popular Edwardian Music Hall song (about a lame horse) called 'The Hammer Hammer Hammer down the Hard High Road'.

CAPABILITY

The modern marathon is based on the 1896 event introduced by Baron de Coubertin to the first modern Olympic games. He had read the famous poem by Robert Browning (1879) glorifying the myth of the Greek runner Pheidippides bringing news to Athens on foot of their victory at Marathon over the Persians and then dying (a myth as there is no contemporaneous account even by the historian Herodotus). Messengers, hemerodromoi, went on foot, not horseback.

The marathon is an endurance test for which most people are not well adapted and from which they take several weeks to recover fully.

AGE

Despite being regarded as a major challenge by the majority of participants, runners as young as 8 years and as old as 80 years have run the marathon in about 200 min (3 h 20) and a man of over 90 has run it in under 360 min (6 h). Fields for major marathons now exceed 30,000, with females making up about one third of the runners. The most populated 5 year age group in the entries is

35–39 inclusive and the next is 40–44 inclusive. Most entrants train for several months and in many events >95% of the starters finish. However, in the London Marathon, about 25% of the entrants who sign up 5 months before the event do not start.

In the London Marathon as many as 75% are raising money for charities.

Illness and disability

Many of the charity entrants walk the whole way. Many enter with ongoing medical problems and pursue a form of illness denial (Table 1). Medical charities benefit dramatically from the cult of sponsored marathon runs to the extent that some organise their own events.

Enlarging the challenge

However one marathon is not enough for some adherents and last year 2 runners completed the New York Marathon as a seventh marathon after running 6 previous marathons on the 6 previous

TABLE 1: Examples of illness denial in marathon runners

HEART DISEASE post infarct, post CABG, transplant

CANCER, LEUKAEMIA

ASTHMA, LUNG TRANSPLANT

DIABETES insulin dependent

COLECTOMY

PARAPLEGIA wheel chair athletes

MOTOR NEURONE DISEASE

EPILEPSY

ADDISON'S DISEASE

GROSSLY DISABLED congenital and acquired

days. The marathons were in 6 different continents. One of the runners (Ranulph Fiennes) ran within 4 months of cardiac bypass surgery. A British runner has won a foot race across the US from the Pacific to the Atlantic oceans (approximately 5000 km) winning the event by averaging 80 km a day for 62 days consecutively. Double marathons, marathons in extreme environmental conditions and triathlons are becoming increasingly popular.

Physiological limits

Speed of running a marathon is limited to <6 m/sec, by the aerobic power of man, his maximum glycogen storage capacity and inability to burn fat at a fast enough rate when the glycogen stores are depleted. There is also a need for heat dissipation of as much as a kilowatt, and ideal marathon running conditions favour ambient temperatures between 5 and 10°C. Elite marathon runners are often small, and extremely thin, some weigh <45 kg. A disproportionate number of the fastest times has been achieved by Kenyans from a small area of the African Rift Valley.

Women run >8% more slowly than men and seem unlikely to fulfil previous predictions that they would run faster than men.

Physiological stresses

Marathons may be run under a huge range of conditions which will be handled differently by different runners As running can produce a heat output of as much as a kilowatt, weather conditions are crucial, but a cool day in England can produce runners with both hyperthermia (a heavily built body builder) and hypothermia (an elite Kenyan runner who became rapidly chilled when he could not continue running). Heat loss is primarily by evaporation of sweat, with one litre of sweat equivalent to 580 kcals (2.4 MJ). Heat and fluid balance are therefore crucial. Inadequate fluids may result in dehydration, loss of running efficiency and heat disorders; excess hypotonic fluids can produce hyponatraemia.

Other stresses

Running a marathon produces muscle stiffness which is delayed (DOMS) especially when some of the course is down hill, necessitating eccentric muscle contractions. Muscle biopsies show microtrauma and the muscle can take weeks to recover. Knee injuries, lower leg 'shin splints' and Achilles' tendonitis are common in training and fatigue fractures are not uncommon particularly in women who have amenhorrea.

The much publicised 'wall' is a physical and probably psychological event that occurs particularly in runners who are novices, have trained inadequately and start too fast. It can occur at half distance onwards but more usually at about 20 miles.

There is a strong feeling of fatigue, muscles ache or burn and there is an overwhelming desire to stop or at least walk rather than run. It often coincides with depletion of muscle glycogen. The muscle stores of glycogen can be boosted but in most individuals are insufficient to supply all the fuel for aerobic running for more than about 90 min. In the well trained runner a combination of glycogen and fat is utilised and the glycogen supplies in the muscle are eked out to the end of the race. Glycogen depletion makes exercise uncomfortable, is associated with a drop in running speed and often an increase in ventilation. For the same mechanical energy output, fat requires more oxygen than does glycogen, but even with maximum availability of oxygen the running pace is considerably lower than when burning glycogen because of mechanisms within the muscle fibres which limit the rate of lipid metabolism

VULNERABILITY

Casualties

Major marathons have frequent aid stations along the route and usually a more elaborate Field Hospital at the finish to deal with runners who collapse at the finish or shortly afterwards.

The total morbidity of a mass marathon can never be assessed. Luxury medical support (frequent inviting first aid stations) attracts runners with minor ailments, such as cramps and blisters which the majority of runners suffer without seeking medical aid.

Surveys of marathon runners have a notoriously low response rate. Even checking race related hospital Accident and Emergency departments for runner casualties would give incomplete statistics of runners coming from outside the immediate environment of the marathon. Troubled by persistent or new symptoms they would go to their local hospital

TABLE 2: Constitutional medical problems associated with marathon running

GASTROINTESTINAL vomiting, diarrhoea, stomach cramps, GI bleeding and bowel ischaemia

NEUROLOGICAL exertion headache CVA, epilepsy

RESPIRATORY ASTHMMA hyperventilation

CARDIAC ARREST myocardial infarction Arrhythmia

EXERCISE ASSOCIATED COLLAPSE hyperthermia, hypothermia, hypoglycaemia, hypovolaemia, heat stroke

RENAL TRACT haematuria, haemoglobinuria, renal failure rhabdomyolysis

HYPONATRAEMIA FROM EXCESS FLUIDS

TABLE 3: Exercise associated collapse

Intense exercise causes:
- Profound vasodilatation
- Depletion of effective circulating blood volume
- Reduction of pre-load and stroke volume
- Depletion of muscle and liver glycogen/blood glucose

Cessation of exercise:
- Loss of calf muscle venous return pump
- Loss of diaphragmatic venous return pump
- Increased leg blood pooling
- Sudden drop in pre-laod, cardiac output and BP

Treatment:
- Lithotomy position
- Fluids ++ (iv if necessary)
- Carbohydrate oral or iv
- Cool, if hot
- Strip, dry and warm if cold

TABLE 4: London Marathon deaths for 24 years compared with ETSC travel risks 2001–2002 >580,000 marathons, 25 million Km, 8 deaths

Mode of transport	Deaths/ 100 Mkm	Deaths/ 100 mhrs	Deaths/ 100 years	Normalised death risk/time exposed
LM 1981–2004	32	308	2.67	12
Motorcycle	13.8	440	3.81	18
Bicycle	5.4	75	0.65	3
Car	0.7	25	0.21	1
Airline	0.035	16	0.138	0.7
Rail	0.035	2	0.017	0.08

after they have left the finish.

Classification

The first aid contacts are classified as:

Social: Stop for a drink, shoe lace or self administered petroleum jelly.

Topical: Blisters, groin chafing, runners nipple, subungual haematoma.

Musculoskeletal: Cramp, joint pain, fatigue fractures, injuries from falls.

Constitutional: These are the more serious 'medical' problems and include all the collapses (Table 2).

Exercise itself may produce collapse from fatigue and fluid and glycogen depletion (exercise associated collapse) (Table 3).

It may be complicated by hypothermia, hyperthermia or even heat stroke.

Cardiac arrest and arrhythmias, hypoglycaemia, vomiting, diarrhoea, ischaemic colitis, rhabdomyolysis leading to haemoglobinuria and renal failure, can all occur but muscle cramp and blisters are overwhelmingly the most frequent complaint.

Race and race hospital experience varies dramatically from year to year and the 2 hot London Marathon days (1996 and 2003) produced very different spectra of hospital cases, with hyponatraemia very rare in 1996 being the commonest diagnosis at St Thomas's hospital in 2003 (14 cases). In 2004, after considerable race medical education, there was only one case from 32,200 runs, but it was a cool very wet day.

Mortality

There is a popular misconception, probably partly based on the Pheidippides myth, that 'unfit runners can kill themselves with over exertion'. The London Marathon had no deaths until the 10th year, but has now had 8 (in 580,000 runs) and all the runners were previously symptom free and considered very 'fit' by their friends and relatives.

Five had severe coronary artery disease, 2 had hypertrophic cardiomyopathy, of which one collapsed after finishing but could not be resuscitated. A young runner collapsed at the finish from a subarachnoid haemorrhage and died subsequently in hospital. There have been 5 successful cardiac resuscitations, all with coronary heart disease.

The death risk of running the London Marathon has been compared with the exposure risk of different forms of transport within the European Union (Tunstall Pedoe 2004). Running the London marathon is less risky on a time of exposure basis than riding a motor bicycle (2/3 rd risk) but 4 times more risky than riding a bicycle. In other words 2 h on a motor bike has the same death risk as running for 3 h in the London Marathon or 12 h of cycling (Table 4).

MEDICAL SERVICES

There have been 5 successful cardiac resuscitations in the London Marathon and this is a great credit to the medical services, principally supplied by St John Ambulance Brigade.

The Medical Director works closely with the race organisers and St John and we recruit doctors, chartered physiotherapists, podiatrists and nurses to work with the >1,300 St John volunteers at >30 aid stations and finish medical facilities. The Medical Director gives medical advice to the runners and treatment advice to St John and the hospital Accident and Emergency Departments who have to deal with the serious marathon casualties, usually relatively few in number.

FURTHER READING

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