Ankle injuries in basketball: injury rate and risk factors

G D McKay, P A Goldie, W R Payne, B W Oakes

Abstract

Objectives—To determine the rate of ankle injury and examine risk factors of ankle injuries in mainly recreational basketball players.

Methods—Injury observers sat courtside to determine the occurrence of ankle injuries in basketball. Ankle injured players and a group of non-injured basketball players completed a questionnaire.

Results—A total of 10 393 basketball participants were observed and 40 ankle injuries documented. A group of non-injured players formed the control group (n = 360). The rate of ankle injury was 3.85 per 1000 participations, with almost half (45.9%) missing one week or more of competition and the most common mechanism being landing (45%). Over half (56.8%) of the ankle injured basketball players did not seek professional treatment. Three risk factors for ankle injury were identified: (1) players with a history of ankle injury were almost five times more likely to sustain an ankle injury (odds ratio (OR) 4.94, 95% confidence interval (CI) 1.95 to 12.48); (2) players wearing shoes with air cells in the heel were 4.3 times more likely to injure an ankle than those wearing shoes without air cells (OR 4.34, 95% CI 1.51 to 12.40); (3) players who did not stretch before the game were 2.6 times more likely to injure an ankle than players who did (OR 2.62, 95% CI 1.01 to 6.34). There was also a trend toward ankle tape decreasing the risk of ankle injury in players with a history of ankle injury (p = 0.06).

Conclusions—Ankle injuries occurred at a rate of 3.85 per 1000 participations. The three identified risk factors, and landing, should all be considered when preventive strategies for ankle injuries in basketball are being formulated.

Keywords: basketball; ankle; injury; risk; prevention

Subjects and method

In Melbourne, Australia, an elite basketball competition and three recreational basketball competitions were observed to identify injuries prospectively. Injury observers were instructed to view games and note the occurrence of injuries during the game. At the end of a game, all players were asked about their injury status, and injured players completed a questionnaire. A control group was obtained by administering a questionnaire to entire teams of players who were not injured on a particular day but were from the same competition as the injured players. All games observed were played indoors on wooden floors.

Table 1 outlines the areas of questioning. The ankle injured players were telephoned to monitor the progress of their injury, to obtain information about time missed, treatment sought, and changes in shoes, protective equipment, and warm up on returning to play.
Boxing and the brain

P McCrory

Revisiting chronic traumatic encephalopathy

Chronic traumatic brain injury or chronic traumatic encephalopathy (CTE) is considered by some authorities to be the most serious health problem in modern day boxing. The condition is often referred to by a number of names in the medical and non-medical literature including dementia pugilistica and “punch drunk” syndrome.

Whilst there exists great controversy regarding the ethics of boxing, one of the key medical issues is the risk of a boxer developing CTE either during or after his boxing career. Recent evidence suggests that exposure to boxing alone is insufficient to cause this condition.

It is believed that CTE represents the cumulative long term neurological consequences of repetitive concussive and sub concussive blows to the head. CTE is more common in professional rather than amateur boxers, however, CTE has been documented in other sports such as American football, ice hockey, rugby, horse racing, and soccer.

CTE is clinically characterised by a combination of speech and gait disturbance, pyramidal tract dysfunction, memory impairment, extrapyramidal features, behavior or personality changes, and psychiatric disease. In the early stages of this condition, the symptoms are transient and reversible, however, in the later stages they are progressive. The neurology of CTE includes characteristic neuropathological features of cerebral atrophy, septal fenestration, cerebellar tansillar scarring, cavum septum pellucida, loss of pigmented cells, and prominent neurofibrillary tangles.

It is salient to review the original paper discussing the neuropathology of CTE. Although individual case reports had been published of boxers with chronic dementing illnesses, the seminal paper discussing the association of neuropathological findings in boxers was published by the English pathologist, John Corsellis. He studied the brains of 15 retired boxers and retrospectively studied their fight histories. While a number of characteristic changes were noted in these brains, it is the boxers’ histories that deserve specific note. Of the fighters studied, their exposure to boxing ranged between 300 and 700 bouts in the course of their careers. This was in addition to sparring and other fight training that would have occurred.

The issue then that needs consideration is that in this day and age we would seldom see a fighter with such a record. Even the top professionals report fight careers of 30–50 fights before retirement, an order of magnitude less than that described in Corsellis’ landmark study.

Recent research in boxers has also suggested that CTE in boxers may be associated with a particular genetic predisposition. The apolipoprotein E-ε4 gene (ApoE), a susceptibility gene for late onset familial and sporadic Alzheimer’s disease, may be associated with an increased risk of CTE in boxers.

In a non-boxing population, ApoE polymorphism was significantly associated with death and adverse outcomes following acute traumatic brain injury as seen in a neurosurgical unit. In a recent prospective study, ApoE genotypes were tested for their ability to predict days of unconsciousness and functional outcome after six months. There was a strong association demonstrated between the ApoE allele and poor clinical outcome.

Furthermore, ApoE deficient (knockout) mice have been shown to have memory deficits, neurochemical changes, and diminished recovery from closed head injury when compared to controls. It is suggested that ApoE plays an important role in both neuronal repair and antioxidant activity resulting in ApoE knockout mice exhibiting an impaired ability to recover from closed head injury.

How then does this help the debate on the risks of boxing? Firstly we need to reconsider the original evidence on exposure as a risk factor for CTE. The simplistic assumption based on epidemiological data from previous studies that CTE is a manifestation of the length of a boxer’s career and hence exposure to punches needs to be readdressed.

Similarly the development in understanding of the genetic risk that a boxer may carry developing CTE means that this area may need to be re-examined in light of current day research. This issue also raises a number of ethical issues, if a boxer is found to be homozygous for the ApoE-ε4 phenotype should his boxing career be curtailed? At the very least, informed consent, and genetic counseling should be undertaken.

Whilst one may argue the ethics and morality of boxing, it behooves us as scientists and clinicians to at least place the medical arguments regarding risk of injury on a scientific footing.

REFERENCES


Author’s affiliation

P McCrory, Editor
Brain injury and heading in soccer

Head to ball contact is unlikely to cause injury but head to head contact might

Whether repeated concussive or subconcussive blows cause permanent or cumulative brain injury is a complex and controversial question. Press coverage highlighted the case of Jeff Astle, a former England international football player, where the coroner ruled the cause of his death as an “industrial disease”—suggesting that repeated heading of balls during his professional career was the cause of his subsequent neurological decline. This case was at odds with that of Billy MacPhail, a former Glasgow Celtic player, who in 1998 lost a legal battle to claim benefits for dementia that he said was due to heading the old style leather footballs. Concern has been raised over whether heading in soccer may be the basis for injury and cognitive impairment, and in the United States this has led to calls advocating the use of protective headgear for soccer players.

Soccer players don’t just head the ball; their heads can collide with each other, and players in positions where heading is common are also more likely to have head to head collisions more often. Although uncommon, most concussive injuries seen in soccer derive from such head to head rather than ball to head contact.

Heading a soccer ball results in head accelerations of less than 10 g (or less than 1000 rad/s²) whereas the minimum values for the development of sport related concussion are 40-60 g (or 3500-5000 rad/s²). In contrast, head to head contact can generate enough of the forces required to cause brain injury as in any conventional head injury. Recent biomechanical research has found that commercially available soft helmets fail to reduce even this degree of head trauma to a safe level, which implies that these helmets have only a limited protective role in this setting.

There is no evidence that sustaining several concussions over a sporting career will necessarily result in permanent damage. Research on experimental animals provides some supporting evidence against the concept that recurrent concussive injuries alone cause permanent damage. In studies of experimental concussion, animals have been subjected to repeated concussion 20-35 times in a two hour period. Despite the unusually high number of injuries no residual or cumulative effect was shown.

Can repeated subconcussive trauma such as might be seen in heading the ball cause a cumulative neurological injury in this setting? Although this was indicated by early retrospective studies, more recent studies have not supported this idea.

In a series of retrospective studies including retired Scandinavian soccer players, cognitive deficits were noted. The results of these studies are flawed, with appreciable methodological problems. These problems include the lack of pre-injury data, selection bias, failure to control for acute head injuries, lack of blinding of observers, and inadequate controls. The authors conclude that the deficits noted in these former soccer players were explained by repetitive trauma such as heading the ball. However, the pattern of deficits seen is equally consistent with alcohol related brain impairment—a confounding variable that was not controlled for.

Matser et al from the Netherlands have also implicated both concussive injury and heading as a cause of neuropsychological impairment in both amateur and professional soccer players. Reanalysis of the data from these papers, however, indicates that purposeful heading may not be a risk factor for cognitive impairment.

Prospective controlled studies using clinical examination, neuroimaging, or neuropsychological testing have failed to find any evidence of cognitive impairment in soccer players.

We do not know for certain whether heading the ball in soccer may result in chronic cognitive impairment. It seems unlikely that subconcussive impacts such as seen in head to ball contact will cause chronic neurological injury. Although head to head contact may cause concussive injury, it is both uncommon and unlikely to result in cumulative brain injury. It has been speculated from other sports that particular genotypes may place athletes at heightened risk in
association with head trauma, although this is yet to be validated in other studies. For football players the avoidance of exposure to brain injury is important, although currently there are few means by which this may be achieved. Most head to head contact is inadvertent, and coaching techniques and visual perception training may help in a few cases but are unlikely to eliminate this problem entirely. Soft shell helmets or head protectors currently do not have the biomechanical capability to prevent concussive trauma and hence cannot be recommended.

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Competing interests: None declared.

1 Shaw P. Heading the ball killed England striker Jeff Astle. Independent 2002 Nov 12.

Self reports in research with non-English speakers

The challenge of language and culture is yet to be met

A ssessment of the health and healthcare needs of ethnic minority populations, often relying on self reported data, is important in health and social services. Major problems exist with the reliability of such information, particularly among recent and older immigrants and refugees who may have little or no competency in English and may be at high risk of health problems. Approximately 25% of immigrants to Britain born in China, Bangladesh, India, and Pakistan have no functional skill in English, and 70% cannot function fully in an English speaking social environment.

When a measure is probing differences within a group it must be appropriate, valid, and reliable for the group concerned. However, if the data are to be used to make comparisons between groups as in clinical trials and most epidemiological studies, then the questions must be conceptually and functionally equivalent and appropriate for all the groups compared. Non-English speakers are often excluded from clinical trials and epidemiological studies, for reasons including the lack of valid and reliable cross cultural measurements.

In clinical and epidemiological studies questions developed for native English speakers are usually translated into other languages. It is assumed that the modes of inquiry appropriate for native English speakers are applicable to other linguistic groups. These assumptions may reflect pragmatic issues relating to time and finance or lack of understanding of the complexities of language and culture. Translations, even by experts, may fail to achieve questions that are comparable to the original English in terms of appropriateness and meaning. It is therefore important to consider conceptual matters, cultural relevance, and the subtle connotations of words and phrases.

In multilingual studies, if each language is written and spoken forms are not the same—for example, smoking tobacco. This strategy requires an participatory approach whereby monolingual and bilingual representatives of the target group(s) are
**SUPPLEMENT**

**Erythropoietin and blood doping**

N Robinson, S Giraud, C Saudan, N Baume, L Avois, P Mangin, M Saugy

**Objective and method:** To outline the direct and indirect approaches in the fight against blood doping in sports, the different strategies that have been used and are currently being used to fight efficiently against blood doping are presented and discussed.

**Results and conclusions:** The paper outlines the different approaches and diagnostic tools that some federations have to identify and target sportspeople demonstrating abnormal blood profiles. Originally blood tests were introduced for medical reasons and for limiting misuse of recombinant human erythropoietin (rHuEPO). In this way it became possible to prevent athletes with haematocrit levels well above normal, and potentially dangerous for their health, competing in sport. Today, with nearly a decade of blood testing experience, sports authorities should be familiar with some of the limitations and specially the ability of blood tests performed prior to competitions to fight efficiently against the misuse of rHuEPO, blood transfusion, and artificial haemoglobin.

Erythropoietin (EPO) is a 30 400 molecular weight glycoprotein hormone produced mainly in the kidney, and also in the liver (<10%) and, in very little quantities, in the brain. The physiological stimulus for EPO production is tissue hypoxia, which, in the large majority of instances, is directly related to the number of circulating erythrocytes. Thus, EPO and erythropoiesis are part of a negative feedback cycle that keeps tissue oxygen delivery within a narrow range by controlling the number of erythrocytes circulating in the blood. In a normal individual, any loss of erythrocytes, such as by bleeding or haemolysis, decreases delivery of oxygen to the tissues. When this tissue hypoxia is sensed by cells in the kidney and liver capable of producing EPO, they produce and secrete EPO into the plasma. EPO is carried to the bone marrow, where it binds to specific cell surface receptors on its target cells—the CFU-E, pro-erythroblasts, and basophilic erythroblasts. The binding of EPO by these cells increases their ability to survive and reach the reticulocyte stage and thereby contribute to the population of circulating erythrocytes. The increased numbers of circulating erythrocytes in turn deliver more oxygen to the tissues. This increased oxygen delivery is sensed by the EPO producing cells, which then reduce EPO production so that the normal steady state number of erythrocytes is restored.

The response of the kidneys to hypoxia has been shown to be exponential—that is, in individuals with a normal capacity to produce EPO, a linear decline in haematocrit is accompanied by an exponential increase in plasma EPO levels. This exponential increase is not based on the release of stored, preformed EPO. Rather, the hypoxia is sensed by an intracellular molecule that interacts with an enhancer element of the Epo gene and thereby induces transcription of the gene. The increase in EPO production in the hypoxic kidney is achieved by recruitment of more cells to produce EPO. The EPO producing cells of the kidney are a minor subset of cortical interstitial cells. Under normal conditions, only a few scattered cells produce EPO. When a threshold level of hypoxia is achieved, the cells capable of producing EPO do so at a maximal rate. The greater the areas of renal cortex in which the hypoxia threshold is met, the greater the number of cells that produce EPO.

**MECHANISM OF ACTION OF ERYTHROPOIETIN**

In the bone marrow, EPO binds to receptors displayed on the cell surface of CFU-E, proerythroblasts, and basophilic erythroblasts. The mature EPO receptor, with a molecular

**Abbreviations:** BFU-E, burst forming unit-erythroid; CFU-E, colony forming unit-erythroid; EPO, erythropoietin; IOC, International Olympic Committee; LAD, Swiss Laboratory for Doping Analyses; rHuEPO, recombinant human EPO; sTFR, soluble transferring receptor
Human growth hormone doping in sport

M Saugy, N Robinson, C Saudan, N Baume, L Avois, P Mangin

Background and objectives: Recombinant human growth hormone (rhGH) has been on the list of forbidden substances since availability of its recombinant form improved in the early 1990s. Although its effectiveness in enhancing physical performance is still unproved, the compound is likely used for its potential anabolic effect on the muscle growth, and also in combination with other products (androgens, erythropoietin, etc.). The degree of similarity between the endogenous and the recombinant forms, the pulsatile secretion and marked interindividual variability makes detection of doping difficult. Two approaches proposed to overcome this problem are: the indirect method, which measures a combination of several factors in the biological cascade affected by administration of GH; and the direct method, which measures the difference between the circulating and the recombinant (represented by the unique 22 kD molecule) forms of GH. This article gives an overview of what is presently known about hGH in relation to sport. The available methods of detection are also evaluated.

Methods: Review of the literature on GH in relation to exercise, and its adverse effects and methods of detection when used for doping.

Results and conclusion: The main effects of exercise on hGH production and the use and effects of rhGH in athletes are discussed. Difficulties encountered by laboratories to prove misuse of this substance by both indirect and direct analyses are emphasised. The direct method currently seems to have the best reliability, even though the time window of detection is too short. hGH doping is a major challenge in the fight against doping. The effect of exercise on hGH and its short half-life are still presenting difficulties during doping analysis. To date the most promising method appears to be the direct approach utilising immunoassays.

GROWTH HORMONE AND EXERCISE

The effect of acute exercise on production of GH in the body has been widely described in the literature. The concentration of hGH in blood increases with time for a given work intensity and can increase 10-fold during prolonged moderate exercise. During more intensive exercise (with accumulation of lactate at 70% \( V_{\text{O}2}\) max for a short term period such as 10–20 minutes) hGH will increase by 5–10-fold. With short exercise durations, levels of GH will generally peak at 15–30 minutes after the exercise. Furthermore, it appears that hGH response is more closely related to the peak intensity of exercise than the total work output. Endurance training generally amplifies the pulsatile release of growth hormone, elevating the GH amplitude. This appears evident when the training is very hard and above the aerobic threshold.

Apart from exercise related increase, GH secretion can be affected by other factors—for example, GH secretion is increased in hypoglycaemia, increased temperature, and stress, whereas it decreases in obesity, or with a carbohydrate-rich diet and intake of \( \beta_2 \) adrenergic agonists. Thus, it is hard to differentiate between the physiological increase in hGH levels seen in exercise and what can be from external hGH administration (as in doping). This problem makes the purely quantitative approach of measuring directly the total circulating GH not feasible in case of doping, except if the conditions of collection of biological samples are well controlled.

Abbreviations: GH, growth hormone; IGF, insulin-like growth factor; rhGH, recombinant human GH
REDUCING THE PREVALENCE OF EXERCISE RELATED CARDIAC DEATH

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INTRODUCTION

Non-traumatic sudden death during or immediately following strenuous sporting activity is an event of which most sportsmen are aware. The subject has been the concern of sportsmen, sports governing bodies and the medical profession, and has often excited alarmist media reports.

Although there appears to be a statistically small increased risk of sudden death during strenuous exercise (Opie, 1975; Thompson et al, 1982), there remains an appreciable mortality (Northcote and Ballantyne, 1983, 1984). The majority of sudden deaths are attributed to cardiovascular disease, particularly coronary heart disease (CHD) and although a wide age range is affected, most subjects are over the age of 35 years (Northcote and Ballantyne, 1983).

It is the intention of this article to examine ways in which the prevalence of sudden cardiac death in sport can be reduced.

DEMOGRAPHIC DATA

At present, we are aware of 151 cases of sudden death which have occurred in association with strenuous sport and have been documented in the medical literature (Northcote and Ballantyne, 1984). Only six non-cardiac causes of death are recorded, four being attributed to intracranial haemorrhage, one to heat stroke, and one to an acute gastro-intestinal haemorrhage. Of the cardiac causes, CHD accounted for 110 cases, followed by structural cardiovascular abnormalities such as hypertrophic obstructive cardiomyopathy (HOCM), coronary artery anomalies and valvular heart disease. A surprisingly small number (3) are attributed to myocarditis. Most of the deaths occurred in association with running, field sports and squash. The subjects ranged in age from 16-66 years, but were mostly over the age of 35 years (mean = 40 ± 9.1 years). Thirty-eight per cent suffered prodromal symptoms or conditions and 52% of those with CHD had documented risk factors for CHD. The prodromal symptoms included chest pain, dyspnoea, fatigue and gastrointestinal upsets. The most prevalent CHD risk factor was smoking.

In summary, it would appear from this data that most sudden deaths are attributed to cardiac disease, most frequently CHD, and a proportion of individuals may be at greater risk because of the presence of pre-morbid conditions and CHD risk factors.

Cardiovascular Effects of Strenuous Sport

Sudden and excessive exercise is believed to be more likely to precipitate “heart attacks” in the unfit (Shephard, 1974). In addition, competitive sports may be more likely to precipitate sudden death than exercise training alone (Opie, 1975). It is probable, also, that aggressive individuals taking part in highly competitive sports are at greater risk.
A number of workers have reported the heart rate (HR) responses to various sports. In squash we have recently confirmed HR responses of 80% of the predicted maximum heart rate (PMHR), sustained for up to 40 minutes in healthy volunteers (Northcote et al, 1983). Similar studies in tennis have reported HR response of 60-70% of PMHR for periods of rally play only (Misner et al, 1980). Heart rate has been shown not to rise above 100 bpm in golf, bowls or cricket (Kozar and Himischer, 1963).

Cardiac Arrhythmias

Cardiac arrhythmias, including ventricular tachycardia, have been demonstrated in a group of healthy male individuals during squash (Northcote et al, 1983). One can assume, that such arrhythmias would be more likely in the presence of CHD. Apart from myocardial ischaemia, a number of reasons exist which may explain this phenomenon during or immediately after exercise.

Serum catecholamines rise during strenuous exercise and may result in myocardial ischaemia and arrhythmias in the presence of CHD (Raab et al, 1962). In the immediate post-exercise period, serum concentrations of catecholamines and free fatty acids are known to be elevated (Dimsdale et al, 1984; Johnson et al, 1969). Both of these changes may be responsible for cardiac arrhythmias at this time (Northcote et al, 1983; Gooch and McConnel, 1970). Other biochemical changes such as exercise induced hyperkalaemia, may also contribute to the generation of arrhythmias (Lim et al, 1981). Likewise, environmental influences such as humidity and high temperature, are known to elevate heart rate and increase cardiac ectopic activity (Taggart et al, 1972). Smoking is capable of stimulating both catecholamine release and an increase in free fatty acids in the serum (FFA’s) (Ball and Turner, 1974; Kershbaum et al, 1961).

Heat Stroke

Heat stroke, noted particularly in marathon running (Wyndham and Strydom, 1969), may result in haemocoagulation, which could make thrombus formation and subsequent coronary artery occlusion more likely. It has also been shown to cause patchy, subendocardial necrosis (O’Donnel and Clowes, 1972).

The potentially hazardous effects of strenuous exercise outlined above are probably more harmful in the presence of CHD or other cardiovascular abnormality. It is likely, that in the normal heart, these influences are benign.

PREVENTION

Recognising the Problem

Study into the phenomena of sudden death during vigorous exertion, is hindered by inadequate registration and investigation of each case. At present, a death is registered without information of the activity of the deceased prior to death. Thus, the frequency of death during exercise is not known. There is a need for greater recognition of this problem in view of the growth in the number of reported deaths and increasing age of participants. Sports governing bodies can assist by attempting to document the extent and nature of sudden death in their own sport. The Squash Rackets Association (SRA) is to be commended for its efforts in this way. They have gone as far as circulating some sensible suggestions to prospective players in an effort to reduce any complications occurring when playing squash.

Which Sport?

It is perhaps unfortunate, that squash has been subject to much media coverage because of the prevalence of sudden death. This may be a reflection of the individuals engaged in the sport. The SRA themselves, estimate that there may be 165,000 players over 45 years of age playing in the UK. As it is such a physically demanding sport, it may be wiser for individuals in the coronary prone age group to consider more gentle forms of exercise if taking up sport for the first time after some years of inactivity. The present boom in marathon running is also potentially hazardous to the coronary prone or those with undetected, overt, cardiovascular disease. Only a few years ago, we would have questioned the sanity of recommending a 26 mile run to a 50 year old, but now this activity is encouraged! There is no evidence that such endeavours are more beneficial to the cardiovascular system, than other, more gentle forms of exercise. Why has jogging a few miles a few times a week become “unfashionable”? We would recommend that individuals should be encouraged to exercise, but to participate in sports which are suited to their age, physical ability and general health. Walking, jogging, swimming and cycling would be suitable in this respect.

Education

There is a requirement for more adequate education and supervision of the embryo sportsman. All sportsmen should be aware of the small risks attached to vigorous exercise, and should be able to recognise warning symptoms. Aggressive, competitive sportsmen have been noted to deny prodromal symptoms (Opie, 1975; Northcote et al, 1984). Individuals should be encouraged to refrain from strenuous exercise if such symptoms occur. It has been suggested that individuals should not exercise when suffering from an upper respiratory tract infection or other pyrexial illness, as this can be accompanied by a potentially lethal subclinical myocarditis (Kocnar and Rous, 1973). Although we have not been able to implicate myocarditis as an important cause of sudden death in sportsmen, we agree that vigorous exercise should be discouraged at this time. The medical profession and those concerned with the supervision of sportsmen should be aware of the consequences of such
prodromal symptoms and conditions. In a recent study (Northcote et al, 1984), of 30 sudden deaths in association with squash, we found that one third of the individuals had hypertension documented on two or more occasions, but this had been treated in only one subject. Three subjects had hyperlipidaemia, three had angina or previous myocardial infarction (MI) and one subject was known to have suffered a myocardial infarct 10 years previously and also had aortic regurgitation, requiring regular review at a cardiology clinic. This subject had been told by his doctors that there was no harm in playing squash!

Pre-participation Medical Screening

Individuals such as those outlined above may benefit from medical screening which would detect overt cardiovascular disease and would indicate a relatively increased risk. However, having been advised of this increased risk, it is of course the individual’s decision whether he persists in playing a particular sport.

The practicality of medical screening has been questioned. However, often a simple interview and clinical examination would suffice. This would allow detection of CHD risk factors, and overt, potentially lethal cardiovascular disease. If a subject was found to have one or more CHD risk factors or was symptomatic, we would advocate screening using exercise electrocardiography. This policy has also been suggested elsewhere (Chung, 1980; Levy, 1981; Nye, 1983).

The value of exercise electrocardiography in this respect is not solely related to the detection of CHD. It can be a useful investigation for predicting future coronary events. There is good evidence that it does so (Table I). Bruce and McDonough (1969) studied 221 asymptomatic men and found that those with an abnormal appearance on the exercise electrocardiogram had a relative risk of subsequent CHD, 13.6 times greater than normal over the subsequent five years. Other workers have reported similar results ranging from a relative risk of 10-20 (Aronow, 1973; Froelicher et al, 1974; Cumming et al, 1975). We believe that the evidence of the exercise test result, when added to the other risk factors assessed, would enable one to provide better advice to the subject contemplating strenuous sporting activity. In the present state of knowledge, however, it would not be justifiable to proceed from a positive exercise test result in an asymptomatic subject to either thallium scintigraphy or coronary angiography.

We appreciate the potential cost of such a programme, but feel this could be minimised by screening the population at greatest risk, i.e. males > 35 years. Facilities for this could perhaps be made available on a payment basis within the NHS or could be provided by individual sports centres.

Changes in individual sports

Certain sports could be made safer by alterations in their rules. In boxing, title fights are now conducted over 12 rounds instead of 15, and Olympic boxers now require to wear “head guards”. Possibly by improving the environment of some sports, a reduction in mortality may occur. Improved ventilation and temperature control on squash courts may be beneficial. Although it would seem commercially unviable at the present, alterations in court design may be desirable. Various changes have recently been suggested such as removing the back wall, or making the court bigger — both of these measures would reduce the length of rallies and perhaps place more emphasis on racquet head skill, rather than physical effort.

Useful Precautions

Adequate warming-up and warming-down should be encouraged, this may reduce the number of dysrhythmic deaths. Likewise the discouragement of smoking and

### Table I

Follow-up studies using maximal treadmill test to screen asymptomatic men for latent coronary heart disease.

<table>
<thead>
<tr>
<th>Main investigator</th>
<th>Years followed-up</th>
<th>Exercise test used</th>
<th>ST segment response</th>
<th>No. (%) of total population</th>
<th>No. developing CHD (%) of ECG response group</th>
<th>Predictive value</th>
<th>Relative risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bruce and McDonough (221 subjects)</td>
<td>5</td>
<td>Bruce test (CB)</td>
<td>Abnormal</td>
<td>22 (10)</td>
<td>3 (13.6)</td>
<td>13.6%</td>
<td>13.6</td>
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<tr>
<td>Aronow (100 subjects)</td>
<td>2.5</td>
<td>Bruce test (V)</td>
<td>Abnormal</td>
<td>13 (13)</td>
<td>3 (23)</td>
<td>23%</td>
<td>20</td>
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<td>Froelicher et al (1390 subjects)</td>
<td>6.3</td>
<td>Balke test (CC)</td>
<td>Abnormal</td>
<td>140 (10)</td>
<td>28 (20)</td>
<td>20%</td>
<td>14.3</td>
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CHD = Coronary heart disease  
ECG = Electrocardiography
avoidance of a hot bath or shower may reduce the harmful effects of increased levels of catecholamines, free fatty acids and potassium after exercise. Because of the risk of hyperpyrexia and heat stroke, vigorous activity in extreme heat should be avoided. The Boston Marathon in 1973, in which one runner died of a myocardial infarct was run when the environmental temperature was between 30°-35°C (Sheehan, 1976). Fluid and electrolyte loss should be compensated, particularly during endurance events such as marathon running. Although feeding stations are available at most marathon events, not all runners utilise this service adequately. Hypothermia following cessation of activity, particularly in marathon runners, should be avoided by preventing heat loss. However, the practice of supplying aluminium foil "space blankets" to finishing competitors is an expensive and ineffective way to do this. It may be better for competitors to have their tracksuits made available at the finishing point.

CONCLUSIONS

Many may take the view that the preventive measures above are unnecessary because of the low statistical risk of sudden death in sport. However, the extent of the problem is probably grossly underestimated. We do not know the prevalence of non-fatal coronary events during exercise, and are dependent on media reports for information on sudden deaths. It has been estimated, that in squash alone, 27 deaths occur per annum in the UK (Fowler, 1980). If this is true then there are likely to be a considerable number of deaths in other sports, all amounting to an appreciable mortality. The suggestions expressed in this paper may help reduce the occurrence of sudden cardiac death in sport.

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MARATHON MEDICINE MEETING
20th April, 1985 (Saturday before London Marathon)
Held in The Royal Festival Hall
Details from Dr. Dan Tunstall Pedoe, Cardiac Dept., St. Bartholomew's Hospital, London EC1A 2BE

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INTERNATIONAL SYMPOSIUM ON SPORTS CARDIOLOGY
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