HEART RATE RESPONSE AND ECG MONITORING IN VETERAN SQUASH PLAYERS.

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January 1997

Completed in partial fulfillment of the degree of M Med Sci.

In memory of Mr R R Sibbald who suffered a fatal coronary thrombosis, in 1995, at the age of 56, within 1 hour of playing tennis.

I, Helen Mary Sibbald, declare that the work presented in this thesis is original and has been carried out by myself except where acknowledged otherwise. This work has not been presented for any other degree at this or any other university.

Signed ....HMSibbald

Date ....18·2·1997

Place ....DURBAN....

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ABSTRACT

The incidence of sudden death during or after squash play has become a source of concern. In order to screen for coronary artery disease, exercise stress testing has been advocated, by the American College of Sports Medicine (1986), for those at or above the age of 45 already exercising or before embarking on exercise.

Eighteen veteran squash players (mean age 49 ± 3 yr) took part in the study. Heart rate response was monitored throughout a squash match and for an hour after play. ECG changes were monitored for one hour after squash play.

Mean heart rate, throughout playing time was 148 ± 16 beats per minute (range 118 - 168 bpm), representing 86.7% of Predicted Maximum Heart Rate (PMHR). Mean maximal heart rate was 169 ± 14 bpm (range 141 - 186 bpm), representing 98.8% of PMHR. Thus squash represents a very high intensity activity for these players. On subsequent ECG monitoring, no abnormalities were detected.

The results of this study confirm that squash is an extremely high intensity sport and that even veteran players play at a level close to their maximal. This level of play did not provoke subsequent cardiac arrhythmias in this small group of players, contrary to an earlier study that reported arrhythmias in one third of a group of younger players in the post match period.

CHAPTER 1

INTRODUCTION

The game of squash has become increasingly popular in South Africa and throughout the world. In particular, there are a large number of veterans (i.e. over the age of 45), participating in squash. An indication of this is the 1996 Veterans Interprovincial Tournament in South Africa, which with 738 people participating, is the largest squash tournament in the world. With this growth in popularity, there have been several reports of sudden death associated with squash, either during the game or in the hour after play. In a study in the United Kingdom which investigated 60 sudden deaths associated with squash, the mean age of the subjects was 46 years (Northcote, Flannigan, and Ballantyne, 1986). The American College of Sports Medicine recommends a stress electrocardiogram (ECG) for those at or above the age of 45 years, desiring to begin exercising or for those at the age of 45 years already participating in exercise (American College of Sports Medicine, 1986).

Very few studies, however, have been conducted to elucidate the incidence of an underlying cardiac condition in veteran sportsmen that may justify mass exercise stress test screening prior to participation.

Northcote, MacFarlane, and Ballantyne (1983), showed that in younger squash players (under the age of 43 years), ECG changes occurred during and after squash which were not found on subsequent exercise stress testing. Furthermore, a recent study showed that a typical physician's exercise stress test failed to elicit the high heart rates observed during the squash game itself and perhaps not surprisingly, no ECG abnormalities were detected by the stress test. (St Clair Gibson, personal communication). These findings question the necessity for and the value of the cardiac stress test.

The aim of the current study was to determine the heart rate observed during a squash match and to investigate the incidence of cardiac arrhythmias in the hour after the match in veteran squash players.

CHAPTER 2
LITERATURE REVIEW

2.1 RECOMMENDATIONS FOR STRESS TESTS PRIOR TO EXERCISE PARTICIPATION :

The American College of Sports Medicine (ACSM) (1986), recommends that ,"at or above the age of 45, it is desirable for these individuals to have a maximal exercise test before beginning exercise programs. It is also desirable in those who are already exercising once they reach age 45."

The stress test is described by McArdle and Katch (1986), as the systematic use of exercise for i) ECG observations and ii) to evaluate the adequacy of physiological adjustments to the metabolic demands of exercise, that exceed the resting requirements. These authors believe that the exercise stress test provides a "reliable and quantitative index of the person's functional impairment if heart disease is detected. This enhances the accuracy of diagnosis and subsequent exercise prescription."

Chung (1980), states that, the stress ECG is of great value for those planning to participate in vigorous or intense physical activity (be it sport or occupation). This author argues that the exercise ECG not only provides useful information regarding the identification of coronary artery disease (CAD) and therefore yidentification of the individual who may be at increased risk of a cardiac event whilst exercising, but it also provides an "essential" guideline concerning the individuals functional capacity (physical capacity). For example, a sport or occupation which requires an exercise workload beyond the individuals' physical capacity (as determined by the exercise ECG test) is hazardous and sudden death may be more likely to occur in the susceptible individual. If a ventricular arrhythmia is provoked by the exercise ECG test, the individual should not engage in any sport/exercise program until the arrhythmia is fully evaluated, treated and all aspects of cardiac status are assessed. Chung (1980) concludes that an exercise ECG test is recommended before engaging in an exercise programmme or sport for men above 40-50 years old or individuals with one or more known risk factors or proven coronary heart disease.

2.2 STRESS ECG - LIMITATIONS :

Limitations exist when using exercise ECG testing of asymptomatic patients as a screen for future cardiac events. Firstly, there are a large number of abnormal tests that represent false positive results. Secondly, exercise ECG testing fails to identify the majority of patients who later have cardiac events including myocardial infarction (MI) and sudden death (Coplan and Fuster, 1990).

2.2.1 False positives:

ST-segment depression is the most common ECG manifestation of exercise-induced myocardial ischaemia. The probability and severity of coronary artery disease (CAD) is directly related to the amount of depression. A false positive test is one in which an abnormality such as exercise induced ST-segment depression, is detected in an asymptomatic person with a normal coronary angiogram. There are, however, a number of known factors, other than CAD, which can cause a positive result with respect to ST-segment depression. These are digitalis, mitral valve prolapse syndrome, Wolff-Parkinson-White syndrome, pre-existing left bundle branch block or left ventricular hypertrophy, or other cardiac diseases. Furthermore, there is often a failure of a physician to recognise the "labile ST-T wave change" or non-specific ST-T wave change in many healthy but anxious individuals. This is a normal variant that should not be misdiagnosed as a positive exercise ECG test (Chung, 1980). Exercise cardiac scintigraphy has been shown to be effective in detecting individuals with exertional ST-segment depression but normal coronary angiograms (Sheffield, Reeves, Blackburn, Ellestad, Froelicher, Roitman, and Kansal, 1977). Exercise scintigraphy makes use of Thallium injection followed by myocardial imaging and interpretation of the images for perfusion defects.

2.2.2 False negatives :

Failure to identify patients who later have acute cardiac events is a serious limitation of a screening exercise test. McHendry et al. (1984) conducted a study involving a screening exercise test in 916 asymptomatic men and then follow-up for a period of 8 - 15 years. They found that the number of men in the group of 833 with a negative stress test, suffering acute cardiac events (25 myocardial infarctions and 7 sudden deaths), far exceeded the number of acute events (1 myocardial infarction and 1 death) seen in the group of 83 patients with a positive stress test.

The patients with a positive stress test had a higher risk factor profile for sudden death than the patients with a negative stress test but more patients in the negative stress test group actually experienced sudden death. The authors' explanation was that those who suffered myocardial infarctions or sudden death, may have had thrombosis or vasospasm at the site of a coronary stenosis that was not sufficiently critical to stimulate the development of a collateral circulation. Those with abnormal ST-segment response, on the other hand, did develop collateral circulation to "protect" them from myocardial infarction (McHendry, O'Donnell, Morris, and Jordan, 1984).

Coplan and Fuster (1990) examined the pathophysiology of acute cardiac events to determine why the exercise test fails to detect patients who subsequently have MI and sudden death, and discussed implications for screening.

They concluded that the ECG exercise test is designed to detect ischaemia resulting from an imbalance of myocardial oxygen supply and demand during exercise. This usually occurs secondary to a flow-limiting obstruction in a coronary artery. Failure of an exercise test to identify a patient at risk for an acute cardiac event may result from :

i) failure to perform sufficient exercise to provoke cardiac stress.
ii) inadequate sensitivity of the test to detect ischaemia.

Investigations, however, have suggested that many victims of sudden death / MI may not have had flow-limiting coronary artery stenosis when tested. The acute coronary syndrome develops secondary to an active process that can quickly change a non-occlusive stenosis to one associated with total obstruction. This is used to explain why the screening exercise test fails to show signs of myocardial ischaemia. At the time of testing, there was no "critical" obstruction present. Critical obstruction may result when a non-occlusive atherosclerotic plaque is transformed into a severe occlusion by overlying thrombus. This may occur following plaque rupture, where collagen and other factors are exposed to the blood, promoting platelet adhesion and aggregation. The non-occlusive plaque would not have produced sufficient ischaemia to be detected as an abnormal exercise stress test. The 'new' occlusion may however lead to sudden death, particularly in the absence of collaterals (Coplan and Fuster, 1990).

With regard to the implications for screening, Coplan and Fuster (1990) felt that tests are required that are more sensitive to the presence of occlusive and non-occlusive coronary lesions. Thallium scans have been found to be abnormal in 75% of patients with false negative ECG stress tests (Hakki, De Pace, Colby, and Iskandrian, 1983). If however, rupture of an atherosclerotic plaque is the primary mechanism initiating acute ischaemic syndromes and a plaque was not previously sufficient to result in an abnormal thallium scan, then these scans also have limitations in their ability to identify patients at risk of sudden death. Furthermore these scans are expensive and not practical for screening large numbers of asymptomatic individuals.

Sheffield et al., (1977), concluded that exercise stress testing should be performed in asymptomatic persons only to determine the relative risk of future coronary events and not to predict the angiographic appearance of their coronary arteries. Since coronary artery disease is prevalent in patients consulting physicians for chest pain, the stress test is most relevant in this group. The stress test is most useful in aiding the investigation of chest pain and therefore aiding appropriate medical management.

The exercise stress test is perhaps not as relevant in asymptomatic patients. The conclusion of a study by Detrano and Froelicher (1988), was that although exercise testing is helpful in evaluating the presence and severity of cardiac arrhythmias, it is of little assistance in determining their prognostic significance. "Careful evaluation of exercise testing as a screening device for asymptomatic patients reveals that it is somewhat disappointing in this application" (Detrano and Froelicher, 1988). Epstein and Maron (1986) conclude that, after screening evaluation, it must be understood that people with the designation, "low risk", can never entirely rule out the possibility of sudden death as a result of a cardiac event. Also those with the designation, "some risk", may warrant additional testing to establish more definitively, the diagnosis and its functional implications.

In summary, it is apparent that there are limitations of the exercise stress test that need to be considered when it is used as a screening test in asymptomatic patients, and other possible methods considered. The recommendation of the exercise stress test as a necessary screening test in older sportspeople is questionable.

2.3 HEART RATE AND SQUASH:

Several studies have examined heart rate responses to squash play. Maximum heart rates, expressed as a percentage of predicted maximum heart rate (PMHR), in various studies, are compared in table 1.

TABLE 1 MAXIMUM HEART RATE (HRMAX) OBTAINED DURING SQUASH IN VARIOUS STUDIES.

		Subjects	Mean age (yrs)	HRMAX (bpm)	% PMHR
Blanksby et al. (1973)	middle-aged	25	42.4	174.8	98.4%
	middle-aged active	25	43.9	151*	85.5%
	"a-grade"-active	25	25.6	162.7	83.6%
Blanksby et al. (1980)	middle-aged	9	44.8	157	89.6%
	middle-aged active	9	44.2	160	91%
	"a-grade"-active	9	25.8	167	86%
Docherty and Howe (1978)		30	29		80 - 85%
Beaudin et al. (1978)		10	29.4	185 ± 13	97%
Northcote et al. (1983)		21	33	170 ± 16	90%
St Clair Gibson (1997)	league players	10	49	177 ± 8	104%
	social players	10	53	172 ± 8	103%

* value calculated as not given by the author in original paper

The two studies by Blanksby et al. (1973) and Blanksby et al. (1980) appear to show conflicting results. In the 1973 study, the maximum heart rate in the middle-aged sedentary group was found to be higher ; and the maximum heart rate in the middle-aged active group was found to be lower than in the 1980 study. The authors' explanation for these results was that, in the 1980 study, an extra 20 second rest was taken every 3 minutes. This could have been sufficient to allow greater recovery between rallies.

Blanksby, Elliot, Davis and Mercer (1980), noted that the maximum heart rates, during a game, of otherwise sedentary middle-aged players (mean age 45 years) tended to be

generally lower than those in habitually active players. It was concluded that the active group could probably maintain a higher level of activity.

Both the middle-aged groups (sedentary and active) had lower heart rates than younger, "A-grade" players (mean age 26). But when related to PMHR, both middle-aged groups were exercising at a higher percentage of PMHR than the younger players (89.6% and 91% versus 86%). It is evident, when comparing these results to those in the studies shown in table 1, that "A-grade" players recorded heart rates similar to those found in Blanksby, Elliot, and Bloomfield (1973) and Docherty and Howe (1978).

St Clair Gibson (personal communication) compared the cardiovascular responses in a routine clinical stress test to the cardiovascular responses in different sports, including squash, in veterans aged between 45 and 55. Squash players were divided into 2 groups. The first group were league players (mean age 49 ± 5 yr) and the second group were social squash players (mean age 53 ± 5 yr). Both groups played 2 squash games with heart rate monitors. All subjects performed an exercise stress test. Maximum heart rates (177 ± 8 bpm and 172 ± 15 bpm; 104% of PMHR and 103% of PMHR respectively) achieved during the squash games were significantly higher than the maximum heart rates attained during the exercise stress tests (153 ± 8 bpm and 156 ± 12 bpm). The author concludes that "caution should be exercised when advising patients to exercise freely on the basis of a routine stress ECG." It would appear however that these exercise stress tests were submaximal and therefore their results cannot be compared to those attained during squash. Neither can it be confidently claimed that the maximum heart rates achieved during squash reflect the individuals' true maximal heart rate.

Several studies have examined average heart rates during squash play. Average heart rates all exceeded 75% of predicted maximum heart rate (PMHR) (see Table 2). This corresponds to an intensity of exercise that would place unfit individuals under a high circulatory strain.

TABLE 2 AVERAGE HEART RATES, DURATION OF PLAY AND % PMHR DURING SQUASH IN VARIOUS STUDIES.

		mean age	duration	mean	%
Beaudin et al. (1978)		29	45	155 ± 8	81%
Docherty et al. (1978)		30	30		79%
Mercier et al. (1987)		29	30	154 ± 16	81%
Montpetit et al. (1987)		27	50	147 ± 18	76%
Northcote et al. (1983)		33	40	149 ± 18	80%
Van Rensburg et al. (1982)		26	68	161 ± 12	83%
St Clair Gibson (1997)	league players	49	26	159±10	93%
	social players	53	26	155±12	93%

In comparison to tennis, squash places a more severe workload on the myocardium as the body is subject to sudden and vigorous demands. In squash, the playing environment is usually one with a high ambient temperature and humidity with poor ventilation, leading to an increase in heart rate. The intervals between rallies are shorter, and less time is spent retrieving the ball. The game is more continuous and little recovery time is allowed during a game. The nature of the game is one of longer and more intense rallies. Northcote, MacFarlane, and Ballantyne (1983) state that squash is usually played by individuals of a highly competitive or aggressive nature who would rather win a point than submit to fatigue or discomfort, increasing the risk of a cardiac incident. Fowler and Cox (1984), describe squash as highly competitive, demanding fast reactions leading to the release of large quantities of catecholamines. Squash has a large anaerobic component resulting in a considerable oxygen debt (Fowler and Cox, 1984).

In summary, mean game heart rates indicate that squash is a high intensity activity potentially placing unfit players under cardiac strain or at risk of a cardiac event. During a squash game, near-maximal heart rates may be experienced.

2.4 ECG AND SQUASH:

Squash, as a result of its high intensity, is potentially more liable to provoke cardiac arrhythmias than many other sports. Northcote, MacFarlane and Ballantyne (1983) monitored heart rate and ECG changes during and 30 minutes after squash play in 21 healthy, fit, male squash players. Mean age was 33 ± 6.5 yr (range 23-43 yr). The following ECG abnormalities were found:

1) ventricular extrasystole during play (in 7/21), ventricular extrasystole after play in 7/21,of whom 6/7 had ventricular extrasystole during play.

2) ventricular tachycardia during play in 2/21, ventricular tachycardia after play in 1/21.

3) premature atrial contraction during play in 13/21, premature atrial contraction after play in 6/21, of whom 4/6 had premature atrial contraction during play.

Of interest, is the high proportion of ventricular extrasystoles found in the 30 minutes after play (as many as during play itself), a time referred to by many as the "vulnerable post exercise period." Ventricular extrasystoles induced by exercise may induce sudden death and therefore these subjects are at increased risk.

In this study, by Northcote et al. (1983), however, preparticipation clinical assessment and subsequent exercise ECG testing failed to identify those subjects who developed ventricular arrhythmias when playing or after playing. This indicates that exercise stress ECG testing may not be sensitive enough to detect arrhythmias which may occur during or after squash.

The authors, offered several possible causes of these extrasystoles:

1) myocardial ischaemia, on the basis of the presence of asymptomatic coronary atherosclerosis in some players,
2) cardiac arrhythmias occurring in response to thermal stress, as squash is often played in excessively warm environments which tend to be poorly ventilated,
3) the increase in catecholamines during exercise leading to myocardial ischaemia and arrhythmias in the presence of coronary artery constriction,
4) metabolic changes, such as hyperkalaemia, precipitating arrhythmias.
5) venous pooling at the end of play, lowering venous return. This, with continuous sympathetic nervous system stimulation may precipitate coronary artery insufficiency,

leading to arrhythmias. This may be exacerbated by the thermal effects of a hot bath or shower.

Kohl, Powell, Gordon, Blair and Paffenbarger (1992), believed these potentially fatal arrhythmias to result from the combination of a triggering event, usually a premature ventricular contraction, and a susceptible myocardium. In the case of exertion-related sudden death, a number of exercise related factors combine with pre-existing cardiac disease to culminate in the genesis of a susceptible myocardium and fatal triggering event. These factors include: excessive increase in myocardial oxygen demand, changes in sympathetic and parasympathetic tone, release of coronary vasoconstrictor substances, increased blood coagulability, lactic acidosis, electrolyte derangements, elevated free fatty acid concentration, the sudden decrease in cardiac output on abrupt cessation of exercise, and excessive increase in body temperature. Kohl et al. (1992) postulated that it is the combination of exercise and underlying heart disease rather than exercise alone that usually leads to the final common pathway of fatal arrhythmia. The healthy heart, even when subjected to strenuous exertion, appears to be protected from fatal arrhythmias except where profound electrolyte disturbances, heat stroke or drug abuse occur.

In summary, several possible causes of ECG abnormalities, accompanying exercise, have been offered and it is clear that the individual may not only be at risk during the game itself but may also be at risk immediately after the game. It is suggested that an ECG abnormality may be more likely to be detected during squash and in the hour immediately after than in a clinical exercise stress test.

2.5 SUDDEN DEATH AND EXERCISE / SQUASH :

Sudden cardiac death in people over 35 is most often related to coronary artery disease (CAD). CAD is found at autopsy in 80% of people over 35 who die suddenly during or shortly after exercise. It is estimated that 43% of these are reported to have been previously asymptomatic and unaware of their underlying cardiac disorder (Kohl et al., 1992). Myocardial ischaemia is said to create the setting in which a trigger event such as premature ventricular contraction and a susceptible myocardium combine to evoke a potentially lethal arrhythmia. Such a trigger may be myocardial ischaemia accompanied by dramatic cellular electrophysiological changes in the affected area as well as the delayed consequences of reperfusion producing electrophysiological irritability. Mechanisms by which fatal myocardial ischaemia develops in people with CAD who perform an acute bout of exercise are possibly:

1) exertion related fissuring of a fragile atherosclerotic plaque with resultant thrombus formation, transforming a previously non-occlusive plaque into a total or near-total occlusion.
2) a plaque, itself, preventing myocardial oxygen supply from meeting the increased myocardial oxygen demand that accompanies exercise.
3) secondary to exercise-induced coronary artery spasm (Kohl, Powell, Gordon, Blair, Paffenbarger, 1992).

Epstein and Maron (1986) concluded that the primary mechanism of sudden death in healthy athletes is cardiovascular in nature. In the middle-aged or older athlete, CAD is the most significant cause of death. The major cause of sudden death in the young athlete is hypertrophic cardiomyopathy or related conditions characterized by left ventricular hypertrophy, aortic rupture or congenital coronary artery abnormalities.

Kohl et al. (1992) described hypertrophic cardiomyopathy to be characterized by inappropriate myocardial hypertrophy of a non-dilated left ventricle. Most of the associated deaths were presumed to be due to ventricular arrhythmias that resulted from the interaction between exercise and the structural and functional abnormalities of hypertrophic cardiomyopathy. The cause of death in hypertrophic cardiomyopathy may have been as a result of :

1) myocardial ischaemia which occurs during exercise in people with hypertrophic cardiomyopathy as a result of :

i) an imbalance between the oxygen supply and demand of a markedly increased muscle mass,

ii) narrowing of intramural coronary arteries and

iii) prolonged maintenance of left ventricular wall tension with associated slower than normal decrease in impedance to coronary blood flow during diastole; or

2) an abrupt decrease in stroke volume perhaps due to impaired ventricular filling, due to increased left ventricular wall thickness or impaired ventricular emptying due to left ventricular outflow tract obstruction.

Northcote, Flannigan, and Ballantyne, (1986), reported on sixty deaths in the United Kingdom associated with squash. Eighty-nine sudden deaths associated with squash occurred in 8 years but in only 60 of the cases was there sufficient information for a detailed investigation. In this study, 59 of the 60 were males, and the mean age was 46 ± 10 yr (range 22 - 66 yr). Sudden death was defined as death occurring within 1 to 24 hours of onset of the symptoms. In this study, all of the subjects collapsed while playing squash or *within one hour* of playing. All except one died within an hour of play. The remaining subject died 6 hours after play. The causes of death were CAD (51), valvular heart disease (4), cardiac arrhythmia (2), non-cardiac (2), and hypertrophic cardiomyopathy (1). Northcote et al. (1986) suggested that coronary artery spasm may be the cause of coronary artery occlusion during vigorous exercise, especially in those where a fresh occlusive thrombus was not found. Recently it has been suggested by Ciampricotti et al. (1990) that cardiac arrhythmias resulting from reperfusion of previously ischaemic areas of myocardium may be responsible for the frequent occurrence of sudden death after the cessation of exercise.

Northcote, Flannigan and Ballantyne (1986), observed that many individuals with known medical conditions continue to play squash. "Men in middle age seem reluctant to acknowledge that they may be in poor physical condition / health. We and others (Northcote, Evans, and Ballantyne, 1984), have noted that sportsmen tend to deny physical infirmity and prodromal symptoms."

Many people play squash because they hope that regular vigorous exercise may help to alleviate or prevent heart disease. Siscovick, Weiss, Fletcher and Lasky (1984), found that

the risk of primary cardiac arrest is transiently increased during vigorous exercise. High intensity activity was classified as that which required approximately 6 KCal or more per minute. Squash has been recorded to require as much as 14,8 KCal per minute in a 70-kg man. (McArdle, Katch, and Katch, 1986). Because the increase in risk was particularly large for men with low levels of habitual activity, their data supported the clinical impression that unusual exercise may be associated with increased risk. Even among men who were habitually vigorous, the risk of primary cardiac arrest was increased during exercise. However, although intense physical activity may be one of the factors that can precipitate primary cardiac arrest, *habitual* participation in this activity is associated with an overall reduction in the risk of primary cardiac arrest and CAD. Of interest, is that in Northcote's study (1983), all subjects had been playing squash at least twice a week for two years. They were involved in regular physical activity but still showed potentially dangerous ECG changes during and after squash play (Northcote, MacFarlane, and Ballantyne, 1983).

Northcote and Ballantyne (1983) concluded that a small risk of sudden cardiac death exists in subjects participating in vigorous sport but that this risk is greater in those with asymptomatic cardiovascular disease. Preparticipation medical screening would detect overt, potentially fatal cardiovascular disease. There is, however a group of people, in general, over 40 years of age, with asymptomatic coronary artery disease who would not be identified by screening. Those over the age of 40 years with an increased risk profile for coronary artery disease, therefore, should participate in less vigorous sport. Sportsmen should not ignore untoward symptoms, and if symptoms do occur, they should either stop or avoid participation and seek medical advice (Northcote and Ballantyne, 1983). Practical recommendations with respect to cardiac risk factors are shown in appendix 1.

The present study is similar to the one by Northcote et al. (1983), except that it involves subjects of 45 years and over who are potentially a higher risk group. This study aims to quantify the average and maximal heart rates experienced during a game of squash and to determine the incidence of ECG changes after the match in the older age group, who might otherwise be advised to have an exercise stress test if already exercising or prior to embarking on exercise. The possibility exists that, by a natural process of selection, those with cardiac problems or potential problems, have already stopped playing of their own accord or, that those who continue to play have adopted a healthier lifestyle and have relatively fewer risk factors and may not exhibit the high incidence of post match ECG changes reported by Northcote et al. (1983) in younger players.

CHAPTER 3

METHOD

18 male volunteers consented to participate in this study. All signed an informed consent form and a questionnaire relating to risk factors was answered, which is included in Appendix 2. All players were of similar ability and had been playing squash for several years. Electrodes were positioned prior to the game so that leads could be quickly attached to minimise the delay at the end of the match. A preparticipation resting ECG was recorded. A match was considered the best of 5 games. Heart rate was monitored by means of a "POLAR" heart rate monitor (Polar Electro Oy - Finland) and was recorded every 15 seconds during the game and for the hour after play. On completion of the match, subjects were rapidly fitted with a five-lead Holter monitor. Electrodes were placed over the manubrium or mid-clavicular line, (in line with the manubrium), in the fourth intercostal space on the right and in the fifth intercostal space in the mid-clavicular line on the left and right. The ECG was recorded for one hour after the game during which time the subjects went about their normal post match activities. The ECG was not recorded during the game as the Holter monitor was found to be too bulky to allow normal play. Holter tape analysis was carried out in a routine cardiac monitoring laboratory by a blinded technician. The reports were read independently by a cardiologist who was blinded to history and risk factors. In the event of post match ECG abnormality, an exercise stress test would be performed.

Statistical methods:

Averages were calculated for playing time, temperature, heart rate and maximum heart rate during squash play.

Range was calculated for heart rate and maximum heart rate.

Analysis of variance was performed to investigate the intergame variability of percentage PMHR.

Analysis of variance was performed to investigate the percentage playing time spent at different percentages of PMHR.

A paired T-Test was performed to compare average heart rates in the easiest and hardest games.

CHAPTER 4

RESULTS

All subjects were right handed. Their mean age was 49 ± 3 yr (range 45 - 54 yr). Subject characteristics are displayed in table 3. Table 4 shows the cardiac risk factors determined from the risk questionnaire (see Appendix 2). An explanation of the risk factors is provided in Appendix 3.

From Table 4 it can be seen, that 5/18 had 1 risk factor, 8/18 had 2 risk factors, 4/18 had 3 risk factors, 1/18 had 4 risk factors. As all subjects were males over 45, age and gender was a risk factor common to all 18 subjects. All exercised regularly (range 3 - 10 hours a week). Eighty-three percent consumed alcohol and 83% consumed caffeine.

Mean playing time was 42.1 ± 14.1 minutes (range 18.3 - 61.0 minutes) in a mean ambient temperature of 25.6 ± 2.2 °C (range 21 - 28°C).

HEART RATE :

The heart rate monitor malfunctioned once, therefore only 17 subjects are included in the heart rate analysis.

The mean *maximum* heart rate recorded, for the whole group, during play, was 169 ± 14 bpm (range 141-186). This represents a mean of 98.8% of the predicted maximum heart rate for the whole group, where PMHR as defined by Astrand (1986) and McCardle, Katch and Katch (1986) is 220 bpm minus the age of the subject. Individual maximum heart rates during play are displayed in Table 5.

Several players' individual absolute maximum heart rate values, recorded during play, were higher than their PMHR's (see fig 1). Nine subjects recorded absolute maximum heart rates during squash greater than their PMHR, and 8 subjects' absolute maximum heart rates during squash were below their PMHR. Fourteen out of seventeen subjects fell within the 2 standard deviations described by Astrand for PMHR in a population (i.e ± 20 beats) (see fig 2). It is not possible to conclude, from the results, that the maximum heart rate during squash represents the true maximal heart rate possible for all the individuals.

If one considers intergame variability, analysis of variance showed no significant difference in % PMHR between the games (see Table 6). Three subjects' *average* game heart rates, as a percentage of PMHR, in their hardest game, exceeded their PMHRs. In fact, all three of them had average heart rates greater than PMHR in more than 1 game. Figure 3 shows individual average heart rates and individual average heart rates in the hardest game, again indicating the intensity of the games. It is evident that there is some variability amongst individuals' hardest games (average heart rates ranged from 72% to 104%).

The range for average heart rates in the easiest game was 66% - 96% PMHR (mean 83 ± 8 bpm) and the range for the hardest game was 72% - 104% PMHR (mean 91 ± 9 bpm). There was therefore an 8% difference between average heart rates in the easiest and hardest games. ($p < 0.0001$). Furthermore, it is noticeable that some individuals' average heart rates, in their easiest game, were higher than some individuals' average heart rates in their hardest game.

Percentage of playing time spent at < 80%, 80 - 90%, and >90% of PMHR is represented in figure 4. It is clear that a large portion of the playing time is spent at >90% of PMHR (see fig 4). Analysis of variance showed no significant difference in the percentage of playing time spent at the intensity categories listed above.

The *average* heart rate, for the group, throughout the playing time was 148 ± 16 bpm (range 118 - 168 bpm) (see table 5). This represents 86.7% of the PMHR. Individual average heart rates are displayed in fig 3, as a percentage of PMHR.

ECG :

All resting ECGs were normal. Of the post-exercise recordings, 13 out of 18 subjects had ECG recordings suitable for study, 3/17 had poor recordings and were therefore excluded from the ECG analysis. 2/17 reports were not returned from the laboratory and were therefore excluded. No abnormal ECG changes were detected in any of the subjects.

Unifocal ventricular ectopics (Lown grade 1, see Appendix 4) were noted in one of the subjects but reported to be a normal variant and of no consequence.

CHAPTER 5
DISCUSSION

The first major finding of the current study, is that the maximum heart rate achieved during squash play represented a very high percentage of PMHR. This can be compared to the mean findings of previous studies, undertaken in players of various ages, that were summarized in table 1. Generally, these studies report a maximum heart rate during squash that is very close to the individuals' predicted maximum heart rate. Of interest, is St Clair Gibson's finding that maximum heart rates obtained during squash, in his group of veteran players, exceeded the PMHR (see Table 1).

Blanksby et al. (1973) commented on maximum heart rate and the corresponding percentage of PMHR (as defined by Astrand and Rodahl, 1986), in 3 groups of squash players. The middle-aged sedentary group reached 98.4% of PMHR; the middle-aged active group reached 85.5%; and the "A-grade" active group reached 83.6% of PMHR (see Table 1). The middle-aged sedentary group, who in their everyday activities have a limited work output, abruptly exposed themselves to prolonged and severe workloads, attaining 98.4% of maximum heart rate. The authors concluded that the increase in heart rate and blood flow may lead to cardiac arrhythmias and that this problem could be accentuated in individuals of a competitive nature who are prepared to suffer discomfort and acute feelings of fatigue in order to win and stated that, "For men in this category, squash could be a dangerous game" (Blanksby, Elliot, and Bloomfield, 1973). The middle-aged active and "A-grade" players participated within more tolerable limits of heart rate, and there appears to be less risk for them to continue playing. The present study group were more like the middle-aged active group in that they all exercised a minimum of 3 times a week. The maximum heart rate, however, corresponded to 98% of PMHR. This is closer to the values achieved by the middle-aged sedentary group.

A possible explanation for Blanksby's results is that when comparing heart rates of trained athletes vs sedentary people, heart rates increase with exercise to a lesser extent in trained athletes. The middle-aged active group would therefore be expected to have a lower absolute maximum heart rate.

TABLE 3 : SUBJECT CHARACTERISTICS

SUBJECTS	AGE (yr)	WEIGHT (kg)	HEIGHT (cm)	PMHR (bpm)
1	48	62	173	172
2	54	81	184	166
3	51	77	170	169
4	54	77	166	166
5	51	86	170	169
6	46	93	179	174
7	48	83	185	172
8	52	78	171	168
9	48	92	170	172
10	48	90	179	172
11	48	80	178	172
12	52	68	178	168
13	45	105	184	175
14	45	75	170	175
15	50	75	168	170
16	48	82	175	172
17	50	76	187	170
18	51	80	180	169
MEAN	49	81	176	171

TABLE 4 : RISK FACTORS

SUBJECTS	AGE (yr)	HYPERTENSION	SMOKING	INACTIVITY	OBESITY	DIABETES	FAMILY HISTORY	HIGH CHOLESTEROL	CARDIAC HISTORY	PSYCHOSOCIAL STRESS
1	48						yes			
2	54									
3	51									
4	54									
5	51							yes		
6	46						yes	yes		
7	48									yes
8	52						yes			
9	48				yes		yes	yes		
10	48									
11	48						yes			
12	52						yes			
13	45				yes			yes		
14	45		yes						yes	
15	50									yes
16	48									
17	51						yes			
18	50		yes			yes				

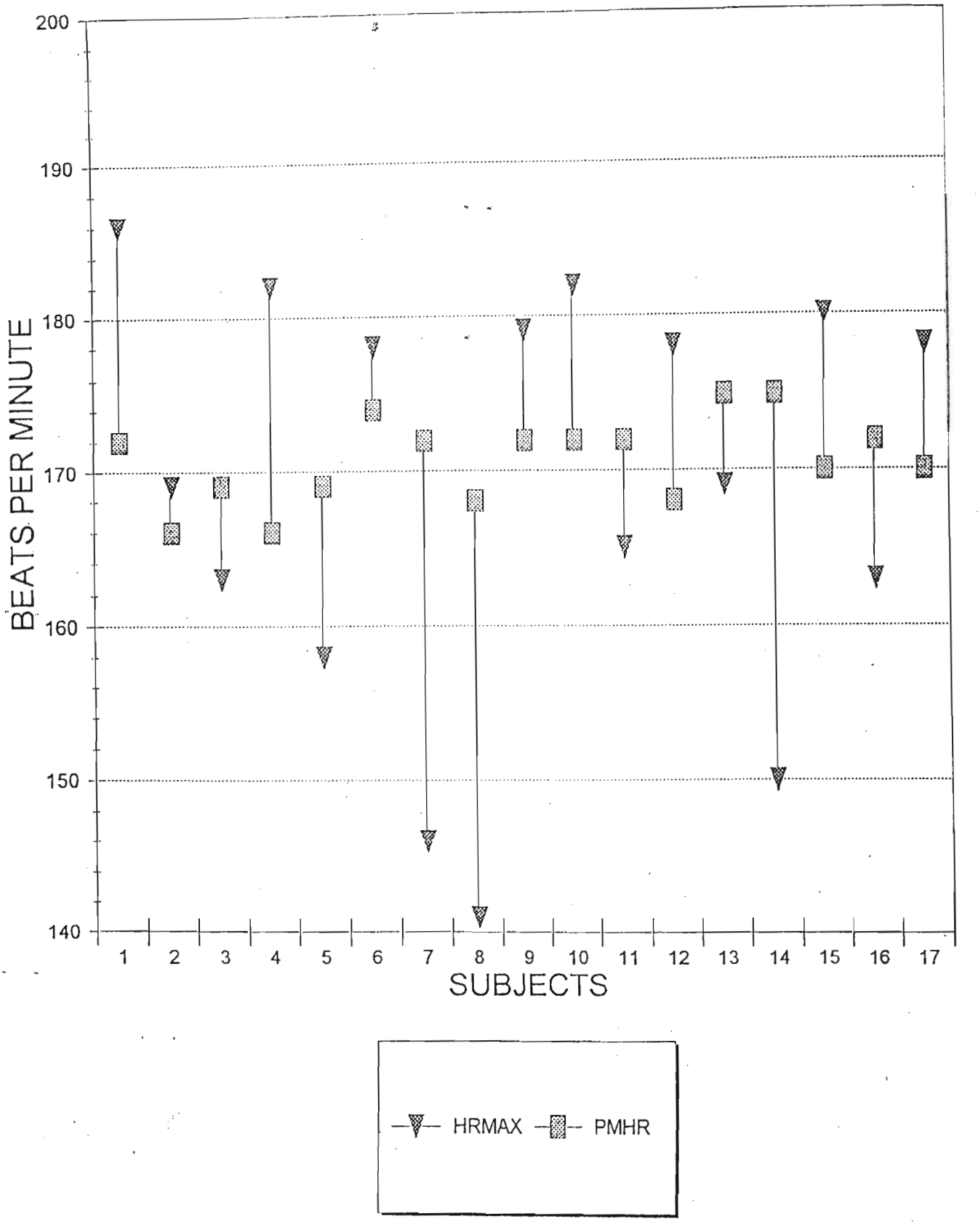

FIG 1 - ABSOLUTE MAXIMUM HR VS PMHR

The second major finding of the current study is the high average heart rates achieved during squash play. This confirms the findings of previous studies undertaken in younger players (Table 2). The subjects in this study achieved an average heart rate of 148 ± 16 bpm and were therefore playing squash at an intensity at least as high as was shown in previous studies, despite their older mean age.

However, it should be noted that when both these maximal and average values are expressed as a percentage of PMHR, an overestimation of the intensity could result. The equation commonly used to calculate PMHR (220 - age), may not be equally applicable to all individuals. Koeslag and Sloan (1976), investigated two groups of men aged 18 to 33 years, all of whom performed strenuous sport at least five times a week. One group consisted of cross-country runners and the other group of rowers, swimmers, rugby players, badminton and squash players. These authors report that the actual maximum heart rate for the runners, obtained during the VO2 max test, was 183 bpm. The PMHR as described by Astrand and Rodahl (1986) was calculated as 220 - 22.5 = 197.5bpm. The PMHR was therefore an overestimate of true maximal heart rate. In the group of other sportsmen (mean age 21.5 yr), the calculated PMHR was 220 - 21.5 = 198.5 bpm, whilst the actual maximal heart rate achieved during the maximal exercise test was 195 bpm, which was slightly lower than the PMHR. In the present study the mean age was 49 years, PMHR was therefore 220 - 49 = 171 bpm. Mean maximum heart rate during squash was 169 bpm, only 2 bpm lower than PMHR. Although it cannot be inferred that this was a truely maximal effort or maximum heart rate, it is clear that it is likely to be close to maximal. True maximum is therefore likely to be similar or *higher* than that reported by Koeslag and Sloan, 1976.

Although the mean maximal heart rate during squash for the whole group was similar to PMHR, many player's individual maximum heart rates were higher than their PMHR's as shown in figure 1. This could be an indication of the intensity of the game of squash or the potential for error when using PMHR. Astrand and Rodahl (1986), state that the standard deviation for maximum heart rate during exercise is ± 10 bpm. All but 3 subjects in the present study fell within 2 standard deviations of their PMHR during the squash game (i.e. ± 20 beats) (see figure 2). If the maximum heart rate achieved during squash was not a reflection of *true maximal heart rate,* these percentages are likely to be an overestimate of intensity.

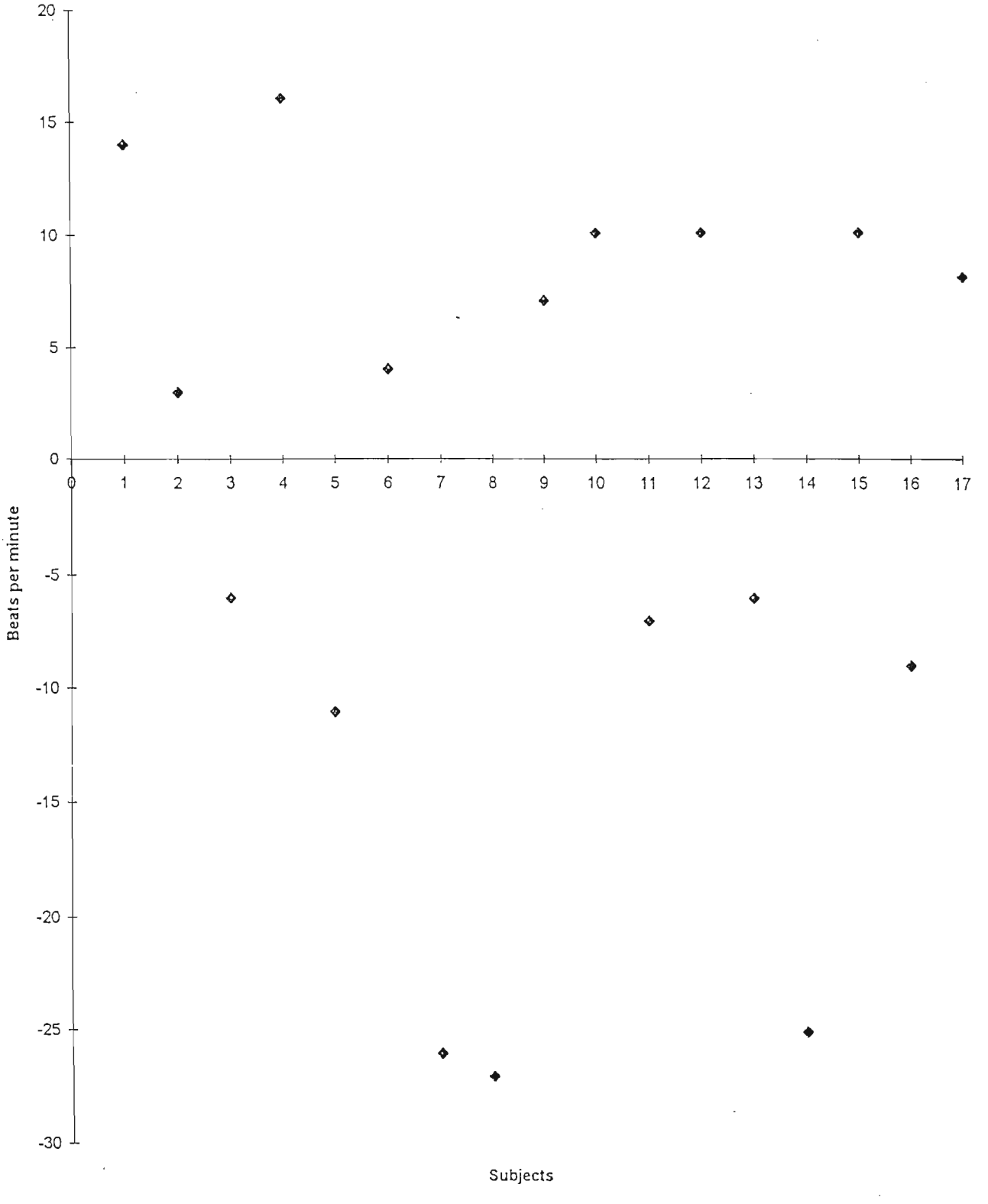

Fig 2: Individual deviation from PMHR

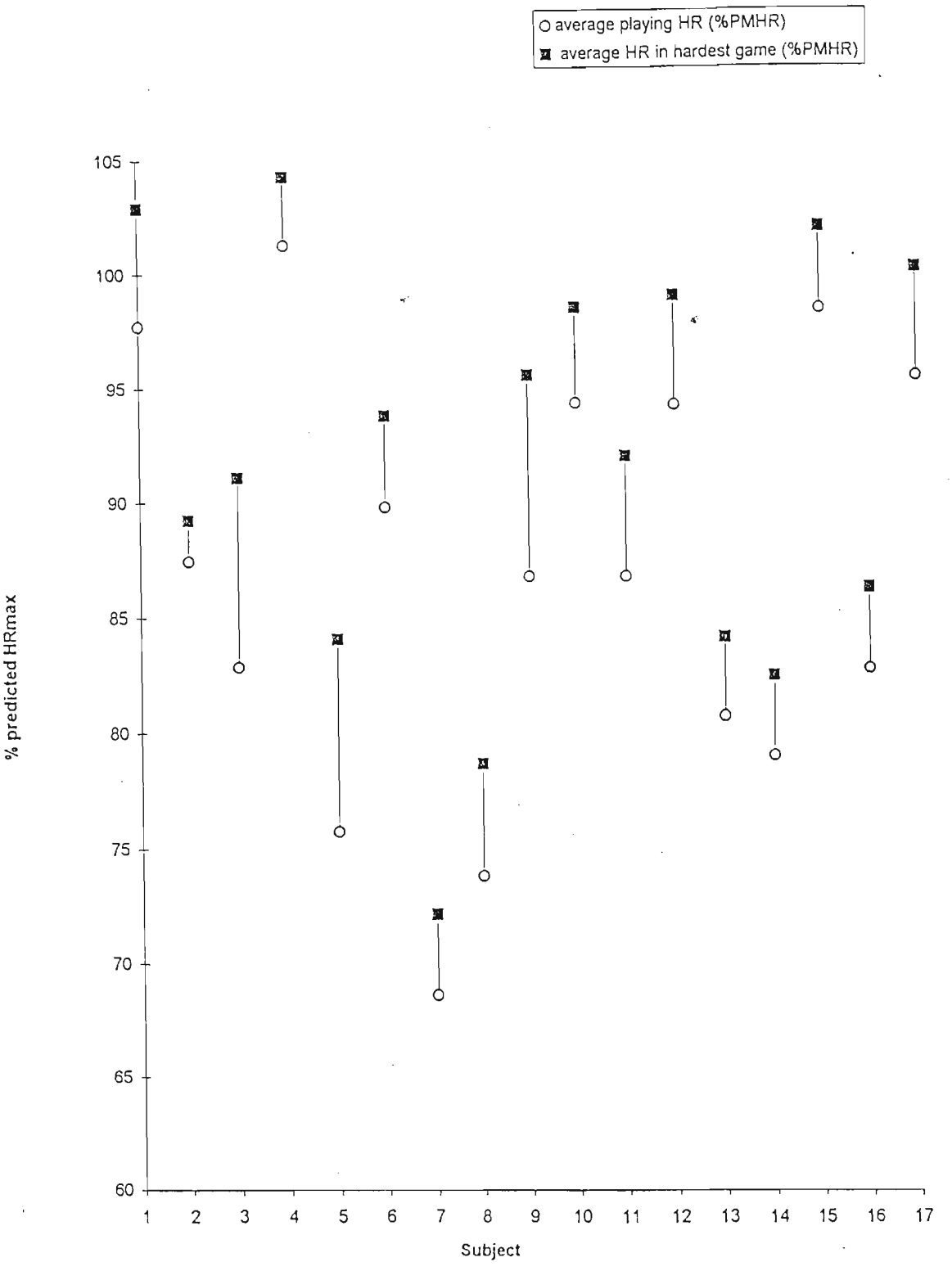

Fig 3 : Individual overall average playing HR and average playing HR in the hardest game (% predicted max HR)

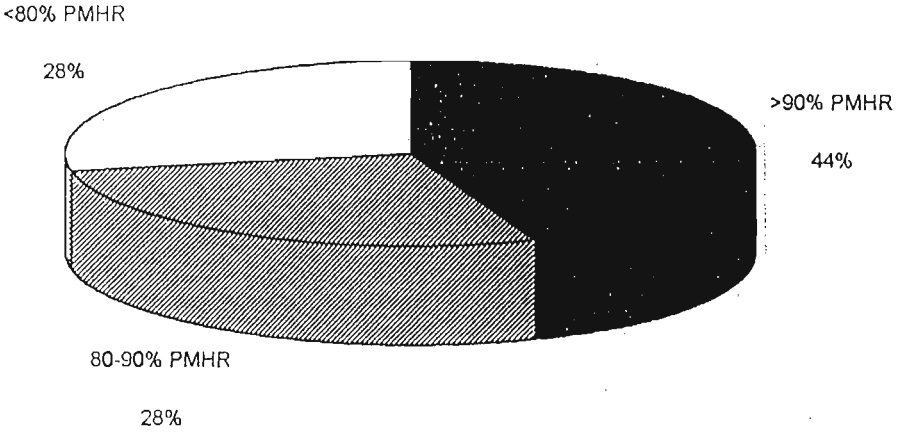

Fig 4: Percentage of match at different %PMHR

TABLE 5 : MEAN HEART RATES AND MAXIMUM HEART RATES

SUBJECTS	AGE (yr)	MeanHR (bpm)	HRMAX (bpm)
1	48	168	186
2	54	145	169
3	51	140	163
4	54	168	182
5	51	128	158
6	46	156	178
7	48	118	146
8	52	124	141
9	48	149	179
10	48	162	182
11	48	149	165
12	52	158	178
13	45	141	169
14	45	138	150
15	50	167	180
16	48	142	163
17	50	162	178
18	51	-	-
MEAN	49	148	169

TABLE 6 : INTERGAME VARIABILITY OF % PMHR

SUBJECTS	AGE (yr)	GAME 1	GAME 2	GAME 3	GAME 4	GAME 5	MAXAVE
1	48	92	100	98	102	103	103
2	54	-	87	89	-	-	89
3	51	82	79	83	86	91	91
4	54	95	102	104	104	102	104
5	51	76	75	72	80	84	84
6	46	88	91	89	87	94	94
7	48	66	66	70	72	72	72
8	52	68	79	76	-	-	79
9	48	79	87	95	-	-	95
10	48	87	95	98	96	-	98
11	48	83	83	88	92	88	92
12	52	88	99	94	98	95	99
13	45	82	78	83	84	-	84
14	45	78	82	80	81	80	82
15	50	92	98	102	101	-	102
16	48	83	86	82	78	82	86
17	50	86	96	97	99	100	100
MEAN	49	83	87	88	90	90	91

Heart rate values greater than 80% of PMHR suggest that squash, as an activity, is capable of maintaining or improving an individual's cardiovascular fitness. High levels of physiological stress accompany exercise of this intensity. As previously mentioned, while the heart rate response is high, the exercise intensity, estimated from this variable, may be an overestimation of the actual intensity. Other factors playing a role in these observed heart rates include the fact that squash involves a lot of arm work which causes an increase in heart rate response above that caused by leg exercise alone (Beaudin et al., 1978; Mercier et al., 1987; Van Rensburg, 1982). Secondly, there is the stress of rapid rallies, concentrating on the ball, and reacting instantaneously. Thirdly, a raised body temperature leads to an increased heart rate (Mercier et al., 1987).

It is, however, clear that irrespective of %PMHR, the absolute heart rate is very high, (mean 169 ±14 bpm). It is therefore possible that arrhythmia may result during or in the hour after the game.

Northcote, MacFarlane, and Ballantyne (1983) found 7/21 players to have ECG changes (most notably ventricular extrasystoles) in the 30 minutes after play. In fact, as many abnormalities were found after play as during play. Unfortunately, in the current study, it was not possible to record an ECG during the game and we report, therefore, findings from the hour after play. Northcote's subjects were exercising at similar intensities (see Table 2) as the subjects in the present study and yet, in the present study, no ECG abnormalities were detected in the post-exercise period in any of the subjects. There are several possible explanations for this finding.

Northcote et al. (1983) possibly happened to choose a subset of people with abnormalities while in the current study and that of St Clair Gibson (personal communication), who found no ECG abnormalities in exercise stress testing of veteran squash players, a low risk group may have been studied. It is possible that the symptomatic people in the older age group studied in the latter two investigations, stopped playing and that those still playing with this amount of regularity and with so few cardiac risk factors are a low risk group. This would explain the lower incidence of abnormalities in the older group, but it is not possible to confirm this in the current study. The study by Northcote et al. (1983) was conducted in Scotland with ambient temperatures between 20 and 24°C . The present study was conducted in South Africa, in summer, with ambient temperatures of a mean of 25.6 ± 2.2°C.

Prolonged exercise in a hot environment causes a greater heart rate than exercise at a low room temperature. Players here are possibly more adapted to their environment. Reapeated exposure to hot environments, especially when combined with exercise, results in improved capacity for exercise and less discomfort on heat exposure (McArdle, Katch and Katch 1986).

In Northcote, MacFarlane, and Ballantyne's study, of the 8 subjects who developed ventricular extrasystoles during or immediately after play, 5 were recognised as having at least 1 CAD risk factor. 2 gave a family history of MI under the age of 45 years, and 4 smoked. In the 13 subjects who did not develop ventricular arrhythmia, 8 had no identifiable risk factors and 5 had at least 1. There was no significant difference in prevalence of risk factors between the two groups. In the present study, where no arrhythmias were found, 5/18 had 1 risk factor, 8/18 had 2 risk factors, 4/18 had 3 risk factors and 1/18 had 4 risk factors. The subjects in the present study, therefore appeared to have a higher risk factor profile (all had at least 1 risk factor, whilst in the study by Northcote et al., 11/21 had no risk factors). It would appear then, that risk factor profile cannot be used to explain the results of Northcote et al. (1983)

Stress testing may not elicit heart rates as high as were found in the squash game. St Clair Gibson found maximum heart rates during exercise stress testing to be significantly lower than those attained during squash. It is questionnable whether these subjects performed maximally and an exercise stress test of this submaximal nature may not be sufficient to detect ECG abnormalities. Similarly, in the study by Northcote et al. in 1983, all but one of the players with positive ECG during and after squash failed to produce a positive stress test result. Coplan and Fuster (1990) warn that failure to perform adequate exercise in an exercise stress test, may result in failure to identify patients at risk of a cardiac event.

Furthermore, in the current study, no abnormalities were detected in any of the veteran players after the game (i.e. in the high risk period). We therefore found no indication for stress testing these individuals. We cannot, however say that no abnormalities would have been found on exercise stress testing these individuals, and a follow up investigation is necessary, where an exercise stress test is carried out on all 18 subjects. From our results, however, there certainly seems to be no indication for mass exercise stress test screening of this asymptomatic group of veterans.

CONCLUSION

In summary, the mean heart rate, throughout the playing time, was 148 ± 16 bpm (range 118 - 168 bpm), representing 86.7% of PMHR. This is comparable with results of similar studies. Maximal heart rates achieved during squash were extremely high (98.8% of PMHR), confirming the very high intensity nature of the sport. No ECG abnormalities were observed, in the hour after play, a result conflicting with the study by Northcote et al. in 1983. This study found no indication for an exercise stress test in this small group of veteran squash players. A larger study is warranted.

SUGGESTIONS FOR FURTHER STUDY :

This group of individuals will be followed up with a maximal exercise stress test to detect the incidence of ECG abnormalities and if the exercise stress test elicits as high heart rates as observed here. Furthermore, a larger group of veteran players will be assessed.

ACKNOWLEDGEMENTS :

Professor M Mars

Dr A Weston

Dr D P Naidoo

A Peterson

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Mrs M Hurley

Mr B Legassick

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APPENDICES

APPENDIX 1 :

PRACTICAL RECOMMENDATIONS WITH RESPECT TO CARDIAC RISK FACTORS

Northcote and Ballantyne (1983) conclude that there exists a small risk of sudden death attached to vigorous activity, particularly squash, which constitutes a threat to players. It has been recommended, in order to minimise this risk, that subjects at an increased risk of coronary heart disease (eg: those over age 40) should perhaps not begin playing squash, and, that subjects in this age group already playing squash should consider changing to a less dangerous sport. This is clearly a subjective judgement and poses the question: What advice should be given to the 45 year old and older players who continue to play?

An increased awareness of the following risk factors may reduce the number of deaths associated with vigorous exercise :

1) Unfit people should not begin to participate in vigorous/highly competitive sport without a period of preparation. Exercise training is likely to increase the absolute exercise intensity that is needed to evoke sudden cardiac death during exercise i.e. get fit to play squash, do not play squash to get fit.
2) Avoid vigorous exercise in extreme heat because excessive fluid and electrolyte loss and high temperatures increase heart rate and may induce arrhythmias. As arrhythmias have been noted in the post-exercise period, avoid a hot bath or shower immediately after exercise.
3) Smoking is associated with :
   a) increased carboxyhaemoglobin levels,
   b) the release of catecholamines which leads to increased heart rate, blood pressure, risk of arrhythmias, platelet adhesiveness, and increased blood lipid concentration and
   c) increased blood viscosity associated with increased fibrinogen levels, haematocrit and red cell volume. This represents a "dangerous triad of risk factors for cardiac hypoxia and arrhythmias" so it is therefore advisable not to smoke in the hour immediately after playing.
4) always warm up and warm down
5) never play when feeling ill or within 48 hours of recovering from an illness which gave a raised temperature. (Sharp, 1990).

APPENDIX 2

QUESTIONNAIRE : RISK FACTORS FOR CARDIAC DISEASE.

Do you smoke ?.....(If YES : how many per day ?).....

How much alcohol do you drink (per week) ?.....

Are you overweight ?.....Are you on diet ?.....

How much caffeine (including coke) do you drink ?.....

Do you have a history of :

- heart attack
- coronary bypass
- other cardiac surgery
- hest discomfort (especially with exertion)
- high blood pressure
- extra, skipped, rapid heart beats or palpitations
- heart murmurs, clicks or unusual cardiac findings
- rheumatic fever
- ankle swelling
- peripheral vascular disease
- phlebitis or emboli
- unusual shortness of breath
- lightheadedness or fainting
- pulmonary disease (asthma, emphysema,bronchitis)
- abnormal cholesterol levels
- diabetes
- stroke
- emotional disorders

Are you on any medication ?

Are you allergic to any drugs ?

Have you recently been ill, hospitalised or undergone any surgical procedure ?

Do you have any orthopaedic problems eg arthritis ?

Has anyone in your family had any of the following ?

- coronary disease.....If yes, what age?.....

- sudden death.....If yes, what age ?.....

- congenital heart disease.....

How many hours of exercise do you undertake per week ?

- regular.....frequency.....
- type.....duration.....
- intensity.....

Height ?.....

Weight ?.....

Occupation ?.....

Age ?.....

APPENDIX 3

RISK FACTORS

1. Age and gender : after the age of 35, in males, and 45, in females, the chance of dying from coronary heart disease increases progressively.
2. Cholesterol levels greater than 200 mg.dl increases the risk of heart attack. In the present study, 2/18 subjects reported high cholesterol levels and 2/18 reported to be at the higher part of normal. No actual cholesterol test was performed.
3. Smoking : the probability of death from heart disease in smokers is almost twice as great as for non-smokers. Risk increases with an increased number of cigarettes smoked per day and the number of years as a smoker.
4. Hypertension : with each increment in both systolic and diastolic pressure, the risk for adverse cardiovascular effects increases with time.
5. Physical inactivity : physical activity has a protective effect on coronary heart disease and helps to eliminate other risk factors like obesity, glucose intolerance, insulin insensitivity, and mild hypertension.
6. Obesity : is associated with a number of cardiac deaths, especially sudden death in males and CCF in females. This high death rate results from the influence of obesity on blood pressure, blood lipid levels, and the risk of precipitating the onset of diabetes.
7. Diabetes / glucose intolerance : glucose intolerance is often associated with hypertriglyceridaemia, increased blood pressure, increased low-density lipoprotein cholesterol values and decreased high-density lipoprotein values. The most common macrovascular manifestation of diabetes is CAD. The risk of coronary heart disease (CHD) is 2 times greater for men and three times greater for women with diabetes.
8. Psychosocial stress : people with an overdeveloped sense of time-urgency, drive and competitiveness (type A behaviour) develop CHD. The major CHD risk factors seem to have greater impact amongst type A persons.
9. Family history : a history of premature fatal or non fatal stroke or myocardial infarction or sudden coronary death in siblings or parents suggest an increased risk of CHD. Furthermore, a family history of diabetes, hypertension or high cholesterol also increases the risk of CHD. (McArdle, Katch, Katch, 1986; ACSM, 1993).

APPENDIX 4

LOWN CLASSIFICATION USED TO CLASSIFY VENTRICULAR ARRHYTHMIAS

Grade 0 : not a single ventricular ectopic beat throughout the entire recording.

Grade 1 : occasional, isolated ventricular premature beats.

Grade 2 : frequent ventricular premature beats at a rate greater than 30 in any hour of recording.

Grade 3 : multiform ventricular premature beats.

Grade 4 : repetitive ventricular premature beats

Grade 4A :couplets.

Grade 4B : salvos

Grade 5 : early ventricular premature beats. (Lown and Wolf, 1971).