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Ambulatory electrocardiography in squash players

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SUMMARY Ambulatory electrocardiography was carried out in 21 healthy, fit, male squash players (aged 23–43 years) before, during, and after match play. Resulting electrocardiograms were analysed with respect to heart rate and changes in rhythm. The results indicate that squash increases the heart rate to 80% of an individual's predicted maximum heart rate for the duration of a game. Ventricular arrhythmias were detected in seven of the subjects during play and in seven in the immediate post-exercise period, an incidence which was not reproduced on subsequent maximal treadmill exercise testing.

This study indicates that squash is a physiologically demanding sport which places a severe strain on the myocardium for considerable periods of time and is capable of generating cardiac arrhythmias. These findings are particularly important for an individual already at risk of sudden death from coronary artery disease or structural cardiovascular abnormalities. Medical advice before participation in the game will identify those at high risk of cardiovascular disease. Subjects in this study who developed arrhythmias were not, however, identified by history, examination, or exercise electrocardiography. Thus, it seems unwise to begin playing squash after the age of 40 years. Whether subjects in this age group already participating in the game should continue to play remains a matter for individual judgment.

The game of squash has enjoyed a huge increase in popularity in the United Kingdom and throughout the world in recent years. Individuals are encouraged to take up the sport in the belief that it will provide them with an increased standard of fitness. In addition, because it is played indoors in a warm environment, it has become a more acceptable form of winter exercise in Britain than outdoor sports such as running or field sports.

With this increase in participation has come a disturbing growth in the number of reports of sudden death associated with the sport. Such reports appear predominantly in the national press and have concerned players of poor, average, and superior fitness[1] in all age groups. The number of deaths has been estimated at approximately 27 a year in the United Kingdom.[2] Although frequently quoted, such a figure is, however, unreliable and may represent only part of the problem. At present, it is impossible to determine accurately the incidence of squash fatalities in the United Kingdom as any figures rely heavily on newspaper and personal reports through agencies, such as the Squash Rackets Association, and will be incomplete and possibly misleading.

It is generally assumed that most sudden unexpected deaths during or shortly after squash are related to the cardiovascular system. We therefore decided to assess heart rate and rhythm changes by investigating the electrocardiographic events during and immediately after playing in 21 healthy male volunteers. Several previous studies have determined heart rate response by radiotelemetry in various activities including ice hockey,[3] water polo,[4] swimming,[5] tennis,[6] and squash,[7] but they have, however, concerned themselves with heart rate responses only.

Subjects and methods

Male volunteers were recruited from squash clubs in the west of Scotland, of whom 27 participated in the study. Of these, 21 had ambulatory electrocardiograms which were considered suitable for study; six were excluded for several reasons. One subject removed his monitor before playing and failed to reassemble the apparatus properly; in the remainder loss of electrode-skin contact resulted in poor quality of the electrocardiograms.

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All players were of similar average ability and had played at least twice weekly for two years. Their mean age was 33 (SD±6·5) years (range 23–43 years) and their weight 77·49 (SD±8·65) kg. All subjects were right handed. None had previous evidence of cardiovascular disease and no abnormalities on clinical examination, 12 lead electrocardiogram, or chest x ray films. None of the players was receiving drugs at the time of the investigation.

Subjects attended the cardiology department at least 90 minutes before their prearranged squash match. A medical history was taken with special reference to the cardiovascular system and presence of coronary heart disease risk factors.[8] Clinical examination, resting electrocardiogram, and radiography of the chest were also undertaken to exclude major cardiovascular disease.

Subjects were then fitted with an Oxford Electronic Instruments series 4·24 single channel electrocardiographic monitor. The exploring electrodes (Medicotest) were positioned over the manubrium sterni and in the fifth intercostal space and anterior axillary line of the chest wall on the left side, thus producing a modified chest lead V5. These electrodes were secured with Transpore surgical tape. This technique minimised interference from the pectoralis group of muscles and produced excellent quality recordings (Fig. 1). Recording was started at least 45 minutes before play. After 10 minutes warming up each subject played for 40 minutes against an opponent of similar ability and with balls of an identical speed. The ambient temperature during each match was between 20°C and 24°C. Recording was continued throughout play and for at least 30 minutes after.

Tape analysis was carried out with computer assistance as described.[9] Mean heart rates were recorded on a minute by minute basis before, during, and after play. A visual search using a Reynold's high speed electrocardiogram analyser was undertaken to confirm abnormalities detected by computer analysis and samples recorded on to electrocardiographic paper. All findings reported here are derived from these recordings.

Results

HEART RATE

The maximum recorded heart rate for the group during play was 170·28±16·2 (mean±SD) beats/minute (range 144–197 beats/minute). When expressed as a percentage of predicted maximum heart rate, as defined by Astrand and Rodahl,[10] this represented a mean of 90% (SD±8·2) of the predicted maximum heart rate for the group as a whole. The mean heart rate throughout the 40 minute period of play was 149±17·8 (range 120–182) beats/minute, which represents 80%±10·2 of the predicted maximum heart rate maintained for the duration of play.

One subject (aged 32 years) with a calculated predicted maximum heart rate of 186±10 beats/minute developed a maximum heart rate of 197 beats/minute after thirty-seven minutes of play and maintained a mean heart rate of 182 beats/minute throughout the 40 minute period, equivalent to 98% of his predicted maximum heart rate.

Fig. 2 shows the heart rate trend for the whole group measured for each minute time plot and expressed as a percentage of predicted maximum heart rate. Despite an adequate warming up period of 10 minutes, there was a steep rise in pulse rate at the start of play. A commensurate rapid fall in heart rate occurred within the first minute or two of the post-exercise period.

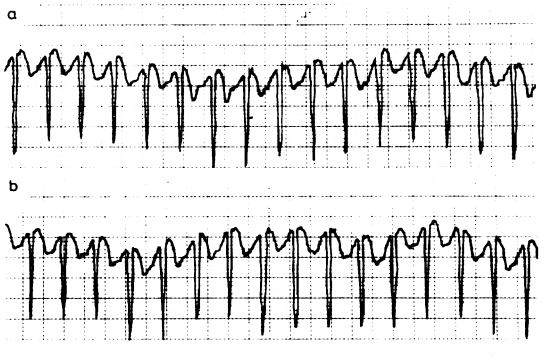

Fig. 1 *Representative portions (a) and (b) from ambulatory electrocardiograms of two individual subjects showing the quality achieved at heart rates over 150 beats/minute.*

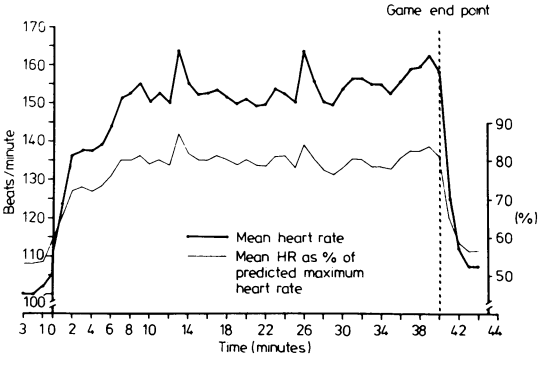

Fig. 2 *Mean heart rate response on a minute by minute basis for the group during and immediately after play expressed as beats/minute and as a percentage of predicted maximum heart rate (HR).*

HEART RHYTHM

Both ventricular extrasystoles and premature atrial contractions were detected during and after play. Seven (33%) subjects developed at least one ventricular extrasystole during play but only three (aged 33, 40, and 36 years) had more than five extrasystoles during the 40 minutes (11, 11, and 84 respectively). Two of these subjects (aged 36 and 40 years) had evidence of one episode of ventricular tachycardia (defined as three or more ventricular extrasystoles in succession at a rate of more than 100 beats/minute). These occurred after six and thirty minutes of play and extended over four and nine cycles respectively; the latter subject also had coupled ventricular extrasystoles. No significant relation was observed between the age of the subject and the development of ventricular extrasystoles.

In the first 30 minutes after play, a period often referred to as the post-exercise vulnerable period,[11] seven (33%) subjects had at least one ventricular extrasystole and all but one also had evidence of ventricular extrasystoles during exercise. Only three subjects had more than five post-exercise ventricular extrasystoles (six, 10, and 26), and most of these (63·2%) occurred in the first nine minutes of the post-exercise period; 94% occurred within 17 minutes of the end of play. In addition to those developing ventricular tachycardia during play, one player had an episode of ventricular tachycardia after exercise; this occurred four minutes after play and extended over five cycles. All ventricular extrasystoles recorded were unifocal, long cycle in type. The frequency distribution of ventricular extrasystoles with respect to time is shown in Fig. 3(b). These data refer to the eight subjects having ventricular extrasystoles during play or in the post-exercise period.

Thirteen (62%) subjects had at least one premature atrial contraction during play (defined as an R-R interval less than 70% of the average for the minute in which it occurs and, in addition, is less than 70% of the preceding R-R interval). Only four subjects, however, had more than five such contractions (13, 17, 24, and 34 respectively); six subjects (29%) had at least one premature atrial contraction post-exercise but only four developing post-exercise premature atrial contractions had evidence of these during play. Because of their nature and the high heart rates recorded in this study the computer findings could not always be confirmed by direct inspection of the electrocardiogram. It is appreciated that confusion

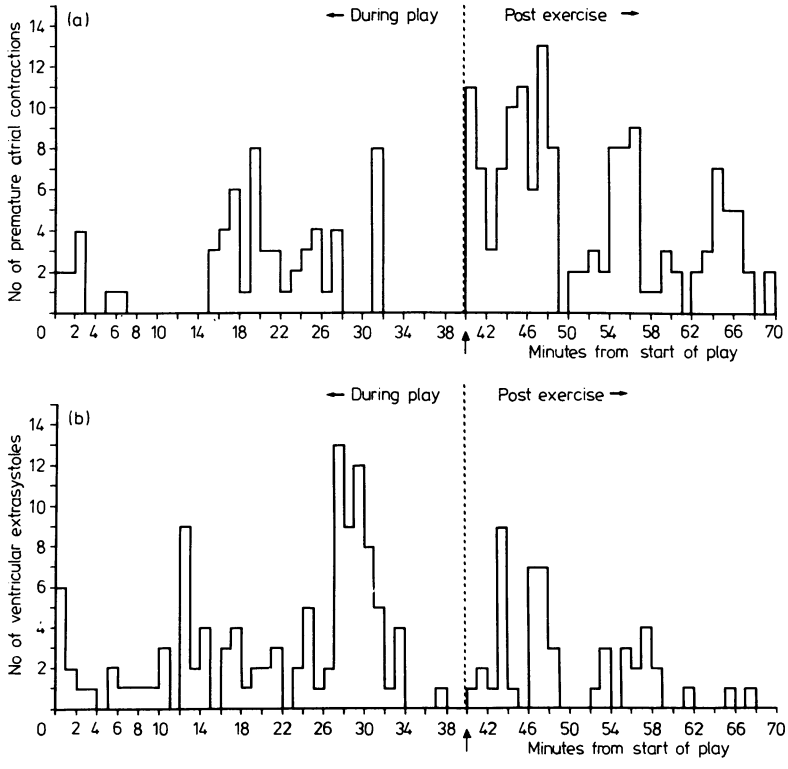

Fig. 3 *Mean frequency distribution of (a) supraventricular extrasystoles and (b) ventricular extrasystoles in relation to time from the start of play.*

may arise in distinguishing premature supraventricular contractions from sinus arrhythmia, but we considered that, during such vigorous exercise, this would not influence results. Fig. 3(a) shows the distribution of premature atrial contractions with respect to time for the 13 subjects developing these.

Of the eight subjects who developed ventricular extrasystoles during or immediately after play, five were recognised as having at least one coronary artery disease risk factor, two gave a history of early myocardial infarction (under 45 years) in a first degree relative, and four smoked more than 10 cigarettes a day. In the 13 subjects who did not develop a ventricular arrhythmia, eight had no identifiable risk factors whereas five had at least one. The χ^2 test did not show any significant difference in the prevalence of risk factors between the two groups.

Each subject who developed ventricular extrasystoles during or immediately post-exercise underwent maximal exercise electrocardiography on a treadmill linked to a three channel recorder using a Bruce protocol.[12] All subjects had an excellent exercise tolerance with maximal heart rates of $182 \pm 8 \cdot 3$ (mean $\pm$ SD) beats/minute; only one subject developed ventricular extrasystoles consisting of three isolated unifocal ventricular complexes.

Only one subject had ventricular extrasystoles in the 60 minutes before play (three isolated ventricular complexes). In this particular case, no further extrasystoles occurred during or after play.

Discussion

Our results show that squash places a prolonged and severe workload on the myocardium, the body being subjected to sudden and vigorous demands. In this study, the mean heart rate was increased to about 80% of the predicted maximum heart rate for the duration of play in moderately fit male players. A previous study[13] has shown that even higher heart rates can occur in sedentary men playing squash, compared with those of active men, and have approached those observed in very fit squash players. Unfit players may therefore achieve heart rates higher than those reported in this study. In addition, after the end of play, an abrupt fall in heart rate occurred within the first few minutes post-exercise. The results of this study confirm the heart rate responses found by others.[13 14] These studies, however, used radiotelemetry to relay electrocardiographic events and heart rate was recorded only every 30 seconds and averaged for each five minutes of play. In the present study continuous monitoring allowed the mean heart rate to be calculated every minute. Previous studies also investigated the heart rate response over 25 to 30 minutes of play only and did not investigate the post-exercise period. No mention was made of rhythm changes.

In the present study the rapid rise in heart rate was tempered by a 10 minute warming up period. Thus, if such a warming up period does not take place, the heart rate may rise from a resting rate of about 70 beats/minute to more than 150 beats/minute in a very short time. This practice may be potentially dangerous and should be discouraged. Compared with squash the heart rate response in other sports is not as pronounced; for example, tennis will raise the heart rate to between 60% and 70% of the predicted maximum heart rate in men,[14 15] but only for periods of actual rally play, whereas in squash our results show that an increase to 80% of the predicted maximum heart rate occurred throughout play. This discrepancy in heart rate response may be explained by the physical environment in which the games are played. A squash court has a higher ambient temperature and humidity with poor ventilation compared with a tennis court. Such thermal and environmental stresses increase heart rate considerably.[16] Unlike tennis, minimal time is spent on retrieving the ball between points.

Because of the nature of the game squash players are likely to overextend their physiological capabilities in an effort to win, since longer and more intense rallies are possible compared with other sports such as tennis or badminton—these being played on an "open court"—in which the margin for error during play is much less. This series of events may be more probable with individuals of a highly competitive or aggressive nature, who would rather win a point than submit to fatigue or discomfort. Such individuals may be more attracted to squash than more passive individuals.

Psychological stress has been found to increase heart rate[17 18] and in some instances has been implicated in causing sudden death.[19] Perhaps the stress of a one-to-one conflict in an enclosed space, as in squash, precipitates similar events.

The incidence and frequency of cardiac arrhythmias in squash players during and after exercise are higher than would be expected on routine 24 hour ambulatory electrocardiography in subjects with no evidence of cardiovascular disease,[20] although ventricular extrasystoles and short bursts of ventricular tachycardia can occur in apparently healthy subjects.[21 22] Those arrhythmias associated with vigorous exercise may be more serious.[23] Of the three subjects who developed an episode of ventricular tachycardia in the present study, one showed evidence of coupled ventricular extrasystoles and a prolonged run of ventricular tachycardia during play extending over nine cycles. He was advised against further participation in squash.

The cause of these arrhythmias must be a matter for

conjecture, as there are several possibilities. There is good evidence that exercise can precipitate arrhythmias in the coronary prone population.[24] Since myocardial ischaemia is known to cause arrhythmias[25] it is possible that asymptomatic coronary atherosclerosis is present in some players. Cardiac arrhythmias can occur in response to thermal stress,[16] which may be important in squash since it is often played in an excessively warm environment. Catecholamines rise during exercise and may precipitate both myocardial ischaemia and arrhythmias in the presence of coronary artery constriction.[26] Other metabolic changes such as hyperkalaemia[27] during exercise[28] may precipitate arrhythmias.[29] Our results showed a high proportion of extrasystoles occurring in the vulnerable post-exercise period.[11] Players who smoke after a squash match, with a consequent rise in blood free fatty acids[30] and release of catecholamines,[31] would be at greater risk at this time.

At the end of play the sudden cessation of activity increases venous pooling in the tissues and therefore a diminished cardiac return. In the face of continued sympathetic nervous system stimulation this may precipitatate coronary artery insufficiency and thereafter an arrhythmia. This may be exacerbated by the thermal stresses inflicted by a hot bath or shower after the game. The possibility that a viral myocarditis can cause arrhythmias or sudden death has been considered.[32 33] Evidence for this relation is, however, tenuous at present.

Ventricular extrasystoles induced by exercise may induce sudden death.[34] Therefore, a proportion of these deaths are likely to be arrhythmic. If this is correct, then those subjects developing ventricular extrasystoles during play are at an increased risk. Competitive sports, such as squash, may be more liable to cause serious arrhythmias than other sports.

Medical screening before participation in the game has been advocated by others,[35] including exercise electrocardiography.[36] In the present study, subjects developing ventricular arrhythmias, and therefore at an increased risk of sudden death, were not identified by preparticipation clinical assessment or by subsequent exercise electrocardiography. Routine screening will identify subjects with more overt evidence of cardiovascular disease. There will, however, remain a group which cannot be identified. Despite this, subjects over 30 years should be encouraged to seek medical advice before participating in this sport.

We are aware of the benefits of physical exercise and acknowledge that it may have a part to play in the primary or secondary prevention of coronary artery disease.[37] In addition, we realise that concern expressed about squash may deter the inactive majority from sporting activity rather than the squash playing population. Nevertheless, the small risk of sudden death attached to vigorous activity—and particularly squash—remains and will constitute a threat, however small, to all players. It would seem wise to make attempts to minimise this risk. In the light of available evidence subjects at an increased risk of coronary artery disease—for example, over 40 years of age—should possibly not begin playing squash. We also recommend that subjects in this age group already playing squash should consider changing to a less dangerous sport, although this will remain a matter for individual judgment.

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