EXERCISE AND SUDDEN DEATH

Dan Tunstall-Pedoe, MRCP, DPhil,

Senior Lecturer and Honorary Consultant Cardiologist

St. Bartholomew's Hospital, London EC1 A 2BE

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The theme of this conference. . Exercise a prescription for health? has to look not only at the possible benefits of exercise, but also its possible dangers. Like Williams who has been given the brief to discuss over-use injuries, I am in the position of devil's advocate and have researched the most harmful effect of exercise... sudden death associated with exertion. Some of the Media have been very anxious to promote exercise as being beneficial to the 'Circulation', but the impression they occasionally give is that they are more interested in their own circulation than their readers'. It is therefore reasonable to point out that there are risks associated with exercise and attempt to quantitate that risk, so that the benefits can be weighed against it. Having taken the position of devil's advocate I would like to say that I take regular vigorous exercise myself, and have competed in track, road and cross country races for more than 20 years.

How dangerous is exercise? Unfortunately the risk calculated from supervised exercise programmes such as those described by Shephard in this volume are probably not applicable to unsupervised and competitive situations encountered outside the gymnasium. The statistics of sudden death and the events associated with it are difficult to obtain. Sudden death is the commonest non-violent, non-suicidal form of death in young people, and is often defined as death occurring within 24 hours of the onset of symptoms. Unexpected death in a young person will naturally be blamed on any slightly unusual antecedent activity such as severe exertion. To show that that activity itself precipitated the sudden death, requires data on the amount of time spent in that activity, or recovery from it, compared with the overall sudden death rate associated with that activity, and not associated with it in the population at risk. This type of information is not available for the population as a whole, so that most of the available studies have been made on circumscribed groups, such as members of the armed services.

Coroners' post mortems.

One of the advantages of using data obtained from studies on sudden death in the armed forces, is that not only is there a defined population at risk, whose activities are scheduled and recorded, but where sudden death does occur, it is likely to be investigated more

thoroughly than in a coroner's post mortem. The chief function of a coroner's post mortem is to exclude death from violence and death from drugs or alcohol. If any natural cause (any pathology of the heart or brain) is found in the absence of evidence of violence, death from natural causes can be proclaimed, and the exact mechanism of the death, and the finer points of the aetiology can be neglected. The armed services post mortem studies particularly those from the United States have been much more meticulous than those from the routine coroner's pathologist. Any non-accidental death of a conscript requires very full explanation, not only for the sake of the relatives but for the service authorities as well.

It is evidence from these sources rather than diffuse anecdotal evidence that I will principally discuss.

Surveys

Koskenvuo (1976) examined the records of the Finnish services for the relationship of sudden death to exertion. There were 45 cases of sudden death in 660,000 man years (6.8/100,000 man years) in this population who were drafted at 19. Of these deaths

36% were ascribed to disease of the heart or great vessels.

29% were ascribed to cerebrovascular disease.

93% of these deaths were evaluated by post mortem.

The cardiovascular deaths, of which there were 16, were found to be very related closely to exertion, with only 5 exceptions. Nine conscripts were at nearly maximal exertion at the onset of their preagonal symptoms, one was dancing and one swimming. Only one had the onset of his symptoms during sleep. In contrast to this the cerebrovascular deaths were heralded by symptoms principally developing during sleep (9/13).

There were three additional deaths from unknown causes, that occurred during strenuous exertion which seem most likely to have been cardiogenic. (Possibly due to cardiac arrhythmias.)

The cardiovascular causes of death in this very young population who should have been screened for any obvious heart disease on being conscripted, included coronary artery anomalies and atheroma, myocarditis and abnormalities such as hypoplasia of the aorta.

A similar survey on young people has been reported by Maron (1978) who reviewed the autopsy registers of the United States National Institute of Health and Armed Forces Institute of Pathology, collecting 19 cases of sudden death in competitive athletes, in whom there was adequate necropsy information. The age range was 14-30 (mean 20) and death occurred during or immediately after exercise in all of these cases. The sports represented included basket ball (7) football (4) distance running (3) wrestling, boxing and swimming. Two of these athletes died suddenly and no apparent abnormality of the heart was found, but the conduction system of the heart was not closely examined, and it seems likely that these could have been arrhythmic deaths (see below).

The listed causes of death in order of frequency were:

Hypertrophic cardiomyopathy	6
Coronary atherosclerosis	3
Coronary artery anomalies	3
Ruptured aorta	2
Idiopathic concentric	
left ventricular hypertrophy	2
Wolff-Parkinson-White syndrome	1

The various types of pathology will be considered after the next study, that of Opie, 1975 who garnered newspaper reports of sudden death in sportsmen from the South African press and then investigated them. He found reports of sudden death in 21 sportsmen and found evidence for a cardiac cause in 18 all of whom died during or shortly after taking part in their sport: rugby 7, refereeing 4, soccer 2, tennis 2, golf, jogging, yachting, and mountaineering.

Only 7 of these deaths were investigated by post mortems, but the other 11 had either ECG and clinical evidence or strong clinical evidence (chest pain etc) of ischaemic heart disease. Nine of these sportsmen had had episodes of angina-like chest pain before taking part in their final game.

Not surprisingly from the preponderance of ischaemic heart disease, this group of sportsmen had much greater average age than the Finnish or American series.

The risk of death calculated by Opie for rugby players (from the number of deaths and the number of games of rugby taking place) was 1 death for every 50,000 man hours of rugby (6 years) the average age of men who died was 26. Much more alarming was the death rate among referees who were older (average age 53) and who averaged 1 death for every 3,000 hours of refereeing.

Only three of the deaths were in non-smokers and there was a high level of predisposing factors for ischaemic heart disease in this group of subjects. Ischaemic

heart disease is very common in white South Africans and a certain pig-headed insistence on taking part in one's chosen sport seems to have been a unifying feature in many of these cases, which unlike the deaths in the younger athletes described in the series above, might well have been prevented, since nearly all had premonitory symptoms or were highly at risk from ischaemic heart disease.

It is of some interest that various diseases known to cause sudden death are only found in one or other of the three surveys. Thus Maron lists hypertrophic cardiomyopathy, to cardiologists a notorious cause of sudden death in young people, while the Finnish study lists myocarditis which is unrepresented in the series reported by Maron. Owing to the relatively few post mortems in Opie's series, it is not possible to know whether all the cardiac deaths were indeed due to coronary artery disease, or whether hypertrophic cardiomyopathy or myocarditis might also be responsible. Are the differences in the causes of death real in these series or do they represent fashions or trends in post mortem assessment? In discussing the commoner causes of sudden death listed in these series, with this question in mind, one might also ask whether the deaths from each cause were preventable.

Hypertrophic cardiomyopathy

Maron listed this as the cause of death in 30% of his subjects. The disease consists of an unphysiological hypertrophy of the heart muscle whose fibres lose their normal orientation. The hypertrophy is asymmetric and often leads to obstruction of the outflow tract of the left ventricle. It is often familial, being associated with a family history of sudden death, and may cause symptoms of angina, breathlessness, palpitations and syncope. Where there is outflow tract obstruction there are characteristic physical signs, but milder cases may only be suspected on the basis of an abnormal ECG and echocardiogram. (Ultrasonic measurements made of intracardiac dimensions and movements). One of the characteristic abnormalities is that the interventricular septum may be considerably thicker than the free wall of the left ventricle, the so called 'asymmetric septal hypertrophy' or 'ASH'. This was found in the six dead athletes, labelled as having hypertrophic cardiomyopathy. Since it has been shown that a certain degree of asymmetric septal hypertrophy is not uncommon in the normal population, and appears to be even more common in athletes whose hearts have hypertrophied as part of a physiological response, it is possible that not all of these deaths were in fact due to this form of cardiomyopathy.

Certainly one of them, a well known tennis player, had the fully developed form of this heart disease. She was known to have a heart murmur and an abnormal

ECG and her fainting attacks, had rather incredibly in retrospect been blamed on hypoglycaemia. Had the diagnosis been established before death she should have been advised against competition, and perhaps her death could have been prevented.

Myocarditis

This is a listed cause of sudden death, and again post mortem criteria may well vary between pathologists. Inflammatory disease of the heart can be severe enough to cause heart failure and death from this cause in myocarditis is fortunately rare. More frequently during the course of an acute pyrexial illness, very often viral, there is a mild myocarditis, recognised clinically by the appearance of ECG changes, conduction abnormalities and often cardiac arrhythmias, and pathologically by the appearance of inflammatory cells and later patchy fibrosis in the myocardium.

Mild attacks of myocarditis are probably very common. Routine ECGs on all patients reporting an attack of influenza to their general practitioner during an epidemic of A2 influenza, revealed that 43% of patients had ECG changes, although these attacks of influenza were not sufficiently severe to warrant hospital admission. In two cases there were permanent ECG changes, one mimicking ischaemic damage and the other showing permanent complete heart block (Verel et al., 1976). There are several anecdotal reports of athletes collapsing while out for a run 'to sweat out a cold or fever' or doing so shortly after exertion in these circumstances. They presumably die from a lethal cardiac arrhythmia precipitated by a low grade myocarditis and potentiated by exercise.

Any severe exertion, competition or training during the course of, or in the immediate convalescent period of an acute pyrexial illness is therefore hazardous and should be severely discouraged. Cardiac arrhythmias are a common manifestation of myocarditis and are the most common 'final common path' for other cardiac causes of sudden death. Could these deaths be prevented by routine ECGs on athletes with pyrexial illness? Probably the caution that I have given above is the most practical preventive. Electrocardiography in the prevention of sudden death in athletes will be discussed in more detail below.

Ischaemic heart disease

Depending on the age group studied, this is either the result of coronary atheroma (Opie's study) or congenital anomalies of the coronary circulation.

Coronary atheroma

Exercise may well act as a preventive in reducing the

risks of developing ischaemic heart disease in those who take regular vigorous exercise, but unfortunately it is not totally protective and may actually precipitate an infarct in somebody who has developed coronary atheroma. The coronary atheroma may be totally asymptomatic until the onset of the infarct.

A fifty year old man was recently admitted under my care with an anterior myocardial infarction. He rowed regularly for a club veteran eight, and while out training developed classical chest pain and collapsed. His colleagues had to haul him back into the boat and row him to hospital as fast as possible!

As Opie has pointed out, many athletes in middle age have a number of risk factors for coronary disease, they may smoke heavily, be hypertensive, have excess levels of fat in their blood and have a strong family history of coronary artery disease. Exercise does not make them totally immune from the risks of myocardial infarction, and in the presence of coronary atheroma exercise may well render part of the myocardium ischaemic. Whether this results in a self limiting attack of angina, a myocardial infarction, or a lethal cardiac arrhythmia resulting in collapse and sudden death (or instantaneous death as some authors prefer to call it in these circumstances) is a matter of individual circumstance.

Thus whilst exercise is beneficial and safe for the vast majority of the population, there are dangers for those with coronary artery disease whether recognised or not. The jogging epidemic is probably exposing a certain number of middle aged men with undiagnosed coronary disease to these hazards and it is unfortunately paradoxical, that those who might benefit most from exercise, the middle aged, obese, possibly hypertensive smoker, are most at risk.

Coronary artery anomalies

Many of these anomalies may be found as part of a complex of congenital heart disease and we will only mention those that occur as the sole abnormality. The majority of the anomalies of origin of coronary arteries from the aorta are benign and are found in about every two hundredth coronary angiogram. (Origin from the pulmonary artery of the left coronary artery causes gross cardiac ischaemia and early death, of the right coronary artery is usually well tolerated.)

The anomaly that has been closely linked with sudden death on exertion is where the left coronary artery arises from the anterior sinus of Valsalva. In this situation the left coronary artery passes obliquely between the aorta and the pulmonary artery. There is an accumulating literature describing sudden death associated with exertion in young people found at post mortem to have this anomaly, Cheitlan et al 1974. The

total known cases of this anomaly are 33, nine of whom died suddenly and unexpectedly, seven of these were less than 22 years of age and died while exercising, five of them while running. This anomaly is very uncommon, but illustrates again that under certain circumstances exercise can precipitate sudden death.

Recognition of coronary anomalies depends on routine investigation of possible cardiac symptoms, such as syncope or chest pain on exertion even in young athletes. Many of these cases have had dramatic symptoms before the final fatal event, but these have not been investigated with exercise electrocardiogram and coronary angiography, where indicated. Cheitlin et al describe a case in which the diagnosis was made after successful resuscitation of a 14 year old boy with this condition, who then had cardiac surgery. He had a normal exercise test, but had had two episodes of exertional syncope.

Cardiac arrhythmias and sudden death

I have said that a arrhythmia is the most likely final common path of exercise induced sudden (or particularly instantaneous) death. Most post mortem examinations do not include careful examination of the conduction system of the heart, although certain ECG conduction abnormalities are well known to be associated with an increased risk of sudden death. One of Maron's cases was said to have Wolff-Parkinson-White syndrome. In these people there is a conduction pathway between the atria and the ventricles that bypasses the normal slow passage of the impulse through the atrioventricular node. They are prone to tachycardias, have an abnormality of the ECG, which comes and goes, i.e. the ECG may be quite normal at times, and run an increased risk of sudden death, although this is not particularly associated with exertion in the literature on the subject.

It is probable that more than the usually recognised 1/1000 of the population have aberrant conduction pathways in the heart which under suitable circumstances could form part of a pathway for a dangerous arrhythmia, and some of the sudden deaths on exertion that at post mortem are found to be associated with minimal evidence of myocarditis, or doubtful evidence of hypertrophic cardiomyopathy may be explained by such a mechanism. Recently at St. Bartholomew's Hospital we had as a patient a professional footballer of 27, who collapsed while playing, with chest pain. He was found to have not only Wolff-Parkinson-White syndrome

and a very rapid tachycardia, but also severe coronary artery disease.

Screening of athletes and the prevention of sudden death

Although I have dwelt on this subject, sudden death in fit young athletes is dramatic but uncommon. By its nature it is a terminal event, not a symptom of which the athlete complains. However the near misses, the syncope on exertion, the chest pain on exertion, the severe bout of palpitations associated with breathlessness or chest pain, are all warning symptoms that are often ignored by the athlete or his medical advisor, but should be fully investigated.

Should all athletes have routine ECGs? In some countries such as Italy this is done, at considerable expense. ECGs are easy to record but athletes often have abnormal traces, Lichtman et al 1973, Tunstall Pedoe & Thomason, 1976 and the danger is that fit young athletes may well be given a cardiac neurosis by their medical attendants as a result.

The athlete's heart is often enlarged, beats irregularly and slowly at rest, and may exhibit a fairly wide range of ECG abnormalities some similar to those of heart disease (Tunstall Pedoe and Thomason, 1976). I have seen a fit young boxer, who was asymptomatic, but who had a cardiac catheter and myocardial biopsy before his medical advisors were happy that there was nothing wrong with him, because of the changes they had discovered of 'athlete's heart'.

I therefore feel that symptoms should be very thoroughly investigated by doctors aware of athletes' peculiarities, but routine screening of a fit, young population is unlikely to be rewarding.

In summary therefore, sudden death in athletes on exertion is a rare but well documented risk. Deaths from violence in sport are more common, but the increasing participation of middle aged men, who have not been habitual exercisers, in jogging and other vigorous sports, may increase the incidence of sudden death associated with exercise. Some of these men instead of dying from their coronary disease under other circumstances, may do so while exercising. In the same way that an old car tends to break down on the motorway, the heart is rather more likely to have its defects exposed by maximum demands being made on it. These deaths may be the price we have to pay for a fitter, and happier population.

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