Reasons for Sudden deaths of Sportsmen Taking Part in Strenuous Activity during Hot Humid Climate

Sajeev Jos[a], Cicily Pearly Alex[b] & Santhosh J[c]

[a]St. Albert's College (Autonomous), HoD, Department of Physical Education, Ernakulam, Kerala, India
[b]Associate Professor and Head, Department of Physical Education, St. Xavier's College for Women Aluva, Kerala, India
[c]Associate Professor and Head, Department of Physical Education, Nirmala College, Muvattupuzha, Kerala, India

To reduce the mortality rate due to sudden death of sportsmen by finding the causes, symptoms and prevention which leads to it. Systematic evaluation of clinical information and circumstances associated with sudden deaths; interviews with family members, witnesses, and coaches

Environmental Conditions

Maximum day temperature in the tropics generally averages more than 30^{o} C all the year-round. In the wet season, humidity is also high. Hot and/or humid conditions limit cooling, because the temperature and vapour gradients between the skin and air are not conducive to loss of heat. In such conditions, body temperature becomes a factor limiting performance of high-intensity endurance exercise. Strategies that reduce resting body temperature or enhance dissipation of heat can therefore enhance performance.

Athletes practicing in environmental conditions such as high ambient temperatures, humidity, and solar radiation may experience a thermal stress increase on the body and the risk of heat illness. Common guidelines for health care professionals monitoring conditions for practice or competition time are set at specific ranges. Temperatures between 26.7° to 32.3° C with relative humidity under 70% indicate that athletes should be watched closely for any signs of distress. Yet, with the same temperatures with relative humidity over 70% athletes should not only be monitored but they should also take ten minutes rest at least every hour. Temperatures ranging from 32.2° to 37.8° C with a relative humidity under 70%, the same recommendation should be needed. Conditions with temperatures between 32.3° to 37.8° C or higher with a relative humidity over 70% should warrant a suspension in practice or a shortened practice time with little clothing (Arnheim et al,1997). A study by Galloway under different ambient temperatures found a decrease in time to exhaustion, VO2max, and CHO oxidation at only 31° C. He also reported an increase in rectal temperature, sweat rate, rate of perceived exertion, and heart rate in the same condition compared to less stressful environmental conditions (Galloway et al, 1995).

Ambient temperatures higher than 35° C with a relative humidity of 60% can cause a detrimental effect in evaporation of sweat and maintenance of steady state temperatures

Global Warming

Global warming is the increase in the average measured temperature of the Earth's near-surface air and oceans since the mid-twentieth century, and its projected continuation.

The average global air temperature near the Earth's surface increased 0.74 ± 0.18 °C (1.33 ± 0.32 °F) during the hundred years ending in 2005**.** The Intergovernmental Panel on Climate Change (IPCC) concludes "most of the observed increase in globally averaged temperatures since the mid-twentieth century is very likely due to the observed increase in anthropogenic (man-made) greenhouse gas concentrations via an enhanced greenhouse effect. Natural phenomena such as solar variation combined with volcanoes probably had a small warming effect from pre-industrial times to 1950 and a small cooling effect from 1950 onward.

Climate model projections summarized by the IPCC indicate that average global surface temperature will likely rise a further 1.1 to 6.4° C (2.0 to 11.5° F) during the twenty-first century. Remaining scientific uncertainities include the amount of warming expected in the future, and how warming and related changes will vary from region to region around the globe. Most national governments have signed and ratified the Kyoto Protocol aimed at reducing greenhouse gas emissions, but there is ongoing political and public debate worldwide regarding what, if any, action should be taken to reduce or reverse future warming or to adapt to its expected consequences.

Cause for cardiovascular strain during strenuous activity in hot humid cimate

Environmental heat reduces the thermal gradient between the environment and the skin surface, and between the skin surface and body core, thus imposing an added resistance to body heat loss. It is already proved that body heat can actually be gained when the temperature of the environment is greater than that of our skin. Increased humidity imposes a heat loss barrier to the evaporative mechanism by decreasing the vapour pressure gradient between the moisture in the air and the sweat on our skin. Such a heat loss barrier causes an excessive increase in rectal temperature and severely limits the capacity for work.

Vasodilatation to the skin, arms, legs, and trunk are affected by the nervous system’s sympathetic response to an increase in body temperature (Backx et al, 2001). With the stress of an increase in blood flow to the skin in an effort to decrease the thermal load and supply oxygen to the active muscles, the cardiovascular system cannot keep up with the demands (Casa, 1999). A redistribution of blood is needed because of the increased body temperature. Blood pressure is more vital compared to the skin trying to cool the body or the performing muscles, and as a result hyperthermic and metabolic inefficiencies occur (Sawka et al, 1989). The hyperthermic and metabolic inefficiency response is especially relevant to athletes in the heat during practice. With this inefficiency comes an increase in lactate production as a result of a decrease in hepatatic blood flow and an increase in muscle temperature, which can eventually lead to fatigue. In an attempt to help waste removal, delivery, and buffering capacity; blood vessels in the muscles try to vasoconstrict but are unable because of the decrease in blood flow, thus in turn causing more inefficiency and fatigue (Fortney et al, 1985).

Even though heart rate increases with exercise, stroke volume decreases because of the decrease in cardiac filling. The decrease in stroke volume is largely due to the decrease in blood flow to the skin and muscles and dehydration. The body reacts by increasing the heart rate even more in an attempt to compensate for the decreased stroke volume. Eventually, VO2, performance, and fatigue are affected and a decrease in exercise tolerance occurs in the heat (Greger et al, 1996).

Causes

CORONARY HEART DISEASE

Although we realise that case reports may be incomplete and therefore may not be entirely representative, a few obvious features emerge from the table. The sample included a preponderance of male subjects, and coronary artery disease accounted for 80 (730O') of all 109 deaths, 34 (425 0o) of them occurring in men with documented risk factors for the disease. Those dying from coronary artery disease were significantly older (p<0-001) than those dying from structural cardiovascular abnormalities (mean ages 40 (SD 9 1) and 19 (6 2) years respectively). In addition, of the group as a whole, 34 subjects (310o) suffered prodromal symptoms such as angina or extreme fatigue.

Immunity to atherosclerosis has been reported who take part in ceremonial runs.'8 Much controversy surrounded an assertion by Bassler in 1977 that fatal coronary artery disease had never been foundin a marathon runner.'9 He postulated that marathon running may protect against atherosclerosis but that running shorter distances mayhave no protective effect. Opie later described three cases of fatal myocardial infarction in marathon runners,20 and subsequently further reports appeared.2' Bassler also claimed that coronary arteries of marathon runners were "enlarged" and "widely patent." This was based on a pathology report of a marathon runner by Currens and White.22 On review of this case, however, the authors clearly stated that atherosclerosis of the coronary arteries was present, the lumen ofthe coronary arteries being reduced to 30o/ diameter at several sites.

Several long distance runners have died from myocardial infarction: perhaps the most notable was Vladimir Kuts, who was gold medallist in both the 5000 and 10 000 metre events in the 1956 Olympic games. He died in 1975 after a myocardial infarction at the age of 48.23 Hence although moderate and regular physical exercise may reduce the risk of coronary atherosclerosis, regular participation in strenuous sport does not confer immunity, as suggested. Perhaps more precise information will result from a current prospective study by the American Medical Joggers' Association,24 whose 4000 members are taking part in a study comparing them with a group of randomly selected physicians with respect to coronary artery disease, exercise behaviour, and risk factors for coronary heart disease. 25 A series of case reports have analysed the factors surrounding sudden death in sport. Opie described 21 sudden deaths in sportsmen occurring within one hour after onset of symptoms, in which 18 were
attributed to coronary artery disease.5 These cases concerned predominantly
rugby football players and referees. In a study of 18 deaths during or after jogging, Thompson et al concluded that 13 had been caused by coronary heart disease.26 Waller and Roberts described five cases of sudden death during running in male runners over 40.21At necropsy all were found to have severe coronary artery disease. On Rhode Island there were 11 cases of sudden death in joggers during 1975-80 attributed to coronary artery disease, the subjects
having an age range of 28-74 years (mean 46 years).Thus coronary artery disease is the most frequent cause of sudden death in sport, particularly in those over 40.

STRUCTURAL CARDIOVASCULAR ABNORMALITIES

Structural cardiovascular abnormalities are the commonest cause of sudden cardiac death in young people. Maron et al reported 29 cases of sudden death in young (13-30 years), highly conditioned competitive athletes.27 In 22 of these death occurred during or in the first hour after sporting activity. In the group as a whole 28 were found later to have structural cardiovascular abnormalities such as hypertrophic cardiomyopathy, which was present in 14.Other abnormalities have included congenital anomalies of the coronary arteries, which account for a small proportion of deaths.2 27 26 The most frequent abnormality is anomalous origin of the left main coronary artery from the right coronary cusp. The risk of sudden death in subjects with this abnormality is 27°o.29 Hypoplasia and atresia of the coronary arteries have also been described and are associated with sudden death.30Valvular heart disease has not been a feature in published reports as a cause of sudden death in sport. As suggested by Lynch,2 this probably reflects the easier detection of such abnormalities at routine medical examination.

OTHER CONDITIONS

Myocarditis has been suggested as a cause of death in sportsmen.3' 32
Few cases, however, have been explained by myocarditis alone, although isolated reports describe myopericarditis2 and chronic myocarditis in young sportsmen.28 Myocarditis is probably not an important factor in sudden death.
Dysfunction of the conduction pathways in the heart leading to fatal arrhythmias has been reported.27 33 In these cases the arteries supplying the sinus node have exhibited bizarre medial hyperplasia and intimal proliferation, thus leading to ischaemia of the sinus node. In many cases no gross pathological cause can be found for sudden cardiac death, but such a conclusion should be reached only after thorough pathological examination of the cardiovascular system, with particular reference to histopathology of the conduction systems.

Prevention

PREPARTICIPATION MEDICAL SCREENING

Since sudden and unaccustomed physical activity is believed to be
more likely to precipitate heart attacks in the unfit,34 it may be more important to "screen" these people before they begin a new sporting activity. Preparticipation medical screening, which has been advocated, would detect overt, potentially lethal cardiovascular disease, but it may be that such disease would have appeared earlier. Routine medical examination would, however, be useful in detecting asymptomatic hypertension but would be relatively insensitive in identifying asymptomatic coronary artery disease or those at risk of fatal arrhythmia. Exercise electrocardiography might help to improve the
detection of those at increased risk and we agree with Chung,35 who advocated screening by exercise electrocardiography for subjects over 40 or in younger subjects with positive risk factors for coronary artery disease before engaging in an exercise programme.

AVOIDANCE OF EXCESSIVE EXERCISE

All sportsmen should be aware of the small risk of sudden cardiac death associated with vigorous sporting activity. Subjects should select a sport suitable for their age and general physical condition. Unfit people should not begin to participate in vigorous, highly competitive sports without a period of preparation. Johnson et al advocated that exercise should be regular, and that sub maximal, rhythmical efforts are preferable until a greater work tolerance has been developed.36 Also the heart rate should not exceed 170 beats/min, particularly if the subject is over 35. In order to adapt to high work rates sportsmen should make an effort to warm up adequately. In addition, possibly as important is a "warm down" at the end of an exercise period. This may reduce the likelihood of arrhythmias in the immediate post exercise period. Exercise programmes for subjects at increased risk should ideally be supervised initially.

PRODROMAL SYMPTOMS

Prodromal symptoms such as chest pain or extreme fatigue are common before sudden death.38 Exercising adults, who may deny these symptoms,5 6 should be encouraged to seek medical attention should any untoward symptoms occur during or before vigorous exercise. In these circumstances play should be discontinued. We are concerned about the over enthusiasm of marathon runners and spectators who help runners to the finishing line when they are on the brink of collapse. This should be discouraged, and may be potentially dangerous. It would also be wise, until further information is available, to avoid participation in vigorous sport when feverish or having symptoms of a respiratory tract infection.

AVOIDANCE OF THERMAL STRESS

Since high temperature increases heart rate and may induce arrhythmias 39 a hot bath or shower immediately after exercise should possibly be avoided. Vigorous activity in extreme heat should also be avoided-in this instance the additional factors of fluid and electrolyte loss may play a part in causing death. Such environmental factors are evident on many squash courts in Britain. In endurance sports such as marathon running fluid and electrolyte loss should be compensated as far as possible. Although "feeding stations" are available at most marathon meetings, not all athletes use this service adequately.

SMOKING

Smoking should be avoided by all sportsmen. Not only is smoking incriminated in coronary artery disease,25 but it can cause an increase in serum free fatty acids40 and release of catecholamines,41 which may interact with one another to cause arrhythmias,4' particularly in the immediate post exercise period.

Conclusions

There is a small risk of sudden cardiac death in subjects participating in vigorous sport. This risk is greater in those with asymptomatic cardiovascular disease,

the more overt forms ofwhich might be detected by medical screening. There is a group of patients, in general over 40, with asymptomatic coronary
artery disease who would not be identified by screening. It may be that those at risk of coronary artery disease-for example, those over 40-should participate in less vigorous sport.Sportsmen should not ignore untoward symptoms, and if such symptoms develop, they should either stop or avoid participation before seeking medical advice. In addition, sportsmen and those concerned with sport should be aware of the risks. Following the precautions outlined above will, we hope, reduce the number of these tragic deaths.

References

1Herodotus. The histories. De Selincourt A, transl. New York: Penguin Books Inc, 1954:397-8.
2 Lynch P. Soldiers, sport and sudden death. Lancet 1980;i:1235-7.
3National Safety Council. Accident facts. Chicago: NSC, 1981.
4Thompson PD, Funk EJ, Carleton RA, Sturner WQ. Incidence of death during jogging in Rhode Island from 1975 through 1980. JAMA 1982; 247:2535-8.
5Opie LH. Sudden death and sport. Lancet 1975;i:263-6.
6 Vuori I, Makarainen N, Jaaskelainen A. Sudden death and physical activity. Cardiology 1978;63 :287-304.
7 Jokl E, McLellan JT. Exercise and cardiac death. Baltimore, Md: University Park Press, 1971.
8 Moritz AR, Zamcheck N. Sudden and unexpected deaths in young soldiers. Archives of Pathology 1946;42:459-593.
9 Adelson L. Sudden death from coronary disease the cardiac conundrum. Postgrad Med 1961 ;30:139-47.
10 Pell S, D'Alonzo CA. Immediate mortality and five year survival of employed men with a first myocardial infarction. N Engl J Med 1964;270:915-22.
11 Kuller L, Lillienfeld A, Fisher R. An epidemiological study of sudden and unexpected deaths in adults. Medicine 1967;46:341-61.
12 Spain DMC, Bradess VA. The relationship of coronary thrombosis to coronary atherosclerosis in ischemic heart disease. Am3' Med Sci 1960; 240 :701-10.
13 Armstrong A, Duncan B, Oliver MF, et al. Natural history of acute coronary heart attacks. A community study. Br HeartjI 1972;34:67-80.
14 Wikland B. Medically unattended fatal cases of ischaemic heart disease in a defined population. Acta Med Scand 1971 ;suppl 524.
15 Koplan JP. Cardiovascular deaths while running. JAMA 1979;242:2578-9.
16 Fowler AW. Cause of death on squash courts. On Call 1980;14:7.
17 Biss K, Ho KJ, Mikkelson B, Lewis L, Taylor CB. Some unique biological characteristics of the Masai of east Africa. N Engl Med 1971 ;284:694-9.
18 Groom D. Cardiovascular observations in Tarahumara Indian runners the modern Spartans. Am Heart3' 1971 ;81 :304-14.
19 Bassler TJ. Marathon running and immunity to athero-sclerosis. Ann NY Acad Sci 1977;301:579-92.
20 Opie LH. Long distance running and sudden death. N EnglJ7 Med 1975;293:941-2.
21 Waller BF, Roberts WC. Sudden death while running, in conditioned runners aged 40 years or over. Am J Cardiol 1980;45:1292-1300.
22 Currens J, White PD. Half a century of running. N Engl3r Med 1961 ;265:251-5.

23 Anonymous. Death of Vladimir Kuts. New York Times 1975 Aug 19.
24 Jones RJ. Mortality of joggers. YAMA 1982;247:2569.
25 Kannel WB, Doyle JT, McNamara PM, Quikenton P, Gordon T. Precursors of sudden coronary death. Factors related to the incidence of sudden death. Circulation 1975;51:606-13.
26 Thompson PD, Stern MP, Williams P, Duncan K, Haskell W, Wood P.Death during jogging or running: a study of 18 cases. JAMA 1979;242:1265-7.
27 Maron BJ, Roberts WC, McAllister HA, Rosing DR, Epstein SE. Sudden death in young athletes. Circulation 1980;62 :218-29.
28 Jokl E, McLellan JT. Exercise and cardiac death. JAMA 1970;213:1489-91.
29 Cheitlin MD, De Castro CM, McAllister HA. Sudden death as a complication of anomalous left coronary origin from the anterior sinus of Valsalva; a not-so-minor congenital abnormality. Circulation 1974;50:780-4.
30 Ogden JA. Congenital abnormalities of the coronary arteries. Am J Cardiol 1970;25 :474-9.
31 Barlow JB. Exercise, rugby football and infection. S Afr MedJ7 1976;50:1351.
32 Kocnar K, Rous J. Preventive approach to sudden cardiac death at sports performance. Br J Sports Med 1973;7:166-7.
33 James TN, Froggatt P, Marshall TK. Sudden death in young athletes. Ann Intern Med 1967;67:1013-21.
34 Shepherd AJ. Sudden death-a significant hazard of exercise. Br J Sports Med 1974;8:101.
35 Chung EK. Exercise ECG testing: is it indicated for asymptomatic individuals before engaging in any exercise program ? Arch Intern Med 1980;140:895-6.
36 Johnson PB, Updyke WF, Stolberg DC, Schaefer M. Physical education: a problem-solving approach to health andfitness. New York: Holt-Rinehart-Winston, 1966.
37 Carruthers M, Nixon P, Murray A. Safe sport. Lancet 1975;i:447.
38 Alonzo AA, Simon AB, Feinlab M. Prodromata of myocardial infarction and sudden deaths. Circulation 1975;52:1056-62.
39 Taggart P, Parkinson P, Carruthers M. Cardiac responses to thermal, physical and emotional stress. Br MedJ 1972;iii:71-6.
40 Kurien VA, Yates PA, Oliver MF. The role of free fatty acids in the production of ventricular arrhythmias after acute coronary artery occlusion. EurJf Clin Invest 1971 ;1 :225.
41 Opie LH, Norris RM, Thomas M, Holland AJ, Owen P, Van Noorden S. Failure of high concentrations of circulating free fatty acids to provoke arrhythmias in experimental myocardial infarction. Lancet 1971;i:818.
42 Johnson RH, Walton JL, Krebs HA, Williamson DM. Metabolic fuels during and after severe exercise in athletes and non-athletes. Lancet 1969 ;ii :452-5.