Sudden death

Sudden death risk in older athletes: increasing the denominator

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Excluding the older athlete should be a last resort

Publicity and campaigning surrounding the tragedy of sudden death in young athletes (incidence 1 in 200 000 young athletes per year)[1] has rather overshadowed the mortality risk of older competitors aged >30.

Population studies show that death rates during sports participation increase dramatically with age[2] as the incidence of coronary heart disease increases. Is this just coincidental, or is the sport triggering the deaths? The highest overall mortality (numbers dying—"the numerator") is in recreational sports favoured by the middle aged and elderly, such as fishing and lawn bowls. This is because of the large numbers of participants and their lengthy exposure (time spent participating in the sport) ("the denominator") in assessing comparative risk. The latter will vary with different populations of participants.

$$\text{Sport exposure risk} = \frac{\text{Number of deaths (Numerator)}}{\text{Number of participants} \times \text{Time of exposure to risk (Denominator)}}$$

Lack of information about the denominator means that in most sports and recreational activity the exposure risk cannot be calculated and so compared. Collecting the death statistics without the denominator is almost meaningless, and can lead to illogical deductions, for instance that recreational fishing is more dangerous than hang gliding.

> "His death had seriously held up play, and the ambulance had damaged the grass."

Many sports have their share of older coronary prone participants. I recall visiting a golf club many years ago the day after a sudden coronary death on the 12th fairway. Members felt that it was very inconsiderate of the deceased, who had had previous cardiac events. His death had seriously held up play, and the ambulance had damaged the grass. He should not have played.

Should we try to prevent older athletes with high risk from participating, and possibly upsetting other participants? This would mean screening them, stratifying the risk, trying to exclude those with high risk, and giving those passing the screening regular subsequent checks. This would be expensive for rather poor predictive value[3] and likely to inhibit healthy, beneficial exercise for the majority.

Or should we, as one of my (now deceased) patients suggested to me, encourage our ageing population to take up increasingly risky pursuits including dangerous sports in order to reduce the risks of them becoming a long term geriatric burden? A heretical and provocative view! Older people are on the whole more risk averse. Dangerous sports and pursuits cause not only death but can cause chronic disability. However, when aged >75, with limited hearing, eyesight, and mobility, even crossing the road can become a dangerous "sport".

Older "athletes" are being encouraged by publicity surrounding mass participation events, such as marathons, and by health education to exercise and "have a go" in many sports. There have been remarkable performances by what one hesitates to call "the elderly". A 70 year old has climbed Mount Everest and another has become the oldest successful English Channel swimmer. The 2004 London Marathon reported several 80 year olds and a 92 year old runner, who finished in 6 hours 7 minutes.

The age distribution of the London Marathon shows the largest numbers in the half decade 35–39 years old inclusive, the next largest is 40–44 inclusive.

What are the death risks in these older athletes? These are overwhelmingly from coronary heart disease.[4 5] Predicting the risk is complicated. Whereas regular aerobic exercise reduces the risk of coronary events overall, and reduces risk factors for coronary artery disease, there is no doubt that exertion increases the risk of coronary events in those who have ischaemic (and other) heart disease.[6] Exercise (exertion) prevents, but also precipitates cardiac events.

To predict the risk for any particular event such as the London Marathon with its 32 000 participants you would need to know:

- the age and sex distribution of the competitors
- the incidence of coronary disease in the various population subgroups entering the marathon
- the duration of exposure to risk
- the intensity of exercise and its accompanying increased risk.

This list contains a lot of unknowns, but there are more.

Is the risk linear with time spent running in the marathon? Probably not, but data recording reduced risk from road races of shorter distance suggest that time of exposure is important rather than just peak intensity of exercise, which would be higher in shorter distance races and would give the opposite effect.[7]

Calculations are complicated by the fact that marathon runners are not a randomly selected subgroup of the population. Some older athletes take up exercise as a lifestyle change. They aim to reduce their known high risk of coronary events and may believe the claims of the now discredited 1970s running "gurus" James Fixx (author of *The complete book of running*) and Dr Tom Bassler, that if they take enough exercise they are immune from, or can even reverse, coronary artery disease.

"I had coronary artery surgery 15 years ago and have cured my heart disease by running marathons," says a runner raising money for the British Heart Foundation, in a report from an East Anglian newspaper. Such naivety is not uncommon and may lead to a dangerous denial of symptoms.

The distribution of coronary risk may therefore be distorted by these factors, making prediction difficult. What are the measured risks? Road running is one of the few sports with large numbers of participants and measured exposure.[8 9] Associated with 580 000 runs in the London Marathon since 1981, there have been eight deaths. One was from subarachnoid haemorrhage, two from hypertrophic cardiomyopathy, and five from coronary heart disease. (There have also been five successful cardiac resuscitations, all with coronary heart disease.) Counting all the eight deaths (including the 22 year old runner with subarachnoid haemorrhage) and postulating the average time of exposure as 4.5 hours, this gives the following statistics on the exposure death risk of running the London Marathon (table 1).

The death rate normalised for "time of exposure" can be compared with day

Table 1 London Marathon deaths over 24 years compared with European Transport Safety Council travel risks 2001–2002 (>580 000 marathons, 25 million km, eight deaths)

Mode of transport	Deaths/100 million km	Deaths/100 million hours	Deaths/ 100 years	Normalised death risk/ time exposed
London Marathon 1981–2004	32	308	2.67	12
Motorcycle	13.8	440	3.81	18
Bicycle	5.4	75	0.65	3
Car	0.7	25	0.21	1
Airline	0.035	16	0.138	0.67
Rail	0.035	2	0.017	0.08

to day risks of road vehicular transport in Europe and is less than that for motorcycles (two thirds), but four times that of riding a bicycle for the same length of time. The transport figures are from the European Transport Safety Council and have been updated since those used in earlier publications.[9 10]

In the last few years, the death risk of European travel has become safer with a larger denominator (number of people travelling on the roads).

Increasing participation of older athletes (denominator), with all the health benefits for the vast majority, will probably result in an increase in the total number of sports deaths. However, this should be more than balanced by a decrease in the overall risk of death in those taking regular exercise.

Dying on the golf course, although unfortunate, should become more socially acceptable as the benefits of exercise even for the coronary prone are more generally appreciated.

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Tendinopathy

Reactive oxygen species and tendinopathy: do they matter?

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Reactive oxygen species are probably involved in tendinopathy

We propose that a molecular link between the exaggerated dysfunctional repair response in overuse tendinopathies and the subsequent orchestration of effective tendon healing is the control of the production and persistence of reactive oxygen species within the intracellular and extracellular milieu of the tendon tissue. Reactive oxygen production and the ensuing cellular response can be strongly influenced by lifestyle factors such as the intensity and frequency of exercise.

''Reactive oxygen species'' (ROS; also referred to as active oxygen species, AOS; reactive oxygen intermediates, ROI) is a collective term for both radical and non-radical but reactive species derived from oxygen. A free radical, is ''any species capable of independent existence that contains one or more unpaired electrons''.[1] The presence of such unpaired electron(s) often imparts considerable reactivity. Commonly detected and potentially physiologically relevant ROS include the superoxide anion, hydrogen peroxide (H_2O_2), the hydroxyl radical, singlet oxygen, and peroxyl radicals. A further and interrelated group are the reactive nitrogen species (RNS)—for example, peroxynitrite.[1]

ROS are continually produced during normal cell metabolism. The mitochondrial respiratory chain, NADPH-cytochrome P_{450} enzymes in the endoplasmic reticulum, phagocytic cells, lipoxygenase, and cyclo-oxygenase are also sources of basal ROS production.[1] Trauma and environmental and physiological stimuli may enhance ROS production.[1]

Traditionally, ROS are viewed as imposing cellular/tissue damage through lipid peroxidation, protein modification, DNA strand cleavage, and oxidative base modification, although the relative reactivity and susceptibility of the molecular targets vary. Thus, ROS production is implicated in numerous aspects of pathophysiology including tumorigenesis, coronary heart disease, autoimmune disease, overuse exercise related damage to muscle, and impairment of fracture healing.[1 2]

This association with cellular damage and pathology has predisposed much of the literature to consider decreased ROS production de facto a universally desirable phenomenon. This, however, belies the complexity of ROS action, in which subtle changes in ROS type and concentration may exert profound effects on cell metabolism and development including proliferation, differentiation, and adaptive responses. At higher levels, ROS may initiate and/or execute the demise of the cell. The ability of H_2O_2 to diffuse across membranes imparts potential to exert effects at sites distant