

Downhill Skiing: A Putative Model of Hypoxia Preconditioning?

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Abstract

Sudden cardiac death (SCD) represents the leading non-traumatic death during downhill skiing, the most popular winter sport worldwide. Remarkably, about 50% of all SCDs happened on the first skiing day particularly when the preceding sleeping altitude was low. From these observations it may be inferred that the SCD risk can be modified by short-term adaptation, i.e. hypoxia preconditioning (HP). Downhill skiing at moderate or high altitude may represent a unique model of HP. In fact, analyses of our data on SCDs in male downhill skiers are consistent with an episode of early protection for about 3 hours and a subsequent vulnerable episode during the first skiing day at altitudes at or above 1700 m.

Sudden cardiac death (SCD) represents the leading non-traumatic death during downhill skiing, the most popular winter sport worldwide. More than 90% of all SCDs are attributed to male skiers over 34 years with pre-existing cardiovascular diseases [1]. Remarkably, about 50% of all SCDs happened on the first skiing day particularly when the preceding sleeping altitude was low [2]. From these observations it may be inferred that the SCD risk can be modified by short-term adaptations in terms of preconditioning. Both exercise and hypoxia are capable to induce such adaptations, e.g. including the respiratory, cardiovascular, metabolic and autonomic nervous systems, potentially contributing to subsequent cardio protection [3,4]. Since sleeping at moderate altitude before exercising at altitude reduced the risk of SCD, hypoxia preconditioning (HP) seems to be of particular importance. Preconditioning describes a powerful endogenous mechanism protecting an organ against damage. After single or repeated exposures to a noxious stimulus below the threshold of damage, e.g. hypoxia, the organism develops tolerance to similar or even different stimuli [5]. Hypoxia has been used as a preconditioning stimulus since the 1990ties and has emerged as an easily and safely applicable stimulus preventing several organs including the heart against subsequent injury [6].

The protective effects of HP result from a single exposure or several short episodes (2 to 10 min) of hypoxia. Protection occurs immediately (early protection), lasting for a few hours, or delayed (late protection after a vulnerable phase of about 24 hours), lasting for several days [3,4]. The preventive efficacy of HP has extensively been studied in animals but experimental and clinical data from humans are lacking. However, studies performing remote ischemic preconditioning confirmed the time course of early and late protection, as stated above, against endothelial ischemia-reperfusion injury in humans [7]. Thus, downhill skiing at moderate or high altitude may represent a unique model of HP. Skiers perform repeated downhill runs of a several minutes duration using ski lifts or cable cars to ascend repeatedly from lower to higher altitude. At about 2000 m arterial oxygen saturation values remain relatively high during rest (>90%) but drop below 85% during short physical activities especially

on the first day at altitude [8]. If cardio protective HP effects are generated during downhill skiing they should become evident from the time-dependent occurrence of SCDs at altitude. In fact, analyses of our data on SCDs in male downhill skiers are consistent with an episode of early protection for about 3 hours and a subsequent vulnerable episode during the first skiing day at altitudes at or above 1700 m (Figure 1).

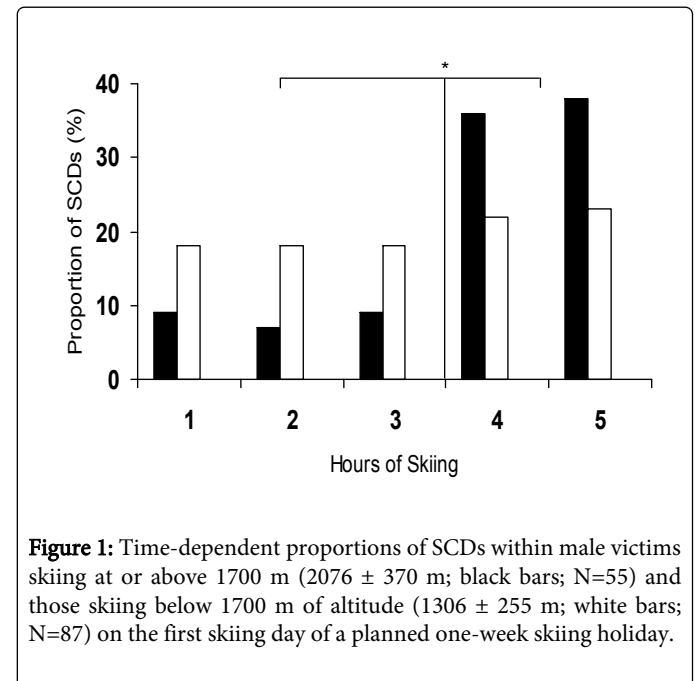


Figure 1: Time-dependent proportions of SCDs within male victims skiing at or above 1700 m (2076 ± 370 m; black bars; N=55) and those skiing below 1700 m of altitude (1306 ± 255 m; white bars; N=87) on the first skiing day of a planned one-week skiing holiday.

*indicates significant between-group differences in SCD frequencies during the first 3 skiing hours vs. those during the hours 4 and 5 ($P < 0.05$). There are no differences between groups regarding age, body mass index, cardiovascular risk factors, regular physical activity, skiing skills, living altitude, daily skiing duration, and day-time of skiing start.

In addition, after 3 hours of skiing several triggers like lactic acidosis, increased serum free fatty acid concentrations, hypoglycaemia, or intra- and extracellular electrolyte derangements may contribute to the increase of the SCD risk. No altitude-dependent differences in SCD frequencies were found from the second to the fifth skiing day. Thus, skiers at risk might actually benefit from HP but should urgently avoid skiing longer than 3 hours on the first day at altitudes above 1700 m. In fact, this clinical setting could represent a real-life model for HP but is clearly limited by the largely varying altitudes of ski areas, the varying individual skiing intensities and exposure times, and the small number of patients as well.

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