Mortality on Mount Everest, 1921-2006: descriptive study

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Abstract

Objective To examine patterns of mortality among climbers on Mount Everest over an 86 year period.

Design Descriptive study.

Setting Climbing expeditions to Mount Everest, 1921-2006.

Participants 14 138 mountaineers; 8030 climbers and 6108 sherpas.

Main outcome measure Circumstances of deaths.

Results The mortality rate among mountaineers above base camp was 1.3%. Deaths could be classified as involving trauma (objective hazards or falls, n=113), as non-traumatic (high altitude illness, hypothermia, or sudden death, n=52), or as a disappearance (body never found, n=27). During the spring climbing seasons from 1982 to 2006, 82.3% of deaths in climbers occurred during an attempt at reaching the summit. The death rate during all descents via standard routes was higher for climbers than for sherpas (2.7% (43/1585) *v* 0.4% (5/1231), P<0.001; all mountaineers 1.9%). Of 94 mountaineers who died after climbing above 8000 m, 53 (56%) died during descent from the summit, 16 (17%) after turning back, 9 (10%) during the ascent, 4 (5%) before leaving the final camp, and for 12 (13%) the stage of the summit bid was unknown. The median time to reach the summit via standard routes was earlier for survivors than for non-survivors (0900-0959 *v* 1300-1359, P<0.001). Profound fatigue (n=34), cognitive changes (n=21), and ataxia (n=12) were the commonest symptoms reported in non-survivors, whereas respiratory distress (n=5), headache (n=0), and nausea or vomiting (n=3) were rarely described.

Conclusions Debilitating symptoms consistent with high altitude cerebral oedema commonly present during descent from the summit of Mount Everest. Profound fatigue and late times in reaching the summit are early features associated with subsequent death.

Introduction

The summit of Mount Everest is the highest point on earth (8850 m above sea level). Thousands of mountaineers have tried to scale Everest, and many have died. We examined the circumstances of such deaths to establish patterns of mortality among the mountaineers.

Methods

We carried out a retrospective study of deaths during expeditions on Everest during 1921-2006. The population included all members and employees of these expeditions. We identified deaths through published accounts of expeditions and by searching the Himalayan Database, a registry of expeditions on the Nepalese Himalayas.[1] We counted repeat visits and successes on reaching the summit as independent events. We defined the altitude of death as that at which a fatality occurred or was estimated to have occurred, from where a fall occurred, or from where a non-survivor was evacuated. A fatal incident was defined as an event involving one or more people on an expedition, or climbing together, between camps. We estimated altitude in incidents where mountaineers died at different altitudes.

Four experienced doctors independently examined the accounts, with disagreements resolved by consensus. Deaths involving trauma were classified by the type of trauma. We categorised high altitude cerebral or pulmonary oedema as high altitude illness, using accepted criteria[2] or reliable reports. We also classified deaths as high altitude illness if a diagnosis of cerebral or pulmonary oedema could not be established, symptoms of acute mountain sickness were present,[3] and no other primary cause of the symptoms was evident. Death due to exposure was classified as hypothermia. Sudden death was defined as abrupt death without previous progressive symptoms. We listed death as unclassified when a primary cause could not be established. Deaths were classified as disappearances if the death or immediate cause of death was not witnessed and the body not found.

We examined the accounts of deaths in mountaineers who reached 8000 m to determine the stage of the climb, speed of the climber compared with team mates, and mention of visual disturbances and symptoms of high altitude illness.[3] From the Himalayan Database we obtained the times mountaineers reached the summit.[1]

Statistical analysis

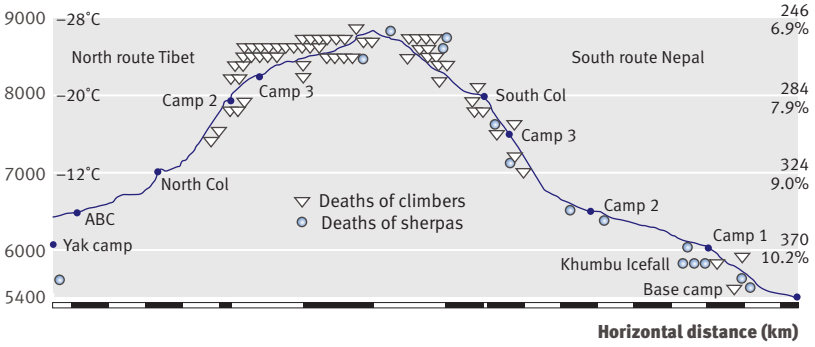
We used Fleiss' kappa method to analyse inter-rater agreement for classification of death.[4] For univariable analyses of binary and categorical outcomes we used a χ^2 test or Fisher's exact test. We used the two sided *t* tests or a Wilcoxon rank sum test to compare continuous outcomes, including summit time and number of deaths due to objective hazards per incident, whenever appropriate.

Results

The table outlines the characteristics of the study population. Overall, 341 accounts were analysed, including 136 notes from the Himalayan Database, 106 journal reports, 31 books, 32 direct accounts by 22 climbers, 14 web based accounts, and 7 miscellaneous sources. In total, 154 fatal incidents resulting in 212 deaths were identified. For the classification of all 212 deaths, there was unanimous independent agreement for 165 (78%). The Fleiss' κ value for inter-rater agreement was 0.63, P<0.001 (substantial agreement).[4] Of deaths classified at all altitudes, fatal cerebral oedema occurred at a higher altitude than fatal pulmonary oedema (7 *v* 5; 8276 (SD 791) m *v* 6229 (SD 104) m, P=0.001). Table 2 on bmj.com provides the classification of deaths above base camp; see www.himalayandatabase.com for further data.[1]

Above base camp, 103 incidents involving climbers occurred at a higher mean altitude than 44 involving sherpas (7854 (SD 918) m *v* 6927 (SD 116) m, P<0.001). Thirty seven incidents involved objective hazards, with more sherpas killed per incident than climbers (1.18 *v* 0.54, P=0.02). The mean altitude of events involving objective hazards was 6381 (SD 661) m.

The death rate during all descents from the summit via standard routes was higher for climbers than for sherpas (2.7% (43/1585) *v* 0.4% (5/1231), P<0.001; all mountaineers 1.9%). The figure presents the mortality and distribution of deaths on the standard routes during the spring climbing seasons, 1982-2006. Of 77 deaths, 55 occurred during bids for the summit (51 climbers) and 22 during route preparation (11 climbers).



Route	Mountaineers	Death rate during descent from summit (%)	P value*
North	Climbers	3.4	0.0001
	Sherpas	‹0.2	
South	Climbers	1.7	0.02
	Sherpas	0.4	
Combined north and south	Climbers	2.5	0.0001
	Sherpas	0.2	
North	Mountaineers	2.0	0.1
South	Mountaineers	1.1	

Deaths on standard north and south routes of Everest during spring climbing season (April-June) 1982-2006. Deaths during descent are above route profile and deaths before summiting or during bids for summit with unknown outcome are below. One sherpa died below Yak camp during evacuation. Right axis shows estimated barometric pressure during May,[5] and percentage of oxygen at sea level (760 mm Hg) that exerts equivalent partial pressure to atmospheric oxygen at relevant altitude. Left axis shows estimated ambient air temperature during May.[6 7] Scale on x axis is expanded by factor of two for route above 8000 m. *Two sided Fisher's exact test

Population characteristics of mountaineers on Everest, 1921-2006. Values are numbers (percentages) unless stated otherwise

Variables	Climbers (n=8030)	Sherpas (n=6108)	Total
Male	7404 (92.2)	6106 (99.9)	13 510
Female	626 (7.8)	2 (0.1)	628
Mean (SD) age (years) (range)	36.5 (8.9) (12-74)	—*	—
Overall total	8030	6108	14 138
Summit ascents:			
1953-81 (all)	94 (5.3)	23 (1.8)	117 (3.8)
1982-2006:			
S-spring	663 (37.5)	661(51.2)	1324 (43.3)
N-spring	732 (41.4)	484 (37.5)	1216 (39.8)
Other†	279 (15.8)	122 (9.4)	401 (13.1)
Total	1768	1290	3058

S-spring=standard Nepalese route via South Col or Southeast Ridge, or minor variants, during April to June; N-spring=standard Tibetan route via North Col or Northeast Ridge, or minor variants, during April to June.
Population includes estimates of expedition size and roles of mountaineers (climbers or sherpas) for Chinese expeditions, 1960-79, on north side of Everest. Percentages may not add up to 100% owing to rounding.
*Data were unreliable.
†All other routes such as West Ridge or North Face approaches, or attempts on all routes during seasons other than spring.

Table 3 on bmj.com presents data on the mountaineers who died after reaching 8000 m. Fifty three (56%) died during the descent, 16 (17%) after turning back, and nine (10%) during the ascent. The stage of the summit bid was unknown for 12 mountaineers (13%), and four (5%) died before leaving camp. Of 23 mountaineers who died while climbing without supplemental oxygen, 11 (48%) died during the descent, 4 (17%) after turning back, and 4 (17%) during the ascent; the stage was unknown in 4 (17%). Median time to reach the summit was earlier for survivors than for non-survivors (0900-0959 (interquartile range 0800-0859 to 1100-1159) *v* 1300-1359 (1100-1159 to 1600-1659), P<0.001). Thirty seven climbers died or were in extremis in the presence of witnesses, 34 fell behind team members before becoming debilitated or dying, 11 were unknown, 11 climbed alone, and one could not be classified owing to insufficient information. Thirty three had symptoms of high altitude illness and six had symptoms of high altitude illness or hypothermia. The frequency of symptoms of high altitude illness was noticeable fatigue or exhaustion (n=34), confusion or coma (21), ataxia (12), respiratory disturbances (5), nausea or vomiting (3), and headache (0).

Discussion

This study examined the patterns of deaths over 86 years (1921-2006) on Mount Everest. The mortality above base camp was 1.3% (see bmj.com). Most sherpas were killed in incidents on the lower slopes, whereas most climbers died above 8000 m. Climbers typically died during descent from the summit, often developing cognitive impairment and ataxia, symptoms of high altitude cerebral oedema. Profound

fatigue, late summit times, and the tendency to fall behind companions were common early features of non-survivors.

Strength and limitations

The strength of this study lies in the records kept over four decades on the Himalayan Database.[1] An assessment of deaths was, however, limited by the variability of circumstances. While there was substantial agreement between the reviewers on classification of the deaths,[4] categorisation relied on a descriptive system. This may underestimate underlying problems, such as neurological dysfunction leading to falls, disappearances, or vulnerability to hypothermia at extreme altitude. Although a retrospective study can only show an association and not prove causality, this simple descriptive technique allows broad patterns of mortality to be detected.

Distribution of deaths

The largest class of deaths involved objective hazards such as avalanches or falling ice. Sherpas were killed at a greater rate per incident than climbers (1.18 *v* 0.54, P=0.02). These incidents typically occurred on the lower sections of routes, passing below slopes prone to avalanches. The higher death rate per incident among sherpas can largely be explained by more time spent transporting equipment in these areas. During the spring climbing seasons of the last 25 years of the study period, deaths from objective hazards were rare. By contrast, 85.4% of deaths on the north route and 43.9% of deaths on the south route occurred above 8000 m (figure). While a typical expedition to Everest now lasts about 60 days, 82.3% of deaths among climbers occurred during or after the day of a summit attempt.

Deaths above 8000 m

Climbers died during descent at a greater rate than sherpas (2.5% *v* 0.2%, P<0.001). Gross cognitive impairment and ataxia were common among non-survivors. These progressive symptoms and delayed presentation are consistent with high altitude cerebral oedema, a vasogenic oedema predominantly caused by failure of vascular endothelial fluid regulation after inadequate acclimatisation to hypoxia.[2 8 9] Acute hypoxaemia during descent due to the exhaustion of supplemental oxygen supplies is another potential explanation.[10] A similar pattern of death during descent is, however, seen in climbers with and without supplemental oxygen above 8000 m. Arterial oxygenation decreases during strenuous exercise at high altitude,[11 12] so maximal hypoxaemia would occur during vigorous exertion below the summit. However, we identified only two cases of sudden death that occurred during ascent, both in climbers using supplemental oxygen. Among non-survivors neurological dysfunction, and possibly the susceptibility to acute hypoxia after exhaustion of supplemental oxygen, therefore typically progresses with time at extreme altitude. This is characteristic of the cerebrovascular leak and disruption of fluid homoeostasis of high altitude cerebral oedema, rather than of acute hypoxaemia alone. In addition, hypoxic neurological dysfunction at extreme altitude may involve other pathophysiological mechanisms not prominent at lower elevations.

Respiratory distress, nausea, vomiting, and headache were rarely noted in non-survivors above 8000 m. This may be because people with these symptoms turn back earlier. The mechanisms of the headache of cerebral oedema are unclear but may relate to the acute distension of blood vessels and meninges at the base of the brain during raised intracranial pressure.[13] An acute decrease in intravascular volume is a normal physiological response to high altitude,[14] and dehydration, due to the increased physiological demands and need to melt sufficient snow, is a common problem above 8000 m. Acute decreases in fluid volumes may slow the increase in intracranial pressure and related headache after fluid extravasation. Ataxia and confusion may occur with a lesser degree of oedema due to the hypoxaemia at extreme altitude. We speculate that at extreme altitude headache, nausea, and vomiting are unreliable heralds of fatal cerebral oedema, whereas debilitating ataxia and impaired consciousness present earlier than these symptoms. Profound fatigue, reflected in significantly later summit times, was an early symptom of subsequent non-survivors.

Acclimatisation to extreme altitude

Since neurological symptoms are present in many non-survivors, critical questions include whether adequate acclimatisation is possible at this altitude and, if so, how can it best be achieved. Climbers died at over six times the rate of sherpas during the descent from the summit. The differing mortality during descent may be related to better acclimatisation by sherpas, since they spend more time at higher altitude transporting equipment. However, most sherpas are of Sherpa extraction, an ethnic group from the Nepali highlands that may have congenital and acquired adaptations to hypoxia compared with their lowland employers.[15 16] While one subpopulation of mountaineers can acclimatise sufficiently to climb Everest using supplemental oxygen with relatively low mortality,

WHAT IS ALREADY KNOWN ON THIS TOPIC

What is already known on this topic

The death rate on Mount Everest is greater than that of lower mountains attempted by similar populations of climbers

The death rate among climbers is higher than that among sherpas

WHAT THIS STUDY ADDS

Most climbers on Mount Everst die above 8000 m, usually during descent from the summit

Cognitive impairment and ataxia, symptoms of cerebral oedema, are often present

Profound fatigue and late summit times are early features of non-survivors

the interaction and relative importance of the differences between populations and acclimatisation profiles requires further study.

Competing interest: RAS has a financial interest in the Himalayan database.

Ethical approval: This study was approved by the institutional review board of Massachusetts General Hospital.

Provenance and peer review: Not commissioned; externally peer reviewed.

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Voices in the air

Jeremy S Windsor wonders how to explain the benign presence he met on Mount Everest

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After leaving Eric a strange feeling possessed me that I was accompanied by another … The "presence" was strong and friendly. In its company I could not feel lonely, neither could I come to any harm, it was always there to sustain me on my solitary climb up the snow covered slabs. Now as I halted and extracted some mint cake from my pocket, it was so near and so strong that instinctively I divided the mint into two halves and turned round with one half in my hand to offer it to my "companion."[1]

I first met Jimmy on the Balcony, a cold windswept snow shelf high up on the southeast ridge of Mount Everest. At an altitude of more than 8200 metres our introduction had been brief, with little more than a muffled "hello" and a few words of encouragement passing between us. Over my right shoulder, obscured by the bulky oxygen mask and the rim of down that smothered my face, I was sure I could see Jimmy moving lightly in the darkness. But despite him remaining close by me for the rest of the day, I didn't see him again. At the time, it hadn't worried me; instead I was warmed by the thought of human company and too breathless to question what seemed so real. If the truth be told, my thoughts were really nothing more than brief flickers of images or sounds that vanished with the onset of each new breath. Not only was I "stupid from lack of oxygen," as one Mount Everest mountaineer once so memorably put it, but I was exhausted. After nearly three months on Everest I had lost almost a fifth of my

DUNCAN SMITH

body weight, owing in large part to bouts of altitude sickness and my fussy vegetarian diet. Higher on the mountain, the weight loss had made matters worse. Without a decent amount of fat to insulate me, it had become difficult to sleep at night and I would doze through the day ignoring the need to drink and consume precious calories.

The climb to the Balcony had been achieved on little more than willpower, and standing beside my new companion I soon realised that even this energy had begun to fade. Jimmy seemed to have fared much better. His few words had been measured and calm, filled with a confidence that I badly lacked: "Come on, change your cylinder and get moving." Jimmy's voice was firm and commanding, stirring me into activity. Here my memory falters. I don't recall changing the cylinder or even the decision to carry on along the fixed ropes, but I do remember Jimmy. Sometimes as I inched my way along I would hear the rhythmic scratches of his crampons on the icy path and even feel the smooth tugs of his ascender on the rope that we shared. As we rested, my new climbing partner would stand so close that I'd be able to hear the soft whisper of oxygen entering his facemask and the rattle of breath as he exhaled. Throughout the hours we spent together Jimmy would often speak, answering my questions and offering a mixture of firm commands and gentle words of encouragement to drive me on. As we climbed higher we even indulged in making plans for what we'd do on the summit and how we'd celebrate when we returned to Kathmandu. Finally, after almost 10 hours of climbing we emerged on the South Summit, the sun beginning to stir out to the east, emphasising the curvature of the earth visible far on the horizon. Like a football match commentator describing Wembley stadium on Cup Final day, Jimmy pointed out the route ahead, the famous Hillary Step, and the location of the fixed ropes that skirted along the icy cornice and finished on the summit. I knew at that point that I'd make it, and so did Jimmy. After a few more encouraging words he whispered a final "cheerio" and was gone.

I hadn't been the first to encounter someone like Jimmy on Everest. Over the course of almost a century of climbs on the mountain, many similar experiences have been recounted. For some mountaineers, these "companions" have had a clear visual form, such as that described by Nick Estcourt during a climb to Camp 5 on the first successful ascent of the southwest face in 1975: "I turned round and saw this figure behind me. He looked like an ordinary climber, far enough behind, so that I could not feel him moving up the fixed rope, but not all that far below. I could see his arms and legs and assumed that it was someone trying to catch me up."[2]

For others, such hallucinations have tended to be almost invisible, ranging from faint noises or "voices in the air" to familiar characters capable of holding lengthy conversations. During a night spent in a snow hole close to the summit, Dougal Haston later recalled: "I was locked in suffering silence except for the occasional quiet conversation with Dave Clarke. Hallucination or dream? It seemed comforting and occasionally directed my mind away from the cold."[2]

For many of us, companions like Jimmy have tended to be a source of comfort, providing not only a sense of companionship but sometimes practical help as well. In 1988 Stephen Venables completed an extraordinary ascent of the Kangshung Face without supplemental oxygen and was forced to sit out the night on a small ledge just below the summit. At times, Venables realised that a crowd had formed around him: "Sometimes they offered to look after parts of my body … Perhaps it was then that Eric Shipton, the distinguished explorer so closely involved with the history of the mountain, took over warming my hands. At the end of the ledge my feet kept nearly falling off where I had failed to dig a thorough hollow in the snow. I was aware of several people crowding out the feet, but also trying to look after them."[3]

What is to be made of these benign figures? As a doctor I want to believe that Jimmy's appearance was linked to the physical and psychological stresses that I felt that day. Herbert Tichy, the first to climb Cho Oyu (8201 metres) in 1954, summed it up well when he wrote: "Things like this arise because the spirit has somehow broken free from the anchorage which holds it fast far down in the valley, and strays right up to the very frontiers of insanity."[4]

But as a superstitious mountaineer I'm swayed by an alternative explanation. Peter Habeler, who with Reinhold Messner in 1978 became the first to climb Everest without supplemental oxygen, draws a different conclusion and links those climbing the mountain to previous generations: "There is a saying that whoever is killed up on the mountain wanders forever after his death, and guides the living mountaineers during their last metres to the summit."[4]

Today, my memories of Jimmy are so clear and vivid that it sometimes makes it impossible to embrace the scientific explanations that I should wholeheartedly accept. But whatever the explanation for Jimmy's appearance, I know now that I wouldn't have reached the summit without him.

Competing interests: None declared.

Provenance and peer review: Not commissioned; not externally peer reviewed.

JSW was a member of Caudwell Xtreme Everest, a research project coordinated by the UCL Centre for Altitude, Space and Extreme Environment Medicine, University College London. The aim of the project was to conduct research into hypoxia and human performance at high altitude to improve understanding of hypoxia in critical illness. JSW reached the summit of Mount Everest on 24 May 2007 accompanied by Ang Kaji Sherpa, Dawa Tenji Sherpa, Lila Chhombe Basnet, Michael Brown, Pemba Gyalzen Sherpa, Pemba Nuru Sherpa, Michael O'Dwyer, and Roger McMorrow.

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Rugby (the religion of Wales) and its influence on the Catholic church: should Pope Benedict XVI be worried?

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Abstract

Objective To explore the perceived wisdom that papal mortality is related to the success of the Welsh rugby union team.

Design Retrospective observational study of historical Vatican and sporting data.

Main outcome measure Papal deaths between 1883 and the present day.

Results There is no evidence of a link between papal deaths and any home nation grand slams (when one nation succeeds in beating all other competing teams in every match). There was, however, weak statistical evidence to support an association between Welsh performance and the number of papal deaths.

Conclusion Given the dominant Welsh performances of 2008, the Vatican medical team should take special care of the pontiff this Christmas.

Introduction

In recent times, an intriguing urban legend has arisen in Wales: "every time Wales win the rugby grand slam, a Pope dies, except for 1978 when Wales were really good, and two Popes died" (http://news.bbc.co.uk/sport1/hi/funny_old_game/4449773.stm). We used historical data to examine whether the Vatican medical team caring for Pope Benedict XVI should be especially vigilant in this, a year in which Wales won the grand slam (http://en.wikipedia.org/wiki/Grand_Slam_(Rugby_Union) and http://en.wikipedia.org/wiki/Grand_Slam_(Rugby_Union)).

Methods

We investigate both parts of this claim, and refer respectively to them as the special and general theories of papal rugby. The special theory indicates the

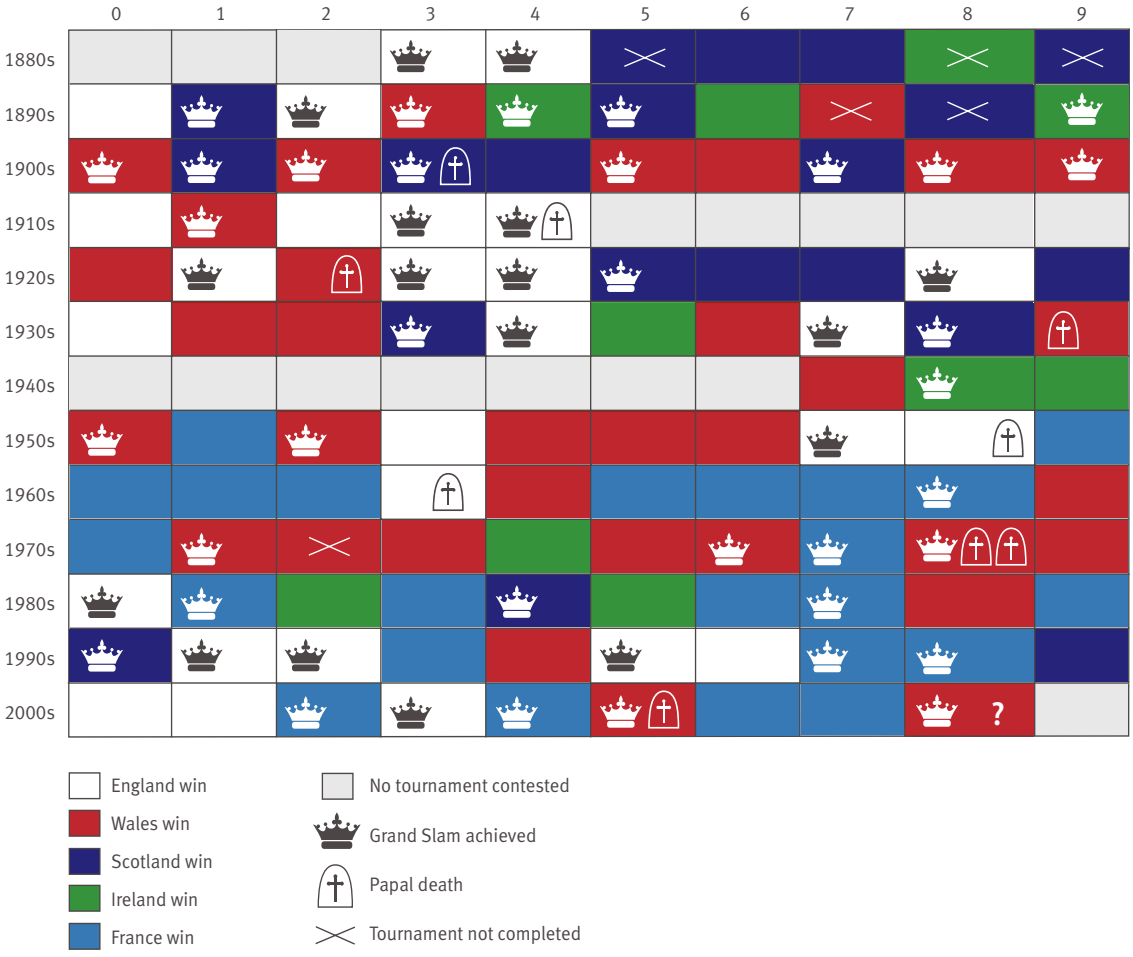

Fig 1 | **The winners of the rugby tournament with the years of grand slams and papal deaths marked**

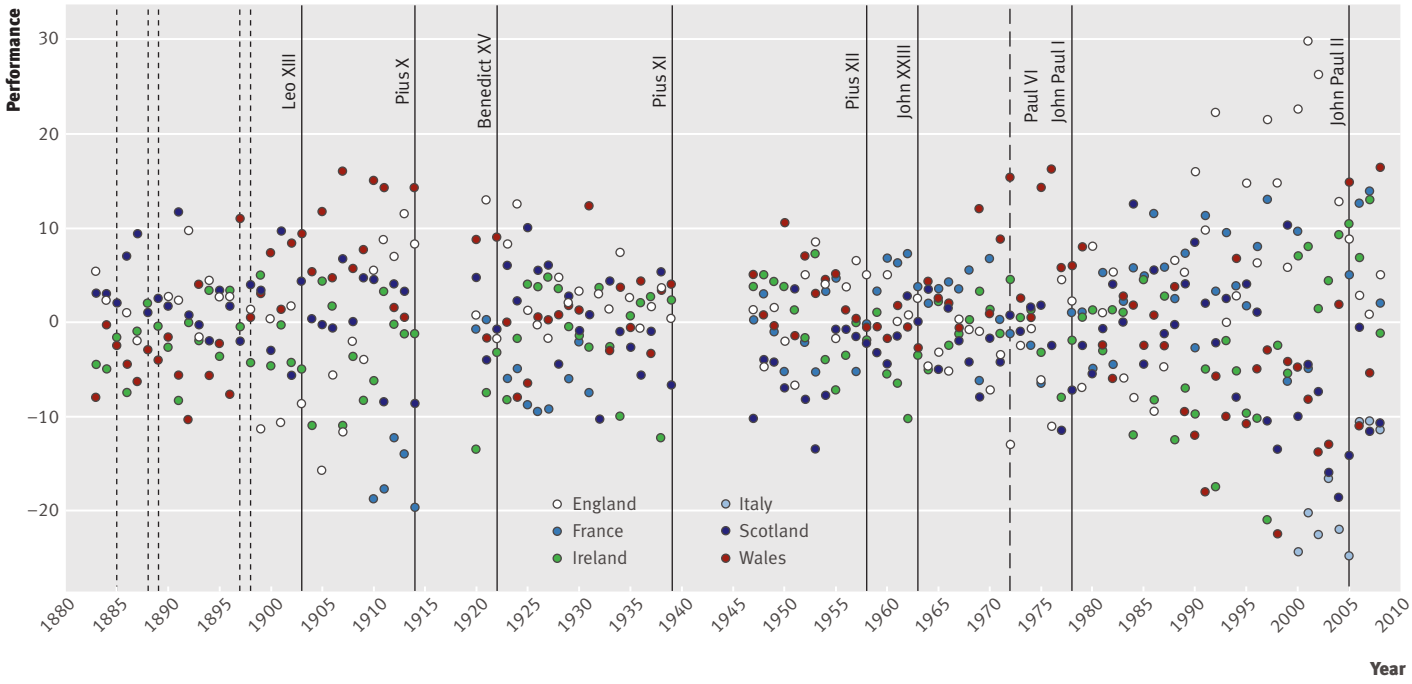

Fig 2 | Performance of the rugby nations, measured by average points difference per game, showing the years of papal deaths. Dashed lines represent years when the tournament was not completed

direction of the effect: when Wales win a grand slam, the chance of a papal death in that year increases. The general theory suggests a dose-response relation: when Wales perform particularly well, the expected number of papal deaths increases.

A grand slam is achieved when, in a given season, one nation succeeds in beating all other competing teams in every match. We discard results from the six years in which not all scheduled matches were played (1885, 1888-9, 1897-8, 1972).

To our knowledge, every pope from St Peter to Pius IX (who was pope from 1846 to 1878) died without a rugby union grand slam being contested or won. Although rugby union was invented in 1823, the year 1883 offered the first opportunity for a rugby grand slam, when England, Ireland, Scotland, and Wales completed their first annual international rugby union tournament. France entered the competition in 1910 (though did not compete during the years 1932-9). In 2000, Italy began to compete in the event, which is now known as the Six Nations Championship. Under our working definition, 53 grand slams have been achieved to date.

PHIL GILHAM GETTY IMAGES SPORT

Since 1883, eight pontiffs have died, five of whom did so in grand slam years: Leo XIII (1903) when Scotland won, Pius X (1914) when England won, and Paul VI (1978), John Paul I (1978), and John Paul II (2005) when Wales won. The deaths of Pius IX (1922) and Benedict XV (1939) coincided with Wales winning the tournament, though without achieving the grand slam. Each papal death in this period coincided with victory for a predominantly Protestant nation (England, Scotland, or Wales) rather than a predominantly Roman Catholic nation (France, Ireland, or Italy).

In all our investigations, we used the calendar year of completion of the northern hemisphere rugby union championship as our unit of analysis. Our sample size—that is, the number of completed competitions from 1883 to 2007—was therefore 107. Figure 1 shows the winning teams for each of these years, and indicates grand slams, papal deaths, and incomplete championships.

Results

To investigate the special theory of papal rugby, we used logistic regression to relate the years in which popes have died (since 1883) to home nation (England, Ireland, Scotland, and Wales) grand slams. The binary outcome of interest was whether at least one pope died in that year. There was, however, no evidence of a significant association between any individual home

WHAT IS ALREADY KNOWN ON THIS TOPIC

Rugby is followed religiously in Wales
Wales is a long way from Rome

WHAT THIS STUDY ADDS

Welsh grand slams coincide coincidentally with Papal deaths

nation grand slam victories and the years of papal deaths (P>0.1).

To investigate the general theory of papal rugby, we constructed a measure of a nation's rugby performance: the ratio of points difference (points against subtracted from points for) to the number of games played. In other words, we used the average (signed) difference in score per game as an indication of how well a team played in that year. Note that we did not adjust for changes to rugby scoring laws, nor for the beginning of the professional era in 1995. Figure 2 plots our measure of performance for all six nations against the year of competition and also indicates the years of papal death.

We then used Poisson regression to relate the number of popes dying in a given year (with possible values 0, 1, 2, ...) to all four home nations' levels of performance (simultaneously). We found a borderline significant (P=0.047) association between Welsh performance and the number of papal deaths but no significant associations between papal mortality and performance of any other home nation.

Discussion

The special theory of papal rugby is nothing more than an urban myth, based largely on two Welsh grand slam wins in recent memory. This comes as something of a relief, as we are at a loss to see how the events could be linked, especially given the continuing rapprochement between Catholic and Protestant churches.

Nevertheless, using the Six Nations data from 2008, our model for the general theory of papal rugby predicts that 0.62 (about 3/5) of a pope will die this year. It could be argued that Wales' strong win over Italy artificially inflates their measure of performance; however, based on the historical evidence, we do not believe the Vatican medical staff can fully relax until the new year arrives.

This project was based on a suggestion by Geraint Fuller. We are grateful to Christine Connolly of Six Nations Rugby Ltd for providing us with the historical data used in our analyses.

Contributors: GP and RPP cowrote the paper and collected the historical data. DF cowrote the paper and performed the statistical analysis. GP is guarantor.

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Is golf bad for your hearing?

M A Buchanan and colleagues investigate the possible hazards of modern drivers

A 55 year old right handed man presented to the ear, nose, and throat outpatient clinic with tinnitus and reduced hearing in his right ear. Clinical examination was unremarkable. His pure tone audiogram showed an asymmetrical sensorineural hearing loss, worse on the right, with a decrease on that side at 4-6 kHz (fig 1) typical of a noise induced hearing loss.[1] He had been playing golf with a King Cobra LD titanium club three times a week for 18 months and commented that the noise of the club hitting the ball was "like a gun going off." It had become so unpleasant that he had been forced to discard the club.

Magnetic resonance imaging of his internal acoustic meati showed no abnormality, and we deduced that his asymmetrical sensorineural hearing loss was attributable to the noise of the golf club. Other than regular golf, he had no history of prolonged occupational or recreational exposure to loud noises (such as shooting) or exposure to ototoxic substances to account for this noise induced loss.

Our internet search of reviews for the King Cobra LD club found some interesting comments:

"Drives my mates crazy with that distinctive loud 'BANG' sound. Have never heard another club that makes so distinctive a sound. It can be heard all over the course, it is mad!!"

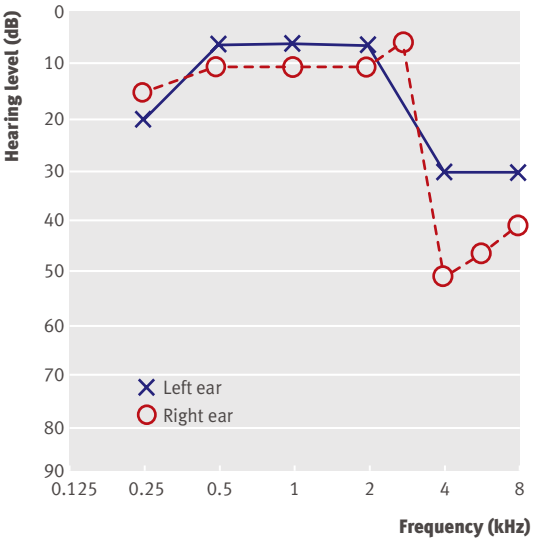

Fig 1 | Pure tone audiogram showing sensorineural hearing loss on the right, with a noise induced drop at 4-6 kHz

"A very forgiving club . . . albeit the 'unusual' clanking sound."

"I don't mind the loud BANG as it sounds like the ball goes a really long way. It sounds like an aluminium baseball bat, so some may not like it."

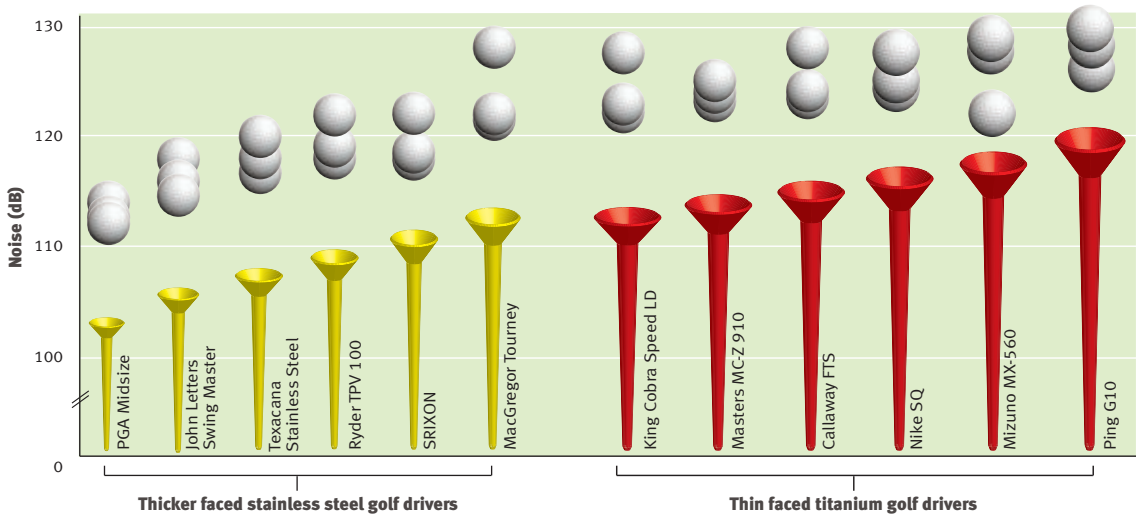

Fig 2 | Comparison of peak emitted sound levels (dB) between thicker faced stainless steel (yellow) and thin faced titanium (red) golf drivers when hit three times by a professional golfer

"This is not so much a ting but a sonic boom which resonates across the course!"

Diagnosis of noise damage

Guidelines exist to help diagnose noise induced hearing loss, setting out three requirements and four modifying factors that must be considered to formulate a firm diagnosis.[1] Our patient's audiogram met the requirements for a high frequency hearing impairment. His hearing was at least 10 dB worse at 4-6 kHz than at 1-2 kHz, and there was a downward notch of at least 20 dB in the 3-6 kHz range (fig 1). The remaining requirement—continuous noise exposure of 100 dB (or 90 dB for susceptible individuals)—does not apply in this case as we are dealing with impact (50 µs) noise. The modifying factors were also consistent with noise induced impairment.

He had no previous history of noise exposure, and the tinnitus described was a characteristic of noise exposure. In addition, calculation of Robinson-Sutton's equations[2] confirmed that in a man of 55 years, age induced hearing loss (presbyacusis) could not account for the loss at 4-6 kHz in his right ear, and that it must have been due to noise exposure.

Noisy clubs

The coefficient of restitution (COR) of a golf club is a measure of the elasticity or efficiency of energy transfer between a golf ball and club head. The United States Golf Association, in conjunction with the Royal and Ancient, St Andrews, Scotland, stipulates that the upper limit of COR for a golf club in competition use is 0.83.[3] This means that a club head striking a ball at 100 miles per hour (mph) will cause the ball to travel at 83 mph. Thinner faced titanium clubs, such as the King Cobra LD, have a greater COR and deform on impact more easily, the so called trampoline effect, not only propelling the ball further, but resulting in a louder noise. The King Cobra LD and Nike SQ both have CORs above 0.83, making them non-conforming for competitions.[3]

The experience of our patient prompted us to study the sound levels produced by different golf drivers. A professional golfer hit three two-piece golf balls with six thin faced titanium golf drivers and six standard thicker faced stainless steel golf drivers. We used a modular precision sound level meter (Brüel and Kjær) to record the levels of sound impulse (dB). The distance from the right ear of the golfer to the point of impact between the golf club and ball was 1.7 m. We therefore positioned the sound meter 1.7 m from the point of impact.

The thin faced titanium clubs all produced greater sound levels than the stainless steel clubs (fig 2). Interestingly, the club used by our patient (King Cobra LD) was not the loudest. Our results show that thin faced titanium drivers may produce sufficient sound to induce temporary, or even permanent, cochlear damage, in susceptible individuals. The study presents anecdotal evidence that caution should be exercised by golfers who play regularly with thin faced titanium drivers to avoid damage to their hearing.

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