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Edward Wilson: medical aspects of his life and career

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ABSTRACT. Edward Wilson's career plans were changed by a diagnosis of pulmonary tuberculosis. This diagnosis is reviewed as are the 'allergies', hypothermia, vitamin C deficiency and other medical conditions encountered by Wilson between 1901 and 1912. Edgar Evans' death on the return from the South Pole and a possible infective cause, is discussed.

Contents

Introduction

This paper considers the effect that illness may have played in determining Edward Wilson's career pathway and whether health matters could have determined outcomes in his fellow-explorers. Wilson suffered many of the conditions endured on early exploration: vitamin deficiency, hypothermia, altitude problems, malnutrition and dehydration. In addition he suffered two potentially fatal medical episodes: a respiratory disorder diagnosed as tuberculosis and an anaphylactic reaction. He also had an axillary abscess that was duly drained.

Wilson's tuberculosis

In the nineteenth century, tuberculosis was 'that perpetual spectre in the background, carrying off the young, the beautiful and the talented' (Keers 1978: vii). The disease is caused by a pathogenic mycobacterium, first identified by Robert Koch in 1882, which is transmitted from person to person. Then as now, overcrowded conditions and poor nutrition can increase the risk of developing the illness. In 1897, Wilson was living and working in the Caius Mission, Battersea, situated in a high-density population of poor housing. At the same time he was pursuing his medical studies at St George's Hospital, London. He ate frugally. At Christmas of that year he felt unwell with a fever and subsequently suffered from weight loss, a cough and chest pain. Pulmonary tuberculosis was diagnosed in March 1898.

At that time the diagnosis was made on the history of symptoms, on clinical examination, by microscopical identification of the mycobacterium in the spit and by culturing the bacterium where possible. X-rays were discovered in 1895 but, in the early films, soft tissues lacked clarity and the films were difficult to interpret. There is no record of Wilson having had a radiograph, although St George's purchased the equipment in 1898 (St George's Hospital Medical School 1898–1899).

When he consulted his supervisor at the hospital, the diagnosis that he was given was definite. His father wrote in 1898 that 'bacilli, [the mycobacterium] had been seen.' (Wilson 1955: 85). The presence of mycobacterium in spit does not, in itself, make a diagnosis of pulmonary tuberculosis. There are many types of mycobacterium, some are pathogenic, others are not; for example, non-pathogenic saprophytic mycobacteria, found in dust and water, were often present in scrapings from metal cold-water taps (Hynes 1961: 226). These could have been present as a contaminant. Mycobacterium tuberculosis is a pathogenic bacterium that typically causes human clinical disease. The important point in diagnostic differentiation is the cultural characteristics of the mycobacterium. In Wilson's case, there is no record of a positive culture of mycobacterium tuberculosis. This would have confirmed the clinical diagnosis.

Treatment was non-specific. Experience had shown that rest in sanatoria or spas, away from urban communities and at high altitudes could help. No attempt was made to separate patients from their non-infected companions. Patients and visitors ate the approved high-calorie meals seated in communal dining rooms, probably breathing mycobacteria onto each other (Mann 1999: 134). Prolonged periods of rest on verandas in the fresh (cold) air were prescribed and in the early 'cure', exercise and smoking were forbidden. Wilson went to Norway and then to Davos, Switzerland, where he wrote that he 'hardly dared clear his throat without feeling he had said something tuberculous' (Wilson 1955: 95). His health improved gradually and he returned to England after seven months.

Wilson accepted the diagnosis but the evidence is not conclusive. What is certain is that he had a debilitating febrile illness with weight loss, chest symptoms and subsequent scarring in the lung. This could have been due to a number of respiratory conditions other than tuberculosis. He could have had, for example, a subacute bacterial pneumonia. Such an infection could damage the bronchi and result in blood in the sputum. Certainly his recovery was impressive. He gained weight rapidly

and later in the Antarctic, he became a strong 'man-hauler'.

In parenthesis, it may be noted that tuberculosis remains a public health issue and although effective and proven anti-tuberculous drugs are available nowadays, the emergence of a drug-resistant mycobacterium is of serious concern.

When he was a student Wilson's ambition was to be a surgeon. With his 'label' of tuberculosis this was considered inadvisable. Undecided about his career, he felt the need of practical experience in a hospital. He became a junior doctor in Cheltenham in September 1900. Within a month he had developed a serious hand infection following a needlestick injury. This occurs when a needle accidentally perforates the skin of the hand, usually the palm. The infection spread rapidly to glands in the upper arm, which suppurated. In bed, he endured 'the worst week of illness and pain it has ever been granted to me to experience' (Wilson 1966: 23). Only regular morphine controlled the pain. The abscess in his armpit was drained under ether anaesthetic. Wilson had to resign from his post. This was his only hospital experience.

Wilson and the *Discovery* expedition

The possibility of obtaining the post of zoologist and junior surgeon on the 1901Antarctic *Discovery* expedition was brought to Wilson's attention and he applied. His arm infection had not completely healed when he met Robert Falcon Scott in November 1900 but Scott was positive about his appointment, provided he was fit. Pay was started in January 1901. Subsequently, the Admiralty Medical Board questioned his fitness to join the expedition on the basis of the past diagnosis of tuberculosis but Scott took him on his (Wilson's), responsibility. The appointment was confirmed in July 1901.

After the *Discovery* expedition in 1904, Wilson lectured to his colleagues at St George's (Wilson 1904). He had few complaints, though he reported that metal bolts driven through the hull of *Discovery* caused water to condense on the bunks, causing mattresses and bedclothes to rot. The clothes under the bunks were soaked or frozen into icy lumps. Wilson postulated that the persistent damp exacerbated his tendency to the muscular aches and pains that troubled him throughout the expedition.

He told his colleagues that when the wardroom carpet was beaten, as part of a general clean up in Antarctica, many of the crew suffered from severe cold-like symptoms plus general aches and pains. Symptoms appeared shortly after the cleaning and some men were so badly affected that one group, out on an expedition, had to return. Wilson thought that this was due to 'infection that had laid dormant for many months being liberated' (Wilson 1904) and he may have been right, as atopy and allergy were relatively uncommon in the early 1900's. Alternatively the symptoms could be due to a toxic reaction, now called 'organic dust toxic syndrome' caused by exposure to an organism 'thermophilic actinomycetes'. This causes influenza-like symptoms, shivering, cough and chest pain. The symptoms last for 24–72 hours and can be debilitating (C.A.E. Pickering, personal communication, March 2006). Otherwise there were few 'colds' on the expedition.

An early sortie to Cape Crozier in March 1902, proved how right Scott had been to worry about his men's inexperience in Antarctic conditions (Scott 1929: 170). When conditions became atrocious the leader, Lieutenant Charles Royds, split the party. Nine men were sent back to base under Lieutenant Michael Barne's inexperienced command.

Barne's group became caught in a blizzard close to the ship and he decided to try to make a break for *Discovery*. Steward Clarence Hare and Able Seaman George Vince were wearing fur finneskoes, rather than boots. None of the party had crampons. Vince shot down a slope and disappeared forever. Hare, a thin eighteen-year old, went back for his boots but also disappeared, it was feared, permanently. His arrival at the ship after forty-eight hours absence, with no signs of frostbite or any other problem was a huge relief. He had been unconscious for thirty-six hours. His last memory was of going towards a patch of rocks, which he hoped would provide some shelter, wearing his heavy woollen blouse and gabardine outer clothes over warm underclothes (Scott 1929: 186). Perhaps he survived the sub-zero temperatures because he managed to position himself in the lee of a rock, pulling his arms inside his blouse and covering the opening of his hood (Scott 1929: 186). He must have been covered with enough snow to give insulation and warmth but allowing sufficient air for breathing, a sort of primitive snow hole (a technique since developed for survival in extreme conditions). At base, Hare was found to be hypothermic. Wilson put him in the magnetic laboratory at an initial environmental temperature of 17°F, (−8°C), higher than the temperature recorded on the ship, which was 0°F (−18°C) and warmed him slowly to 34°F (+1°C) when he moved his patient to the sickbay (Wilson 1966:125). Hare had no long-term effects.

Generations of seafarers had believed that eating citrus fruits relieved scurvy symptoms, but by 1900, views had changed and it was widely held that ptomaine poisoning (a product of putrefaction of meat) was the prime cause of scurvy. This view was held by Dr Reginald Koettlitz, the senior surgeon on *Discovery* and by the senior medical establishment of the day. In March1900, Koettlitz wrote in the *Guy's Hospital Gazette* that 'scurvy would not be cured by seeking for substances that have ignorantly been called antiscorbutics' and that lime (or orange or lemon) juice had no useful effect against scurvy (Koettlitz 1900: 152). One of Wilson's duties was to smell and sample every newly opened tin of meat daily. 'Tainted' tins were discarded.

Early signs of scurvy are languor, loss of strength, depression and inertia, followed by swollen spongy gums, loosening of teeth, skin discolouration (like bruises which coalesce), sores over the body and widespread muscle

and bone aches. In severe cases there is blood loss from nose, mouth, bowels and bladder. Exertion can cause fainting and there can be sudden death (*Virtue's Household Physician* 1926: 530). Scurvy is now known to be due to vitamin C deficiency.

Early in the expedition on Dr Koettlitz's regimen, monthly examinations on the ship were satisfactory. Only one man had any suspicious signs. But in September 1902, Albert Armitage's group had to return from a sortie because of clinical scurvy (Wilson 1966: 192) and the ineffectiveness of the measures was clear; the tins were thrown out and fresh seal meat, mutton, skua or penguin was eaten at least six times per week. Lime juice was not insisted upon, but the doctors increased the jams and bottled fruits ration (Wilson 1904).

In spite of the improved diet, Scott, Wilson and Ernest Shackleton developed clinical scurvy on the southern journey of 1902–1903. On this expedition, fifty-two days after they had left base on 2 November, Wilson noted that Scott and Shackleton had developed spongy gums and small skin haemorrhages. He, himself, developed clinical signs later. Shackleton deteriorated badly. His cough, that had been present for days, worsened and he became very breathless. He coughed blood, and once, he was too ill to get out of the tent (Wilson 1966: 243). On 16 January, Scott wrote that Shackleton had paroxysms of coughing, was panting, dizzy, spitting blood, exhausted and excitable. Wilson feared that Shackleton would not survive. Double rations of seal meat were given. Since it was essential to keep going, Shackleton skied slowly beside the sledges, forbidden to haul or assist with camp work. All three had symptoms and signs of scurvy on their return to *Discovery* on 3 February and scurvy was the purported reason for Scott sending Shackleton home on *Morning.* Scott may have considered that the risk of a further collapse was too high

Fresh supplies, especially vegetables brought on *Morning*, helped their recovery (Scott 1929: 535). Notably Wilson's clinical scurvy does not appear to have affected his scarred lung. In scurvy, old scars can break down as normal collagen production is arrested (C. Bates, personal communication, December 2006). If the original diagnosis of tuberculosis were correct, scurvy might well have caused a recurrence, as pathogenic mycobacteria can survive in scarred lung tissue and may reactivate if the scar breaks down.

Wilson thought that further expeditions would not get clinical scurvy, because the *Discovery* years had shown the preventative value of the fresh food available in Antarctica. He did not specify, or know, that it is animal's offal (liver, kidney and lungs) that has the significant vitamin C content. Muscle has practically no vitamin C (J. Woods, personal communication, November 2006). He could not know how the longer southern expedition of 1910 would exhaust some of the men's supplies.

It is now accepted that, in humans, a pre-scorbutic phase exists before clinical scurvy develops. In an experiment on human scurvy conducted in 1940, reduced levels of vitamin C in platelets and white cells were recorded at three months, in the absence of clinical signs of scurvy, when a diet with no vitamin C was taken. At this time an experimental wound healed rapidly and normally. Skin dryness with hyperkeratosis developed in four months, (the earliest clinical sign), skin petechiae a month later. At six months a further experimental wound failed to heal. During this experiment it was noted that gum lesions did not occur in the volunteers whose mouths were previously healthy (Crandon and others 1940: 356–357).

To anticipate, it may be noted that in 1912, on the *Terra Nova* expedition, Lieutenant Edward Evans had clinical scurvy when he had been away from base camp for just over twelve weeks, (eighty-six days) (Smith 2000: 132). This suggests that he was in a pre-scorbutic state when he left base camp. By contrast Chief Petty Officer Edgar Evans had been away from the base for over fifteen weeks, (108 days), when he died on his way back from the Pole. If Evans had been in a pre-scorbutic phase when he left camp it is likely that some clinical signs of scurvy would have developed by this time. Since bleeding is a manifestation of established disease it is less likely that Evans' death, some days after minor trauma, was associated with an intracranial event related to a low vitamin C level.

Wilson after *Discovery*

After *Discovery*, Wilson living in Bushey, Hertfordshire, had a narrow escape from death. He was stung on the temple by a wasp. This caused an anaphylactic reaction, (a very serious allergic–type reaction). In minutes he suffered generalised itching, wheezing and coughing, could hardly breathe, had pains in his chest and a racing pulse. He felt faint and became confused (probably related to low blood pressure and lowered oxygen levels). The life saving treatment was, and is, subcutaneous injection of adrenaline. By an astonishing coincidence, the attending physician was a Dr. Shackleton, a cousin of Ernest Shackleton, who practised in Bushey. Wilson slowly improved but his chest rattled and wheezed for some hours. White wheals on his bright red skin took days to resolve (Wilson 1995. 150). He had no other episodes.

The final expedition

During the 1911–1912 pole expedition Scott, Wilson, Henry Bowers, Lawrence Oates and Edgar Evans were on the plateau, at an altitude of 9,500–10,500 feet, (2,900–3,200 meters) for seven weeks. The temperature was regularly around −20°F (−29°C). The extreme cold of Antarctica reduces the barometric pressure and so exaggerates the effects of altitude, which means that the men were experiencing the same physiological effects that are found at higher altitudes on equatorial mountains (West 2001: 559). They would have had to hyperventilate to maintain an adequate oxygen level in the 'thinner' dry air with resultant dehydration. Their summit rations, consisting of butter, pemmican, biscuit, cocoa, sugar and tea (Cherry-Garrard 1994: 572), provided just over 4,500

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calories per day. After Evans' death his companions would initially have had an extra 1100 calories each, still insufficient for hauling at sub-zero temperature. A negative calorie balance results in loss of muscle as well as insulating body fat. As the central (core) temperature drops, so the ability to coordinate is reduced. Wilson must have realised this as he and his colleagues fumbled slowly with accustomed tasks. He understood that the effects of the cold were becoming more acute.

On 29 January 1912, Wilson developed a swelling of his tibialis anterior muscle (his shin), after twenty-two miles of difficult skiing. The front of the leg was swollen and tight, the skin red and oedematous (Wilson 1972: 238). Wilson's description is considered to be the first record of a syndrome that can follow strenuous activity or injury and which is now recognised in athletes (Freedman 1953: 183; Mubarak 1981). Ischaemia and pain are due to an increased pressure within the rigid bony-fibrous anterior compartment that surrounds the muscle. In addition to his exertions, Wilson may have caught his ski on a rut, bruising his left leg and causing a small bleed into the muscle (Fiennes 2003: 324), although he does not mention this in his diary.

Evans died on 17 February 1912. The exact cause of death cannot be known but is likely to have been by compounding medical problems: malnutrition, dehydration, and infection. He cut his knuckle, (probably on the non-dominant hand) on 31 December 1911, whilst reducing the length of the sledges. This injury 'had a lot of pus in it' by 7 January 1912 (Wilson 1972: 230). At the South Pole, ten days later, his nose and cheeks were severely frostbitten, (as were those of Oates and Bowers), but Evans' hands were so troublesome that an early camp stop was made (Wilson 1972: 232). By 28 January, as they pulled north in the biting wind and sub-zero temperatures, Evans had five 'badly blistered fingers tips from frostbite, which he got at the Pole' (Wilson 1972: 238). At the end of the month the nails were falling off leaving the fingers raw.

Wilson dressed Evans' fingers every other day, sterilising snow and making an antiseptic lotion with a spirit lamp, which he had taken from a broken hypsometer that he had found in the Norwegian tent near the pole (Wilson 1972: 232). Evans' fingers were 'still quite sweet' on 4 February but by the following day they were suppurating and his nose was 'bad and rotten-looking' (Wilson 1972: 240). His nose became almost as bad as his fingers (Scott 1935: 438). As they marched on 9 February, the ambient, damp temperature rose to plus 10°F (–12°C).

On 16 February, Evans collapsed with sickness and giddiness. He could not walk and camp was made early. Scott wrote that Evans had stopped the march on a trivial excuse (Scott 1935: 446). The day after, Evans had difficulty keeping his ski shoes on, possibly he could not tie his laces with his infected hands, and possibly he was in an unrecognised confused state. Whatever the reason he could not pull effectively. He was ordered to unhitch and trailed well behind his companions. When they went back for him he was dishevelled, on his knees, unable to stand. He was comatose by the time he was carried to the tent. He died at 10 pm.

His companions did not show much understanding of Evans' deterioration. In discussion with his religious minded mother, Wilson had accepted the concept of 'the healing power of sickness', taking the view that the lack of previous suffering made a patient less able to withstand illness. As late as 16 February, he wrote that Evans' collapse had 'much to do with the fact that he had never been sick in his life' (Wilson 1972: 243) implying, with no evidence, a psychosomatic component to Evans' decline. These uncharacteristically unsympathetic comments were made at a time when he himself was greatly debilitated. The fact that Evans struggled to cooperate, even up to the day he died makes depression and withdrawal unlikely.

Infection could have been a major factor in causing Evans' death. Several possibilities are considered. *Staphylococcus aureus* is a likely cause of Evans' hand infection. Abscess formation occurs typically seven days after a wound infection, as happened here. *Staphylococcus aureus* is a bacterium carried in the nose and skin. It is not eliminated by the low temperatures of the Antarctic, and it has been isolated from sleeping bags exposed to those temperatures (Hadley 1981: 231). By February 1912, Evans' cuts and fingers were suppurating, and in addition Wilson noted that Evans' nose was very bad and rotten looking. A possible sequence is: nasal carriage resulting in wound infection and followed by a bacteraemia, probably repeated. There need have been no signs in the arm; the bacteraemia could have silently gained ascendancy. The final clinical picture can be interpreted as collapse due to hypotension, dehydration and accelerating infection. A streptococcal infection is less likely, these pathogens do not stay locally for long and the septicaemia kills quickly (H. Pennington, personal communication, June 2006).

Alternatively, pathogenic staphylococci could also have gained direct entry to Evans' cranium from his 'rotten-looking' nose, a description suggesting infection. It may be noted that Oates and Bowers had frostbitten noses and cheeks but there was no suggestion that they were rotten. The relatively high ambient temperature of 9 February and subsequently, could have promoted bacterial growth in the decaying tissue under its frost bitten surface skin. Evans' obvious deterioration on 16 February, seven days after this higher temperature, would fit the pattern of a frontal lobe abscess secondary to sinus infections. Although others have discounted the likelihood of a brain abscess, it remains a possibility.

A cavernous sinus thrombosis, (a clot in the venous blood draining the face or sinuses and invariably fatal in pre-antibiotic days) should be considered. This condition is associated with infection of the nose and lips and could have resulted from progression of infection from the thrombosed nose into the brain (Bayley and Love 1964: 463). Dehydration would increase this risk.

The British Antarctic Survey medical unit found bacillus species, anaerobes and enterococci, in a human faecal

sample passed over thirty years previously and exposed to temperatures of 0°F (−18°C) to −90°F (−58°C) (P. Marquis, personal communication, March 2005). Faecal contamination of Evans' fingers is theoretically possible. *Escherichia coli* is a common wound infection and present in faeces. A gram-negative septicaemia would have a relatively slow clinical course but progress to final dramatic collapse.

Conclusions

In summary, a significant respiratory illness first delayed Wilson's qualification in medicine, then forced him to revise his ambition to be a surgeon. Serendipity gave him the opportunity to explore Antarctica as a natural scientist, artist and doctor. In Antarctica, imperfect understanding of dehydration, nutrition, vitamin deficiencies and infection contributed to his and his colleague's deaths. His medical contributions related importantly to his steady, loving and supportive nature, his common sense and his calm. He believed that his life was in God's hands and it was God's intention for him to go with Scott (Seaver 1946: 75). He was one of the most successful of Scott's appointees.

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