

# ‘Polar anaemia’: cardiac failure during the heroic age of Antarctic exploration.

H.R. Guly

Derriford Hospital, Plymouth, PL6 8DH (hguly@aol.com)

Received February 2011; First published online 8 July 2011

**ABSTRACT.** On the *Belgica* expedition (1897–1899), Dr F.A. Cook described a disease that he called ‘polar anaemia’ and on this expedition it affected most of the expedition members and caused one death. The symptoms were shortness of breath, abnormalities of the pulse and oedema (swelling of the legs) and the disease was clearly cardiac failure. During the heroic age of Antarctic exploration a similar disease affected at least eight other expeditions causing five other deaths. This disease was very similar (and probably identical) to a disease affecting (mostly) Scandinavian seamen and called ‘ship beri-beri’. Both diseases were almost certainly what is now called wet beri-beri due to thiamine (vitamin B1) deficiency though most sufferers were probably also vitamin C deficient and some may have had both beri-beri and scurvy. It may have been exacerbated by invalid diets. This paper describes the disease and how it was considered and treated at the time.

## Introduction

Almost every book written about the heroic age of Antarctic exploration mentions scurvy and others have also written about the scurvy that occurred on some of these expeditions (Kendall 1955; Evered and Goldsmith 1985; Carpenter 1988: 150–157) but if one discounts the arguments concerning the cause of death of R.F. Scott and his companions in 1912, there was only one definite death from scurvy during the heroic age – that of A. Spencer-Smith of Shackleton’s Ross Sea party in 1916. However there were six deaths from heart failure (excluding the death of Shackleton who died of coronary heart disease). In addition there were at least eight others who had a similar disease but who did not die.

The aim of this paper is to describe this disease including the symptoms and signs, the way it was considered at the time, and the way it was treated and to suggest a cause for it.

## Methods

This study was undertaken as part of a larger study of medicine concerning the heroic age of Antarctic exploration and the author believes that he has read most of the relevant literature in English and much of that in French.

## Symptoms and signs

The symptoms of ‘polar anaemia’ were those of cardiac failure with shortness of breath, abnormalities of the pulse, and oedema (swelling of the legs). The deaths and non-fatal illnesses from this disease are listed in Table 1.

On the Belgian expedition, E. Danco died and most of the crew was affected to a greater or lesser extent. Dr F.A. Cook wrote that:

‘Physically we are steadily losing strength though our weight remains nearly the same, with a slight increase in some. All seem puffy about the eyes and ankles and the muscles, which were hard earlier are now soft, though not reduced in size. We are pale, and the skin is unusually

oily. The hair grows rapidly, and the skin about the nails has a tendency to creep over them, seemingly to protect them from the cold. The heart action is failing in force and is decidedly irregular. Indeed this organ responds to the slightest stimulation in an alarming manner. If we walk hurriedly around the ship the pulse rises to 110 beats, and if we continue for fifteen minutes it intermits, and there is also some difficulty in respiration. The observers going only one hundred yards to the observatories come in almost breathless after their short run. The usual pulse, too, is extremely changeable from day to day. Now it is full, regular and vigorous; again it is soft, intermittent and feeble. In one case it was, yesterday, 43, today it is 98 but the man complains of nothing’ (Cook 1980: 291).

Elsewhere he writes: ‘The heart is unsteady, easily disturbed, and mitral murmurs, which I have not heard before, are audible...the breathing is often difficult, the blood retreats from the skin, but the larger veins are abnormally full’ (Cook 1980: 306–307). In addition there were other symptoms including indigestion and gastrointestinal symptoms. The situation was also complicated by psychiatric illness.

A similar illness occurred in both of J.-B.E.A. Charcot’s expeditions but without death. On the *Français* expedition (1903–1905), Charcot writes of A. Matha: ‘the oedema in his legs was serious, and examination of his heart and circulation left me in no doubt that the diagnosis had to be inflammation of the cardiac muscles’ (Charcot 2004: 119). He did not improve with the introduction of fresh meat. The most detailed description of this illness is given by E. Gourdon who wrote a doctoral thesis on medical aspects of the expedition (Gourdon 1913). Gourdon also says that Charcot, had similar problems (Gourdon 1913: 82) but Charcot himself does not mention that he was ill.

On the *Pourquoi Pas?* expedition (1908–1910), there were similar problems in R. Godfroy and Charcot himself. Charcot diagnosed himself as having ‘pronounced

Table 1. Cases of 'polar anaemia' during the heroic age of polar exploration.

Expedition	Name	Onset	Death	Heart disease	Haemorrhages	Other symptoms
<i>Belgica</i>	Danco	March	June	*		
	Several others	June/July		*		Psychiatric symptoms
<i>Southern Cross</i>	Hanson	July	October	*		Loss of sensation legs, vomiting
Swedish ( <i>Antarctic</i> )	Wennesgaard	March	June	*		Cough, muscular rheumatism
Scott ( <i>Discovery</i> )	Crean	June		*		
1st German ( <i>Gauss</i> )	Enzensperger	October	February	*		Cough
	Werth	August				Chest and arm pain, leg stiffness
<i>Scotia</i>	Ramsay	March	June	*		Chest pain
1st French ( <i>Français</i> )	Matha	July		*		Loss of sensation in legs
	Charcot	September				Chest pains, pin & needles, cramps in limbs
2nd French ( <i>Pourquoi Pas?</i> )	Godfroy	June		*	September	
	Charcot	June		*	September	Chest pains
	Gourdon	October		*		
2nd German ( <i>Deutschland</i> )	Vahsel	June	August	*		Albumin in urine

myocarditis' (Charcot 1978: 199). However three months later, both had developed some evidence of scurvy in the form of purpuric spots. Gourdon later developed some oedema and Charcot noted how this disease had only affected the officers and not the crew although later Chollet fell 'a victim in his turn, but to a much more ordinary form of scurvy. He has great black spots on his thighs and can no longer keep on his feet' (Charcot 1978: 247). Godfroy's illness initially responded to a diet of seal meat but he later relapsed and both he and Charcot were still having problems in January 1910, seven months after their symptoms started.

On the *Southern Cross* expedition (1898–1900), L.C. Bernacchi described N. Hanson's illness: '[he] has suffered severely from headaches and has nearly lost the use of his legs. The limbs are swollen and almost dead to sensations. Pins can be stuck there without inflicting pain. He is scarcely able to stir out of the house...' (Crawford 1998: 135). C. Borchgrevink, the expedition leader, described the same symptoms: 'Mr. Hanson was in rather a low condition; he lost feeling in his legs and the flesh on them took lasting impressions from pressure' (Borchgrevinck 1980: 139) This is clearly a description of oedema. He also suggests that others had problems as well. Confirmation that Hanson was oedematous is shown by his weight: between the beginning of August and the beginning of October, Hanson's weight increased 15 lb whereas the mean weight increase of the rest of the party was 4.3 lb. (Borchgrevinck 1980: 317)

A. Ramsay's illness on the *Scotia* expedition (1902–1904), led by W.S. Bruce, is not well described. He was thought to have been malingering until Dr. Pirie examined him and, to his surprise, made a diagnosis of mitral stenosis, narrowing of the mitral valve of the heart

(Bruce 1992). His main symptom would appear to have been shortness of breath.

On the Swedish expedition (1901–1903) O.C. Wennesgaard's illness is even less well described but Dr. E. Ekelöf says that he 'was attacked by acute articular rheumatism ... [in] August 1902. In March 1903 ... he had a fresh attack. In addition to the other symptoms there also appeared insufficiency of the heart, and after severe sufferings, lasting about six weeks, Wennesgaard at last died on the 7th of June 1903' (Ekelöf 1904).

A similar illness occurred on R.F. Scott's *Discovery* expedition (1901–1904). R. Skelton records that 'Crean ... is on the sick list with I believe "dropsical" symptoms. ... I believe his weight has gone up to 209 lbs, almost 20 lb in a month' (Skelton 2004). This presumably is the person of whom Dr. E.A. Wilson wrote a month later: 'One of the seamen about whom we have had some anxiety, owing to the swelling of his legs in a way which suggests scurvy and reminded one unpleasantly of Hanson's illness on the *Southern Cross* Expedition, has taken a decided turn for the better' (Wilson 1966: 159).

We have some objective data on T. Crean from the routine measurements that Koettlitz made of the crew of the *Discovery* (Koettlitz 1904). Crean's weight and calf circumference are shown in Fig 1. Unfortunately there is no weight recorded in April 1902 but his weight increased from 191 lb in May to 209 lb in June and July. Later weight measurements when he was well might indicate that his normal weight was 180–185 lb and so his weight in May might have been greater than normal. This was associated with an increase in calf circumference, presumably due to oedema. His spirometry (lung capacity) measurements show no obvious pattern of getting worse and then better again.

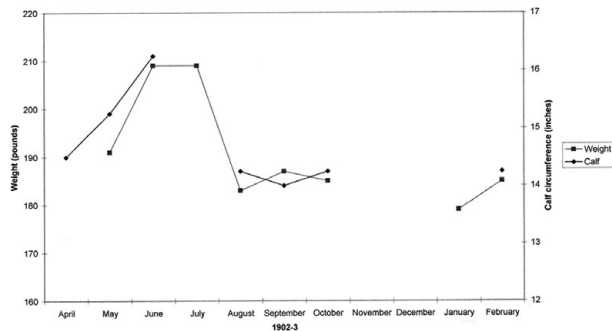


Fig. 1. Weight and calf circumference of Thomas Crean.

As has been noted, two members of the second French expedition developed bleeding. Charcot included bleeding as part of his description of ‘la maladie des conserves’ but said that ulceration of the gums was very rare (Charcot 1931b: 750).

#### How was this illness considered at the time?

On the *Belgica* expedition (1897–1899), Cook called it ‘polar anaemia’ (Cook 1980: 303) and E. Gourdon (geologist on both the French expeditions) described it on one occasion, similarly, as ‘l’anémie de la longue nuit’ (anaemia of the long night) (Gourdon 1936). However polar anaemia is a misnomer as there is no record that Cook measured the haemoglobin level and thus no evidence of anaemia. In fact, all who measured the haemoglobin level in the expeditions of the time found that it often increased (Ekelöf 1904; Wilson 1905; McLean 1919).

R.E.G. Amundsen refers to the illness on the *Belgica* as scurvy (Amundsen 1913) though Cook never used that word. De Gerlache, the expedition leader, also notes that: ‘At one point the doctor believed it his duty to tell me that he had detected evidence in my condition that was symptomatic of scurvy’ (de Gerlache de Gomery 1998). Cook probably believed that the disease was scurvy but wanted to avoid causing panic.

E.A. Wilson thought that the polar anaemia on the *Belgica* ‘was probably incipient scurvy’ (Wilson 1966: 121). Even when it was differentiated from scurvy, it was considered to be related in some way. Dr. Alexander Macklin, a surgeon on Shackleton’s second and third expeditions, describing polar medical problems, refers to ‘scurvy (and allied conditions)’ (Macklin 1923).

On the *Southern Cross* expedition (1898–1900), Bernacchi said that Hanson appeared to have the symptoms of scurvy (Bernacchi 1901) and although the cause of death was said to be ‘an occlusion of the intestines’, (Crawford 1998: 156) this would not cause oedema.

There was also significant psychiatric disease on the Belgian expedition. It is clear that Cook considered this as part of polar anaemia which he blamed on the tinned food and the environment: ‘the long darkness, the isolation, the tinned foods, the continuing low temperature, with increasing storms and a high humidity, finally reduced our systems to what we call “polar anaemia”...

Most dangerous of all were the cardiac and cerebral symptoms. The heart[‘s] . . . action was weak, irregular, and entirely unreliable through the night. . . The men were incapable of concentration, and unable to continue prolonged thought. One sailor was forced to the verge of insanity, but he recovered with the returning sun’ (Cook 1980: 303). Elsewhere he said that ‘the sun seems to supply an indescribable something which controls and steadies the heart. In its absence it is like an engine without a governor’ (Cook 1980: 291).

Charcot’s views changed over time. He recognised cardiac disease by describing his illness as ‘polar myocarditis’ (Charcot 1978: 199–200) but was uncertain as to the underlying cause. Initially, like Cook, he blamed the environment: ‘this condition, which is sometime incorrectly called polar anaemia, could be attributed to a hyperactive circulation which the nerve centres cannot naturally be familiar with, and which I think is probably caused by changes in the nature of the atmosphere in these regions in winter. It is certain that the symptoms have never been noted in summer and it is difficult . . . to blame the winter darkness. This is equally long in Iceland and even longer in the north of Norway, where such things have never been observed’ (Charcot 2004: 136).

In his second expedition, his confusion can be seen by the comments in his book. On 21 June 1909 he wrote: ‘The so-called “polar anaemia” – or perhaps it is scurvy, which is just as much to be feared – has made its appearance on board’ (Charcot 1978: 199) but six days later he said: ‘. . . I abandon the idea of scurvy to fall back upon polar myocarditis, the origin of which is as yet unexplained. We have nothing wrong with our gums, none of the classical symptoms of scurvy. . .’ (Charcot 1978: 200). The traditional remedies for scurvy did not work. However by September he and Godfrey had developed some purpura and so he reconsidered: ‘After thinking it out, I have come to the conclusion that we are suffering from scurvy, or more precisely, from preserved-food sickness’ (Charcot 1978: 214). He later reasoned: ‘All the so-called polar anaemias turn out to be nothing but maladies allied to scurvy. In the past when crews lived almost entirely on salt meat they were attacked by the well-known variety of scurvy, with large black spots, ulceration of the gums, etc. But everything changes, even diseases, and with the modern preserved food the classical scurvy has been replaced by the curious kind from which we suffered, characterised especially by oedema of the lower limbs and myocarditis without anything wrong with the gums’ (Charcot 1978: 216).

He also called it ‘scorbut moderne’ [modern scurvy] but also attributed an underlying cause to the disease with the name ‘la maladie des conserves’ [disease of preserved food] (Charcot 1931a, 1931b) although on one occasion used the words ‘so-called beri-beri’ (Charcot 1931a, 1931b). Gourdon called it ‘le mal des conserves’ (Gourdon 1913: 77).

He was convinced that this disease was caused by a toxic substance in the tinned food. He described his

Table 2. Scurvy during the heroic age of Antarctic exploration

Expedition	Names	Onset	Notes
Scott ( <i>Discovery</i> )	Heald, Ferrar, Cross	September	Started after sledging
2nd French ( <i>Pourquoi Pas?</i> )	Scott, Wilson, Shackleton	January	Started while sledging
	Charcot, Godfroy	September	Started three months after developed beri-beri
	Chollet	November	No preceding illness. Did not eat seal meat
Scott ( <i>Terra Nova</i> )	Lt Edward Evans	January	Started while sledging
Ross Sea party	Spencer-Smith, Mackintosh, Hayward	January	Started while sledging. Spencer Smith died in March

reasoning: ‘Gourdon and Godfroy came to tell me . . . that they had oedema of the legs. . . . I put them, therefore, on an exclusive diet of seal’s meat. Surely enough, at the end of three days, every symptom has disappeared . . . What proves completely that it is provoked by the preserved-foods and not by the absence of fresh meat is that the symptoms disappear, not by adding fresh meat to the diet, but by the total suppression of preserved-food’ (Charcot 1978: 235).

He continued with this belief for another 20 years, long after the discovery of vitamins. In 1931 he again described ‘maladie des conserves’ or ‘scorbut moderne’ in two papers and wrote that ‘it will serve no benefit to take in large quantities of fresh meat, vegetables, fruits etc – let us say the word – and vitamins if one continues to consume preserved foods, even in small quantities . . .’ (Charcot 1931a, 1931b). He quotes his Antarctic experience and some deaths but some of the cases he described were people with a mild degree of oedema only, and these were clearly not due to vitamin deficiency. For example he wrote: ‘In 1923, during a crossing from Cherbourg to Gibraltar which took eleven days on the *Pourquoi Pas?* where we only ate preserved meats with an abundance of potatoes, vegetables and fruits, I started to have pretibial oedema which disappeared immediately on abandoning preserved foods’ (Charcot 1931b). The author cannot fully explain his observation but eleven days is too short for vitamin deficiency to have occurred.

#### The first German expedition (1901–1903)

On the first German expedition, led by E.D. von Drygalski, a party of five was left on Kerguelen while the main party went to the Antarctic continent in *Gauss*. Some of the party and some of the supplies were taken to the island by the steamer *Tanglin* in November 1901 before *Gauss* arrived at the end of December. Many of the Chinese crew of the *Tanglin* were suffering from beri-beri and two died and were buried on Kerguelen. When E. Werth and then J. Enzensperger became ill on the island, they diagnosed themselves with the same illness and Werth’s illness was confirmed as beri-beri when he eventually reached Australia in April 1903. At the time opinions were divided as to whether beri-beri was caused by a deficiency of some kind or by an infection (Carter 1977; Carpenter 2000) and Drygalski believed that they

had been infected by the Chinese and that the infection had been transmitted via either rice or via the building materials of their hut (Drygalski 1989). The medical report of the expedition was not published for another 24 years, by which time the cause of beri-beri was well-recognised and H. Gazert (surgeon to the expedition) described the illnesses and said that they were due to ‘the hydropic form of beri-beri’ (Gazert and Renner 1927).

#### What was this illness?

The information in Table 1 was not compiled as a result of formal medical reports and so the fact that an abnormality was not recorded does not indicate that it was not present. All the illnesses seem very similar and all the deaths seem to have been cardiac deaths; all were conscious to the end and several had neurological symptoms. All illnesses started in the austral autumn or winter and were not related to sledging. They are very different from the proved cases of scurvy which are shown in Table 2 (this excludes the deaths of Scott and his companions, the cause of whose death is still much debated). Charcot and Godfroy did develop scurvy symptoms but these started three months after their cardiac symptoms. Most of the other cases of scurvy were related to heavy exertion and sledging.

Almost certainly these diseases were due to wet beri-beri: the symptoms correspond to the disease. Beri-beri was known to affect other ships at the time and the food they brought with them was vitamin deficient. This conclusion is not a new idea: as noted above, the illnesses on the first German expedition were diagnosed as beri-beri and Charcot said that many cases of polar anaemia were misdiagnosed as either scurvy or beri-beri (Charcot 1931a). Hanson’s death has also previously been described as due to beri-beri (Crawford 1998: 135).

Beri-beri is caused by thiamine (vitamin B1) deficiency. Thiamine is produced by plants and fungi and the best sources are in cereal grains and yeast. In grains, the thiamine is concentrated in the husk and germ which are removed during milling and so concentrations of thiamine are significantly lower in refined grains. Hence the greater risk of beri-beri in the officer class who tended to eat more refined foods (whereas scurvy tended to be more common on the lower deck). Beri-beri takes two main forms, dry and wet. In dry beri-beri the symptoms

are primarily those of nerve damage, particularly in the lower limbs causing heaviness and stiffness, progressing to weakness, pins and needles, numbness and muscle wasting although this may be masked by some oedema. Wet beri-beri causes heart failure as a result of a cardiomyopathy (the myocarditis that Charcot describes). The signs of heart failure include oedema, a rapid pulse and shortness of breath. In the acute fulminant form, the heart may be enlarged, cardiac murmurs and death may occur and death usually occurs with the patient fully conscious (Brabin and Coulter 2003). It is not clear why some patients get the wet, and others the dry form of the disease but it is thought that wet beri-beri is caused by an acute vitamin deficiency whereas dry beri-beri is caused by longstanding deficiency of a lesser degree (Truswell 2007). However there is much individual variation.

Other possibilities must be considered for some of the deaths. Bruce says that 'Outside accidents, certain forms of heart disease have been about the only cause of death and in these cases the trouble was probably present in its initial stages before the person joined the expedition. . . .' (Bruce 1911). Danco and Ramsay were both said to have had previously undiagnosed cardiac conditions. This is possible as rheumatic heart disease was much more common 100 years ago than now, but is not necessarily correct. Most expeditions would have organised medical examinations for participants as part of the selection process and cardiac murmurs should have been picked up then. A doctor finding a murmur in a patient whose heart he has not previously listened to may assume that it is of long-standing. Ramsay was said to have the murmur of mitral stenosis. This is a difficult murmur to hear, but, if present, this would not be caused by beri-beri. Psychologically, a death is easier to accept (for doctor, expedition members and patient's family) if it can be thought of as, somehow, inevitable. However pre-existing cardiac disease may have been a factor in some of these deaths.

The cause of Richard Vahsel's death on the second German expedition (1911–1913) was said, in one place to be 'heart dropsy and kidney failure' (Filchner 1994: 174) and, in another, 'pericardial dropsy' (Filchner 1994: 210). Dropsy is a term that used to be used to describe severe oedema: constrictive pericarditis produces symptoms that, without specialised investigation, may be difficult to differentiate from heart failure. Vahsel is widely believed to have had syphilis (Filchner 1994: 207; Murphy 2002) and this can cause a variety of heart diseases. However he was also a heavy drinker and this, too, is associated with heart failure. The cause of this, in most cases, is uncertain but beri-beri can cause heart disease in alcoholics due to thiamine deficiency as a result of most calories being taken in the form of alcohol (McIntyre and Stanley 1971). Kidney disease may cause a similar picture of oedema but this is associated with albumin in the urine. Charcot says of Matha's illness on the first French expedition that his 'symptoms resembled

those of Bright's disease [glomerulonephritis – a form of kidney disease] without albuminuria' (Charcot 1931b).

As noted above, Hanson's cause of death was given as occlusion of the intestines. This might explain his vomiting and the fact that he was treated with a stomach pump (Crawford 1998: 153) but this would not have caused his other symptoms. If this was a true diagnosis, then it was superimposed on beri-beri.

Both Hanson and Ramsay were said to have been ill on the ship before reaching Antarctica. There is little detail about Ramsay but Hanson had a diarrhoeal illness. There is some evidence that dysentery, itself, may predispose to beri-beri (Carpenter 2000: 197) but the diet used to treat it would have been devoid of vitamins and probably exacerbated the problem. A report on vitamins in 1924 said: 'A large proportion of medical casualties on foreign service consists of intestinal diseases, and both during the acute stage and the subsequent convalescence the diet is largely restricted to bread, condensed or tinned milk, and invalid foods, which may be regarded as either free from vitamin B or very poor in it' (Committee of Lister Institute and Medical Research Council 1924: 122).

### Ship beri-beri

Charcot claims that although 'scorbut moderne' had particularly been observed in polar and sub-polar regions, there was no reason for it not to manifest itself at all latitudes (Charcot 1931a). A similar (probably identical) disease did exist but with a different name.

In the nineteenth century, what was known as beri-beri was the disease seen mostly in Asia among rice eating populations. Thiamine in rice is found mainly in the husk and germ which is removed when rice is polished. Its history has been well described by Carpenter (Carpenter 2000). It was primarily a neurological disease with loss of sensation and weakness of the lower limbs though many also had oedema and cardiac problems and there was a high death rate. Braddon, reviewing the subject in 1907 said that 'dropsy is rare and never occurs in absence of nerve involvement' (Braddon 1907: 453).

However in 1894, crews on Norwegian ships began to suffer from a disease very similar to beri-beri. As on Charcot's expedition, it was more common in ships' officers. These seamen had the oedema of beri-beri with non-specific neurological symptoms but mostly without the major neurological features of beri-beri. This had some of the clinical features of scurvy and also responded to a change of diet as quickly as did scurvy whereas dry beri-beri in the far east did not respond. This disease became known as 'ship beri-beri'. Conditions on the polar exploring ships are likely to have been similar to those on other merchant ships. Ship beri-beri is thought to have resulted when Scandinavian ships changed from serving rye bread made with yeast to bread made from white flour and baking powder (Carpenter 2000: 134).

Braddon says that in both true beri-beri and ship beri-beri both dropsy and neuritis occurred but that in true beri-beri the neuritis predominated whereas in ship beri-beri the dropsy predominated (Braddon 1907: 453).

Descriptions of the disease are very variable. Holst compared ship beri-beri to 'true' beri-beri, saying that 'The symptoms of this disease consist, in the great majority of cases, in weakness and a prominent dropsy of the lower limbs, extending often to other parts of the body. There also exists shortness of breath and other symptoms of a weak heart, causing often sudden deaths from acute paralysis of the heart. But . . . symptoms of neuritis of the limbs are comparatively rare' (Holst 1907). He describes two series of cases. In one series, typical neurological signs of beri-beri were only found in men from four of 34 ships and in the other, they were only found in four of 57 ships. However scurvy also occurred with typical scurvy signs (sore gums and haemorrhages in the skin and muscles) being found in 12 of 34 ships in the first series though in none in the second series.

Hess, however, emphasised the closeness of ship beri-beri to scurvy, saying that ship beri-beri was characterised by spongy, hemorrhagic gums and other signs encountered in scurvy but that what differentiated it from scurvy, was anaesthesia of the extremities (Hess 1920).

#### Why was this considered as scurvy?

In the 18th and 19th centuries in western Europe, scurvy was largely a disease of seafarers. They were deficient in vitamin C but would also have been deficient in other vitamins. The clinical features of pure vitamin C deficiency were not fully established until experiments were made in the 1940s with diets deficient in vitamin C alone (Vitamin C Subcommittee of the Accessory Foods Committee 1953) and it is interesting to compare the symptoms of scurvy as it is understood now, with those described in Osler's textbook (perhaps the standard textbook of medicine of the era) of 1907. In addition to the gum disease and haemorrhages which are now known to be caused by vitamin C deficiency, Osler also describes oedema, feebleness and irregularity of the heart, depression and occasional ocular symptoms such as night blindness or day blindness (Osler 1907: 750–753). It is clear that the 1907 definition of scurvy encompasses much more than what is now known as scurvy and includes features due to thiamine and vitamin A deficiency. Much of what was diagnosed as scurvy may have been either scurvy with beri-beri or even beri-beri alone. Braddon quotes Nocht as saying 'a few years ago no one would have thought of describing cases of swelling and weakness of the limbs, even without any definite gum lesions, occurring in the course of sea-voyages other than as scurvy' (Braddon 1907: 465).

It is thus not surprising that ship beri-beri and polar anaemia were initially thought to be a variant of scurvy. Once vitamins had been discovered, ship beri-beri was

considered a deficiency of both vitamin B and C (Committee of Lister Institute and Medical Research Council 1924: 135) and while most people with ship beri-beri almost certainly had what is now known as wet beri-beri, clearly some had scurvy as well.

#### Why did some explorers get scurvy and others, beri-beri while some remained well?

The tinned foods that the explorers took with them from Europe would (with the exception of some of the luxury items) have all been similarly prepared and vitamin deficient but there were other differences in the diets of the expeditions. There would probably have been differences in the amounts of fresh food taken on board at the last port of call, in late December or early January, before finally leaving for the Antarctic. There would also have been differences between expeditions as to how much they relied on fresh meat from seals and penguins. Differences in diet have largely been discussed with regard to the calories and vitamin C content of the sledging diets (Kendall 1955; Evered and Goldsmith 1985; Huntford 2000: 565); Pugh 1972). As regards thiamine intake, Huntford (2000: 565) states that on Scott's *Terra Nova* expedition, it was 1.26 mg per day compared to a requirement of 1.8 mg (for 4,500 calories intake). There is no evidence of beri-beri on this expedition. Amundsen's intake was 2.09 mg due to wholemeal bread leavened by fresh yeast (a good source of thiamine) (Huntford 2000: 372, 565). Tyler-Lewis (2006) states that the thiamine intake of the Ross Sea party was less than half of the requirement. I have found no analysis of the diet of the expeditions that developed beri-beri.

The Medical Research Council wrote in 1924, that 'a diet deficient in both anti-scorbutic and anti-beri-beri accessory factors will occasion both diseases, but the likelihood is that symptoms of beri-beri will be the first to appear' as beri-beri comes on after 80–90 days of a diet consisting mainly of polished rice whereas on a vitamin C deficient diet, scurvy took about four months to appear (Committee of Lister Institute and Medical Research Council 1924: 122). More recent work has shown that with a vitamin C deficient diet, scurvy changes may take even longer. Skin changes started after 21 weeks but that by 26 weeks only six of ten volunteers had bleeding into the skin follicles and by 35 weeks there was still one who had no bleeding. Gum changes first appeared at 26 weeks (Vitamin C Subcommittee of the Accessory Foods Committee 1953). Studies of thiamine deficient diets have been briefly reviewed by Carpenter and give less clear cut results. Symptoms such as apathy, fatigue and loss of appetite may appear after as few as two weeks and objective muscle weakness was present within three months but classical beri-beri was not produced (Carpenter 2000: 159–160). This difference in the time to produce symptoms is borne out by the fact that Charcot and Godfroy on the second French expedition suffered

from both diseases but while the beri-beri appeared in June, the scurvy did not appear until September.

There is some evidence that heavy exertion may increase the requirement for vitamin C and thus speed the onset of scurvy (Norris 1983) and this would explain why most of the scurvy occurred in sledging parties.

There is also much variation between individuals. During the World War II, the British interned in Hong Kong were fed a poor diet and the first cases of beri-beri appeared in about three months but over the next four months, only 9% developed beri-beri (Carpenter 2000: 156). Some of this variation is genetic but other variations will be in their size, degree of exertion and their tastes. Danco said he would rather die than eat penguin (Cook 1980: 333). Chollet on *Pourquoi Pas?* did not eat seal meat and this would have predisposed him to his scurvy. On Shackleton's *Endurance* expedition, one the sailors complained that providing fresh seal meat rather than tinned meat was 'a — cheap way of running the expedition' and the crew preferred tinned meat but were won over by seal being served to the ward room which produced the response: 'if the wardroom can have it, we can, can't we'. (Huntford 2000: 417). Thiamine concentrations in muscle are low but are much higher in liver and kidney (Carpenter 2000: 128–129) and so individuals who ate lean meat but avoided offal would have a lower intake of thiamine.

### Treatment

As the disease was considered to be scurvy, or a variant thereof, the mainstay of treatment was fresh food. The only fresh food available to the polar explorers was meat. Lean muscle contains some vitamin C and so while lightly cooked meat may help to treat scurvy (Carpenter 1988: 230–232) it contains small amounts of thiamine (Carpenter 2000: 128–129) and so was ineffective in treating beri-beri. Concentrations of both vitamins are higher in liver.

Specific remedies were tried for the cardiac disease, though with little success. Cook wrote:

'Medicament, I find, is of little service. A temporary relief is sometimes effected by well-directed drugs, but the lasting effects are disappointing. Iron and arsenic, and many of the ordinary tonics effective in home anaemias, are entirely inert. After considerable experiment, I have abandoned drugs as an important aid. Fresh food, artificial heat, a buoyant humour, judicious clothing, and the least possible humidity are the conditions which suggest a rational treatment. . . . I prohibit all food except milk, cranberry sauce and fresh meat, either penguin or seal steaks fried in oleomargarine. The patient is not allowed to do anything which will seriously tax the heart. His bedding is dried daily and his clothing is carefully adjusted to the needs of his occupation. Laxatives are generally necessary, and vegetable bitters with mineral acids, are a decided help. Strychnine is the only remedy which has given me any service in regulating the heart,

and this I have used as a routine. But surely one of the most important things was to raise the patient's hopes and instil a spirit of good humour' (Cook 1980: 331–332).

Strychnine was not effective when Ramsay was terminally ill. Bruce reports that 'Ramsay was very far gone by then, and had bid good-bye to all. . . Pirie administered sulphonal [a sedative] and morphine, as well as strychnine [a stimulant], after which he slept for five hours, his breathing and heart improving all the time.' Later he deteriorated and 'a further injection of strychnine was of no avail' (Bruce 1992: 133) and he died.

It is well known that Cook treated patients by exposing them to an open fire and this has been said to have been the first recognition and treatment of seasonal affective disorder (Myerson 2001) but in his opinion he was treating the cardiac disease as much as the psychiatric disease as he wrote: 'The effect is most noticeable in the action of the heart which during the long night, is deprived of its regulating force; now quick, now slow; then strong; again feeble, but never normal. The best substitute for this absence of the sun is the direct rays of heat from an open fire. From an ordinary coal or wood fire this effect is wonderful. I have stripped and placed men, before the direct rays of heat, whose pulse was almost imperceptible, and in the course of less than an hour had a heart action nearly normal' (Cook 1980: 317).

In the English translation of his book describing the *Français* expedition, Charcot is quoted as saying: 'I shall adopt Dr. Cook's treatment, since it is sensible and can do no harm. It consists of having the patient sit naked in front of a red hot stove; but I am relying more on absolute rest and the beneficial effects of milk, fish and nature, together with a little digitalis and caffeine' (Charcot 2004: 137). However the original French talks about '... les bons effets du lait, de la somatose et du poisson. . .' [the beneficial effects of milk, somatose and fish] (Charcot 1908). Somatose was a beef extract used as an invalid food.

Hanson was treated with electrical treatment to his legs (presumably because of the loss of sensation), Bernacchi writing that 'the doctor is very reticent upon the subject and devotes much attention to the patient, applying a galvanic battery to his legs daily' (Crawford 1998: 135).

### Acknowledgments

This research was funded by the Wellcome Trust which awarded the author a research grant to study medicine during the heroic age of Antarctic exploration. I would also like to thank Dr. P. Witt for translating parts of Dr. Gazert's medical report of the first German Expedition and Professor K.J. Carpenter for advice on the cause of ship beri-beri.

### References

- Amundsen, R. 1913. *The south pole. An account of the Norwegian Antarctic expedition in the 'Fram', 1910–1912. Vol. 1.* Translator, A.G. Chater. London: John Murray.

- Bernacchi, L. 1901. *To the south polar regions: expedition of 1898–1900*. London: Hurst and Blackett Ltd.
- Borchgrevink, C.E. 1980. *First on the Antarctic continent*. London: C. Hurst and Co.
- Brabin, B.J., and J.B.S. Coulter. 2003. Nutrition-associated disorder. In: Cook, G.C., and A. Zumla (editors). *Manson's tropical diseases*. 21st edition. London: Saunders.
- Braddon, W.L. 1907. *The cause and prevention of beri-beri*. London: Rebman Ltd.
- Bruce, W.S. 1911. *Polar exploration*. London: Williams and Norgate.
- Bruce, W. S. 1992. *The log of the 'Scotia'*. Edinburgh, Edinburgh University Press.
- Carpenter, K.J. 1988. *The history of scurvy and vitamin C*. Cambridge: Cambridge University Press.
- Carpenter, K. J. 2000. *Beriberi, white rice and vitamin B*. Berkeley: University of California Press.
- Carter, K.C. 1977. The germ theory, beriberi, and the deficiency theory of disease. *Medical History* 21:119–36.
- Charcot, J-B. 1908. *Expédition Antarctique Française (1903–1905)*. *Journal de l'expédition*. Paris: Masson et Cie.
- Charcot, J-B. 1931a. La maladie des conserves, *La Presse Médicale* 55: 1057–1058.
- Charcot, J-B. 1931b. Le traitement du scorbut moderne ou maladie des conserves. *Bulletin de L'Académie de Médecine* 60: 748–756.
- Charcot, J-B. 1978. *The voyage of the 'Pourquoi Pas?'*. Translator, P. Walsh. London: C. Hurst and Co.
- Charcot, J-B. 2004. *Towards the south pole aboard the 'Français'*. Translator, A.W. Billingham. Huntingdon: Bluntisham Books.
- Committee of Lister Institute and Medical Research Council. 1924. *Report on the present knowledge of accessory food factors (vitamins)*. 2nd edition. London: HMSO.
- Cook, F.A. 1980. *Through the first Antarctic night*. London: Hurst and Co.
- Crawford, J. 1998. *That first Antarctic winter*. Christchurch NZ: South Latitude Research Ltd. 135
- De Gerlache de Gomery, A. 1998. *Fifteen months in the Antarctic*. Translator, M.M. Raraty. Huntingdon: The Erskine Press and Bluntisham Books.
- Drygalski, E.D. von. 1989. The southern ice continent. Translator, M.M. Raraty. Bluntisham: Bluntisham Books and Erskine Press.
- Ekelöf, E. 1904. Medical aspects of the Swedish Antarctic expedition October 1901–January 1904. *Journal of Hygiene* 4: 511–540.
- Evered, D.F., and R. Goldsmith. 1985. Vitamin C supplies on the British Antarctic expedition 1907–09, *Polar Record* 22(141): 680–601.
- Filchner, W. 1994. *To the sixth continent: the second German South Polar expedition*. Translator, W. Barr. Huntingdon: Bluntisham Books.
- Gazert, H., and O. Renner. 1927. Die beriberifälle auf Kerguelen. In: *Deutsche Südpolar-Expedition 1901–1903 im Auftrage des Reichsamtes des Innern. Vol. 4(1)*. Berlin: Walter de Gruyter and Co. 355–386.
- Gourdon, E. 1913. *Thèse pour le Doctorat en Médecine; un hivernage dans l'Antarctique*. Paris: G Steinheil,
- Gourdon, E. 1936. Hivernage dans l'Antarctique en 1904. In: *Jean-Baptiste Charcot 1897–1936*. Paris: Yacht Club de France. 131–133.
- Hess, A.F. 1920. *Scurvy, past and present*. Philadelphia: J.B. Lippincott and Co.
- Holst, A. 1907. Experimental studies relating to 'ship-beri-beri' and scurvy. *Journal of Hygiene*. 7:619–633.
- Huntford, R. 2000. *The last place on Earth*. London: Abacus.
- Kendall, E.J.C. 1955. Scurvy during some British Polar expeditions 1875–1917. *Polar Record* 7:467–485.
- Koettlitz, R. 1904. Physiological measurements and bacteriological notebooks. London: Natural History Museum MS National Antarctic Expedition 1901–1904.
- McIntyre, N., and N.N. Stanley. 1971. Cardiac beri-beri: two modes of presentation. *British Medical Journal* 3:567–569.
- Macklin, A.H. 1923. Appendix V – Medical. In: Wild, F. *Shackleton's last voyage: the story of the 'Quest'*. London: Cassells and Co Ltd.
- McLean, A.L. 1919. *Bacteriological and other researches*. Australasian Antarctic Expedition 1911–14. Sydney (Scientific reports series C vol. VII, part 4): 100–106.
- Murphy, D.T. 2002. *German exploration of the polar world: a history 1870–1940*. Lincoln and London: University of Nebraska Press.
- Myerson, R.M. 2001. Frederick A Cook, MD: the art and science of medicine aboard the *Belgica*. In: Declair, H., and C. de Broyer (editors), *The 'Belgica' expedition centennial. Perspectives on Antarctic science and history*. Brussels: Brussels University Press.
- Norris, J. 1983. The 'scurvy disposition': heavy exertion as an exacerbating influence on scurvy in modern times. *Bulletin of Medical History* 57: 325–538.
- Osler, W. 1907. *The principles and practice of medicine*. 6th edition. London: Appleton.
- Pugh, L.G.C. 1972. The logistics of the polar journeys of Scott, Shackleton and Amundsen. *Proceedings of the Royal Society of Medicine* 65:42–47.
- Skelton, J. 2004. *The Antarctic journals of Reginald Skelton*. Cheltenham: Reardon Publishing.
- Truswell, S. 2007. The B vitamins In: Mann, J., and A.S. Truswell (editors). *Essentials of human nutrition*. 3rd edition. Oxford: Oxford University Press: 184–200.
- Tyler-Lewis, K. 2006. *The lost men*. New York: Viking.
- Vitamin C subcommittee of the Accessory Foods Committee. 1953. *Vitamin C requirement of human adults*. London: HMSO
- Wilson, E.A. 1905. The medical aspect of the *Discovery's* voyage to the Antarctic, *British Medical Journal* 2:77–80.
- Wilson, E.A. 1966. *Diaries of the 'Discovery' expedition to the Antarctic Regions 1901–1904*. Editor, A. Savours. London: Blandford Press.