

Military Cold Injury During the War in The Falkland Islands 1982: An Evaluation of Possible Risk Factors

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SUMMARY: Throughout the history of war, there have been many instances when the cold has ravaged armies more effectively than their enemies. Delineated risk factors are restricted to negro origins, previous cold injury, moderate but not heavy cigarette smoking and the possession of blood group O. No attention has been directed to the possibility that abnormal blood constituents could feasibly predispose to the development of local cold injury. This study considers this possibility and investigates the potential contribution of certain components of the circulating blood which might do so.

Three groups of soldiers from two of the battalions who served during the war in the Falkland Islands in 1982 were investigated. The risk factors which were sought included the presence or absence of asymptomatic cryoglobulinaemia, abnormal total protein, albumin, individual gamma globulin or complement C3 or C4 levels, plasma hyperviscosity or evidence of chronic alcoholism manifesting as high haemoglobin, PCV, RBC, MCV or gamma glutamyl transpeptidase (GGT).

No cases of cryoglobulinaemia were isolated and there was no haematological evidence to suggest that any of those men who had developed cold injury, one year before this study was performed, had abnormal circulating proteins, plasma hyperviscosity or indicators of alcohol abuse. Individual blood groups were not incriminated as a predisposing factor although the small numbers of negroes in this series fared badly.

Although this investigation has excluded a range of potential risk factors which could contribute to the development of cold injury, the problem persists.

Two areas of further study are needed: the first involves research into the production of better protective clothing in the form of effective cold weather boots and gloves and the second requires the delineation of those dietary and ethnic factors which allow certain communities to adapt successfully to the cold. A review of the literature in this latter area is presented.

Introduction

Local cold injury may greatly reduce effective combatant troops in war and can result in considerable morbidity during exercises in peacetime. Its significance and occurrence is underestimated and frequently under-diagnosed. Until the Falklands War of 1982 the last occasion in which British servicemen fought in a cold climate was in Korea and the remaining medically qualified veterans of that conflict have nearly all retired.

There are historical instances in which the cold has inflicted more battle casualties than the enemy. Larrey¹ reported the loss of 11650 out of 12000 men of the 12th division of Napoleon's Grand Army during the Russian Campaign and Hitler's advance into, and subsequent retreat from, the USSR during World War II resulted in both sides losing catastrophic numbers of men from the cold². The British Army documented 115361 cases of frostbite and trench foot in the official records of World War I but the majority of these occurred early in the conflict. With the introduction of duckboards, the issue of dry socks and strictly imposed foot and hygiene discipline the incidence dropped during the

later years of the campaign³. The influence of these measures in combating cold injury was again manifest during World War II in North West Europe where British casualties were much lower than the 91000 suffered by the United States Army of whom some 87% were infanteers. There were times during the winter of 1944-1945 when the cold resulted in up to one-third of American battle casualties^{4,5}.

No accurate figures for the number of British and Argentinian troops who fought in the Falklands and sustained cold injury are available although symptoms were recorded in 28.5% of 3 Para and 20-30% of 2 Scots Guards⁶.

Non-freezing and freezing cold injury not only reduces fighting capability in war but also occurs sporadically in the United Kingdom and in North West Europe amongst soldiers either on exercises or as a result of sleeping rough whilst intoxicated.

Any blood constituent which would impair flow at reduced temperature is likely to predispose to damage in a cold environment. Cryoproteins are known to do so⁷.

The digital necrosis seen in patients with cryo-

globulinaemia is clinically indistinguishable from that produced by freezing and this appearance raised the possibility that there might be a group of otherwise asymptomatic individuals who had small quantities of cryoglobulins circulating in their blood which might predispose them to developing cold injury. The further possibility that there could be a number of Servicemen who were polycythaemic and hyperviscid due to the effects of chronic alcohol ingestion could explain why some but not all personnel who served in the Falklands campaign sustained cold injury whilst others subjected to similar conditions did not do so.

This study examines these possibilities by comparing venous blood obtained from British Servicemen who had clinical cold injury during the campaign with a similar group who experienced the same environmental conditions but did not do so and a further group who did not participate.

Subjects and Methods

Six groups of soldiers, three from the 3rd Battalion, The Parachute Regiment and three from the 2nd Battalion, Scots Guards were studied. The first two groups (A) consisted of men from these battalions who were diagnosed as having sustained cold injury in the Falklands. Confirmation of this diagnosis has been substantiated in most cases by objective measurement of impaired nerve conduction and by abnormal vasomotor response to a cold stimulus observed by strain-gauge plethysmography, (McCaig 1983)⁸. The second two groups (B) consisted of men of similar age who had gone to the Falklands but who did not sustain injury despite being subjected to an identical environment. Selection of these subjects was made by the sub-unit commanders who had led them during the war. The third group (C) consisted of a similar number of soldiers who did not go to the Falklands and who had not previously suffered the effects of cold elsewhere in the world.

All personnel gave informed written consent for venipuncture, which was performed two to three hours after a midday meal. Consent forms were numbered serially and allocated randomly. Thus the sampling and analysis was performed blind and the groupings constructed after the results were obtained from lists provided by the units.

Samples of 20ml venous blood were withdrawn from the antecubital fossa using a venous tourniquet into syringes and needles warmed to 37°C. 10ml of this blood was immediately transferred to EDTA lined bottles previously warmed to 37°C and replaced in a warmer at the same temperature. The warmed specimens were centrifuged at 37°C for 10 minutes at 1000 rpm followed by 15 minutes at

2000 rpm. Thereafter, the supernatant plasma was collected into plain bottles at room temperature and transferred for cryoglobulin, total protein, albumin, IgC, IgA, IgM, complement C3 and C4 estimation. These plasma samples were divided into three aliquots, one placed at 4°C, one at 37°C and the third retained at room temperature. Regular inspection was carried out for 72 hours but no cryoproteins were observed. Immunoglobulin (IgG, IgA, IgM, C3 and C4) levels were estimated by immunonephalometry on a Disc 120 laser nephelometer (Hyland Laboratories, USA) using goat antisera to IgG, C3 & C4 (Atlantic Antibodies, USA), IgA, IgM (ICL, Scientific, USA). Total protein and albumin levels were obtained by standard laboratory techniques.

The other specimens were analysed by routine methods on a Coulter S Senior, (Coulter Electronics, Linton, Beds.) for haemoglobin, packed cell volume, red cell count and MCV. Plasma viscosity was determined on a Harkness Coulter Viscometer⁹ and gamma glutamyl transpeptidase levels were estimated by an automated method using the technique of Szasz¹⁰ on a Coulter Kem-o-mat autoanalyser.

Reference ranges were: total protein, (55-79g/l), albumin, (30-42g/l), IgG, (5.4-16.1g/l), IgA, (0.9-3.4g/l), IgM (0.5-2g/l), C3, (0.7-1.7g/l), C4, (0.1-0.7g/l), plasma viscosity (1.5-1.72cp) and GGT, (6-28iu/l).

Quantitative data were compared between groups using an unpaired test and blood group data were analysed using a Chi Squared test with Yates correction where applicable.

Results

Although the mean ages of the groups in 2 SG were higher than those in 3 PARA, no statistical differences were evident. They are shown on Table 1.

Table 1
Subjects Studied

	Number	Age (Yrs)
3 Para		
Group A	14	22.2 ± 2.33*
Group B	15	21.4 ± 2.10
Group C	16	20.3 ± 2.55
2 SG		
Group A	16	24.4 ± 3.10
Group B	16	23.8 ± 4.02
Group C	16	25.8 ± 4.74

* ± 1 SD

Information was obtained from the RMO's of the two battalions^o on the distribution of blood groups as was data on those soldiers who were studied. Tables II(a) and II(b) show these distributions.

The variation in the proportions of blood group genotypes between the two battalions reflects regional differences in the distributions of blood groups. In this small series no protection due to the carriage

Table II (a)

Blood Groups	A	B	O	AB
3 Para				
Rh Pos.	178	41	183	25
Rh. Neg.	30	0	51	0
Totals	208(40.9%)	41(8.1%)	234(46.0%)	25(4.9%)
2 SG				
Rh Pos.	165	52	263	18
Rh. Neg.	17	9	19	2
Totals	182(33.3%)	61(11.2%)	282(51.7%)	20(3.64%)

Table II (b)

ABO and Rhesus Distribution between the Study Groups

	A	B	O	AB	Rh+	Rh-
3 Para						
Group A	8	1	4	1	10	4
Group B	6	1	8	0	13	2
Group C	7	0	8	1	12	4
2 SG						
Group A	5	0	9	2	15	1
Group B	4	2	10	0	16	0
Group C	5	2	9	0	14	2

of blood group A was found nor were there any statistical indicators suggesting an increased tendency to suffer cold injury in holders of blood group O.

The results of haemoglobin, packed cell volume, red cell count and mean corpuscular volume are delineated in Table III.

Haemoglobin levels were found to be higher in Group C of 2 SG when compared with Group C of 2 Para ($P < .05$) but MCV levels were comparable between all groups despite being numerically higher in 2 SG.

Table IV gives the results of plasma viscosity and gamma glutamyl transpeptidase (GGT) levels. Once again these figures were similar, the GGT levels in 2 SG being higher than those in 3 PARA. The levels in Group B of 3 PARA were higher than Group C ($P < .05$).

Finally, Table V shows the results of total protein, albumin, IgG, IgA, IgM, complement C3 and C4. No statistical differences were evident.

Both total protein and albumin levels were at or above the upper limit of normal for the laboratory reference range which is thought to reflect the excellent state of nutrition and fitness seen in these soldiers.

Cryoglobulins were not found in any of the soldiers who were studied.

Six soldiers had GGT levels in excess of 50iu/l. In four of these the plasma viscosity was raised but MCV was above 95fl in only one and $C3 > 1.70$ g/l in three. There were eight men with raised MCV in whom only one had an elevated GGT but four had raised plasma viscosity and C3. Plasma viscosities higher than 1.72cp were measured in 20 individuals. In this group two had an elevated MCV, four a raised GGT and five a raised C3.

It can be concluded that the incidence of alcohol

Table III
Haematology Results \pm ISD

	Hb g/l	PCV	Red Cell Count ($10^{-12}/l$)	MCV (fl)
3 Para				
Group A (n=14)	15.02 \pm 0.92	45.50 \pm 2.34	4.885 \pm 0.357	89.14 \pm 3.11
Group B (n=15)	15.47 \pm 1.16	46.09 \pm 3.17	4.989 \pm 0.357	88.50 \pm 3.34
Group C (n=16)	14.91 \pm 1.04†	45.38 \pm 3.00	4.912 \pm 0.351	88.56 \pm 2.52
2 Scots Guards				
Group A (n=16)	16.10 \pm 0.88	47.54 \pm 2.72	5.033 \pm 0.241	92.13 \pm 4.00
Group B (n=16)	15.50 \pm 0.71	45.83 \pm 0.47	4.893 \pm 0.201	91.30 \pm 2.55
Group C (n=16)	16.18 \pm 1.02†	47.74 \pm 3.02	4.998 \pm 0.290	93.25 \pm 4.16

† $P < .05$

Table IV
**Plasma Viscosity (c p) and Gamma Glutamyl
Tranpeptidase GGT (IU/l) Results \pm ISD**

	Viscosity (cp)	Gamma GT (IU/l)
3 Para		
Group A (n=14)	1.688 \pm 0.121	16.93 \pm 8.84
Group B (n=15)	1.682 \pm 0.079	23.00 \pm 10.78*
Group C (n=16)	1.655 \pm 0.057	12.75 \pm 4.88*
2 Scots Guards		
Group A (n=16)	1.663 \pm 0.067	31.19 \pm 35.73
Group B (n=16)	1.644 \pm 0.087	27.19 \pm 23.75
Group C (n=16)	1.691 \pm 0.092	26.5 \pm 11.55

*P .05

adapted individuals would cause damage. This capacity would appear to be related to maintenance of higher skin temperatures and has been reported in Arctic Indians¹⁴, Alakaluf Indians¹⁵, and in Arctic Eskimos¹⁶. It remains unclear as to what the aetiology of this adaptive response can be ascribed although an Eskimo's basal metabolic rate is higher than a Caucasian of comparable surface area and lean body mass¹⁷. By inference, this excess energy expenditure could be taking place in the vasodilated extremities of these peoples. Little et al¹⁸ (1971) tested skin temperature responses in groups of young and adult Peruvian Indians. One group was compared with adult American and British Caucasians. The adult Indians, regardless of their place of

Table V
Protein Results (g/l \pm 1SD)

	Total Protein (g/l)	Albumin (g/l)	IgG (g/l)	IgA (g/l)	IgM (g/l)	C3 (g/l)	C4 (g/l)
3 Para							
Group A (n=14)	84.57 \pm 3.78	44.86 \pm 3.25	11.71 \pm 0.01	2.40 \pm 0.96	1.49 \pm 0.52	1.25 \pm 0.24	0.45 \pm 0.24
Group B (n=15)	84.2 \pm 4.39	44.0 \pm 1.89	12.08 \pm 1.81	2.30 \pm 0.83	1.55 \pm 0.69	1.33 \pm 0.23	0.49 \pm 0.41
Group C (n=16)	81.19 \pm 4.12	42.25 \pm 3.11	11.25 \pm 1.47	2.55 \pm 0.56	1.35 \pm 0.63	1.38 \pm 0.29	0.39 \pm 0.15
2 Scots Guards							
Group A (n=16)	86.88 \pm 4.22	44.06 \pm 3.43	12.51 \pm 1.85	1.98 \pm 0.58	1.72 \pm 0.54	1.43 \pm 0.30	0.59 \pm 0.42
Group B (n=16)	84.31 \pm 4.96	44.00 \pm 3.18	11.84 \pm 2.00	1.63 \pm 0.51	1.76 \pm 0.63	1.34 \pm 0.21	0.61 \pm 0.41
Group C (n=16)	86.75 \pm 3.82	42.44 \pm 1.86	13.09 \pm 1.63	1.92 \pm 0.79	1.74 \pm 0.54	1.42 \pm 0.18	0.52 \pm 0.19

abuse based upon haematological and biochemical markers in this series of 93 soldiers was very low one year after 61 of them had served in the South Atlantic. In only one individual were all markers positive and although he was in the affected group of 2 SG there is no evidence on these results to suggest that alcohol abuse played any significant part in the development of cold injury in the Falklands.

There were no negroes in 2 SG but all four of those in 3 PARA suffered cold injury.

Discussion

There are several factors which may cause an individual to be more susceptible to local cold injury. Caucasians fare better in a cold environment than negroes who have a 2.8 to six fold increased likelihood of developing symptoms, although the reasons for this difference have yet to be defined^{11,12,13}. Animals or man either adapt to their environment or perish. There are numerous recorded examples of man's ability to avoid cold induced injury despite exposure, which in non-

dwelling, maintained higher skin temperatures after cold exposure than the whites implying a strong genetic effect, but they also maintained higher temperatures than the Indian children which would suggest a degree of adaptation during life. The influence of genetic mixing between Indians and Caucasians was studied by measuring the rate at which cold induced vaso-dilation (CIVD) occurred after hand-immersion at 5°C in two groups of Canadian Algonkian Indians from two villages¹⁹. They concluded that the onset of CIVD occurred later in the Indians with greater Caucasian admixture.

In addition to these genetic factors there is a real possibility that diet may contribute to adaptation to environmental cold. Laursen¹⁷ (1983) has noted that the capacity of Alaskan Eskimos to cope with freezing conditions appears to be lost if they transfer their diet to that of Westerners and stop consuming seal blubber and fish. Sinclair²⁰ (1953) failed to find any cholesterol deposition in the corneas of Eskimos on a traditional diet which contains the highest known proportion of dietary fat. Coronary heart disease is not observed in these people²¹. Fish

and blubber fat is rich in linolenic acid as is the dietary fat consumed by the Japanese in whom atherosclerosis is also rare. He extended his experiments by joining a community of long living Eskimos in Greenland in 1976 and subsequently consumed seal and fish as his sole nourishment for 100 days²². This resulted in extension of his bleeding time from four minutes to in excess of 50 minutes and greatly decreased his platelet aggregation. He postulated that this effect was caused by the high intake of linolenic acids resulting in the production of prostacyclin, (PGI₃) and thromboxane (TXA₃) rather than PGI₂ or TXA₂ which are derived from linoleic acid. Whereas PGI₂ and PGI₃ both deaggregate platelets, TXA₂ promotes thrombosis whilst TXA₃ has little or no effect upon platelets. These interesting findings do suggest that diet may be an important contributor to the circulation of blood in the extremities of Eskimos who consume traditional food and may reduce the likelihood of intravascular thrombosis during periods of impaired flow and hyperviscosity in a cold environment^{20,22}.

Rather more difficult to explain on either genetic or dietary grounds is the capacity to adapt to occupations involving the handling of cold objects or immersion of feet in cold water. Examples include Gaspé fishermen²³, fish filleters²⁴, and Canadian lumberjacks²⁵ whose feet remain immersed and cold for long periods without deleterious effect. A fish diet in the first instances and active movement and exercise in all examples may result in improved circulation in these cases along with an acquired capacity to vasodilate.

Raynaud's phenomenon whether it be idiopathic or secondary to peripheral vascular disease, thromboangiitis obliterans, vibration injury, previous cold injury or to mixed connective tissue disease greatly increase predisposition to the effects of cold²⁶.

Other factors which have been delineated include moderate but not heavy cigarette smoking, fatigue, ethanol consumption, hyperhidrosis, reduced lean body mass, blood group O individuals, inadequate clothing and footwear, hygiene, rank and motivation^{3,11,25}.

With the exception of ABO blood groupings no attention has been paid in the literature to the possibility that the physical properties or composition of the blood itself could predispose to the development of cold injury. Nor is it clear whether the pathological effects of the cold upon the extremities is the direct result of intravascular thrombosis alone or due to an inflammatory response with endothelial damage and vascular destruction in addition. Eady et al²⁷, (1981) in a study of cold induced urticaria and vasculitis, demonstrated the appearance of complement C3 in dermal blood

vessels following a cold stimulus which preceded deposition of fibrin and immunoglobulin within the vessel wall and was associated with mast cell degranulation. There was later perivascular infiltration by inflammatory cells and later still vascular endothelial disruption. Controls showed no such response.

Cryoglobulinaemia has been recognised since 1933²⁸, and the conditions in which it has been observed include myelomatosis, lymphoma, mixed connective tissue disorders including rheumatoid arthritis, systemic lupus erythematosus, Sjogren's disease, cirrhosis, Crohn's disease and disseminated malignancy²⁹⁻³³. There were a number of cases reported above in whom the aetiology for cryoglobulinaemia was unknown and they were described as being essential or idiopathic. Mcgrath and Penny⁷. (1978) demonstrated greatly increased blood and plasma viscosity in cases of cryoglobulinaemia with an associated increased red cell aggregation on cooling at low shear rates. They suggested that this finding explained at least in part the localisation of damage to the skin in those peripheral tissues of the extremities most exposed to cold.

Dehydration due to the combined effects of excessive sweating within the clothing required in a cold environment along with a reduced intake of water also results in hyperviscosity³⁴. Chronic alcohol abuse is associated with a raised MCV and gamma glutamyl transpeptidase, (GGT)³⁵⁻³⁸.

This study has investigated several components of the circulating blood in soldiers who served in the Falklands Campaign of 1982 and has effectively excluded many of them as being aetiological factors in the development of non-freezing cold injury. Thus the reasons why some and not all individuals who are subjected to similar adverse environmental circumstances develop symptoms remains unanswered.

The study was performed one year after the event. As no cases of cryoglobulinaemia were found in any of the soldiers in any of the three groups who were studied, it would seem most unlikely that small groups of individuals with asymptomatic cryoglobulinaemia exist. Whereas it is recognised that epinephrine release produces distal vasoconstriction in association with fear, the concomitant production of cryoglobulins in these circumstances has not been investigated. Perhaps venous sampling of parachutists prior to their first jump might confirm or refute this possibility.

It is known that the circulation in the fingers can be reduced to 3% of control levels following immersion in water at 13°C for two hours³⁹ or to zero at temperatures between 0°C and 8°C⁴⁰. Associated with this diminution of flow is a tendency

to sludging and microvascular occlusion. Thus, polycythaemia or hyperviscosity would tend to accentuate this phenomenon⁴¹. Although high haemoglobin levels have been recorded in chronic alcoholics⁴² it is still disputed whether this is entirely due to the alcohol or whether it derives from the stimulus produced by consistently high levels of carbon monoxide found in heavy smokers who also drink. The possibility that certain soldiers might be found to have biochemical or haematological evidence of chronic alcohol abuse was explored bearing in mind that one year had elapsed between the exposure to cold and also that alcohol intake was restricted to two cans of beer per day during the sea voyage between Ascension Island and the Falklands. This restriction, however, would not necessarily permit a considerably greater intake by alcohol abusers who could have increased their consumption by certain forms of barter.

The concept of hyperviscosity and an increased tendency to sludging associated with a high haematocrit and polycythaemia may contribute to the high proportions of chronic alcoholics who form the bulk of most of the civilian series of frostbite⁴³⁻⁴⁵. More likely is that these cases occur due to acute inebriation causing them to sleep rough in cold weather. It is nonetheless worthwhile to consider chronic alcoholism particularly in troops who are to be exposed to the extremes of cold either on exercise in peacetime or in war.

The tendency for negroes to be more susceptible to the cold than Caucasians was confirmed in this study albeit with small numbers.

The proportion of the population with Group A blood is higher in Scandinavia than in the rest of Europe and it has been postulated that this is associated with an evolutionary capacity to withstand cold⁴⁶. Group B blood becomes increasingly common through Eastern Europe and into Asia⁴⁶. Group O American soldiers have been found to have a 1.8 times increased chance of cold injury during training in Alaska. Whereas 3 Para have a pattern of blood groups which is in close parallel with England despite drawing from all areas of the nation, the 2 SG who recruit 60% of their troops from north of the border had a pattern of distribution which is identical to that found in SW Scotland with a Group A proportion of only 33% and with 52% group O²⁵. This would appear to reflect a Celtic rather than Scandinavian ancestry. There is no indication, however, to restrict soldiers' employment because of their blood genotypes.

Complement C3 and IgM deposition in the walls of dermal blood vessels has been shown to be an early finding in the development of vasculitis associ-

ated with cold urticaria²⁷. In this series, immunoglobulins, complement C3 and C4 levels were all indistinguishable between groups and none of these proteins have been shown to be of aetiological importance in the development of non-freezing cold injury.

It was not possible to obtain information on the smoking habits of the men from these two battalions who served in the Falklands. Information from the 93 who were investigated as regards smoking was not sought. Previous work carried out in Alaska^{11,12,26}, has revealed that light smokers (less than 20 per day) had a higher risk of sustaining frostbite than non-smokers or heavy smokers (more than 20 per day).

Conclusions

The cold produced considerable morbidity and loss of fighting strength during the Falklands Campaign as it has on innumerable occasions throughout the history of war. This study has excluded some of the possible factors within the circulating blood which might have predisposed to its development.

The following areas of research warrant further evaluation. Improvements in cold weather clothing and particularly foot and hand wear is a major priority. Further data on the microscopic, electron microscopic and biochemical nature of the effects of cold upon blood vessel walls, nerves and dermal cells is required. The possible contribution of diet and specifically those rich in omega 3 polyunsaturated fatty acids similar to that consumed by Eskimos who have successfully adapted to their environment needs investigation. Ways in which the prolonged bleeding time produced by this diet could be rapidly corrected in the event of wounding should be sought. Evaluation of vasodilator drugs as prophylactics might prove a worthwhile avenue for study.

Finally, standardisation and quantification of the effects of previous cold injury is required in order to assess prognosis, future employment standards and the possibility of improving late vasospastic symptoms by sympathectomy the value of which in the acute phases of frostbite remains questionable^{44,45}.

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