The Independent Medical Expert Group (IMEG)

Report and recommendations on medical and scientific aspects of the Armed Forces Compensation Scheme

March 2015

Topic 3 – Compensation aspects of Non freezing cold injury (NFCI)

History and Background

1. Non-freezing cold injury (NFCI) has been recognised in the military context from Roman times. While the conditions in the 1982 Falklands conflict predictably led to cases in Royal Marines, in recent memory NFCI has not been a major disabling disorder amongst serving UK troops nor in UK civilian practice. However from the winter of 2005/6 onwards more cases began to appear with consequences for operational capability, healthcare and compensation. Typically NFCI was occurring in army recruits undertaking winter training in UK in the Brecon Beacons, North York Moors and Northumbria. Despite refresher training and new instructions on prevention and management for soldiers and the chain of command, and a new Surgeon General Policy Letter on clinical care, cases continued to present during subsequent winters. During the Falklands conflict, Royal Marine personnel were almost all UK-born Caucasian males while those affected more recently are typically foreign and Commonwealth troops, British born Afro-Caribbeans or Caucasians born and raised in Africa. In January 2012 the Surgeon General set up an independent expert Review Group chaired by Prof Hugh Montgomery, University College London to examine all aspects of NFCI. The report was completed in February 2013 (1).

2. NFCI was rarely claimed under the War Pension Scheme nor was it a common reason for medical discharge even in Royal Marines who fought in the Falklands. Of the 2,000 – 3,000 men exposed to the cold conditions of that campaign only about 12 left service in the following two years because of NFCI.

3. From 2006 however, the picture in the UK military, notably army personnel, is different. The 2013 Report on the Health of the Armed Forces records that in 2012/2013 there were 604 referrals to the Cold Injury Clinic, Institute of Naval Medicine, the great majority being army (2). Defence Statistics confirm that the number of UK personnel medically discharged due to NFCI as the principal or contributory cause from 6 April 2005 until 31 March 2014 is 518. Of these about 330 have AFCS awards for NFCI at some level of severity. In addition to no fault compensation, 707 claims for civil damages have been made between May 2007 and 31 December 2014. 470 have been settled with 197 remaining active. The cost so far is £17.2 million damages and £11.3 million claimant legal costs.

4. If medical discharge occurs due to NFCI, armed forces ill-health pension award will usually be appropriate. Most personnel affected are members of the Armed Forces Pension Scheme 2005 (AFPS 05) scheme where benefits are awarded as a lump sum if the invaliding disorder does not compromise civilian employability. For more serious injuries and disorders, a pension is paid at two levels (Tiers) dependent on the degree of functional compromise for suitable civilian work.

5. The AFCS no fault compensation figures above are a minimum because claims may be accepted up to seven years from “the day on which the injury occurs”. In addition the early editions of the Tariff included no NFCI descriptors. Claims in the early period were few and Tariff Table 4 Physical disorders descriptors were used. Cold injury descriptors (two) were introduced into Table 2 in September 2008:

Item 66 Level 15 - Cold injury which has caused, or is expected to cause, symptoms and significant functional limitation and restriction at 6 weeks, with substantial recovery beyond that date.

Item 62 Level 14 - Cold injury with persisting symptoms and significant functional limitation and restriction.

and the three current descriptors for NFCI at levels 14, 13 and 10 followed IMEG's consideration and recommendations in the first report, January 2011:

Item 65 Level 14 - Non freezing cold injury which has caused, or is expected to cause, neuropathic pain and significant functional limitation or restriction at 6 weeks with substantial recovery beyond that date.

Item 55 Level 13 - Non freezing cold injury which has caused or is expected to cause neuropathic pain and significant functional limitation or restriction at 26 weeks, with substantial recovery beyond that date.

Item 27 Level 10 - Non-freezing cold injury with persistent local neuropathic pain and severe compromise of mobility or dexterity, and evidence of permanent damage to small nerves on thermal threshold testing.

Footnote for all three is

A descriptor for non-freezing cold injury refers to either unilateral or bilateral damage to the upper or lower extremities.

6. The AFCS aims to make consistent and equitable awards for listed injuries and disorders. The likely prognosis and impact of the treated condition on functional capacity for civilian employment over the person's working lifetime is then used to assess injury severity and award level. Injuries and disorders should be diagnosed when they meet defined generally accepted medical criteria e.g. according to the World Health Organisation (WHO) International Classification of Diseases (ICD) and the intention is to make awards full and final following best practice treatment at optimum functional state.

7. The findings of the Montgomery report call into question whether these criteria are met by the current NFCI descriptors. The report found a lack of reliable scientific evidence in the international literature on many aspects of NFCI, including its definition, a systematic description of its clinical features, its natural history, the role of specialist tests in diagnosis and assessment of severity, its prognosis and best practice treatment. The pathogenesis also remains incompletely understood. There is experimental evidence that impaired blood flow due to vasoconstriction leads to ischaemic neural damage. In turn the neural and vascular elements interact causing further ischaemic damage. There is also good experimental evidence that cold directly damages nerve fibres, independent of vascular factors (1).

8. The natural history of NFCI requires further research, but from clinical experience it is possible to state that in the majority of those who develop NFCI, the symptoms and signs resolve completely over weeks or a few months, while pain persists in a minority. Available evidence strongly suggests that this pain is due to damage to small diameter sensory nerve fibres supplying the skin and deeper tissues in the affected extremities (small fibre peripheral neuropathy, SFN

paragraph14-17 inclusive). This neuropathic pain, NP, may be continuous and is frequently exacerbated by mechanical and thermal stimulation of the affected extremities (paragraph18-19 inclusive). In addition, sensitivity to cold, due to abnormal vascular reactivity, is a common consequence of an acute episode of NFCI, and in many subjects, this persists and can be easily demonstrated using thermography. However, the symptoms arising as a result of such cold sensitivity only develop when the affected extremities are exposed to cold, they are usually mild, and they resolve rapidly on re-warming. Abnormal thermography, on the other hand may continue long after symptoms have resolved completely (1).

9. A major recommendation of the Montgomery report which IMEG strongly endorses, is for further research on several other aspects of NFCI. To better understand the natural history of NFCI, a systematic prospective longitudinal study starting at the time of recruitment to the services is needed. The Montgomery report also recommended studies on how NFCI is investigated, diagnosed and treated. Such research will take time and for the foreseeable future military no fault compensation claims for NFCI will be informed by clinical information similar to that available at present. Detailed physical examination findings, general and neurological, are not routinely and systematically recorded in Service primary care medical records, nor at the specialist Cold Injury Clinic, Institute of Naval Medicine (CIC, INM).

10. Based on the limited published peer-reviewed evidence base, IMEG has reviewed and made recommendations on the NFCI descriptors and tariff levels. Another source of information has been discussion of clinical matters with Dr Howard Oakley, recently Head of Survival and Thermal Medicine, INM. Dr Oakley ran the CIC there for many years, assessing up to 1,000 patients with suspected NFCI each year. He has given IMEG access to a number of unpublished papers, including his own 2014 audit of clinical features and observations on patients with NFCI seen at INM during the preceding sixteen months (3). IMEG also had the benefit of a paper from Major-General RP Craig, L/RAMC prepared as an introduction for the Royal British Legion (RBL)'s Medical Advisory Committee (4).

11. Of the total 644 new cases recorded in Dr Oakley's 2014 analysis, there were 271 where injury was sustained before 1 September 2012. The average time interval between injury and the date seen in the CIC was 7.8 months (SD 2.8), with a range of 2 to 15 months. Medical diagnoses other than cold injury were recorded in 89 cases i.e. 14% of the total, with the majority having primary Raynaud's disorder. Of the 284 winter 2012/13 NFCI cases, most were army junior ranks with an average age of less than 30 years; the majority of injuries occurred in the UK. Half had NFCI of hands and feet; in 36% feet only were affected and in 6% hands only. Mixed freezing and non-freezing cold injuries occurred but were rare.

12. The most frequent and troublesome presenting symptom was pain, more or less continuous and lasting more than two hours on re-warming after cold exposure. The proportion of patients experiencing pain diminished exponentially with time from cold exposure, but 23, (8%) were still experiencing pain 32 weeks after the injury and two patients reported having pain 4 years after the initial cold exposure. In addition to pain, numbness was reported in 25% and cold sensitivity i.e. feeling pain on cold exposure by 68%. Other non-neurological signs such as change in skin colour, blistering and skin peeling were uncommon. At INM, diagnosis of NFCI depends on the given history and special investigations, including infrared thermography (IRT) and thermal threshold testing (TTT). However, while abnormal results may support the clinical diagnosis of NFCI, neither of these tests can be regarded as diagnostic. In normal subjects there is a wide reference range of responses to a cold challenge. Abnormal results may be demonstrable in the absence of symptoms and in the case of thermography, even without a history of cold exposure. In the 2014 INM series IRT was near-normal in 55% of those tested and showed mild to moderate cold sensitivity in 39%. Cold

sensitivity was moderate in 22% and worse than that in 12%. TTT was more likely to be normal in the hands (67%) while 59% had abnormal thresholds in the feet.

13. Cases at INM are seen on referral from primary care ideally at three to four months after cold exposure. The recent increase in case numbers, without a commensurate increase in clinical resources, has resulted in many cases being seen only at around nine months or longer, after cold exposure, as noted above (para 11). Where the time interval coincides with the warmer temperatures of spring and early summer there may be natural improvement in symptoms. In terms of prognosis, in Dr Oakley's experience about a third of new cases overall can be recommended for immediate return to full duties; about 55% are advised to keep extremities warm and dry and use foot spa treatment. Normal practice is then for their review at about six months, when most are found to be fit to return to duty in some degree. Neuropathic pain management is needed from the initial visit in about 10% cases and about 4% at the first visit are thought to be so severely affected by pain as to be likely to require medical discharge.

Small Fibre Peripheral Neuropathy and Neuropathic Pain

14. **Small Fibre Peripheral Neuropathy (SFN)**. A small fibre neuropathy results from structural damage selectively affecting small diameter unmyelinated and myelinated fibres. The unmyelinated fibres include both sensory fibres, carrying sensations of heat, mechanical or chemical stimuli, and sometimes all three, and autonomic motor fibres controlling blood vessels and sweat glands. SFN may complicate systemic diseases such as diabetes mellitus, sarcoidosis, vasculitis, rheumatoid arthritis and other auto-immune conditions as well as infections including HIV and Hepatitis C; it may also occur in alcohol misuse and be associated with certain therapeutic drugs and rare genetic pain syndromes (5). Most SFNs in these conditions are length-related, affecting the feet initially and then the hands, progressing proximally in a glove and stocking distribution.

15. The most severe symptom of SFN is neuropathic pain (NP), often continuous and described as burning, gnawing, stinging or raw, with paroxysmal lightning, stabbing or electric shock-like pains superimposed. Normally harmless stimuli such as light touch or gentle stroking, wearing socks or the bed-sheets can also evoke pain, a symptom and sign known as allodynia (5). In its most extreme form, seen most commonly in some alcoholic and diabetic patients, neuropathic pain may severely compromise function, preventing weight-bearing, walking and use of the hands. On neurological examination, signs of SFN are often difficult to detect. However, with careful sensory testing, impairment of temperature and pin prick (pain) sensation may be detected, while light touch, vibratory and joint position sensations, mediated by larger myelinated nerve fibres, are normal.

16. Diagnostic tests for SFN in routine clinical practice are limited. Standard nerve conduction tests (NCS) measure conduction in large myelinated fibres and so are normal. Thermal Threshold Testing (TTT) yields data on perception of warm and cold, but is a psychophysical test that relies on consistent and accurate subjective report, and is open to error. Within the last decade, skin biopsy and measurement of the reduced density of free nerve endings in the epidermis, using standard histochemical techniques has emerged as the first validated measure of damage to small nerve fibres, and there are now agreed diagnostic criteria. Skin punch biopsy of the lower limb is suitable for most patients and has a low complication rate. International normative standards of nerve fibre density in skin epidermis are now available and the diagnosis of SFN is made when the patient's value lies below the fifth centile for age and sex matched controls (6). The test correlates well with clinical symptoms and signs and has high sensitivity and specificity (7). Quality diagnostic services

with samples sent by courier are now becoming more widely available in UK central neuropathology laboratories (5).

17. In summary, SFN can be a difficult diagnosis to make with confidence, requiring a careful and detailed clinical assessment by specialist neurologists. A clinical expert panel has set out criteria for the diagnosis of SFN in diabetes, based on clinical features coupled with special investigation (8). More recently another group has recommended that these criteria should apply in any case of clinically suspected neuropathy (9). Improved access to skin biopsy in routine clinical practice will greatly increase diagnostic accuracy.

18. **Neuropathic Pain (NP)**. In the peripheral neuropathy of NFCI, neuropathic pain is distributed according to the parts affected in the initial episode of NFCI. Both in its symptoms and pathogenesis NP is distinct from nociceptive pain, the type of pain experienced normally, which has an essential protective function and is signalled by an intact nervous system (10) (11). Damage to small sensory nerve fibres in SFN causes a range of abnormal properties in the damaged nerve fibres and secondary changes in the spinal cord. NP is variable in severity and may be continuous or intermittent. At its worst, it is unremitting and debilitating, severely limiting normal dexterity or mobility or both. Treatment of NP is difficult. A wide range of interventions may be employed, including local measures, systemic drug therapy, and occasionally surgery (10). For NP related to NFCI, most commonly used systemic drugs include amitriptyline, gabapentin or pregabalin. Doses should be titrated carefully to the patient, but at best they commonly produce only partial pain relief, and medication related adverse effects, typically dizziness, sedation and fatigue, are common.

19. As expected with any painful condition, low mood, disturbed sleep and anxiety are common. Longer term consequences of NP include social isolation, reduced physical function and employability, relationship difficulties and substance misuse (10) (12). Robust studies on discrete psychiatric diagnoses in NP are limited by study design, numbers of subjects, lack of controls and because studies often consider pain in general, not solely or specifically NP. The evidence is that in NP the prevalence of a discrete diagnosable mental health disorder, meeting ICD 10 criteria is relatively low although more common than in control populations who do not suffer NP (13). There is a link between a person's attitude to their pain and the development of mood disorder or anxiety state. The risk is highest in those who are pessimistic, feel hopeless, believe that they will be unable to cope and feel overwhelmed by the pain. These findings have led to a biopsychosocial model and approach to treatment of the disorder, rather than simply a biomedical one. Psychological interventions, notably cognitive behaviour therapy are increasingly recognised as having a part to play in improving the patient's emotional state.

Findings and recommendations on diagnosis and clinical course

20. Consideration of the published evidence, the unpublished papers and discussion has led to the following findings and recommendations on NFCI diagnosis and clinical course.

a. Diagnosis.

NFCI is a pathological state arising from the sustained cooling of peripheral tissue with temperatures in the range from just above freezing to about 20 degrees C. Non-freezing temperatures are often associated with exposure to persistent wetness, and it is usually the combination of cold and wet that leads to the development of NFCI. The temperature at

which NFCI may develop has been extended to around 20 degrees C because symptoms and signs similar to NFCI have been reported in warm climates following prolonged foot immersion (1). However the vast majority of those affected in the military around the world have been reported in those with prolonged exposure to colder non-freezing temperatures associated with persistent wetness of the affected extremities (1) and this relates directly to the UK experience of NFCI. Symptoms of acute NFCI are always maximal at the distal parts of the affected digit or limb and are due to prolonged vasoconstriction and direct tissue damage. As long ago as 1945, Ungley described four discrete stages in the presentation and evolution of NFCI (14) but symptoms and signs often overlap, the time course of transition through the various stages is variable and not all symptoms are present in every patient. NFCI should be diagnosed from the combination of clinical history, clinical examination and special tests. There should be

1) A history of cold exposure, with the onset of appropriate symptoms during cold exposure and typical re-warming symptoms and signs. In those with persistent symptoms first seen at intervals of up to several months after the cold exposure it is important to seek corroborative evidence from contemporaneous medical records.

2) Physical examination should include inspection of skin, a vascular assessment and neurological examination of large and small peripheral nerve fibre function. Symptoms and signs of acute NFCI resolve completely in the majority of cases but abnormal vascular reactivity may lead to ongoing abnormal cold sensitivity, which may be asymptomatic. Abnormal signs attributable to large nerve fibre dysfunction usually resolve within weeks of injury, with restoration of light touch, vibration and proprioception and normal tendon reflexes, while abnormal small nerve fibre functions may persist, with impairment of sensitivity to pin prick and temperature.

3) The special investigations, IRT and TTT, may be difficult to interpret. As commented already TTT is a psychophysical test dependent on reliable subject report, and can yield abnormal results in asymptomatic individuals. With IRT there is a wide range of responses to a cold challenge and abnormal results can be seen in individuals with no history of NFCI (1). Thermal thresholds are however always abnormal in those with small fibre neuropathy. In those with the vascular sequelae of NFCI, i.e. cold sensitivity, thermal thresholds are typically normal in the hands. In the feet thresholds are normal to cooling but abnormal, i.e. slow, to re-warming (Dr Oakley, personal communication). Both the Montgomery Report and discussion with Dr Oakley suggest that especially in Afro-Caribbean personnel there must be doubts about the use of Infra-Red Thermography for claims assessment at all. Thermal Threshold Testing can be used as an adjunct but alone, it does not establish the diagnosis nor confirm NFCI severity.

b. Clinical course of NFCI

The characteristic clinical features of NFCI include:

1) **NUMBNESS**. Numbness occurs as an almost universal symptom in acute cold exposure.

2) **PAIN**. During re-warming after acute cold exposure, the feet become hot and pain (often described as burning in quality) is reported in most cases. In Dr

Oakley's experience, pain usually develops within 2 weeks of cold exposure and almost never at an interval of more than 4 weeks; the longest interval he would accept for the onset of NFCI-related pain that then persists is 12 weeks. Pain starting de novo after this time would not, in his view, be due to NFCI. The numbers of patients with pain diminishes with time after cold exposure. Almost all those with acute NFCI recover within 6 months and do not develop functionally limiting symptoms, though cold sensitivity may be demonstrable.

3) **OTHER SYMPTOMS**. Longer term additional neurological symptoms reported in a minority of cases, and in descending order of frequency, are numbness, paraesthesiae and allodynia. These symptoms are transient and resolve on re-warming. While the prevalence of psychological symptoms in NFCI is unknown, such symptoms and functional limitations are well recognised in patients with other types of peripheral neuropathy. A relatively small minority of those with a peripheral neuropathy may also suffer from a discrete mood disorder.

4) **BODY AREAS AFFECTED**. In the Falklands cohort almost all cases had NFCI of the feet and hands were only very rarely involved. By contrast, in the 2014 audit, half had NFCI affecting hands and feet; in about 40% only feet were affected and in less than 10%, hands only. Where hands and feet were both involved, typically severity was different in the two body areas.

5) **OTHER CLINICAL ISSUES**. Current INM treatment of acute NFCI includes avoidance of further cold exposure and foot spa therapy. The latter has not been subjected to study in a well-controlled clinical trial. Neuropathic pain is treated with either amitriptyline or pregabalin, but again, use of these drugs in NFCI is not based on controlled clinical trials. Dr Oakley is of the view that there are probably some individuals who, when exposed to repeated 'low level' cold, may develop features of chronic NFCI in a cumulative fashion without a clear prior clinical episode of acute symptoms and signs. The number likely to be affected in this way is not known, but in his opinion is probably small. While intuitively those with a history of a previous episode of NFCI might seem likely to be at increased risk of another episode and it is sensible for those with NFCI to avoid further cold exposure, as yet the evidence is unclear. A recent UK military study found men of Afro-Caribbean origin to be about 30 times more likely to develop NFCI than Caucasians (15). The chronic sequelae of NFCI tend to be either mainly vascular or neurological. There are racial differences, with Caucasians much more frequently developing vascular sequelae (95%) compared with 5% neurological sequelae. By contrast, in Dr Oakley's experience, 25% of Afro-Caribbeans develop neurological sequelae.

Definitions of Acute and Chronic NFCI

21. Because in most cases the acute features of NFCI resolve completely within 12 weeks of symptom onset, with only about 10% going on to develop chronic problems, IMEG takes the view that, for clinical and compensation purposes, it is useful to separate the acute and chronic phases of NFCI. In the current state of knowledge and reflecting clinical documentation in military records the following definitions are recommended.

Acute NFCI results from exposure to sustained cooling of a limb or limbs at temperatures ranging from just above freezing to 20 degrees C. Symptoms develop during the period of

cold exposure and include initial coldness and numbness in the affected part and changing colouration of the extremity. Signs include limb(s) cold to the touch, reduced or absent peripheral pulses, impaired cutaneous sensation and reduced or absent tendon reflexes. Pain, often severe, develops at an interval of days to weeks after the incident event, but not longer than 12 weeks following cold exposure.

22. In those who do not go on to develop persistent disabling clinical features, abnormal vascular reactivity may be demonstrable in the longer term. Abnormalities of sweating, particularly hyperhidrosis, skin blistering and swelling are common but not universal features of acute NFCI.

23. The clinical features of chronic NFCI include persistent abnormal vascular thermal reactivity and a sensory neuropathy affecting solely or predominantly small nerve fibres and giving rise to chronic continuous or intermittent neuropathic pain, frequently accompanied by cold allodynia.

Chronic sequelae of NFCI (Chronic NFCI) comprise symptoms and signs persisting at more than twelve weeks following an episode of cold exposure and the features of acute NFCI. Symptoms include neuropathic pain, accompanied by signs consistent with abnormal vascular reactivity to cold, and a sensory small fibre peripheral neuropathy, characteristically associated with cold allodynia.

24. In the AFCS, the award aims to take account of the disabling effects of the injury or disorder over a lifetime. In NFCI, serious challenges include the lack of objectivity in diagnosis, particularly in those with persistent symptoms, and the limited current understanding of the natural history or treated course of NFCI. Current understanding is that in the great majority of cases, there is complete resolution of symptoms and restoration of function. Dr Oakley estimates that about 10% have persisting symptoms and variable functional compromise, but even then and at up to twelve years post exposure, there can be marked improvement in symptoms and function. The INM experience is that in Caucasians, except those from tropical regions, ongoing symptoms, if present, are likely to be vascular (95%) i.e. cold sensitivity and only mildly disabling. Only approximately 5% have ongoing troublesome neurological symptoms, notably neuropathic pain. The proportions for Afro-Caribbeans with persistent neurological features are 25%; mixed vascular and neurological features are seen in 10%; and vascular features alone in 65%. Dr Oakley reports that in a small number of those assessed at INM (less than 2-3%), symptoms and signs are consistent with the development of Raynaud's phenomenon (i.e. secondary Raynaud's phenomenon due to NFCI). It is concluded that this may occur as a result of an episode of acute NFCI, but it has not yet been the subject of prospective systematic investigation.

Recommended descriptors

25. The recommended scope, format and elements for the three new descriptors are:

1) to cover both acute NFCI which resolves by 12 weeks, and acute NFCI with symptoms persisting after 12 weeks, but with recovery at 26 weeks:

- acute NFCI with resolution of symptoms and signs within 26 weeks of symptom onset

2) for chronic NFCI with persistent cold sensitivity:

- acute NFCI progressing to chronic NFCI at 12 weeks of symptom onset with

persistent cold sensitivity beyond 26 weeks.

3) for chronic NFCI with persistent cold sensitivity, neuropathic pain and severe functional limitation of feet or hands or both:

- acute NFCI progressing to chronic NFCI within 12 weeks of symptom onset with verified small fibre neuropathy, persistent cold sensitivity, neuropathic pain and severe functional limitation or restriction beyond 26 weeks.

26. Recommended revised AFCS descriptors

Item 65 Level 14

Non-freezing cold injury which has caused pain in the feet or hands or both, with functional limitation or restriction at 6 weeks and substantial recovery by 12 weeks. Continuing cold sensitivity may be present beyond 12 weeks.

Item 55 Level 13

Non-freezing cold injury which has caused neuropathic pain in the feet or hands or both, with significant functional limitation or restriction at 26 weeks and substantial recovery beyond that time. Continuing cold sensitivity may be present beyond 26 weeks.

Item 27 Level 9

Non-freezing cold injury in feet or hands or both, with small fibre neuropathy diagnosed clinically and by appropriate tests* with continuing neuropathic pain beyond 26 weeks, and severely compromised mobility and, or dexterity.

*diagnosis should be by a non-treating consultant neurologist

The “acute” NFCI definition in paragraph 21 above applies to the Level 14 descriptor while the other two descriptor categories should meet the criteria for the “chronic” definition discussed above in paragraph 23. These definitions should be included in footnote to the Table 2 as well as criteria for neuropathic pain. As with all descriptors in the AFCS tariff those for NFCI and the associated awards take account of recognised psychological consequences of NFCI short of a discrete diagnosable disorder.

References:

(1) The non-freezing cold injury (NFCI) review group report on Non Freezing Cold Injury. London MOD: 2013.

(2) Report on the Health of the Armed Forces. London MOD: 2014.

(3) Oakley, E.H.N. Results of an audit of cases seen in the INM Cold Injury Clinic in the 16 months to January 2014. London MOD: 2014.

(4) Craig, R.P. A paper on NFCI for the Medical Advisory Committee. London the Royal British Legion; 2013

(5) Themistocleous A.C. et al The clinical approach to small fibre neuropathy and painful channelopathy. Pract Neurol 2014; 14: 368-379.

(6) Lauria, G. et al et al Intraepiderrmal nerve fiber density at the distal leg: a worldwide normative reference study. J Peripher Nerv Syst 2010; 15: 202-207

(7) Lauria, G. et al Skin biopsy as a diagnostic tool in peripheral neuropathy. Nat Clin Pr Neurol 2007; 3: 546- 557

(8) Tesfaye, S. et al Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity and treatments. Diabetes Care 2010; 33: 2285-93

(9) Lauria, G. Small fibre neuropathies. Current Opinion in Neurology 2005; 18: 591-597

(10) Scadding, J.W. Neuropathic Pain. In: Brain's Diseases of the Nervous System. M Donaghy. (ed) Twelfth edition. Oxford OUP. Chapter 17, pp 453-475. 2009

(11) Treede, R.D. et al Redefinition of neuropathic pain and a grading system for clinical use: consensus statement on clinical and research diagnostic criteria. Neurology 2008; 70: 1630–5

(12) Jensen, M.P. et al The impact of neuropathic pain on health related quality of life. Neurology 2007; 68: 1178-82

(13) Radat, F. et al Psychiatric co-morbidities in patients with chronic peripheral neuropathic pain: A multicentre cohort study. European Journal of Pain 2013; 17: 1532-2149

(14) Ungley, C.C.B. et al The Immersion Foot Syndrome. Brit J Surg 1945; 33: 17-31

(15) Burgess, J. et al Retrospective analysis of the ethnic origins of male British army soldiers with peripheral cold weather injury. J R Army Med Corps 2009; 155(1); 11-15