IMEG

The Independent Medical Expert Group

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

17 May 2013

Topic 3 – Recognised Diseases

Introduction

1. Lord Boyce in his review of the AFCS raised the issue that while under the War Pensions scheme the majority of medical discharge cases suffering from physical disorders receive entitlement to war pension; this is not the case under the AFCS. This is a reflection of the different standards of proof required in the two schemes. The standard of proof in AFCS is 'on the balance of probabilities' (or 'more likely than not'), which is the standard of proof in both civil compensation and the statutory compensation scheme for civilian occupational injury and disease, the Industrial Injuries Scheme.

2. At its inception in 1917, the standard of proof used in the War Pensions Scheme was "on the balance of probabilities". This was changed in 1943, at the height of the Second World War, when for injuries and disorders arising in service, the burden of proof, transferred to MoD to demonstrate that a service cause was "beyond reasonable doubt" not the cause of the disease. The change was introduced at this time because inadequate record keeping was leading to large numbers of claimants unfairly not receiving compensation.

3. In his report, Lord Boyce proposed that the IMEG should develop a list of Recognised Diseases for the AFCS. By this he meant that IMEG should review the medical literature and receive evidence from experts to provide guidance about the circumstances when "on the balance of probabilities", a disease having onset in or around service was more likely than not to be attributable to service in the Armed Forces.

4. The normal burden of proof in civil compensation and other statutory compensation schemes such as the Industrial Injuries Disablement Benefit (IIDB) Scheme is "on the balance of probabilities". For claims under AFCS this implies demonstrating that military service is more likely than not (more than 50:50) the predominant cause of the injury or disease in the individual case. In the Industrial Injuries Disablement Benefit Scheme, for those conditions where there is sufficient evidence that this level of proof is satisfied, the disease is 'prescribed', i.e. attributable in the individual case to the particular cause in relation to clearly specified circumstances of exposure.

5. In the individual case, attribution is usually based on sufficient evidence to answer the questions:

- Does the particular agent or exposure cause the disease, at least in some circumstances?
- If so, were the circumstances of the individual case such that the agent or exposure is more likely than not to have been the cause of the disease?

6. Recognition of a particular agent as the cause of a disease, and attribution in the individual case, is most clear when the cause is specific to the disease, or nearly so, and the probability of causation is high. Such conditions are now relatively uncommon but a relevant example is

occupational asthma, asthma whose primary cause is an agent inhaled at work. The majority of cases of occupational asthma are due to the development of an allergic reaction to the specific cause encountered in the workplace (e.g. flour in a baker). Asthma develops after an initial symptom-free period of exposure and recurs on re-exposure to the specific cause, in concentrations which do not cause respiratory symptoms in others similarly exposed or previously in the affected individual. Inhalation testing with the specific agent will provoke an asthmatic reaction in the sensitised individual (but not in others not sensitised). Also, for many agents evidence of a specific immunological reaction (i.e. specific IgE antibody) will be found. In principle, the specific cause of asthma can be demonstrated in the individual case.

7. The majority of diseases however are not specific to a particular cause. A particular cause may increase the frequency of occurrence of a disease, which can have other recognised causes. As an example, lung cancer is well known to be caused by smoking cigarettes. More than 90% of cases in the general population occur in cigarette smokers. A smoker of 20 cigarettes a day during adult life will increase his or her chances of developing lung cancer by some twenty-fold. In the case of lung cancer in a smoker of 20 cigarettes a day for 40 years we can say with confidence that it is likely that the lung cancer is attributable to the smoking of cigarettes.

8. However, there are also other causes of lung cancer, such as asbestos and ionising radiation. When are we entitled to attribute lung cancer in an individual to asbestos exposure? The lung cancer caused by asbestos is indistinguishable from a lung cancer of other cause, such as smoking, so it has no specific distinguishing features. We have to ask the question: in what circumstances would it be more likely than not that the lung cancer was caused by exposure to asbestos. As the individual case has no distinguishing (or specific) features, we have to look at populations of people exposed in their work to asbestos. Among these, are there any circumstances where the frequency of the disease has increased sufficiently to make it more likely than not in the individual case that the lung cancer would be unlikely to have occurred in the absence of occupational exposure to asbestos? The answer is that, among other circumstances, the frequency (or incidence) of lung cancer was more than doubled in asbestos textile workers, both smokers and non smokers, who worked for 20 years or more in an asbestos textile factory. In these circumstances we can conclude it is more likely than not the lung cancer is attributable to asbestos).

9. Why is a greater than doubling in the frequency of the disease so critical in determining attribution to a particular cause? We can consider a hypothetical 100 men working in a particular occupation (fig 1). Among these 100 men, as in the general population, the number of new cases of a particular disease is 10 each year, i.e. no different.

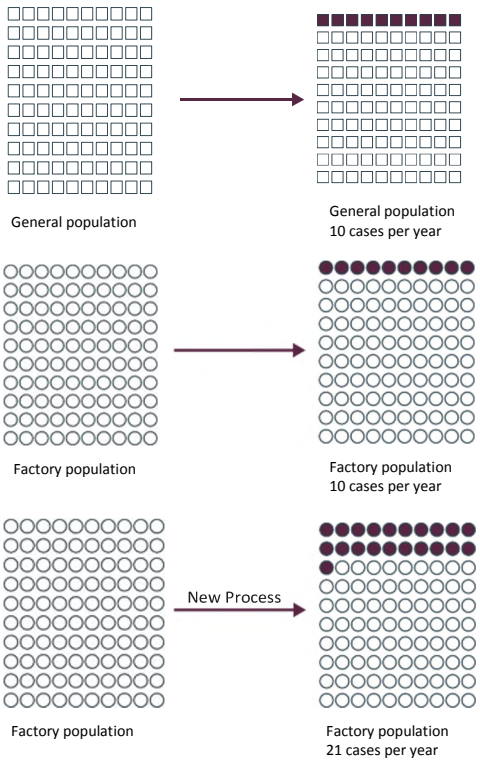

Fig 1. Increased incidence of disease from ten per year to 21 per year in factory population following the introduction of a new process.

Sometime later, after the introduction of a new process, the number of cases of the disease in these 100 men increases to 21 each year, i.e. more than 2 times the previous frequency. We cannot distinguish the additional 11 cases from the 10 in whom the disease would otherwise have occurred. What we can say is that in any particular individual among the 21 cases, there is a more than 50:50 chance, or a greater than doubling of risk, that the disease would not have occurred without exposure to the particular cause. On the balance of probabilities it is therefore more likely than not that the disease is attributable to the particular cause in the individual case. We can say that 'but for' his working in this factory it is unlikely the man would have developed the disease. The balance of probabilities has shifted to 'more likely than not' and in this circumstance the disease can be attributed to the particular cause.

10. In the case of Recognised Diseases in the AFCS, we are therefore looking for evidence that service in the Armed Forces is consistently associated with an increase in the frequency of a particular disease or illness and whether there are circumstances where the frequency is more than doubled, making it more likely than not in the individual case that the disease was attributable to a cause in service.

11. It is also important to distinguish "all or none" diseases from "more or less" diseases. A well-recognised "all or none" physiological condition is pregnancy: one cannot be a bit pregnant. In contrast, many important conditions including high blood pressure, hearing loss and mental health disorders are 'more or less' conditions. These have a continuum of frequency of symptoms without a clear distinction between those with and without the condition. The definition of disease is therefore less clear and subject to expert opinion

12. The epidemiological evidence informing these determinations should be of high quality, drawn from several independent studies and sufficiently consistent and robust that further research at a later date would be unlikely to overturn it.

A. Epilepsy

Clinical issues

1. The prevalence of epilepsy is about 5-10 per 1000 in the UK with an incidence of about 80 per 100,000 per annum. The risk is higher in childhood/young adulthood and in older age. In a general practice-based study about 60% of patients with epilepsy had tonic-clonic seizures, with 12% each, complex and mixed partial seizure types (1). Other types are uncommon.

2. An epileptic seizure occurs when there is an abnormal and excessive synchronised discharge of a set of cerebral cortical neurones. Epilepsy is a condition in which the sufferer is prone to recurrent unprovoked epileptic seizures. In clinical practice, epilepsy is said to be present when two or more attacks have occurred. Single seizures in people without epilepsy can be caused by the same trigger factors that may cause seizures in those with epilepsy.

3. Because of the complexity and tendency for seizures of different types to occur together, seizures and epileptic syndromes are difficult to define and no single classification system is ideal. The WHO and International League Against Epilepsy (ILAE) classification (1989) is most widely used for clinical epidemiological and research purposes (2).

   Clinically, epileptic seizures may be classified under four broad headings:

   a) Simple partial seizures are usually brief and intense, with a variety of symptoms including focal motor, autonomic (flushing, sweating, vomiting), somatosensory, special sensory or psychic depending on the site of the epileptic discharge. Consciousness is preserved throughout the attack.

   b) Complex partial seizures give rise to the same symptoms but by definition there is also always impairment of consciousness. There may be automatic behaviour and reactive automatisms during complex partial seizures. The source of 60-70% of all simple and complex partial seizures is the temporal lobe, giving rise to the typical aura and motor symptoms.

   c) Secondarily generalised seizures are partial seizures in which the epileptic discharge spreads to both cerebral hemispheres, resulting in a generalised seizure, most often of tonic-clonic type.

   d) In primary generalised seizures (Idiopathic Generalised Epilepsy) there is bilateral

involvement of the cerebral cortex at the onset of the seizure. Consciousness is lost at the start of the fit and so there is usually no warning of an attack. Generalised tonic-clonic fits (also known as grand mal) may occasionally be associated with a prodrome of malaise, usually brief, but no specific symptoms. In some types of epilepsy, an increasing frequency of another generalised seizure type, including myoclonic jerks or absences, may herald a tonic-clonic seizure. Absence attacks (petit mal), associated with a characteristic EEG pattern, present in childhood or early adolescence and only very rarely for the first time in adult life

Causes of epilepsy

4. Epilepsy is always a symptom of an underlying brain disorder and may present years or even decades after the development of the causal lesion. Causes of epilepsy in childhood are not considered further here. There are many causes of adult onset epilepsy (3)(4). Idiopathic epilepsy and epilepsy resulting from birth injury and structural abnormalities including hippocampal sclerosis and neuronal migration defects may all have onset in adult life. Other common causes include head injury; alcohol and drug abuse; infections including meningitis, encephalitis, pyogenic cerebral abscess, and toxoplasmosis in immunocompromised patients; cerebrovascular disease (including vascular malformations); and brain tumours. In some parts of the world, malaria and cysticercosis need to be considered as possible causes.

5. Head injury is an important cause in military populations; post-traumatic epilepsy is more likely in people with family history of epilepsy. Brain tumours account for the development of seizures in about 30% of adults aged 30-50 years. Multiple sclerosis is an occasional cause of fits, and seizures sometimes occur in degenerative conditions such as Alzheimer's disease. With increasing access to specialist clinics and high quality MRI, an underlying cause for epilepsy can be determined in about 50% of patients, and this percentage is likely to increase.

6. In terms of genetic contribution, there are two broad groups (5). In one group, epilepsy is associated with developmental brain abnormality and other neurological or cognitive difficulties. This includes some well defined syndromes, and is a form of symptomatic epilepsy. Genetic and epigenetic causes, but without identifiable gross brain abnormality, are also the main explanation for the group of epilepsies usually referred to as idiopathic or cryptogenic.

7. Post-traumatic epilepsy may follow head injuries and takes the form of focal or generalised seizures (6). It is more common in societies with a higher rate of personal violence and in military personnel who are injured in action

- Some patients may have only a single seizure, but in a group of patients who had a single late (>1 week after injury) seizure, 86% had another within 4 years (6). After a severe head injury, the risk of onset of recurrent seizures is known to be increased for at least 10 years (7).

- The risk is related to the severity of the injury. After mild blunt injuries with only a brief loss of consciousness, the risk is only slightly greater than in a control population (8)

- With moderate injury (fractured skull and/or unconsciousness lasting between 30 minutes and 24 hours) the risk is approximately double that in a control group at 2% at 5 years, 2.5% at 10 years and 3% at 20 years. (9).

- When the injury has been severe, with cerebral contusion, intracranial laceration and/or unconsciousness lasting more than 24 hours, the risk is much higher, at some 6% at 1 year, 10% at 5 years and 16% at 10 years. (9).

- With penetrating head injuries, 50% of patients have active epilepsy after 15 years. (9)

- Overall, the Relative Risk (RR)[1]* of developing epilepsy after head injury falls with time, being 12.7 after 1 year, 4.5 up to 5 years and 1.4 after 10 seizure-free years.

8. In people with epilepsy, many factors can precipitate seizures (3). These include electrolyte disturbances (sodium, potassium, calcium, magnesium), toxins (particularly alcohol), and therapeutic and recreational drugs (including tricyclic antidepressants, anti-psychotics, anti-cholinergics, anti-histamines, methylxanthines, cocaine, ecstasy, amphetamines, some antibiotics; and withdrawal from barbiturates, benzodiazepines and therapeutic antiepileptic drugs).

9. Seizures may also be induced by other metabolic disturbances such as hypoglycaemia, hypoxia and ischaemia. Sleep deprivation is a powerful precipitant in many patients. Major systemic diseases, including renal and hepatic failure and porphyria, may also cause seizures. In some women, there is a link with phases of the menstrual cycle, fits tending to occur more frequently in the days preceding menstruation (catamenial epilepsy).

10. Photosensitive epilepsy is the most common form of reflex epilepsy, accounting for 0.5-8.0% of patients with epilepsy in different reported series. Patients experiencing photosensitive fits usually have idiopathic generalised epilepsy. Attacks may be triggered by flashing lights, including flickering television screens. The great majority of patients with photosensitive epilepsy present to medical attention during childhood and adolescence. Other reflex epilepsies, including reading, writing, eating and musicogenic epilepsies are very rare.

11. Non-specific psychological stress is often cited as a precipitant for seizures. However, objective data have failed to show on the balance of probabilities that this is an independent causal factor.

12. Whilst fever is a common cause of fits in childhood (febrile seizures between the ages of 6 months and 6 years), fever as a cause of fits per se in adults has not been clearly established. Transient fever and peripheral blood leucocytosis (and indeed, a rise in the white cell count in cerebrospinal fluid) are common after tonic-clonic seizures. Thus fever is frequently a consequence rather than a cause of fits in adults.

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[1]* **Relative Risk (RR) is the ratio of risk, usually expressed as disease incidence in exposed and unexposed populations. In this case, the ratio of the incidence of epilepsy in those who have had a head injury to the incidence in those who have not.**

13. Some patients have tonic-clonic seizures only during sleep (sleep epilepsy). Sleep may enhance focal epileptogenic discharges, and tonic-clonic seizures limited to sleep in adults should be regarded as being likely to have a focal, partial onset. In patients in whom a pattern of fits occurring only during sleep has been established, the risk of fits occurring while awake is only 13% over 6 years (3). Seizures occurring shortly after waking (awakening seizures) are common in the idiopathic generalised epilepsies, usually presenting in childhood. Fits in patients with these epilepsies are also particularly likely to occur with sleep deprivation or sudden arousal from sleep (3).

Specific precipitants

Shift work

14. There is little research evidence that shift work causes an increase in fit frequency in those with epilepsy. However, sleep deprivation can provoke seizures in some people with epilepsy, and night shift working undoubtedly changes sleep patterns and reduces overall sleeping. Shift work is best avoided in those with epilepsy.

Alcohol

15. Alcohol misuse is strongly associated with increased risk of epileptic seizures. With binge drinking, seizures usually relate to alcohol withdrawal, but may also occur as a direct toxic effect, or due to associated causes including hypoglycaemia and head injury. Seizures frequently occur in chronic alcohol abuse. In people with epilepsy from another cause, there may be an increased propensity to alcohol-induced seizures.

Photosensitivity and visual display equipment

16. Reflex epilepsy is the term applied to epilepsy in relation to specific precipitants. As mentioned above, photosensitive epilepsy is relatively rare in adults and occurs more frequently in women.

Epilepsy and work in the military

17. The Equality Act (2010) means that the majority of civilian jobs are, or can be made, suitable for people with epilepsy. Consideration of whether a person with epilepsy should enter a particular employment or, where there is onset in work, whether they should be retained in employment is a matter of individual facts, hazards and risks integral to the job, scope for adjustment and the medical evidence. Driving, especially of HGV and PSV, is one of the few activities for which there is a statutory bar.

18. The Act does not apply to the military. People with proven epilepsy or who have suffered a single seizure less than four years before entry cannot enlist. An individual who suffers a single seizure while in service will be downgraded with restricted duties. He will be investigated and observed for 18 months. If by that time there have been no further fits, and following consultant neurological and occupational physician opinion, he may be reinstated. Aircrew who suffer a single fit are grounded permanently, and where aircrew personnel have more than one fit they will be medically discharged.

Epilepsy as a recognised diseases in the AFCS

19. Table 6 of the tariff is headed Neurological Disorders including spinal, head and brain injury. A footnote confirms that awards for brain injury paid at Levels 1, 2 and 4 include compensation for associated epilepsy, recognising the high risk that head injuries of such severity will have associated epilepsy. Item 29 Table 6, AFCS Order 2011, provides for other cases of post-head injury epilepsy. Where post-head injury epilepsy is uncontrolled, an additional award is payable (Item 15 Table 6 of the Order 2011). Epilepsy may also occur as a possibility rather than a probability, following meningitis or encephalitis or in relation to a tumour. Where that occurs and the primary disorder is accepted as due to service after 6 April 2005 and an award paid, an additional award for epilepsy will be considered.

20. Where a first fit is precipitated by one of the factors noted above, e.g. sleep deprivation, due to service, or in relation to a therapeutic drug dispensed by or on behalf of Defence Medical Services, the fit will be accepted as due to service. If the individual then has a second fit, the position in relation to AFCS compensation for the epilepsy will depend on the case specific facts. If recurrent fits are clearly due to sleep deprivation due to service, an award is likely, but most such patients will go on to have fits unrelated to sleep deprivation, proving to have Idiopathic Generalised Epilepsy. In other words, constitutionally they have a low epileptic threshold. In this latter circumstance no award is payable. Therapeutic drug-related seizures are usually one-off, and a second clearly drug related seizure, either to the same or another drug, should also be regarded as a single seizure (i.e. not epilepsy). Where such a pattern arises in service and the therapeutic drug has been administered by, or on behalf of, Defence Medical Services, two awards, each for a single fit, may be appropriate. The legislation provides (Article 12 AFCS Order 2011) that recurrent seizures related to alcohol or other recreational drugs are not compensated under the AFCS.

21. Because of the restrictions on military employability, circumstances where worsening of epilepsy by service, after 6 April 2005 might arise will be very rare. Where the case specific facts meet the terms of Article 9 AFCS Order 2011, worsening may be accepted. Potentially, this might arise where an individual has an initial fit more than four years before service entry, is allowed to enlist and then experiences a further fit in service, satisfying the diagnosis of epilepsy. In addition, the fit in-service must have been precipitated by a service related factor.

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B. Meningitis and Encephalitis

Clinical Issues

1. Meningitis is inflammation of the meninges. Encephalitis is diffuse inflammation of the brain, which may have a lobar, multifocal or diffuse distribution, reflected in the presenting clinical features. When they occur together they are termed meningo-encephalitis. Most cases of these disorders are caused by infection entering the body through the upper respiratory tract, the gastro-intestinal tract, the skin or at the site of trauma. When organisms infect previously healthy adults through these routes they usually affect only the tissues at and around their site of entry or, in a few cases, cause systemic illness. It is rare for them to directly invade the central nervous system. Infecting organisms are a necessary cause of these disorders in that without them the disorder will not occur. However by themselves, they are not a sufficient cause. Factors increasing the risk of disease include immune compromise and poor general health. Other factors as yet unknown also increase the risk, as cases of meningitis and encephalitis frequently occur in young previously healthy individuals.

2. Exposure to an infecting micro-organism, which may be a bacterium, virus or of other type, e.g. Rickettsia or protozoan, is a necessary cause of these disorders. These organisms are ubiquitous and the disorders occur in civilian as well as military populations. In respect of infectious disorders the aim of the AFCS is to pay benefit where on the balance of probabilities service on or after 6 April 2005 has increased the risk of an illness beyond that of the general UK civilian community. Reflecting that aim, Article 12 of the 2011 AFCS Order includes a provision which excludes payment of benefit for injury or death in certain circumstances. These include the following in relation to infections:

- no award is payable where injury or death is due to an illness which is an endogenous infection or
- an exogenous infection except where:
  - the infection is acquired in a non-temperate region and the person infected has been exposed to the infection in the course of service; or
  - where, in a temperate region, there has been an outbreak of the infection in service accommodation or a workplace.

Endogenous infections are taken to be those having origin in the person themselves e.g. appendicitis, in which the infecting organism is a normal gut commensal bacterium. By contrast, exogenous infections enter the body from outside.

3. Acute bacterial meningitis usually occurs sporadically in Western countries (1-5 per 100,000 per year) (1) but can also occur in epidemics, as it does commonly, in some parts

of the world, e.g. sub-Saharan Africa. In the UK, small outbreaks of meningococcal meningitis are well-recognised. Bacterial meningitis occurs sporadically as a complication of infection elsewhere in the body, following trauma to the skull or spine or in relation to neurosurgery, e.g. insertion of cerebrospinal fluid (CSF) shunts and drains. The organisms most commonly involved are Neisseria meningitidis (2) or Streptococcus pneumoniae. Both spread from the respiratory tract. Less commonly, Staphylococcal aureus, Listeria monocytogenes, Escherichia coli and Haemophilus influenzae are the infecting organisms. Meningitis following injury to the skull or spinal column usually occurs within a few weeks of the trauma but cases with intervals longer than 20 years post-trauma have been reported (3). Recurrent bacterial meningitis is uncommon and usually related to an anatomical defect which may be congenital, e.g. dermal sinus, or related to skull trauma (4).

4. Mycobacterial meningitis, most commonly due to Mycobacterium tuberculosis, is a life-threatening infection that is more common in some parts of the world but may occur in isolated cases anywhere. Treatment has to be continued for a year or longer, and infection with drug resistant organisms is an increasingly common problem.

5. Article 5 of AFCS 2011 Order provides that a descriptor is intended to cover the expected effects of the primary injury and its appropriate clinical management. In relation to surgery, post-operative pain and subsequent scarring are virtually inevitable consequences, and so they are taken into account in the primary descriptor and associated award. On the other hand, development of bacterial meningitis or encephalitis is not to be expected. They are possible, but not probable consequences and so where they do occur as result of service, a separate additional award would be payable.

6. Aseptic meningitis refers to meningitis not due to bacteria. Causes may include spirochaetes, fungi, parasites or viruses. Therapeutic drugs can occasionally produce a similar clinical picture (5) and it can occur in relation to cancer or leukaemia (malignant meningitis) (6). Viral meningitis is more common than bacterial meningitis in Western countries. Viruses usually invade the meninges via the blood stream. Many viruses are capable of causing meningitis by a variety of modes of transmission, and the clinical course is consequently variable. In some cases viral meningitis follows a flu-like illness, while in others, clinical onset is acute or sub-acute, with headache, vomiting and painful eye movements. Neck stiffness may be absent in mild cases and is usually less severe than in bacterial meningitis. Tuberculous meningitis is usually insidious in onset and is uncommon in young, otherwise healthy adults in Western countries.

7. Viral encephalitis is less common in the UK than meningitis (7). In the UK, herpes simplex virus is the commonest cause. The characteristic clinical features include headache, increasing drowsiness and impairment of consciousness, and focal neurological deficits including dysphasia and hemiparesis, reflecting the frontal and temporal lobe focus of the pathology. Convulsions are also common. In the Far East, Japanese B virus infection is the commonest cause of encephalitis. A wide variety of other viruses may also cause encephalitis, and clues to the possible cause include geographical location (arboviruses); season (arboviruses, enteroviruses); animal bite (rabies); preceding illness or immunisation (e.g. measles, influenza, varicella); other present illness (Herpes zoster, infectious mononucleosis, Mycoplasma pneumoniae, mumps, etc); or an immunocompromised state (HIV, JC virus). Other severe viral encephalitides include those caused by Ebola and West Nile viruses. Viral encephalitis is associated with considerable mortality, and persistent neurological deficit is common in those who recover. Specific drug treatment is available for some causes of viral encephalitis (Herpes Simplex, Herpes Zoster, HIV), but not others. Prevention, by appropriate

immunisation, is possible for some (e.g. Japanese B encephalitis).

8. Viral meningitis may be caused by many different viruses. Mumps virus is the commonest identified cause of viral meningitis in the UK. Viral meningitis has a much better prognosis than viral encephalitis or tuberculous meningitis, and full recovery can be expected in the majority.

UK military immunisation policy

9. The policy on the immunological protection of UK personnel is published in a Joint Service Publication (JSP) 950 leaflet (8). Military immunisation policy normally reflects the UK national immunisation schedule as set out in the department of Health Green Book (9), with some military specific variation. These variations may apply to all service personnel, e.g. the need, because of possible short notice deployment, for all personnel to have in-date yellow fever certificates. Other measures apply to recruits/new service entrants. For example, those without a BCG scar or other evidence of immunisation are offered Mantoux testing and BCG immunisation; and similarly if non-immune, all recruits, new entrants and those transferring to Defence Medical Services are immunised against varicella. Those whose principal service occupation puts them at high risk, e.g. health care staffs, are tested for Hepatitis B seroconversion after a primary course of Hepatitis B immunisation. Specifically in relation to meningitis and encephalitis, all serving personnel aged less than 25 years, all recruits, new service entrants and new members of the Reserve, regardless of age, are offered a single dose of meningococcal conjugate (men C) unless previous immunisation is documented. Immunisation against Japanese B encephalitis is given for deployment to the Far East.

Meningitis and encephalitis as recognised diseases in the AFCS

10. As rare diseases with small absolute numbers of cases and marked annual variation, there is no published evidence on the incidence of meningitis and encephalitis in different occupations, and there are no robust epidemiological studies in military populations. Since the introduction of recruit immunisation against meningococcal disease in 1992, no clusters of central nervous system infections have occurred in UK military communities (10). Following adoption of this policy, while the reduction in disease incidence for recruits post-immunisation was not statistically significant, rates in unimmunised trained personnel decreased. A similar disease pattern and fall in rates amongst unimmunised trained personnel were seen in Norwegian troops during a trial of recruit vaccination (11). These findings suggest that unimmunised recruits may act as an infection source for older trained personnel. Where cases of meningitis do occur in service, the clinical evidence, incubation periods, etc, confirms they are sporadic in nature.

11. Meningitis and encephalitis can be accepted as due to service on balance of probabilities and awards made under the Scheme where meningitis or encephalitis is appropriately diagnosed, the infective agent identified and the incubation-period determined and;

- the illness is part of an outbreak in a military work place or camp anywhere in the world and the affected person lives or works there
- the illness is sporadic and due to an exogenous infection which has been contracted while the person served in a non-temperate zone
- post-traumatic acute bacterial meningitis may follow injury to the skull or spinal column or occur in relation to neurosurgery. Where the primary injury, or injury or illness leading to surgery is due to service, an additional separate award is payable.

12. Because of the nature and pathogenesis of meningitis and encephalitis, worsening by military service is not a relevant concept. In some cases, service may begin before and continues after 6 April 2005, when the AFCS was introduced. In that situation, if the case facts in terms of infecting organism and its incubation period, and timing of clinical presentation confirm that infection pre-dated 6 April 2005 and was due to service, entitlement would be given under the War Pensions Scheme even if the disorder did not present clinically until after 6 April 2005.

13. AFCS tariff descriptors do not make explicit reference to meningitis or encephalitis. It is most likely that any award would meet descriptors in Table 4 or exceptionally, Table 6. To date no claim for meningitis or encephalitis has been made.

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C. Multiple Sclerosis

IMEG consideration of this topic was informed by literature scrutiny, discussion with subject experts and the Chairman, Dr Scadding and Dr Braidwood also had a useful meeting with the Chair of Mutual Support, the armed forces multiple sclerosis support society.

Clinical Issues

1. Multiple Sclerosis (MS) is the most common serious neurological disorder in young people affecting some 80,000 people in the UK, with a prevalence of approximately 1 in 800. Typically it has onset in previously fit young adults and thus new cases are not uncommon in the military population. The causes of MS are not fully understood but it is considered, from familial, genetic and epidemiological studies, to be a disease due to interaction between genetic and environmental factors.

2. MS is a disease of the central nervous system caused by inflammatory demyelination of nerve fibres in the brain, brain stem and spinal cord. Localised areas of inflammation and demyelination arise unpredictably in different parts of the nervous system over time. The usual age of onset is 20 - 40 years with a peak at age 30 years, although it may develop at any age including during childhood. Characteristic sites for lesions causing clinical episodes include optic nerves, brain stem, cerebellum and spinal cord. Three main temporal patterns of the disease are recognised: relapsing-remitting, in which there are punctate relapses, often with partial or complete symptom resolution, particularly in the early stages of the disease; primary progressive, in which there is gradual onset, no remission and gradual worsening of the neurological deficit and development of progressive symptoms attributable to lesions at new sites within the central nervous system; and secondary progressive MS, in which an initial relapsing-remitting pattern is followed by a progressive course. Intervals between relapses are very variable, from weeks to several years. Occasionally, MS runs a benign course, with few, widely spaced relapses, with good recovery from each relapse, occurring over many years, but in the majority, the disease leads to increasing disability.

3. Determination of the timing of onset of MS can be problematic. At the time of presentation with a first episode of neurological disturbance, accountable for clinically by a lesion at a single site in the central nervous system, a diagnosis of MS cannot be made on purely clinical grounds, without additional information from Magnetic Resonance Imaging (MRI). Prior to the introduction of MRI, the diagnosis was made on the basis of further episodes, typical of demyelinating lesions, disseminated in time and within the nervous system, supported by evidence of asymptomatic optic nerve lesions using visual evoked potentials, and the presence of oligoclonal bands in the Cerebrospinal Fluid (CSF) on protein electrophoresis. With the introduction of MRI, a combination of clinical, MRI and CSF criteria can now be employed (1) (2). The need for confident earlier diagnosis has been emphasised by the introduction of disease-modifying treatments in recent years.

4. MRI has also permitted detailed study of prognosis. In prospective investigation of patients presenting with a first demyelinating episode affecting optic nerve, brain stem or spinal cord, 50 - 70% had multiple lesions on T2-weighted MRI, and 82% of these patients went on to have further clinical attacks within the next 10 - 20 years, establishing the diagnosis of clinically definite MS (3) (4). Thus an individual might have had an episode of transient sensory disturbance or optic neuritis, often minor and not always leading to neurological assessment and investigation, years before a second episode that clinically, supported by investigation, clearly indicates that he/she has MS. In retrospect, it can be recognised that the illness began at (or before) the time of the initial clinical episode.

5. Recognition of a relapse, rather than the fluctuation of symptoms (and to some extent signs) that is so commonly experienced by those with MS can sometimes be difficult. It is straightforward when there are new symptoms related to a lesion in a previously unaffected part of the nervous system and more difficult when there is exacerbation of existing symptoms/deficits. For example, fatigue, so common in MS and exacerbated in many patients by exertion or overdoing things, can lead to presentation with considerable transient worsening of symptoms (e.g. of a paraparesis) that can mimic a relapse, though such exacerbations usually last for less than 24 hours and, by definition, a relapse lasts for at least 24 hours. In many cases it is difficult to tell acutely, and even if there is recovery to an objective level that is much the same as the pre-deterioration state, it can be hard in some patients, even in retrospect, to be completely certain as to whether or not a relapse has occurred.

6. The factors that can lead to temporary exacerbation, mimicking relapse, include hot weather, fever from intercurrent illness, and other environmental increase in temperature, e.g. hot baths or working in hot environments, as part of military duties. Likewise, lack of sleep, physical exertion or fatigue may be identified by patients as causing a relapse or exacerbating their illness.

7. Quite apart from these factors, many patients report wide fluctuation in the severity of their symptoms, without obvious external provocation. Investigation, for example with MRI, is often unhelpful in this situation. There is frequently a mismatch between neurological symptoms and signs and MRI findings. This is largely because the easily seen plaques of demyelination are present in the cerebral hemispheres (subcortical white matter and corpus callosum), where they are often asymptomatic, whereas the common neurological deficit-producing lesions in the brain stem, cerebellum (to some extent) and spinal cord are less easily demonstrable, though with high quality MRI, lesions at these sites can be seen. MRI is most useful as an investigation to support or establish the diagnosis of MS, and less useful in the later stages of the disease.

8. In conclusion, it can be hard to be definite about whether or not a relapse has occurred, particularly in the setting of pre-existing deficits. Many patients report relapses occurring at non-stressful times, noting that during a previous stressful period (of one type or another), they remained well, when they had expected that the stressful circumstances might have caused a relapse. There is, of course, a natural tendency to relate the development or exacerbation of all diseases to identifiable life events, and MS

is no exception to this rule.

Causation and attribution to occupation

9. There is no evidence that MS is uniquely occupational and in military personnel, it is clinically indistinguishable from the disorder as it occurs in the wider population. Literature scrutiny identifies no published papers on MS incidence in any occupational groups including the military, in comparison to the incidence in the general population. The evidence suggests that MS is an autoimmune disorder whose incidence is influenced by genetic and racial predisposition, family history and migration; e.g. the risk is generally low for black and white South Africans but increases for white English speakers who migrate to South Africa as adults rather than children (5). Work on birth order looking at the proposal that the disorder is more common amongst first-borns has produced conflicting results (6). Risk of developing MS is affected by gender and hormonal balance. It is more common in women, and in women with MS relapse rate is influenced by pregnancy. There is reduced risk of relapse as pregnancy proceeds while in the puerperium there is, conversely, a twofold higher risk of relapse (7).

Possible exposures/circumstances for investigation

10. The pattern of disease suggests that MS is triggered by an environmental factor in individuals who are genetically susceptible. Potential environmental agents have been the subject of much speculation and research of varied quality and study design. Infection, diet, toxic chemicals, soil constituents, head injury and physical and psychological trauma have all been investigated.

11. Infection. An infectious agent has long been suggested as a trigger for MS and relapses are reported following upper respiratory infection. (8). In support of the infective hypothesis is the varying disease frequency in Caucasian populations, dependent on latitude and migration and observed case clustering in small communities. Some studies have indicated an increase in the risk of relapse following systemic infection, together with evidence of increased lesion activity on MRI. These infections include bacterial urinary tract infection and viral respiratory or gastrointestinal infection (9) (10). However, despite a substantial body of research, some of high quality, investigating specific infections including measles, mumps, EB virus, herpes simplex, rubella, varicella, adenoviruses, Chlamydia and mycoplasma, no direct causal relationship has been established between infection with any of these microbes and onset or exacerbation of MS (11). There is some evidence that the total number of childhood infections before age 7 years is higher in MS sufferers than in controls (12).

12. Immunisation. Clinical onset and relapse of MS after immunisation for various diseases have been reported (13) especially in respect of Hepatitis B. Subsequent epidemiological studies did not confirm this and the US Institute of Medicine has concluded that current evidence does not support a causal link between commonly administered immunisations including those for tetanus, (alone or combined with polio and diphtheria), polio, influenza, hepatitis B, varicella, BCG and MS onset or relapse (14) (15). For some infections there is currently insufficient evidence on which to base recommendations for immunisation (e.g. human papilloma virus, rabies). A recent small study showed an

increase in rate of relapse following immunisation for yellow fever (16), and this is probably best avoided. Most neurologists recommend avoidance of immunisation during periods of disease activity (i.e. during or shortly after relapse), and there are reasonable grounds for the avoidance of administration of live attenuated vaccines to patients receiving immunosuppressive or immune-modulating treatments (16).

13. Toxic chemicals and metals. The fact that MS is common and occurs sporadically in previously fit young adults has led to speculation, case reports etc. looking at possible neurotoxicity of a range of substances including organic solvents, trichloroethylene, lead, mercury and zinc. However there is no reliable evidence of an association (17) (18).

14. Diet. Numerous studies have investigated the role of lifestyle including diet e.g. junk food, synthetic additives, absence or preponderance of dairy products, food contaminants and allergy. There are several strands of evidence for a possible role for vitamin D deficiency as a causative factor for MS. MS frequency increases with increasing latitude, which is strongly correlated with exposure to Ultraviolet type B radiation (UVB) from sunlight and vitamin D concentration; the prevalence of MS at high latitudes is lower than expected in populations with high consumption of vitamin D-rich fatty fish; and MS risk decreases with migration from high to low latitudes (19). The possibility that vitamin D supplementation might decrease MS rates in vulnerable populations is currently under investigation. In patients with established MS, a single small controlled trial failed to demonstrate any benefit of vitamin D treatment (20). For other dietary constituents, there is no compelling evidence to support a dietary link with the cause or course of the disease.

15. Climate. Although it is well recognised that a rise in body temperature may temporarily exacerbate symptoms attributable to demyelinating lesions in the nervous system (see paragraph 6 and 11) there is no evidence that living in hot climates, or heat stress illness, either causes MS or induces relapse of established disease.

16. Physical trauma. Previous suggestions that physical trauma causes or exacerbates MS have not been borne out by careful epidemiological studies (21). The hypothesis that physical trauma contributes to the genesis of the disease is based on the observation that physical injury causes breakdown of the blood-brain barrier and auto-reactive lymphocytes then cause demyelination and neuronal loss or damage. The barrier breakdown lasts some 2 - 12 weeks, so exacerbation or onset is likely to relate to that time frame. Evidence supportive of an association between physical trauma to head and neck and MS onset or exacerbation is predominantly based on animal models supported by case reports and case control series. However, overall current evidence, including literature review (22) and prospective studies (23), does not support a causal role for physical injury in causation or exacerbation of MS. There is no consistent evidence concerning surgical trauma and disease activity. There is also MRI evidence demonstrating a lack of correlation of the site of MS plaques within the cervical spinal cord and levels of compression resulting from cervical spondylosis (24).

17. Psychological stress. There are particular challenges in considering psychological stress. A substantial amount of research has been undertaken to attempt to clarify the role of mental stressors in the aetiology of a wide variety of diseases. Methodological difficulties and limitations include the following (25):

a) Self-reporting. Mental stressors have different effects in different individuals, and assessment of the severity of the stress experienced inevitably requires some form of self-reporting, based on a simple description of feelings or on a questionnaire applied by an experienced interviewer. While the latter is more reliable, complete elimination of subjective bias is not possible. People who suspect that their medical condition is due to mental stress are more liable to have examined their past experiences for stress-related symptoms than individuals who remain healthy. Finally, remembering events can be difficult enough, remembering the way one felt at various times in the past can be much harder.

b) Multiple 'confounding' factors. The very complexity of the factors that can affect the way an individual reacts to stress, including constitution and personality, coping strategies, social support and behaviour and lifestyle change, means that studies that examine associations between stress and disease are at risk of confounding by these other factors, which may be independently associated with the disease. Even where studies attempt to neutralise such confounding in the analysis of findings, this can be only partially achieved, since constitution, personality, coping strategies, behaviour and lifestyle are all unique to each individual and cannot readily be statistically separated out.

Use of the term 'confounding' suggests that various factors may be involved and operate independently of each other. In some cases factors influence disease aetiology through working in combination and modifying individual influence. This makes assessment of the effect of one factor in isolation, such as mental stress, very difficult. One study showed that a group who experienced stress at work also showed features of anger, hostility, depression, anxiety and social isolation (26).

An individual's whole lifetime behaviour and lifestyle with regard to diet, exercise, smoking, and consumption of alcohol and other drugs can have an important influence on the development of disease. Such behaviour is unique to the individual and can vary from day to day and according to mood, making attempts to classify behaviour types over the whole period of a study only approximate at best. Changes in behaviour and lifestyle may occur due to mental stress and many other factors, and in any case will be self-reported.

c) Association, cause, and effect. Where studies detect an association between a stressor and a medical condition, considerable care must be taken before concluding that cause and effect have been established. The association may operate through an underlying factor common to both elements of the association. Stress may be associated with a particular medical condition because the individual is constitutionally prone not only to the condition but also to a tendency to feel stressed. Also, even if a causal link were established, which element is the cause and which the effect would remain to be proven. Depression may be associated with cardiovascular disease (CVD) (27), but does depression cause CVD, does having CVD give rise to depression, or are depression and CVD both independently due to some other cause, perhaps of a constitutional nature? All are questions which challenge both clinician and researcher.

d) Defining mental stressors. Acute mental stressors are usually dramatic and easily recognised by others, so studies involving their effects can adopt firm definitions without difficulty. The effects of chronic mental stressors, on the other hand, vary substantially from individual to individual. An experience which may be stressful to one person can be constructively challenging to another. Therefore, defining any problem, activity, or event as a chronic mental stressor can never be absolute. Studies of potential chronic mental stressors suffer an inherent weakness of definition from the outset.

e) Study design. Many of the studies into the association of mental stress with physical disease attempt to address these difficulties, but few, if any, fully succeed. Most are cross-sectional and retrospective, at least in terms of recall of the stressor experience, whereas establishment of mental stress as a cause of development of pathological change requires prospective, longitudinal cohort studies of considerable duration, incorporating strictly matched controls throughout. Those studies that do perform follow-up assessments and do so at infrequent intervals will inevitably fail to account adequately for fluctuations in stress levels, behaviour and lifestyle over the whole study period. The many limitations already described render completely conclusive studies of this topic extremely difficult.

18. MS was described in the mid 19th century and its possible association with emotional stress first suggested by Charcot in 1872 (28). This was followed in quick succession by a series of supportive case reports .In 1958 (29) and 1970 (30) case studies were published concluding that psychologically stressful situations immediately preceded the onset of MS in 35 out of 40 and 28 out of 32 patients respectively.

19. Since that date many more studies have been published. These are of varied design, including some case control (31) and some prospective studies looking both at stress and onset of MS (32) and as a trigger for relapses (33). Although the longitudinal studies involved retrospective recall of stressor episodes, they suggested some association between stress and MS, particularly low grade chronic domestic or workplace stress.

20. An Israeli study in 1993 (34) came to a different conclusion. It considered disease exacerbations using SCUD missile attacks during the 1990/91 Gulf War as the stressor. The study was small with only 32 patients. The number of relapses during the war and in the following two months was reported as significantly lower than expected based on the frequency during the preceding two years. In other words, these severe life-threatening stressor events seemed to protect the patients from exacerbations, for at least a period. In addition to the small sample size, other factors which we need to bear in mind in relation to the study are: the short follow-up period; the effect of different stressors; and variable impact of trauma on different samples because of different expectations of, and preparation for, trauma, such as missile attacks. Military and civilian samples are particularly likely to differ with regard to these factors.

21. Other studies have attempted to consider possible mechanisms of disease where psychological stressors are potential factors. This includes the role of increased

permeability of the blood brain barrier, as well as changes in brain MRI and their relation to clinical signs (35). Another study (36) considered the possible moderating effects of psychological, social and biological factors. The study was clear that previous investigations all had methodological limitations. While it supported a possible correlation between psychological stress and MS, it did not provide clear evidence on balance of probabilities that stressful life events cause or contribute to it.

22. The 33-day Israeli-Lebanese conflict of 2006 led to two further studies of MS exacerbation (37) (38) both providing evidence that exposure to war-related events increased disease activity. A further study indicated a possible association between coping strategies and a reduced relapse rate (39). However, a recent systematic review concluded that stress as a risk factor for MS onset or exacerbation is not yet proven, and again drew attention to methodological issues. These included the heterogeneity of measurement of stress in different studies, and the need to incorporate a multidisciplinary approach to stress measurement and clinical and radiological criteria for MS (40). It is also worth emphasising that all the published research studies on this topic have been conducted in civilian rather than military populations. Furthermore, it should be recalled that UK military personnel known to have MS are judged to be non-deployable on medical grounds and removed from combat situations.

23. Multifactorial causation and prognosis of MS. It is widely acknowledged that MS is due to a combination of genetic and environmental factors (see paragraph 9 above). A question raised in relation to the military is whether the combination of putative stressors, outlined in the preceding paragraphs, might act in an additive or synergistic way to either cause MS or produce relapses/progression of existing disease. If this was the case, and given that physical and psychological stressors are frequently experienced in military life, it might be expected both that the incidence and prevalence of MS would be higher in the military, and that the rate of progression of the disease and degree of disability related to duration of disease might also be greater. Furthermore, one might expect the life expectancy of those affected by MS in the military to be reduced. However, there is no published evidence to support any of these outcomes.

Multiple sclerosis as a recognised disease in the AFCS

24. As outlined above, and despite a large body of research into aetiology, the cause or causes of MS remain unknown. Reported studies are of varying design quality, are rarely prospective, are often small and heavily reliant on patient or family recollection, there may be issues about diagnosis and study findings are inconsistent. As a result, current understanding of the causation of MS does not allow it to be a recognised disease, as defined, in terms of attribution on the balance of probabilities. In terms of service worsening, Article 9 AFCS Order 2011 must be met and service worsening must be the predominant cause of the downgrading and medical discharge. As with other conditions which may be associated with a wide spectrum of disability and where safety of the person or colleagues may be an issue, retention of a person in their principal service occupation or in some other in-service role after diagnosis, depends on individual circumstances. In line with many other chronic illnesses, best clinical advice and occupational health practice seek to keep the person engaged productively, socially connected, and part of a team for as long as possible. Overall evidence is that almost all but the heaviest jobs are good for people's health. The armed forces are committed to high standards of human resource management and occupational

medicine and aim to retain people in service as long as appropriate for both them and the organisation. Care is taken to ensure that working environments, including any modifications are provided, tasks allocated, suitable working hours and patterns arranged and the individual monitored so as to avoid harm to him/her or any possible worsening of his/her disorder. In respect of claims, each case must be looked at on its merits but where medical employability downgrading and medical discharge is an issue for personnel with MS, it is much more likely to be related to the natural progress of the disorder itself than to service worsening.

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D. Asthma

Clinical Issues

1. Asthma is an inflammatory condition of the bronchial (conducting) airways of the lung characterised clinically by reversible airways obstruction and hyper-responsiveness of the air passages. Symptoms are episodic and improve between attacks although in chronic or inadequately treated asthma a variable degree of airways obstruction may persist (1). The disorder commonly starts in childhood. It is estimated to affect 7 - 10% of adults in UK with some having asthma persisting from childhood and others developing it as adults without a previous history. Childhood asthma may improve in adolescence but relapse in later life (2).

2. Asthma is considered to be due to a combination of genetic and environmental factors. It probably occurs when a person with genetic pre-disposition encounters an

environmental factor that induces airways inflammation and hyper-responsiveness. Children of parents with asthma, who are atopic or have allergies, are more likely to develop asthma.

3. Although the boundaries between them can overlap it is helpful to distinguish:

Initiators: Agents which initiate asthma, i.e. an identifiable primary cause, in those previously without it, by causing airway inflammation and increased airway responsiveness to non-specific stimuli e.g. exercise, cold air. The best characterised initiators of asthma are the causes of occupational asthma, where a specific agent encountered in the workplace causes asthma in an individual previously without it.

Inducers: Agents which on re-exposure in a patient with asthma increase airway inflammation and airway responsiveness. This includes both agents which can initiate asthma (e.g. inhaled flour in a baker) and others which include viral respiratory infections such as caused by rhinovirus, the common cold virus which is the commonest cause of exacerbations of asthma as well as allergens such as grass pollen, house dust mite and cat.

Provokers of asthma cause transient acute airway narrowing in patients with asthma. These include physical factors such as inhaled cold, dry air, the cause of so called exercise-induced (more properly provoked) asthma and inhaled chemicals such as sulphur dioxide which provoke acute airway narrowing in individuals with hyper-responsive airways. Exercise can provoke an attack even in elite athletes (3). This is a provocation of existing asthma, which can occur in an individual who, in the absence of a sufficient exercise stimulus, may not previously have experienced symptoms or have been symptom free since childhood. Exercise does not initiate, but can provoke, asthma. Certain common therapeutic drugs which also can provoke asthma include, aspirin, non-steroidal anti inflammatory drugs and β-blockers, used in the treatment of high blood pressure and chronic heart failure. Anxiety and stress do not induce asthma but can be associated with a worsening of symptoms.

4. Several of these agents occur in the workplace and asthma can be both initiated/induced and provoked by the work environment. The term occupational asthma usually refers to asthma initiated by an agent encountered at work, while work -related asthma covers both this and asthma where attacks are provoked by an agent in the work environment.

5. Asthma caused by an agent inhaled at work can occur due to toxic damage to the airways, (irritant induced asthma) or as the outcome of an acquired specific hypersensitivity or allergic response (hypersensitivity - induced or allergic asthma). Irritant - induced asthma results from inhalation of irritant chemicals in high concentration while allergic asthma can be caused by a variety of substances inhaled over a period of time in usual day to day (non-toxic) concentrations. These include, inhaled proteins such as grain, flours, latex and animal proteins, inhaled chemicals such as isocyanates and platinum salts which bind to body proteins; (so-called haptens) and complex biological molecules such as pine wood resin (colophony) and hard wood dust. Occupational asthma (hypersensitivity-induced) is a prescribed

disease under the Industrial Injuries Disablement Benefit Scheme. As well as recognising over 20 specific allergens, the terms of prescription include a further category, "any other sensitizing agent inhaled at work". Provided other factors have been excluded, a causal link is accepted if symptoms develop and subsequently recur, following exposure to a known sensitizer at work (4). Claims for irritant-induced asthma can be made under the accident provisions of the Scheme.

6. Hypersensitivity-induced asthma differs clinically from irritant induced asthma in the time course of symptoms. Irritant induced asthma develops within 24 hours of exposure to an irritant chemical in toxic concentrations and subsequently persists for at least three months. Hypersensitivity-induced asthma is the manifestation of an allergic reaction and occurs in a minority of those exposed to its cause. There is an initial asymptomatic period (the period of sensitisation), usually of months, after initial exposure to the causal agent. After the development of hypersensitivity-induced asthma, exposure to the causal agent in low doses, to which others are exposed without symptoms, and to which the individual was previously tolerant, can cause exacerbations of asthma with increased airway inflammation and responsiveness.

7. IMEG has identified no published papers on asthma incidence in the armed forces or any of the single services. Service personnel in the three services undertake a variety of principal service occupations and professions as in the civilian world. Some occupations seen in military context e.g. bakers, lab technicians, human and animal health professionals, animal handlers, environmental health technicians, painters, especially spray painters, are at similar risk for occupational asthma as their civilian counterparts.

8. For irritant-induced asthma, symptoms should begin within 24 hours of exposure and persist for at least three months. Possible service exposures for irritant asthma include inhalation of chlorine, sulphur dioxide in high dose, ammonia smoke, and sealant (5) (6).

9. Provokers do not initiate asthma nor worsen the underlying asthma, airways inflammation and airway responsiveness. They provoke attacks in patients with pre-existing asthma so avoidance can reduce the frequency but not the existence, or severity of, asthma. Where asthma is initiated by an occupational agent, subsequent exposure should be avoided as rapidly and completely as possible to minimise the risk of increasingly severe and chronic asthma (7). However despite avoidance of exposure initiated by an agent inhaled at work, asthma may persist for many years, in some cases indefinitely.

Asthma and military service

10. Respiratory problems are a common cause of rejection for military service. Candidates with a history of wheezing including on exercise are normally refused entry. An exception is where a person with a history of wheeze or chest tightness has been free from symptoms and off all treatment for at least the previous four years and where

- the history of wheeze was before the age of four with no episodes since that age
- there is a proven history of a single episode of wheeze associated with respiratory infection since the age of four, but more than four years ago.

11. If a person has asthma in childhood there is significant risk of recurrence as an adult and this includes while serving (8). Fitness, employability and retention decisions for serving personnel are based on the individual case facts including the person's service and principal service occupation e.g. aircrew diver or chef. Issues for consideration include: confirmation of diagnosis; assessing clinical severity; optimum treatment; the presence of workplace allergens and irritants; and the scope for their reduction or elimination as well as alternative service employment (9).

Asthma as a recognised disease in the AFCS

1. To accept asthma as a recognised disease due to AFCS service we need:

i) onset and of symptoms after initial exposure at work to an agent recognised as able to induce irritant or hypersensitivity induced asthma and subsequent clinical pattern consistent with the specific occupational link

ii) pattern of symptoms consistent with irritant or hypersensitivity induced asthma

iii) symptoms and pulmonary function test results are consistent with asthma, i.e. reversible airway narrowing, and other types of respiratory disease are excluded.

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