











Medical Adjudication Guidance

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| <p>General Guidance following the High Court Judgement on the "Bennett" case  Role of Medical and Lay Officers  Changes in Medical Understanding Affecting War Pensions  Conditions of Unknown Aetiology  - Policy Statement </p> | <p>Alcohol Guidance where this is an issue </p> |
| <p>Atherosclerosis Guidance for Claims Myocardial Infarction Psychosocial Factors DSS Response to 1997 Paper by Professor P Sleight FEPOWs and Atherosclerosis</p> | <p>Gastro-intestinal disease Helicobacter Pylori </p> |
| <p>Gulf cases Guidance for Claims</p> <p>Depleted Uranium Adjudication Guidance Background Note</p> | <p>Hypertension Guidance for Claims</p> |
| <p>Ionising radiation Guidance for Claims Genetic Damage Policy Update June 2003  Guidance on evidence of Sue Rabbitt Roff'</p> | <p>Malaria Guidance for Claims</p> |
| <p>Noise Induced Hearing Loss Policy Statement Ageing in Noise Exposed Ears Noise Injury and Balance Disorders Transitional Guidance</p> | <p>Parkinson's disease FOMD on Mortality FEPOWS and Parkinson's</p> |
| <p>Psychiatric conditions Schizophrenia Guidance when Bullying is Contended</p> | <p>Smoking Guidance where this is an issue Passive Smoking </p> |
| <p>Head Injury Head Injury and Dementia </p> | |

Guidance following the High Court Judgement on the "Bennett" case

Background

1. This Note provides guidance about reliable evidence when considering claims to War Pension. The information is a result of a High Court case of BENNETT.

Reliable Evidence

The Law

2. In claims falling under Article 5, the burden of proof rests with the claimant. The claimant **must** show, by evidence, that the disablement is due to an injury which is attributable to or aggravated by service. It is only where a reasonable doubt exists and the reasonable doubt is based on **reliable evidence** that the benefit of that doubt must be given to the claimant. Before the question of attributability can be addressed, it must first be decided whether the evidence is "**reliable**".
3. Article 5 also says that where there is no note in contemporary official records of a material fact on which the claim is based, other reliable corroborative evidence of that fact may be accepted.
4. So there are two questions to be answered:
 - Is the evidence reliable?
 - Does the (reliable) evidence raise a reasonable doubt that the injury is due to service?
5. This guidance only covers the first question, although the two may overlap (ie almost by definition, evidence which is considered to be unreliable will not raise a reasonable doubt).

The Bennett Judgement

6. In the High Court case of Bennett, the judge decided that there is no requirement in law for "corroboration of the claimant's evidence". He said "That which is only permitted can never be required. I construe this as meaning that contemporary official records (which I construe widely) are the best corroboration, but other corroborative evidence can be accepted in lieu."
7. It is implicit in the judgement that the evidence has to be reliable. Where the SoS does not consider that to be so, no award of War Pension may be made.

Providing evidence

8. We can expect the claimant to do everything possible to support his claim. It is not for the SoS to do this. Although the Bennett judgement means we cannot insist on reliable corroborative evidence, that does not prevent us encouraging claimants from the outset, and in their own interests, to provide any evidence they have to support the claim.
9. VA will normally obtain evidence to which the claimant draws attention and is held in Departmental, official service or medical records (ie by DWP, MoD or any part of the NHS).

Evidence

10. Evidence which should be considered includes:
 - the claimant's own statements on the claim form and any subsequent information given on WPA68/WPA68A, at medical/psychiatric boards, or in other correspondence
 - any records provided by the claimant, eg press cuttings, diary entries, family letters written at the time of the alleged incident, accident or injury
 - all service records from the MoD, including any additional records from his personal file and the injury report form (usually obtained from MoD) and whether the alleged incident etc. Occurred on or off duty
 - medical records from the Medical Records Centre at Filestore
 - GP or any medical professional records
 - hospital casenotes (NHS and MoD where relevant)
 - NI records showing claims to sickness/invalidity/incapacity benefits
 - statements made by witnesses who were present at the time. If witnesses are identified on the claim form, follow them up where required. If not supplied, ask the claimant, as appropriate, for regiment, rank and serial number of any witnesses. Occasionally, it may be reasonable to check details with MoD.
 - the claimant's occupational history since leaving service.
11. This list is not exhaustive and other evidence may be relevant in an individual case. Judgement must be exercised in deciding what evidence is necessary in the light of a particular case.
12. As mentioned above, it is in the interest of both the claimant and VA to obtain as much relevant evidence as possible, and as early as possible, during the course of the claim. You must request information as close as possible to the outset of a claim.

What is reliable? - The test

13. Evidence from contemporary official records is reliable and if it confirms the claimant's account of an incident or injury, it is also corroborative. The same applies where reliable evidence from another source or sources confirms the claimant's account.
14. In some cases, however, when all leads have been pursued as far as possible, we will be left with the claimant's statement only, or with just partial confirmation of the alleged incident or fact, eg confirmation that a man played football, but not that he was on duty or representing the regiment.
15. We must then decide whether the statement as a whole can be accepted as trustworthy and safe. The reliability of the statement must be judged in the

context of all the other factors of the case. It should not be judged in isolation. It is essential to take a rigorous approach to testing evidence for reliability, especially as claims are often made in respect of alleged incidents occurring many decades ago. The approach should also be consistent and equitable. Inevitably, with the passage of years, memories may become dim and recollections imperfect. However, it should not be overlooked, that the unsupported evidence of the claimant **can** be accepted as reliable.

Weighing up the evidence

16. It is not possible to devise a precise formula to determine whether the evidence is reliable. Each case must be considered on its merits and a conclusion drawn as to whether the test outlined above at paragraph 15 above is satisfied. In most difficult cases, as part of the team approach to decision-making, it is important to bring in the Medical Adviser (MA) at an early stage to consider medical points which might shed light on the reliability of the evidence.
17. The issues covered in this paragraph are essentially medical relating not just to the occurrence of an injury but to the likely degree of consequent damage. The following questions should be considered in liaison with the MA when assessing evidence for reliability:

In the absence of an official contemporary record or other reliable evidence, is it reasonable to accept as credible the claimant's statement?

- bear in mind that the absence of a reference to a claimed incident, accident, significant injury or hospital treatment in an otherwise detailed service record might constitute evidence that it did not occur in the circumstances claimed, casting doubt upon the reliability of the claimant's account
- it may be reasonable to ask the claimant about the omission, eg why did he not report the accident?
- are VA or even the claimant aware of any other similar cases?

Why did the claimant not mention the injury on discharge and/or sign a statement saying that he had suffered no injury or disease during service?

- at the very least, either might suggest that the effect of the injury was slight, although by itself this does not mean it did not occur.

Why was the claimant classed "A1" on discharge?

- equally, "A1" on later entry into the reserve would suggest that the service injury was, at the very most, a minor one.

Are there any inconsistencies in the claimant's statements which may suggest that the evidence is unreliable?

- where necessary, the claimant should be questioned about his statements in an effort to resolve any inconsistencies.

Does the nature of employment since discharge suggest that the service injury was slight, or that disablement is due to a cause other than service?

- this could be particularly relevant where the injury was not noted in service records and where the claimant has, since service, done heavy manual work in civvy street, or work where there is a rigorous pre entry medical.

Is the time between the alleged injury, first recorded treatment for the claimed condition and the claim, a factor?

- this may be relevant, particularly if the claimant now says he has suffered disablement from the start, but has never sought treatment for it or only sought treatment after a time gap, or has not made a previous claim to a War Pension.
- the time gap could suggest that the alleged service injury has no connection with the current disablement.

Is the nature of the current disablement consistent with the claimant's alleged service injury?

- if the injury and disablement are not consistent, there is good reason for taking the view that the claimant has not provided reliable evidence to raise a reasonable doubt that the disablement is due to service.

Deemed destroyed cases

18. Lawyers advise that "wrongful destruction of official records cannot change the legal position" and that there are no exceptions in legislation for the official records being destroyed. The legal requirement must be applied in **all** cases.

Conclusion

19. Where there is no official record of a service injury, each case must be examined on its merits and in the light of all the circumstances in order to decide whether the claimant's statement is trustworthy and safe.
20. As much relevant evidence as possible must be obtained and the overall picture presented by that evidence considered. In considering each case, it might be helpful to set out separately the factors which support the claim and those which apparently contradict it.
21. It is important in every case that decisions, reasons for decisions, telephone calls and any discussions with MAs should be clearly recorded on file.

War Pensions: roles of Medical Officers and lay officers in decision-making

Background to War Pensions entitlement

Entitlement to a War Pension on account of service-related disablement and death for those who served in HM Forces, or their dependants, is provided for by The Naval, Military and Air Forces Etc (Disablement and Death) Service Pensions Order 1983 (the SPO). Except in prescribed circumstances, such as when a serviceman is invalided from the Services or dies in Service, a claim for War Pension from the disabled or bereaved person is required before the Veterans Agency (VA) can consider entitlement.¹ Entitlement exists from the time of an injurious incident; it is brought into effect by making a claim. War Pension cannot be paid whilst still in service. There are no time limits within which a claim must be made. Entitlement can therefore come into effect at any time after release from service. If entitlement is established, the amount of war pension payable depends principally on the extent to which the ex-serviceman is disabled due to service. This is assessed, and a war pension awarded by the Secretary of State in the form of a lump sum gratuity (for disablement of less than 20%) or an ongoing pension (for disablements assessed at 20% or more).

War Pensions can only be paid in respect of disablement due to, or aggravated by, service; or death due to, or hastened by, service. They cannot be paid for conditions due to non-service causes such as ageing or life-style.

Consideration of Entitlement

The Secretary of State for Social Security makes decisions in relation to eligibility and entitlement to war pensions.

Who is the Secretary of State?

These decisions are made by lay officers of Veterans Agency acting as the Secretary of State.²

Determining entitlement

1. Firstly, lay officers determine facts³. For example, whether a claim has been made, for what, and the date on which it has been made, whether the claimant was a member of HM Forces, whether he has "shown disablement", whether he suffered a service-related injurious incident.
2. Then, if entitlement can be determined without an answer to a medical question, for example where a claimant does not satisfy a basic lay condition (eg former membership of HM Forces) the lay officer gives a rejection decision. There is no need for a Medical Officer's certificate because there is no medical question to be determined.
3. Where the lay officer has accepted the facts of a case, but cannot make a decision on entitlement without the answer to a medical question, the lay officer asks a Medical Officer appointed for the purpose to answer that question. The Medical Officer, who has professional medical expertise and experience in application of the

¹ Articles 3A & 3B The Naval, Military And Air Forces Etc (Disablement & Death) Service Pensions Order 1873

² *Carltona Limited V Commissioner Of Works* (1943) 2 All ER 560

³ *Gillan V The Minister Of Pensions*; ROSWPA, Vol 5, P 286

provisions of the War Pensions Scheme (in practice a Medical Adviser employed by Veterans Agency) will determine that medical question.⁴ Medical Officers provide a reply in the form of a certificate on questions such as:

- whether a claimed disablement is due to an injury which is attributable to, or aggravated by, service; or
 - whether a death is due to, hastened by, or substantially hastened by, an injury that was attributable to, or aggravated by, service.
4. The ultimate responsibility of the lay officer (acting as Secretary of State) who is taking the decision is to ensure that it is a reasonable one. Where the determination of a claim involves a certificate from a Medical Adviser on a medical question, the lay officer must therefore be satisfied that it is reasonable for him to follow that certificate. The lay officer shall accept the certificate, subject to the following conditions:

(a) Matters of fact

If there is a factual statement in the certificate which he knows to be inaccurate (eg where a certificate records an amputation of the left leg, but the lay officer knows from other evidence before him that it is the right leg which has been amputated). In such situations the lay officer should refer the certificate back to the Medical Adviser for discussion. If a question of law arises legal advice may be sought in consultation with the Medical Adviser.

(b) Medical matters

The Secretary of State appoints or recognises doctors to provide certificates on medical matters. In the vast majority of cases it is therefore reasonable for him to rely on the certificate of his expert Medical Advisers. If, exceptionally, a case does arise where the lay officer has a query relating to the certificate, the case should be referred back to the Medical Adviser for discussion.

Entitlement decision

If the Medical Adviser's certificate states there is a causal link between disablement and service then, subject to the considerations set out in paragraph 4, the lay officer decides entitlement to War Pension in accordance with that certificate.⁵

If the Medical Adviser's certificate states there is no causal link then, subject to the considerations set out in paragraph 4, the lay officer gives a decision refusing an award.

Assessment

Where entitlement to a war disablement pension is established it is then necessary to determine the degree of the claimed service-related disablement. War disablement pension can be claimed for any disablement, and awards under the Scheme depend not on the nature of the disablement but on its degree. The legislation sets out the method of assessment, which is by comparison of the disabled person with a normal healthy person of the same age and sex.

⁴ Art 1 (4)(b)(i) and Arts 4 (1) and 5 (1) of the SPO

⁵ Art 1 (4)(b)(i), 4 (1), 5 (1) & 8 of the SPO

The resultant level of assessment is expressed as a percentage. As assessment of disablement involves a medical question, the lay officer asks the Medical Adviser to provide a certificate answering the question as to what degree the ex-serviceman is disabled as a result of service⁶. The lay officer then determines the degree of disablement in accordance with the Medical Adviser's certificate (subject to the conditions set out in paragraph 4 above).⁷

Award

A lay officer decides the rate of War Pension due (taking into account adjustments that may be necessary on account of factors such as serious negligence by the pensioner, awards of third-party compensation, and imprisonment). A lay officer also decides the date from which pension is awarded.⁸

Supplementary Allowances

War disablement pension awards provide compensation for the service-disablement itself. The disabling effects of the injury on the individual, such as the need for care, or assistance with mobility, are addressed by supplementary allowances.

In relation to supplementary allowances, a Medical Adviser advises a lay official on medical matters, in cases where the Secretary of State deems this appropriate. There is no requirement for questions relating to supplementary allowances to be certified, so a Medical Adviser's certificate on such questions is inappropriate.⁹ It is for a lay official to determine entitlement and make award or rejection decisions on supplementary allowances as appropriate.

Guidance

In order to support consistency and equity in decision-making, guidance can be given to medical and lay Agency personnel on the legal, procedural and policy parameters which they should take into account in exercising their decision-making roles. This guidance takes the form of Departmental policy statements, instructions, medical appendices and medical adjudication guidance. Each case must however always be considered on its individual merits.

Where medical certification or advice is required it should reflect the circumstances of the individual case, including facts accepted by the Secretary of State, and correctly apply the legislation, taking account of relevant case law on how it should be interpreted. Decisions must also be medically sound, reflecting the relevant medical evidence in the particular case, and in line with contemporary accepted medical and scientific understanding.

⁶ Art 1 (4)(b)(i), Art 9 (3) & (4), SPO

⁷ Art 1 (4)(b)(i) & Art 9 (6) SPO

⁸ Starr And Others V The Minister Of Pensions, ROSWPA, Vol 1, P 109

⁹ Part III, SPO

Changes in Medical Understanding Affecting War Pensions

The legal context

1. In deciding whether or not new medical or scientific evidence impacts on war pensions, the standard of proof to be applied is not scientific proof. Rather, it is reasonable doubt. War Pensions legislation and case law provide guidance on this consideration.

2. When considering whether there is reliable evidence to raise a reasonable doubt case law has established that all the available evidence must be weighed:

".... the intention of the paragraph [Article 5(4) of the Service Pensions Order 1983] is that it is the duty of the claimant to produce reliable evidence to establish his claim, but if (after hearing and considering that reliable evidence and making a comparison between such evidence and other evidence which is called on behalf of the Ministry to contradict or controvert it) the Tribunal has a reasonable doubt, then under those circumstances the plain meaning of that paragraph of the Article is that the benefit of that doubt shall be given to the claimant" (Dickinson).

3. Case law also establishes that it is reasonable to conclude that a published work presenting a hypothesis based on a limited study, which later comes to be the generally accepted view, would not at the time of its publication create a reasonable doubt. It was for the Secretary of State to decide at what later stage, when the hypothesis becomes sufficiently supported, a reasonable doubt was raised. This was before it became the generally accepted view:

"I see no reason why, the Secretary of State should not be entitled to hold that what has now become the generally accepted view was a mere hypothesis based on a limited study which would not have created a reasonable doubt within the terms of Article 5(4). The stage by which it becomes sufficiently supported to raise such a doubt in his mind is a matter for the Secretary of State. Accepting, however, that the shift of opinion was a gradual process and that by February 1980 it was the generally accepted view, there must have been an earlier stage when, if asked to consider the matter, he would have found that there was a "reasonable doubt" (Edwards).

4. The Edwards judgement was in retrospect - looking back from a position of general acceptance to establish the point at which the initial hypothesis had become a reasonable doubt. In some cases the proposition eg that noise and age related hearing losses are more than additive is not presently generally accepted - and so the consideration is prospective.

Consideration of evidence

5. Factors to be considered in deciding whether there is reliable evidence to raise a reasonable doubt would therefore include:

- whether the new view being expressed was a mere hypothesis and whether there was any supporting evidence for it and the basis for that support (eg empirical evidence)

- the weight of the evidence on both sides
 - publication of evidence (although the fact that work is not published does not, on its own, mean that it cannot be considered to raise a reasonable doubt - it might do so, for example, if it was unpublished but was widely supported by those in the field.
6. The courts have also held that a conflict of medical opinion does not, in itself, mean that a reasonable doubt has been established and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of *Tigg v The Ministry of Pensions* the presiding Judge stated "Merely because a doctor of eminence, and I have no doubt the doctor in this case was of very great eminence, is expressing a view contrary to the view expressed by the medical witness called on behalf of the Ministry, does not mean there is a doubt and the appellant must therefore be entitled to a pension. It is a question of fact for the tribunal" (cases of [Tigg ROSWPA Vol 5, p141](#) and [Howard ROSWPA Vol 5, p515](#)).
7. Identification and accreditation of changes in medicine broadly follow the scientific process. An individual has an idea on a topic and if, usually following informal communication with colleagues, the idea is considered worth pursuing, he goes on to formulate a hypothesis. Whether the facts fit the hypothesis is then tested by experiment. Many experiments may be required and empirical findings need to be repeatable. In time a paper is prepared and submitted for publication in peer reviewed journal. Here referees consider such matters as:
- whether the material is original and not trivial
 - whether the paper takes due account of and cites previous related work and the more general literature on the subject
 - whether arguments are plausible and clearly expressed with appropriate use of tables and figures
 - whether the experimental procedures appear technically competent
 - whether conclusions reached are in line with and not contrary to indisputable facts
8. It is not the function of referees to validate the material presented in a paper and publication does not in itself accredit the contents. Rather it allows wider expert reaction and other groups to take up the matter. The further progress of a scientific or medical idea may be diverse and prolonged.
9. There may be a period of controversy with further studies alternately replicating and refuting the original hypothesis. The publication, and hence hypothesis, may gain credence by being cited by other authorities and in Review articles on the topic.

10. In this way a body of evidence will be accumulated to support or refute the original concept. That evidence may then be evaluated, peer reviewed and finally weighed against the existing body of evidence and knowledge. By this process an idea may become generally accepted and recognised as a change in medical understanding.

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War Pensions Causation - Conditions Of Unknown Aetiology

1. Understanding of the aetiology of medical conditions is fundamental to quality War Pensions medical decision-making. Early War Pension claims were for disablement due to traumatic war related injury but later the legislation extended the meaning of 'injury' to include wound or disease. In the forties most medical conditions were considered constitutional and therefore never attributable to service. Today the pattern of War Pensions claims is different, with much less emphasis on disablement due to traumatic war injury. Over the years, case law has changed the standard of proof and for many diseases medical understanding now accepts both genetic and environmental causes. All of this, and the fact that War Pensions is an individual jurisdiction, make decisions on War Pensions causation complex.

Medical Appendices

2. Since there are no standard texts on disease aetiology, the **appendices** were developed. These are dated and aim to represent at date of writing, contemporary accepted medical understanding of the conditions discussed. They do not represent a specific Departmental viewpoint. Care is needed in their interpretation in the context of War Pensions 'reasonable doubt' standard of proof. The appendix standard of proof is the 'medical/scientific' proof normally applied in medical and scientific writing. This implies sufficient evidence not just to raise a reasonable doubt but to become generally accepted medical understanding. Where a cause of a condition is rejected as unproven in the appendix it cannot be assumed that the requirements of Article 4 are met. It is important to look at the language used. Is it simply that there is no evidence to suggest an association or is actual evidence quoted which discounts a causal effect, beyond reasonable doubt? Only the latter satisfies Article 4 standard.
3. A particular possible causal factor or circumstance may not have been investigated and therefore no evidence has emerged as to whether or not the factor is associated with development of a particular disease. There is therefore 'lack of evidence of an effect'. It is important to note that that is not the same as 'evidence of lack of effect'. 'Evidence of lack of effect' results when a matter has been investigated with studies concluding that there is no such causal association.

Conditions Of Unknown Aetiology - The Coe Rules

4. Conditions of unknown aetiology are medical diagnoses where it is generally accepted medically that research has not identified specific causes to account for the majority of cases. **The concept and the case law concern Article 4 of the SPO.** There is no case law on conditions of unknown aetiology under Article 5. The Coe judgement (RSWPA Vol 5, Page 725, 1966) sets out 3 rules:

Rule 1: If the medical evidence is simply to the effect that nothing is known about the cause of the disease, the presumption of entitlement in the claimant's favour created by Articles 4(2) and 4(3) is not rebutted, and an application for a pension on the ground of attributability must succeed.

Rule 2: If there is evidence that, although its aetiology is unknown, the disease is one which arises and progresses independently of service factors, the claim may be rejected.

Rule 3: It will not suffice to rebut the presumption to offer evidence merely to the effect that:

'.....in the light of modern knowledge, it cannot be accepted that service factors are associated in any way with the onset of the disease or that any circumstances of service hastened its course'.

For evidence of that nature does not establish that service factors played no part in causing a disease in such conditions there would have to be an award on the basis of attributability.

5. The judgement uses the term 'factors of service'. Recent in-depth investigation of the term by policy and legal colleagues confirms the concept as wide-ranging and specific to individual War Pension claims. In short, the term applies to any material fact relevant to the claim.
6. Interpretation of Rule 1 of Coe is straightforward. **If nothing is known about the aetiology of a condition underlying a claimed disablement** (and the other conditions of the SPO are fulfilled) entitlement must follow.
7. Rule 2 clarifies the scope for the Secretary of State to reject a condition of unknown aetiology under Article 4. In essence the Secretary of State needs to be able to **point to evidence dissociating service factors from the claimed condition before he can reject under Rule 2**. To reject, he must point to evidence which shows beyond reasonable doubt that **no aspect** of service played a part in cause or course of the condition. Suitable evidence would be published peer reviewed scientific evidence covering all the relevant material facts. There may be scope for interpretation of research, eg research may be available on certain factors such as physical exertion but not necessarily in a service setting. It would be reasonable to argue that this 'by default' dissociates service factors. Given the broad and idiosyncratic nature of service factors as indicated above, this is clearly a very stiff test.
8. Rule 3 is even more constraining in terms of medical logic. It covers the situation where there is no (or incomplete) evidence about the **causal role of service** in a particular condition of unknown aetiology but there is no evidence suggestive of a service role. As a result, the general medical understanding of the condition would be that there is no association with service. That view meets the medical standard of proof and will reflect generally accepted medical understanding. Legal advice is, however, that it does not meet the Article 4 standard of proof.
9. The penultimate paragraph of the Coe judgement contains the following:

'.....Medical services said that "the aetiology of this disorder is for the moment unknown". The judgement goes on to say, "From the published literature, it would appear that it is highly improbable that any of the factors common to

service life are in any way responsible for the origin or aggravation of Behcet's Syndrome. Despite numerous investigations which have been carried out, it has never been shown that trauma, dietetic or climatic hardships, physical or mental stress or strain, sudden, cumulative or prolonged, play any part in the causation of the disease. To hold, on these grounds, that the applicant was disentitled to a pension is, in my judgement, in effect to place some onus of proof upon him, and that of course, is wrong".

In rejecting the claim simply on the basis that there is no evidence of a service link is effectively to transfer the onus of proof to the claimant. To reject requires **evidence** that service played no part.

Decisions On The Same Disease In Different Claims

10. An important issue frequently raised over the last few years is the desirability of consistency of War Pensions **decisions for the same disease in different claimants**. The Coe judgement comments on this, stating that it would obviously be:

"neater and more satisfactory, at least in the eyes of the lay man, if all decisions in respect of a particular disease were uniform, whether they be for or against the applicant for a pension. But to lay this down as a rule would be to exalt a **finding of fact in a particular case into a principle of law applicable to all cases**. That would be wrong for, as Lord Wright said in *Tidy vs Battman* (1934) 1st KB Division, page 319: "It is unfortunate that questions that are questions of fact alone should be confused by importing into them as principles of law a course of reasoning which has no doubt been properly applied in deciding other cases on other sets of facts and later.....". The use of formulas and categories may cause injustice. The task of the Tribunal is to decide each case on the material before it.'

Practical Decision Making

11. So far we have established that to lawfully reject conditions of unknown aetiology under Article 4 is a considerable challenge requiring a particular type of evidence. How can we best approach this issue operationally? One potentially attractive way would be to provide a list of conditions of unknown aetiology which may be normally rejected. There are however limitations to lists, not least an ongoing need to review and revise. There is also the intrinsic risk of fettering judgement in an individual jurisdiction. Cases must be considered on their individual merits.

12. In the individual claim, having established that the injury underlying the claimed disablement is a disease, one then first goes on to consider whether or not it is a condition of unknown aetiology. To do this reference should be made to the relevant medical appendix and the following questions addressed:-

- How much is known about the disease overall?
- Does the appendix give a clue as to the quality and quantity of the research? ie are there many references? Are these recent? Are the studies by well known research groups? Are they published in mainstream publications?

- Can you say that causes accounting for the majority of cases are identified and accepted medically or
- **Are we really dealing with a condition of unknown aetiology ?**

13. If the conclusion is yes, under Article 4, unless there is evidence quoted in the appendix, covering all the relevant case specific material facts and dissociating these from the condition, rejection is not appropriate. Some "worked examples" of the approach are at **Annex A**.

14. It is important to note that, using the definition in paragraph 4 above, and the suggested approach to the appendices, many conditions, eg many cancers, hypertension and atherosclerosis are **not** conditions of unknown aetiology. In these diseases, research has identified causes to account for the majority of cases.

The Coe Rules Do Not Apply To Conditions Of Known Origin.

15. Current understanding of the general causes of cancer emphasises the importance of **lifestyle factors** particularly smoking, alcohol and diet. In some individual cancers – eg, particularly non-small cell lung cancer - the causal link with cigarette smoking is very strong – with up to 90 per cent of tumours due to this - and critically depends on the numbers of cigarettes smoked and length of the habit. A similar relationship is seen in bladder cancer, cancer of the larynx and mouth. Smoking and alcohol consumption are also key in oesophageal malignancy in the western world. Dependent on the individual case facts, an MA may well judge that the evidence allows defensible rejection of such claims under Article 4.

16. On the other hand there will be cases of **conditions of known origin**, where in the individual case, the known causal factors accounting for the majority of cases may not be present. In this situation (although Coe is not relevant), the normal, already very considerable Article 4 burden of proof, applies and lawful application to the individual case facts may result in certification.

17. In conclusion, in determining war pension claims for conditions where causes to account for the majority of cases are not known:-

- Under Article 5, it is for the claimant - by reliable evidence - to raise a reasonable doubt in his favour of a service link. All the evidence is weighed and the benefit of doubt given to the claimant. Hypothesis or speculation on causation which might be covered by language such as "it is thought that" "there is some evidence that" - does not meet the criteria for reliable evidence.
- Under Article 4 whether 4(2) or 4(3), rejection of conditions of unknown aetiology, requires evidence beyond reasonable doubt that relevant service factors or the material facts specific to the claim are not involved in causation of the claimed disablement.

Summary

- The Coe Rules applicable to conditions of unknown aetiology are relevant only under Article 4.
- Conditions of unknown aetiology are medical diagnoses where it is generally accepted medically that research has not identified specific causes to account for the majority of cases.
- Under Article 4, for conditions of unknown aetiology, rejection of a service link is only appropriate where the Secretary of State is able by evidence beyond reasonable doubt to dissociate the condition in the individual case from service causal factors.
- War Pensions is an individual jurisdiction. Article 4 claims for conditions of unknown aetiology must be carefully considered on their case specific facts.
- No blanket guidance on attributability of conditions of unknown origin is appropriate. Consistency in War Pensions certification of conditions of unknown origin lies not between the specific conditions underlying the claimed disablement but between closely similar individual cases.
- Where in any individual Article 4 case, the claimed disablement is due to a disease other than a condition of unknown aetiology and the known causes are not present, the normal Article 4 considerations apply.
- The Coe rules effectively introduce – for conditions of unknown aetiology – an even greater hurdle for lawful rejection than is presented by the provisions of Article 4 alone.

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Conditions Of Unknown Aetiology

This note briefly contains some 'worked examples' of the approach described in the Practical aspects section of the desktop guidance. The current in use editions of the medical appendices on motor neurone disease, brain tumours, osteoarthritis and atherosclerosis are used as the medical evidence.

Motor Neurone Disease

i How much is known about the causes of the condition or disease overall?

Comment: No firm generally accepted causes have been identified.

ii What is the quality of the research?

Comment: Not known from the appendix.

iii What is the quantity?

Comment: The condition is described as sporadic but research has been intensive. Difficult to tell from the appendix but case series likely to be small.

iv Can we say that causes accounting for the majority of cases are identified and accepted medically?

Comment: No.

v Are we really dealing with a condition of unknown aetiology?

Comment: Yes.

Brain Tumours

i How much is known about the causes of the condition or disease overall?

Comment: Apart from a familial component and ionising radiation, no firm generally accepted causes have been identified.

ii What is the quality of the research?

Comment: Not known from the appendix. It is noted that the research quoted refers to brain tumours generically. In fact the appendix makes clear that this is a highly heterogeneous diagnosis with many sub groups having origin in different cell types. That suggests no real basis to assume common aetiological factors. ? Does the grouping together frequently seen in research papers to increase numbers compromise research quality.

iii What is the quantity?

Comment: Considerable. Also up to date. In mainstream journals and published by well known researchers.

iv Can we say that causes accounting for the majority of cases are identified and accepted medically?

Comment: No. Many causal influences have been proposed but the evidence is conflicting with later studies failing to confirm first research.

v Are we really dealing with a condition of unknown aetiology?

Comment: Yes.

Atherosclerosis

i How much is known about the causes of the condition or disease overall?

Comment: The appendix discusses 17 major groups of risk factor.

ii What is the quality of the research?

Comment: Not immediately known from the appendix. However the references list work done in many major academic centres by some of the world's leading cardiologists and epidemiologists.

iii What is the quantity?

Comment: Enormous.

iv Can we say that causes accounting for the majority of cases are identified and accepted medically?

Comment: Yes. The appendix notes occlusive vascular disease as the major cause of morbidity and death in developed countries and that 3 independent risk factors account for half the variation in ischaemic heart disease incidence. Goes on to list 14 other factors. Otherwise there is no direct reference. However atherosclerosis is the subject of intensive publicly funded primary and secondary prevention actions. Such programmes only arise when major causes of the condition are known and such action is likely to be effective in both human and financial cost terms.

v Are we really dealing with a condition of unknown aetiology?

Comment: No

Osteoarthritis

- i How much is known about the causes of the condition or disease overall?

Comment: The appendix discusses general factors, constitutional and local factors and covers 22 paragraphs. Although the precise mechanisms are not always clear, there are very many major identifiable risk factors.

- ii What is the quality of the research?

Comment: Not known.

- iii What is the quantity?

Comment: Appendix refers to epidemiological research studies in relation to obesity, genetics, hormones, bone density, injury etc. A very common condition which has attracted considerable research.

- iv Can we say that causes accounting for the majority of cases are identified and accepted medically?

Comment: Yes. The appendix shows that primary osteoarthritis is often constitutionally related and that the secondary type, by definition, has a known cause.

- v Are we really dealing with a condition of unknown aetiology?

Comment: No

Conclusion: It is likely in individual cases of Motor Neurone disease or brain tumours certification under Article 4 will occur.

Latent periods

For war pensions certification where tumours are in the frame it is important to bear in mind latent periods. Tumours – benign and malignant – have long latent periods. Unfortunately cell kinetic studies are not yet sophisticated enough to allow definite latent periods for individual tumour types to be established. However, it is known that acute leukaemias develop more quickly than solid tumours (from the Radiation Effects Research Foundation follow-up studies of atomic bomb survivors). In general the latent period for leukaemias is two years, and for other solid malignant tumours, around ten.

WAR PENSIONS: CONDITIONS OF UNKNOWN AETIOLOGY: POLICY STATEMENT

Summary

Department's Policy:

A war pension may be awarded for any disablement causally linked to service. The scheme does not provide awards for disablement that is not attributable to, or aggravated by, service. Anyone who has served in the armed forces can claim a pension at any time from service release. Decisions are evidence based, reflecting the facts of the individual case, the relevant law and policy, and contemporary scientific and medical understanding of the aetiology (cause) of the claimed condition.

Conditions of unknown aetiology are medical diagnoses where it is generally accepted medically that research has not identified specific causes to account for the majority of cases.

Medical Officers within the War Pension Agency establish the **medical condition (diagnosis) underlying the claimed disablement**. Diagnosis has to be established to **enable consideration** of possible causal link between the disablement and service. Medical adjudication guidance on conditions of unknown aetiology is issued separately.

Each case is determined on its own individual circumstances and evidence.

Legal Summary

Under **Article 4** of The Naval, Military and Air Forces etc. (Disablement and Death) Service Pensions Order 1983 (SPO), the onus is on the Secretary of State to show, beyond a reasonable doubt, that service has not caused or aggravated the claimed condition. Consideration of claims for conditions of unknown aetiology falling to be considered under Article 4 varies **depends on the contemporary published evidence**.

- i when nothing is known about the aetiology of a condition **then** the Secretary of State cannot disprove a service link **and** it must be accepted as attributable to service;
- ii if the aetiology of the claimed condition is unknown but **appropriate research evidence of suitable quality** shows beyond reasonable doubt that the disease is one that occurs and progresses independently of service factors the claim can be rejected.
- iii if the available medical evidence is **simply** that "it cannot be accepted that service factors are associated with the onset or hastening of the disease", then this does not establish that service played no part but simply refuses to accept the positive assertion that service factors played a part in causing the claimed condition. If the Secretary of State cannot prove **by evidence** beyond

a reasonable doubt that service factors did not play a part in causing the onset or hastening of the disease the condition must be accepted as attributable to service.

Where a claim falls to be considered under **Article 5** of the SPO, the onus is on the claimant to raise a reasonable doubt, through reliable evidence, that service has caused or aggravated the claimed condition.

If the position in relation to a condition is simply that the cause is totally unknown, then it is **likely** that under Article 5 a claimant will be unable to raise a reasonable doubt in his favour by way of reliable evidence. A claim will **therefore not** normally succeed. However, if, **in the individual case**, the claimant produces reliable evidence raising a reasonable doubt in comparison with evidence held by the Department, then the benefit of that doubt must be given to the claimant.

The Issue

War pension is awarded for disablement due to injury due to service. The definition of injury includes wound or disease. When the claim is in respect of a disease, the aetiology (cause) of disease is therefore the basis for war pension considerations. The fact that a disease is diagnosed does not mean its aetiology is known. This statement considers how to approach war disablement pension claims for conditions of unknown aetiology.

The Law

How the scheme works

- i The war pensions scheme is administered under the authority of the Service Pensions Order. A war pension may be awarded for disablement **due to service in the armed forces**. "Disablement" is defined as "physical or mental injury or damage, or loss of physical or mental capacity"¹. "Injury" includes wounds and diseases².
- ii A war pension may be claimed for any disablement by anyone who has served in the British armed forces. Claims may be made at any time after service release. Decisions are evidence based and each case must be determined on its own individual circumstances using the relevant burden and standard of proof, as explained at paragraph 5. Awards may be made where, within the relevant law, the evidence (including service and medical **facts** and the contemporary medical understanding of the condition claimed), is that service has caused or made worse the claimed condition. When the aetiology of a disease is unknown or poorly understood entitlement considerations **may be complex**.

¹Item 22, Part II, Schedule 4 to the SPO - in this statement the term "Service Pensions Order", and the abbreviation "SPO", refer to The Naval, Military and Air Force etc. (Disablement and Death) Service Pensions Order 1983, SI 1983 No. 883, as amended.

² Item 27, Part II, Schedule 4 of the SOP (amended by Article 5(3) of SI 1994/772

- iii When a claim is made, the **medical diagnosis underlying** the claimed disablement has to be established. Diagnosis is a medical matter and the approach is therefore similar to that which would be applied in a clinical setting – where diagnosis will inform the most appropriate medical treatment. Diagnosis has to be established before considering any possible causal link between the disablement and service.
- iv The test of proof for deciding whether service was the cause of disablement is peculiar to the war pensions scheme:
 - a) for claims made not more than 7 years after leaving the armed forces, **Article 4** of the SPO places the onus on the Secretary of State to show beyond a reasonable doubt that the claimed disablement is not due to an injury (or disease) that is attributable to, or aggravated by, service;
 - b) for claims made more than 7 years after the end of service, **Article 5** of the SPO puts the onus on the claimant to raise, by way of reliable evidence, a reasonable doubt that the claimed condition is due to an injury (or disease) that is attributable to, or aggravated by, service.

Case Law

Article 4

Over time, the High Court has produced a number of judgements which provide interpretation of war pensions provisions when dealing with conditions of unknown aetiology;

In the case of Bourne³, in February 1946, the court upheld the notion that under certain circumstances even when the precise cause of a condition remained unknown, there might be adequate material "... of a scientific or statistical nature, as known to the medical profession, to enable doctors to exclude factors as having any influence upon the disease". Similarly, in November 1947, (the then) Mr Justice Denning found that, "... in many cases although the aetiology is unknown, experience and statistics are able to throw light on the circumstances in which the disease arises or develops"⁴.

In the case of Brown and others (July 1946), the Lord Justice-Clerk (Lord Cooper)⁵ referred to expressions used by the Ministry such as a disease being "*of unknown origin*", "*insidious in onset*" and "*of endogenous aetiology*". The Court held that if no one knows what causes the disease the Ministry could not possibly discharge the onus of proving beyond reasonable doubt that service conditions did not cause it.

Moreover, should the claimant provide **any** facts based on reliable evidence to suggest that service may have played a part in causing the disability, entitlement should be accepted on the basis of attributability: "... if there is anything reasonable to suggest...that any incident of war service may have played a part, such as

³ Bourne, ROSWPA, Vol 1, P 109

⁴ King, ROSWPA, Vol 1, P 817

⁵ Brown & Others, ROSWPA, Vol 2, P 461

exceptional stress or strain immediately preceding the onset of symptoms the claim must be allowed⁶."

However it is the 1966 High Court case of Coe⁷ v Minister of Pensions and National Insurance which is now considered when the Secretary of State makes an entitlement decision. The Coe case provided three rules, which evolved after consideration of previous judgements, in relation to medical evidence/attribution.

It should be noted that Coe rules were made with specific reference to an Article 4 case and therefore only have meaning in the context of Article 4.

Rule 1:

"If the medical evidence is simply to the effect that nothing is known about the cause of the disease, the presumption of entitlement in the claimant's favour created by Articles 4(2) and 4(3) is not rebutted, and an application for a pension on the ground of attribution must succeed"

- a) If nothing is known about the aetiology of a condition underlying the claimed disablement, Coe Rule 1 applies and attribution must be accepted.
- b) However this rule will seldom be appropriate nowadays, as most diseases have been subject to some research and there will therefore be at least some degree of scientific and/or medical knowledge.

Rule 2:

"There is evidence that, although its aetiology is unknown, the disease is one which arises and progresses independently of service factors, then an award should not be made."

The Secretary of State can reject a condition of unknown aetiology under rule 2 of Coe, if he has evidence that:

- a) disproves a service link, even though the actual cause of the condition cannot be stated; and
- b) shows beyond reasonable doubt that the disease is one which occurs and progresses independently of service.

However, to make such a rejection the evidence disproving a possible service link must be such as to remove all reasonable doubt. If the evidence does not reach this degree of certainty, Coe Rule 3 applies and entitlement should be accepted.

⁶ Miller, ROSWPA, Vol 1, P 615, 625 (July 1947)

⁷ Coe, ROSWPA, Vol 5, Page 725

Rule 3:

It will not suffice to rebut the presumption in the claimant's favour to offer evidence merely to the effect that "... in the light of modern knowledge, it cannot be accepted that service factors are associated in any way with the onset of the disease or that any circumstances of service hastened its course. Evidence of that nature does not establish that service factors played no part, merely declines to accept the positive assertion that service factors played a part in causing a disease. In such circumstances there would have to be an award on the basis of attributability."

Coe rule 3 should be considered along side the penultimate paragraph of the judgement which provides:

"From the published literature, it would appear that it is highly improbable that any of the factors common to service life are in any way responsible for the origin or aggravation of Behcet's syndrome. Despite numerous investigations which have been carried out, it has never been shown that trauma, dietetic or climatic hardships, physical or mental stress or strain, sudden, cumulative or prolonged, play any part in the causation of the disease." The judge then noted "To hold, on these grounds, that the applicant was disentitled to a pension is, in my judgement, in effect to place some onus of proof upon him, and that of course, is wrong."

For the Secretary of State to reject a case, he must refer to evidence which must specifically have researched and excluded service factors playing a part in the causation of the disease. For example he must show that trauma, dietetic or climatic hardships, physical or mental stress or strain, sudden, cumulative or prolonged play no part in the causation of the disease. The research does not necessarily have to have been conducted in a service setting.

Where there is no, or only limited, evidence about the causal role of service in a condition the cases cannot be rejected. To reject a claim on the basis that there is no evidence of a service link incorrectly places the onus of proof to the claimant: *"It was not for the appellant to prove that he had been exposed to some relevant infection, it was for the Secretary of State to prove that he had not"*.⁸

Suitable evidence would be published peer reviewed scientific evidence covering all the relevant material facts. The research referred to does not necessarily have to have been conducted within the service setting but must dissociate service factors.

Should the claimant provide any reliable evidence to suggest that service may have played a part in causing the disablement, entitlement should be accepted on the basis of attributability:

⁸ Bennett V Secretary Of State For Social Security 6 June 1984

"A Cause is the Cause"

The test of proof in war pensions is reasonable doubt - a condition is accepted as due to service even if there is no more than a reasonable doubt, based on reliable evidence, that the disablement was attributable to, or aggravated by, service. It is irrelevant how great an impact war service had on the disability as long as there is a causal link; a minor contributory cause which is service related is as significant as a major cause.

On 10th November 1947 Denning J (as he then was) held in the case of Marshall:⁹

"If war service is a cause of the disease arising, even though not a predominant cause, the disease is attributable to war service; but, if it is not a cause of it arising the only remaining question is one of aggravation, which depended on whether the injurious process is accelerated or intensified by war service. The burden is upon the Minister on all these questions to negative firstly, attributability, and secondly, aggravation."

The High Court principle "A cause is the cause" stems from the Marshall case. The principle being:

- a) if a disease arose during war service, if war service was one of the causes of it arising, then it is attributable to war service. If war service was not a cause of it arising, it cannot be attributable, but may be aggravated;
- b) on the issue of attributability/aggravation; the first question is when the disease arose. The burden here is on the Minister [now Secretary of State] to show that the disease existed before service; a pre-service susceptibility or predisposition is not enough to discharge the burden;
- c) the task of the Minister [now Secretary of State] and Tribunal is to ascertain what are the causes of the disease arising, not to assess their relative potency. If one of the causes is war service, the disease is attributable to war service, even though there may be, more powerful causes, operating, and to which it is also attributable;
- d) war service must be a cause, as distinct from being part of the circumstances on or in which the cause operates; (This point stems from the earlier decision of the same judge in Minister of Pensions v Chennell in November 1946).¹⁰

Cases often occur where the disease would have arisen in any event, war service or no war service, in such cases the disease is predestined and not attributable to service.

⁹ Marshall V The Minister Of Pensions, ROSWPA, Vol 1, P 785

¹⁰ Chennell V Minister Of Pensions, ROSWPA, Vol 1, P 253

Article 5

Article 5 differs from Article 4 in placing the onus on the claimant, and in requiring reliable evidence to raise a reasonable doubt which establishes a causal link between the injury or disease and service. There is no case law on cases of unknown aetiology for Article 5 cases.

There is case law on the general interpretation of Article 5. The High Court has held that the word "reliable", in the context of Article 5, cannot have been intended to mean "convincing", but means more than "fanciful". In the case of *Edwards*¹¹ the judge held, with particular reference to "changes of medical opinion", that: "*there are... in my judgement, three stages: no reasonable doubt, reasonable doubt, and consensus.*" A war pensions claim under Article 5 would pass the test at the point where the (reliable) evidence raised a reasonable doubt, but: "*a mere hypothesis based on a limited study.... would not have created a "reasonable doubt" within the terms of Article 5(4)*". The real question, however, is whether the evidence raises a reasonable doubt in the mind of the Secretary of State (SoS). If he finds the evidence unreliable, it obviously will not raise a reasonable doubt in his mind.

When considering whether there is reliable evidence to raise a reasonable doubt, case law has established that all the available evidence must be weighed: The Courts have held that:

*"it is the duty of the claimant to produce reliable evidence, to establish his claim but if (after hearing and considering that reliable evidence and making a comparison between such evidence and other evidence which is called on behalf of the Ministry to contradict or to contravert it) the Tribunal has a reasonable doubt, then under those circumstances the plain meaning of that paragraph of the Article is that the benefit of that doubt shall be given to the Claimant".*¹²

Conflict of Medical Opinion

The Courts have also held that a conflict of medical opinion does not, of itself, mean that a reasonable doubt has been established, and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of *Tigg v The Minister of Pensions* the presiding Judge stated:

*"Merely because a doctor of eminence, and I have no doubt the doctor in this case was of very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean there is a doubt and the Appellant must therefore be entitled to a pension. It is a question of fact for the Tribunal".*¹³

¹¹ Edwards 1992 H C J No CO/2281/90

¹² Dickinson, ROSWPA, Vol 5, P 242 & Edwards, 1992 H C J No CO/2281/90

¹³ Tigg, ROSWPA, Vol 5, P 141

The above is reinforced by Howard in 1955:

*"the mere fact that there is a difference of medical opinion does not mean that there is a doubt and therefore the Appellant must have the benefit of it. There is only a doubt if the Tribunal, having considered the whole of the evidence, are left in doubt, in which case the burden of proof has not been discharged."*¹⁴

Both cases quoted are article 4 cases as they were made within seven years of leaving service however the principles cited are also applicable to article 5.

Reliable evidence

There may be research which suggests a service link and which raises a reasonable doubt, even if "the cause" of the condition is unknown. In considering, under Article 5, whether there is reliable evidence to raise a reasonable doubt that service caused the disablement of unknown aetiology regard has to be paid to whether conclusions of studies are hypotheses; or whether there is supporting evidence, which goes beyond hypotheses, and if so its basis, the reaction of other experts in the field to the evidence and the weight of overall evidence on the matter.

Conclusion

Article 4

The fact that the aetiology of a disease is unknown is insufficient to reject a claim on the basis that the disease is not due to service. Even if there has been some scientific study of the condition, and this has failed to identify any **material facts related to service** life that could be involved in its origin or aggravation, this is not sufficient to reject a claim under Article 4.

Under Article 4, whether 4(2) or 4(3), rejection of conditions of unknown aetiology is an onerous task. In each individual case, the Secretary of State has to have sufficient evidence to show beyond reasonable doubt that service played no part in causing or aggravating the condition.

This does not mean that it is impossible for the Secretary of State to reject a claim. If research evidence positively disproves a service link and there is no reasonable doubt that the evidence is reliable rejection will follow.

Article 5

Where disablement is claimed under Article 5, it is for the claimant to raise a reasonable doubt, by reliable evidence, that the claimed condition is attributable to, or aggravated by, service.

The Coe Rules are not applicable to Article 5 cases.

¹⁴ Howard, ROSWPA, Vol 5, P 515

If the position in relation to a condition is simply that the cause is totally unknown, then it may be that under Article 5 a claimant will be unable to raise a reasonable doubt in his favour by way of reliable evidence and thus a claim will normally not succeed. However, if the claimant produces reliable evidence which, after comparison with evidence held by the Department, causes the Secretary of State reasonable doubt then the benefit of that doubt is given to the claimant.

27 February 2001

Medical Adjudication Guidance on Alcohol

1. This is a reminder note on Departmental policy and the 1994 legislative change on injuries due to the use or effects of tobacco or consumption of alcohol SI 1994/772. You may find it helpful, in recalling the background, to re-read the guidance on [smoking](#).
2. Article 5(3) of the amendment Order 1994 amends Schedule 4 of the principle Service Pension Order by inserting a provision which limits the definition of "injury" in the principle Order. Schedule 4 part II of the SPO 1983 now defines injury as

"includes wound or disease but excludes any injury due to -

 - a. the use or effects of tobacco; or
 - b. the consumption of alcohol;

except that paragraph a., in so far as it relates to the use of tobacco, and paragraph b. above shall not apply where the person suffers from a mental condition which is attributable to service if -

 - i. the degree of disablement in respect of that condition has been assessed at 50% or more; and
 - ii. he started or continued to use tobacco or to consume or to continue to consume alcohol due to that condition".
3. The 1994 amendment did not introduce a new policy into war pensions. The Department, supported by case law, has long taken the view that disablement due to the effects of tobacco or alcohol cannot normally be attributable to service. This is because the decision to use these substances is a matter of personal choice.
4. In keeping with the generosity of the War Pensions Scheme and the intention to protect any individual whose disablement is truly caused by service, the amendment makes specific exception to exclusion of "injury due to use of tobacco or alcohol" if two particulars are satisfied. For certification of disablement which is due to alcohol or tobacco to be considered the following must apply. a. the pensioner must suffer from an accepted mental condition assessed at 50% or more and b. he must have started or continued to use tobacco or alcohol as a result of that medical condition's removal of his free will. Expert psychiatric opinion was consulted as to the degree of disablement specified. This included advice from the Addiction Unit at the Maudsley and confirmed that the 50% safeguard is very generous even when dealing with psychotic illness.
5. Legal advice confirms that injuries due to the "effects of consumption of alcohol" start with the first drink. Alcohol abuse syndrome can only develop by consumption of alcohol. This means that the amendment includes alcohol abuse syndrome itself and not just any consequent physical or mental conditions eg cirrhosis, Korsakoff's psychosis and peripheral neuropathy.

Medical adjudication guidance in claims for atherosclerosis

In conjunction with the [atherosclerosis appendix](#) dated February 1998, this minute provides guidance for decision-making in claims for atherosclerosis.

Consideration of each case on its merits remains fundamental. However for quality decisions made on a consistent and equitable basis, the following principles must be taken into account.

The appendix has been informed by literature review, discussion and correspondence with UK experts on cardiology and epidemiology.

The current appendix differs from earlier editions in several important ways.

- Since the Framingham study there is increased understanding of the pathogenesis of atherosclerosis but no increase in the risk factors identified. It is therefore reasonable to say that atherosclerosis is not now a disease of unknown origin.
- The current view of atherosclerosis emphasises the 2 separate processes of atherogenesis and thrombogenesis. This confirms war pensions practice dating back to at least 1961 of considering the underlying atherosclerosis and the acute event separately.
- Expert opinion also confirms that once in a steady state, a myocardial infarction has a direct and discrete amount of disablement. Underlying atherosclerosis is unaffected.
- Being of different pathogenesis the risk factors for atherosclerosis and acute manifestations may be different. Epidemiological studies confirm this.
- The progress of atherosclerosis is intermittent and can be reversed.
- Most war pension claimants are now 70+. We need to bear this in mind when discussing risk factors. Epidemiological studies have almost entirely looked only at young and middle aged men.
- With age there are normal and compromising changes in the cardiovascular system.
- Although it is accepted that the basic pathology of atherosclerosis is the same throughout the circulation, the risk factors may differ at different sites, eg, cholesterol level is a risk factor in myocardial infarction but not in cerebral infarction.
- Acute stress whether physical or mental, occurring within 24 hours of acute event is a risk factor in myocardial infarction. Reliable evidence has not been adduced to raise a reasonable doubt that acute stress is a risk factor for peripheral embolus, cerebral haemorrhage or infarction.
- Cholesterol level is a risk factor for myocardial infarction in the middle aged. Its relevance in myocardial infarction in the elderly or stroke is less clear.

Frequently claims and appeals are being seen where it is contended that exercise, stress or diet have played a part in the claimed condition. The following points are worth noting:-

A. Physical exercise - paras 56-61

Expert opinion is that the role of physical exercise in atherosclerosis is not confirmed.

It is however agreed that it does no harm and for this reason it is advocated in secondary prevention.

Well designed studies suggest that if exercise has a part to play, it is the **level of activity** or **energy expenditure** which is important. There is strong evidence that cardio-protective levels are outwith the scope of healthy old people.

Whether Article 4 or 5 applies, the contention that **immobility** causes or affects the course of atherosclerosis should not be accepted.

B. Stress - paras 66-75

As discussed above where there is reliable corroborative evidence of an acute stress (physical and mental) within 24 hours of an acute myocardial infarction certification will be appropriate.

Evidence does not support this in relation to peripheral embolus, cerebral infarction or haemorrhage.

However where it is contended that myocardial infarction or angina is due to service-related psychosocial stress we should reject under Article 4 or 5.

Please also see '[DSS response to 1997 paper by Professor P Sleight](#)'.

C. Diet - paras 49-50

Diet is usually a matter of personal choice. Even if the Secretary of State accepts dietary compulsions they would be time-limited, eg, during a training exercise. Information from MoD indicates that NAAFI menus have long reflected healthy eating requirements. The time course is broadly in line with civilian awareness. They also allow choice and accommodate special dietary requirements and since the 1970s MoD has been committed to health education.

In addition, atherosclerosis progresses intermittently, and can be reversed.

Service diet should not be accepted as causally related to atherosclerosis or its manifestations.

Other related topics

i. Assessment of MI

We should continue to certify using a dated label. Disablement resulting from an infarction is primarily due to death of cardiac muscle. It may include arrhythmia or heart block, heart failure or embolus to some other part of the circulation.

The associated degree of disablement should be stable when the individual reaches a steady state. Angina first manifesting in close time relation to a myocardial infarction is part and parcel. If angina is stable for a period of months or more following an accepted myocardial infarction, we should be careful to establish the precise reasons for any future increase before accepting increased disablement. Modern cardiology imaging is increasingly routine and will often allow precise aetiology of the angina to be determined, and if appropriate dissociated.

As ever, each case on its merits and many will best be discussed with ECW.

ii. Treatment

The acceptability of current anti anginal and anti hypertensive drug therapy has dramatically altered in the last few years. For an individual who is maintained in good health with repeat prescriptions and occasional general practice supervision an assessment of 6-14% should be considered.

If more disruptive follow up is required, eg regular anticoagulant clinic attendance, a higher assessment may be appropriate.

[Medical adjudication guidance in claims for atherosclerosis continued](#)

[back to Medical Adjudication Contents](#)

Myocardial infarction - relation to atherosclerosis

1. As you know I have been searching for a suitable published reference to support our practice of - as appropriate - accepting myocardial infarction (dated) attributable to service while at the same time rejecting the basic injurious process atherosclerosis. Dr J J Brown, MRC Hypertension Unit, confirmed that this was his understanding in a personal communication and expressed willingness to allow publication of his name. In view of all the issues about disclosure of names it was felt that this was not appropriate.
2. That clinical manifestation of ischaemic heart disease might involve a process other than atherogenesis was first observed by Morris (1951) in an analysis of post-mortem findings at the London Hospital. At a time when there was no increase - in fact a decrease in advanced atheroma - the mortality from coronary heart disease via sudden cardiac death and myocardial infarction was increasing several fold. Morris considered that a process separate from atherogenesis was involved.
3. The term "coronary thrombosis" was introduced in 1912 by Herrick. Despite this, most research until the last 10-15 years has concentrated on atherogenesis rather than the process leading to lumen occlusion.
4. By the late 1980s evidence had confirmed that thrombosis proceeds and causes transmural myocardial infarction (Chandler et al 1974; Davies(1987) and about the same time in the late 1980s the role of thrombosis in sudden cardiac death was established.
5. It is now established that there is a strong relation between haemostatic function and risk of coronary heart disease - in particular high plasma fibrinogen levels are important risk factors for clinical coronary heart disease. Environmental determinants of thrombogenesis include smoking and dietary fat intake. That means that dietary fat influences risk of coronary heart disease both through atherogenesis and separately through thrombogenesis. Evidence is also now emerging that the benefit of fat lowering regimes in reducing clinical incidents is mediated in the short term by effects on coagulability and thrombin production rather than the more difficult process of reversing atheroma.
6. The pathogenesis of atherosclerosis is now well studied (Davies 1996). There is no evidence that thrombogenesis leading to clinical myocardial infarction nor sudden cardiac death influences the underlying atherogenic process.

References

- Chandler AB et al 1974. Coronary thrombosis in myocardial infarction. Report of a workshop on the role of coronary thrombosis in the pathogenesis of acute myocardial infarction. *Am. J Cardiol*, 34, 823-33.
- Davies MJ (1987). Thrombosis in acute myocardial infarction and sudden death. Chapter 10, FA Divis, Philadelphia, PA pp 151-9.
- Davies MJ (1996). Atherosclerosis in Diseases of the Heart. 2nd Ed. 1996. Ed. Julian et al. Saunders London.
- Herrick JB (1912). Clinical features of sudden obstruction of the coronary arteries. *J Am Med Assoc* 23, 2015-20.
- Meade TW (1992). Coronary Heart Disease: Epidemiology from Aetiology to Public Health. Ed. Marmot et al. Oxford 1992
- Morris JN (1951). Recent history of coronary disease. *Lancet* i, 1-7, 69-73.

August 1998

RBL SUBMISSION (MARCH 2000) ON PSYCHOSOCIAL FACTORS AND ATHEROSCLEROSIS

The Issue

1. "What is the evidence that psychological stress or psychosocial factors¹ due to service in the armed forces may be causally associated with atherosclerosis, in particular, with clinical coronary artery disease?"

2. In considering this question in the context of war pensions it is important to remember that the key to war pensions entitlement is the presence of a causal link between service and the claimed disablement. Observed associations are not necessarily causal, but exposures are more likely to be so if:

- the observed association is strong
- the observed association is consistent amongst studies
- the observed association is specific
- the cause clearly precedes the outcome
- "more" of the cause produces "more" of the disease
- the association is biologically sensible
- there is human evidence of the association
- the proposed cause is in line with contemporary understanding.

Background

3. This matter has been carefully considered over the last few years. Following Professor Sleight's paper, the published evidence was evaluated. In March 1999 a Departmental Policy statement was produced, concluding that reliable evidence did not raise a reasonable doubt of a general causal link between service related psychological stress or psychosocial factors and coronary heart disease. Since War Pensions is an individual jurisdiction with each case considered on its own merits, where an acute coronary illness arises soon after a service-related stressful event or circumstance the two may be causally linked. No evidence was found supporting a causal effect where there is a long time interval between the stress and illness.

1999 Evidence

4. In 1999 the debate was further informed by two review papers^{2 3}, which explored the links between psychosocial factors and clinical coronary heart disease. Of the two reviews, the American Rozanski paper has a slightly different focus, being concerned with pathogenesis and implications for therapy. It also cites mainly animal studies. The human evidence reviewed overlaps with that of the other paper, and so this comment addresses only the Hemingway and Marmot Review.

5. The relation of stressor experiences to atherosclerosis and/or high blood pressure has been the subject of a considerable amount of research over the years. However, it is generally accepted that the published studies are of varied quality, often cross-sectional and retrospective, based on self-report and using a wide spectrum of definitions. Particular strengths of the Hemingway and Marmot paper are its focus on prospective studies, definition of "psychosocial factor", and use of a quality filter. Only published evidence meeting defined criteria is included in the review.

6. The paper records the limitations of systematic review in proving causation. It acknowledges the lack of standardised methods for measurement of psychosocial factors and draws attention to the need for an ideal systematic review to include all possible available studies on the topic. The authors confirm that their review paper has not been preceded by exhaustive search of the literature and may well have significant omissions. It is also true that for any review there is a danger of an incomplete picture because studies showing positive outcomes are more likely to be published than those suggesting no association. For a systematic review there should be direct liaison with researchers in the field.

7. The paper itself illustrates publication bias. It has appeared in two editions. That submitted for WPA consideration is an extract from the text "Evidence Based Cardiology" (Yusuf, S et al, eds 1998)². Another edition of the paper subsequently appeared in the British Medical Journal (Hemingway and Marmot 1999)⁴. In the 1998 edition, nine out of ten studies reviewed were reported as supporting an aetiological role for anxiety and depression in cardiovascular disease. In the later BMJ version, eleven out of eleven studies are reported as positive. The negative study included in the 1998 edition of the paper followed three groups of hypertensive American men and women for six years and showed an inconsistent effect of depression across the population. This paper (Simonsick 1995)⁵ was not cited in the May 1999 paper.

8. Turning to the review details, brief comment on the evidence is followed by the authors' conclusion that "in healthy population, prospective cohort studies suggest a possible aetiological role" in clinical coronary disease for four groups of psychosocial factors:

- (i) personality traits – type A personality/hostility
- (ii) social networks/support
- (iii) psychological states – depression and anxiety
- (iv) workplace factors

By their nature personality traits cannot be service exposures and so they are not further considered here.

The Evidence on Social Networks/Support

9. Although the literature does not invariably support this association, effective social network and support (both in terms of numbers and quality) would seem as a matter of common sense supportive of good health. The review looked at eight prospective studies of social networks/support and cardiovascular outcomes, five of which were positive. However, as the authors themselves concede, important questions about social networks and support remain unanswered. There are no agreed definitions nor consensus as to measurement. The structural and functional interactions of social networks and supports, how they might influence cardiovascular outcomes and the role of personality, remain matters of speculation.

10. The studies quoted focus on social support/networks around the time of the clinical incident or outcome. It is difficult to imagine how network/support factors related to service in the armed forces and producing no adverse effects at the time could influence clinical events many years later. While there may be exceptions, it is notable that war pension claimants of all generations, ranks, types and theatres of service speak in warm terms of the long lasting close comradeship, shared interests and values they first enjoyed while in the armed forces.

The Evidence on Psychological States – Anxiety and Depression

11. Examination of the evidence on the link between depression and anxiety and coronary outcomes is important because they may be accepted war pension disablements either as discrete conditions or as symptoms of another condition. The review looked at ten papers and records a grade B6 correlation between depression and anxiety and cardiovascular outcomes. The review does not include detailed narrative analysis of the papers. When this is carried out a rather different impression is obtained.

12. The first issue is the **populations under study** in the various papers. These were often highly selected eg particular cohorts of Harvard students or medical/paramedical personnel. There were also issues of **gender and age bias**. One study involved women only - rather more were exclusively middle-aged males. There was variable attention paid to the issue of selection bias in terms of those who volunteered for the study and complied with follow-up. The attrition rate in some studies was quite high and there is no real explanation for this nor assurance that confounding selection characteristics were not at work. All of these features suggest that caution is needed in elevating the conclusions of the various individual studies to the general conclusion that anxiety or depression causes cardiac disease.

13. Depression and anxiety are given different **definitions** in the various studies. Some studies concerned ill individuals and relied on clinician diagnosis, in some cases using DSM classification. Yet other studies involved well members of the general population and identify anxiety and depression traits purely on the basis of self-assessment. Nebulous subjective concepts like "hopelessness" (Everson 1996)⁷ and "vital exhaustion" (Appels 1990)⁸ were included and one study looked at "worrying" (Kubzansky 1997)⁹. It was questionnaire based and the results "suggested that high levels of worry in specific domains may increase the risk of coronary heart disease in older men". That is to say it was only worrying about some things - in this case affairs of state, political issues - that was claimed to be associated with increased risk of heart disease.

14. The studies were all at least in part prospective ie the anxiety or depression was present at the outset of the study and pre-dated the cardiac outcome. Most of the studies, however, employed a single measurement of the exposure under study made at one point in time. There was no indication as to whether individuals were depressed or anxious during the whole period of follow-up, or for only a very short period at the outset of the study, or whether the psychiatric condition or symptom waxed and waned. In addition episodes of depression and anxiety could not be excluded in the controls since the criteria for being accepted as a control was simply absence of anxiety or depression at the onset of the study. An exception to this was the SHEP Co-operative Research Group Study (Wassertheil-Smoller et al 1996)¹⁰ study which showed among elderly persons an excess risk of death, stroke and myocardial infarction associated with an increase in depressive symptoms over time. In this paper, baseline depressive symptoms were not associated with these outcomes.

The Evidence on Workplace Factors

15. Much of the evidence on psychosocial factors and coronary disease comes from work-place studies and theoretical concepts of occupation strain. The most widely known model is that of Karaseck and Theorell¹¹ which proposes that stressful jobs are characterised by a combination of high psychological workload demands and low control or decision latitude. These concepts are not self-evident but have specific definitions. A job with high demands requires workers to work fast and hard to accomplish an excessive amount of work in too short a time. Low decision latitude or control is present where workers cannot make decisions on their own and where there is also lack of skill, discretion/opportunity to learn new skills, develop aptitudes or abilities and a great deal of repetition. A body of evidence suggests a positive association between job strain and risk of coronary heart disease. The evidence is, however inconsistent and incomplete. Various occupations, eg armed forces, are completely omitted and other groups, eg some races and women, are under-represented in studies. Comparatively little work has been done on possible modifying effects such as personality type and good social support. Some studies suggest clustering of negative factors like job strain, depression, hostility, isolation in some people¹² and other reports¹³ find no correlation between job strain and clinical coronary disease.

16. Against this conflicting picture, proposals for alternative job strain models are emerging. In the effort-reward imbalance model, there is emphasis on both intrinsic effort, ie the individual's need for control – a measure of personality and abilities and extrinsic effort specified by a high workload. On the rewards side of the equation are money, esteem and occupational status control (promotion prospects and job security).

17. The Hemingway and Marmot Review looked at ten workplace studies. In some, job strain was assessed by self-report and in others more objective ecological assessment from job title was available. Six out of the ten studies were reported as positive. How and why job strain might lead to coronary disease remains unknown. The review records the growing emphasis on control at work rather than on demands, and acknowledges that the occupational strain models are being reformulated. The published literature on job strain almost entirely focuses on currently employed subjects in stable work situations and where the clinical illness is sustained while the individuals are still in the workplace.

General Points

18. The studies of the Hemingway and Marmot Review covered a range of pathological outcomes eg angina,

acute myocardial infarction, acute cardiac death with findings inconsistent across the range outcomes. In other words in some studies there was an association with sudden cardiac death, in others with angina. Diagnosis in the papers sometimes depended on clinical examination and medical investigation. In other studies it was based on self-report by the subjects, a frequent outcome being angina pectoris diagnosed by questionnaire with no medical corroboration. At least one of the studies cited makes reference to the fact that a proportion of people claiming angina pectoris at first screening later go on to deny its existence.

19. The quality filter imposed a minimum number of subjects ie numbers of clinical cardiovascular events in the studies suitable for review. However, in all the studies the numbers of subjects experiencing the outcome under investigation were very small. In this situation, small inaccuracies in diagnosis of outcomes could produce large effects on outcome incidence.

20. The review quotes relative risk for the various studies but does not include information on their confidence intervals. In many cases scrutiny of the original papers confirms this is wide.

Conclusion

21. In conclusion there is a clear risk of over-interpretation of the data presented in the review. It should be noted that the review conclusion begins "Large prospective studies examining specific psychosocial hypotheses suggest the importance of psychosocial factors in relation to CHD (coronary heart disease) aetiology and prognosis."

Other Evidence Submitted in 1999

22. At around the same time the report of the proceedings of the 1998 Australian Repatriation Authority Conference on the effects of military stress on psychiatric illness and cardiovascular disease was published.¹⁴ The executive summary and conference overview confirms and expands the conference conclusions quoted in the Department's policy statement. In relation to exposure to stressors and ischaemic heart disease it contains the following:-

"When considering experimental stressors (brief, standardised, replicable, distressing and frustrating activities, for example mathematical manipulation, simulated public speaking) or studies encompassing the effects of natural disasters, life event stress and work stress, support for an association between experiencing acutely stressful events and acute coronary events was found. Negative emotions and certain personality traits may also contribute to negative outcomes in patients with established ischaemic heart disease. The role of stressful experiences long distant in time from the physical disease endpoint is much less clear and the available information is not yet at a level where causality may be inferred."

23. The standard of proof in Australia for veterans' pension is more generous than in the UK requiring only a "reasonable hypothesis" of a link between service and the claimed condition.

24. The overall conclusion on the basis of this further evidence was that the causal role of psychosocial factors in health and disease remained poorly understood and that to inform war pensions decisions, the Departmental policy statement, and current medical appendices on atherosclerosis (December 1998) and hypertension (October 1996) remained appropriate.

2000 EVIDENCE

25. In March 2000 the RBL submitted a brief comment by Professor Sir M Marmot on the Departmental response to the 1999 evidence and a pre-publication overview paper on the impact of socioeconomic status (SES) and psychosocial factors on pathological measures of sub-clinical atherosclerosis¹⁵. This is a chapter

from a new text "Stress and Coronary heart disease" edited by Stansfield, S and Marmot, M and to be published later this year.

26. We note Professor Marmot's comments but remain of the view that for lawful, evidence-based decision-making in war pensions the present Departmental approach remains appropriate, with each case considered on its specific facts.

27. Much work has now been completed on the pathogenesis of atherosclerosis. The process is prolonged from initiation in childhood or youth to clinical events in later life. It is dynamic and there is evidence in humans of progression and regression. While many of the histopathological and molecular changes have been analysed, theories as to the inter-relationships of the various stages both with each other and with clinical outcomes, remain just that.

28. Dr Hemingway's paper notes first the findings of the 1999 Hemingway and Marmot review conceding that such observed associations do not establish causation. He then proposes that a further test of the hypothesis that psychosocial factors cause coronary heart disease may be obtained by looking at sub-clinical atherosclerosis. Amongst advantages of that approach is that outcome cannot be biased by the exposure under study. The paper reviews the evidence that socioeconomic status and psychosocial factors influence arterial and myocardial structure and function. The measures of arterial structure used in the studies reviewed are carotid intima-media thickness (IMT) and coronary angiography. For arterial function, the surrogates are endothelial dysfunction (ETD) and arterial stiffness, and for myocardial structure, left ventricular mass (LVM).

29. At this date, evidence in humans on the link between psychosocial factors and arterial function and myocardial structure, is very limited. Although there is some evidence in healthy humans of an association between the classic coronary risk factors and endothelial dilatation (ETD) there is no evidence on psychosocial factors. The situation is similar for arterial stiffness. The 1992 Framingham study¹⁶ reported that left ventricular hypertrophy was a strong predictor of clinical cardiovascular disease and since then research emphasis on the heart muscle itself rather than the coronary arteries, has increased. Evidence in humans on a link between psychosocial factors and left ventricular mass is emerging but the studies are few and results conflicting.

30. Increasing availability of angiography and B-mode ultrasonography for measurement of atherosclerosis has led to rather more published studies on arterial structure and psychosocial factors. Because of their ready accessibility in the neck, intima-media thickness is usually measured by ultrasonography of the carotid arteries. That this is a reasonable proxy for coronary artery atherosclerosis is suggested by the fact that post-mortem studies show good correlation of the two¹⁷, by the presence of shared risk factors¹⁸ and by the fact that carotid artery IMT correlates with incident clinical coronary events.¹⁹ The reviewer identifies fifteen studies on socioeconomic status and psychosocial factors and IMT, reporting them all as positive.

31. A number of the studies are based on the same population, viz. the Kuopio study group from East Finland comprising middle-aged men at high risk of atherosclerosis. Most studies are of men only, two are of women only.

32. There is variation in the techniques of B-mode ultrasonography and use of measures to minimise potential intra and inter observer bias. The studies measure IMT at different sites of the arteries – some look at mean and some at maximum thickness. Some studies are cross-sectional in design, others are prospective with follow-up from 18 months to ten years. Most include adjustment for confounders. This is sometimes for age alone, for age and baseline pathology and in yet others, for a wide range of the conventional atherosclerosis risk factors.

33. The psychosocial factors considered also vary. Five studies looked at socioeconomic status, variously in childhood, adult and over a lifetime. One reported on social support/networks, one at discontent, one hopelessness and one, anxiety/hostility. Some studies were concerned with occupation and workplace demands and some with cardiovascular reactivity both in isolation and in the context of the workplace. These

diverse features of the individual studies, coupled with the lack of understanding of the inter-relationships of the various factors, caution against over-interpretation or extrapolation to the general of the individual study findings.

34. The review does not provide in-depth comments on the individual studies, their design, robustness etc and it is notable that the tabulated results (Table 2, page 24/25) are selective and not the only findings of the individual studies.

- The first paper listed in the table is the 1995 Diez-Roux²⁰ study which involved about 16000 subjects aged 45-64 years of different race and gender and from four US communities. Table 2 records the result as showing "inverse linear association in each sex, race group for each SES measure". The paper itself qualifies this by noting that the trend linking the education measure of socioeconomic status with IMT was much clearer in whites than blacks. No explanation is available.
- The Agewell (1996)²¹ paper looked at whether quality of life (as measured by the validated Minor Symptoms Evaluation Profile (MSEP) questionnaire) in about 100 hypertensives was associated with increasing IMT of the common carotid artery over the 3.3 year follow-up. The MSEP has three quality of life measures, contentment, vitality and sleep. As recorded in Table 2 progression was associated with discontent. It was not associated with the other two measures. This finding introduces the possibility of a purely random effect.
- The 1997²² Lynch paper showed significantly greater maximum IMT but not mean IMT, in men with low demand, high-income jobs.
- The Kamarck (1997) study²³ associated cardiac reactivity and IMT (max and mean) progression but only in the youngest subjects.

Finally, the 1999 Ebrahim et al²⁴, British Regional Heart Study began by stating that "The reported associations between cardiovascular risk factors, clinical disease, IMT and plaques are inconsistent."

It stressed the importance of plaques in predicting clinical events and took the view that some of the inconsistency in the findings might relate to the method and particularly the site of measurement of intima-media thickness (IMT). Its findings were that IMT of the common carotid artery, IMTCCA, IMT of the carotid bifurcation, IMT bif and plaque are correlated with each other but show differing patterns of association with risk factors and clinical disease. In their population, IMT CCA predicts stroke while IMT bif and plaque correlates with coronary risk.

35. Hemingway's new review concludes that both the Hemingway and Marmot review on clinical outcomes and the new work on sub-clinical atherosclerosis show socioeconomic status and psychosocial factors as "independent" associations of the outcomes. This is based on the fact that observed associations persist after adjustment for the standard atherosclerosis risk factors. Such a finding may however be premature or too simplistic.

36. It is well recognised that where multivariate modelling is used, drawing conclusions about the independence of particular risk factors is risky, particularly where there may be room for multiple measurement error.²⁵ In the studies, single baseline measures of the atherosclerosis risk factors were used with no account taken of possible variation over study duration. The psychosocial factors and Socio-economic status are associated with the classic risk factors, both at the baseline and over time. Further, the "confounder" risk factors included in the studies did not include all known atherogenic risk factors, far less any still to be identified. It is of note that the 1995 Lynch study²⁶ of socioeconomic status and carotid structure on the Kuopio population comes to a quite different conclusion. While recording a strong association between socioeconomic status and carotid atherosclerosis it concluded that "the results show that this association was mediated by known atherosclerotic risk factors".

37. Dr Hemingway himself summarises the main findings of the review as "**suggesting** that sub-clinical atherosclerotic mechanisms are involved in the psychosocial-CHD association". He acknowledges the need for further work, the gaps in the evidence so far in terms of the range of exposures examined, and the desirability of a more systematic approach.

How Will This New Evidence Alter War Pensions Practice?

38. War pensions is paid not for biochemical or sub-clinical pathological change but for disablement caused by injury due to service, disablement being defined in the legislation as "physical or mental injury or damage or loss of physical; or mental capacity". The inter-relationships between socioeconomic changes and clinical disease over a lifetime and the dynamics of atherosclerosis, regression and progression at sub-clinical level remain to be established.

39. Underlying lawful war pension entitlement is the presence of a causal link to service. It is difficult to see how socioeconomic factors can ever be service exposures. Rather they seem matters of constitution, temperament, early and whole life experience and socio-cultural context.

40. The evidence on occupational stress is potentially relevant to individual war pension decisions but at present is complex, contradictory and with new models emerging. There seems a need to fully evaluate not just job characteristics but the possible mediating effects of individual personality, social support and team spirit. This last is particularly relevant in the armed forces' environment. Despite many studies from US, Australia and UK, there remains a lack of evidence that FEPOWs and other ex-POWs, whose occupational stress is unquestionable in terms of high demands and low control, have increased risk of coronary heart disease²⁷. The Department's view of service life remains as set out in the Policy statement.

41. War pension can be claimed at any time after service release with the bulk of claims for coronary disease lodged many years after service. We have carefully considered Dr Hemingway's new work and noted the findings of the 25 year follow-up of civil servants from the first Whitehall study²⁸. This confirmed that while socioeconomic differences in mortality persisted after retirement, the role of occupational status as a measure of socioeconomic status (but not non-work measures) decreased after retirement.

Conclusion

42. From the overall available evidence, we therefore remain in agreement with the Australian RMA Consensus Conference conclusions and continue to consider that, while cases will be decided on their specific facts, reliable evidence does not raise a reasonable doubt that, in general, stressful events or circumstances long distant in time from clinical coronary disease are causal.

1 A measurement which may relate psychological phenomenon to the social environment and pathological changes

2 Hemingway, H and Marmot, M (1998) Psychosocial Factors in the Primary and Secondary Prevention of Coronary Heart Disease: a systematic review Chap 18: 269-285 in Yusuf, S et al 1998 Evidence Based Cardiology, London BMJ Books

3 Rozanski, A et al (1999) Impact of psychological factors in the pathogenesis of cardiovascular disease and implications for therapy. *Circulation*: 99:2192-2217

4 Hemingway, H and Marmot, M (1999). Psychosocial factors in the aetiology and prognosis of coronary heart disease: systematic review of prospective cohort studies. *BMJ* 318: 1460-1467

5 Simonsick, EM et al. Depressive symptomatology and hypertension associated morbidity and mortality in older adults. *Psychosom Med* 1995; 57: 427-435

6 Grade B evidence is defined as "moderate evidence from observational studies suggesting an effect but the evidence is conflicting or observation studies constitute the only evidence, ie, no random controlled trials".

7 Everson, S A et al. Hopelessness and risk of mortality and incidence of myocardial infarction and cancer. *Psychosom Med* 1996; 58: 113-121

8 Appels, A, Mulder, P. Excess fatigue as a precursor of myocardial infarction. *Eur Heart J* 1990;9: 758-764

9 Kubzansky, LD et al. Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the Normative Ageing Study. *Circulation* 1997; 95:818-824

10 Wassertheil-Smoller, S et al. Change in depression as a precursor of cardiovascular events. *Arch Intern Med* 1996; 156:553-561

11 Karaseck RA and Theorell, T (1990), *Healthy Work: Stress, Productivity and the Reconstruction of Working Life*, New York. Basic Books Inc.

12 Williams, R et al (1997). Psychosocial correlation of job strain in a sample of working women. *Ann Gen Psych*: 54:543-548

13 Hlatky, M et al (1995). Job strain and the prevalence and outcome of coronary artery disease. *Circulation*: 92:327-333

14 Morris, P et al (eds) *Stress and Challenge. Health and Disease Proceedings of the RMA Consensus*, Brisbane, 9-11 February 1998, Brisbane RMA

15 Hemingway, H (2000) Arterial and myocardial structure and function: contributions of socioeconomic status and chronic psychosocial factors

- 16 Kannel, WB, Cobb J. Left ventricular hypertrophy and mortality - results from the Framingham study. *Cardiology* (1992) 81: 291-298
- 17 Mather, K et al (1963) Correlation of the extent and severity of atherosclerosis in the coronary and cerebral arteries *Circulation* 27: 929-934
- 18 Sharrett, A et al (1999) Relative importance of various work factors for asymptomatic carotid atherosclerosis versus coronary heart disease incidence: the ARIC study, *AM J Epidemiology* 149: 843-852
- 19 Salonen, JT et al 1993 Ultrasound B-mode imaging in observational studies of atherosclerotic progression, *Circulation* 1993 87: 1156-1165
- 20 Diez-Roux, AV, et al (1995). Social inequalities and atherosclerosis. *A. J Epidemiol*; 141: 960-972, 21 Agewall, S, Negative feelings (discontent) predict progress of intima-media thickness of the common carotid artery in treated hypertensive men at high cardiovascular risk. *AM J Hypertens* 9: 545-550
- 22 Lynch, J et al (1995). Socioeconomic status and carotid atherosclerosis. *Circulation* 92: 1786-1792
- 23 Kamark, TW, (1997). Exaggerated blood pressure responses during mental stress are associated with enhanced carotid atherosclerosis in middle-aged Finnish men: findings from the Kuopio Ischemic Heart Disease Risk Factor Study. *Circulation* 96: 3842-3848
- 24 Ebrahim, S, et al (1999). Carotid plaque, intima media thickness, cardiovascular risk factors, and prevalent cardiovascular disease in men and women: the British Regional Heart Study. *Stroke* 30: 841-850
- 25 Lui, K (1988) Measurement error and its impact on partial correlation and multiple linear regression analyses. *Am J Epidemiol* 127: 864-874
- 26 Lynch, J et al (1995). Socioeconomic status and carotid atherosclerosis. *Circulation* 92: 1786-1792
- 27 Gale, C et al (1999) , Morality from Parkinson's disease and other causes in men who were prisoners of war in the Far East, *Lancet*, Vol 354: Dec 18/25 , 1999
- 28 Marmot, MG and Shipley, MJ (1996). Do socioeconomic differences in mortality persist after retirement? 25 year follow-up of civil servants from the first Whitehall study. (*BMJ* 313:1177-1180)

June 2000

Dr E A Braidwood

War Pensions: Psychological stress/psychosocial factors and coronary heart disease: DSS response to 1997 paper by Professor Peter Sleight:

Contents:

[Policy Statement](#)

[The Law](#)

[Basic Concepts](#)

[Study by Australian Department of Veterans Affairs](#)

[Conclusion](#)

Policy statement

Policy summary

War Pension is awarded for any disablement causally linked to service. Anyone who has served in the armed forces can claim pension at any time from service release. War Pension is medically certified. Decisions are evidence based reflecting the facts of the case, the relevant law and policy and contemporary medical understanding of the causes of the claimed condition.

Psychological stress is a normal and essential part of life. There is no agreed definition - the term is variously applied to both demands and the associated response on an individual - and it is highly subjective. One man's stress is another's stimulus. Service in the armed forces is therefore not, of itself, stressful.

Professor Sleight's paper refers to studies which propose a causal link between psychological stress and various diseases including coronary artery disease.

Atherosclerosis, the process underlying coronary disease is due mainly to constitution and ageing. In addition, major environmental risk factors have been identified. In many individual cases of coronary disease the principal risk factors, such as cigarette smoking and high serum cholesterol, do not appear to be present; other possible causal factors, including psychological stress, have therefore been explored. Much of the evidence on stress relates to the workplace, particularly the individual's control over events and decisions.

The contemporary evidence has been studied and it is concluded that a reasonable doubt is not raised that there is a general causal link between service linked psychological stress/psychosocial factors and coronary heart disease. Where an acute coronary illness arises soon after a service related stressful event or circumstance, the two may be causally linked.

Each case must be determined on its own individual circumstances and evidence.

The issue

1 In a paper dated October 1997, Professor Peter Sleight, emeritus Professor of Cardiovascular Medicine at Oxford, proposes a causal association between psychological stress/psychosocial factors in the service setting and coronary disease. Informed by contemporary medical evidence including papers cited by Professor Sleight the Department examines that alleged association in this policy statement.

Medical adjudication guidance in claims for atherosclerosis continued

[back to Medical Adjudication Contents](#)

Annex A

Atherosclerosis and ex-FEPOWs

Since the early follow up studies (1) on the health of ex-FEPOWs, information on ischaemic heart disease and hypertension has been included. The early results based on data from the 1950s and 60s were inconsistent, tending to support (2) higher morbidity rates from ischaemic heart disease and high blood pressure with at the same time lower mortality (3). Richardson's 1965 Canadian Study on Hong Kong veterans (4) produced opposite findings, ie, no increase in high blood pressure or coronary disease, but slightly higher death rate overall and highly significant death rate for coronary disease. Possible explanations include small study numbers, their reliance on self-report and self-diagnosis and the young age of the ex-FEPOWs at this time and corresponding expected low prevalence of these conditions.

By the 1980s a more consistent picture was emerging and studies in both the US (5) and UK (6 and 7) found no evidence of increased morbidity or mortality from ischaemic heart disease. This pattern has continued to be observed in Australia (8, 9, 10), the US (11 and 12) and in the UK (13).

Because they are predominantly diseases of old age, data on cerebrovascular and peripheral vascular disease is limited. There are in any case few systematic data on prevalence of these conditions in populations (14) in general. At present (13, 15) reliable evidence does not raise a reasonable doubt of increased prevalence of these conditions in the ex-FEPOW population.

References

1. Cohen, BM et al (1954) A follow-up study of World War II prisoners of war. *Can Med Assoc J*; 56: 475-481
2. Beebe, GW (1975) Follow-up studies of World War II and Korean war prisoners: II. Morbidity, disability and maladjustments. *Am J Epidemiol*; 101: 400-422
3. Nefzeger, Md (1970) Follow-up studies of World War II and Korean war prisoners. I. Study plan and mortality findings. *Am J Epidemiol*; 91:123-138
4. Richardson, HJ (1965) Report of a study of disabilities and problems of Hong Kong veterans 1964-1965. Canadian Pensions Commission, Ottawa
5. Keehn, CZ (1986). Follow-up studies of World War II and Korean war prisoners. III. Mortality to January 1, 1976. *Am J Epidemiol*; 111: 194-211
6. Gill, GV, Bell, DR (1981). The health of former prisoners of war of the Japanese. *Practitioner*; 225: 531-538
7. Gill, G (1983) Study of mortality and autopsy findings amongst former prisoners of war. *J R Army Med Corps*; 129: 11-13
8. Adena, MA (1989) The Health of Australian Veterans. *Med J Austral*; 150:356-357
9. Dent, OF et al (1989) Postwar mortality among Australian World War II prisoners of the Japanese. *Med J Austral*; 150:378-382
10. Venn, AJ et al (1991) Chronic morbidity of former prisoners of war and other Australian veterans. *Med J Australia*; 155:705-712.

11. Eberly, RE et al (1991) Prevalence of Somatic and Psychiatric Disorder Among Former Prisoners of War. *Hosp and Comm Psychiat Vol* 42:807-813
12. Oboler, S (1987) American prisoners of war – an overview. In Williams T (ed). *Post-traumatic Stress Disorders. A Handbook for Clinicians, Disabled American Veterans*, Cincinnati, Ohio, pp 131-143
13. Gale, C et al (1999) Mortality from Parkinson's disease and other causes in men who were prisoners of war in the Far East. *Lancet*; 354: 2116-2118
14. Page, WF (1992) *The Health of Former Prisoners of War*. Washington, National Academy Press
15. Creasey, H et al (1999) Is Experience as a Prisoner of War a Risk Factor for Accelerated Age-Related Illness and Disability? *J Am Geriatr Soc*; 47:60-64

Medical adjudication guidance on H pylori and gastro-intestinal disease

This paper expands the [Training Minute dated 25 April 1996](#).

Under Article 4 and with onset in service, the dyspeptic conditions, gastritis, peptic ulcer and gastric cancer are usually certified as attributable.

In Article 5 cases if there is in service "gastritis" (NB not gastro-enteritis) circumscribed and self limiting, and no further dyspeptic illness for many years when a peptic ulcer or gastric cancer is confirmed, gastritis (dated) attrib nil should be certified and the post-service condition rejected.

In a claim for dyspepsia with no record of in service upper gastro-intestinal problem and symptomatic onset of peptic ulcer or gastric cancer many years post service rejection is appropriate.

Recent advances in the understanding of H pylori requires review of these practices.

The organism now known as H pylori was first observed in the human stomach in a case of gastric cancer in 1906. Little more was heard of it until the development of the flexible endoscope. In the 1970s it was observed in different clinical situations in the upper gastrointestinal tract. In 1984 the organism was cultured. Biochemical and ultrastructural characterisation followed and in 1989 it was assigned its own genus.

Advances in serology then enabled H pylori disease associations to be studied. The prevalence rate of H pylori was established for different communities, ages, sexes and in relation to different clinical and pathological entities. As usual many of these initial studies were open to criticism, statistical and technical, but a number of consistent observations were established. Immunological evidence also supports the view that H pylori in dyspeptic disease is not a harmless commensal.

In the Western World H pylori infection is common in the community and increases with age from around 20% in 20 years olds to approximately 75% of 60 year olds. In the Third World infection is even more common in young people and by middle age the evidence suggests almost universal carriage of the organism in some communities.

H pylori may be associated with histological abnormality. Most commonly it is seen in chronic active gastritis where a causal relationship is favoured.

70% cases of gastric ulcer have H pylori present and in duodenal ulcer 95-100% infection is seen. However in peptic ulcer a causal role is not yet universally accepted by medical experts. The evidence is that peptic ulcer, whether gastric or duodenal, is multifactorial in origin. Of those infected with H pylori only a proportion develop peptic ulcer disease, ie H pylori is one factor in the development in the clinical syndrome. It has however been shown that there is a decrease in the clinical relapse rate of peptic ulcer if treatment includes a course of anti-infective therapy. This suggests that H pylori is a major risk factor in the genesis of peptic ulcer disease.

The relation of H pylori to gastric cancer is even less clear. Again this condition is multifactorial in origin. H pylori may be present in gastric cancer but the nature of

this association is not known. In Africa there is a high H pylori prevalence but the gastric cancer rate is low. This tends to reduce the prospect of a possible causal role.

Effect of these observations on war pensions certification

- The present medical understanding is that the case causally linking gastritis and H pylori is strong. A reasonable doubt is raised of a causal association with peptic ulcer but that position has not yet been reached for gastric cancer and H pylori.
- In certification terms current policy in Article 4 cases is sound.
- In Article 5 cases where the dyspeptic process has earliest manifestation in the post service period rejection remains appropriate. Review of present practice is appropriate in cases where there is both "in" and "post service" dyspepsia.
- Service medical documents may contain reference to alcohol or therapeutic drugs as a cause of gastritis but in many cases the cause of in-service gastritis is not clear and there will be a reasonable doubt that it was caused by H pylori. In such cases, in-service gastritis (dated) should be certified attrib.
- The in-service episode of gastritis may be very short and there may be no evidence of ongoing dyspeptic problem until many years later when a peptic ulcer is confirmed. We should not now automatically reject peptic ulcer. Rather we need to consider a causal link beginning with the in-service gastritis and leading to certification of peptic ulcer attrib.
- In cases where the onset of dyspepsia due to gastritis or peptic ulcer is post-service and there is no evidence that the claimant experienced upper gastrointestinal symptoms in service, then the condition(s) should be rejected.
- The claimant may contend that he became infected with H pylori during service, with subsequent development of peptic ulcer. Current bacteriological knowledge does not support this contention. In particular, there is no evidence that military conditions predispose the individual to H pylori infection. The present practice of rejecting these claims should remain unchanged.
- The relationship between H pylori and gastric cancer remains to be elucidated. At present, if there is an episode of in-service gastritis (circumscribed) and gastric cancer arises late post-service, this will be for rejection.
- If there is an in-service dyspepsia followed by ongoing problems, then that case should be decided on its own merits. It would be sensible to discuss such a case with the Training Officer.

Summary

Article 5

1. Gastritis (dated) arising in service - attributable.
2. Peptic ulcer arising subsequent to in-service gastritis - attributable. The date of change of medical opinion is 1 March 1993.
3. Gastric cancer arising post-service where there has been a circumscribed episode of gastritis in service - NANA.
4. Gastric cancer arising post service, dyspepsia in service with ongoing problems - each case to be decided on its own merits.

Article 4

Gastritis, peptic ulcer and gastric cancer arising in service are normally attributable. The date of change of medical opinion is 1 March 1993 which is the publication date of the paper by Tytgat, S N, Loads, L A, Rawws, E A. H Pylori Infection And Duodenal Ulcer Disease. Gastroenterology Clinics of N America 22(1): 127-39.

MEDICAL ADJUDICATION GUIDANCE IN GULF CASES

Because of the uncertainty, speculations and sensitivities of Gulf-related illness it is particularly important that our decision-making is consistent and equitable reflecting War Pensions law, policy and current sound medical evidence.

This guidance aims to present current medical understanding. It provides:-

- i. background information on the conflict and the context of Gulf-related illness,
- ii. summarises medical policy aspects of Gulf claims processing,
- iii. comments on the various contentions seen in these claims and finally
- iv. there is a brief claims analysis to date.

Background information

About 51,000 British troops took part in the Gulf War 1990-91. The air war began on 16. 1.91, lasted 39 days and was followed by a ground war of 4 days.

The conflict had a number of distinctive features:-

1. Combat casualties and non-battle injuries and disease were much lower in absolute numbers and in incidence than in other military campaigns.
2. There was a high proportion of reservists for whom the Gulf was their only experience of active service.
3. The conflict was brief, lacked a clear outcome and did not result in a triumphant return to UK.
4. Many personnel came back to a redundancy notice as part of the post Cold War defence cuts.
5. The conflict was in an alien environment with risk of unusual infectious disease. The decision was made to employ an accelerated pre-deployment vaccination programme (including unlicensed vaccines).
6. The use of chemical and biological weapons was anticipated and countermeasures were arranged.
7. The MOD has acknowledged that record keeping in the Gulf was inadequate.
8. Appropriate preventive preparation for PTSD and debriefing after the event were not widely available. In addition most of the troops returned by air, not sea and lost the opportunity of a "DIY" psychological debriefing as seen eg in the Falklands.
9. In September 1996, five years after the war, the Ministry belatedly confirmed the use of organophosphorus pesticides in the Gulf.
10. It must be remembered that the majority of claims made by ex-Gulf personnel (defined by War Pensions Agency as those where service includes service in the Gulf) related to illness and incidents at some other part of the service. At the end of

October 1998 there had been approximately 3,300 Gulf claims of which 875 relate to Gulf-related illness.

11. It should be noted that it is unlikely that all Gulf claims have yet been made. Many regulars deployed to the Gulf are not yet discharged from service. Gulf participants who were regular servicemen generally returned to units and continued to serve - with no documented adverse effect.

[to top of the page](#)

12. By late 1993 a small number of ex-Gulf personnel began to complain of vague ill-defined illness whose symptoms often had onset at an interval from the conflict. A similar pattern was emerging in US. In the US with 600,000 troops involved, a Presidential committee was set up and an extensive programme of research including epidemiology/toxicology/psychological evaluation was begun. From the outset progress updates and results have been available to the UK.

13. In October 1993, the MOD set up the Gulf Assessment Programme. From then until January 1996 the programme attracted only a trickle of personnel (about 300). In January 1996 the Gulf Assessment Programme was opened to serving soldiers and since then the numbers being screened has risen.

14. Gulf-related illness has attracted much media and political speculation and uninformed comment. No research anywhere in the world has yet identified a specific Gulf-related illness. The label Gulf War Syndrome/Desert Fever etc remains therefore inappropriate for War Pension purposes.

Medical policy aspects of Gulf-related illness claims handling

In essence claims from Gulf participants are exactly as any other claim with decisions made in the light of relevant law:policy guidance and the specific facts of the case. At present Article 4 - usually 4(2) - applies to Gulf claims and there is a corresponding high rate of certification.

The Agency and Department has been subject to much criticism about delays in handling Gulf claims. Much of this relates to the nature of the conditions claimed and the evolving nature of the claims.

a. Conditions claimed are characterised by their vagueness/minor nature and symptoms are frequently within normal variation. They often develop at an interval from the conflict and frequently claimants have not sought medical attention prior to the claim being made. It is essential to remember that it is first for the claimant to show, on the balance of probabilities, to the satisfaction of SoS that the claimed disablement is present. Merely listing the claimed symptoms on the claim form does not fulfil the requirement.

b. Disablement shown - the onus of SPO Article 4 becomes operative.

c. Evidence gathering in Gulf cases is complex and often protracted. Records of the Gulf Assessment Programme are now usually available and, following express permission of the claimant, are a useful source of evidence often able to replace Consultant reports.

d. Claims analysis confirms that accepted disablements generally fulfil specific diagnostic criteria. Where, following a reasonable amount of evidence gathering, a specific diagnosis remains unclear, the ICD-9 designation, SSIC (symptoms, signs,

ill-defined conditions) should be used. This label can be defended with a suitable explanation at appeal. (Article 67(3aa) will allow review and revision in the light of future research if entitlement has been accepted. We of course have carte blanche if there is no certificate).

[to top of the page](#)

Labels

Where the labels CFS:PTSD:fibromyalgia are considered they must fulfil the accepted Department standard criteria as indicated in previous minutes. If these diagnoses have been used based on any other criteria, this should be explained and the label changed.

The label organophosphorus toxicity should not be used unless there is clinical evidence of acute reaction in the Gulf. This is because present medical understanding - including the recent Institute of Occupational Medicine study - do not suggest the existence of a defined syndrome of chronic low dose organophosphorus toxicity.

Similarly Vaccination Reaction. Much has been made in claims of the possibility of illness resulting from the use of vaccine combinations. Even in the very young combination vaccination is not infrequent and most countries have approved rapid immunisation programmes for foreign travel. Such a regime presents a considerable immunological challenge. Indeed that is the purpose. There is no current evidence that this sort of approach compromises the immunological system either short or long term.

Notes on the specific exposures contended by claimants as causal

a. Vaccination

It is well known that vaccines can produce immediate minor localised (or very rarely) more generalised reactions. The evidence is that the majority of these subside in 48 hours. There is at present no evidence that vaccination can lead to delayed onset symptoms in the absence of severe acute reaction.

The vaccines used in the Gulf divide into 2 groups -

i. Routine. All UK military personnel are routinely administered tetanus:polio:yellow fever:typhoid. Medical workers also receive Hepatitis B. For operation Granby troops would receive boosters as required and

ii. in addition for Gulf. Cholera:plague:anthrax (with pertussis as adjuvant). This programme was drawn up following a full risk assessment. All vaccines except plague were licensed.

b. Smoke from oil-well fires

This is a contention made by many claimants. The Americans have extensively researched it and concluded that it, exceptionally, might temporarily aggravate pre-existing chest disease eg asthma, but by itself does not cause illness.

[to top of the page](#)

c. NAPS tablets

Pyridostigmine bromide was used as nerve agent prophylaxis (NAPS). This drug, an anticholinesterase, has been used since the 1950s. The dose used in the Gulf was much less than is standard in myasthenia gravis eg 90 mg/day compared with 500 mg daily. Side effects are nausea, diarrhoea and colic. They are reversible within hours of stopping the tablets.

d. Combination of NAPS with OP substances or vaccines

There is a hypothesis that toxic effects similar to that claimed by some Gulf participants particularly the vague central nervous system symptoms of irritability, concentration impairment, mood change, might occur due to combination effects of NAPS with OPs or the complex vaccines. This is currently being studied in animals. The difficulty is that again in these circumstances maximum effect would be expected at the time of exposure followed by improvement with time. This is not the pattern recorded in a single case to date. Rather we have interval - usually post-service - development of symptoms.

e. Exposure to depleted uranium

Quite a lot is known about the hazards of depleted uranium. At this date from the Gulf, toxic effects, mainly on the kidney, would dominate. Clinically this usually presents in some form within a short time of exposure. For the future the potential ionising radiation late effects need to be kept in mind. Secretary of State should enquire of the MOD re any claimed incident and apply clinical judgement to the medical and service factors of the individual case.

f. Infectious diseases

There have been few claims for infectious diseases in Gulf participants. The US experience is 31 cases (total) of Leishmaniasis with 12 of the previously unreported, unique to the Gulf, viscerotropic form. This has not been seen in UK forces but the MOD Gulf Assessment Programme has identified one case of giardiasis and several of amoebiasis.

[to top of the page](#)

g. Exposure to chemical and biological weapons

It was originally thought that no chemical or biological weapon was deployed in the war. In June 1996 the US government announced a potential exposure to sarin and cyclosarin at Al-Khamasiyah. Where such an contention is made the Secretary of State should make enquiries of the MOD.

Even at extremely low doses nerve agents produce acute effects (within minutes). The substances are OP molecules and the side effects are those anticipated where acetyl choline breakdown is prevented. There is evidence of possible longer time side-effects where there has been an acute effect. Where this has not been so, present evidence does not support development of medium or long term effects. The US Department of Defence is now investigating the possible long term sequelae of sub-toxic exposure levels.

h. Exposure to OP insecticides

OP insecticides were used. Chemically related to the nerve agents, their structure is modified to reduce mammalian toxicity and increase insect toxicity. Specific substances used were Malathion powder - used for dusting Iraqi POWs : fenitrothion: neocidal (supplied in error) and alfacron. These were used to spray tentage to reduce risk of insect-borne diseases particularly diarrhoeal disease.

Malathion and fenitrothion were approved at the time of the Gulf War. Alfacron was and is approved still and neocidal is a sheep dip not for human use and was used in error. MOD details of the specific exposures of particular individuals is restricted to Iraqi prison warders and environmental health personnel. Use of the compounds was overseen by environmental health professionals with specialist training.

OP insecticides are acetylcholinesterase inhibitors and acute effects of miosis:abdominal cramps:nausea:dry mouth:diarrhoea along with dizziness:anxiety tremor:confusion are well known. Certain OPs are associated with a delayed neuropathy (PIDN) developing 2-3 weeks after acute intoxicating exposure and consisting of a distal symmetrical sensori motor mixed neuropathy affecting the lower

limbs first and subsequently the upper limbs. It is not thought that this occurs due to acetylcholine inhibition. In addition, more recently a paralytic intermediate syndrome has been described. This has onset within 24-96 hours of toxic exposure and lasting about 3 weeks. Clinically it is like Landry-Guillain-Barré Syndrome.

More controversial are long-term effects. Some long-term effects following chronic (10-15 years) lower dose exposure on central and peripheral nervous system have been described in case studies. The studies are few and findings are not consistent.

Prior to the MOD Gulf announcement there was already concern about OP in use as sheep dip. In December 1993 MAFF announced new safety measures on sheep dips and the setting up of a panel of medical and toxicological experts to investigate the long-term side effects of organophosphates. The Committee considered - that there was at present no firm scientific evidence to support the hypothesis that the chronic health effects are associated with exposure to OP.

A study set up at the Institute of Occupational Medicine, Edinburgh looking at long-term side effects of organophosphates has now reported. The findings are complex but do not identify a specific syndrome of chronic low dose OP exposure.

What is the relevance of all this to the Gulf situation? The answer must be we do not yet know. A critical fact is the definition of "chronic" exposure. Nearly all published agricultural studies are 5-15 years not weeks or months as in the Gulf.

[to top of the page](#)

In his 1995 paper Jamal wrote - there have been few high quality epidemiological studies which have addressed the question of whether chronic effects can result from small levels of exposure over a long period of time and this question is therefore unresolved.

The present War Pensions medical position (confirmed by literature search and discussion with medical experts and toxicologists) is that the quality and quantity of evidence does not raise a reasonable doubt that, in the absence of acute toxicity, organophosphate insecticide exposure causes medium or long term side effects. This is the position we should reflect in certification where OP poisoning is claimed. The judgement will depend critically on the facts of the individual case, bearing in mind that war pension is awarded for disablement due to service not specific diagnoses and contemporary understanding of Gulf related illness.

Claims analysis

| a. Most frequent complaints | % |
|-----------------------------|-----|
| 1. Tiredness | 55% |
| 2. Muscular and joint pains | 40% |
| 3. Irritability | 30% |
| 4. Sleeping difficulty | 20% |
| 5. Memory loss | 20% |
| 6. Breathlessness | 15% |
| 7. Rashes | 12% |
| 8. Tingling in limbs | 15% |

| | |
|------------------------------------|-----|
| b. Diagnostic categories by system | % |
| Psychological | 33% |
| Infectious disease | - |
| Neoplasm | - |
| Endocrine | - |
| CNS/Peripheral | - |
| CVS | 2% |
| Respiratory | 10% |
| GIS | 10% |
| GUS | 8% |
| Skin | 10% |
| Musculo-skeletal | 10% |
| Other | 15% |

| | |
|------------------------|---|
| c. Total diagnoses | % |
| Minor physical illness | 40% |
| Psychiatric illness | 35% |
| | - - This divides into PTSD - more than half all psychiatric diagnoses, 20% of total diagnoses) and others (ie anxiety, depression, adjustment reaction, 15% of total diagnoses) |

Major physical illness 10%

Fatigue or unexplained 15%

References:

Jamal G A. Long term neurotoxic effects of organophosphate compounds. Adverse Drug React. Toxicol. Rev 1995 14(2)) 85-99 Oxford.

Chronic neurological effects of organophosphate pesticides. BMJ 312 1996 1312-1313.

August 1999

Diagnostic criteria:

[Fibromyalgia](#)

[Chronic Fatigue Syndrome](#)

[back to Medical Adjudication contents](#) -

[back to top of the page](#)

MEDICAL ADJUDICATION GUIDANCE NOTE ON DEPLETED URANIUM

Depleted Uranium

1. It has occasionally been contended by Gulf veterans at war pension claim and in the press that depleted uranium may be a causal factor in the illnesses they are now experiencing. Following recent reports of cases of leukaemia in NATO troops who have served in Kosovo where depleted uranium tipped weapons were used, there may be more war pension claims where depleted uranium is the proposed service link. It is hoped that this note will be helpful in medical decision-making.
2. Uranium is a natural, chemically toxic and radioactive element made up of 3 isotopes. Natural uranium is only slightly radioactive – 0.7% of the total comprises the radioactive isotope. When the isotope used in nuclear reactions is extracted, depleted uranium is the by-product. By definition this has a lower concentration of radioactive isotope than natural uranium.
3. Depleted uranium is nearly twice as dense as lead. This property is used to improve the performance of armour and missiles to penetrate armour. Uranium tipped missiles produce shrapnel and an aerosol dust when impacted on armour or if accidentally ignited.
4. Depleted uranium is chemically toxic and radioactive. Most of the radiation does not penetrate the skin and while external to the body, depleted uranium is considered to be a low hazard to health both from chemical and radiation effects. This view is held generally both in occupational health eg uranium mines or weapons manufacture and in relation to military use. HSE exposure limits are set for soluble depleted uranium and expressed in terms of chemical toxicity.
5. **Chemical effects** If ingested/inhaled or entering by the skin (normally via wounds) the effects depend on dose/type of exposure and solubility of the ingested particles. If insoluble, uranium is swallowed, the particles pass through the body and out via faeces. Soluble substances swallowed or inhaled as gas/fume/aerosol travel to target organs – the chief of which is the kidney via the blood stream.
6. **Radiation effects** Because depleted uranium is radioactive it is a potential carcinogen. The evidence suggests it has a low cancer risk relative to other radionuclides. The external radiation hazard of depleted uranium includes alpha, beta, gamma and X-rays. In practice alpha radiation is stopped by the skin and poses no hazard. There is no conclusive evidence from animal or human studies that natural uranium causes cancer. Studies of miners experiencing high life-time exposures show an increased death rate from lung cancer but careful examination of the evidence suggests this is due to concurrent exposures such as radon, tobacco, silica. Only one animal study – involving more than 5 years inhalation by dogs – resulted in tumours in animals.
7. The time course of carcinogenesis must be remembered. It is generally accepted medically that 2 years minimum – from carcinogen exposure to clinical onset – is required for development of leukaemia, other than chronic lymphatic leukaemia which is not caused by radiation exposure For solid tumours the time lag is about 10 years. The Gulf War took place in 1990/91 when a

total of 300 tonnes of depleted uranium were fired – mostly by the US. Only about 100 rounds of depleted uranium weapons were fired by UK forces.

8. Both US and UK research, including from the Institute of Medicine, Department of Defence and the Rand corporation, supports the view that the possible depleted uranium exposures of soldiers in the Gulf produced only minimal – if any – risk of harmful health effects and that effects of depleted uranium are unlikely to explain any of the Gulf-related disablements claimed to date. There is no evidence that UK personnel sustained shrapnel injury from depleted uranium ammunition. 33 US personnel with embedded depleted uranium shrapnel have been monitored at the Baltimore Veterans' Agency since 1993. To date there are no adverse health effects.

9. One hazard about which documentation is incomplete is the possible exposure of UK personnel sent to retrieve Iraqi tanks which had been damaged by US depleted uranium shells. (Retrieval of tanks was appropriate because scrutiny of Iraqi tanks provides access to information on Soviet style weapons). In practice depleted uranium weapons are very accurate and tanks hit by them are unlikely to be suitable for such scrutiny. Precautionary guidance about the safe way to approach recently struck vehicles possibly hit by depleted uranium was issued to UK troops in Kosovo.

10. To inform estimate of health risk due to depleted uranium, AWE/DRPS have performed some calculations.

a. Personnel would need to be in a tank fully loaded with depleted uranium for 600 hours before 20 m SV dose ie the current permissible annual dose for radiation workers is reached by external radiation. No UK soldier was in this situation.

b. Depleted uranium metal is covered by protective coating therefore ammunition handlers are not subject to internal ionising radiation hazard.

c. For soldiers handling depleted uranium ammunition in stores there is no significant external radiation hazard.

d. Radiation hazard can be produced by burning depleted uranium in air. If inhaled over a **long period** (not a single or low number of incidents) this could travel to the lungs.

11. MoD already has in place (March 1999) screening for Gulf veterans where their own doctor or Medical Assessment Programme physicians think depleted uranium may be responsible for illness. Depleted uranium has not been confirmed as responsible in any case. Kidney disease has been rare and where it has occurred an underlying pathology has been established. In addition about 40 Gulf veterans have had some urine samples analysed for depleted uranium in Canada and a number have been reported positive programme to re-test urine samples in UK has also been set up but up-take of the tests (which includes testing at a laboratory of the ex-serviceman's choice) has been very low. Following media speculation linking cases of leukaemia in Italian NATO soldiers to depleted uranium in Kosovo, MoD Ministers have announced the setting up of an additional screening programme. This is to be based on best available scientific evidence and details are awaited.

January 2001

BACKGROUND NOTE ON DEPLETED URANIUM

Current media and political interest in DU has followed reports from Italy and other NATO countries linking adverse health effects, particularly leukaemia, with use of DU tipped weapons in the Balkans. Although first used militarily as recently as the Gulf, DU has had industrial use for many years, for example in the keel plates of ocean going yachts and in aircraft and as part of the radiation shielding of therapeutic radiation sources. As a result it has been subject to extensive research on possible health effects and its use is governed by Health and Safety statute.

What is it?

DU is derived from natural uranium by removal of the most radioactive isotopes and is 40% less radioactive than natural uranium. It is a very dense heavy metal and its military use is as a kinetic penetrator to increase targeting accuracy of weapons. A total of 300 tonnes was used in the Gulf (almost exclusively by the US), and in the Balkans 3 tonnes were used in Bosnia in 1994-95 and 9 tonnes in Kosovo in 1999.

How are people exposed?

DU is a pyrophoric material and when it impacts on a surface a plume of particulate smoke is produced. These particles can then be ingested or inhaled or can enter the skin via wounds. This might occur if individuals are inside tanks hit by DU weapons or are on the ground in the vicinity of such attack. People can also theoretically be exposed when handling weapons in stores or while travelling in tanks or other vehicles containing tipped ammunition. In both these situations risk is very insignificant as the weapons are appropriately coated. It has also been suggested that there might be some slight risk to personnel dealing with bodies or prisoners who have themselves been contaminated. Finally people could be subject to shrapnel injury.

Effects on health

The effects on health depend on the dose/type of exposure and the solubility of the ingested material. Insoluble uranium normally passes through the body and out through faeces. It may occasionally lodge in the lungs and other parts of the body if the source is shrapnel. Soluble substances go via the blood stream to the chief target organ, which is the kidney. Damage to the kidney is typical of a heavy metal and takes the form of acute tubular necrosis. This result, which is considered the main hazard of DU, is rare and has never been detected in Gulf veterans anywhere in the world. In both the US and the UK some Gulf participants have suffered kidney complaints, but the underlying pathologies have been established and clearly shown to be unrelated to any possible exposure to DU. The solid particles are potential carcinogens. The evidence is that DU has a low cancer risk relative to other radionuclides. Its most important radiation hazard is alpha radiation which is stopped by the skin and so poses no hazard. There is no conclusive evidence that natural uranium causes cancer. No UK Gulf participant sustained shrapnel damage, but some US soldiers did. They have been monitored at the Baltimore Veterans' facility since 1993 and to date no adverse health effects have been detected. Although research has been carried out into reproductive effects there is no evidence to suggest that DU exposure has been associated with reproductive difficulties or children born with congenital abnormalities.

Research

There is a wealth of research in relation to industrial use and since the Gulf the US

Institute of Medicine, Department of Defence and the Rand Corporation have systematically reviewed the literature. Similar investigation has taken place in Canada, and an independent review by the Royal Society is awaited. Publication date is expected to be this spring. All this work has provided no evidence that DU itself, even in battle conditions, is likely to be a health hazard.

Screening

Since March 1999, uranium screening has been available for Gulf participants where a physician (either their own or one at the Medical Assessment Programme) considers that DU exposure might be linked to their health. No evidence of such a link has been found. About 40 Gulf vets have had their urine tested by Canadian scientists and some have been reported positive. The protocols and methodology used have not been published and there is concern about the reliability and validity of these tests. Despite invitations to discuss DU with MoD officials, Drs Sharma and Durakovic have always declined to do so. The MoD established a protocol for verifying the Canadian results. This included opportunity for veterans to choose at least one laboratory to carry out the tests. They have however declined to participate. Precautionary guidance about handling DU weapons and approaching vehicles which may have been hit by DU weapons is provided to UK troops.

Following the recent media interest, MoD ministers have announced the setting up of an additional screening programme. They have emphasised that this is to be based on best available scientific evidence, including advice from the UK National Screening Committee of the Department of Health and the report "currently under preparation by the Royal Society". Details are awaited.

The main issue of public disquiet is the potential of DU, however small, to produce cancer. It should be noted that the latest report of the United Nations' Scientific Committee on Environmental Causes of Cancer considers that less than 2% of all cancers are occupationally caused. In addition, the Italian soldiers with leukaemia became ill at the latest in early 2000 having served in Kosovo in 1999. The latent period for ionising radiation linked leukaemia is generally accepted as a minimum of two years.

Medical Adjudication Guidance has been available to all War Pensions Medical Advisers since 1998. For revised edition [click here](#)

Medical Adjudication Guidance On Hypertension

A new edition of this [appendix dated October 1996](#) is now available.

The appendix is quite straightforward. Whether the case falls to be considered under Article 4 or Article 5 essential and primary hypertension cannot be attributable to service.

Secondary hypertension, however, may fall to be certified. The relation between renal disease and blood pressure is complex and it will often be appropriate to discuss these cases with the ECW. Specialist opinion may be required.

Of the factors involved in essential hypertension discussed in the appendix, stress is the most likely contention.

The current position is that:-

1 Acute fear or physical stress causes a **transient** rise in blood pressure. This has not been shown to lead to sustained hypertension.

2 Authoritative opinion is also of the view that reliable evidence does not raise a reasonable doubt that stress is a cause of sustained hypertension.

The last edition of the appendix discussed the spiral hypothesis. No recent work on this has been published. Supporting evidence is lacking and well designed more recent studies are not supportive eg the 1995 Whitehall study.

Claims for hypertension in relation to UDR/RIR or other active service may pose special problems. In general, the guidance set out above applies.

Where there is **corroborative evidence** of more than one episode of acute, physical or mental stress, with onset of **sustained** high blood pressure in close time relation to the acute stresses, the specifics of the case should be discussed with ECW.

October 1996

Medical adjudication guidance on hypertension continued

[back to Medical Adjudication Contents](#)

Medical Policy Adjudication Guidance On Claims Where Ionising Radiation Claims Is An Issue

Claims where service related ionising radiation exposure is an issue arise in several situations. The claimant may have been in the vicinity of Hiroshima or Nagasaki in the aftermath of the detonations or he may claim a job specific exposure in nuclear submarines or as a medical technician. Information on this type of case to inform Secretary of State acceptance of ionising radiation exposure is obtained from the Defence Radiological Protection Service.

The majority of ionising radiation claims are from nuclear test veterans. 21,000 UK servicemen took part in the UK atmospheric nuclear tests between 1952 and 1958.

1. Any case where service related exposure to ionising radiation is confirmed

In any case (nuclear test or other) where the claim concerns a condition which is aetiologically linked to ionising radiation, and reliable evidence confirms in the opinion of Secretary of State, that there has been service exposure to excess ionising radiation, certification will follow. Medical opinion as to whether a condition is considered causally linked to ionising radiation will normally be found in the relevant appendix. If that is not so, the matter should be discussed with ECW/TO who will discuss with MPM as required.

2. Participation at UK atmospheric nuclear test sites

The NRPB study is important in nuclear test cases. Firstly it should be remembered that the study concerned "participation" at UK atmospheric nuclear test sites, rather than ionising radiation exposure.

The 1988 NRPB report's overall conclusion was that participation had no detectable effect on expectancy of life or the participants' total risk of developing cancer.

Generously interpreted the report did suggest a possible excess of leukaemia (other than CLL) and multiple myeloma. This led to the Departmental policy of certification of attributability for multiple myeloma and leukaemia (other than CLL) in anyone who participated at a UK atmospheric test site. This was later extended to include polycythemia rubra vera.

CLL (chronic lymphatic leukaemia) is not causally related to ionising radiation and hence is excluded.

The NRPB findings in 1988 were not conclusive and the study was extended to December 1990 to clarify the position. A follow-up report was published on 11 December 1993.

[back to Medical Adjudication Contents](#)

Genetic Damage

1. "Genetic damage" is most frequently seen as a war pension claim in relation to alleged ionising radiation exposure or exposure to chemical toxins. Similar claims have also been made in relation to Gulf service. Recently, the findings in the European NTVA case (Messrs McGinley and Egan) have been published. One of Mr McGinley's claims related to infertility and I felt it important that before providing advice to WPA that I had time to study the report. (The MPM's copy of the report may be borrowed on request.)

2. Claims for "genetic damage" usually concern infertility or arise when a child - or even grandchild - is born with a congenital defect. Awareness of the incidence of congenital disablement in the general UK community is low and perception of the effects of ionising radiation equally uninformed.

3. There is first a need in war pensions to show disablement. Where the issue is infertility, support for the presence of disablement is usually forthcoming. Where genetic damage - leading to congenital disablement in the next generation is the issue, the situation is more difficult. There is no current routine test of genetic damage and certainly not one which could reasonably be used for war pensions claims determination. Since DNA is in a constant state of mutation and subject to many influences including possible ageing, background toxins (not necessarily identified) and natural radiation etc another difficulty arises because even if genetic damage can be shown its aetiology cannot be specified.

4. In relation to Gulf claims it should be noted that recent US research (Cowan et al 1997) has confirmed no difference in incidence of congenitally disabled children born to Gulf veterans as opposed to persons who did not serve in the Gulf. No study has yet been published concerning UK participants but Dr Ann Doyle at the London School of Hygiene and Tropical Medicine - as part of the MRC - MoD sponsored research - is specifically looking at the incidence of these defects in the progeny of Gulf service personnel.

5. The adverse health effects associated with ionising radiation exposure are of two types - deterministic and stochastic.

6. Deterministic effects arise shortly after exposure to a radiation dose (hours to weeks) but they occur only if the dose exceeds a certain threshold value. For different organs of the body there are different thresholds. If the dose is beneath threshold no effect is seen. Thresholds for all tissues are comparatively high, several hundred times above natural background levels of radiation. If the threshold is breached the severity of resultant effects increases with dose. Deterministic effects include damage to bone marrow, gastrointestinal tract, CNS, lung and skin. Usually the effects are not fatal - include vomiting and diarrhoea and together constitute the radiation syndrome. If there is a very high dose death will result.

7. Stochastic effects arise years to many years (2 to 40+) after the ionising radiation exposure and here the probability depends on the level of the dose and the severity is not related to dose. There is no known threshold but the probability that the effect will occur decreases as the dose decreases. This means that even at the dose of natural radiation there is a finite possibility of a stochastic effect. At the same time some individuals exposed to very high levels will manifest no effect. The 2 main effects are cancer in the exposed individuals and hereditary disease in subsequent generations. Hereditary effects have not been shown to occur in humans although

they may do so in animals.

8. Infertility - The effects of radiation on sperm count are deterministic in nature ie are subject to a threshold and increase in severity with dose. Reduction may be permanent at high doses. Relatively low acute doses may cause a temporary reduction in the sperm count, which may last for 1 to 2 years. The ultimate degree and duration of depletion of the sperm depends on the magnitude of the dose. The range of doses reported and their effects are very variable. However, a whole body dose sufficient to cause permanent changes in sperm count (reduced or absent sperm over many years) would cause moderate to severe radiation sickness, unless the dose was delivered directly to the testes to avoid such effects (Mettler et al 1995). Local contamination with fallout would not provide a sufficient dose to produce this effect. There is no reasonable likelihood that a documented low sperm count is causally related to radiation exposures at UK atomic tests.

9. Studies of 88,000 children of the Hiroshima and Nagasaki survivors have not shown excess of congenital abnormality, mortality or cancer incidence in the first 2 decades of life. (Neill and Schull 1988).

10. In summary then - subject to the usual individual case consideration - ionising radiation claims for infertility will generally fail along the lines of paras 6-8 above and claims for "genetic damage" related to congenital disablement will also generally fail "not found".

References

Mettler Jr F A et al (1995). Medical Effects of Ionising Radiation (2nd Edition). Philadelphia (1995).

Neel JV and Schull WJ (eds). The Children of the Atomic Bomb Survivors: A Genetic Study. Washington DC, National Academy Press (1991).

October 1998

[back to Medical Adjudication contents](#)

Policy Statement on Claims for Ionising Radiation Related Conditions Summary

The Department's normal policy is that:-

1. In general an award of war pension will be considered in any case where there is reliable evidence of service related ionising radiation exposure **and** there is a recognised causal link between the claimed condition and such exposure.
2. The Secretary of State does not accept, as a matter of course, that those present at UK atmospheric nuclear test detonations and clean up operations were exposed to harmful levels of ionising radiation as a result of service in these locations in the armed forces.
3. **Service related ionising radiation exposure** means exposure to a measurable level of ionising radiation due to service in the armed forces and as determined by radiological dosimetry specialist report.
4. Awards can be made for leukaemia (other than chronic lymphatic leukaemia) and primary polycythaemia rubra vera if their clinical onset is within 25 years of first visiting the sites, based on **presence only** at the tests (ie exposure to service-related ionising radiation does not need to be shown).
5. Present overall evidence does not raise a reasonable doubt that ionising radiation causes atherosclerosis or related coronary heart diseases.
6. A Glossary setting out some key technical terms is found at the end of this policy statement.

The Purpose

7. This statement sets out the Department's policy on deciding claims for war pensions where service related ionising radiation exposure is alleged to have caused disablement or death. It also states the reasoning and evidence on which the policy is based. Situations where claims covered by the policy would be expected to arise include:
 - participation in the UK nuclear tests in the Pacific and Australia or as a result of the subsequent clean up operations at the test sites
 - Prisoners of War held in Nagasaki or Hiroshima during WWII and the subsequent clean up operations
 - accidents on board nuclear submarines or ships
 - handling nuclear weapons
 - employment as a radiographer or other medic eg where X-ray equipment is in use

About 27,000 UK servicemen participated in the UK nuclear tests and the largest number of claims relate to presence at the nuclear test sites. Because the effects of excess ionising radiation can take a long time to become apparent most claims are made under Article 5 of the Service Pensions Order (1983).

The Law - How the scheme works

8. A war pension may be claimed for any disablement by anyone who has served in the British Armed Forces. Claims may be made at any time from service release. Decisions are evidence based and each case must be determined on its own individual merits. Awards may be made where, within the relevant law, the evidence including service and medical facts and the contemporary medical understanding of the condition claimed, shows a causal link between service and the claimed condition, on the basis of the relevant burden and standard of proof as referred to in paras [9] to [10] below.
9. For claims made not later than 7 years after leaving the armed forces, Article 4 of the Service Pensions Order provides that the onus is on the Secretary of State to show beyond a reasonable doubt that the claimed disablement is not attributable to, or aggravated by, service, or that death was not due to, or hastened by, any such condition. If he cannot show this an award of War Disablement Pension or War Widow's Pension, as appropriate, may be made.
10. For claims made more than 7 years after the end of service, Article 5 of the Service Pensions Order puts the onus on the claimant to raise, by way of reliable evidence, a reasonable doubt that the claimed condition is attributable to, or aggravated by, a service injury or that death was due to or substantially hastened by an attributable injury or the aggravation by service of an injury. If he does so, an award of war pension may be made.

Case Law -

11. The High Court has held that the word "reliable", in the context of Article 5, cannot have been intended to mean "convincing", but means more than "fanciful". A High Court Judge held that, with particular reference to "changes of medical opinion" that "there are... in my judgement, three stages: no reasonable doubt, reasonable doubt, and consensus". A war pensions claim under Article 5 would pass the test at the point where the (reliable) evidence raised a reasonable doubt, but: "a mere hypothesis based on a limited study... would not have created a "reasonable doubt" within the terms of Article 5(4)."The real question, however, it held, is whether the evidence raises a reasonable doubt in the mind of the Secretary of State (SoS). If he finds the evidence unreliable it obviously will not raise a reasonable doubt in his mind. (case of Edwards 1992 HCJ no. CO/2281/90).
12. The Courts have also held that a conflict of medical opinion does not, of itself, mean that a reasonable doubt has been established, and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of Tigg v The Minister of Pensions the presiding Judge stated " Merely because a doctor of eminence, and I have no doubt the doctor in this case was of very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not

mean there is a doubt and the Appellant must therefore be entitled to a pension. It is a question of fact for the Tribunal" (cases of Tigg ROSWPA vol.5 p.141 and Howard ROSWPA vol.5 p.515) .

13. In particular in assessing evidence regard has been paid to:
 - whether conclusions of studies are hypotheses; or whether there is supporting evidence, which goes beyond hypothesis, and if so its basis
 - the reaction of other experts in the field to the evidence
 - the weight of overall evidence on the matter.
14. Evidence for any new approach in science must always be considered and weighed relative to the existing body of evidence on a subject, with account taken of the robustness and authority of new studies. In particular the design and methods, sample size, case and control selection, statistical validity, repeatability of findings, approach to bias and possible alternative factors and hidden influences. Other important factors include whether the findings have been replicated by other independent researchers and the overall plausibility/consistency relative to contemporary understanding.

Ionising radiation

15. Exposure to radiation in all its forms is part of being alive. Ionising radiation is taken to mean radiation of high enough energy to displace electrons from atoms and includes cosmic rays, gamma rays, X-rays, alpha and beta radiation. The average level of exposure to natural background radiation varies throughout the world dependent mainly on the geology of the underlying earth. In the UK it is about 2.2 mSv (millisieverts) per annum average. There is, however, a range and it is much higher in areas of igneous rock such as Scotland.
16. Tissues vary in their sensitivity to ionising radiation and different types of ionising radiation have different capacity to cause tissue damage and hence adverse health effects. As no dose of ionising radiation is considered safe, natural background is generally considered the cause of a proportion of the cancers which occur in a population. In the UK about a third of the population is affected by, and about a quarter will die of, malignant disease. However there is no clear correlation of background level and cancer incidence and areas with high total backgrounds (such as Kerala and the Andes) demonstrate no excess malignancy. By contrast the UK with one of the lower average backgrounds has one of the highest cancer incidences in the world. A synopsis on **ionising radiation dose, radiological protection and the health effects of ionising radiation is at Annex A.**

UK atmospheric nuclear tests

17. Between 1952 and 1958 the UK carried out 21 atmospheric nuclear tests (12 in Australia, 9 at Christmas Island), in the South Pacific. The radiological safety standards at the UK atmospheric nuclear trials in the 1950s were based on the

then consensus of international scientific opinion as formulated by the International Commission on Radiological Protection. A fundamental principle was to keep any exposure as low as possible. Many of the detonations involved high air bursts falling freely. The risk of significant contamination of land occupied by service or civilian participants from these air bursts was avoided by careful selection of weather conditions and environmental monitoring following the tests. The natural background radiation at Christmas Island is very much less than that of average UK locations. Overall it is considered that almost all the British servicemen involved in the UK nuclear tests received little or no additional radiation exposure as a result of participation. Further details of the tests will be found at **Annex B**.

The National Radiological Protection Board (NRPB) nuclear test follow up studies

18. As a result of concern amongst some test participants about the effects that participation could have had on health, in 1983 the Ministry of Defence commissioned an independent study by the NRPB to investigate whether the health of participants showed any correlation with radiation exposure.
19. This comprehensive cohort study compared the mortality and cancer incidence in over 20,000 test participants with that of a similar-sized control group of ex-servicemen who had not participated in the test programme.

The term ‘test participant’ has a particular definition and includes servicemen present at the due dates, at any of the following test sites and experimental programmes

| Operation | Site | Date |
|-----------------------------------|----------------------------|---------------------------|
| Hurricane Mosaic | Monte Bello W Australia | April 1952-June 1956 |
| Totem | Emu Field S Australia | August 1953-August 1957 |
| Buffalo Antler Minor trials | Maralinga S Australia | April 1955-August 1967 |
| Grapple X Y Z Brigadoon | Christmas Island S Pacific | June 1956-June 1964 |
| Grapple | Malden Island S Pacific | October 1956-June 1964 |
| | RAAF Pearce W Australia | May 1956-August 1956 |
| | RAAF Edinburgh S Australia | August 1956-November 1960 |

There is no requirement for presence at actual detonations.

At the RAAF sites the work included cloud sampling and handling contaminated aircraft. RN ships were associated with tests at Monte Bello, Malden and Christmas Island. The Minor Trials, did not involve nuclear detonations. They took place at

Maralinga (Tims, Rats and Vixen A and B) while Kittens was at Emu field. Major clean up operations took place at Christmas Island in 1964 and Maralinga in 1964 and 1967.

20. The main conclusions of the first NRPB Report (Darby et al 1988) were that presence at the nuclear weapons test sites had increased the risk of multiple myeloma and leukaemia (other than chronic lymphatic leukaemia) compared with service controls. This was not considered to be due to ionising radiation exposure. There was a particularly low rate of the conditions in the controls and those sub groups considered most highly radiation exposed did not show the highest rates of the conditions.
21. Otherwise presence at the sites
 - did not have a detectable effect on the participants' expectation of life,
 - did not have a detectable effect on participants' risk of developing any other malignancy.
22. The study was extended and the second NRPB Report (Darby et al 1993) produced an additional 7 years data
 - confirmed the overall conclusion of the 1988 Report; that participation in the tests had no detectable effect on the participants' expectation of life nor on their risk of developing most cancers.
 - concluded that the small hazard of multiple myeloma suggested by the 1988 Report was not supported by the additional data although the possibility of some small risk of developing leukaemia (other than chronic lymphatic leukaemia) in the first 25 years after participation could not be ruled out.

With regard to other cancers the report concluded that:

- overall the number of deaths and cancer incidence amongst participants is lower than amongst the control group
 - as expected because a large number of diseases were considered, any excesses in participants are due to chance.
23. Following pressure for a further investigation into the alleged effects of exposure a 3rd NRPB study was commissioned. The report of the study which extended the follow up period to 1998 was published in February 2003 (Muirhead et al 2003). Key findings were :-
 - reaffirmed the overall findings of the 1988 & 1993 reports that participation in the Tests had no detectable effect on the participants expectation of life nor on their risk of developing most cancers.

- confirmed the conclusion of the 1993 report on the alleged association between participation in the UK test programme and multiple myeloma that there is no evidence to support a link.
- suggested particularly in 2 – 25 years after first test participation a small increase in risk of leukaemia (excluding chronic lymphatic leukaemia) among test participants relative to controls although the difference in rates between the 2 groups is narrowing with longer follow up.

24. Applying the test set out at para 13 and 14 of this statement, the Secretary of State considers the National Radiological Protection Board Reports, of which a principal author is Sir Richard Doll, to be reliable evidence.

In particular the following points are noted:-

- The study identified the test participants, and followed them up to monitor the occurrence of disease and death in the participant population. It then compared this, over the same time period with the rates in both a service and civilian control population.
- The study involved 20,000 subjects and an equal number of controls
- The reports describe in detail the efforts made to ensure sample completeness and to control bias.
- The study limitations are discussed by the authors and conclusions are reasoned and restrained.

The Secretary of State's opinion as to the reliability of the evidence in the reports is in accord with the general opinion of the scientific community. Positive reactions include comment from Prof John Kaldor of New South Wales (Kaldor 1999) and the US Presidential Advisory Committee on Human Radiation Experiments (Thomas 1998).

Impact of the NRPB reports on the Secretary of State's normal policy

25. Based on the first report, Secretary of State's normal policy became to award war pension for claims for leukaemia (other than chronic lymphatic leukaemia) and multiple myeloma in those present at test sites. The policy also included awards for primary polycythaemia rubra vera, the red blood cell equivalent of leukaemia. In light of the 1993 report, Secretary of State's normal policy was revised. Since then, on the basis of presence at atmospheric nuclear test sites new claims for multiple myeloma are rejected but awards continue to be made for leukaemia (other than chronic lymphatic leukaemia) and primary polycythaemia rubra vera having clinical onset within 25 years of first presence at the test sites. On the basis of the findings of the 2003 report the Secretary of State's current normal policy remains unchanged

26. **The reports did not causally link development of those conditions to ionising radiation exposure and the policy is not an acknowledgement that those**

present at the tests were exposed to harmful levels of ionising radiation. The accepted service link is purely presence at the test sites.

27. Having carefully considered the reports the Secretary of State is of the opinion that they do not provide reliable evidence to raise a reasonable doubt that other cancers (eg liver and bladder) might be attributable to service in the armed forces because of presence at the nuclear test sites. Consequently it is his normal policy that awards may not be made for solid cancers on the basis of presence at atmospheric nuclear test detonations or clean-up operations alone. However it is also his normal policy that an award of war pension may be made for cancer in any case where there is reliable evidence of service exposure to sufficient level of ionising radiation and there is a recognised causal link between the claimed condition and such accepted exposure.

Children of test participants

28. The sample on which the 1988, 1993 and 2003 NRPB Reports was based did not include the children of test participants and was solely concerned with a study of the test participants themselves and not with any possible affect their participation might have had on their progeny.

29. Any claim for compensation for a child in respect of disablement or death said to be due to the parent's participation in the UK Tests would not fall within the scope of the SPO.

Position of civilian test participants eg MOD civilian employees

30. Compensation for civilians or their widows employed by the MOD who participated in the tests and who claim that disablement or death is due to participation is similarly not covered by war pensions legislation. In addition to civil action against MOD there is a possibility of a successful claim to Industrial Injuries Benefit under the Industrial Injuries Scheme administered by the DWP. MoD civilian employees are also covered by the Compensation Scheme for Radiation Linked Disease.

Cases falling outside the general policy guidelines

31. Any reference to the Secretary of State's "normal policy" indicates that the policy should not be a rigid one. The merits of each individual case should be considered and discretion should be used when deciding whether to make a payment.

Evidence of cancer causation by ionising radiation

32. Evidence that ionising radiation can cause human cancer has come from several sources. These include follow-up of patients therapeutically irradiated for malignant conditions, such as cancer of the cervix, and non-malignant conditions like ankylosing spondylitis, follow-up studies on UK, US, Australian and New Zealand service personnel present at atmospheric nuclear test sites, and most notably from the Japanese atomic bomb survivor studies.
33. The Japanese atomic bomb survivor data shows evidence of an increase in cancer incidence but only in individuals exposed to levels of ionising radiation of 50 mSv and above. (The UK natural background radiation is 2.2 milliSieverts per annum average.) There is no firm evidence from any human low dose epidemiological

studies which unequivocally demonstrates an increase in cancer incidence.

34. Since everyone is exposed to ionising radiation and not everyone develops cancer other factors must be relevant. Cancers induced by ionising radiation are indistinguishable from those due to other more common causes such as diet, tobacco, alcohol etc. In addition to the dose of radiation delivered, the type of radiation, its duration of exposure ie an acute high dose or a chronic low dose, the particular tissue irradiated and the age of the individual at the time of the radiation are all known to be important. Taking the overall evidence on these matters into account and in the absence of a positive threshold dose of ionising radiation, the convention is to accept that no dose of ionising radiation is completely free from risk of cancer and that the risk increases linearly with dose.
35. There is, however, a spectrum of risk dependent on the factors discussed above and a standard international approach to estimation of the probability that a particular cancer in a particular patient is causally linked to ionising radiation has been established. (IAEA - Tech - Doc 870 (1996)). The probability of causation approach requires dosimetry information on the individual's exposure. The Secretary of State's normal policy in war pensions is that there is reliable evidence to raise a reasonable doubt that there is a causal link between ionising radiation exposure and the following cancers:-

leukaemia (other than chronic lymphatic leukaemia)
multiple myeloma
non-Hodgkin's lymphomas
polycythaemia rubra vera
female breast
lung
oesophagus
stomach
colon
primary cancer of the liver
gall bladder
thyroid
urinary bladder
non-melanoma skin
brain
salivary gland
bone
ovary
uterus and vagina
testis
kidney
pancreas (Pierce (1996): Thompson (1994))

In war pension claims for disablement or death due to these conditions **and** where the Secretary of State has accepted **service related ionising radiation exposure**, a war pension award will be considered. The Secretary of State does not accept

evidence of participation in nuclear tests as itself equating to proof of service related ionising radiation exposure.

Evidence of radiation induction of non-cancer conditions

36. Reports of the atomic bomb survivor follow-up studies, suggests that ionising radiation exposure may also be associated with non-cancer diseases. (Kodama (1996): Schull et al (1998)). Associations have been described with uterine fibroids and certain non cancerous thyroid and para-thyroid tumours. The issue of a possible link between ionising radiation exposure and cardiovascular disease has also been raised in relation to war pensions. A review of the current evidence is at Annex C.
37. On present evidence the Department does not accept that a reasonable doubt is raised by reliable evidence that ionising radiation exposure is causally related to atherosclerosis or any of its manifestations.

References

Darby et al (1988) Report on Mortality and Cancer Incidence in UK Participants in UK Atmospheric Nuclear Weapon Tests and Experimental Programmes NRPB-R214

Darby et al (1993) Report on Mortality and Cancer Incidence 1952-1990 in UK Participants in the UK Atmospheric Nuclear Weapon Tests and Experimental Programmes NRPB-R266

Kaldor (1999) Report to the Minister assisting the Minister for Defence on recent studies of nuclear test veterans. University of New South Wales, Australia.

Thomas (1998) Letter to the Editor J Radiol Prot; Vol 18; No 3: 209-210

Pierce (1996) Studies of the mortality of atomic bomb survivors. Report 12, Part 1. Cancer: 1950-1990. Rad. Res. 146, 1-27.

Thompson (1994) Cancer incidence in atomic bomb survivors Part II: Solid tumours, 1958-1987. 1994. Rad. Res. 137: S17-67.

IAEA – Tech – Doc 870 (1996) Methods for estimating the probability of cancer from occupational radiation exposure.

June 2003

Annex A

Radiation dose

1. The first definition of a unit of radiation dose was made in 1928 by the International Congress of Radiology. The rontgen (R) was defined as that quantity of radiation which produces in 1cm of air one unit of charge of either sign, thus defining a unit of exposure. Units of **absorbed dose**, the actual energy absorbed in the tissue being irradiated are now used. The radiation absorbed dose or **rad** is now cited in SI (System Internationale) units - joules per kg - of absorbing material. The fundamental unit - 1 joule/kg is 1 gray (1Gy) equivalent to 100 rads (R).
2. Different radiation types have greater or lesser effect per unit dose so they are all expressed relative to the effects of X-rays ie a unit equivalent dose is used. To calculate the rontgen equivalent in man (**rem**) - the absorbed radiation dose is multiplied by a radiation weighting factor - dependent on type and energy of the radiation. The current SI unit of equivalent dose is the **sievert**. For X-rays and gamma rays the equivalent dose in sieverts and the absorbed radiation dose in grays are the same. The relationship between the different dose units is:-

1 gray (Gy) = 1 joule/kg = 100 rads (R) = 100 rems (r) = 1 sievert (Sv) = 1,000 millisieverts (mSv) = 1,000,000 microsieverts (microSv)). Typical doses of radiation include:-

Chest X-ray - 0.02 mSv

Brain scan - 7 mSv

Bone scan - 4 mSv

Average annual UK dose from cosmic rays - 0.26 mSv

Average annual UK dose from gamma rays - 0.35 mSv

Average annual UK dose natural background radiation – 2.2 mSv

Radiological protection

3. Since the days of Marie Curie it has been appreciated that ionising radiation exposure may be hazardous to health. Radiation dose limits were first recommended for ionising radiation exposure in 1928. The statutory limit on the amount of radiation to which the general public may be exposed in excess of natural background radiation and excluding medical exposure is set, from 1 January 2000 at 1 mSv per annum.
4. The most important source of man made exposure is medical investigation which accounts for 90 per cent of man made exposure. Average natural background radiation is raised to 2.6 mSv by all man made exposure. UK estimated experience excluding medical investigation is 0.04 mSv. Other statutory limits include occupational dose limits. From 1 January 2000 these are 20 mSv per annum for classified workers and 6 mSv per annum for unclassified workers.

Health effects of ionising radiation

5. Adverse health effects of ionising radiation are independent of the source of radiation and are of 2 types. Early and late.

Early effects (also called deterministic)

- These effects usually arise shortly after exposure, usually within hours or weeks.
- There is a threshold dose, beneath which no effects are seen.
- This threshold is relatively high, exceeding natural background radiation levels at all parts of the planet by several hundred fold.
- The severity of the effect varies directly with dose.
- Duration of exposure is also important and for a given total dose, acute exposure is more harmful than a protracted dose.
- The tissues affected are those whose cells have a high turnover rate ie bone marrow - skin - gastro-intestinal tract.

Late effects - also called stochastic/probabilistic

- These effects arise years (2-40 or more) after exposure and the probability depends on the level of the dose.
- There appears to be no threshold and the severity of the effects is not dose dependent.
- This means that there is a finite risk even from low level natural background radiation. At the same time persons exposed to high dose may suffer no ill effects.
- The 2 main late effects are induction of cancer and hereditary disease in subsequent generations.
- All diseases which can be radiation induced can also occur naturally or in relation to other exposures - cigarette smoke, alcohol, diet (both excesses and deficiencies), occupational exposures - and are not distinguishable on the basis of cause.
- Current best evidence is that radiation of all types gives rise to less than 2% of all cancers worldwide. The most important carcinogenic type of radiation is in fact ultra-violet light (UVB) not ionising radiation.
- Not all types of cancer have been shown by evidence to be caused by ionising radiation.
- Hereditary effects have not been demonstrated in humans but there is such evidence in some types of animals.

Effects of total body irradiation

| Equivalent dose (Sv) | Effect |
|--------------------------------------|---|
| Sublethal to man 0.0001 (0.1 mSv) | Around 2 weeks' natural background radiation, no detectable effect |
| 0.001 (1 mSv) | Around 6 months' natural background radiation, no detectable effect |

| | |
|---------------|--|
| 0.01 (10 mSv) | No detectable effect |
| 0.1 (100 mSv) | Minimal decrease in peripheral lymphocyte count, no clinical effect |
| 1 (1000 mSv) | Mild acute radiation sickness in some individuals (nausea, possible vomiting), no acute deaths, early decrease in peripheral lymphocyte count, decrease in all WBC and platelets at 2-3 weeks, increase in late risk of leukaemia, solid tumours |

| Equivalent dose (Sv) | Effect |
|----------------------------------|--|
| Lethal to man 10 (10,000 mSv) | Severe acute radiation sickness, severe vomiting, diarrhoea, death within 30 days of all exposed individuals. Severe depression of blood cell and platelet production, damage to gastrointestinal mucosa |
| 100 (100,000 mSv) | Immediate severe vomiting, disorientation, coma, death within hours |
| 1000 (1,000,000 mSv) | Death of some micro-organisms, some insects within hours |
| 10,000 (10,000,000 mSv) | Death of most bacteria, some viruses |
| 100,000 (100,000,000 mSv) | Death of all living organisms, denaturation of proteins |

Annex B

UK atmospheric nuclear tests

1. From information available the Ministry of Defence estimated that only about 10% of all participants were likely to have been exposed to measurable levels of ionising radiation. The relevant groups of personnel were in order of likelihood of exposure:-
 - RAF aircrews involved in sampling from airburst clouds (205 men). Mosaic. Totem. Buffalo. Antler. Grapple.
 - RAF decontamination flight crews who sluiced the aircraft (129 men).
 - RN personnel on HMS Diana when she sailed through the fallout at Operation Mosaic. (282 men)
 - The officers of the Buffalo Indoctrinee Force and Target response group. They assembled to observe at first hand the effects of the detonation (249 men).
 - Others – with recorded exposures greater than zero (1123 men).
The total of the five groups equals almost 2,000 men.
2. A nuclear explosion can produce effects on health via visible light, heat, air blast and ionising radiation.
3. The visible light from a nuclear detonation can cause flash-blindness at considerable distances and permanent eye injury at short ranges. (There is no absolutely safe range for this effect). Protection at UK trials was assured by mustering all persons and ordering them to face away from the detonation with eyes covered.
4. The heat from a nuclear detonation can cause first-degree burns to bare human skin at ranges up to three kilometres from a ten-kiloton detonation or twenty-one kilometres from a one-megaton detonation. Protection is by maintaining a sufficient distance.
5. Air blast is unlikely to cause injury to a person more than three kilometres from a ten-kiloton burst or six kilometres from a one-megaton burst. Protection is again by maintaining a sufficient distance.
6. The ionising radiation exposure associated with nuclear detonations is of two types. Firstly, a large pulse of radiation is emitted by the device as it explodes. This is absorbed by the air over distances of a few kilometres. To be close enough to receive a significant dose of ionising radiation an individual would also be within the lethal range of the air-blast and heat. Secondly, ionising radiation, under certain circumstances, is also emitted by the radioactive particulate fallout. The fallout particulates are carried downwind after the detonation. Wherever possible, UK trial detonations were carried out as high airbursts to minimise fallout. All UK atmospheric nuclear trials devices produced yields at, or very close to, the design figure.
7. Specialist instrumentation was used at the trials to measure ionising radiation. Personal

dosemeters, designed to estimate the dose to an individual from photons and beta particles, were carried typically for a month. The film badge consists of a piece of photographic film, sealed in a light-tight package bearing a unique number, and contained in a cassette adapted for securing to the clothing. Exposure to ionising radiation causes blackening of the film. After conventional photographic developing, the film is compared with a standard and an estimate of dose obtained. It is sensitive to photons (gamma rays and X-rays) beta particles and low-energy neutrons, and can distinguish between them.

8. The Atomic Weapons Establishment, Aldermaston holds the film badge records of the test participants. Badges were not issued to all personnel – Ministry of Defence estimate that 21% of total participants had badges. In general more badges were issued for the earlier tests (96% of those present at Operation Hurricane had a badge while only 20% of those at Operation Grapple). The reducing percentage of people monitored was informed by the actual exposure levels and characteristics of preceding operations. In general those men most likely by the nature and location of their duties to be exposed to measurable doses were monitored. Not all of those monitored showed a recordable dose. Less than 500 individuals received 5mSv or more and about 80 of these received 50 mSv. Doses recorded refer to the entire test programme for the individual and in some cases this will be several years. Of the 80, the majority were RAF crew who took part in cloud sampling.
9. Dose from ionising radiation can also arise by internal contamination, through breathing or swallowing contaminated dust. Although alpha-emitting materials would be the most hazardous in this respect, they are a very small component of fallout compared to beta and photon-emitting materials. The risk of internal dose was minimised at the trials by ensuring that only essential and fully protected personnel were ever in areas where internal contamination was possible. In addition, while a film badge did not measure internal dose directly, to receive a significant internal dose, an individual would have to enter an area where there were high levels of resuspendable fallout emitting photon and beta radiation. It is highly unlikely that this could happen without at the same time there being a measurable external dose received as indicated by his film badge.
10. The Ministry of Defence records identified those men present at the minor Maralinga trials who were at highest risk of radionuclide ingestion or inhalation. There were 847 in total. In the NRPB study, this group was considered separately. It did not show any increased risk of multiple myeloma, leukaemia or other malignancies relative to the rest of the participant group.
11. In conclusion, almost all the British servicemen involved in the UK nuclear tests received little or no additional radiation as a result of participation. If personnel who served at Christmas Island at that time had been stationed in the UK in an average location their dose of naturally occurring ionising radiation would have been 3 times greater than it was at Christmas Island.

Ionising radiation and heart disease

1. Until the 1960s the heart and blood vessels were thought to be completely resistant to ionising radiation (Warren 1942). Since then reports have appeared describing inflammation of the heart lining, and conduction disorders from damage to the electrical system following high dose mediastinal irradiation of malignant tumours. Today these effects of high dose ionising radiation exposure are generally accepted. (Stewart et al 1984).
2. The situation regarding a link between ionising radiation - particularly low dose ionising radiation - and atherosclerotic disease is less clear. An association between ionising radiation and atherosclerosis has not been established. (Corn et al 1990).
3. In animal studies, coronary sclerosis has been found in rabbits, rats and pigeons which have been irradiated **and** which have high serum cholesterol. The evidence is that both elements are required (Gold 1961).
4. In 1958 a human case study reported a myocardial infarction following deep X-ray therapy and since then there have been further reports linking death due to coronary disease following radiotherapy for medical conditions including Hodgkin's disease, breast cancer, seminoma. (Prentice 1965: Stewart et al 1967: Tracy et al 1974: McReynolds et al 1976). In these studies confounders were present eg they did not control serum cholesterol, blood pressure or cigarette smoking. In addition the study subjects were already ill. The reports do not prove a causal relation between ionising radiation and ischaemic heart disease.
5. Further information from long term follow-up studies of heavily irradiated populations (Host and Loeb 1986) has shown excess mortality from myocardial infarction in these populations. There are also case reports of cerebral infarction following radiotherapy to head and neck and of peripheral vascular disease of the lower limbs following pelvic irradiation. However, these effects have only been reported where the irradiation procedure delivered a very large dose of ionising radiation (20,000-60,000 mSv) and the results cannot be extrapolated to populations exposed to low doses of irradiation. Studies involving up to 20 years follow-up of patients irradiated according to more recent radiotherapy procedures ie using much lower doses have shown no significant difference in myocardial infarction death rate between irradiated and control populations. (Hancock et al 1988). A detailed discussion of these studies was presented in a review paper by Kodama (1995).
6. An American 50 year follow-up study of 30,000 radiologists suggested that in those who started practice between 1920 and 1929 there were an excess of coronary deaths compared with other medical specialists. However, a British 60 year follow-up study of 25,000 radiologists did not confirm this effect and similarly follow-up studies of 14,500 patients treated with deep X-ray therapy for ankylosing spondylitis over 30-50 years suggested no increase in coronary deaths. Results are therefore inconsistent.

7. Most follow-up studies have focussed on mortality rates, which is subject to many uncertainties and inaccuracies. A more accurate estimate of the association would come from incidence studies in large populations with lengthy follow-up and controlled risk factors.
8. The issue of association between ionising radiation and stroke or coronary heart disease in non-medical settings has been addressed periodically in the atomic bomb studies. In particular in the Radiation Exposure Research Foundation Life Span Study (RERF LSS) Technical reports. Until the report, LSS6, summarising the results for period 1950-70 there was no suggestion of a relation between atomic bomb radiation exposure and mortality from stroke or coronary disease. That analysis reported an increased mortality from coronary disease in women exposed to 100 mSv or more. The increase was particularly marked where dose exceeded 500 mSv. The trend was not however confirmed in the subsequent report, LSS9 for the period 1950 - 1978, although this did show increased mortality from "all diseases other than cancer" where exposure exceeded 2000 mSv.
9. The report on the period 1950-85 (Shimuzu et al 1992) used a new method of exposure dose estimate and showed clearly increased mortality from circulatory disease - including stroke and cardiac disease again in heavily exposed survivors.
10. The issue of accuracy of death certificates for the RERF studies has been examined (Carter et al 1991) and it is apparent that death certification for cardiovascular disease is less accurate than for malignancies. In addition it is the case in these mortality studies that other known cardiac risk factors cannot be controlled.
11. Only a few studies have yet been published which look at the **incidence** of coronary heart disease and stroke in relation to ionising radiation exposure associated with the atomic bombs. For the period 1958-1964, Johnson et al (1966) found no association. A later report covering the period 1958 - 1974 suggested an increase of stroke and coronary disease in females heavily exposed (over 2000 mSv) in Hiroshima. The effect was not seen in men or in Nagasaki survivors.
12. Kodama's 1994 study, now covering the period up to 1990, again confirmed an increase in myocardial infarction incidence in heavily exposed survivors regardless of age, gender or location, although the excess of myocardial infarction was very small compared with cancers. (The relative risk of myocardial infarction at 1,000 mSv exposure was 1.17. The associated p value is 0.02 with a confidence interval (95%) of 1.01 - 1.36). Lifestyle risk factors for coronary disease were not adjusted for. At this date, therefore, further studies are needed to determine the matter.
13. The Department's position in the context of exposure to low dose ionising radiation exposure is that present overall evidence does not raise a reasonable doubt that such exposure is causally associated with atherosclerosis or any of its manifestations.

References:

Warren S. Effects of radiation on the cardiovascular system. Archives of pathology. 34:

1070-1079 (1942).

Stewart J R & Fajardo, L F. Radiation induced heart disease: An update. *Progress in cardiovascular diseases.* 28: 173-194 (1984).

Corn B W et al. Irradiation-related heart disease. *Journal of clinical oncology.* 8: 741-750 (1990).

Gold H. Production of arteriosclerosis in the rat. Effect of X-ray and high fat diet. *Archives of pathology.* 71: 268-272 (1961).

Pearson H. Incidental changes of X-ray therapy. *Lancet, I:* 222 (1958).

Prentice R T W. Myocardial infarction following radiation. *Lancet, 2:* 388 (1965).

Stewart J R et al. Experimental radiation-induced heart disease in rabbits. *Radiology,* 91: 814-817 (1968).

Tracy G P et al. Radiation-induced coronary artery disease. *Journal of the American Medical Association.* 228: 1660-1662 (1974).

McReynolds R A et al. Coronary heart disease after mediastinal irradiation for Hodgkin's disease. *American journal of medicine.* 60: 39-15 (1976).

Host H & Loeb M. Post-operative radiotherapy in breast cancer: Long-term results from the Oslo Study. *International journal of radiation onconogy, biology, physics,* 12: 727-732 (1986).

Hancock S L et al. Intercurrent death after Hodgkin's disease therapy in radiotherapy and Adjuvant MOPP Trials. *Annals of internal medicine,* 109: 183-189 (1988).

Kodama K. Circulatory diseases. In Shigematsu. I et al. (eds). *Effects of A-bomb radiation on the human body.* Tokyo, Japan. Harwood Academic Publishers, 1995. Pp 182-194.

Shimizu Y et al. Studies of the mortality of A-bomb survivors, 9. Mortality, 1950-85: Part 3. Noncancer mortality based on the revised doses (DS86). *Radiation research,* 130: 249-266 (1992)

Carter R L et al. Combining diagnostic categories to improve agreement between death certificate and autopsy classification of cause of death for atomic bomb survivors, 1950-87. RERF technical report. 15-91, Radiation Effects Research Foundation. Hiroshima (1991).

Johnson K G et al. Cerebral vascular disease in Hiroshima. Report of a six year period of surveillance. 1958-64. ABCC technical report. 23-66. Atomic Bomb Casualty Commission, Hiroshima (1966).

Kodama K. Cardiovascular disease in atomic bomb survivors. RERF update, 5(4): 3-4, Radiation Effects Research Foundation, Hiroshima (1994).

Glossary

Absorbed dose See **dose**.

Acute radiation syndrome (ARS) The onset, within hours of high **dose** whole body **irradiation**, of nausea and vomiting followed by destruction and diminished (or absent) replacement of essential blood cells resulting in vulnerability to serious infection and bleeding; recovery is possible but with increasing **doses** these effects are more severe and death more likely.

Alpha particle A particle consisting of two protons plus two neutrons. Emitted by a radionuclide.

Background radiation **Ionising radiation** from naturally occurring **radionuclides** both in the environment (from soil, rock and building materials and from space – cosmic radiation) and in the body.

Beta particle An electron emitted by the nucleus of a radionuclide. The electric charge may be positive, in which case the beta particle is called a positron.

Contamination The suspension in air or deposition of **radionuclides** upon, or in, the ground, water and other surfaces, and personnel and equipment.

- **External contamination.** Of a person - deposition, general or localised, of **radionuclides** upon all or any of clothing, hair, skin and, or equipment.
- **Internal contamination** Of a person - deposition within the body, usually by inspiration, by ingestion or sometimes through penetration of (usually broken) skin by **radionuclides** which will then **irradiate**, the cells of surrounding body tissues.

Cosmic rays High energy ionising radiations from outer space.

Decay The process of spontaneous transformation of a radionuclide. The decrease in the activity of a radioactive substance.

Dose The amount of **ionising radiation** received as deduced from the energy absorbed from an external radiation source.

- **Absorbed dose** Quantity of energy imparted by ionising radiation to unit mass of matter such as tissue. Unit gray, symbol Gy. $1\text{Gy} = 1 \text{ joule per kilogram}$
- **Equivalent dose** The quantity obtained by multiplying the absorbed dose by a factor to allow for the different effectiveness of the various ionising radiations in causing harm to tissue. Unit sievert, symbol Sv.
- **Effective dose** The quantity obtained by multiplying the equivalent dose to various tissues and organs by a weighting factor appropriate to each and summing the products. Unit sievert, symbol Sv.

Dosimeter A small device worn on the person to measure absorbed energy and from which a record of **Absorbed Dose** may be obtained.

Dosimetry The estimating, recording and maintaining of records of **dose**.

Emitter A **radionuclide** decays by emission of certain radioactive particles and, or electromagnetic radiation. A particular **radionuclide** may be described as an **alpha** or **beta** or **beta/gamma** emitter.

Fallout The transfer of radionuclides produced by nuclear weapons from the atmosphere to earth; the material transferred.

Fission products The two, invariably radioactive, fragments remaining after an atom has been split (undergone fission).

Gamma ray A discrete quantity of electromagnetic energy without mass or charge. Emitted by a radionuclide. Cf X-ray.

Ionising radiation Radiation that produces ionisation in matter. Examples are alpha particles, gamma rays, X-rays and neutrons. When these radiations pass through the tissues of the body, they have sufficient energy to damage DNA.

Ionisation The process by which a neutral atom or molecule acquires or loses an electric charge. The production of ions.

Monitoring The process of searching for the presence of and then measuring, reporting and recording radiation **dose rates** found within a given area or on a person.

Neutron A nuclear particle (similar to a hydrogen atom but without electrical charge); emitted during fission and fusion by only a few **radionuclides**; long range (kilometres) in air and highly penetrating; an external **hazard** only at detonation; densely **ionising**.

Non-ionising radiation Radiation that does not produce ionisation in matter. Examples are ultraviolet radiation, light, infrared radiation and radiofrequency radiation. When these radiations pass through the tissues of the body they do not have sufficient energy to damage DNA directly.

Radiation weighting factor (RWF). A factor intended to take account of the relative biological effectiveness of different types of radiation according to both their energies and how densely ionising they are.

Radionuclide An unstable nuclide that emits ionising radiation.

X-ray A discrete quantity of electromagnetic energy without mass or charge. Emitted by an X-ray machine. Cf gamma ray.

Medical Adjudication Guidance on the evidence of Ms Sue Rabbitt Roff

Submissions have been received from Sue Rabbitt Roff in service-related ionising radiation cases. The evidence submitted includes extracts from her report on the health and mortality of members of the British Nuclear Test Veterans Association who were present at UK atmospheric nuclear test sites and clean-up operations.

This note first provides Departmental comment on Ms Roff's study. Part B deals with the case specific submissions.

A Departmental comment on "Mortality and Morbidity of Members of the British Nuclear Test Veterans Association and the New Zealand Nuclear Test Veterans Association and their Families"

Summary

The format of the paper does not conform to a scientific study suitable for publication in a mainstream peer review journal.

It is purely descriptive and contains no statistical analysis.

It does not compare the health experience of the study populations - either in terms of mortality or symptom occurrence - with the general population.

The study is based on self-report. The associated questionnaires are undisclosed.

The paper causally associates participation at an atmospheric nuclear test site with deleterious health effects with the implication that these effects are due to ionising radiation exposure. There is no comment on actual levels of ionising radiation exposure experienced. Rather an underlying assumption is made that all participants were subject to harmful ionising radiation exposure.

The case selection is subject to very considerable bias. Only about a tenth of all nuclear test participants joined the BNTVA. The aims and objectives of the association make it very likely that membership already implies selection on the grounds of poor health. The mortality study involves only a small sub-group of the total membership and the self-report mortality/morbidity study used data obtained from only half of the questionnaires issued.

General Comments

3. In late 1998, Ms Roff, a medical sociologist, submitted a study to the Department on the long-term health effects of participation in the UK nuclear test programme. The findings have been presented at scientific meetings but have not been published in a peer reviewed scientific or medical journal. The study is in 4 parts, two of which are relevant in a war pension context. An examination of 608 deaths of British Nuclear Test Veterans Association (BNTVA) members notified to that organisation between 1983 and mid 1997.

A description of mortality and morbidity among BNTVA members; an examination of 1041 responses to a questionnaire mailed to the 2087 current members of the BNTVA in December 1997.

A major difficulty of the report is that its data were collected from a self-selected cohort of BNTVA members. The total membership of BNTVA amounts to only about 10% of the total test participants. The aims of the Association make it unlikely that the BNTVA membership is truly representative of the total population who were present at the tests. That problem is obviously magnified in this study because it was based on subsets only of the already selected BNTVA membership.

The report relies on information about causes of death or illnesses provided by families of deceased veterans or in the case of illness, by the men themselves. It is not corroborated medically. The report is purely narrative and contains no formal statistical analyses. There is no control population against which comparisons are

made nor are comparisons made between her cohort and the health and mortality statistics readily available for the general population. The inferences drawn are largely based on the absolute numbers of deaths or diseases reported. There is no comparison of the death or health experience of the study populations with the general population. There is no data presented on likely radiation levels experienced, rather there is an underlying presumption that the men were exposed to excess ionising radiation which was deleterious to health.

Multiple Myeloma

The first sub-study, a mortality profile of the group, reports an excess of deaths from multiple myeloma. It should be noted as a general point, that in epidemiological work on causation, studies which relate causal exposures to disease outcomes in terms of incidence (ie determination of rate of occurrence of new cases) are more robust than those which use death certificate information as a measure of the condition under discussion. This is because of the inaccuracy and uncertainties found in death certification. In Ms Roff's study only a minority of the multiple myeloma cases reported were identified from death certificate information. The remainder are even less firmly based and arise from questionnaire responses and self-reporting.

The NRPB follow up studies

As a result of concern amongst some test participants about the effects that participation could have had on their health and the public pressure because of that, in the mid 1980s, the Ministry of Defence (MoD) commissioned a study by the independent ICRF and NRPB "To Investigate the Mortality and Cancer Incidence 1952-1990 in the UK Participants in the UK Atmospheric Nuclear Weapons Tests and Experimental Programme".

The study compared the mortality and cancer incidence in over 20,000 test participants with that of a similar sized control group of ex-servicemen who had not participated in the test programme.

The first NRPB follow-up study (Darby et al 1988) found that participation might have caused a small risk of multiple myeloma. The evidence was weak because of the exceptionally few cases arising in the control population. In the 1993 study, the additional data (follow-up until December 1990) did not support the earlier findings on multiple myeloma and the report concluded that the apparent increase in participants compared with controls reported in the earlier study was a chance finding. That remains the NRPB position.

To discuss whether or not the comparative likeness in the number of reported multiple myeloma cases currently recorded for the test vets and controls in the NRPB test vet database (which tracks the records of some 22,000 veterans and a similar number of matched controls) is statistically valid, the MOD has commissioned a further NRPB study. The conduct of this independent study is subject to oversight by a management committee including academics, medical specialists. The BNTVA has observer status on the management committee. It is likely to take about 2 years to complete and the report will be subject to peer review and publication in the mainstream scientific press.

There have been recent reports in the scientific and medical press of an increase in incidence of multiple myeloma in the general population world-wide. This may be a real effect or may relate simply to improved diagnosis of the condition. Multiple myeloma is predominantly a disease of old age - the minimum age of veterans is now 57 years and many are well over 70 years.

The Department's current position in relation to war pension claims is that a service causal link with multiple myeloma may be accepted in a nuclear test veteran but only where there is evidence of service related excess ionising radiation exposure and not purely as a result of participation in Britain's nuclear tests.

This policy came into effect in December 1993 - the date of publication of the second

NRPB report - and replaced an earlier policy, based on the first follow-up study, which accepted multiple myeloma as attributable to service. Cases may still occasionally be seen where multiple myeloma was accepted purely on the basis of participation. These decisions were correct at the time they were made and do not require revision.

The data in **The Second Rabbitt Roff Sub-Study** which is again narrative, looks mainly at disablement in the veterans. Information was obtained from responses to questionnaires sent to all members of the BNTVA and to relatives of deceased members. The actual questionnaire used has not been made public. Only 1,000 responses were received from the 2087 members of the organisation. There is no medical verification of any of the diagnoses reported.

All of these factors lead to considerable potential for inaccuracy and bias. It is likely, based on experience of other studies similarly based, that the health status of either the test veteran or his family would influence the likelihood of obtaining a response to the questionnaire. Among those who did respond, the amount of recall bias is also likely to vary depending on whether the veteran himself or his family completed the questionnaire.

The data presented show that there is an age bias among the responders with those replying being on average younger than expected from the age distribution of the whole cohort. Much of the discussion of the paper focuses on disorders of skin, bone and the muscular skeletal system which the author considers are unduly common in the study population. These problems are of course common in the general population with skin conditions accounting for about 15% of GP consultations in the UK, a point not made by Rabbitt Roff.

SAPHO Syndrome

The term SAPHO syndrome (**S**ynovitis, **A**cne, **P**ustulosis, **H**yperostosis, **O**steitis) was coined by French physicians in 1987 to designate a group of co-existing manifestations of which the most commonly occurring was osteitis of the anterior chest wall. The first ever report was a 1961 description of musculo skeletal problems accompanying severe acne (acne conglobata). (Windom et al 1961). There followed reports of bilateral clavicular osteomyelitis occurring in the presence of palmoplantar pustulosis (Kato et al 1968) (Giedon et al 1972). These initial reports were in children but in the late 1970s and 1980s further case reports appeared in adults.

The condition, as a diagnostic entity, however remains controversial and to date there have been only 2 cases reported in the literature in the UK. Not all features require to be present for the term to be applied, but the key feature is **osteitis of the clavicles**. Acute pseudoseptic arthritis may also occur and sacro-iliac involvement is common.

19. It is difficult at first sight to see the logic behind grouping these features together but the basis is as follows:

- i) Pustulosis and severe acne share the same basic process ie pseudo abscesses.
- ii) The bone involvement is radiologically and anatomically identical whether there is skin involvement or not and whatever its basis.
- iii) The anterior chest wall is invariably involved and in many cases which have been imported there is sacro-iliac involvement
- iv) Acute pseudo-septic arthritis may be involved with pustulosis, acne or psoriasis.

The actual pathophysiological link in the view of some authors is the organism propionibacterium acnes. While usually benign in course, the condition as reported is seriously disabling.

The paper discusses the likely existence of SAPHO syndrome due to ionising radiation exposure. The SAPHO syndrome is not universally accepted and is at most a case definition and not a pathological entity. The syndrome has not previously been discussed in terms of ionising radiation as a cause and no evidence is presented for that view. Rather it is hypothesis on the part of Ms Roff. From the

literature, it is clear that the description, SAPHO syndrome, is applied to individuals who are significantly disabled. The paper also contains speculation about palmo plantar pustulosis as a common condition in nuclear test veterans, again speculating about a link with ionising radiation. This is a very serious highly disabling condition which would not fail to come to medical attention. Anecdotally, it has rarely, if ever, been claimed for war pension.

Rheumatology and dermatology, particularly in relation to general medicine, are responsible and expert disciplines in the UK. There are well organised regular national and regional forums for case discussion, academic presentations and authoritative journals are published. It is therefore most unlikely that SAPHO syndrome or some variant thereof could be occurring and not be the subject of discussion or case report in the UK literature. Similarly, it is unlikely that its occurrence in a particular sub-group such as nuclear test veterans would go unreported.

References

Kahn, M et al (1992). SAPHO syndrome. Rheumatic disease clinics of North America. Vol 18 No 1: 225-246.

Brandsen R et al (1993). SAPHO syndrome. Dermatology: 186: 176-180.

Kahn M (1993). Psoriatic arthritis and synovitis, acne, pustulosis, hyperostosis and osteitis syndrome. Current opinion in rheumatology; 5: 428-435.

Medical/scientific reaction to the study.

Since Ms Roff's work has not appeared in the scientific press there has been limited opportunity for published comment from other researchers in the field. The Department's view is however echoed by recent comment in Australian and New Zealand government reports. Report details are to be found at the end of this document.

New Zealand

In an annex to the report of the Inquiry into the Health Status of the children of Vietnam and Op Grapple Veterans published in June 1999, there is the following comment on "Press releases and web site postings of Sue Rabbitt-Roff". – "Roff's evaluations are speculative and without foundation"

Australia

Ms Roff submitted her findings to the Australian government. (She is Australian and there is of course an interest, in that Australia was both the site of some of the tests and there was involvement of Australian personnel).

As a result earlier this year, Professor John Kaldor, Epidemiology, New South Wales was invited by the Minister of Veterans' Affairs to do a review of recent studies on nuclear test veterans. This included Ms Roff's work and the NRPB follow up studies.

In the final report Professor Kaldor's detailed comment on the NRPB studies, in line with general international reaction is very positive.

"comprehensive cohort study of high quality"

"within the framework of the epidemiological cohort study design, the NRPB studies appear to have been carried out to a very high standard".

The report also carefully scrutinised Ms Roff's work. Comments included:

"The studies did not use any standard epidemiological method for comparing the occurrence of death or illness in the study population with a relevant unexposed population".

"the possibility that the BNTVA membership was unrepresentative of the overall population of test participants was not adequately addressed in the methodology".

"Due to the substantial methodological limitation the Rabbitt Roff studies as currently presented do not provide any new information about the health risks of exposure to ionising radiation, nor about any health risks that may have been experienced by Australian participants in the UK tests".

B. Guidance notes on Ms Roff's case specific submissions

Ms Roff is an Australian sociologist working at Dundee University Department of Medical Education. She is not medically or scientifically qualified. She has a long association with the anti-nuclear movement and is a member of the BNTVA. Her recent study is discussed at A. above. It has been presented at a variety of scientific and medical meetings but it has not been published either in the proceedings of the meetings nor importantly in a widely recognised peer reviewed scientific or medical journal. It has recently appeared in the journal, *Medicine Conflict and Survival*. This is a medico political journal published by the Frank Cass publishing group and described as an officially designated journal of International Physicians for the Prevention of Nuclear War. The format and content of the paper do not conform to studies published in the mainstream scientific press.

Ms Roff's comments on war pensions cases are frequently submitted on University of Dundee notepaper. Some MAs have commented on this. Our understanding is that she writes on her own behalf and not in the name of the Department or University. While the practice is not one DSS or the WPA would endorse, Sol Branch have advised that no comment should be made or action taken on this account.

Ms Roff occasionally makes reference to her "other publications". It should be noted that she has occasionally contributed to the letters column of the *Lancet*. In January of this year she provided an article for the *New Statesman* on nuclear test veterans and she has also been quoted in journals such as the *New Scientist* where there is editorial sympathy for her viewpoint.

Another source of evidence cited by Ms Roff includes rather sensational articles from local newspapers or the Australian tabloid press. These frequently centre around residual environmental pollution. The articles are often highly political involving aboriginal resettlement issues and of limited relevance.

Ms Roff's evidence suggests a lack of appreciation of radiation dose issues. Because the convention is that there is no threshold for the carcinogenic potential of ionising radiation, she takes the view that there is no need to differentiate levels of radiation differing by orders of magnitude.

Ms Roff's submissions frequently quote (apparently without permission of appellant's or their estate) previous War Pension Agency and particularly, PAT decisions. In practice on many occasions there is very little common ground between the cases cited by Ms Roff and that under contention. The following advice has been received from Sol Branch on the issue.

War pensions is an individual jurisdiction with each case decided on its individual merits. As a general principle therefore Tribunals should decide each case on its own facts and not be bound by earlier PAT decisions. A leading case on the issue is the case of *Merchandise Transport Ltd v British Transport Commission* (1962) 2 QB 173. High Court cases decide legal principles and in that respect are precedent setting. However, again for direct extrapolation of case specific details, the compared cases would need to be absolutely similar

The submissions seek to undermine the validity and reliability of AWE dosimetry and the NRPB reports. Our response to that should simply take the line that the reports are in the public domain, are peer reviewed and accepted internationally. (See the discussion above). Fuller comment on the dosimetry issues is to be found in the ionising radiation statement.

Finally, on occasion Ms Roff quotes other jurisdictions eg Australia and US concluding that the UK provisions are less generous. Not infrequently she misquotes the provisions. Detailed information on overseas arrangements are held by medical policy - these are available and we would be happy to discuss. On most occasions

however it would be appropriate to simply say that the basis of the provisions is very different. In reality neither the Australian nor US schemes are markedly more generous than the UK approach. Both are highly influenced by political considerations, not science based.

References:

Roff (1999). Mortality and Morbidity of Members of the British Nuclear Test Veterans Association and the New Zealand Nuclear Test Veterans Association and their Families". *Medicine, conflict and Survival*. Vol 15. Supp 1. July-September 1999.

Darby et al (1988). Report on Mortality and Cancer Incidence in UK Participants in UK Atmospheric Nuclear Weapon Tests and Experimental Programmes NRPB-R214.

Darby et al (1993). Report on Mortality and Cancer Incidence 1952-1990 in UK Participants in the UK Atmospheric Nuclear Weapon Tests and Experimental Programmes NRPB-R266.

Kaldor J (1999). Report to the Minister assisting the Minister for Defence on recent studies of nuclear test veterans. University of New South Wales, Australia.

New Zealand Government 1999. Inquiry into the Health status of the children of Vietnam and Operation Grapple Veterans. Wellington.

July 2001

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MEDICAL ADJUDICATION GUIDANCE ON MALARIA

This note provides a guide on the medicine.

Malaria in humans is a protozoal disease caused by plasmodium falciparum: vivax: ovale: or malariae. It is transmitted by mosquito-bite and inside the mosquito the parasite undergoes its development cycle. Malaria is a disease of hot, wet climates and is endemic in the tropics and subtropics below 1500m, the exception being the Mediterranean hinterland, the USA and Australia.

The clinical effects of malaria are explicable in terms of the life cycle of the parasite. The female mosquito feeds on human blood and becomes infected with the gametocyte, ie, the sexual form of the malarial parasite. This develops in the mosquito into a **sporozoite**. The mosquito then bites a human and the **sporozoite** quickly homes to the liver. The transformation to **merozoite** occurs and leaving the liver, these enter the red blood cells where asexual replication occurs, to **schizonts**. These rupture, causing fever, whose periodicity depends on the species of the parasite.

Malignant tertian malaria, the most severe form, is due to plasmodium falciparum. The incubation period is 8-15 days. Blackwater fever may be seen when malignant tertian malaria is treated with quinine. Tertial periodicity is particularly marked during relapses. Relapses regularly occur up to one year and rarely **up to 4 years**.

Benign tertian malaria is caused by plasmodium vivax. This is the most common relapsing form. Incubation period is variable from 2-3 weeks to 38 weeks. Relapses may occur **up to 8 years**.

Quartan malaria (plasmodium malariae) has an incubation period of 15-30 days. Attacks are usually mild with relapse for **up to 50 years**, often associated with some stressful situation, eg surgical procedure.

Plasmodium ovale is rare and mild and relapses are uncommon.

Mixed infections are common in certain endemic areas.

In general a documented primary attack of malaria during service is attributable to service. If a malaria attack is documented post-service with no in-service reference, in reaching a decision on attributability, regard should be paid to the length of time the particular type of malaria can persist.

Reactivation: While not a common event, claims for re-infection are possible.

a. If there is documented pre-service malaria – which apparently persists during service but if there is no re-infection during service, malaria should be rejected NANA.

b. If a reactivation of a pre-service malaria occurs due to climate or other service compulsion, we should consider aggravation.

c. It is important to note that if service malarial infection involving a different type of malaria occurs, this should be accepted as attributable.

Assessment of malaria (dated) will usually be nil or 1-5%.

Disablement due to sequelae of malaria, such as splenomegaly, thrombocythaemia/thrombocytopenia and perhaps splenectomy, may be attributable

and result in an increased degree of disablement. Another contention is disablement due to the effects of malaria therapy.

With recommended doses serious adverse effects of quinine are rare. Quinine produces a reversible hearing loss and tinnitus that disappears upon withdrawal of the drug. The degree of threshold shift correlates closely with the concentration of the drug in blood plasma.

CINCHONISM (quinine is derived from the bark of the cinchona tree) is a common syndrome which occurs when a patient is taking the drug, appearing usually within 1-3 days and subsiding quickly when the drug is withdrawn. It comprises tinnitus, headache, nausea, abdominal pain, minor visual disturbances, transient loss of hearing and tremors.

February 2001

WAR PENSIONS POLICY STATEMENT NOISE - INDUCED HEARING LOSS

Summary

The Department's policy is that:

a war pension may be awarded for any disablement causally linked to service. The scheme does not provide awards for disablement that is not caused by service. Anyone who has served in the armed forces can claim a pension at any time from service release. Decisions are evidence based, reflecting the facts of the individual case, the relevant law and policy and contemporary scientific and medical understanding of the claimed condition;

disablement is defined as "physical or mental damage, or loss of physical or mental capacity". In this case the disablement is bilateral noise-induced sensorineural hearing loss due to noise damage in service;

if a disablement is accepted as caused by service, the degree of disablement due to service is assessed by comparison of the person as so disabled with a normal healthy person of the same age and sex;

the contemporary evidence on the relationship between noise-induced hearing loss and age-related hearing loss has been studied: an expert review of the evidence in 1997-98, and the further Departmental review in 1999, found that there was no reliable evidence to raise a reasonable doubt that noise-induced hearing loss increases after removal from the source of injurious noise, or as age-related hearing loss is added to it;

to ensure that an assessment in respect of service-related noise-induced hearing loss does not include hearing loss which arises after service and is due to non-service causes, assessment is based on evidence of the amount of hearing loss present at service release;

the degree of disablement in respect of hearing loss of less than 50 decibels in both ears, averaged over the frequencies of 1, 2 and 3 kilohertz, is to be assessed at under 20 per cent;

where the degree of disablement in respect of noise-induced hearing loss caused by service is assessed at under 20 per cent, no account is to be taken of related conditions or symptoms in assessing the total degree of disablement due to service;

no award is payable if the assessment of the degree of disablement in respect of noise-induced hearing loss due to service is under 20 per cent;

each case must be determined on its' own individual circumstances and evidence.

[back to top of the page](#)

WAR PENSIONS POLICY STATEMENT - NOISE-INDUCED HEARING LOSS

The Issue

In recent years, the majority of claims for war pension have been for deafness or, more specifically, hearing loss. Although there are several medical conditions that may give rise to hearing loss and lead to the award of a war pension, the most common is **bilateral noise-induced sensorineural hearing loss**.

This statement considers theoretical and practical aspects of the approach to claims for noise induced hearing loss under the war pensions scheme, including:

A. the law - the legislative context and case law ([paragraphs 3 to 15](#))

B. diagnosis of noise-induced hearing loss ([paragraphs 16 to 18](#))

C. causation - whether there is reliable evidence of service cause, including the isolation of service related noise-induced from age related hearing loss ([paragraphs 19 to 26](#))

D. the assessment of noise induced hearing loss caused by service ([paragraphs 27 to 33](#))

E. the approach to associated and consequential symptoms and conditions ([paragraphs 34 to 35](#))

F. awards for noise-induced hearing loss ([paragraph 36](#))
[back to top of the page](#)

A. The Law

How the scheme works

The scheme is administered under the authority of the Service Pensions Order (the "SPO"). A war pension may be awarded for disablement **due to service in the armed forces**. "Disablement" is defined as "physical or mental injury or damage, or loss of physical or mental capacity". "Injury" includes wounds and diseases.

A war pension may be claimed for any disablement by anyone who has served in the British armed forces. Claims may be made at any time after service release. Decisions are evidence based and each case must be determined on its own individual circumstances. Awards may be made where, within the relevant law, the evidence (including service and medical factors) and the contemporary medical understanding of the condition claimed, shows a causal link between service and the claimed condition. Decisions are made using the relevant burden and standard of proof as referred to in paragraphs 5 to 10 below.

The first stage in any claim is to **show disablement**. Article 3 of the SPO puts the onus on the claimant to show disablement. The standard of proof at this stage is the **balance of probabilities**. In effect, the claimant needs to show that it is more probable than not that he or she has suffered the disablement.

The nature (diagnosis) of the claimed disablement also has to be established. Diagnosis is a medical matter and the approach is therefore similar to that which would be applied in a clinical setting – where diagnosis will inform the most appropriate medical treatment. These matters have to be resolved before considering

any possible causal link between the disablement and service.

7. The test of proof for deciding whether service was the cause of disablement under the war pensions scheme is peculiar to the scheme:

a) for claims made not more than 7 years after leaving the armed forces, **Article 4** of the SPO provides that the onus is on the Secretary of State to show beyond a reasonable doubt that the claimed disablement is not attributable to, or aggravated by, service;

b) for claims made more than 7 years after the end of service, **Article 5** of the SPO puts the onus on the claimant to raise, by way of reliable evidence, a reasonable doubt that the claimed condition is attributable to, or aggravated by, a service injury.

8. If it is accepted that the claimant is suffering from a disablement due to service, the degree or level of that disablement is assessed. The assessment forms the basis of the award which may be paid. The law sets out the method of assessment. Assessment is on a percentage basis, with 100 per cent representing maximum disablement, and is made by comparing the condition of the claimant, as disabled by service, with that of "a normal healthy person of the same age and sex". Earning capacity and "the effect of any individual factors or extraneous circumstances" are not taken into account. The scheme contains a range of allowances which recognise the disabling effects of the accepted condition on the individual, for example restricted mobility or the need for attendance. There is also an age allowance, which increases in rate with age and the degree of assessed disablement.

A war pension may be awarded for any disablement due to service. To maintain consistency and equity in assessment across the range of conditions, the SPO contains a number of scheduled assessments, which also serve as benchmarks for the assessment of disablements not listed in the schedules. Article 9 also contains specific provisions on the assessment of hearing loss.

The standard of proof when making an assessment is the normal civil law test of the **balance of probabilities**.

Once the percentage degree of disablement due to service has been assessed, the level of award is determined.

[back to top of the page](#)

Case Law

12. Over time, the High Court has produced a number of judgements, which provide legal interpretation of war pensions provisions. These relate only to questions of entitlement— there is no right of appeal to the High Court on matters of assessment.

13. The High Court has held that the word "reliable", in the context of Article 5, cannot have been intended to mean "convincing", but means more than "fanciful". In the case of *Edwards* the judge held, with particular reference to "changes of medical opinion", that: "*there are,.... in my judgement, three stages: no reasonable doubt, reasonable doubt, and consensus.*" A war pensions claim under Article 5 would pass the test at the point where the (reliable) evidence raised a reasonable doubt, but: "a

mere hypothesis based on a limited study.... would not have created a "reasonable doubt" within the terms of Article 5(4)". The real question, however, is whether the evidence raises a reasonable doubt in the mind of the Secretary of State (SoS). If he finds the evidence unreliable, it obviously will not raise a reasonable doubt in his mind.

14. The Courts have also held that a conflict of medical opinion does not, of itself, mean that a reasonable doubt has been established, and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of *Tigg v The Minister of Pensions* the presiding Judge stated:

"Merely because a doctor of eminence, and I have no doubt the doctor in this case was of very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean there is a doubt and the Appellant must therefore be entitled to a pension. It is a question of fact for the Tribunal".

15. The issues to be decided when determining a claim are therefore:

whether the claimant is suffering from noise-induced hearing loss

whether there is reliable evidence of service cause: i.e. sufficient exposure to levels of loud noise in service to cause noise-induced hearing loss

the degree of hearing loss due to that service cause

[back to top of the page](#)

B. Diagnosis of noise-induced hearing loss

16. Diagnosis of noise induced hearing loss in the individual case can be difficult. Noise injury damages hearing, but individuals vary widely in their susceptibility, as well as in the amount of noise they are exposed to. Noise injury first gives rise to a temporary hearing loss, which recovers on removal from the noise. The term noise induced hearing loss refers to the permanent bilateral sensorineural hearing loss that results from repeated or continuous exposure to chronic or intermittent loud noise for several years. There are no specific signs or symptoms to distinguish noise induced hearing loss from other forms of hearing loss.

17. The Medical Research Council's recent National Study of Hearing has shown that significant sensorineural hearing loss is common in the UK population as a whole, particularly as people age (Davis 1989). The study also shows that noise injury related to work of all types probably contributes less to the hearing loss of the population than previously thought. As part of general improvements in health and safety, hearing conservation measures have been increasingly in place in the armed forces since 1963, culminating in implementation of the Noise at Work Act 1989. New cases of hearing loss due to chronic exposure to noise in service will therefore become increasingly rare. (Hearing loss due to other potentially service-related causes, such as infection and physical trauma, may still occur and may lead to pension.)

18. Hearing loss is normally measured using pure tone audiometry. The audiogram pattern across the frequencies 0.5 to 8 kHz is used in diagnosis of noise induced hearing loss. There are many possible causes of sensorineural hearing loss other than noise, but noise damage always produces greater loss at 3, 4 and 6 kHz than at 0.5, 1 and 2 kHz. The maximum is frequently at 4 kHz, producing a characteristic notch in the audiogram. However, with time, hearing loss at adjacent frequencies may catch up due to the effects of ageing. In steady state conditions, the loss at 3, 4 and 6 kHz due to exposure to noise reaches a maximum in 10-15 years. Diagnosis of noise induced hearing loss in war pension cases is a medical matter informed by several factors. In addition to the audiometric evidence, war pension medical officers must also take into account the service history, particularly the evidence of the claimant's exposure to service noise.

C. Causation

19. Most claims for hearing loss are made under Article 5 of the SPO. For a current claim to be considered under Article 4 of the SPO, service would have to have terminated less than 7 years ago. Under Article 5, noise-induced hearing loss may be accepted as due to service if there is reliable evidence to raise a reasonable doubt that it is so caused.

[back to top of the page](#)

Isolation of service related noise-induced hearing loss

20. An individual's hearing is subject to many influences over a lifetime. Firstly, there is a wide constitutional variation in hearing amongst individuals (Lutman and Davis 1996). As well as noise, sensorineural hearing loss may be due to infection and certain medicines, and in addition hearing is significantly affected by age (Davis 1989).

21. Since a war pension can be claimed at any time after discharge from the forces, sometimes fifty years or more after leaving service, people may claim when they are quite old. Important questions for war pensions and other compensation schemes are, therefore:

- a. whether hearing loss due to noise injury gets worse after removal from noise or
- b. whether it gets worse when age-related hearing loss is added to it.

If exposure to noise in service were to cause an increase in the level of overall hearing loss following removal from the noise, i.e. after service, war pensions assessments might be increased to reflect this.

22. Since the 1970s, a substantial body of scientific literature has built up on different aspects of noise-induced hearing loss (see Annex A for details). The scientific evidence on the relationship between hearing loss due to age and noise, In

the context of the War Pensions Scheme, was reviewed by a team of independent experts in 1997/98, and again by the Department in early 1999.

23. The specific questions addressed by the reviews were therefore whether, having regard to war pensions legislation and case law, there was reliable evidence to raise a reasonable doubt that noise-induced hearing loss increases following removal from the noise injury or as age related hearing loss is added to it.

[back to top of the page](#)

Reliable evidence

24. In assessing the evidence particular regard has therefore been paid to:

whether conclusions of studies are hypotheses; or whether there is supporting evidence going beyond hypothesis, and if so its basis

the reaction of other experts in the field to the evidence

the weight of overall evidence on the matter.

25. Evidence for any new approach in science must always be considered and weighed relative to the existing body of evidence on a subject, with account taken of the robustness and authority of new studies. In particular the design and methods, sample size, case and control selection, statistical validity, repeatability of findings, approach to bias and possible alternative factors and hidden influences. Other important factors include whether the findings have been replicated by other independent researchers and the overall plausibility/consistency relative to contemporary understanding.

26. The expert review of the evidence in 1997-98 and the further Departmental review in 1999, which was informed by an extensive search of the scientific literature and the advice of a range of experts on evidence relevant to the consideration, found that there was no reliable evidence to raise a reasonable doubt that noise-induced hearing loss increases after removal from the source of injurious noise, or as age-related hearing loss is added to it.

[back to top of the page](#)

D. The assessment of noise induced hearing loss caused by service

Measurement of hearing loss

27 There are many compensation schemes dealing with hearing loss but no single agreed method for its assessment. Most high volume schemes use pure tone audiometry as a reliable measure of hearing loss. The DSS method derives from the Industrial Injuries Advisory Council (IIAC) and the British Association of Otolaryngologists (BAOL) (IIAC Report 1973). Hearing threshold level is measured by pure tone audiometry at 1, 2 and 3kHz. These are the principal frequencies involved in distinguishing speech. They also produce reliable, repeatable audiometric results. The same frequencies are used in United Kingdom courts in negligence cases. Where the results of PTA are considered unreliable, unrepeatable or in some other way unrepresentative of true hearing ability, cortical evoked response audiometry ("cERA") may be carried out. CERA is more complex and testing is more prolonged, but the results are more objective, involving minimal subjective response from the person being tested. Technical aspects of hearing loss assessment will be

kept under review for possible application to the scheme

Evidence of the degree of hearing loss caused by service

28. Since noise induced hearing loss does not increase following removal from the hazardous noise or as age-related loss is added to it, and as a war pensions assessment may only include that which is caused by service, the war pensions approach is to assess the individual's hearing as it is at or about service release, using evidence from as close to that date as possible. Service personnel undergo a release medical examination including measurement of hearing ability. The nature of the medical evidence at release may vary depending on the dates of service. In cases since the mid-1980s PTA should be included, while before that date, clinical testing, normally by forced whisper and/or conversational voice, was the standard method.

29. It is accepted that hearing loss measured at service release will include some loss due to constitution and ageing, as well as loss possibly due to other non-service factors. While it is understood that noise-induced hearing loss does not get worse after removal from the noise - and that any deterioration in overall hearing in an individual cannot therefore be due to service noise - there is no generally accepted method of accurately separating the measured loss due to service noise from other causes operating at the same time. The Department's policy is therefore to include, in an assessment of noise induced hearing loss caused by service, **all** hearing loss present at the point of service release. The same approach is taken to occupational deafness under the industrial injuries scheme.

[back to top of the page](#)

Applying the evidence

30. Evidence recorded in official documents - i.e. in records of service medical examinations performed in service and at discharge - is assumed reliable **unless there is other relatively contemporary evidence to challenge it**. For example, if a service discharge medical record shows hearing to be normal clinically, but there is evidence from another source, such as civilian GP or hospital notes from around the same time, showing hearing loss present. In such cases, if the alternative evidence is reliable, it may be accepted in preference to the service record.

31 If there is no evidence from service release - for example because records are missing - the decision should be informed by the earliest available evidence.

Assessment

32. The legal basis for assessment in war pensions is described above. The scheduled assessments generally relate to traumatic physical injuries typically seen in war. Loss of senses is included, and absolute deafness is given an assessment of 100 per cent. There is otherwise no reference to hearing loss in the schedule.

33. Where hearing loss is not total, the current approach to translating measured hearing loss caused by service into a percentage assessment is:

where there is audiometric evidence from at or close to service release, the measured hearing loss, expressed in decibels (dB) and averaged over 1, 2 and 3 kHz, is applied to a scale linking levels of hearing loss to percentage assessment. The scale was advised by IIAC and the BAOL and takes into account the scheduled assessments. On the scale, bilateral hearing loss of 50 dB averaged over 1, 2 and 3 kHz equates to 20%. ("Absolute deafness" - a statutory 100% assessment - equates to a bilateral loss of 106 dB on the scale). In line with this, Article 9(2A) of the SPO provides that hearing loss of less than 50 decibels in both ears - averaged across the frequencies of 1, 2 and 3 kilohertz - shall be assessed at less than 20 per cent;

where there is no audiometric evidence from at or close to service release, but there is clinical evidence from that time, the assessment should be informed by that clinical evidence (e.g. response to forced whisper or conversational voice).

[back to top of the page](#)

E. Associated/consequential conditions and symptoms

34. Claims for noise induced hearing loss may include associated symptoms (such as tinnitus) or contend that the noise-induced hearing loss (or tinnitus) has caused a further discrete disablement.

35. The SPO provides that, where noise-induced hearing loss itself is assessed at under 20 per cent, neither the noise-induced hearing loss itself, nor related conditions or symptoms, shall be taken into account when determining the total degree of disablement due to service. The approach in practice depends on whether the case involves (a) a related **symptom**, or (b) a further **condition**.

Related symptoms – tinnitus

Tinnitus is a common symptom in the adult population occurring at some time in about 30% of adults and increasing in incidence with age. Tinnitus is subjective and may be intermittent or improve over time. (Axelsson and Coles 1996). It is considered seriously disabling in only a small proportion of people.

There is no consensus approach to tinnitus in compensation schemes. Indeed because of its subjective nature, many schemes do not compensate for tinnitus while others give a flat rate addition. Details of some approaches in other jurisdictions are at Annex B.

Under the war pensions scheme, tinnitus is treated as part of the underlying hearing loss. Cases are considered on an individual basis. An assessment for noise-induced hearing loss may be increased to make allowance for tinnitus when the noise-induced hearing loss itself is assessed at 20 per cent or more. The addition made for tinnitus is a matter for medical judgement

[back to top of the page](#)

Related conditions - consequential psychiatric disablement

On occasions, a claim may be made that a further disablement has developed because of the accepted hearing loss or tinnitus. The most commonly claimed conditions are depression and anxiety.

Rarely, where the claimed condition is a separate diagnostic entity, and it is due to the service-related noise-induced hearing loss (including any tinnitus), that condition may also be accepted as due to service.

As it is a separate condition, it should not be considered as part of the noise-induced hearing loss. It should be assessed separately from the hearing loss, but it can be included in the overall composite assessment **only if** the underlying noise-induced hearing loss (excluding tinnitus) is assessed at 20 per cent or more.

[back to top of the page](#)

F. Awards

36. If the degree of disablement due to the hearing loss alone is assessed as being **less than 20 per cent**, the SPO provides that no award shall be made for:

the noise-induced hearing loss itself, or

any related symptom, such as tinnitus, or

any related condition, such as a consequential mental disablement.

This applies regardless of the assessment that might be appropriate to the related symptom or condition

CONCLUSION

Assessments and awards for noise-induced hearing loss

37. Under the rules of the war pensions scheme, the degree of disablement in respect of hearing loss of less than 50 decibels in both ears, averaged over the frequencies of 1, 2 and 3 kilohertz, is assessed at under 20 per cent;

38. Where the degree of disablement in respect of noise-induced hearing loss caused by service is assessed at under 20 per cent, no account is taken of related conditions or symptoms in assessing the total degree of disablement due to service;

39. No award is payable if the assessment of the degree of disablement in respect of noise-induced hearing loss due to service is under 20 per cent;

The relationship between noise-induced hearing loss and age related hearing loss

40. A war pension may be awarded for disablement causally linked to service. The scheme does not provide awards for disablement that is not caused by service

41. The contemporary evidence on the relationship between noise-induced hearing loss and age-related hearing loss has been studied and carefully evaluated. An expert review of the evidence in 1997-98, and the further Departmental review in 1999, found that there was no reliable evidence to raise a reasonable doubt that noise-induced hearing loss increases after removal from the source of injurious noise, or as age-related hearing loss is added to it;

42. To ensure that an assessment in respect of service-related noise-induced hearing loss does not include hearing loss which arises after service and is not caused by service, assessment is based on evidence of the amount of hearing loss present at service release.

[back to top of the page](#)

ANNEX A

HISTORY OF THE UNDERSTANDING OF THE RELATIONSHIP BETWEEN NOISE-INDUCED HEARING LOSS AND AGE RELATED HEARING LOSS

1. From the 1950s, it was held that hearing losses due to age and noise were simply additive. The thinking was that noise destroyed cochlear hair cells and so was localised to this part of the inner ear, while the effects of age were more widespread in the auditory system. The two processes of change due to noise and age were therefore independent. Therefore, when they were both present in the same person, they were simply additive. At the same time, it was known that either age or noise injury acting alone could produce total hearing loss. That being so, they could not be fully additive at their extremes - a person totally deaf because of noise, for example, could not suffer greater hearing loss with age - so at the limits noise and age must be less than additive.

2. In the classic study on Hearing and Noise in Industry, (Burns and Robinson 1970) a modified, less than additive formula was proposed for the combination of hearing loss due to age and noise. In 1975 the Burns and Robinson proposal became the basis of the **International Standard** (the present edition is ISO 1999: 1990). This underpins most medico-legal work, including court settlements, in the UK and overseas. (Hanley, 1998) In 1987, on review of the data (Robinson 1987), it was pointed out that ageing had a greater effect than previously appreciated. Several studies suggest that, at the age of 80 years, hearing is the same whether the ear is noise exposed or not (Macrae 1971; Rosenhall, Pederson and Svanborg 1990).

3. In 1992 a modelling study on noise-induced hearing loss in the medico-legal context, including a detailed discussion on prognosis and prognostic assessment, was published (Lutman 1992). The discussion concluded, "in the interests of scientific parsimony it is recommended that prognosis allowances should be nil", i.e. no allowance should be made for future increase in overall hearing loss.

4. Also in 1992, a report of an expert working group on Assessment of Hearing Disability - for use in medico-legal practice was published. (King, Coles, Lutman and Robinson 1992). The report, known as the **Black Book**, recommended an assessment approach which is now standard in negligence cases in the British courts. It discusses the further course of the hearing of a noise exposed individual in

the years following removal from noise injury and, in line with Lutman (1992), concludes "For the purposes of this report, the anticipated increase in NIHD [noise induced hearing disability] after the date of assessment, sometimes referred to as the prognosis allowance, is deemed to be zero".

5. In March 1998 the Irish government, Department of Health and Children published "Hearing Disability Assessment", a report containing the recommendations of an expert working group on a system and criteria for the assessment of disability associated with hearing loss, particularly noise induced hearing loss. The report discusses the relationship between age and noise induced hearing loss. The line taken in this report also reflects the International Standard:

"Excessive noise may damage the same hair cells in the cochlea that degenerate with advancing age causing hearing loss and possibly tinnitus. The effects on hearing of ARHL [age related hearing loss] and NIHL [noise-induced hearing loss] are additive, though less than the sum of the two causes (Williams 1996). Obviously, if hair cells have been damaged by one of these causes, they cannot be re-damaged by the other. The effect of the noise component on the true hearing threshold progressively diminishes with time. Thus, at the age of 80 years it makes virtually no difference to an individual's hearing ability what his/her noise exposure has been (Robinson 1988)".

[back to top of the page](#)

ANNEX B

THE APPROACH TO THE ASSESSMENT OF TINNITUS

In the UK civil courts tinnitus has generally been accepted as occupational in origin where it occurs with noise induced sensorineural hearing loss and arises within a year of removal from noise injury. There is no agreed method of assessing the tinnitus and approaches elsewhere therefore vary considerably.

Many compensation schemes do not compensate for tinnitus at all. Others recognise the effect of tinnitus by giving a notional flat rate addition to the assessment where tinnitus accompanies a level of noise induced hearing loss for which compensation is payable, for example in Australia where an additional 5 per cent can be allowed.

In schemes which allow a variable addition to an assessment of hearing disablement the additions for tinnitus are usually low, for example in Germany where tinnitus accompanying compensatable noise induced sensorineural hearing loss can attract an additional 2.5 to 10 per cent.

In the USA, the US Department of Veterans' Affairs Guidelines sets out six criteria which must be satisfied before tinnitus can be recognised:

- a. the claim must be unsolicited;
- b. for compensation to be paid there must be a compensatable degree of hearing loss;
- c. there must have been an attempt to relieve the tinnitus prior to the claim - eg through counselling, or drugs or hearing devices;
- d. there must be evidence to support personality change/sleep disorders;
- e. there must be no history of substance abuse or use of therapeutic drugs, and

f. any complaint of tinnitus must be substantiated by family or someone described as a "significant" other.

A new system of hearing disability assessment has recently been proposed in Ireland (Irish Department of Health and Children 1998). This recommends compensation should be paid for tinnitus only where evidence confirms that it occurs for 50% of the time and has existed for 2 years. Onset must also have close time relation to the hearing loss and the complaint should be medically documented prior to the benefit claim. Suggested assessment additions are: mild tinnitus nil %; moderate 2% and severe 6%.

[back to top of the page](#)

REFERENCES/BIBLIOGRAPHY

General Texts

Alberti P (1997). Noise and the ear in Kerr, A (ed) Scott-Brown's Otolaryngology. 6th edition. Vol 2, 11: 1-34 Oxford. Butterworth Heineman.

Davis A C (1995). Hearing in Adults. London. Whurr.

Davis A (1997). General Background - Epidemiology in Kerr A (ed) Scott-Brown's Otolaryngology. 6th ed. Vol 2, 3: 1-38 Oxford, Butterworth Heinemann.

Orgler et al. (1987) American Occupational Medical Association noise and hearing conservation committee guidelines for the conduct of an occupational hearing conservation program. J. Occup. Med; 29: 981-982.

Sataloff J (1957) Industrial Deafness. New York. Dekker.

Sataloff R S (1993) Occupational Hearing Loss. 2nd ed. New York. Dekker .

Included in the policy statement and annexes

Burns W and Robinson DW (1970). Hearing and Noise in Industry. London: HMSO.

Davis A (1989). The prevalence of hearing impairment and reported hearing disability among adults in Great Britain. Int. J. Epidemiol; 18(4): 911-7.

Hanley v the Minister for Defence, High Court (1996/5599P). Irish Current Law Digest; August/September 1998.

Industrial Injuries Advisory Council 1973 Report on occupational deafness. Cmnd 5461. London HMSO.

International Organisation for Standardisation (1990). Acoustics - Determination of occupational noise exposure and estimation of noise induced hearing impairment. International Standard. 2nd ed. 1999:1990

Irish Department of Health and Children (1998). Dublin. Hearing Disability Assessment.

King P F, Coles R R A, Lutman M E and Robinson D W (1992) Assessment of Hearing Disability. London. Whurr.

Lutman M E (1992). Apportionment of noise-induced hearing disability and its prognosis in a medico-legal context: a modelling study. Brit. J. Audiol; 26: 307-319.

Lutman M and Davis AC (1996). Distributions of hearing threshold levels in populations exposed to noise in Axelsson A et al (eds) Scientific Basis of Noise

Induced Hearing Loss. New York. Thieme.

Macrae J H (1971). Noise induced hearing loss and presbycusis. *Audiology*; 10: 323-333.

Robinson D W (1988) Impairment and disability in noise induced hearing loss. *Adv. Audiol*; 5: 71-81.

Rosenthal U et al (1990). Presbycusis and noise induced hearing loss. *Ear and Hearing*; 11: 257-263.

Williams R G (1997). Review paper: the diagnosis of noise induced hearing loss. *J. Aud. Med*; 6(1): 45-58.

[back to Medical Information Contents](#)

Departmental Comment on the Further Evidence Submitted by Royal British Legion on Ageing in Noise Exposed Ears

Synopsis of the Gates Study¹

1. The paper reports a prospective study using data from the Framingham Heart Study which examines whether the ageing process in noise damaged ears is the same as in unexposed ears. It looked at the 15 year changes in hearing threshold level of 203 men whose average age at the outset was 64 years (range 58 - 80 years). A proportion of the men had worked in heavy manufacturing industry. It was assumed at the outset of the study that the subjects were retired and that over the fifteen years of follow-up they had no further noise exposure. Rather any changes in hearing threshold level would be due to age.

2. The first audiometric sign of noise induced hearing loss is a loss of sensitivity in the frequencies 3 - 6 kHz leading to a characteristic audiometric "notch". Presence of such a high frequency "notch" is taken as evidence of noise damage. A validated mathematical method was used to identify high frequency notches with depth to width ratios of 0.5 or greater in the initial audiograms of the 203 men. Audiograms were classified as having no notch, N0 (0 - 15 dB), a small notch, N1 (16 - 35 dB) or a large notch, N2 (more than 35 dB).

3. The study reports the 15 year pattern of change in mean age adjusted pure tone threshold as a function of notch category. At frequencies 0.25 - 1 kHz there was no difference in threshold change across the notch groups. In the frequencies 3 - 6 kHz ie those already damaged by noise, the change for those with N2 over the 15 year period was less than for N0 and N1. This is as expected as there are a finite number of cells to damage in the cochlea and if already damaged by noise, cells cannot be further damaged by ageing. At 2 kHz ie on the margin of noise sensitivity there was a clear increase in threshold shift change over the follow-up period dependent on notch category.

4. The authors conclude that in the population studied, noise damaged ears do not age at the same rate as non-noise damaged ears and that contrary to conventional wisdom² which holds that the effects of noise injury do not progress on removal from the noise, the increased loss over time at 2 kHz suggests that noise injury effects continue after noise exposure has ceased. Careful analysis of the paper and of studies referenced in it suggests that this interpretation, even constrained to the study population, is an overstatement.

5. The paper discusses only **mean** hearing loss and gives no idea of variation both amongst individual initial hearing levels within the various notch groups nor of the variance over time. The change in dB/15 years was calculated from measured hearing threshold levels at only 2 points over the follow-up period. The paper briefly acknowledges the complexities of noise and age related hearing loss, the confounding nature of noise and age and the fact that for any measured overall hearing level, accurate quantitative apportionment on basis of noise and age is not possible. The paper does not expand on these issues. With an average age of 64 years in the study population at the outset there would already be a considerable age-related component to the overall hearing loss, regardless of noise exposure.

6. The paper makes the important **assumption** that the men were retired and therefore had no further occupational or other noise exposure during the 15 year follow-up phase. No evidence was specifically gathered or recorded on that issue. It should be noted that a companion paper³ using the same population states that the

subjects were not questioned about exposure to non-occupational noise sources at the outset. However at the 15 year examination 63% of those tested indicated that they had used guns. Recreational shooting is common in US and there is no obvious/inevitable age barrier. World wide, DIY and gardening pursuits often using noisy equipment, are popular amongst the retired population. There must therefore be serious reservations about such a blanket conclusion.

7. In the discussion section the paper records that "the finding of an accelerated rate of loss over time in the frequency area adjacent to the noise notch" is a new finding. This is not so. It later makes reference to a similar report in a study⁴ on 16 retired jute weavers who had been noise exposed for 30 years. A 1990 longitudinal study⁵ of noise exposed men examined at ages 70, 75 and 79 years also recorded this. "In the group of men previously in their life exposed to long-lasting noise the hearing deteriorated much more from age 70 to age 75 at 2 kHz than those not exposed to professional noise". This paper goes on to interpret this finding thus: "this further supports the concept that socio-economic factors other than exposure to noise are important in the group of males with a history of occupational noise". It makes no suggestion that the finding supports the proposition that noise injury effects continue following removal from noise injury.

Age-related hearing loss

8. The paper makes reference to, but does not discuss in detail, the complexities of age-related hearing loss. It identifies chronic noise exposure as its major component. Studies of primitive populations in virtually noise-free environments⁶ and who have excellent high frequency hearing into old age, support this view. However other processes including time related degeneration, cumulative effects of extrinsic insult due to diseases, particularly cardiovascular, and intrinsic insult due to wear and tear and age-related susceptibility to disease are also involved. It is of note that the rural African populations are free not just of noise but also of atherosclerosis, high blood pressure and have low cholesterol. There may also be issues about their precise ages and genetic factors are likely to be important. Evidence on the role of these various elements in age-related hearing loss remains an enigma. It is at best conflicting and certainly it is not possible to correlate anatomical, functional and audiometric change with ageing accurately.

9. Since 1965 the Baltimore Longitudinal Study of Ageing⁷ has measured hearing threshold levels from 500 Hz to 8 kHz to establish longitudinal patterns of change in hearing thresholds over time in a population, screened to exclude otological disease, unilateral hearing loss and importantly noise induced hearing loss. The results - even in this highly selected group with minimal possibility of noise-age interaction - show highly variable patterns of change in hearing level at the various frequencies over time. Important findings include the fact that age associated hearing loss starts young and is not confined to high frequencies, the loss at 500 Hz being detectable earlier than at the higher frequencies. The longitudinal decline at 8 kHz is relatively constant after age 50 years but the rates of change in other frequencies continue to increase with age until they catch up with the 8 kHz. It is also shown that individuals do not necessarily track on the same hearing level over time. These facts are relevant to the Gates' study and its assumptions. In essence, they mean that even in a highly screened non noise exposed population no "normal" pattern of hearing loss with age can be established.

Noise Induced hearing loss

10. The 1990 NIH Consensus Development Conference on Noise and Hearing loss confirmed that noise injury causes initial high frequency damage with development of a notch on the audiogram. With additional hearing loss from noise or ageing the threshold at 8kHz may worsen, eliminating the clear audiometric pattern. The Consensus Conference report goes on "... the presence or absence of noise induced

hearing loss cannot be established on the basis of the audiometric shape per se." That must apply to the elderly subjects of the study.

11. The submitted paper lists some caveats and **does** state that a high frequency notch is not prima facie evidence of noise damage. It does not however offer alternative explanations of notching and in its interpretation of its results retains the underlying assumption that high frequency notches are due to noise. By assertion only, the paper states that the cohort studied was suitable because noise induced hearing loss is common in the population, its prevalence supported by histories of noise exposure, self-attribution of hearing loss to noise and characteristic audiometric findings. To support this statement, cross-reference is made to an earlier companion paper on the same population.³

12. The Framingham Heart Study began in 1948 following up more than 5,000 subjects, male and female, initially aged 30-62 years. The focus being on cardiovascular disorders and subjects seen every two years.

13. Pure tone audiometry was carried out at biennial examinations, 15, 18 and 22. At every routine two-year examination, subjects were asked if they had experienced sufficient noise or vibration at work to cause bodily harm if endured day after day. As part of the 15, 18 and 22 biennial examinations they were also asked if they thought that exposure to noise was a factor in their hearing status. The answers were not subject to corroboration by evidence and no measured noise exposures were available.

14. Mathematical curve fitting to detect audiometric notches (as described at paragraph 2 above) was carried out at examination 15. The notch categories had slightly different definitions from those described in the later paper. In N0 the notch equated to 0-19 dBs, for N1, 20-39 dB and N2 described notches with depths great than 40 dBs.

15. In the male subjects there was a clear relation between years of occupational noise exposure and the presence and size of audiometric notches. However, there were important discrepancies.

Table 1

The relation of occupational noise exposure history to the notch size category and self-report of hearing loss attributable to noise

| Men | | | | |
|----------------------|------------------|----|------------------|-----|
| Maximum Notch Depth* | Negative History | | Positive History | |
| | Years | N | Years | N |
| 0-19dB | 0.3 +/- 1.2 | 50 | 7.7 +/- 10.9 | 75 |
| 20-39dB | 1.5+/- 3.9 | 44 | 6.8+/-10.1 | 72 |
| 40dB or more | 4.7 +/- 9.4 | 48 | 9.1 +/- 11.5 | 167 |

*No notch indicates notch of 0-19 dB in both ears: small notch indicates a notch of

20-39 dB in at least one ear: large notch indicates a notch of 40 dB or more in at least one ear

16. As can be seen in the table, a group of 75 men regarding their hearing loss as attributable to noise, giving a history of 7.7 +/- 10.9 years occupational noise exposure, had audiograms showing no notch in either ear. Equally, 48 men with large notches on their audiogram did not relate their hearing loss to noise and had experienced only 4.7 +/- 9.4 years occupational noise exposure.

17. On the basis of these data the authors conclude that a self-reported history of noise exposure is insufficient evidence on which to base a diagnosis of noise induced hearing loss in people with high frequency sensorineural hearing loss. They recommend that other causes of notching should be considered, and that the history of noise exposure in subjects should be documented by measurement in the workplace and should include reference to recreational and other non-occupational noise exposure.

18. This paper therefore rejects equating the presence of a high frequency audiometric notch with evidence of noise damage. The later Gates' study¹ submitted by RBL as evidence that effects of noise damage on hearing continue long after noise exposure has stopped, fails to take that advice.

19. In deciding whether or not new medical or scientific evidence impacts on war pensions, the standard of proof to be applied is not scientific proof. Rather, it is reasonable doubt. The consideration in the current context is - whether the scientific evidence of the Gates' paper suggesting that the effects of noise damage on hearing may continue long after noise exposure has stopped - constitutes reliable evidence to raise a reasonable doubt. War pensions legislation and case law provide guidance on this consideration.

20. When considering whether there is reliable evidence to raise a reasonable doubt case law has established that **all** the available evidence must be weighed:
"..... the intention of the paragraph [Article 5(4) of the Service Pensions Order 1983] is that it is the duty of the claimant to produce reliable evidence, to establish his claim, but if (after hearing and considering that reliable evidence and making a comparison between such evidence and other evidence which is called on behalf of the Ministry to contradict or controvert it) the Tribunal has a reasonable doubt, then under those circumstances the plain meaning of that paragraph of the Article is that the benefit of that doubt shall be given to the claimant" (Dickinson).

21. Case law also establishes that it is reasonable to conclude that a published work presenting a hypothesis or postulate based on a limited study, which later comes to be the generally accepted view, would not at the time of its publication create a reasonable doubt. It was for the Secretary of State to decide at what later stage, when the hypothesis became sufficiently supported, a reasonable doubt was raised. This was before it became the generally accepted view:

I see no reason why, the Secretary of State should not be entitled to hold that what has now come to the generally accepted view was a mere hypothesis based on a limited study which would not have created a reasonable doubt within the terms of Article 5(4). The stage by which it became sufficiently supported to raise such a doubt in his mind is a matter for the Secretary of State. Accepting, however, that the shift of opinion was a gradual process and that by February 1980 it was the generally accepted view, there must have been an earlier stage when, if asked to consider the matter, he would have found that there was a "reasonable doubt" " (Edwards).

22. The courts have also held that a conflict of medical opinion does not, of itself, mean that a reasonable doubt has been established, and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of *Tigg v The Minister of Pensions* the presiding Judge stated "Merely because a doctor of eminence, and I have no doubt the doctor in this case was of a very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean there is a doubt and the appellant must therefore be entitled to a pension. It is a question of fact for the Tribunal" (cases of *Tigg ROSWPA Vol 5, p141* and *Howard ROSWPA Vol 5, p515*).

23. The proposition that noise induced sensorineural hearing loss damage continues after noise exposure has ceased is not presently generally accepted scientifically as confirmed by the war pensions expert Independent Review of Hearing Loss in 1998 and later in a letter dated January 1999 by Professor Robert Sataloff, Philadelphia. He stated – "The notion that noise induced hearing loss does not progress following removal from noise has been widely accepted for decades."

24. Taking that existing body of evidence into account the submitted evidence was carefully, as described above, examined and evaluated. In particular, the following points are noted:

- its assumption, by assertion only, that the study population was not noise exposed during the follow-up period
- the failure of the paper to reflect the authors' previous conclusions on the same study cohort on the significance of audiometric shape in diagnosis of noise induced hearing loss
- the failure to address the complexities of age related hearing loss, the confounding effects of age and noise and the fact that age related decline in hearing is highly variable even in screened populations with no noise exposure

25. It is therefore concluded that the Gates' paper, *Longitudinal threshold changes in older men with audiometric notches*, *Hearing Research*, 141: 220-228 (2000) does not constitute reliable evidence to raise a reasonable doubt that the effects of noise injury on hearing continue after noise exposure has ceased.

1. Gates et al (2000), *Longitudinal threshold changes in older men with audiometric notches* *Hearing Research* 141: 220-228
 2. NIH Consensus Development Conference, *Noise and Hearing Loss* (1990), *JAMA* 263:2185-2190
 3. Gates, GA et al. (1999), *High frequency audiometric notches in older people*, *J of Occup hearing loss*, 2: 1-10
 4. Taylor, W et al (1964), *Study of noise and hearing in jute weaving*, *J Acoust Soc Am*, 38: 113-120
 5. Rosenhall, U et al (1990), *Presbycusis and noise-induced hearing loss*, *Ear and Hearing*, 11: 257-263
 6. Rosen et al (1962), *Presbycusis: study of a relatively noise free population in the Sudan*. *Ann. Otol. Rhinol. Otolaryngol*, 82:236-253
 7. Pearson, JD, et al (1995), *Gender differences in a longitudinal study of age associated hearing loss*, *J Acoust Soc Am*, 97(2): 1196-1205
- 3 See 3 above

July 2000

Dr E A Braidwood

Medical Adjudication Guidance on Noise Injury and Balance Disorders June 2000

Summary

- Recently there have been a number of claims where it is claimed that noise injury can cause balance disorders/dizziness/vertigo/dysequilibrium. Given the war pensions age group and its high prevalence of dizziness, this is important.
- This view is contrary to war pensions long standing medical understanding.
- Review of the literature and discussion with Dr Linda Luxon, Professor of Audiological Science at University College, London, and an international authority on vestibular disorder has confirmed the war pensions position that there is no relationship between noise injury and balance disorder.
- No current standard UK or US post graduate text on otolaryngology/audiological science supports such an association. There is not a wealth of published peer reviewed scientific evidence. Most studies are old and on animals.
- The most recent and outstanding study (Hinchcliffe, R et al) was published in 1992. This was part of a general consideration by the Inter Society Working Group on Hearing Disability on the group of disabilities arising from noise and other hearing disorders. It concludes that "the balance of evidence is that such vestibular symptoms do not persist after occupational noise injury has ceased".

NOISE INJURY AND BALANCE DISORDERS

1. Recently a number of claims have been received where it is contended that the disablement vertigo/dizziness/dysequilibrium is due to bilateral noise induced sensorineural hearing loss. The matter has therefore been investigated using literature search via Medline and the Internet and discussion with authorities on hearing loss and balance. A list of pertinent peer reviewed published references is below.

2. Standard UK and US post-graduate texts on otolaryngology and occupational hearing loss contain no reference to such an association. In particular, the current sixth edition of Scott-Brown's Otolaryngology published in 1997 contains chapters on the following subjects:-

Noise and the ear
Sensorineural hearing loss
Vertigo
Meniere's disease
Overview of balance
Causes of balance disorder

The authors of these are all international experts. None contains reference to noise injury as a cause of balance disorder.

3. The standard US texts in the area, Sensorineural Hearing Loss and Occupational Hearing Loss, both written by Sataloff and Sataloff of Philadelphia and with latest editions dated 1993, similarly contain no reference. In fact in the chapter on dizziness in Occupational Hearing Loss there is the following:

"In addition to deafness and tinnitus, vertigo is an important symptom associated with disorders of the ear. The intimate relationship of the vestibular portion of the

labyrinth to the cochlea makes it easy to understand the reason why many diseases and lesions, such as Meniere's disease, head trauma, and vascular accidents, affect both balance and hearing. Some diseases, like mumps, classically affect only the cochlea. Certain toxins and viruses affect only the vestibular portion without affecting the hearing. Intense noise affects only the cochlea."

Individual Published Research Papers on hearing loss and balance.

4. Of the papers identified by literature search, it is of note that many were published up to forty years ago. This is not an area which has been subject to much recent peer-reviewed published research.

5. It is also relevant that several papers cited are animal studies involving guinea pigs and pigeons. They are mainly histopathological and all admit to technical difficulties describing their results as "hypothesis" or "preliminary".

6. Before considering in detail the remaining studies, it seems worthwhile to revisit the modern medical approach to causation and in particular to be clear as to the differences between mere observed associations and causal associations.

7. Epidemiological studies on causation frequently make use of complex statistical analyses. In evaluating the studies it must be remembered that publication of a paper in a peer reviewed scientific journal does not guarantee a suitable study design or appropriate use of statistical tests. Epidemiological papers usually quote associations between exposures and disease outcomes. It is critically important to remember that **statistical association** – even when the correct test has been done on appropriate populations – **does not equal causation**.

8. Consider the following observations. Drinking coke and whisky on a Sunday, coke and vodka on a Monday, coke and rum on a Tuesday may well all be followed by headache. There is then an observed association between coke and headache. But is it causal? To separate causal from non-causal observed associations the "standards" set out by Bradford Hill (1965) remain helpful. Exposures associated with disease outcomes are more likely to be causal if:-

- The association is strong
- The association is consistent from study to study
- The association is specific
- The proposed cause precedes the disease, ie temporality
- More of the proposed cause leads to more disease, ie there is a biological gradient
- The association makes sense biologically – plausibility
- The proposed cause is in line with current accepted understanding of the disease
- There is evidence from human experiments
- The association is analogous to previously proven causal association.

9. It should be noted that these standards or criteria are accompanied by reservations and exceptions and for causation they do not all need to be present. The one exception is temporality – causes must always precede effects.

10. Another important issue which must be kept in mind in considering the aetiology of balance disorders is their prevalence in the general population. In a report published in 1961 of two adult rural populations in the UK 17% in the age group 18-24 years gave an affirmative answer to the question, "do you suffer, or have you ever suffered from, dizziness or giddiness?". (Hinchcliffe, R 1961). For the age group 55-64 years the prevalence rose to 35%. A more recent general population survey

involving 17,000 respondents yielded a prevalence of giddiness or dizziness of 61% (Cole, R R A et al 1998).

11. The human studies identified by the literature search generally involved small numbers of subjects. In only one case were there more than 80 subjects. The studies were cross-sectional in design and generally did not provide pre-noise exposure information on either hearing level or objective vestibular function. It is not uncommon for noise induced hearing loss to co-exist with other hearing disorders. In many cases any observed differences between cases and controls were small and not statistically significant. There was no clear relationship of balance dysfunction with severity of hearing loss and in some studies no relationship between noise induced hearing loss and vestibular function was shown. Two studies in asymptomatic patients discussed sub-clinical measures of balance and did not include any pre-study information. The clinical relevance of the findings are unknown.

12. It is important to note that all the studies focussed on individuals who had been, and continued to be, noise exposed. That being so, it is not clear whether the vertiginous symptoms and signs recorded as causally related to occupational deafness were in fact examples of the well-described Tullio phenomenon (where vestibular symptoms may follow acute acoustic trauma).†

13. None of the papers addresses the issue and further they provide no evidence as to whether or not any claimed vestibular symptoms or signs are sustained or become evident following removal from the noise. Previously published evidence on the Tullio phenomenon (Dickson E and Chadwick D, 1951) suggests that the vestibular symptoms do not persist after noise exposure has ceased.

14. In 1992 a review of the evidence for the possibility that exposure to noise may damage the vestibular receptors in the internal ear as well as the cochlea was carried out for the Inter-Society Working Group on Hearing Disability (ISWGHD) (Hinchcliffe, R et al). This was part of a general consideration by the group of disabilities arising from noise and other causes of hearing damage. In conclusion, the authors stated:

- "It would appear that vestibular symptoms may arise from concurrent exposure to high sound levels. As pointed out previously, however, these symptoms are not sustained after removal from the noise source. The mechanism appears to be in the nature of a Tullio phenomenon. The balance of evidence is that such vestibular symptoms do not persist after occupational noise exposure has ceased."

CONCLUSION

15. From the overall evidence we are of the opinion that at this date a reasonable doubt is not raised that noise induced hearing loss is causally related to vestibular damage.

**Dr E A Braidwood
Medical Policy**

June 2000

†Tullio phenomenon. *First described in 1929 in an animal experiment. Vertigo is directly and acutely induced by loud high-pitched sounds. The sounds produce stimulation of the ampullary cristae of the semicircular canals of the inner ear and thus vertigo.*

REFERENCES – HEARING LOSS AND BALANCE

1. Kilburn K H, Warshaw R H & Hanscom B. Are hearing loss and balance dysfunction linked in construction iron workers? *Br J Indust Med* 1992; 49: 138-141
2. Man A, Segal S & Naggan L. Vestibular involvement in acoustic trauma. *J Laryngol Otol* 1980; 94: 1395-1400
3. Manabe Y, Kurokawa T, Saito T & Saito H. Vestibular dysfunction in noise induced hearing loss. *Acta Otolaryngol (Stokh)* 1995; Suppl 519: 262-264
4. Mangabeira-Albernaz P L, Covell W P, & Eldridge D H. Changes in the vestibular labyrinth with intense sound. *The Laryngoscope* 1959; 69: 1478-1493.
5. McCabe B F and Lawrence M. The effects of intense sound on the non-auditory labyrinth, *Acta Otolaryngol* 1958; 49: 147-157.
6. Oosterveld W J, Polman A R & Schoonheit J. Vestibular implications of noise-induced hearing loss. *Br J Audiol* 1982; 16: 227-232.
7. Paperella M M & Mancini F. Trauma and Meniere's syndrome, *Laryngoscope* 1983; 93: 1004-1012.
8. Paperella M M. Pathogenesis of Meniere's disease and Meniere's syndrome. *Ascta Otolaryngol (Stockh)* 1984; Suppl 406: 10-25.
9. Pyykko I, Aalto H & Ylikoski J. Does impulse noise induce vestibular disturbances? *Acta (Stockh)* 1989; Suppl 468: 211-216.
10. Shupak A, Bar-El E, Podoshin L, Spitzer O, Gordon C R & Ben-David J. Vestibular findings associated with noise-induced hearing impairment. *Acta Otolaryng (Stokh)* 1994; 114: 579-585.
11. Sirala U & Lahikainen E. Studies of deafness in shipyard labourers. *Acta Otolaryngol* 1948; Suppl. 67: 107-122.
12. Van Eyck M. Sound produced labyrinthine trauma. *Acta Otolaryng* 1974 100: 464-466
13. Weiss A D & Weiss E R. Acoustic trauma: tinnitus and vertigo *J Laryngol Otol* 1984; Suppl 9: 82-83.
14. Ylikoski. Impulse noise induced damage of the vertibular end organs of the guinea pig. *Acta Otolaryngol (Stockh)* 1987; 103: 415-421.
15. Ylikoski J. Delayed endolymphatic hydrops syndrome after heavy exposure to impulse noise *Am J Otol* 1988; 9: 282-285.
16. Ylikoski J, Juntunen J, Matikainen E, Ylikoski M & Ojala M. Sub-clinical vestibular pathology in patients with noise-induced hearing loss from intense impulse noise. *Acta Otolaryngol (Stockh)* 1988; 105: 558-563.
17. Dieroff, H G, Scholtz. Zur Frage der Larmbedingten Vestibularisschaden bei begutachteten Larmarbeitern. *Zeit-schrift fur Laryngologie, Rhinologie und Otologie* 1967; 46: 746-57
18. Hinchcliffe, R et al. Occupational noise induced vestibular malfunction? *Brit J Ind Med* 1992; 49: 63-65
19. Hinchcliffe, R. Prevalence of the common ear, nose and throat conditions in the

adult rural population Brit J Prev and Social Medicine 1961; 15: 128:40

20. Coles, R R A et al Vestibular disorders in Edwards, F et al. Eds Fitness for Work: the medical aspects, Oxford Medical Publications 1988: 90-100

Version 2

WAR PENSIONS - CLAIMS IN RESPECT OF NOISE INDUCED SENSORINEURAL HEARING LOSS (NISHL) CASES - TRANSITIONAL GUIDANCE

This document comprises guidance on transitional cases arising from the change to the method of assessment of hearing loss announced to members of the Central Advisory Committee (CAC) on War Pensions on 5 December 1996

Definition

Transitional cases are those where an award in respect of NISHL was in payment before 22 October 1996 or where there was a first claim in respect of NISHL, or an application for review of NISHL in respect of deterioration of that condition, before 1 March 1996 but outstanding on 22 October 1996

Contents

[ANNEX A - Summary](#)

[ANNEX B - Processing Transitional Cases](#)

[The law](#)

[Past and current medical understanding - the interaction of service related NISHL and](#)

[non-service related NISHL](#)

[NISHL - history of past claims](#)

[Ministerial commitment](#)

[Categories of claims for revision](#)

[Appeals](#)

[Supplementary Allowances](#)

[ANNEX C - Information on, and worked examples, of :-](#)

[Category A cases](#)

[Category B cases .](#)

Type 1

Type 2

[Category C cases](#)

[Category D cases](#)

Type 1

Type 2

FOMD on evidence submitted on Parkinson's disease mortality¹

1. We note with interest the letter to the Lancet by Drs Page and Tanner sent in response to the Gale study². We do not however consider that it challenges the earlier findings.

2. The study published in the December Lancet was funded by the Department of Social Security and carried out by the independent MRC Environmental Epidemiology Unit in Southampton under the direction of Dr CN Martyn FRCP. Where correspondence in a scientific/medical publication is considered to challenge or contradict findings of an earlier published study, it is customary for the editors to invite the authors of the paper criticised to provide comment. No such invitation was provided in this case

3. The Gale paper was based on the records of over 11,000 former British FEPOWS and as the paper explains, this figure exceeds the target sample size for the study. It was calculated that a figure of 10,000 cases would give an 84% chance of detection of a two-fold difference in risk of Parkinson's disease among former prisoners of the Japanese compared with the general population (two-tailed error 0.05). By contrast there are serious concerns about the power of the American data. The POW (Pacific) population was only about 25% of this size, with total deaths from Parkinson's disease only 9 (7 in POWs and 2 in controls). The confidence interval surrounding the twofold risk rate is very wide (0.51-12.7) and most importantly not statistically significant ($p=0.26$). In other words, as the authors clearly state the findings "... could be due to chance, as the statistical analyses suggest".

4. It is of note that Dr Page has long been associated with the US Medical Follow-Up Agency study of former prisoners of war, a longitudinal study begun in the early 1950s and comparing the medical status of American POWs with non-POW controls. The report of the part of the study begun in 1986 was published in 1992³. Although its principal focus was not mortality the report does contain brief reference to causes and rates of death. It acknowledges, as does the Gale paper, that there is a lack of evidence on explicit cause specific mortality on those ex POWs who died early after captivity. The report does not provide any hint of an excess rate of Parkinson's disease in ex-POWs (Pacific) compared with the general US population. It specifically states:

"... however, at the time of the last completed mortality follow-up in 1975, overall death rates for former POWs did not differ significantly from those of the US male population of comparable age and colour. Moreover excess cause specific mortality could be attributed to three causes: trauma; TB; cirrhosis of the liver. In general most of the differences between POW and general population mortality have lessened over time. Thus there is no striking evidence from earlier mortality follow-up that POWs die sooner than men in the general population."

5. The research questions for the 1992 report include one to detect differences in self-reported illnesses over time and another comparing physician reported physical examinations over time. Both contained information on nervous system disease, and neither self-report nor physical examination suggested an increased prevalence of Parkinson's disease – nor indeed of any other central nervous system problem. Rather the only recorded difference in terms of neurological disease was for nutritional disorders of peripheral nerves. As expected, ex prisoners of the Japanese had a higher rate of peripheral neuropathy.

6. The earlier MFUA (1975)⁴ report specifically noted that while psychiatric impairment was common there was a low rate of organic brain disease in ex-POWs

(Pacific) compared with other groups. In particular attention was drawn to the conspicuous absence of organic brain syndrome. This was previously reported in concentration camp survivors, the enormity of whose physical malnutrition and deprivation must be considered comparable with that of ex prisoners of the Japanese. The findings reported in the Page letter are therefore counter to his own earlier published work and to the bulk of existing published international evidence (including studies from the US, UK, Canada and Australia) none of which records increased rates of Parkinson's disease in ex-POWs (Pacific)^{5 6 7}.

Significance of the further evidence in the context of War Pensions

7. In deciding whether or not new medical or scientific evidence impacts on war pensions, the standard of proof to be applied is not scientific proof. Rather, it is reasonable doubt. War pensions legislation and case law provide guidance on this consideration.

8. When considering whether there is reliable evidence to raise a reasonable doubt case law has established that **all** the available evidence must be weighed:

"..... the intention of the paragraph [Article 5(4) of the Service Pensions Order 1983] is that it is the duty of the claimant to produce reliable evidence, to establish his claim, but if (after hearing and considering that reliable evidence and making a comparison between such evidence and other evidence which is called on behalf of the Ministry to contradict or controvert it) the Tribunal has a reasonable doubt, then under those circumstances the plain meaning of that paragraph of the Article is that the benefit of that doubt shall be given to the claimant" (Dickinson).

9. Case law also establishes that it is reasonable to conclude that a published work presenting a hypothesis based on a limited study, which later comes to be the generally accepted view, would not at the time of its publication create a reasonable doubt. It was for the Secretary of State to decide at what later stage, when the hypothesis became sufficiently supported, a reasonable doubt was raised. This was before it became the generally accepted view:

"I see no reason why, the Secretary of State should not be entitled to hold that what has now come to the generally accepted view was a mere hypothesis based on a limited study which would not have created a reasonable doubt within the terms of Article 5(4). The stage by which it became sufficiently supported to raise such a doubt in his mind is a matter for the Secretary of State. Accepting, however, that the shift of opinion was a gradual process and that by February 1980 it was the generally accepted view, there must have been an earlier stage when, if asked to consider the matter, he would have found that there was a "reasonable doubt" " (Edwards).

10. The courts have further held that a conflict of medical opinion does not, of itself, mean that a reasonable doubt has been established, and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of *Tigg v The Minister of Pensions* the presiding Judge stated "Merely because a doctor of eminence, and I have no doubt the doctor in this case was of a very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean there is a doubt and the appellant must therefore be entitled to a pension. It is a question of fact for the Tribunal" (cases of *Tigg ROSWPA Vol 5, p141* and *Howard ROSWPA Vol 5, p515*).

11. The Page and Tanner data is published only as a letter, not as a formal hypothesis or report setting out details of methods, results, discussion etc. Further, its general tone "our findings of an apparent two-fold increase ..." is not definitive or authoritative. It freely admits to the likely role of chance and is unable to explain why

its findings differ from those of Gale et al. It is not presented, nor was it interpreted by the editor of the Lancet, as a challenge to the Gale paper.

12. In conclusion it remains the opinion of the Agency that, in the terms described above, the Page and Tanner letter does not annul the findings of the Gale study. At present, there is no reliable evidence to raise a

reasonable doubt that the experience of Japanese captivity generally increased the prevalence or mortality risk from Parkinson's disease as compared with that of general UK male population. That is a general finding. War pensions is however an individual jurisdiction. For reasons previously discussed in the Opinion dated, the Agency view also remains that in this case a reasonable doubt is not raised that service caused or influenced the course of the claimed Parkinson's disease.

July 2000

Dr E A Braidwood

- 1 Page, WF et al (2000) Parkinson's disease and Motor Neurone Disease in former POW (letter) Lancet, 355: 843
- 2 Gale, C et al (1999) Mortality from Parkinson's disease and other causes in men who were prisoners of war in the Far East, Lancet 354: 2116-2118
- 3 Page, WF (1992) The Health of Former POW. Washington DC National Academy Press
- 4 Beebe, GW. Follow-up studies of World War II and Korean war prisoners: II, morbidity, disability and maladjustments. Am J Epidemiol 1975: 101: 400-22
- 5 Creasey, H et al. Is experience as a prisoner of war a risk factor for accelerated age-related illness and disability? J Am Geriat Statement of Case 1999: 47:60-64
- 6 Gill GV, Bell, DR. The health of former prisoners of war of the Japanese. Pract Med 1981; 225-531-38
- 7 Guest, CS, Venn, AJ. Mortality of former prisoners of war and other Australian veterans. Med J Aust 1992; 157: 132-135

EX-FAR EASTERN PRISONERS OF WAR (FEPOWS) AND RISK OF PARKINSON'S DISEASE: SUMMARY OF EVIDENCE IN THE CONTEXT OF WAR PENSIONS SCHEME JAN 2001

The Issue

It is contended that ex-FEPOWs are at increased risk of Parkinson's disease¹ and the condition should be accepted as attributable to their service.

Summary

- The prevalence of neurodegenerative conditions including Parkinson's disease (the most common) has been increasing in the UK and other Western countries over recent years in line with population ageing. Literature search and discussion with experts confirms that the aetiology of most cases of Parkinson's disease is unknown.
- It is contended that ex-FEPOWs are at increased risk of Parkinson's disease and based on the work of Professor Peter Spencer, a neuro epidemiologist from Oregon, that this is due to the ingestion of cycad neurotoxins whilst in captivity.
- This suggestion is not confirmed by other researchers and indeed there has been no published work on cycad as a possible cause of neurological disease since 1991.
- An epidemiological study by Dr Christopher Martyn of the Medical Research Council Environmental Epidemiology Unit, published in the Lancet in December 1999 compared mortality of over 10,700 ex-FEPOWs with that of the general population. It concluded that the mortality of FEPOWs both from all causes, and from Parkinson's disease, was lower than in the national male population.
- This study thus provides no evidence that rates of Parkinson's disease in ex-FEPOWs differ from those in the population generally.
- The contended cycad link remains an hypothesis, without supporting reliable evidence sufficient to raise a reasonable doubt that the condition is due to the FEPOW experience.

The War Pensions Context

(a) How the Scheme works

1. A war pension may be claimed for any disablement by anyone who has served in the armed forces. Claims may be made at any time from service release. Decisions are evidence based and considered individually on the specific facts of the case. Awards are made where, within the relevant law, the evidence including service and medical facts and the contemporary medical understanding of the condition claimed, shows a causal link between service and the claimed condition, on the basis of the relevant burden of proof as referred to in paragraphs 4 and 5 below.

2. For claims made within 7 years of leaving the armed forces, Article 4 of the Service Pensions Order provides that the onus is on the Secretary of State to show beyond a reasonable doubt that the claimed condition is not attributable to or aggravated by, service. If he cannot show this, an award of War Disablement Pension may be made.

3. For claims made more than 7 years after the end of service, Article 5 of the Service Pensions Order puts the onus on the claimant to raise, by way of reliable evidence, a reasonable doubt that the claimed condition is attributable to, or

continues to be aggravated by, a service injury.

(b) Case Law

4. The High Court has held that the word "reliable", in the context of Article 5 cannot have been intended to mean "convincing", but means more than "fanciful". It held that, with particular reference to "changes of medical opinion" that "there are ... in my judgement, three stages: no reasonable doubt, reasonable doubt, and consensus."

5. A war pensions claim under Article 5 would pass the test at the point where the (reliable) evidence raised a reasonable doubt, but "a mere hypothesis based on a limited study ... would not have created a "reasonable doubt" within the terms of Article 5(4)." The real question, however, it held, is whether the evidence raises a reasonable doubt in the mind of the Secretary of State (SoS). If he finds the evidence unreliable it obviously will not raise a reasonable doubt in his mind. (Case of Edwards 1992 HCJ no Colin Pike/2281/90).

6. The Courts have also held that a conflict of medical opinion does not, of itself, mean that a reasonable doubt has been established, and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of Tigg v the Minister of Pensions the presiding Judge stated "Merely because a doctor of eminence, and I have no doubt that the doctor in this case was of very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean there is a doubt and the Appellant must therefore be entitled to a pension. It is a question of fact for the Tribunal." (Cases of Tigg ROSWPA vol 5 p 141 and Howard ROSWPA vol 5 p 515).

(c) Reliable evidence

7. In considering under Article 5 whether there is reliable evidence to raise a reasonable doubt on whether Parkinson's disease in an ex-FEPOW is attributable in any way to service, the Department has taken into account relevant case law and had regard to factors including:
the weight of evidence on both sides
the degree of support from others in that field of medicine
whether opinions are hypotheses: or whether there is support for them; and the basis of that support.

Assessment of evidence

8. New evidence must always be considered and weighed relative to the existing body of evidence on a subject. Account must be taken of the robustness and authority of new studies in particular their design and methodology, the sample size and case selection, their statistical validity, and the study's approach to bias. Other important factors include the consistency of findings with existing evidence, and the response to it of other established experts.

9. The following paragraphs set out the detailed analysis made by the Department on the medical evidence relating to Parkinson's disease, and its relationship to the FEPOW experience.

Recent medical studies

10. There has been extensive international research into the health effects of

Japanese captivity. In the aftermath of the war numerous studies were carried out in the UK, US and Australia on the health of ex-FEPOWs. It was shown that some conditions such as TB, liver cirrhosis, peripheral neuropathy, some psychological disorders and suicide were more common in FEPOWs in the period following repatriation. Equally, no increase in incidence or prevalence of a wide spectrum of diseases including cardiovascular and CNS diseases was recorded. In general, the published evidence is that if an individual survived the experience of Japanese captivity for 10-15 years after release, health and mortality thereafter is no different from that of non-FEPOWs of the same age and gender.

11. Since the early 1980s and the publication of a paper in the Lancet² it has been contended that FEPOWs suffer more neurodegenerative conditions, ie Alzheimer's, motor neurone disease and the most common in our society, Parkinson's disease, than the comparable non-FEPOW population. More recently it was proposed that the particular link was toxins ingested in food or absorbed in the skin when contained in balms or poultices.

12. In support of this idea, the work of Professor Spencer, on Western Pacific Disease, was cited. This condition – a mixture of motor neurone disease/dementia and Parkinson's disease was until recently very common in Guam. For many years it has been suggested that it might be due to ingestion of cycad neurotoxins in flour. Professor Spencer published on the matter in 1990/91 and it later became the subject of a BBC Horizon programme.

13. Even at the time of the programme the hypothesis was not new and had been investigated over a 30-40 year period. Since the Horizon programme, Professor Spencer's ideas on cycad, although subject to further scrutiny, have not been confirmed by other researchers and indeed there has been no new published work supporting cycad as a neurotoxin since 1991.

14. Existing published evidence on the causes of Parkinson's disease was critically appraised by the Department during 1993-1994 and discussion with leading UK experts also took place. This confirmed the Department's view that in most cases the aetiology of Parkinson's disease in ex-FEPOWs was unknown and that even in the context of the War Pensions Scheme, the proposed causal link with cycad was properly described as hypothesis. Supporting reliable evidence sufficient to raise a reasonable doubt that the condition was due to FEPOW experience did not exist.

15. There are other difficulties about the cycad hypothesis in relation to FEPOWs. There is no single cycad palm but rather 9 different genera. *Cycas circinalis* is native to Guam and it is this plant which was proposed as causally linked to Western Pacific Disease. It is not found in any area where there were Japanese camps. FEPOWs might have been exposed to *cycas revoluta* in the camps of the South Kii peninsula of Japan. A single case report linked exposure to this substance in childhood with later motor neurone disease. *Cycad revoluta* also grows in Indonesia but this plant is not edible, being bitter and a powerful emetic. On Kii it was used in traditional Japanese medicine and in Indonesia as a balm for wounds. Given the privations of FEPOW captivity, it is difficult to conceive that such balms or complicated Japanese herbal remedies would have been widely available.

16. Undoubtedly, FEPOWs did eat all manner of vegetable products but whilst there is good evidence of certain plant substances producing acute neurological sequelae in animals and man (chickling pea, cassava and *Chondria armata* – a seaweed) within a short time of ingestion, there is no evidence of such substances causing neurological signs or symptoms with onset only many years after exposure. A recent review of the epidemiology of Parkinson's disease makes no reference to a possible causal role for cycad or any other neurotoxin. (Ben Shlomo 1998)³.

17. The prevalence of neurodegenerative conditions including Parkinson's disease has been increasing in the UK population over recent years in line with the ageing of the population but the precise reasons remain unknown. Recent reviews, articles and current (2000) edition of the Concise Oxford Textbook of Medicine and the 21st editions of Cecil, both hold that Parkinson's disease is a condition of unknown aetiology. All cite possible environmental, and, of increasing research interest, genetic influences.

18. In a 1991 article Calne⁴ and colleagues suggested that Parkinson's disease, as well as other degenerative diseases that arise in old age, could arise from an environmental insult to the nervous system which remains subclinical for several decades and becomes expressed clinically as a consequence of age-related loss of neurones. They did not, however, present empirical evidence to support the hypothesis.

19. In youth, FEPOWs endured a period of severe deprivation and as a result the last Government decided, in 1995, to commission research into the matter. Because of the age group involved it was considered important that any project should produce a reasonably quick outcome without inconvenience to the FEPOWs. Toxicological research is complex, lengthy and expensive and because the initial hypothesis had not been confirmed not even further supported, a toxicological approach was not pursued.

20. As the relevant war pensions legislation requires only reliable evidence to raise a reasonable doubt that a condition may be due to service (ie, it is not necessary to pinpoint a specific service factor), it was decided to conduct an epidemiological study. Were such a study to show an increased prevalence of Parkinson's disease in FEPOWs relative to the general non-FEPOW population, that would be sufficient evidence on which to make awards of war pension. A mortality study was appropriate as it could be documentary using reliable data sources and without inconvenience to FEPOWs.

21. Dr Christopher Martyn, of the Medical Research Council Environmental Epidemiology Unit, who is both a neurologist and an epidemiologist, was chosen as the principle researcher. The study compared mortality of FEPOWs from all causes and from Parkinson's disease, with the general population based on data from the WPA list of former FEPOWs. Members of the Japanese assets cohort were traced through the NHS Central Register, Southport and mortality of the group from all causes and from Parkinson's disease compared with that of the national male population of England and Wales by the person-years method.

Summary of Findings

22. The study was published in the Lancet in December 1995. It considers death from Parkinson's disease during 44 years of follow-up of 10,731 servicemen whose records were traced and who had been FEPOWs (90% of the cohort).

23. Thirty-two ex-FEPOWs were recorded as dying from Parkinson's disease during the period 1952-1995: this number should be compared with an expected number of deaths of 37.1, giving an age-standardised mortality ratio (SMR) of 86.

24. The study also showed that the mortality of ex-FEPOWs from all causes during the period, 1952-1995, was lower than expected. A total of 6,658 FEPOW deaths were observed during the period, to be compared with an expected number of 7,950 giving an SMR of 84. Note: "Our Healthier Nation" describes standard mortality ratios of 80-90 as "well below average". Deaths from Parkinson's disease and from all

causes amongst ex-FEPOWs was therefore lower than in the national male population.

25. The study therefore provided no evidence that ex-FEPOWs are at increased risk of Parkinson's disease.

Review of other evidence published since 1995

26. Since Dr Martyn's study began in 1995, the medical and toxicological literature have been kept under scrutiny and it has been confirmed that little new consistent aetiological evidence on Parkinson's disease has been published in that period. None has provided empirical support for the role of an early environmental insult in development of the condition. The cycad – or perhaps more accurately the broader vegetable neurotoxin hypothesis – remains just that. (It is worth noting that in the published papers suggesting a role for cycad or other neurotoxins even the strongest protagonists refer to the concept in those terms). The role of genetics in early onset Parkinson's disease is well established and research on the field is currently focussed on genetic aspects. It is of note that in December 2000 a paper from Iceland documented that late onset Parkinson's disease also has a genetic component.⁶

27. Scrutiny was extended to the not inconsiderable body of early evidence (1946-1979) – from the USA, Australia, Canada and the UK – on repatriated PoWs. After the war a number of longitudinal studies were set up to compare the health of PoWs (Europe) with those of PoWs (Japan) and the health of both against non-captive population. That project still continues in the USA.

28. The latest 1992 report of the US Medical Follow-up Agency (Institute of Medicine) entitled "The Health of Former Prisoners of War" commented amongst other things whether there is any different pattern of illness in those PoWs – both European and Japanese – who were seriously malnourished at repatriation. There was no evidence of increased prevalence of organic brain disease in general nor Parkinson's disease.

29. In March 2000 a letter appeared in the Lancet in response to the Gale study⁷. The letter is interesting but, for the reasons discussed in the [FOMD](#) on evidence submitted on Parkinson's disease mortality, we do not consider it challenges the findings of the Gale paper. The Gale paper is noteworthy because of the size of the study group. It is considerably larger than any other study of ex-FEPOWs, enhancing the statistical robustness of its results.

30. In conclusion, on present evidence:

- the cause of most cases of Parkinson's disease, including in ex-FEPOWs remains unknown.
- Parkinson's disease seems likely to arise from a causal combination of aetiological factors with genetic and environmental factors involved
- evidence to date, including the Gale paper, does not suggest a higher prevalence of Parkinson's disease compared with the national UK population.
- in the context of War Pensions, reliable evidence does not at this date raise a reasonable doubt that cycad or other neurotoxin has a role in Parkinson's disease in ex-FEPOWs.

31. As in all scientific and medical matters the Department is ready at any time to consider any new evidence of appropriate quality.

Footnotes

1 Parkinson's disease

This disease is a chronic disease of the central nervous system, characterised pathologically by fine, slowly spreading tremor, muscular weakness and rigidity, slow and measured speech, and a peculiar gait.

The cause is unknown

- The disease affects men and women of all races
- The age of onset is rarely less than 50 years
- Thereafter the incidence of the disease increases exponentially with age
- Recovery rarely if ever occurs. The duration is indefinite.

2 Gibberd, FB, and Simmonds, JP (1980) Neurological disease in ex-Far Eastern Prisoners of War, *Lancet*, 1: 135-137

3 Ben Schlomo, Y (1998) Parkinson's disease in Martyn, C and Hughes, RAC (eds). *The Epidemiology of Neurological Disorders*, London, BMJ Books

4 Calne et al, Rinne et al (1991) Parkinson's disease International Workshop Berlin.

5 Gale, CR, Braidwood, EA, Winter, PD and Martyn, CN. Mortality from Parkinson's disease and other causes in men who were prisoners of war in the Far East, *The Lancet*, Vol 354, December 18/25, 1999.

6 Sveinbjornsdottir, et al (2000), Familial aggregation of Parkinson's disease in Iceland. *N Engl J Med* 343: 1765-1770

7 Page, WF, and Tanner, CM, (2000) Parkinson's disease and motor-neuron disease in former prisoners-of-war (letter), *Lancet*, 355: 843

Medical Adjudication Guidance on Schizophrenia

1 For many years medical opinion was that schizophrenia was constitutional and essentially independent of external circumstances.

2 Statistics from the 1914 and 1939 wars showed a similar rate of the condition in people subject to battle stress and the general civilian population. In War Pensions terms, this was strong evidence that military stress played no significant part in the disease. The condition was not therefore certified “attributable” to service. If there was evidence of **severe stress** in close time relation to clinical onset of the condition, entitlement of “agg” was considered.

3 In the 1980s as a result of a review of the medical appendices it was recognised that reliable evidence now existed to raise a reasonable doubt that environmental factors play a more critical role and that, in a predisposed individual, the condition could be precipitated by a much lesser degree of stress. This meant that claims for schizophrenia, having onset in service, could be accepted as attributable to service. The date of change of medical opinion was identified as 1 February 1980. In 1992, the [Edwards judgement](#) resulted in revision of that date to 1 February 1976. The nominated judge was of the view that the quality and quantity of evidence on that date was beyond speculation but sufficient to raise a “reasonable doubt”.

4 Schizophrenia and the other psychoses remain active areas of clinical and research interest and in particular aetiology is still being studied. Schizophrenia presents a spectrum from severe chronic deteriorating illness to shorter psychotic episodes which are treatable with little residual deficit. Family and twin studies suggest a polygenetic inheritance. It is now postulated that the genetic abnormalities arise in genes influencing communication ability, in particular in relation to abstract thought. There is accumulating evidence that this arises due to failure of cell migration in the embryo, possibly due to viral insult. New techniques investigating brain structure confirm that in severe schizophrenia there is neuro-anatomical change with changes unlike those seen in the neurodegenerative disorders.

5 The recently revised [appendix on schizophrenia](#) emphasises heredity and genetics in schizophrenia. The view, increasingly shared by most psychiatrists, is that schizophrenia in its severe form is primarily a neurobiological disorder. External stressors, are significant in the timing of onset and in relapses in those with more severe variants of the condition. In a recent [Social Security Commissioner Hearing C.DLA/8353/95 \(Sylvia Phillips\)](#), on severe mental impairment in DLA, an independent medical expert wrote - “On the basis of these findings I conclude that there is evidence which is growing both in strength and in volume to suggest that at least persons with severe schizophrenic illness with chronic deteriorating course are most likely to have suffered neurodevelopmental damage which has prevented the proper organisation of cells in the meso-limbic system and possibly in the cerebral cortex.

6 “Social stress of its own accord is probably significant in the timing of schizophrenia but is only likely to have a major contribution to cause less serious and more recoverable form of the disorder”.

Medical adjudication guidance on **schizophrenia continued**

[back to Medical Adjudication Contents](#)

Claims For Psychiatric Disablement Where “bullying” In Service etc Are Contended

Claims for psychiatric disablement where “bullying” in service, racial/sexual harassment/or abuse: off-duty assault: initiation ceremonies are cited are increasingly being seen.

There is firstly the requirement as in all war pension claims for Article 3 to be applied. The onus is on the claimant to show to the satisfaction of the Secretary of State on balance of probabilities the presence of disablement. (Guidance on the SoS “normal” policy in this respect is currently being prepared by WII).

Once disablement has been shown, the onus passes to the SoS to consider the “material facts” upon which the claim is based. Particularly where PTSD is in the differential diagnosis and the stressor event is part of the diagnosis, the SoS’s acceptance of the service event may be critical for the outcome. An administrative policy statement on bullying etc is shortly to be issued by WII. This will enable the SoS approach to acceptance or rejection of events to be legal, well informed, consistent and equitable. As far as possible, claims will be submitted to MAs with the SoS decision on the material facts on file. Where consultant referral is appropriate, the view of the SoS on the contended material fact should form part of the terms of reference.

There is as yet no case law on bullying etc. Present consideration has therefore been informed by both Departmental and counsel’s legal advice. Broadly, in deciding an appropriate way forward in bullying cases there is a need to keep in mind the core intention of the War Pensions Scheme ie to provide compensation for those suffering “disablement due to injury **attributable** to service”.

The SPO does not define “attributable to” or “aggravated by” service. These terms are therefore for the SoS to interpret, taking account of relevant case law. Case law supports the view that, regardless of Article, for certification, it is not enough for a condition to arise during service. A key distinction lies between incidents and events occurring within the individual’s personal sphere and those connected with duty or service compulsions. Claims will succeed where disablement results from injury attributable to a compulsion of service or in pursuance of the service’s legitimate objectives.

Where the presence of disablement has been shown and the material facts established many claims where bullying, personal harassment etc is an issue will be for rejection NANA. This is because the disablement results from actions in the personal sphere with service providing a setting not the cause. (Broadly, bullying/sexual, racial harassment or abuse/assaults, initiation ceremonies are within the personal sphere and not compulsions of service. The correct course to reparation is the criminal law not the War Pensions Scheme).

[back to Medical Adjudication Contents](#)

Medical adjudication guidance where smoking is an issue

1 In relation to the recent media publicity, it is wrong to say there has been a change of policy regarding claims for War Pension in respect of medical conditions caused solely by smoking. It has never been the policy of any government to award/allow a War Pension in such circumstances.

2 It is also completely inaccurate to say that the widows of men who were Far Eastern Prisoners of War are to have their War Widows Pensions cut.

3 Over more than 40 years, it has been the general view of successive governments that the War Pensions scheme should not provide compensation for, or a War Widows Pension, in respect of the consequences of personal habits, such as smoking or drinking.

4 The law does, however, provide for awards in respect of smoking-related conditions where there is a severely disabling mental condition (itself attributable to service and assessed at a minimum of 50 per cent) which renders the individual incapable of exercising personal choice, and prevented him giving up the habit.

The effect of the High Court judgement in the [Hunt case](#) (December 1993)

5 The High Court judgement in the case of Mrs Hunt (her late husband was alleged to have taken up smoking to cope with the pain arising from the war pensioned disablement to his feet) was made on the facts of the individual case and should not be regarded as a precedent.

6 Nevertheless, it established that the law as it stood could be interpreted to permit the payment of a War Pension when a person who smokes is disabled or dies from a disease caused by that habit even though there is no severely disabling mental condition. that is why it was considered necessary to clarify the law to ensure that the relevant legislation fully reflected the policy intention. No one lost any money as a result.

7 An exception to the general rule has been made for individuals who are incapable of exercising personal choice in deciding whether to smoke or drink through some service factor. This applies where the claimant is suffering from a mental condition which is attributable to service, is assessed at 50 per cent or more and which causes the individual to smoke or drink or prevents him from giving up the habit.

8 The [May 1996 Training Minute, Smoking Issues](#), gives guidance on answering medical contentions commonly cited at appeal in relation to smoking. These include smoking as a relief for pain and stress. The recent media publicity focuses on the **availability of cigarettes: the special circumstance of FEPOW captivity and the futility of giving up smoking**. The remainder of this minute gives some guidance on handling these issues.

[back to Medical Adjudication Contents](#)

Overview of the evidence on environmental tobacco smoke (ETS) and adult health

1. Smoking cigarettes is associated with increased risk of a range of diseases including various cancers, chronic bronchitis, emphysema and cardiovascular disease. It is therefore inherently plausible that inhalation of ETS would have similar effects.
2. In 1981 a study from Japan (1) and another from Greece (2) reported an increase of lung cancer in wives of heavy smokers. In 1986 the US Surgeon-General published a report (3) on the Health Consequences of Involuntary Smoking. This recorded 13 spouse studies looking at passive smoking and lung cancer, of which 11 were reported as positive.
3. By 1993 the US Environmental Protection Agency (EPA) had published a monograph (4) including 30 epidemiological studies on passive smoking and lung cancer. Twenty-four of these were said to be positive. In addition evidence was presented of links in children with lower respiratory tract infection, middle ear disease and asthma.
4. The 1997 California EPA report (5) further documented a relation between passive smoking and ischaemic heart disease, nasal sinus cancer and in children asthma, sudden infant death syndrome and low birth weight. Similar conclusions were reached by the 1998 UK Scientific Committee on Tobacco and Health (6) (SCOTH).
5. Around the same time UK meta-analyses exploring the link between ETS and lung cancer (7) and ischaemic heart disease (8) were published. A risk of 24% for lung cancer and of 30% for heart disease was reported for those who had never smoked but who lived with smokers.
6. This confirms some conflict. Despite an accumulating body of evidence that there may be a causal association between passive smoking and adverse health effects, some work does not support this view. In addition, over the period there have been dissenting voices (9) (10) (11) expressing serious doubts about a link between ETS and adult health problems.
7. A new paper in May 2003 (12) extended the debate. Although still subject in its methodology to some limitations, the size and study design are important. It was a prospective cohort study following 118,094 adults from late 1959 to 1998. The particular focus was 35,561 never smokers whose spouses smoked. Outcome measures included the relative risk of death from coronary disease. For never smokers married to ever smokers compared with never smokers married to never smokers, the relative (age adjusted) risk for coronary heart disease was 0.94 (0.85-1.05). This does not show a significant

association between passive smoking and death from coronary disease.

8. Closer examination of the individual studies highlights difficulties and limitations in their design, execution and the interpretation of their results.

The individual studies

Power

9. It is axiomatic that studies should be designed so that they are large enough to detect the effect/outcome of interest. Compared with smoking 20 cigarettes a day, the effect of ETS is likely to be very small with parallel small influence on health. That means statistically robust studies need to be large. In fact most of the passive smoking literature involves studies with 100 cases or less (13). In many studies with estimated relative risk of greater than one, the 95% confidence limits include one, ie the reported effect could well be due simply to chance. Evidence of note in this regard is the 1998 International Agency for Research on Cancer (IARC) multicentre case control study (14) which had 650 cases of lung cancer in non-smokers. The relative risk for spousal passive smoking in this very large study was 1.16, with 95% confidence intervals (0.93 – 1.44), ie the observed increase in risk could be a chance finding.

Bias

10. Most of the studies looking at passive smoking and disease are non random case control or prospective in design. These may be the subject of several different types of bias.
 - There may be errors in **determining the outcome** of interest eg cancer studies purporting to consider lung cancer where diagnosis is not based on biopsy nor is there adequate differentiation of primary or secondary tumours. With such serious illnesses or death, another element of uncertainty is introduced because many studies use surrogate reports from patients' relatives. It is recognised that about 15% positive diagnoses of lung cancer made by a doctor are false (15). Errors may lie in either direction producing either over or underestimate of risk. The situation is even less satisfactory for heart disease where self-report of angina etc surrogate animal models or sub-clinical end points were often used (16) and taken as evidence of ischaemic heart disease.
 - Perhaps the biggest single difficulty in passive smoking studies is error in **estimating the ETS exposure**. For anyone over the age of 40 years there is bound to have been some exposure to ETS. Both ischaemic heart disease and lung cancer, the two largest alleged

population effects have long incubation periods, so, ideally, to study causation for these conditions we need regular ETS dosage measurements over many years. In practice most studies use single qualitative measures obtained by self-report with no attempt to estimate dose.

- The studies usually consider either spouse smoking or smoking at work. Few studies enquire, far less measure, the multiple exposures an individual may have from childhood onward eg home – (parents) - home (spouse) – transport – work – and we know of no study which takes into account room size, ventilation or time since exposure etc. Spouse studies assume the spouse spends time at home and smokes at home. Self-report is inherently unreliable. Cultural issues may also be relevant. A number of the passive smoking studies come from Japan where it has traditionally been considered unwomanly for a woman to smoke. There is therefore high risk that smoking will be denied. All of this leads to a risk that smokers will contaminate the non-smoker group.
11. **A more objective measure of ETS is level of cotinine in urine, serum or saliva** and this is used in a number of studies. However, this is still subject to numerous errors including, in its analysis, the fact that it is only one nicotine metabolite and is only relevant to recent passive smoking. Numerous studies of lifetime non-smokers alleging non-exposure to ETS still record measurable cotinine levels (17). Pearson co-efficients for correlation between reported passive smoking exposure and urinary cotinine levels range between only 0.2 and 0.5, ie the levels are not highly correlated. One important often quoted prospective study reporting a positive relation between passive smoking and adverse health looked at 32,000 non-smoking women (with no cancer, stroke, cardiovascular disease at base-line) over 10 years. It however assessed passive smoking by self-report and only once at base-line (18).
 12. Both lung cancer and particularly ischaemic heart disease are multi-factorial. Over 300 alleged risk factors have been identified for ischaemic heart disease. There is therefore potential for **confounding** by variables which themselves correlate both with ETS exposure and the outcome measure. Many studies take no account of the risk factors and in those which do, researchers typically only present 2 relative risks, one totally unadjusted and the other adjusted for a whole string of variables – some having individual very large effects.
 13. As ever, in weighing published evidence on causation there is the issue of **publication bias**. It needs to be remembered that studies showing positive outcomes are likely to be published. There may be many unpublished studies taking the opposite position. Authors rarely take account of this, eg by discussion with research groups known to have an interest to establish the full evidence.

Meta-analysis

14. Because of the relatively small size of the individual passive smoking studies, the procedure of meta-analysis ie the combination of the results from multiple studies, has often been applied to the data. Individually, most of these studies have reported a statistically non-significant increased risk of lung cancer or ischaemic heart disease, a few results have reached statistical significance and some have shown a negative correlation. The subsequent application of meta-analysis is claimed to reveal evidence of statistically significant increased risk both for lung cancer and ischaemic heart disease.
15. Meta-analysis was developed to analyse combined multi-centre drug efficacy studies which used the same study design and protocol ie with resulting highly homogeneous data. As discussed above passive smoking studies on lung cancer and ischaemic heart disease are characterised by wide differences in design, quality and size. It is scientifically inappropriate to apply meta-analysis to such disparate data.
16. In an attempt to overcome this heterogeneity, some analyses apply quality scores to the individual studies but this often results in poor studies or large studies being weighted too heavily and their having potentially disproportionate effect on outcome. Some meta-analyses pay little attention to the quality of data analysed but rather proceed as if the studies were perfect.
17. Ideally aggregated studies should consider all the available data. In the event, in most, few databases have been considered. Publication bias is again important. In general, meta-analyses and systematic reviews do not adequately ascertain/address whether or not non-published work is broadly in line or different from the published studies.
18. Perhaps surprisingly, another issue with respect to the claimed passive smoking effects is the remarkable similarity of the findings. In 18 studies of very different design, size, population looking at passive smoking and coronary heart disease, the range of relative risks was only 1.0 – 2.2 despite the difference in populations, account taken/not taken of different sources of ETS, and no information on “dose”. In this type of situation consistency in results may well relate to the same bias rather than a real effect. Research and medical interest in the topic remains high but recent review papers continue to have the same reservations (19-22).

Conclusion

19. For these reasons at this date it is concluded that contemporary evidence overall does not adequately tell us whether passive smoking increases the risk of coronary heart disease or lung cancer. The

evidence is not consistent and its reliability is in question. In the context of war pensions standards of proof it is considered that a reasonable doubt is not raised.

References

- 1 Hirayama, T. Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan, *Br Med J* 282, 183, 1981
- 2 Trichopoulos, D et al. Lung cancer and passive smoking. *Int J Cancer*, 27, 1, 1981
- 3 US Surgeon General. The Health Consequences of Involuntary Smoking, a Report of the Surgeon General, DHHS (CDC) 87-8398, Rockville, MD 1986, 359
- 4 Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders, NIH Publication No 93-3605, 1993
- 5 Environmental Tobacco Smoke, California Environmental Protection Agency, Sacramento, 1997
- 6 Tobacco and Health, Her Majesty's Stationery Office, London, 1998
- 7 Hackshaw, AK et al. The accumulated evidence on lung cancer and environmental tobacco smoke, *Br Med J* 315, 980, 1997
- 8 Law, MR et al. Environmental tobacco smoke exposure and ischaemic heart disease: an evaluation of the evidence. *Br Med J*, 315, 973, 1997
9. Armitage, AK et al. Environmental tobacco smoke – is it really a carcinogen? *Med Sci Res*, 25, 3, 1997
10. Gori, GB. Science, policy and ethics: the case of environmental tobacco smoke, *J Clin Epidemiol*, 47, 325, 1994
11. Hood, RD, et al. Environmental tobacco smoke exposure and respiratory health in children: an updated critical review and analysis of the epidemiological literature, *Indoor Environ*, 1, 19, 1992
12. Eustram, J et al. Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians 1960-98 *Brit Med J*, 326: 1057, 2003
13. Gillis, C et al. The effect of environmental tobacco smoke in two urban communities in the west of Scotland. *Eur J Resp Dis.* :65 (supp 133): 121, 1984
14. Kreutzer, M et al. Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in Europe. *J Natl*

Cancer Inst. 90, 1440, 1998

15. Lee, PN. Comparison of autopsy, clinical and death certificate diagnosis with particular reference to lung cancer. A review of the published data. *APMIS*, 102(Suppl 45), 1, 1994
16. Glantz, SA et al. Passive smoking and heart disease, Mechanisms and risk. *J Am Med Assoc*, 273, 1047, 1995
17. Tunstall-Pedoe, H et al. Passive smoking by self report and serum cotinine and the prevalence of respiratory and coronary heart disease in the Scottish heart health study. *J Epidemiol Community Health*, 49, 139, 1995
18. Kawachi, MD et al. A prospective study of passive smoking and coronary heart disease., *Circulation*, 95: 2374, 1997
19. Trobs, M et al. Nutrition, antioxidants, and risk factor profile of nonsmokers, passive smokers and smokers of the Prevention Education Program (PEP) in Nuremberg, Germany, *Prev Med Jun*; 34(6): 600, 2002
20. de Groh, M et al. Environmental tobacco smoke and deaths from coronary heart disease in Canada, *Chronic Dis Can*, 23(1):13-6, 2002
21. Jaakkola, MS. Environmental tobacco smoke and health in the elderly, *Eur Respir J Jan*; 19(1): 172, 2002
22. Rosenlund, M et al. Environmental tobacco smoke and myocardial infarction among never-smokers in the Stockholm Heart Epidemiology Program (SHEEP), *Epidem Sep*; 12(5): 558, 2001

28 November 2003

The association between head injury and Alzheimer's Disease or dementia in general

Dementia is characterised by impairment of higher critical functions – thinking, orientation, calculation, comprehension, learning capacity, language and judgement, deterioration in emotional control, social behaviour and personality. It occurs as a result of several different disease processes and there are characteristic neuropathological features which can only be established at autopsy.

Dementia Pugilistica

Severe head trauma can cause dementia at short interval from the trauma. The first medical description of a “punch drunk” fighter was in 1928¹ and since then further reports have clearly established the existence of dementia pugilistica, a form of traumatic encephalopathy. Dementia pugilistica is characterised pathologically by the presence of neuro-fibrillary tangles. Risk of developing this is positively correlated with the length of a boxer's career (ie the number of bouts), being a sparring partner and negatively correlated with the boxer's skill².

From the late 1980s with the increase in the UK elderly population, interest developed in late onset dementia and its causes, and specifically on Alzheimer's Disease, which accounts for at least half of all cases of dementia examined at necropsy³. Since Alzheimer's Disease is also characterised by neuro-fibrillary tangles, amongst the almost 20 putative risk factors proposed for the condition⁴ was head injury.

During the 1990s a large body of research, mainly case-control studies, looked at Alzheimer's Disease or dementia (in general) and its association with head injury. The results were inconsistent. Case control studies tended to be supportive while the few cohort studies were not^{5, 6}.

Methodological problems limit the interpretation of many of the case-control studies on head injury and dementia. Some studies have been small. Diagnosis has been clinical and head injury has been variously defined including with or without loss of consciousness. Most have relied on retrospective recall of head injury, sometimes from the demented person, most frequently from a proxy informant. This approach is subject to random error and bias as individuals are asked to recall events that occurred decades earlier. There must also be questions about the accuracy of the details of the head injury. In addition not all studies have taken account of obvious confounders like alcohol use.

¹ Mortland, H S (1928) Punch Drunk, JAMA, 91:1103 0-1107

² Guterman, A et al (1987) – Neurological Sequelae Of Boxing. Sports Med 4 194-210

³ Deary, I J et al (1988) Recent Research On The Causes Of Alzheimers Disease. BMJ 297:807-809

⁴ Henderson, A S (1988) The Risk Factors For Alzheimer's Disease: A Review And A Hypothesis. Acta Psychiatr Scand 78:257-275

⁵ Wilkins, D B et al (1991) Brain Injury And Neurological Sequelae: A Cohort Study. Neurology 41:1554-1567

⁶ Katzman, R et al (1989) Development Of Dementing Illness In An 80 Year Old Volunteer Cohort. Ann Beurol 25:317-324

Some studies have reported an increased risk of Alzheimer's Disease with head injury with loss of consciousness in males⁷, but not in females, and in those without a family history. Some found an association between head injury (without loss of consciousness) and any dementia, not just Alzheimer's Disease^{8, 9}.

A recent study¹⁰ looked at the suggestion that in those with traumatic brain injury who develop Alzheimer's Disease, the time to clinical disease onset is reduced.

The study provided no evidence of increased risk of Alzheimer's Disease following traumatic brain injury. Using tables of age of onset distribution for a previous cohort of Alzheimer's Disease cases from the same area and without a history of head injury, the results suggested a median time of 10 years to diagnosis rather than the expected 18 years (p=0.015) ie traumatic brain injury reduces the time to clinical onset of Alzheimer's Disease amongst people at risk of developing the disease.

Last year¹¹ a prospective historical cohort study shed some more light on the matter. Using military hospital records, the study on World War II US navy and marine veterans, investigated the association between non-penetrating head injury and Alzheimer's Disease or other dementia in late life. Head injury was defined and dementia was ascertained by a multi-stage detection and assessment protocol. The study also included examination of dose-response effect.

Men were considered to have had head trauma if (i) it was documented in the service medical records (ii) occurred during service, (iii) produced loss of consciousness, post-traumatic amnesia or skull fracture (iv) did not penetrate the dura mater (v) did not result in marked cognitive impairment or neurological sequelae more than three months post-trauma. By "marked" is meant likely to cause substantial limitations in activities of daily living.

A total of 1422 suitable cases were identified and the head injuries rated:

- Category 1 - mild injury – loss of consciousness or post-traumatic amnesia of less than 30 minutes with no skull fracture;
- Category 2 - moderate injury – loss of consciousness or post-traumatic amnesia, more than 30 mins less than 24 hours with or without skull fracture;
- Category 3 severe injury – loss of consciousness/post-traumatic amnesia for more than 24 hours.

The unexposed group were men with diagnoses of pneumonia/laceration or other wound, but no history of head injury pre or post service.

⁷ Mortimer, J A et al (1991) Head Trauma As A Risk Factor For Alzheimer's Disease. A Collaborative Re-analysis Of Case-Control Studies. *Int J Epidem* 20, 2:528-535

⁸ Salih, E (1997) Head Injury And The Risk Of Alzheimer's Disease: A Case Control Study. *International J Ger Psychiatr* 12:363-368

⁹ Rasmusson, D X et al (1993) Head Injury As A Risk Factor In Alzheimer's Disease. *Brain Injury*. 9, 3 213-219

¹⁰ Nemetz, P et al (1999) Traumatic Brain Injury And Time To Onset Of Alzheimer's Disease: A Population Based Study. *Am J Epidem* 101, 149, 1:32-40

¹¹ Plassman, BL et al (2000) Documented Head Injury In Early Adulthood And Risk Of Alzheimer's Disease And Other Dementias. *Neurology* 55:1159-1166

Rates of participation in the study were similar in cases and controls and the majority of subjects were Caucasian. Cases of dementia were identified using a three-stage screening and assessment procedure – involving telephone interviewing and finally full clinical assessment. Similar procedures have been used in similar studies for over 10 years and are accepted as robust. The results showed that in this group of men history of head injury increased the risk of Alzheimer's Disease (HR^{*} = 2.0, 95% CI^{**} = 1.03 to 3.90)¹² and all dementia (HR = 2.23, 95% CI = 1.30 to 3.81).

The risk of Alzheimer's Disease and dementia related to the severity of head injury.

Compared with the group with no head injury there was increased risk of Alzheimer's Disease (HR= 2.32, 95% CI = 1.04 to 5.17) and dementia (HR = 2.39, 95% CI = 1.24 to 4.58) in those with moderate head injury. Similarly severe head injury was associated with increased risk of Alzheimer's Disease (HR = 4.51, 95% CI = 1.77 to 11.47) and dementia (HR = 4.48, 95% CI = 2.09 to 9.63). However, the mildly injured group showed no increased risk of Alzheimer's Disease (HR = 0.76, 95% CI = 0.18 to 3.29) or dementia (HR=1.33, 95% CI = 0.51 to 3.47) compared to the non-head injured group.

Some caution in interpreting these findings is merited. As expected with a study over 50 years there was evidence of attrition at all stages. This is potentially a source of bias if a higher proportion of demented non-head-injured patients went missing from the cohort. Comparison of participants and non-participants did not suggest attrition on that basis but it must be accepted that some such effect is possible.

In addition between service termination and development of dementia these men were exposed to many other factors and influences. This study looked at some of these, eg alcohol and cigarette consumption and did not find that they altered the association between head injury and dementia.

Conclusion

In a war pensions context it can now be accepted from available published peer-reviewed evidence that a reasonable doubt is raised that moderate head injury (defined as sufficient to lead to loss of consciousness or post-traumatic amnesia of more than 30 minutes with or without skull fracture) or severe head injury (ie with loss of consciousness or post-traumatic amnesia of more than 24 hours) is causally associated with onset of Alzheimer's Disease or other dementia even 50 years after the incident.

Dr E A Braidwood, March 2001

¹² HR = Hazards Ratio, CI = Confidence Interval

**High Court of Justice
King's Bench Division
(Ormerod, J)
31st January, 1952
Tigg, J C v The Minister of Pensions**

Evidence - merely because a doctor of eminence is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean that there is a doubt entitling an appellant to a pension.

(Post encephalitic Parkinsonism)

The appellant, John Currey Tigg, applied for an award under the Royal Warrant on the grounds that the post encephalitic Parkinsonism from which he was suffering was due to service. The Minister rejected the claim and, on appeal under s1(1) of the Pensions Appeal Tribunals Act, 1943, the Tribunal upheld the Minister's decision. The appellant contended that the Tribunal's decision was erroneous in point of law and appealed under s6(2) of the said Act to the nominated Judge of the High Court.

Held - that the application for leave to appeal be refused.

Judgement

Mr Justice Ormerod: Merely because a doctor of eminence, and I have no doubt the doctor in this case was of very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean there is a doubt and the Appellant must be thereby entitled to a pension. It is a question of fact for the Tribunal. If they are satisfied on evidence of a certain question of fact, and clearly there was evidence here on which they could be satisfied, it is quite impossible for me to interfere. The application is refused.

[back to Changes in medical understanding affecting War Pensions](#)

**High Court of Justice
Queen's Bench Division
(Ormerod, J)
25th November, 1955
Howard v The Minister of Pensions and National Insurance**

Medical Evidence - where there are conflicting medical opinions as to the cause of a disease, there is no error in point of law if the Tribunal after carefully weighing the evidence decide to accept one opinion in preference to another.

(Vertigo)

The Appellant, Arthur Howard, was awarded a pension under the Air Force Order on the grounds that the tuberculosis from which he suffered was attributable to service. During treatment for this disease in 1947-48 he was given 63 grains of streptomycin over a period of 63 days. In 1952 the appellant made a further claim for pension on account of vertigo, contending that this complaint was caused by the administration of the streptomycin and hence was due to service. The Minister rejected the claim and on appeal under Section 1 of the Pensions Appeal Tribunals Act, 1943, the Tribunal upheld the Minister's decision. The appellant contended that the Tribunal's decision was erroneous in point of law and appealed under section 6(2) of the said Act to the nominated Judge of the High Court.

Held, that the appeal be dismissed.

Judgement

Mr Justice Ormerod: This is an application by Arthur Howard for leave to appeal against a decision of the Ministry dated the 24th February, 1945, when the Tribunal decided that vertigo, of which he was complaining, was not due to the administration of streptomycin, which was part of the course of treatment which he had for tuberculosis, the tuberculosis being admittedly attributable to his war service.

At the present time, the Applicant is about 64 years of age. He joined the Royal Air Force in September, 1939, and served until August, 1945, but he had served before 1939 and in fact had served in the 1914-1918 war. He was discharged in 1945 suffering from tuberculosis. He was admitted to hospital in 1947 and an operation for thoracoplasty was performed upon him, probably in three stages (there is some little doubt about it) up to June of 1948. During that period at the age of about 58, he received an injection of streptomycin, amounting to one grain each day, for 63 days. He was discharged from hospital in October, 1948 and appears to have made no complaint of dizziness even to his own doctor until January or February 1951, more than a year after his discharge from hospital, and probably more than 18 months after the administration of streptomycin.

There was a considerable difference of medical opinion in this case. The opinion of Dr Friend and another surgeon was that the vertigo, from which this man admittedly suffers now, was certainly due to streptomycin. The administration of streptomycin does from time to time, particularly in elderly people, produce symptoms of this kind arising from labyrinthine degeneration in the ear apparently set up by the drug. That is agreed by doctors on both sides. The medical evidence adduced by the Ministry and the evidence of the independent specialists called in was that although it is true that a condition of vertigo may be caused by the administration of streptomycin, in this case the vertigo was not caused in that way. There was evidence that in 1941 Mr Howard complained of vertigo and I think another factor which weighed with the Ministry was that there was this gap from some time in 1948, when the administration of streptomycin ceased, to the beginning of 1951, during which there was no complaint of vertigo.

The Ministry formed the view that streptomycin was not the cause of the condition from which this man unfortunately suffers now, but that it was probably the result of a degenerative condition of the labyrinth of the left ear known as Meniere's disease. That was the evidence before the Tribunal. The Tribunal had before them this conflict of evidence. There was the Report on the one hand of the Medical Services Division of the Ministry and other doctors who had examined the Applicant on behalf of the Ministry, and on the other hand there was the evidence of the two doctors who had examined the man on his own behalf. The Tribunal having considered the whole of that evidence decided that the Ministry had discharged the burden of proof which was upon them to show that this man's condition was probably not due to the administration of streptomycin.

It is argued by Mr Read, who has I think put before the Court every point that could properly be put in this case, that the burden of proof upon the Ministry is a heavier one than is normal because they accepted an additional burden by reason of the fact that symptoms of this kind are frequently set up as a result of the administration of streptomycin. That matter was before the Tribunal and they were fully aware of the burden of proof which rests on the Ministry. Having considered the whole of the evidence they decided that the burden had been discharged and in those circumstances there is nothing this Court can do to interfere with their decision. It may be right or it may be wrong. That is not for me to judge, and indeed it is no part of my duty to

have to do it. It is a matter entirely for the Tribunal to consider the evidence and to come to a conclusion upon the evidence. They have considered the evidence and have come to a conclusion.

As I have said time and again in these cases the mere fact that there is a difference of medical opinion does not mean that there is a doubt and therefore the Appellant must have the benefit of it. There is only a doubt if the Tribunal, having considered the whole of the evidence, are left in doubt, in which case the burden of proof has not been discharged. That was not so in this case. The Tribunal gave a decision and there was evidence to support it, and therefore this court can not interfere.

In these circumstances I propose to grant leave to appeal and dismiss the appeal.

[back to Changes in medical understanding affecting War Pensions](#)

Medical Appendix

Atherosclerosis

Definition

1 In developed countries, occlusive vascular disease due to atherosclerosis and associated thromboembolic phenomena is the major cause of mortality and morbidity.

2 Atherosclerosis is the term applied to a focal condition of the intima (inner lining) of arteries associated with medial (middle coat) changes. The changes are widespread throughout the body, but differ in degree at different sites. In order, the coronary and cerebral arteries, the aorta and the arteries supplying the lower limbs are those most commonly affected.

3 Circulation of blood depends on a complex relationship between components of the blood vessel wall and of the flowing blood. In atherosclerosis we have an abnormal narrowed blood vessel wall causing reduced tissue perfusion. These altered walls also affect blood coagulation.

Clinical manifestations

4 Symptomatic arterial occlusion usually arises from stenosis of a segment of artery or from thrombosis occurring on an stenosed area. Haemorrhage into an atherosclerotic plaque, embolization of thrombotic material or spasm of a diseased vessel may also be involved.

Coronary arteries

5 Atherosclerosis of the coronary arteries results in reduced coronary blood flow and a discrepancy between the supply of and the demand for oxygen by the heart muscle. This causes **ischaemic heart disease** or **myocardial ischaemia**. Clinically, the resultant chest pain or tightness is angina.

6 If the coronary artery lumen is totally occluded, the resultant tissue necrosis of heart muscle supplied is **myocardial infarction**, commonly known as a "heart attack" or "coronary". The terms myocardial infarction and coronary thrombosis are often used synonymously although tissue infarction may occur without thrombosis.

Cerebral arteries

7 Several syndromes arise from atherosclerosis of the cerebral arteries. In most developed countries cerebrovascular disease is the third most common cause of death after ischaemic heart disease and cancer. It is the cause of much physical and mental disablement in the elderly.

[Medical adjudication guidance in claims for atherosclerosis continued](#)

[back to Medical Adjudication Contents](#)

The law

How the Scheme works

2 A war pension may be claimed for any disablement by anyone who has served. Claims may be made at any time from service release. Decisions are evidence based and each case must be determined on its own individual merits. Awards may be made where, within the relevant law, the evidence including service and medical facts and the contemporary medical understanding of the condition claimed, shows a causal link between service and the claimed condition, on the basis of the relevant burden and standard of proof as referred to in paras [3] and [4] below.

3 For claims made not later than 7 years after leaving the armed forces, Article 4 of the Service Pensions Order provides that the onus is on the Secretary of State to show beyond a reasonable doubt that the claimed disablement is not attributable to, or aggravated by, service. If he cannot show this an award of War Disablement Pension may be made.

4 For claims made more than 7 years after the end of service, Article 5 of the Service Pensions Order puts the onus on the claimant to raise, by way of reliable evidence, a reasonable doubt that the claimed condition is attributable to, or continues to be aggravated by, a service injury.

Case law

5 The High Court has held that the word "reliable", in the context of Article 5, cannot have been intended to mean "convincing", but means more than "fanciful". It held that, with particular reference to "changes of medical opinion" that "there are, in my judgement, three stages: no reasonable doubt, reasonable doubt, and consensus". A war pensions claim under Article 5 would pass the test at the point where the (reliable) evidence raised a reasonable doubt, but: "a mere hypothesis based on a limited study ... would not have created a "reasonable doubt" within the terms of Article 5(4)." The real question, however, it held, is whether the evidence raises a reasonable doubt in the mind of the Secretary of State (SoS). If he finds the evidence unreliable it obviously will not raise a reasonable doubt in his mind. (Case of Edwards 1992 HCJ no CO/2281/90).

6 The Courts have also held that a conflict of medical opinion does not, of itself, mean that a reasonable doubt has been established, and that a claim must therefore succeed. This applies irrespective of the eminence or authority of those expressing the opinions. In the case of Tigg v The Minister of Pensions the presiding Judge stated "Merely because a doctor of eminence, and I have no doubt the doctor in this case was of very great eminence, is expressing a view contrary to the view expressed by the medical witnesses called on behalf of the Ministry, does not mean there is a doubt and the Appellant must therefore be entitled to a pension. It is a question of fact for the Tribunal" (cases of Tigg ROSWPA vol 5 p 141 and Howard ROSWPA vol 5 p515).

[Medical adjudication guidance in claims for atherosclerosis continued](#)

[back to Medical Adjudication Contents](#)

Basic concepts

Psychological stress

7 Professor Sleight does not precisely define psychological stress other than as "psychosocial factors (loosely called stress)". For the remainder of this document for brevity the term stress will be used to cover this concept. Stress lacks consensus definition. Current models present stress as part of an interactive life process designed to maintain the status quo. An adverse stress reaction occurs where in an individual there is an imbalance between environmental challenge and his subsequent physical and psychological response. The normal response to a stressful event is well documented. The actual response of the individual is a function of his or her coping resources, personality, previous experience and social support. Stress, then, is unique to individuals and highly subjective.

Coronary artery disease

8 Coronary artery disease results from atheromatous furring of the coronary arteries. In atherosclerosis fatty material accumulates in arterial walls eventually forming plaques which obstruct flow of blood in the artery. Atherosclerosis is constitutional and age-related and major environmental risk factors have been known for many years. These include hypertension, cigarette smoking, high serum cholesterol, diabetes mellitus and sedentary lifestyle. These factors account for only about 50% of episodes of symptomatic coronary disease.

9 Other possible risk factors have therefore been explored. Coronary disease is linked to socio-economic status, one of whose measures is occupation. A considerable body of work on stress and coronary disease has looked at occupational factors, particularly the individual's control over workplace decision-making and practices. (Kaplan and Keil 1993).

Reliable evidence

10 In considering under Article 5 whether there is reliable evidence to raise a reasonable doubt that service related stress might cause atherosclerosis or symptomatic coronary disease, relevant case law has been taken into account. In particular in assessing evidence regard has been paid to:-

- whether conclusions of studies are hypotheses; or whether there is supporting evidence, which goes beyond hypothesis, and if so, its basis
- the reaction of other experts in the field to the evidence
- the weight of overall evidence on the matter.

Assessment of evidence

11 Evidence for any new approach in science must always be considered and weighed relative to the existing body of evidence on a subject, with account taken of the robustness and authority of new studies. In particular the design and methods, sample size, case and control selection, statistical validity, repeatability of findings, approach to bias and possible alternative factors and hidden influences. Other important factors include whether the findings have been replicated by other independent researchers and the overall plausibility/consistency relative to contemporary understanding.

12 The following paragraphs summarise the Department's examination of contemporary medical evidence on stress and coronary disease in the context of the War Pensions Scheme.

13 Part of Professor Sleight's paper considers essential hypertension, a key risk factor for atherosclerosis. The Department has produced a separate medical appendix for hypertension in which the link with stress is addressed, the line taken in the appendix follows current generally accepted UK medical understanding (Julian et al 1996). That is - that there is no aetiological link between acute or chronic psychological stress and sustained hypertension in humans.

14 Applying the test set out in para [10] above, the papers discussed by Professor

Sleight and other pertinent studies have been critically evaluated. As Professor Sleight indicates: there is evidence linking social isolation/support, coping styles, behaviour, anger and hostility with coronary disease (Kaplan and Keil 1993) and very traumatic occurrences such as bereavement and natural disaster may cause temporary increase in risk (Jones 1987: Trevisan et al 1992). Studies on workplace factors and most recently, interactions of these factors, also continue to appear. (Hallquist et al 1998). Lack of precise definitions, reliance on recollection of past events, speculation and conditional language are common in the papers linking coronary disease with stress in the workplace. Most studies discuss stress in relation to symptomatic coronary heart disease (myocardial infarction or coronary thrombosis: cardiac arrhythmia: sudden cardiac death). Few discuss the atherosclerotic process itself and there is little support for a link with stress from prospective or angiography studies (Hollis et al 1990: Tennant et al 1988). Professor Sleight's review concentrates on evidence supportive of his proposition regardless of study design. Other papers offer different conclusions on these issues (Tunstall-Pedoe et al 1997: Light et al 1992).

15 Where psychosocial factors have been proposed as an explanation for observed differences in rates of coronary disease, it is observed that these factors are not the only differences between case and control groups. (Marmot et al 1992). Differences in rates of coronary thromboses in Whitehall civil servants for example **may** be due to psychosocial factors, but since adverse stress reactions and their reasons are unique to individuals, other factors might well be the underlying cause. Among males in the lower civil service grades, where coronary disease more commonly causes symptoms than in higher grades, men are shorter, fatter, smoke more, have a higher prevalence of diabetes and hypertension and are less active in their spare time. The paper refers to the suggestion (Alfredsson et al 1982) that the key issue is control over events. However, there is evidence - including the very nature of public and private sector work today - that people in more "high ranking" jobs still have, and feel they have, little control over what they themselves deem to be important. Yet in this group there is a sustained decline in the incidence of coronary disease.

16 The Department has no generalised view of the stresses of service life. It is accepted that service - particularly active service - provides multiple challenges for individuals who will react in individual ways. Because of the obvious practical difficulties, there are few large general studies of service and particularly ex-service populations. There is nothing, however, to suggest that coronary disease is more common in those who have served in the armed forces.

17 There can be no doubt that in this century, prisoners of war, whether in Europe, Japan or Korea, experienced physical and psychological conditions representing the extremes of stress both in terms of control over life and immediate threat. If there is a link between stress in service and atherosclerosis, one would expect evidence from this group. Since the war numerous follow-up studies of the health of ex-prisoners of war have been carried out. Some of these have continued over many years and the US Medical Follow Up Agency study of former POWs is continuing. **To date there is no published evidence that morbidity or mortality from cardiovascular disease or hypertension is significantly different in ex-prisoners of war. (Page 1992).**

18 Another obvious and important factor in atherosclerotic disease is age. A large proportion of war pension claims are made years after service release for conditions which have only then, around the time of claim, come to light. Reflecting the fact that claims can be made for any disablement, and its prevalence in the community, many of these claims concern coronary disease. They are often accompanied by the contention that an event or circumstance in service caused stress and hence the claimed condition. Atherosclerosis starts to develop from childhood (Berenson et al 1998) but produces symptoms only in middle age or later. Most studies into the causes of coronary disease therefore concern a middle-aged - usually male - population. There is little published evidence on coronary disease in older age groups although clinical and autopsy evidence suggest there may be significant differences between this population and the middle-aged.

19 Theorell (1992) , in a review of evidence linking working conditions to coronary disease, suggested that the failure of Reed et al (1989), in the Hawaii cardiovascular survey to show any association between high demand - low decision latitude and risk of coronary illness, related to the fact that the subjects of the study were all 55 years or older at the start of the follow-up period. A similar age-related phenomenon is seen in a recent Finnish study. (Kamarck et al 1997) where blood pressure changes were demonstrated only in young subjects. Most of the positive studies ie apparently showing a link between stress and coronary disease, have relatively short follow-up periods between the stressful event or circumstance and the coronary event. No published report linking onset of coronary disease symptoms to acute or chronic stress many years previously has been identified.

[Medical adjudication guidance in claims for atherosclerosis continued](#)

[back to Medical Adjudication Contents](#)

Study by Australian Department of Veterans Affairs

20 Since Professor Sleight's paper, the Department of Veteran's Affairs in Australia has investigated this topic. A conference was convened in February 1998 by the Australian Repatriation Medical Authority - a body established to produce Statements of Principle to be agreed by Parliament. The aim was to examine the effects of military stress on psychiatric illness and cardiovascular disease. The conference took place over 3 days and brought together overseas authorities and researchers and ex-service representatives. The conference first confirmed the criteria required before associations would be accepted as causal. They went to develop a definition of "military stressor":-

"the individual experienced, witnessed or was confronted with an event or events that involved actual or threat of death or serious injury, or a threat to the individual's or other people's physical integrity that might evoke intense fear, helplessness or horror".

21 It was recognised that this definition was severe. The conference took the view that less obviously severe stressors might lead to illness but chose not to deal with the data on such effects because of "**lack of consistent systematic definitions for such stressors in the literature and the very diverse methodologies and outcome measures which allowed little opportunity for pulling of data or comparison**". (Quotation from RMA Conference Draft Report, July 1998). It should be noted that the Australian Repatriation Scheme is potentially more generous to the claimant than the UK scheme, requiring no more than a **reasonable hypothesis** of a link between service and the claimed condition. By contrast, the UK scheme requires **reliable evidence** to raise a **reasonable doubt**.

22 The formal presentations, which included detailed literature reviews as well as results of primary research findings, and syndicate and group discussions, included a range of opinion. Of particular interest in the current context was a 50 year prospective study of the psychological sequelae of World War II. This demonstrated that much retrospectively collected material supports mere association and not causal link. It also demonstrated the importance of bearing in mind possible hidden influences including alcohol and cigarette consumption when considering coronary outcomes.

23 The conference reached consensus on a number of matters.

- It was agreed that acute exposure to military stressors (as defined) might be followed by sudden cardiac death or cardiac arrhythmia.
- It was not considered that the body of evidence on exposure to military stressors and the development of hypertension supported a causal link.
- In relation to psychiatric illnesses, panic disorder was considered potentially causally associated with sudden cardiac death.
- The limited data on PTSD and its frequent co-morbidity with other disorders, including substance abuse, precluded recognition of a causal association between PTSD and any cardiovascular outcome.

[Medical adjudication guidance in claims for atherosclerosis continued](#)

[back to Medical Adjudication Contents](#)

Conclusion

The Department's position on claims linking service stress with atherosclerotic coronary disease

24 The evidence has been studied and carefully evaluated; it is concluded that atherosclerosis is a constitutional age-related condition with major environmental risk factors, confirmed over many years in international studies. It is accepted that where acute coronary disease (myocardial infarction or coronary thrombosis: cardiac arrhythmia or sudden cardiac death) occurs in close time relation to acute stress (physical or psychological) the two may be causally linked. **The evidence does not, however, support a causal association between non-acute stress and atherosclerotic coronary disease.**

25 In war pension claims for acute coronary disease (myocardial infarction or coronary thrombosis: cardiac arrhythmia or sudden cardiac death) occurring in service - made within 7 years of service release (Article 4) - **where acute physical or psychological stress is accepted by the SoS as due to service, the claimed condition may, depending on the individual facts of the case, be accepted as attributable to service.** Similarly, for claims beyond seven years (Article 5), decisions are informed by the case facts including service theatre, duration and type of service and the exact circumstances of the claimed stress and its relation to the cardiac condition. There is no blanket rejection or acceptance.

References:

1. Julian et al (1996) disease of the Heart 2nd ed. London Saunders.
2. Tunstall - Pedoe et al (1997). Comparison of the prediction by 27 different factors of coronary heart disease and death in men and women of the Scottish heart health study: cohort study. *BMJ*; 315:722-729.
3. Light et al (1992). Job strain and ambulatory work blood pressure in healthy young men and women. *Hypertension*; 20: 214-218.
4. Theorell (1992). The Psychosocial environment, stress and coronary heart disease in Marmot et al ed. *Coronary Heart Disease Epidemiology from aetiology to public health*. Oxford University Press. 256-273.
5. Reed et al (1989). Occupational strain and the incidence of coronary heart disease. *Am. J. Epidemiol*; 129: 495-502.
6. Kamarck et al (1997). Exaggerated blood pressure responses during mental stress are associated with enhanced carotid atherosclerosis in middle-aged Finnish men. *Circulation*; 96: 3842-3848.
7. Davies (1996). Atherosclerosis and ischaemic heart disease in Julian et al. *Diseases of the Heart*. 2nd ed. London. Saunders. 944-978.

[back to Medical Adjudication Contents](#)

Training note

Date: 25 April 1996

H pylori and gastrointestinal disease Date of change of medical opinion

1. You may remember that Dr Braidwood published a medical update on H pylori related gastrointestinal disease in the 10 November 1994 issue of the BA Medical Services Weekly bulletin. This update included a discussion on certification issues. It was apparent that a review of our certification policy was appropriate, and A1 was asked to consider the implications.
2. The cases at particular issue were those Article 5 cases where there had been an in-service gastritis followed by an asymptomatic period, with post-service development of a peptic ulcer. MAs were asked to stockpile this type of case (both claims and appeals) pending A1 advice.
3. In such cases, viewed quite properly on an individual basis, the outcome, depending upon circumstances, could be either:
 - a) Gastritis (service dates) - ATTRIB
Peptic Ulcer - ATTRIB

OR

 - b) Gastritis (service dates) - ATTRIB
Peptic Ulcer - NANA.
4. Current medical knowledge now raises a reasonable doubt that the two conditions, gastritis and peptic ulcer, have a common aetiology, H pylori. This therefore precludes automatic rejection of peptic ulcer in the in-service gastritis/asymptomatic interval/post-service peptic ulcer case. In such cases, if you are satisfied that the in-service episode was a pure gastritis (**NOT** gastroenteritis), then a certificate should be issued in respect of both the in-service gastritis and the subsequent peptic ulcer, ie:
 - a) Gastritis (service dates) - ATTRIB
 - b) Peptic Ulcer - ATTRIB
5. A1 has concurred with this change and the date of change of medical opinion is 1 March 1993, which is the publication date of the paper by Tytgat G N, Loach L A, Rauws E A, H pylori infection and DU Disease, Gastroenterology Clinics of N America, 22(i): 127-39, 1993 Mar.
6. In cases where the onset of dyspepsia is post-service and there is no evidence that the claimant experienced upper gastrointestinal symptoms in service, then the condition(s) (gastritis or peptic ulcer) should be rejected, NANA. The claimant may

contend that he became infected with H pylori during service, with subsequent development of peptic ulcer. Current bacteriological knowledge does not support this contention. In particular, there is no evidence that military conditions predispose the individual to H pylori infection. The present practice of rejecting these claims should remain unchanged.

7. The relationship between H pylori and gastric cancer remains to be elucidated. At present, if there is an episode of in-service gastritis (circumscribed) and gastric cancer arises post-service, this will be for rejection. If there is an in-service dyspepsia followed by ongoing problems, then that case should be decided on its own merits. It would be sensible to discuss such a case with the Training Officer.
8. I have summarised all the main points of current policy in respect of H pylori and gastrointestinal disease below, together with the main points of this minute. If you require a copy of Dr Braidwood's original article, please contact Medical Library.

Summary

Article 5

1. Gastritis arising in service - attributable.
2. Peptic ulcer arising subsequent to in-service gastritis - attributable. The date of change of medical opinion is 1 March 1993.
3. Gastric cancer arising post-service where there has been a circumscribed episode of gastritis in service - NANA.
4. Gastric cancer arising post service, dyspepsia in service with ongoing problems - each case to be decided on its own merits.

Article 4

Gastritis, peptic ulcer and gastric cancer arising in service are normally attributable.

FIBROMYALGIA

Until now fibromyalgia has been considered a variant of CFS (Chronic Fatigue Syndrome). Present evidence is that it should be considered a separate condition. Although not a topic in the current edition of the Oxford Textbook of Medicine, a useful review of fibromyalgic syndrome was published in the British Medical Journal in 1995 (BMJ, Vol 310, 11 February 1995, 386-389).

The condition is usually primary and more common in females aged 40-60. It is characterised by:-

1. Pain, usually axial, with or without stiffness
2. There is often subjective swelling and paraesthesia of hands and feet
3. Fatiguability, non-restorative sleep
4. Headaches affecting the occiput or bifrontal
5. Diffuse abdominal pain with altered bowel habit
6. Genitourinary problems including dysmenorrhoea

Clinical signs are not remarkable, except multiple hyperalgesic tender sites. There is a discordance between the objective findings and the claimed disablement.

In many patients there is co-existing anxiety and depression, but the precise relation of the psychological symptoms to the condition is not known. Diagnosis of fibromyalgia is by exclusion. Management is aided by explanation. Low dose tricyclic antidepressants and a graded exercise programme improve aerobic fitness.

[back to Medical Adjudication contents](#)

CHRONIC FATIGUE SYNDROME

[Summary of Fukuda criteria](#)

Fukuda K et al: Annals of Internal Medicine 121, 12, 953-959

- 1). Fatigue lasting for six months or longer where other known causes have been excluded from history, physical examination, mental state assessment and appropriate tests.
- 2). Four or more of the following present concurrently for six months or longer:-
 - a.) Impaired memory or concentration
 - b.) Sore throat
 - c.) Tender cervical or axillary lymph nodes
 - d.) Muscle pain
 - e.) Multi-joint pain
 - f.) New headaches
 - g.) Unrefreshing sleep
 - h.) Post-exertion malaise

[back to Medical Adjudication contents](#)

MEDICAL APPENDIX

Hypertension

1 When the heart contracts, blood is forced into the arterial system under pressure. The maximum pressure in the arterial blood during this phase is known as the **systolic blood pressure**. In the interval between contractions, the pressure reduces until the next cardiac contraction. The lowest level in the pressure in the arterial blood during this phase is known as the **diastolic blood pressure**.

2 Arterial blood pressure (both systolic and diastolic) increases with age, and exhibits a wide range in the normal individual both during a 24 hour period and from day to day. In the same individual transient marked variations in blood pressure are common in relation to physical stress, temperature, season, sleep, food intake or sexual activity.

Definition

3 Blood pressure, like height and weight, is a characteristic of the individual. As such blood pressure varies widely among individuals. Some people have blood pressure above the mean, others below. The distribution curve is slightly asymmetrical with a tail to the right particularly with age.

4 In a defined population, there is no dividing line between normal and abnormal blood pressure. The presence of a recordable blood pressure is necessary to sustain life. Two individuals may have the same absolute recorded blood pressure but only one may properly be considered hypertensive.

5 Hypertension is defined as a **sustained** level of blood pressure above the mean blood pressure in the population from which the individual is drawn allowing for age, sex and race. A single raised blood pressure reading is not hypertension.

6 The concept of hypertension as a disease is rather different from conditions such as gout, pneumonia or neoplasm, all of which are either present or absent, that is they are qualitative phenomena. Hypertension is quantitative.

Clinical manifestations

7 Most cases of hypertension come to light at routine medical examination. Earlier this century, many symptoms including headache, dizziness and epistaxis were ascribed to high blood pressure. Evidence now shows that high diastolic arterial pressure alone, up to about 130 mmHg, does not cause symptoms. Even above this level it will remain asymptomatic until there is significant organ damage. It is this organ damage, much commoner in the period before 1960 when drug treatment of hypertension was more limited and less effective, which caused the dizziness, headache and epistaxis. When diastolic blood pressure is sustained about 130 mmHg the condition is described as malignant hypertension. This is a medical emergency. It is characterised by retinal and renal damage caused by fibrinoid necrosis of the renal and retinal arterioles. The condition is fully reversible on treatment.

[Medical adjudication guidance on hypertension continued](#)

[back to Medical Adjudication Contents](#)

ANNEX A

WAR PENSIONS - NOISE INDUCED SENSORINEURAL HEARING LOSS (NISHL)

| DATE | Key Legal/medical changes | Operational approach |
|---|---|---|
| Before 1 Jan 1981 | Science of NISHL not established | Assessments for service related NISHL included all hearing loss at date of claim i.e. age related hearing loss and other forms of hearing loss not due to service. This approach was reasonable in the light of the then understanding. |
| 1 Jan 1981 and after | Validated evidence became available that post service hearing loss due to age or any other cause (e.g. post service industrial noise) cannot be due to, or increased by service related NISHL. Noise induced hearing loss does not increase on removal from the noise injury. Hence it is always possible from now to make final assessments for service related NISHL based on evidence at service release, even though the change was not effected until 22 October 1996. | <p>Although noted in Departmental guidance this understanding was not reflected in the Department's approach to the assessment of NISHL which still included age related hearing loss at the date of claim. This approach was unreasonable in the light of contemporary scientific and medical evidence.</p> <p>To reflect current scientific understanding the relevant assessment for service related NISHL should be made at service release.</p> |
| 7 Jan 1993 and after | <p>Law amended from 7 January 1993 to bring war pensions into line with industrial injuries. As a result:-</p> <p>(i) no awards can be made for service related NISHL assessed at less than 20%; and</p> <p>(ii) no additions can be made to NISHL assessment for symptoms and conditions consequential to NISHL, where hearing loss alone is assessed at less than 20%</p> <p>(iii) less than 20% NISHL assessments cannot be aggregated with assessments for other conditions and included in the overall composite assessment.</p> | <p>From late January 1993, where entitlement to NISHL was given, assessment was made on evidence at service release.</p> <p>It was thought however at that time that, following service release, the accepted NISHL could interact with other types of post service hearing loss to produce a level of hearing loss greater than if the two types were simply added together. That being so Greater Disablement (GD) was considered appropriate and it's application led to NISHL assessments being increased.</p> <p>Again this approach did not reflect the then generally accepted scientific understanding.</p> |
| 22 October 1996 & 5 December | It was recognised that the Department's approach did not reflect accepted scientific | Change of approach to the assessment of NISHL from 22 October 1996 when it was brought |

| | | |
|----------------------|--|--|
| 1996 | understanding. | <p>into line with scientific understanding. Change announced at CAC meeting of 5 December 1996.</p> <p>Subsequently, Ministers decided that :-</p> <p>(i) cases should not be sought routinely but reviewed only when they arise; and that</p> <p>(ii) no pensioner already in receipt of an award for NISHL should have that reduced or taken away, following correction of an erroneous decision.</p> <p>(iii) Claims and applications for a deterioration review made before 1 March 1996¹ and still outstanding on 22 October 1996 would be considered on the basis of the January 1993 approach to handling claims.</p> |
| From 22 October 1996 | <p>New guidelines and procedures introduced to achieve the Ministerial promise on claims and reviews in respect of NISHL</p> <p><u>Detailed guidance on processing transitional cases is at Annex B</u></p> | <p>CLAIMS AND REVIEWS DIVIDED INTO DEFINED CATEGORIES</p> <p><u>CATEGORY A</u></p> <p>Decisions in respect of NISHL made before 1 January 1981 and therefore within the law because they were reasonable at the time they were made.</p> <ul style="list-style-type: none"> • Neither the 20% assessment cut off rule nor the change in approach to the assessment of NISHL apply, although existing assessment for NISHL and therefore, the award in respect of that condition cannot be increased. • Assessments made final • Any level of NISHL assessment can be aggregated with assessments for other conditions |

1. See page 27 Annex C. Category C, para 2

| | | |
|----------|----------|--|
| As above | As above | <p><u>CATEGORY B (TWO SUB TYPES)</u></p> <p>Decisions in respect of NISHL made on or after 1 January 1981 and therefore outside the law because they were unreasonable</p> |
|----------|----------|--|

| | | |
|------------------------|-----------------|--|
| | | <p>(i.e. did not reflect scientific understanding) at the time they were made.</p> <p><u>Type 1</u></p> <p>Decisions in respect of NISHL made on or after 1 January 1981 but before 7 January 1993: -</p> <p>Existing awards in respect of NISHL cannot be reduced or taken away.</p> <p>The "true" NISHL final assessment must be determined. An advisory assessment is also given to maintain the award at the existing level under the authority of Article 67(6) of the SPO.</p> <p>The award is maintained on a mark time basis unless a further claim, or deterioration claim for another condition is accepted and the resultant true assessment exceeds the advisory assessment.</p> <p><i>NISHL assessed at less than 20% can be aggregated with assessments for other conditions</i></p> <p><u>Type 2</u></p> <p>Decisions in respect of NISHL made on or after 7 January 1993 but before 22 October 1996</p> <p>As (i) above but assessments for NISHL of less than 20% cannot be aggregated with assessments for other conditions</p> |
| <p>As above</p> | <p>As above</p> | <p><u>CATEGORY C</u></p> <p>Decisions and applications for review of NISHL made before 1 March 1996 and still outstanding on 22 October 1996.</p> <p>The NISHL assessment which would have been applicable before the change of approach was introduced on 22 October 1996 (i.e. the advisory assessment for payment purposes) is determined together with the "true" NISHL assessment.</p> <p>As with Category B cases the level of award is based on the advisory assessment on a mark time basis. In this case maintenance of the award is under the authority of the Dispensing Instruments² with the</p> |

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|--|--|---|
| | | agreement of Treasury. A list of such awards needs to be presented to the Treasury Commissioners on an annual basis for finance purposes. |
|--|--|---|

2 Maintenance of award using the Dispensing Instruments is usually but not always the case. See para 34, example 7

| | | |
|-----------------|----------|--|
| As above | As above | <p><u>CATEGORY D (TWO SUB TYPES)</u></p> <p><u>Type 1</u></p> <p>Cases where before 22.10.96 a PAT has set an interim assessment which includes GD and where the period for that assessment does not lapse until some time after 22.10.96</p> <p>Whether or not the 20% cut off rule is applied and aggregation of NISHL with other conditions can be allowed depends on when the initial claim in respect of NISHL was made i.e. before or after 7.1.93.</p> <p>At the end of the interim period set by the PAT, a final true assessment can be given and form the basis of award. Category B arrangements apply - identify the "true" NISHL assessment and maintain level of the existing NISHL award under the authority of Article 67(6) and on a mark time basis.</p> <p><u>Type 2</u></p> <p>Claims where a NISHL assessment appeal has been lodged before 1.3.96 but where the appeal is heard after 22.10.96 and the PAT remove finality, increase the assessment from under 20% to 20% or over running from the date of the claim. Category B arrangements apply - identify the "true" NISHL assessment and maintain level of existing NISHL award under the authority of Article 67(6) and on a mark time basis.</p> |
| As above | As above | <p>NISHL TRANSITIONAL CASES - CLAIMS TO SUPPLEMENTARY ALLOWANCE(S)</p> <p>In payment or claimed before</p> <p>22 October 1996 or claimed after 22 October 1996 - See Annex B</p> |

Hearing loss at release - The hearing loss measured at release will include, not just hearing loss due to service noise but some loss due to ageing and the person's constitution . As a matter of preference for the war pensioner that overall level of hearing loss is included in the assessment of the accepted service related NISHL.

Appeals - Where on an appeal against the level of an existing NISHL assessment the "true" NISHL assessment has to be determined, a right of appeal lies against the notified existing NISHL assessment and also the "true" NISHL assessment and the claimant should be advised accordingly. The right of appeal against the notified assessment runs from the original date; the right of appeal against the true assessment runs from the day it was made. The claimant should be informed of his two appeal rights and the relevant time periods and can then decide if he wishes to withdraw any appeal which has been started. If he chooses to proceed with both, then there may be two assessment appeals running concurrently relating to different assessments and time periods. Both appeals should be presented to the Pensions Appeal Tribunal at the same time with an explanation of the circumstances, possibly with a request to combine both in one Statement of Case for one hearing.

ANNEX B

WAR PENSIONS - NOISE INDUCED SENSORINEURAL HEARING LOSS (NISHL) - TRANSITIONAL CASES

THE LAW

THE NAVAL, MILITARY AND AIR FORCES Etc (DISABLEMENT AND DEATH) SERVICE PENSIONS ORDER (SPO) [SI - 1983/883] ENTITLEMENT - ARTICLES 3,4 & 5 OF THE SPO

1. **Article 3** of the SPO provides for an award to be made in respect of disablement due to service in the Armed Forces. On all claims the onus is initially on the claimant to show disablement. **Articles 4 & 5** provide that disablement may be accepted as due to service provided it is **certified** that the disablement is due to an **injury** which is attributable to or aggravated by service. Under **Article 4**, if a claim is made within 7 years of termination of service, the initial onus is on the Secretary of State (SoS) to disprove the claim. Under **Article 5**, after 7 years the onus is on the claimant to prove her/his claim. Under both Articles the benefit of any reasonable doubt that disablement is due to service must be given to the claimant.

MEDICAL UNDERSTANDING AND CERTIFICATION - ARTICLE 1

2. **Article 1(4)** of the SPO requires the SoS to appoint a medical officer to certify any medical question involved in the determination of a claim. Certificates must reflect the current and correct medical understanding. The SoS normally acts on that advice but in making his assessment decision he has a discretion to maintain an award at a different level to that shown on the Certificate, notwithstanding Article 9(6), by virtue of Article 67(6).

3. The SoS also has a medical policy advisor on war pensions to advise on medical matters related to war pensions. If the advisor confirms a change in the medical understanding of the aetiology of a condition or of how it interacts with another condition, medical officers involved in the certification of medical questions need to follow that understanding.

ASSESSMENT - ARTICLE 9

4. **Article 9** of the SPO requires the level of disablement due to service to be assessed on a percentage basis by comparing the condition of the ex-serviceman with that of a normal healthy person of the same age and sex. If there is disablement due to more than one injury it requires a composite assessment of disablement to be made by reference to the combined effect of all injuries which are due to service.

5. **Article 9(2A)** prescribes that average hearing loss measured in both ears at less than 50 decibels (dB) at frequencies of 1, 2 and 3 kilohertz (kHz) shall be assessed at less than 20 per cent.

6. **Article 9(2B)** prescribes that neither Noise Induced Sensorineural Hearing Loss [NISHL] nor a related condition or symptom shall be taken into account in determining the overall level of disablement if the degree of disablement in respect of the NISHL alone is assessed at less than 20 per cent - this would include a psychiatric condition or any other condition which is consequent on NISHL.

AWARD - ARTICLE 8

No awards in respect of NISHL assessed at less than 20 per cent

7. Article 8 of the SPO provides that awards may be made in respect of disablement which is due to service. **Article 8(2A)**, however, provides that **no award** shall be made for NISHL, or a related condition or symptom, unless the NISHL alone is assessed as being at least 20 per cent or more.

TRANSITIONAL PROTECTION FOR CLAIMS MADE BEFORE 7 JANUARY 1993 - ARTICLE 4 OF THE SERVICE PENSIONS AMENDMENT ORDER 1992 (Statutory Instrument [SI] 1992/3208)

8. SI 1992/3208 provides that the new rules introduced under that amendment order from 7 January 1993 (the new Article 8(2A), 8(2B), 9(2A) and 9(2B) of the SPO - see paragraphs 4,5,6,7 and 8 above) should not apply to claims made before that date. This means that:

(a) awards on such claims may still include payment in respect of NISHL assessed at under 20%.

(b) under 20% assessments for NISHL on such claims may still form part of an overall combined assessment.

THE POWER TO REVIEW AND REVISE ASSESSMENTS AND AWARDS - ARTICLE 67

9. Article 67(1) provides that an **assessment** may be **reviewed** by the SoS at any time and on any ground.

10. Article 67(2) provides that an **award** may be **reviewed** if an assessment on which it was based has been revised.

11. Article 67(3) describes the circumstances in which a first tier decision, including an **assessment**, may be **revised** by the SoS to the detriment of the pensioner. These include where the existing assessment was made in consequence of ignorance of, or a mistake as to a material fact, or of a mistake as to the law or in the case of an interim assessment, where there has been a change in the degree of disablement.

12. Article 67(4) - provides that an award may be revised to the detriment of the pensioner (*i.e. an assessment reduced*) where the assessment on which it was based has been revised under Article 67(3).

13. Article 67(6) - provides that, despite the revision of an assessment or award to the detriment of the pensioner under articles 67(3) and (4), the SoS may **maintain the award** at a rate not exceeding that which was appropriate to the assessment applying immediately before it was revised. Such maintained awards may only be increased in line with normal uprating.

14. Article 67(6A) - provides that the SoS cannot review any PAT assessment or decision unless Article 67(7) applies.

15. Article 67(7) - provides that the SoS may review and revise any assessment made by the PAT where there is fresh medical evidence of deterioration.

16. The combined effect of Article 67(6A) and 67(7) is to prevent the SoS from replacing a PAT interim assessment with a new SoS interim assessment at a lower level. However, the position is different if

- a) The period for the PAT interim assessment has expired; and
- b) The condition has stabilised so that the MA can certify the level of assessment under Article 9(2)(d) and 9(3) and on a final basis.

In these circumstances the SoS is not "reviewing or revising" the PAT "interim" assessment but making a new "final". The distinction being that the SoS has changed the type of assessment from interim to final and is not therefore, bound by Article 1(4)(a) and can use the MA's certificate on the medical question of the level of final disablement.

17. For decisions made from 9 April 2001, Article 67 and SPO (1983) 76 of the Personal Injuries (Civilians) Scheme has been amended by introduction of a new paragraph 2A. This allows the Secretary of State to review and vary any assessment or decision made, given or upheld by a Pensions Appeals Tribunal at any time, if he is satisfied that there has been a relevant change of circumstances including improvement or deterioration of the accepted disablement.

THE POWER TO MAKE AWARDS OUTSIDE THE TERMS OF THE SPO - EX-MEMBERS OF THE ARMED FORCES - THE DISPENSING INSTRUMENTS

- 18. (i) The Dispensing Order in Council (19 December 1881) - Royal Navy
- (ii) The Dispensing Warrant (27 October 1884) - British Army
- (iii) The Dispensing Order by His Majesty (14 January 1922) - Royal Air Force

With the agreement of the Lord's Commissioners of the Treasury the Dispensing Instruments allow the SoS to award war pensions under circumstances in which they would not otherwise be awarded, provided that a list of the awards and grounds on which they were made is laid before Parliament on an annual basis.

Minor War Pension Schemes

19. The Dispensing Instruments cannot be applied to claims made under the following schemes - the Polish Scheme, the Mercantile Marine Scheme, the Naval Auxiliary Scheme, the Coastguards Scheme or the Civilians Scheme. Awards on claims made under these schemes which are outside of the provisions of the legislation can only be made on an extra-statutory basis with the agreement of Treasury. Again a yearly list of awards needs to be kept for budgetary purposes.

NISHL - PAST AND CURRENT MEDICAL UNDERSTANDING Past understanding - pre 22 October 1996

20. Until 22.10.96 the Department acted on the basis that service related NISHL could interact with hearing loss due to age to produce a result that was more than additive i.e. the overall hearing loss was more than the sum of the individual parts. Assessments and therefore, awards could include an increase in respect of this additional factor, on the basis that it was due to service, and could be further increased upon deterioration in overall hearing due to age or any other factor such as industrial environment.

Current understanding- 22 October 1996 and later

21. NISHL arises from an injury which causes permanent damage to the cochlear hair cells of the inner ear, for example, by exposure to noise from gunfire, or ship's engine noise. It has been accepted medically for many years that once an individual is removed from the source of noise no further damage to hearing arises directly due to that cause. The level of hearing loss due to that damage remains unchanged and as a consequence, the assessment of disablement in respect of that damage does not change with time.

22. Current medical evidence confirms that in the context of the war pension scheme, service related NISHL and other types of sensorineural hearing loss, such as that due to age or an industrial environment, are "no more than additive". They do not interact so as to cause an increase in the amount of hearing loss due to service, thus there is no additional factor due to service which attracts compensation as a result of that previously supposed interaction.

The assessment of NISHL at service release

23. By law an assessment can only include hearing loss which is **due to service**. Because the disablement due to service noise does not worsen on removal from noise, and because NISHL does not interact with hearing loss sustained after service to produce an overall level of hearing loss that is more than additive, it is logical and reasonable to consider the individual, for the purposes of assessing hearing loss due to service, in terms of his or her condition at service release. On claims made at any point after that date therefore, it will be necessary to consider carefully the evidence of the level of hearing loss due to service.

Evidence to inform assessment

24. **Since the mid 1970s** - the discharge medical examination carried out by MOD has included an audiological examination and an pure tone audiogram. This should normally form the basis of the NISHL assessment.

25. **Before the mid 1970s** - there will usually be no release audiogram, but the release medical will include a clinical assessment of ears and hearing. Expert audiological opinion has confirmed that this may be used to assess hearing loss.

26. If there is alternative evidence (for example, a record from shortly after discharge of the person seeking attention for a noise induced hearing loss, or from an in service examination [PULHEEMS] which shows a finding of hearing loss) which indicates, that in any particular case, the evidence available at the time of discharge is unreliable, the assessment may be based on that alternative evidence.

27. If there is no evidence at or around the date of discharge the assessment may be informed by the earliest available evidence ie usually a hospital audiogram or medical report.

Age Related Sensorineural Hearing Loss (SHL) - presbycusis - sustained before and during service

28. Age related SHL is present and progressive in all individuals. Constitution also determines hearing and both constitutional and age related loss is subject to wide variation amongst individuals. On enlistment every serviceman will have a degree of

age related hearing loss which will increase during service. At the same time he may be exposed to service noise. It is not possible, however, to precisely apportion hearing loss between different causes **operating at the same time**. As a matter of preference for war pensioners, where NISHL is accepted no attempt is made to offset losses due to age or any other cause sustained before or during service i.e. the assessment should include all sensorineural hearing loss present at service release irrespective of the cause.

Age related Sensorineural Hearing Loss (SHL) - presbycusis - sustained after service

29. Age related hearing loss or hearing loss due to other causes e.g. an industrial environment, occurring **after** service, on the other hand, must be considered separately - by definition they arise **after** exposure to service related noise has ceased. Article 9(2)(a) of the SPO requires a comparison to be made with a person of the same age and sex. Logically, therefore, post service SHL cannot be included in the assessment of service related NISHL.

The minor war pension schemes - Civilians, Mercantile Marine etc

30. The legislation for the minor the War Pension Schemes except for the Civilians Scheme applies the provisions of the SPO and will do so in respect of claims in respect of NISHL. That for the Civilians Scheme has equivalent provisions. The minor schemes are slightly more complicated in that awards are only available for disablement which is due to certain types of injury sustained during war time, but as far as claims in respect of NISHL are concerned the same principles apply. However, if a claim or an application for review of NISHL is received under the Mercantile Marine, Naval Auxiliary, Coastguards or the Civilians Scheme, refer it to WPP.

NISHL - HISTORY OF PAST CLAIMS

31. Until about 1980 the natural history of NISHL was not known. Technical advances in microscopy, however, enabled study of the histopathology of NISHL which confirmed that damage did not increase following removal from the injurious noise. This was reflected in Departmental guidance (a medical appendix) produced in 1981. The current view is therefore, that in the war pension context, hearing loss due to noise and age are no more than additive: in fact, the noise related hearing loss acts as a cushion against the subsequent effects of age related hearing loss.

32. The approach to deciding war pension claims did not keep up with these developments:

- * until January 1993 assessments were based on the whole of the hearing loss present at the date of claim, however long this was after service. This was unreasonable in the light of the understanding (1 January 1981) and Departmental guidance at the time that hearing loss due to noise did not deteriorate upon removal from the noise;

- * from January 1993 NISHL was separately identified and assessed and awards in respect of 20% disablement or less excluded but additions were made in respect of interaction with other forms of hearing loss, usually age related SHL, using the "greater disablement" principle (GD). Again, this was unreasonable in the light of the knowledge that losses due to noise and age were no more than additive.

33. Action was taken to bring practice into line with the current understanding from 22 October 1996. Although this had to be done to ensure that decisions on war pensions were within the law, it created ill feeling in parts of the ex-service world. The Royal British Legion, for example, asked for an independent review.

Independent Review - January 1998

34. When the present Government were elected in 1997, they were determined to be satisfied that the Department's approach to the assessment of hearing loss was seen to be in line with current scientific understanding. Baroness Hollis of Heigham (the then Minister for War Pensions) therefore, immediately ordered a review of the scientific evidence behind the Department's approach. That review was conducted by four leading experts in the subject. Its conclusion was that in relation to war pensions, hearing loss due to noise, such as that which may be experienced in service, does not increase after a person is removed from the noise or as subsequent age related hearing loss is added to it. The review team also recommended that the matter be looked at again in a year. As a consequence Baroness Hollis asked the war pensions medical policy adviser to report to her by the end of February 1999 on any relevant developments. The report was received on 26 February 1999.

Further review - February 1999

35. The further review by the medical policy adviser was conducted in a thorough and professional manner, by exhaustive search of the literature, by asking eminent experts for their advice on the existence of any relevant evidence - fourteen experts replied to our request - and by careful scrutiny and evaluation of the new evidence against the existing body of evidence.

36. The conclusion of the further review was that the new evidence, including papers published in peer reviewed scientific journals since January 1998, does not challenge the assumption that the combination of noise and age related hearing loss is no more than additive. This assumption has gained general acceptance and has formed the basis of an International Standard which has been in place for nearly 20 years, successfully providing the basis of medico-legal work internationally.

37. The outcome of the further review did not therefore, change the findings of the expert team in 1998. It means that war pension assessments and awards cannot be increased for service-related noise-induced hearing loss as overall hearing ability deteriorates with age, because that deterioration is not due to service

MINISTERIAL COMMITMENT

38. The failure of the method of assessment of NISHL to reflect contemporary medical evidence since 1 January 1981 means that there are awards in payment for NISHL which, if they are reviewed, will require special action to revise the decision to determine the true level of assessment. Ministers decided that there would be no attempt to seek out these cases for review. They would only be considered when they otherwise present themselves for review (e.g. because of a claim for a further condition, or an application for the review of the assessment of a condition other than the hearing loss).

39. Ministers made two commitments to ensure that no pensioner would be unreasonably disadvantaged by the need to revise decisions because of the failure to reflect contemporary medical evidence. These were that:

(a) no pensioner already in receipt of an award would suffer a reduction in pension or have that pension taken away following the correction of the erroneous decision; and that

(b) claims (both first claims and applications for review for deterioration) made before 1 March 1996, and still outstanding at the time action was taken to bring practice into line i.e 22.10.96, would be considered on the basis of the practice for handling claims introduced in January 1993.

[NB: These claims are identifiable by a yellow medical certificate on the awards file]

CATEGORIES OF CLAIMS FOR REVISION

40. To achieve the ministerial commitment requires use of the powers within Article 67 - especially Article 67(6), the Dispensing Instruments and extra-statutory provision. Cases divide into six categories:

Category A - Awards in respect of NISHL made before 1 January 1981 - and therefore within the law because they were reasonable at the time they were made

Category B - Awards in respect of NISHL made on or after 1 January 1981- outside the law because they were unreasonable in not reflecting medical understanding at the time

Type 1 - Awards in respect of NISHL made after 1 January 1981 but before 7 January 1993

Type 2- Awards in respect of NISHL made on or after 7 January 1993 but before 2 October 1996

Category C - Claims and applications for review in respect of NISHL made before 1 March 1996 and still outstanding at 22 October 1996

Category D - **Type 1** Claims in respect of NISHL where a Pensions Appeal Tribunal has decided on an interim NISHL assessment prior to 22 October 1996 but where the period allotted to that assessment expires after that date.

Type 2 Claims where a NISHL assessment appeal has been lodged before 1 March 1996 but where the appeal is heard after 22 October 1996 and the PAT remove finality, increase the assessment from under 20% to 20% or over to run from the date of the claim and where the period for the PAT interim assessment has elapsed or the claim comes for review for any other reason.

41. A detailed analysis of how these categories of claims or applications for review may be processed is at **ANNEX C** .

APPEALS

42. Where there is an appeal against the level of an existing NISHL assessment, the pre-appeal scrutiny establishes the "true" NISHL assessment. Legal advice is that a right of appeal lies both against the notified NISHL assessment and the "true" NISHL assessment, and that the claimant should be advised accordingly. The right of appeal against the notified assessment runs from the date notified. The right of appeal against the true assessment runs from the day it was made.

43. The claimant should be informed of his two appeal rights and the relevant time periods and can then decide if he wishes to withdraw any appeal which has been started. If he chooses to proceed with both, then there may be two assessment

appeals running concurrently relating to different assessments and time periods. Both appeals should be presented to the Pensions Appeal Tribunal at the same time with an explanation of the circumstances, possibly with a request to combine both in one Statement of Case for one hearing.

Past appeal cases - assessment upheld by PAT

44. The PAT Act does not allow an appeal by either party to the nominated Judge (High Court) on an assessment appeal decision of the PAT. Where a PAT has decided a final or interim assessment the SoS can only review and revise the assessment upwards i.e. when there is fresh medical evidence of deterioration in the accepted disablement. But the current understanding means that this will never be appropriate for NISHL reviews. Where the PAT has made an interim assessment the SoS can review and revise the assessment upwards where there is fresh medical evidence of a deterioration. But the current medical evidence on NISHL means that this will never be appropriate for a NISHL review. When the PAT interim assessment expires the MA can certify a new final assessment at a higher or reduced level, provided the condition has stabilised. Thus the effect of the current medical evidence is that it will always be possible to make a final assessment, but it is unlikely that this will be at a higher level than the interim assessment. The SoS's new final assessment decision will carry a fresh right of appeal.

NISHL - Period of award - Finality

45. Because service related NISHL cannot improve or deteriorate after termination of service any assessment of that condition on a new claim or on review will be a final assessment. A dissatisfied claimant will have a right of appeal against the decision as to the level of the assessment and the decision to make the assessment on a final basis.

46. If a claimant appeals against the SoS's decision on an assessment which includes NISHL and the PAT fail to take account of the current medical understanding on NISHL. The case should be forwarded as quickly as possible to WPP4.

Future appeal cases - PAT not informed of change in medical understanding on NISHL

47. If however, the PAT make a favourable decision on a case which had been awaiting a hearing date since 1996 (i.e. the case was not referred back to WPA for revisions following the change in approach and was heard under the old approach) the decision will have to be implemented and cleared. It should also be noted that where a claim for NISHL has been made before 1 March 1996 and is rejected, and subsequently goes to entitlement appeal. If the PAT allow entitlement, even after 22 October 1996, the subsequent assessment be according to category c principles.

SUPPLEMENTARY ALLOWANCES

Supplementary Allowance in payment before 22.10.96 or claim outstanding at that date

48. At 22.10.96 there was no assessment threshold for ALSO, a 20% threshold existed for the rest of the supplementary allowances payable with a war disablement pension, because as part of their entitlement conditions a continuing pension had to be in place. The lowest percentage disablement for which a pension can be paid is 20%. The assessment threshold for CAA was, and is, 80%. For NISHL claims where an allowance was in payment before **22 October 1996** or where a claim to them was outstanding at that date, the relevant assessment for the purposes of entitlement to

the allowance(s) is the assessment used for payment purposes, eg:-

Where:-

"True" NISHL assessment 30%

"Advisory (payment) assessment" 40%

the 40% assessment should be used. This is in line with the Ministerial promise that no pensioner would have his pension reduced or taken away. This rule does not apply to NISHL claims where a claim to a supplementary allowance has been made after 22 October 1996. After that date only the "true" assessment should be considered.

49. When a NISHL case falls for review the payment of the pension and a supplementary allowance may continue under the usual SPO provisions if the "true" NISHL assessment is sufficient to allow for this. But for cases covered by the Ministerial promise the following rules apply :-

(i) **Category A** - payment of both pension and the supplementary allowance(s) will continue as before, as no review can take place.

(ii) **Categories B & D** -

(a) if the "true" assessment is high enough and the other entitlement conditions for the supplementary allowance are met the allowance may be continued under the SPO without employing the discretion available under Article 67(6)

(b) if the "true" assessment is not high enough and the other entitlement conditions for the supplementary allowance are met, payment of a sum equivalent to the supplementary allowance in question may be continued under the discretion of Article 67(6).

(iii) **Category C** -

(a) if the "true" assessment is high enough and the other entitlement conditions for the supplementary allowance are met the allowance may be continued under the SPO.

(b) if the "true" assessment is not high enough and the other entitlement conditions for the allowance are met, payment of a sum equivalent to the supplementary allowance in question may be continued under the Dispensing Warrants.

Claims to Supplementary Allowances made after 22.10.96

50. The Ministerial promise that no pension in respect of NISHL would be reduced or taken away does not apply to these claims because they were not made before 22 October 1996. Any claim to a supplementary allowance on NISHL case made after 22 October 1996 must be determined on the basis of the "true" assessment. Please remember that legislative amendment (SI 1997/286) from 7 April 1997 introduced qualifying assessment threshold for UnSupp, ALSO and WPMS.

WPP June 2001

ANNEX C

WORKED EXAMPLES

CATEGORY A - PRE 1.1.1981 CASES

Certificates and awards made prior to 1 January 1981 were reasonable at the time that they were made. Therefore, the SoS cannot review and revise the assessment or the award to the detriment of the claimant.

1. Before 1 January 1981 the progress of NISHL following removal from noise was not firmly established and so it was considered reasonable, where NISHL was accepted, to make assessments and awards in respect of overall hearing loss from all causes present at the time of the determination of the claim. As a consequence, decisions to make an award in respect of NISHL before 1.1.81 were correct in the light of the medical understanding at the time. Assessments on those cases remain valid and cannot be altered unless subsequently, it is found that they were made as a result of an error as to a material fact or an error in law - Article 67(3)&(4) refers.

2. If on review another new condition is accepted as due to service or it is accepted that a condition other than NISHL has deteriorated, the new or revised assessment should simply be added to the original assessment.

EXAMPLE

| | |
|--|------------|
| (a) Pre-1.1.1981 assessment for hearing loss (including service related NISHL) | 60% |
| (b) assessment of new condition other than NISHL or application for a review of a condition other than NISHL | 10% |
| (c) New overall composite assessment (a + b) | 70% |
| (d) Award increased to reflect new assessment: | 70% |

Application for review in respect of deterioration of NISHL

3. The pensioner should be informed of the position on NISHL and why an application for a review in respect of deterioration of NISHL cannot succeed.

Appeal to the Pensions Appeal Tribunal [PAT]

4. A right of appeal to the Pensions Appeal Tribunal lies against the SoS's decision on the overall composite assessment (70%). If the claimant appeals, the Statement of Case should also include a detailed explanation of the medical understanding and why the assessment in respect of NISHL cannot be increased. If the PAT increases the NISHL assessment the case should be brought to the attention of WPP4.

CATEGORY B - 1.1.81 & LATER AWARDS

Existing awards in respect of NISHL made on or after 1 January 1981 - outside of the law - (excluding assessments of NISHL made on invaliding or shortly after leaving service) unreasonable because they did not reflect current medical understanding - level of award maintained

1. Decisions to make an award in respect of overall hearing loss which included service related NISHL on or after 1.1.1981, or to include an addition for GD in post 6 January 1993 cases, were not reasonable because they did not reflect the then

current medical understanding. Ministers have agreed, however, that no pensioner should suffer a reduction in pension or have their pension taken away as a result of the revision of an unreasonable decision on review. The power under Article 67(6) is to be used to maintain the level of the award.

Assessment of the level of disablement - Article 9 of the SPO

2. Where a claim for a new condition or an application for a review in respect of deterioration of NISHL or any other condition is accepted, and the previous assessment was not reasonable at the time it was made, the medical adviser has to certify the "true" assessment of disablement according to current medical understanding, including that in respect of NISHL. Article 67(3) of the SPO requires the SoS to reduce the existing NISHL assessment to produce a new **"true"** assessment in line with the MA's new opinion. He is able to do this because the existing assessment was unreasonable at the time it was made.

3. If a claim for a new condition/application for review of an existing condition other than NISHL is accepted, the assessment for that should be added to the **"true"** assessment for NISHL. The resulting composite assessment should be certified by the MA. To include an assessment in respect of non-service related NISHL [GD] within that composite assessment would be unreasonable in law.

Exercise of SoS's discretion to maintain the level of an award in respect of non-service related hearing loss using Article 67(6) of the SPO

4. Article 67(6) permits the continuance of the level of award on the basis of a previous assessment different from the new **"true"** assessment. In any case where Article 67(6) is used to maintain the level of award, the assessment used for that purpose should be separately identified. The use of Article 67(6) is solely a matter of discretion for the SoS **and this assessment cannot form part of the medical certificate**. The notification to the claimant should make clear that the SoS has decided that the rate of pension payable in respect of hearing loss is being maintained by the SoS using discretionary powers.

Claim for a new condition or application for review in respect of deterioration of an existing condition other than NISHL - no increase in the award

5. An assessment for a new condition or for deterioration of a condition other than NISHL must be added to the "true" assessment for NISHL not the previous "unreasonable" assessment protected under Article 67(6). Thus there may be instances where:

- (i) a pensioner who is already receiving a pension for NISHL will claim for a new condition or apply for the review of a condition other than NISHL; and
- (ii) that claim or review is accepted; but
- (iii) at the same time, as part of the review of the case, the NISHL assessment is revised to produce a **"true"** assessment; and
- (iv) the addition of the assessment for the new condition, or the revised assessment for a condition other than NISHL, to the **"true"** assessment for NISHL means that the award cannot be increased; because
- (v) the new composite assessment is the same or less than the **"previous unreasonable"** assessment for NISHL used to maintain the level of the award under Article 67(6)

Level of award maintained on a mark time basis

6. In deciding these claims, care is needed to confirm whether or not they are bound by the 1992 legislative amendment, ie, made before, on or after 7 January 1993 (SI

1992 3208). The award should be maintained under Article 67(6) at the rate appropriate to the assessment in place immediately before the decision was revised. Maintenance of the level of award under Article 67(6) is on a "mark time" basis. In effect, if the claimant makes successful claims to or applications for review of conditions other than NISHL, the addition of a new or revised assessment to the "true" NISHL assessment is used to abate the amount already in payment under Article 67(6). This composite "true" assessment may come to exceed the "previously unreasonable" advisory assessment used for payment purposes, in which case his pension may then be increased accordingly.

7. In some cases the revision of the NISHL assessment may produce a "true" NISHL assessment of under 20%. Further action will depend on when the claim was originally made, ie before or on or after 7 January 1993.

EXAMPLES

The following examples illustrate the effect on NISHL claims, of successful new claims/applications for reviews in respect of conditions other than NISHL.

EXAMPLE 1 - Award made on or after 1.1.81 - successful new claim/application for deterioration review in respect of a condition other than NISHL - "previous unreasonable" NISHL assessment 40% - "true" NISHL assessment 20% - No increase in award payable

| | |
|--|------------|
| (a) "True" assessment for service related NISHL alone | 20% |
| (b) Further condition accepted | 10% |
| (c) "True" composite assessment (a + b) | 30% |
| (d) "Unreasonable" assessment for hearing loss (including service related NISHL) made on or after 1 January 1981 | 40% |
| (e) Award maintained, using SoS power under Article 67(6), | 40% |

Note: (i) The true composite assessment (30%) should be certified by the MA
(ii) the assessment for payment purposes (40%) should be specified and SoS should confirm his decision to exercise his discretion under Article 67(6) to maintain the level of payment.

EXAMPLE 2- Award made on or after 1.1.81 - NISHL claim made before 7 January 1993 - successful new claim/application or review in respect of a condition other than NISHL - "previous unreasonable" NISHL assessment 40% - "True" NISHL assessment 6-14% - No increase in award payable

| | |
|--|-------------|
| (a) "True" assessment for service related NISHL alone 6-14% = | 10%* |
| (b) Further condition accepted - assessment | 20% |
| (c) "True" composite assessment (a + b) | 30% |
| (d) Original assessment for hearing loss including service related NISHL made on or after 1 January 1981 | 40% |
| (e) Award maintained, using SoS power under Article 67(6) | 40% |

Note: (i) * although assessment for NISHL is less than 20%, it can be included in a "true" combined assessment because of transitional powers applying to claims made before 7 January 1993 (Article 4 of SI 1992 No 3208)
(ii) The true composite assessment of 30% should be certified by the MA
(iii) the assessment for payment purposes (40%) should be specified and SoS should confirm his decision to exercise his discretion under Article 67(6) to maintain the level of payment

EXAMPLE 3 NISHL claim made on or after 7 January 1993 - successful new

claim/application for review in respect of a condition other than NISHL - "Original" NISHL assessment 40% - "True" NISHL assessment 6-14% - No increase in award payable

| | |
|---|--|
| (a) "True" assessment for service related NISHL alone 6-14% ie | (10%) but no award* (Article8(2A)SPO) |
| (b) Further condition accepted - assessment | 20% |
| (c) "True" composite assessment (b) | 20% see note 1 |
| (d) Assessment for hearing loss including service related NISHL/GD | 40% |
| (e) Award maintained, using SoS power under Article 67(6), at | 40% |

Note: (i) * as the claim/application for review in respect of NISHL was made on or after 7 January 1993 the NISHL assessment for less than 20% cannot be combined in any "true" composite assessment Article 9 (2B) SPO

(ii) The service related NISHL assessment and the true composite 20% assessment should be certified by the MA

(iii) the assessment for payment purpose (40%) should be specified and the SoS should confirm his decision to exercise this discretion under Article 67(6) to maintain the level of payment.

EXAMPLE 4 Award made on or after 1.1.81 - NISHL claim made before 7 January 1993 -successful new claim/application for review in respect of a condition other than NISHL - "Previous unreasonable" NISHL assessment 40% - "True" NISHL assessment 6-14% - Increase in award payable

| | |
|--|-------------|
| (a) "True" assessment for service related NISHL alone 6-14% = | 10%* |
| (b) Further condition accepted | 50% |
| (c) "True" composite assessment (a + b) | 60% |
| (d) Original assessment for hearing loss including service related NISHL made on or after 1 January 1981 | 40% |
| (e) Award payable at | 60% |

Note: (i) * although assessment for NISHL is less than 20%, it cannot be excluded from the "true" composite assessment because of transitional powers applying to claims made before

7 January 1993 (Article 4 of SI 1992 No 3208) ie before the introduction of the 20% assessment threshold cut off.

(ii) The true composite 60% assessment should be certified by the MA

(iii) in this case assessment and award are made under the SPO on the basis of the "true" assessment. Article 67(6) does not apply because the less than 20% NISHL assessment is protected by the transitional powers

EXAMPLE 5 NISHL claim made on or after 7 January 1993 successful new claim/application for review in respect of a condition other than NISHL - "previous unreasonable" NISHL assessment 40% - "True" NISHL assessment 6 -14% - Increase in award payable

| | |
|--|---------------------------|
| (a) "True" assessment for service related NISHL alone 6-14% = | (10%)but no award* |
|--|---------------------------|

(Article 8(2A) SPO)

| | |
|--|-----------------------|
| <i>(b) Further condition accepted</i> | 50% |
| <i>(c) "True" composite assessment (b)</i> | 50% see note 1 |
| <i>(d) Original 7.1.93 or later assessment for hearing loss including service related NISHL/GD</i> | 40% |
| (e) Award payable at | 50% |

Note (i) * as the claim /application for review in respect of NISHL was made on or after 7 January 1993 the NISHL assessment of less than 20% cannot be combined in any "true" composite assessment

(ii) The service related NISHL assessment and the true composite 20% assessment should be certified by the MA

(iii) in this case Article 67(6) does not apply

Application for review in respect of deterioration of NISHL

8. An application for a review in respect of deterioration of NISHL (hearing loss) should be rejected by the MA and the original assessment maintained. The pensioner should be given a full explanation of why his award in respect of NISHL cannot be increased.

Appeal to the Pensions Appeal Tribunal [PAT]

9. A right of appeal to the Pensions Appeal Tribunal will lie against the SoS's decision on the existing and the new "true" NISHL assessment. If the claimant appeals, the Statement of Case should include a detailed explanation of the medical understanding, why the assessment in respect of NISHL cannot be increased, and of the composition of the current assessment. It should also be made clear that part of the current award is being paid using discretionary powers.

CATEGORY C

New claims to, or applications for review of, NISHL made before 1 March 1996 but still uncleared on 22.10.96

1. Once the need for a change in approach to reflect current medical understanding had been identified, action on existing new claims and applications for review was suspended with effect from 22 October 1996. These included claims outstanding from before 1 March 1996 - the date identified at the time as the date from which it would be appropriate to apply the new approach

Why "1 March 1996" was chosen

2. The choice of "1 March 1996" was based on articles published in February 1996. It later emerged that those articles did no more than reaffirm previous findings. The "1 March 1996" is therefore, no longer regarded as the date from which the new approach should have been applied - it should have applied earlier. The "1 March 1996" remains, however, the key date for the treatment of cases in Category C for the following reasons.

3. In order not to disadvantage individuals simply because of the time taken to decide their claims Ministers decided to action all remaining claims or applications for review from before 1 March 1996, and still uncleared on 22 October 1996, under the old approach (ie including GD). Any part of the assessment of disablement due to non-service related hearing loss cannot, however, be awarded under the SPO, and is paid, with Treasury approval, under the authority of the Dispensing Instruments [DIs] or on an extra-statutory basis for the minor schemes. See paragraphs 11,12 and 13 of the statement dealing with the law.

Category C - Types of case

4. Category C cases are therefore a tightly defined and limited group. This category only applies to claims/applications for review made before 1 March 1996 and still uncleared when action was suspended on 22 October 1996. The following are the variations which may arise depending on the nature of the outstanding claim:

- (1) A new claim solely in respect of service related NISHL, no other post-service hearing loss involved (e.g. claim made on invaliding or shortly thereafter)
- (2) A new claim in respect of service related NISHL, other post-service hearing loss present
- (3) An application for a review of an award in respect of service related NISHL - initial award made and implemented before 1 January 1981
- (4) An application for a review of an award in respect of service related NISHL - initial award made and implemented on or after 1 January 1981
- (5) An application for a review of an award in respect of service related NISHL - initial award made and implemented before 1 January 1981 but reviewed and increased on or after that date.
- (6) An application for a review of an award in respect of service related NISHL and another unrelated condition plus a claim for a new condition - initial award made and implemented before 1981 but reviewed and increased on or after 1 January 1981 in respect of NISHL and the existing condition.

EXAMPLE 1 Outstanding claim is a new claim solely in respect of service related NISHL no other hearing loss involved - e.g. man invalided from service on the basis of NISHL

1. Where there is a new claim solely in respect of service related NISHL an assessment can be made of that condition in the normal way. If the assessment is less than 20% no award can be made. The question of interaction with another type of hearing loss does not arise so there is no need to apply the DIs because the award (if any) is fully covered by the SPO.

| | |
|---|------------|
| (a) "True" assessment of NISHL | 20% |
| (b) Service related NISHL assessment | 20% |
| (c) Award under the SPO | 20% |

2. With the notification of the decision on assessment the claimant should be given an explanation of the medical understanding of NISHL and why his assessment in respect of service related NISHL may not be increased in respect of future deterioration in overall hearing.

Appeals

3. A right of appeal will lie against the SoS decision on the level of the true assessment i.e. (20%) of NISHL in the normal way.

Subsequent claims in respect of conditions other than NISHL

4. Where subsequently, there is a successful claim in respect of a new condition the assessment for that condition should be added to the assessment for NISHL (i.e. the "true" assessment) and an award at the rate appropriate to that combined assessment should be made.

EXAMPLE 2 Outstanding claim is a new claim in respect of service related NISHL, other post-service hearing loss present

1. Where there is a new claim in respect of service related NISHL, but overall

hearing loss includes other post-service hearing loss e.g. due to age. A **"true"** assessment must be made of the service related NISHL which must be certified by the MA. If overall hearing loss is greater than the **"true"** NISHL assessment, the DIs should be used to give an increase in the award equivalent to that which would have been given under the approach in place before 1 March 1996 (i.e. by application of the GD principle)

2. The basis under which payment is made will depend on the **"true"** assessment for NISHL. If the **"true"** assessment is 20% or more, part of the payment will be under the SPO, and part under the DIs:

| | |
|---|------------|
| (a) Service related NISHL - "true" assessment (award under the SPO) | 20% |
| (b) Non service related hearing loss [GD] (award under the Dispensing Instruments) | 20% |
| (c) Award (a + b) | 40% |

if the **"true"** assessment is under 20%, no award can be made under the SPO, and the whole of the payment will be under the DIs e.g. :-

| | |
|---|------------|
| (a) Service related NISHL "true" assessment (6-14%) (award under Dispensing Instruments) | 10% |
| (b) Non service related hearing loss [GD] (award under Dispensing Instruments) | 20% |
| (c) Award (a + b i.e. all under DIs) | 30% |

3. The assessment in respect of the non-service related hearing loss [**GD**] should be specified and the claim noted to the effect that the SoS has agreed an award in respect of non-service related NISHL [**GD**] under the authority of the DIs. A record of payments under the DIs should be kept to ensure that payments are reported to Parliament as required under the DIs.

4. The **"true"** assessment in respect of service related NISHL should be on the assessment notification. With the notification of the assessment the claimant should be given an explanation of the medical understanding of NISHL and why, in his case, discretion has been used to award an increase under the DIs

Appeals

5. A right of appeal will lie against the SoS decision on the level of the **"true"** assessment of service related NISHL. If the claimant appeals against that decision the Statement of Case should include a detailed explanation of the current medical understanding on NISHL and indicate that the award in payment also includes an element paid using discretionary powers

Subsequent claims in respect of conditions other than NISHL

6. The part of the award made under the DIs is on a mark time basis (similar to the use of Article 67(6) in Category B cases, and will be offset against any future additions for further conditions. So, where subsequently there is a successful claim in respect of a new condition the assessment for that condition should be added to the **"true"** assessment for NISHL if that is assessed at 20% or more to give the new **"true"** composite assessment which should be certified by the MA.

If the new addition results in a **"true"** composite assessment equal to or above the existing level of award, the award becomes wholly payable under the SPO: the Dispensing Instruments are no longer required and there is no need for the award to be included in the annual list of DI awards laid before Parliament. If the new **"true"** combined assessment is still lower than the existing award the DIs are still needed to maintain the payment at that rate and the reduced award will need to be included in the list of DI awards.

If the true NISHL assessment is assessed at less than 20% it cannot be combined

with an assessment for another condition but can be offset against the assessment used for payment purposes.

EXAMPLE 3 Outstanding application for a review of an award in respect of service related NISHL - initial award made and implemented before 1.1. 1981

1. The application for a review in respect of deterioration of service related NISHL should be accepted. The SoS cannot revise the original assessment in respect of service and non-service related NISHL because he has no power to reduce an assessment to the detriment of the pensioner under Article 67(3) unless it is wrong in fact or law (i.e. to this extent it is a Category A case). If overall hearing loss has deteriorated since the previous assessment, the DIs should be used to give an increase in award equivalent of that which would have been given under the approach in place before 1 March 1996.

| | |
|---|------------|
| (a) Assessment of service related NISHL made before 1 January 1981(award under the SPO) | 30% |
| (b) Award for deterioration under the DIs | 20% |
| (c) Award (a + b) | 50% |

2. The assessment in respect of the non-service related NISHL [**GD**] should be specified and the claim noted to the effect that the SoS has agreed an award in respect of non-service related hearing loss [**GD**] under the authority of the DIs. A record of payments under the DIs should be kept to ensure that payments are reported to Parliament as required under the DIs.

3. Although the amount of pension payable has increased to that appropriate to a 50% assessment, the assessment notified to the pensioner should be the "**Pre - 1981**" assessment i.e. 30%. The pensioner should be given information on the medical understanding of NISHL, told that the increase in his award in respect of deterioration of NISHL has been made using discretionary powers and that there is no right of appeal against the decision to increase his award to that level.

Appeals

4. A right of appeal lies against the SoS decision on review. In this case that will be against the pre 1.1.81 30% assessment. If the claimant lodges an appeal to the PAT, the Statement of Case must include a full account of the claims but making it clear that the matter before the PAT is the pre 1.1.81 30% assessment of the NISHL. It should also indicate that the award in payment includes an additional element paid using discretionary powers

Subsequent claims in respect of conditions other than NISHL

5. The level of the rate of pension payable will be maintained until such time as the pre 1 January 1981 assessment (i.e. the pre 1981 assessment) can be increased above that rate by the addition of assessments in respect of conditions other than NISHL. (See example 2, paragraph 6 above)

EXAMPLE 4 Outstanding application for a review of an award in respect of service related NISHL - initial award made on or after 1 January 1981

1. The application for a review in respect of deterioration of service related NISHL should be accepted. The existing assessment for hearing loss may be subject of revision (i.e. as in a category B case). The medical adviser should give an

assessment of disablement according to current medical understanding, including in respect of NISHL. Because the original decision was unreasonable at the time it was made Article 67(3) of the SPO requires the SoS to reduce the existing assessment to produce a new "**true**" assessment in line with the MA's new opinion. The level of the existing award, however, will be protected using Article 67(6)

2. If overall hearing loss has deteriorated since the previous assessment, the Dispensing Instruments should be used to give an increase in award equivalent to that which would have been given under the approach in place before 1 March 1996 (i.e. by the application of GD). The award in respect of non-service related hearing loss allowed under Article 67(6) should be noted separately together with that allowed under the Dispensing Instruments.

| | |
|---|------------|
| (a) "True" assessment in respect of service related NISHL6-14% | 10% |
| (b) 1.1.81 or later award for NISHL | 30% |
| (c) Assessment in respect of non service related hearing loss (under the authority of Article 67(6)) (b - a) | 20% |
| (d) Assessment in respect of non-service related hearing loss [GD] (under the authority of the Dispensing Instruments) | 10% |
| (e) Award (a + c + d) | 40% |

3. The level of the rate of pension payable (40% rate) will be maintained until such time as the "**true**" assessment can be increased above that rate by the addition of assessments in respect of conditions other than NISHL.

4. The assessment in respect of the non-service related NISHL should be specified and the claim noted to the effect that the SoS has agreed to maintain an award in respect of non-service related hearing loss under the authority of Article 67(6) **and** has also agreed an award in respect of non-service related hearing loss [**GD**] under the authority of the DIs. A record of payments under the DIs should be kept to ensure that payments are reported to Parliament as required under the DIs.

Appeals

5. The right of appeal lies against the original assessment and the "**true**" NISHL assessment (i.e. 6-14%). If such an appeal is lodged, the Statement of Case must include a full account of the claim and the reasons for decision. However, the Statement of Case should note that the award in payment includes an element paid using discretionary powers.

Subsequent claims in respect of conditions other than NISHL

6. The level of the rate of pension payable will be maintained using Article 67(6) and/or the DIs until such time as the "**true**" assessment can be increased above that rate by the addition of assessments in respect of conditions other than NISHL. (See example 2, paragraph 6 above)

EXAMPLE 5 Outstanding application for a review of an award in respect of service related NISHL - initial award made and implemented before 1.1.1981 - subsequently reviewed and increased on or after 1.1.1981.

1. The application for a review in respect of deterioration of service related hearing loss should be accepted. The SoS cannot revise the original pre-1981 assessment in respect of overall hearing loss including service related NISHL because he has no power to reduce an assessment to the detriment of the pensioner under Article 67(3)

unless it is wrong in fact or law.

2. The decision to increase the NISHL assessment on or after 1 January 1981 may be subject of revision (i.e. as in a category B case). The level of the existing award, however, will be protected using Article 67(6).

3. If overall hearing loss has deteriorated further since the last assessment, the DIs should be used to give an increase in award equivalent to that which would have been given under the approach in place before 1 March 1996.

4. The assessment in respect of non-service related NISHL awarded on or after 1981 and allowed under Article 67(6) should be noted separately together with that allowed for GD under the Dispensing Instruments plus the overall assessment for payment purposes.

| | |
|---|------------|
| <i>(a) Pre 1 January 1981 assessment in respect of service related NISHL</i> | 30% |
| <i>(b) Increase in assessment in respect of non service related hearing loss made on or after 1 January 1981 Article 67(6)</i> | 10% |
| <i>(c) Award, on review, in respect of service NISHL and non -service related hearing loss (a+b)</i> | 40% |
| <i>(d) Assessment of further increase in respect of non-service related hearing loss [GD] (under the authority of the Dispensing Instruments)</i> | 10% |
| <i>(e) Award (a + b (now under Art 67(6)) + d under DIs)</i> | 50% |

5. The level of the rate of pension payable (50% rate) will be maintained until such time as the pre 1.1.81 30% assessment can be increased above that rate by the addition of assessments in respect of conditions other than NISHL.

6. The assessments in respect of the non-service related NISHL [GD] should be specified and the claim noted to the effect that the SoS has agreed to maintain an award in respect of non-service related hearing loss under the authority of Article 67(6) and has agreed an award in respect of non-service related hearing loss [GD] under the authority of the DIs. A record of payments under the DIs should be kept to ensure that payments are reported to Parliament as required under the DIs.

7. The assessment notified to the pensioner should be the pre 1 January 1981 30% assessment. The pensioner should also be given information on the medical understanding of NISHL, told that his award has been increased by making use of discretionary powers.

Appeals

8. A right of appeal lies against the SoS decision on review. In this case that will be against the maintained Pre-1981 assessment (30%) . If the claimant lodges an appeal to the PAT, the Statement of Case must include a full account of the claim.

Subsequent claims in respect of conditions other than NISHL

9. The level of the rate of pension payable will be maintained using Article 67(6) and/or the Dispensing Instruments until such time as the pre 1 January 1981 30%

assessment can be increased above that rate by the addition of assessments in respect of conditions other than NISHL. (See example 2, paragraph 6 above)

EXAMPLE 6 - Outstanding application for a deterioration review of an award in respect of service related NISHL and another unrelated condition plus a claim for new condition - initial award made and implemented before 1.1.1981 but subsequently reviewed and increased on or after 1.1.1981 in respect of NISHL and the first other condition

1. The application for a review in respect of deterioration of service related NISHL should be accepted. The SoS cannot revise the original pre 1.1.1981 assessment in respect of overall hearing loss including service related NISHL because he has no power to reduce an assessment to the detriment of the pensioner under Article 67(3) unless it is wrong in fact or law.

2. The decision to increase the NISHL assessment on or after 1 January 1981 may be subject of revision (i.e. as in a category B case). The existing award, however, will be protected using Article 67(6). Article 67(6) only gives discretion to maintain an award at the level appropriate to the assessment in place immediately before the decision was reversed. The "protected assessment" does not therefore include the new condition.

3. If overall hearing loss has deteriorated further since the last assessment, the DIs should be used to give an increase in award equivalent to that which would have been given under the approach in place before 1 March 1996.

4. Assessments for conditions other than NISHL will form part of the "true" composite assessment.

5. Any addition to be made under the DIs in respect of the outstanding application for deterioration of hearing loss must be added **after** the true/protected award has been established.

6. The assessment in respect of non-service related NISHL awarded after 1981 and allowed under Article 67(6) should be noted separately together with that allowed for GD under the DIs plus the overall assessment for payment purposes.

EXAMPLE 7 - This example is complicated, it is based on the principle that the use of Article 67(6) requires us to immediately offset any level of payment maintained under that article in respect of conditions other than NISHL which are claimed and awarded at the same time (the arm in this example) or later provided that offset does not affect the Ministerial promise. The increase under the Dispensing Instruments, however, can be added on top of the assessment, and would only be offset against the next condition that came along. The rule is to offset against the Article 67(6) award first.

Example 7 is made up of the following steps

Pre 1.1.81 - Rules

- (a) 1.1.79 - Claim in respect of NISHL - untouchable 20% assessment
- (b) 1.1.79 - Claim in respect of leg injury - untouchable 20% assessment

Post 1.1.81 - Pre 7.1.93 Rules

(c) 20.1.92 - Application for review in respect of deterioration of NISHL - NISHL assessment increased by 6-14% - aggregated with previous assessments but unreasonable in the light of current medical understanding - level of payment now maintained under Article 67(6).

Post 1.1.81 & Post 7.1.93 Rules

(d) 5.5.93 - Application for review in respect of deterioration of NISHL - NISHL assessment increased by 6-14% - erroneously aggregated with previous assessments under GD rules also unreasonable in the light of current medical understanding - level of payment now maintained under Article 67(6) .

(e) 5.5.93 - Application for review in respect of deterioration of leg assessment increased by 6-14%.

(f) 2.2.96 - Application for review in respect of deterioration of NISHL - outstanding on 22.10.96 - considered under DIs NISHL assessment increased by 20%

Post 22.10.96 Rules

(g) 12.11.97- Claim in respect of arm - 6-14% assessment - increases the true assessment by 10% but has to be offset against NISHL payments assessments now maintained under Article 67(6) and similarly any subsequent assessment for a new condition would have to be offset until the level of payment can be based on the true assessment without maintenance under either Article 67(6) or the DIs.

The following table sets out the steps in certification. The first column records the increase in assessment, the second column records the composite assessment The third column records the "true assessment" on review in 1997 and the final column records the assessment for payment purposes.

| Action | Contemporary Certified Assessment | | On review post – 22.10.96 True assessment | Assessment - payment purposes |
|---|-----------------------------------|-------------------------|---|-------------------------------|
| (a) NISHL claim made on 1.1.79 - Pre 1.1.81 NISHL assessment - transitional protection applies - assessment not affected by 1993 20% assessment cut off (7.01.93) nor the change of method of assessment of NISHL from 22.10.96 | 20% | Composite 20% | 20% | 20% |
| (b) Claim in respect of leg also made on 1.1.79 - Pre 1.1.81 assessment for leg (SPO) can be aggregated with existing NISHL assessment of 20% at (a) above | 20% | 40% | 40% | 40% |

| | | | | |
|---|-------|-----|-----|-----|
| <p>(c) Application for review of NISHL made on 20.01.92. NISHL assessment increased by 6-14% i.e. before 7.01.93 but after 1.01.81 - unreasonable at the time that it was made - for payment purposes can be aggregated with assessment at (a) & (b) - level of payment maintained under Article 67(6)</p> | 10% | 50% | 40% | 50% |
| <p>(d) Application for review of NISHL made on 5.5.93 i.e. after 7.01.93 and 1.01.81 but before 1.03.96 - unreasonable at the time that it was made - but NISHL assessment increased by 6-14% at that time on the basis of GD (i.e. when it was felt that there was an interaction with age related hearing loss) - level of payment maintained under the authority of Article 67(6).</p> | 10% | 60% | 40% | 60% |
| <p>(e) Application for review in respect of leg (SPO) made on 5.5.93 - assessment increased by 6-14%</p> | 10% | 70% | 50% | 70% |
| <p>(f) Application for a review of NISHL made on 2.02.96 but not considered until 12.11.96 (i.e. after the announcement of the change to the method of assessment of NISHL) - "true" NISHL assessment identified and NISHL assessment for payment purposes increased by 20% under the authority of the DIs - cannot offset at this point.</p> | 20% | * | 50% | 90% |
| <p>(g) Increase in assessment for arm (SPO) of 6-14% on 12.11.97 - can be added to the true assessment but for payment purposes must be offset against the 10% NISHL assessment at (d) above maintained under</p> | 6-14% | * | 60% | 90% |

| | | | | |
|---------------|--|--|--|--|
| Article 67(6) | | | | |
|---------------|--|--|--|--|

[* Theoretically for the purposes of the table the composite assessment at "f" would be "90%" and that at "g" would be "100%" . However, we felt it best to leave these boxes blank because in practice, certificates by that time would have reflected current medical understanding and should have contained the true assessment of "50%" and "60%" with an advisory assessment of "90%".]

THE MINISTERIAL PROMISE WAS THAT NO PENSIONER WOULD SUFFER A REDUCTION IN PENSION OR HAVE HIS PENSION TAKEN AWAY AS A RESULT OF THE CHANGE TO THE METHOD OF ASSESSMENT OF NISHL INTRODUCED FROM 22.10.96 - THUS IN THIS CASE UNLESS THE LEG IMPROVES SO THAT THE ASSESSMENT FOR IT CAN BE REDUCED THE ASSESSMENT FOR PAYMENT PURPOSES CAN NEVER BE REDUCED BELOW 70%

7. The level of the rate of pension payable will be maintained until such time as the **"true composite assessment"** i.e. 60% assessment can be increased above that rate by the addition of assessments in respect of conditions other than NISHL.

8. The assessment in respect of the non-service related NISHL should be specified and the claim noted to the effect that the SoS has agreed to maintain an award in respect of non-service related hearing loss under the authority of Article 67(6) **and** has agreed an award in respect of non-service related hearing loss [**GD**] under the authority of the DIs. A record of payments under the DIs should be kept to ensure that payments are reported to Parliament as required under the DIs.

9. The assessment notified to the pensioner should be the true composite assessment (60%). The pensioner should also be given information on the medical understanding of NISHL, told that his award has been increased by making use of discretionary powers.

Appeals

10. The right of appeal will lie against the new "true" composite assessment (60%). If the claimant lodges an appeal to the PAT, the Statement of Case must include a full account of the claim, including the use of Article 67(6) and the DIs.

Subsequent claims in respect of conditions other than NISHL

11. The level of the rate of pension payable will be maintained using Article 67(6) and/or the DIs until such time as the **"true"** assessment can be increased above that rate by the addition of assessments in respect of conditions other than NISHL.

CATEGORY D

TYPE 1 - Existing awards in respect of NISHL where a Pensions Appeal Tribunal (PAT) has confirmed an interim assessment for a period which commences before 22 October 1996 but ceases after that date. Although these awards did not reflect current medical understanding, the level of award is maintained.

1. PAT interim NISHL assessments determined before 22 October 1996 but expiring after that date did not reflect contemporary medical understanding. Ministers have agreed, however, that no pensioner should suffer a reduction in pension or have their pension taken away as a result of that failure. As with Category "B" cases

therefore, the power under Article 67(6) is used to maintain the level of the award for Category "D" cases.

2. As medical and scientific evidence confirms that the level of service related NISHL cannot change once a person has-left service, all assessments for service related NISHL should be made on a final basis. When the PAT interim assessment expires the MA will therefore be able to confirm that the condition has stabilised and will neither improve nor deteriorate in the future. The MA can then provide a certificate of the "true" final assessment under Article 9(2) and 9(3).

Re-assessment - application of Articles 67(3), 67(6) & 67(7)

3. Instead of then using the certified true assessment as the basis of the award, SoS will exercise his discretion (Article 67(a) to maintain the award at the rate in payment immediately before the decision was revised the advisory assessment used for payment purposes being separately identified. The use of Article 67(6) is solely a matter of discretion for the SoS **and this assessment does not form part of the medical certificate**. The notification to the claimant should make clear that the SoS has decided that the rate of pension payable in respect of hearing loss is being maintained by the SoS using discretionary powers.

4. Maintenance of the level of award under Article 67(6) is on a "mark time" basis. If the claimant makes successful claims to, or applications for review, of conditions other than NISHL, the addition of any such new or revised assessment to the "true" NISHL assessment is used to abate the award paid under Article 67(6). The composite "true" assessment may come to exceed the "maintained assessment" used for payment purposes, in which case his pension may then be increased accordingly

EXAMPLE illustrates the effect on a PAT interim NISHL assessment which has expired after 22.10.96 Award made before 22.10.96 - claimant successfully appeals against the level of assessment before that date - PAT increase assessment on an interim basis for a set period due to expire after 22.10.96 - PAT interim NISHL assessment of 40% reduced on reassessment to a true final NISHL assessment of 6-14%

- (a) "PAT interim NISHL assessment 40%
decision on assessment made on 5 May 1996
i.e. before 22 October 1996 for 3 years
- (b) "True" final assessment for service related NISHL made
on 6 May 1999 i.e. after 22 October 1996 6-14%
- (c) **Award maintained using SoS power under Article 67(6) 40%**

Note (i) The true NISHL final assessment (6-14%) should be certified by the MA

(ii) The assessment for payment purposes (40%) should be specified and the SoS should confirm his decision to exercise his discretion under Article 67(6)"

There are other variations on this basic example which may arise and are the same as those listed for Category "B" cases at Annex B, the only difference being that in those cases the SoS made the original decision as opposed to the PAT. In those circumstances the Category B rules should be applied.

TYPE 2 - Existing awards in respect of NISHL where a NISHL assessment appeal is lodged before 1 March 1996 but is heard by the PAT after 22 October 1996. Where the PAT change the type of assessment from final to interim, increase the

assessment from under 20% to 20% or over and backdate the award to the date of the claim. Although these PAT awards do not reflect current medical understanding, one review the level of existing award is maintained.

1. Existing awards in respect of NISHL where a NISHL assessment appeal is lodged before 1.3.96 but is heard by the PAT after 22.10.96. Where the PAT change the type of assessment from "final" to interim, increase the assessment from under 20% to 20% or over and to run from the date of the claim. Ministers have agreed, however, that no pensioner should suffer a reduction in pension or have their pension taken away as a result of the need to reflect contemporary medical understanding. As with Category "B" cases therefore, when the claim comes up for review at the end of the PAT interim assessment period the power under Article 67(6) is used to maintain the level of award for these Category "D "type 2 cases.

2. As medical and scientific evidence confirms that the level of service related NISHL cannot change once a person had left service, all assessments for service related NISHL should be made on a final basis. When the PAT interim assessment expires the MA will therefore be able to confirm that the condition under consideration has stabilised and will neither improve nor deteriorate in the future. The MA can then provide a certificate on the level of final assessment under Article 9(2) and 9(3)

Re-assessment - application of Articles 67(3), 67(6) & 67(7)

3. Instead of using the certified true assessment as the basis of the award, the SoS will exercise his discretion to maintain the award in place immediately before the decision was revised, the advisory assessment used for that purpose being separately identified.

4. The use of Article 67(6) is solely a matter of discretion for the SoS **and this assessment does not form part of the medical certificate**. The notification to the claimant should make clear that the SoS has decided that the rate of pension payable in respect of hearing loss is being maintained by the SoS using discretionary powers.

5. If a claimant makes successful claims to, or applications for review, of conditions other than NISHL, the addition of any such new or revised assessment to the "true" NISHL assessment is used to abate the award paid under Article 67(6). The composite "**true**" assessment may come to exceed the "**maintained assessment**" used for payment purposes, in which case his pension may then be increased accordingly

EXAMPLE

(a) 23 Dec 1993 Claim in respect NISHL, NISHL assessed at 6-14% indeterminate duration and on a final basis.

(b) 31 Oct 94 NISHL assessment appeal lodged against the SoS's decision as to the level of the NISHL assessment.

(c) 1 Dec 97 Appeal heard - PAT increase NISHL assessment 20% to 20% for an indeterminate duration on an interim basis for a limited period.

(d) 7 Oct.99 On review at the end of the PAT interim assessment period the assessment is reduced to 6-14% and the type of assessment is changed from an interim to a final basis. (i.e the true NISHL assessment) 6-14%

(e) Award maintained using SoS power under Article 67(6) 20%

Note (i) The true NISHL final assessment (6-14%) should be certified by the MA

(ii) The assessment for payment purposes (20%) should be specified and the SoS should confirm his decision to exercise his discretion under Article 67(6)"

There are other variations on this basic example which may arise and are the same as those listed for Category "B" cases at Annex B, the only difference being that in those cases the SoS made the original decision as opposed to the PAT. In those circumstances the Category B rules should be applied.

**Extract from High Court Judgement
Lord Justice McCowan in
The Queen v The Department of Social Security
ex parte John Henry Edwards Date: 10th July 1992**

If the right question is to ask when the present view of the possible causes of schizophrenia became the generally accepted view of medical practitioners, then it is impossible to say that the Secretary of State was not entitled to fix on 1st February 1980 as the appropriate date. I am in no doubt, however, that it is the wrong question and that it involves a misunderstanding of the meaning of Article 5(4). In considering that Article, the word "reliable" cannot, in my judgement, have been intended to mean "convincing". At most it can be construed as "not fanciful". But in fact I doubt whether the word adds anything to the sentence. The real question is: does the evidence raise a reasonable doubt in the mind of the Secretary of State? If he finds the evidence unreliable, it obviously will not raise a reasonable doubt in his mind.

I see no reason why, on the material put before him, the Secretary of State should not be entitled to hold that in 1968, the time of publication of the Brown and Birley study, what has now come to be the generally accepted view was a mere hypothesis based on a limited study which would not have created a "reasonable doubt" within the terms of Article 5(4). The stage by which it became sufficiently supported to raise such a doubt in his mind is a matter for the Secretary of State. Accepting, however, that the shift of opinion was a gradual process and that by February 1980 it was the generally accepted view, there must have been an earlier stage when, if asked to consider the matter, he would have found that there was a "reasonable doubt" and failure on his part to do so would have been challengeable on Wednesbury grounds. There are, in other words, in my judgement, three stages: no reasonable doubt, reasonable doubt, and consensus.

It follows, in my judgement, that the Secretary of State's decision that 1st February 1980, being the date at which consensus was reached, is the earliest date at which he can hold that there was a reasonable doubt, is flawed and cannot stand.

Medical adjudication guidance on [schizophrenia continued](#)

[back to Medical Adjudication Contents](#)

MEDICAL APPENDIX

Schizophrenia, schizoaffective, delusional and acute psychotic disorders

1 Various attempts have been made to formally classify psychiatric disorders, the two major systems being:

1.1 The ICD-10 Classification of Mental and Behavioural Disorders (World Health Organisation, Geneva) is part of the 10th edition of the International Classification of Disease. This appendix follows the common abbreviation of **ICD-10**. It is the international system used by the majority of clinical psychiatrists in Great Britain.

1.2 The Diagnostic and Statistical Manual of Mental Disorders (fourth edition) (American Psychiatric Association Washington DC). References to it in this appendix follow the common abbreviation of **DSM-IV**. It is a system devised mainly by and for workers in the USA, however UK psychiatrists were consulted in its formulation.

2 The two systems above have been in existence for many years but only in their current editions have they been closely comparable.

3 This appendix discusses the clinical features and aetiology of schizophrenia, schizoaffective, delusional and acute psychotic disorders. It is generally based on the ICD-10 system with any major comparisons and distinctions with DSM-IV being discussed where relevant. The ICD-10 codes (numbers usually prefixed with F) are also provided.

Schizophrenia

Definition and clinical manifestations

4 Schizophrenia is characterised by a fundamental distortion of thinking, perceiving and communicating which is accompanied by an abnormal affect (observed mood state), the most common abnormality being lack of reactivity ("blunting").

5 The disordered mental state, and the individual's reactions to it produce the subsequent highly heterogeneous clinical picture with many phases (acute, resolving, residual, remitted) and a widely varying course between individuals. Most patients improve with medication but some go on to "struggle with lingering deficits in areas such as attention, concentration, short-term memory, motivation, planning, decision making, sense of pleasure, empathy and sustained focused behaviour. Such patients often display chronic disabilities in self-care, social relationships and work capacity. They are often unemployed, socially isolated, dependent on their family and public welfare".

Medical adjudication guidance on **schizophrenia continued**

[back to Medical Adjudication Contents](#)

Extract from Social Security Commissioner Hearing

Appeal from decision of a Disability Appeal Tribunal on a Question of Law

Name: Sylvia Phillips (Mrs) Date: 1995

I have had sight of a written report on the nature of schizophrenia by Dr Lawrence Measey, a consultant psychiatrist at Coventry Health Care NHS Trust Mental Health Unit. Moreover, Dr Measey made himself available for questioning at the hearing, so that he was able to enlarge upon and clarify various aspects of his report. I am very much indebted to Dr Measey for his assistance.

Dr Measey explained that some 30 years ago it was often claimed that, as no obvious and consistent change in the neuro-anatomy or histology of the brain could be found in the case of a person who suffered from schizophrenia, the disease was one of dysfunction of the brain rather than the result of defects in its substance.

Thus, schizophrenia was described as a "functional psychosis" as opposed to psychosis and other mental impairments due to organic changes within the brain. However, within the last 20 years or so more vigorous scientific methodology, allied to sophisticated technologies developed for the study of the brain, had seriously put into question the description of schizophrenia as a functional psychosis. Indeed, since the late fifties it had become clear that the spectrum of schizophrenic disorders ranged from severe chronic deteriorating illnesses to a group of psychoses that had a shorter course, were easier to treat, and left little residue behind them.

Dr Measey explained that some forms of schizophrenia were constitutional in origin, whilst others were sparked off by environmental stress. The latter were those which more readily lent themselves to treatment, and eventual cure, but a person with a constitutional condition might also be subject to environmental stress factors. Dr Measey also pointed out that some disorders identical to schizophrenia could arise as a result of damage to the brain in later life, particularly those traumas which affected the temporal lobe.

However, in the case of those suffering from a constitutional condition, it appeared, Dr Measey said, that there was a distortion in the anatomy of the brain. In the words of his written report:-

[Medical adjudication guidance on **schizophrenia** continued](#)

[back to Medical Adjudication Contents](#)

Extract from High Court Judgement

Mr Justice Drake

Grace Hunt v The Secretary of State for Social Services Date: 21/12/1993

The opinion of the Medical Division of the Department of Social Security, relied on by the Respondent and accepted by the tribunal, is that smoking is a matter of personal choice and there is no evidence that the condition of the Appellant's feet caused him to smoke or to continue smoking. I cannot accept that. There is acceptable evidence that this Appellant underwent considerable hardship and suffered very great pain from the condition of his feet. This probably lasted from about the time he was taken prisoner of war in May 1941 until he was demobilised and then continued whilst he underwent repeated operations and treatment for his feet. Indeed, thereafter he continued to require foot treatment about every six weeks throughout his life.

It seems to me highly likely and certainly not free from reasonable doubt that smoking would have given him some relief from that suffering. It is easy to say that it was a matter of personal choice that he did not give it up. As a young prisoner of war, suffering pain without receiving full care and treatment to alleviate it, I do not think that it is realistic to hold that he had a true free choice.

Having, as I find quite reasonably, smoked for many years to alleviate pain, it is likely that he became addicted to the extent that he was unable to give up the habit. He lost the freedom of choice.

I fully accept that the evidence to support the Appellant's case is far from strong. However, having considered it with very great care, I do not think it is possible to say that any tribunal, properly directing itself on this evidence, could find beyond reasonable doubt that there was no causative connection between Mr Hunt's war service and the addiction to smoking which led to his death. In my judgement the evidence does raise a reasonable doubt in the Appellant's favour. Accordingly, this appeal will be allowed.

I will end, however, with a very strong caution against using this decision as a precedent. It depends very much indeed on the facts of Mr Hunt's wartime experience and the pain he continued to suffer for long afterwards as a direct result of his service life. Despite these exceptional facts, I have found this to be very much a borderline case.

It is very unlikely that there will be many other cases where a claimant will be able to raise any reasonable doubt of entitlement to a pension based on a sufficient link between service factors and smoking as a cause of death or disease.

[back to Medical Adjudication Contents](#)

Training minute

Date: 15 May 1996

Smoking Issues

This minute updates and replaces my minute providing guidance on smoking issues. Please destroy my original minute on this topic.

The main changes are a completely revised paragraph on addiction to cigarettes and an additional paragraph providing guidance in relation to the contention that a cigarette after battle resulted in the smoking habit.

As previously, this document addresses the basic issues with suggested "lines to take" and is intended to enable MAs to develop their own responses when asked to provide reasons for decision or when addressing smoking issues in OMDs. Although primarily aimed at appeals MAs, this document may also be of use and interest to claims MAs.

1. Background

PATs are increasingly finding in favour of the appellant in conditions such as chronic bronchitis and atherosclerosis where smoking is an important factor in the cause or progress. We need to address the issue of smoking in a more comprehensive manner than in the past. The argument that the SoS does not accept smoking as a factor of service and that smoking is within the individual's personal sphere is no longer sufficient.

Dr Arrowsmith's minute, dated 14 January 1994, contained guidelines and references for addressing medical contentions. With the exception of paragraph 11 of Peter's minute dealing with the addictive properties of nicotine, I have incorporated these guidelines into this paper unchanged. The paragraph dealing with the addictive effect of nicotine has been re-written.

Current monitoring of PAT decisions shows that the following are the commonest contentions made concerning smoking:

- smoking relieved the pain of an accepted disablement;
- smoking relieved stress/anxiety associated with service or an AD;
- the stress or pain of an AD compelled me to smoke.

[back to Medical Adjudication Contents](#)