Centre for Health and Disability Assessments

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Industrial Injuries Benefit Handbook 2 for Healthcare Professionals The Prescribed Diseases

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Foreword

This handbook has been produced as part of a training programme for Healthcare Professionals (HCPs) who conduct assessments for the Centre for Health and Disability Assessments on behalf of the Department for Work and Pensions.

All HCPs undertaking assessments must be registered practitioners who in addition, have undergone training in disability assessment medicine and specific training in the relevant benefit areas. The training includes theory training in a classroom setting, supervised practical training, and a demonstration of understanding as assessed by quality audit.

This handbook must be read with the understanding that, as experienced practitioners, the HCPs will have detailed knowledge of the principles and practice of relevant diagnostic techniques and therefore such information is not contained in this training module.

In addition, the handbook is not a stand-alone document, and forms only a part of the training and written documentation that the HCP receives. As disability assessment is a practical occupation, much of the guidance also involves verbal information and coaching.

Thus, although the handbook may be of interest to non-medical readers, it must be remembered that some of the information may not be readily understood without background medical knowledge and an awareness of the other training given to HCPs.

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Section 2.3.1 – note added to remind HCP to discuss any case for adjournment with their local IIB lead

Section 2.4 - Information regarding IIB terminal prescribed cancers updated

Section 3.1 – reference to vibration white finger updated with vibration damage to nerves and/or blood vessels of the fingers

Section 3.2 – reference made to mandatory script to be read out when claimant / companion are taking notes during an assessment

Section 3.7.2 – reference to Clinical Standards Team updated to Clinical Project Lead / Clinical Policy and Projects Lead

Section 3.7.2 – missing information regarding occupational deafness on page 27 now re-inserted

Section 3.8 – information on duration of assessment for PD D3, PD D8 and PD D8A updated with reference to other terminal prescribed cancers removed

Section 4 – number of IIB helpdesk updated

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Section 4.6.5 – note added to remind HCPs to discuss any case for adjournment with local IIB lead

Section 5.11 - reference updated to discuss PD C33 cases with Clinical Project Lead and Clinical Policy and Projects Lead

Section 5.12.4 – disablement advice for PD D10 cases updated

Appendix 5 – modified Allen's test added

Appendix 8 – reference made to UE1 process available in the Industrial Injuries Benefit Handbook 1

Outstanding issues and omissions

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Contents

1. The Purpose of this Handbook 9

2. The Prescribed Diseases - General Details 10

2.1 General 10

2.2 Historical Background 10

2.3 Criteria considered in a Prescribed Disease claim 10

2.3.1 Multiple IIB Referrals 11

2.4 Disablement Assessment in Prescribed Diseases 13

3. Providing medical advice to the Decision Maker in a Prescribed Disease report 16

3.1 The Diagnosis of Prescribed Diseases 16

3.2 Taking the statement for a Prescribed Disease 17

3.3 Deciding the date of onset of the diagnosed Prescribed Disease 19

3.3.1 Date of onset - Before the claimant's scheduled employment began 19

3.3.2 Date of onset - Date before the PD was added to the schedule of Prescribed Diseases 19

3.4 Giving an opinion on Loss of Faculty 20

3.5 Subsequent Claims for the Same Disease- previously not diagnosed 20

3.6 Subsequent Claims for the Same Disease - previously diagnosed and assessed 21

3.6.1 Reassessment where there has been a provisional award which is nearing the review date 21

3.6.2 Reassessment where there has been a final award which has, or is about to, come to an end 21

3.6.3 Recrudescence 23

3.7 The Causation Question and the concept of Presumption 24

3.7.1 Causation 24

3.7.2 Presumption 24

3.8 Disablement Questions 28

3.9 How to deal with uncertainty about an earlier Prescribed Disease Diagnosis 29

3.9.1 Assessment of Disablement after Diagnosis by the HM Courts and Tribunals Service 29

3.9.2 Change of Circumstances and Renewal on previously diagnosed Prescribed Disease claim 30
3.10 Justification of Advice in Prescribed Disease 31
3.10.1 When a Prescribed Disease is not diagnosed 31
3.10.2 Giving Adverse Causation advice 31
3.10.3 Justifying the level of disablement assessment 32
3.10.4 The duration of the disablement assessment 32
3.10.5 Recording of Consideration of Further Medical Evidence 32

4. Some of the more common Prescribed Diseases 33
4.1 Prescribed Disease A4 33
4.1.1 Summary of changes to PD A4 33
4.1.2 Dystonias 34
4.1.3 Prescribed Disease A4 36
4.2 Prescribed Disease A6 38
4.3 Prescribed Disease A7 44
4.4 Prescribed Disease A5 45
4.4.1 Cellulitis 46
4.5 Prescribed Disease A8 48
4.6 Prescribed Disease A11 51
4.6.1 Scheduled Occupations 51
4.6.2 Description (Prescription) for PD A11 52
4.6.3 Diagnosis 53
4.6.4 Formal testing of Grip and Manual Dexterity 57
4.6.5 Thermal Aesthesiometry & Vibrotactile Testing 59
4.6.6 Differential Diagnosis in PD A11 62
4.6.7 Assessment of Disablement in PD A11 62
4.6.8 Date of Onset in PD A11 66
4.7 Prescribed Disease A12 66
4.7.1 Carpal Tunnel Syndrome 67
4.7.2 Due to the nature of 71
4.8 Prescribed Disease A14 72

5. Some other Prescribed Diseases 79

5.1 Prescribed Disease A1 79
5.2 Prescribed Disease A2 80
5.3 Prescribed Disease A13 81
5.4 Prescribed Disease B14 83
5.5 Prescribed Disease B15 85
5.6 Prescribed Disease D3 87
5.6.1 Background 88
5.6.2 Handling the claim (Claimants with Work Exposure) 92
5.6.3 Handling the claim (Claimants with No Work Exposure) 93
5.6.4 The Health Assessment Advisory Service involvement in the Scheme 94
5.7 Prescribed Disease D13 97
5.7.1 Background 97
5.7.2 Assessment of PD D13 Claims 99
5.8 Prescribed Disease C3 100
5.8.1 C3a - Phossy Jaw – Osteonecrosis of the jaw 101
5.8.2 C3b – Peripheral polyneuropathy or peripheral polyneuropathy with pyramidal involvement of the central nervous system, caused by organic compounds of phosphorus which inhibit the enzyme neuropathy target esterase. 101
5.8.3 What has been removed from the prescription and why? 102
5.9 Prescribed Disease C31 – Bronchiolitis Obliterans 102
5.10 Prescribed Disease C32 – Carcinoma of the Nasal Cavity or associated Air Sinuses (Nasal Carcinoma) 104
5.11 Prescribed Disease C33 – Chloracne 106
5.12 Prescribed Disease D10 – Lung Cancer In Coke Oven Workers 110
5.12.1 About Coke Ovens and the Coking Process 111
5.12.2 Job titles and job locations at coke works 111
5.12.3 Diagnosis and Treatment of Lung Cancer 112
5.12.4 Disablement Assessment 112
5.13 Prescribed Disease D9 – Unilateral or bilateral diffuse pleural thickening with obliteration of the costophrenic angle 113

6. Occupational Deafness PD A10 116
6.1 Diagnosis 116
6.2 Assessment of disablement 117

Appendix 1 - The Prescribed Diseases 119

Appendix 2 - Information to assist with a claim to Occupational Deafness 136

Appendix 3 - Raynaud's Disease, VWF and HAVS 156

Appendix 4 - Background Information on Thermal Aesthesiometry & Vibrotactile Testing 161

Appendix 5 - Upper Limb Examination Tests 166

Appendix 6 - The Stockholm Scales 167

Appendix 7 - Policy on Passive Smoking 169

Appendix 8 - Faxing of Information and Dealing with Unexpected Findings 170

Appendix 9 - Diagram of a Coke Oven 174

Appendix 10 - PD D9 X-ray changes 175

Appendix 11 – List of Prescribed Diseases which require assessment by RD-trained HCP 176

Appendix 12 - Changes to Presumption rule in IIB 177

Observation form 183

1. The Purpose of this Handbook

The purpose of this second volume of the IIB Handbook is to act as a guide for Healthcare Professionals (HCPs) and for others involved in, or with an interest in, claims for Prescribed Diseases under the Industrial Injuries Benefit (IIB) Scheme.

The Social Security Act 1998 regarding Decision Making and Appeals (DMA) replaced the previous system of independent and multiple Adjudicators with a single status Decision Maker. DMA was introduced for Industrial Injuries Benefit from 5 July 1999. This Handbook provides the HCPs with advice about how Prescribed Disease report forms should be completed to allow the Decision Maker to make justified decisions on such claims.

The reader will find that the Industrial Injuries Benefit Handbook 1 outlines the procedures for medical assessments in all types of IIB claims and that this Handbook 2 provides more specific details of Prescribed Diseases. The Industrial Injuries Benefit Handbook 2 should therefore always be read in conjunction with Handbook 1 in order to understand the full scope of the HCPs' duties to both the Customer (Department for Work and Pensions) and the Claimant (person who is making the benefit claim).

The commonly claimed Prescribed Diseases are covered in broad detail. Where exceptional procedures exist for certain Prescribed Diseases, then these are also outlined.

This Handbook also makes reference to some of the Respiratory Prescribed Diseases. It should be noted that HCPs must also have undergone specific training in Respiratory Prescribed Disease assessments in addition to their training for general IIB before undertaking medical assessments or providing advice in relation to the Respiratory Prescribed Diseases. The Department for Work and Pensions (DWP) recognised that some rare Prescribed Diseases would require a more specialist assessment in order to provide the best quality output. This specialist assessment could be provided by HCPs who have been trained and approved in both general IIB and Respiratory Prescribed Disease. The full list of Prescribed Diseases which require assessment by such HCPs can be found in Appendix 11.

It is hoped that this Handbook provides a good all-round source of Prescribed Disease reference for HCPs, as well as indicating what legislation and other sources of advice should be consulted in cases of difficulty.

2. The Prescribed Diseases - General Details

2.1 General

The Contributions and Benefits Act 1992 provides, amongst other things, benefit for those who have contracted a prescribed industrial disease in employed earner's employment. A disease may be prescribed only in relation to those people with a record of employment in particular defined occupations.

A list of Prescribed Diseases (PDs) and their relevant occupations is shown in **Appendix 1.**

2.2 Historical Background

No fault compensation for specified occupational diseases began with the Workman's Compensation Act of 1897, but the current system was established under the National Insurance (Industrial Injuries) Acts of 1946 and 1975.

The scheme provides benefits to employed earners for certain "prescribed" diseases that have arisen out of and in the course of work.

Diseases are prescribed by the Secretary of State on the advice of the Industrial Injuries Advisory Council (IIAC)

The Social Security Contributions and Benefits Act 1992 states that the Secretary of State may prescribe a disease where he is satisfied that the disease:

a) ought to be treated, having regard to its causes and incidence and any other relevant considerations, as a risk of the occupation and not as a risk common to all persons; and

b) is such that, in the absence of special circumstances, the attribution of particular cases to the nature of the employment can be established or presumed with reasonable certainty.

In other words, a disease may only be prescribed if there is a recognised risk to workers in an occupation, and the link between disease and occupation can be established or reasonably presumed in individual cases.

2.3 Criteria considered in a Prescribed Disease claim

A claim to a Prescribed Disease needs to satisfy a series of criteria before assessment of disablement becomes an issue. These primary concerns are:

1) Is the disease claimed a Prescribed Disease or sequela?

2) Is the claimant working in (or has worked in) a relevant Prescribed occupation?

3) Is it employed earners employment?

These considerations are called the Prescription questions and involve the Decision Maker considering whether the claimant worked in the Prescribed occupation or used the Prescribed tool. In practice the Decision Maker is helped in this respect by the legislation which defines scheduled occupations in which the Prescribed Disease can be contracted. (See Appendix 1)

Usually these three questions are considered and answered by the Decision Maker, without medical advice.

If those first three aspects of the claim are satisfied, then the following must be addressed and these are answered on the basis of medical advice:

4) Medical advice is needed to answer the question of whether or not the claimant is suffering from (or has suffered from) the claimed PD or any other PD or sequela - this area of consideration is known as the Diagnosis Question.

5) When a PD is diagnosed the HCP must also advise the date of onset of the disease.

6) Medical advice is needed to establish if there is a causal link between the diagnosed PD and employment in the prescribed (or scheduled) occupation.

2.3.1 Multiple IIB Referrals

IIB Prescribed Diseases and Accidents are referred to The Health Assessment Advisory Service on a BI8. All the historical BI8's for past claims for other diseases or accidents are attached. There is a separate BI8 for each accident or PD. HCPs may therefore see different versions of BI8s enclosed depending on when the folder was created.

The BI8 was revised in 2014 with removal of many sections which were not in use, such as:

- DO Target
- Details of customer's doctor
- Details of Incapacity
- Details of Injury Benefit
- Assessment for other industrial accidents or diseases
- Other relevant information
- Chargings (continued)

In addition revisions were made to the following sections:

- Renewals: current assessment ends - The number of available boxes was reduced from six to one and renamed 'Assess End'

- Chargings - This now contains an additional column, 'code', to clearly identify the referral code

- Details of the accident or disease – This now only contains the occupation and a description of the accident or PD. The 'date of the accident or PD number' was removed as this information is provided on the front of the BI8 in the top right hand corner (under 'I/A or PD Code')

- Details of existing assessments - This contains information that was previously contained on the BI611 and in the section 'Assessment for other industrial accidents or diseases'. The BI611 is no longer used as the information is recorded on the BI8 jackets itself

- An additional tick box to identify Fast-Track cases has been added at the top of the BI8

The BI8A form is now only included where necessary and at the Decision Maker's discretion.

On occasion there may be more than one BI8 referral to the Health Assessment Advisory Service made at the same time. This may be for more than one PD, or a combination of PD and Accident referrals. In some cases – particularly some Respiratory PDs - it is possible for the HCP to complete the case(s), as a file work exercise, on the basis of the evidence available in the BI8 referral file. If all the referrals can be determined in this way then all cases should be completed before the referrals are returned to the Department for Work and Pensions (DWP).

However where advice can only be given on one or more of the cases based on the evidence in the file without the need for a face to face medical assessment this should be completed and the BI8(s) containing the completed assessment report returned to the DWP IIB office separately. The other case(s) should then be referred for a face to face medical assessment.

The only exception to this may be if there is a potential for the completed case(s) to have an impact on the accident or disease requiring a face to face medical assessment of the claimant.

Fictitious Example 1

PD D1 claimed at same time as PD A11 and referred to The Health Assessment Advisory Service at same time.

The advice on PD D1 may be given using x-ray evidence with advice that the claimant is not suffering from D1.

A face to face assessment is required to assess the PD A11 claim.

In these circumstances the BI8 for PD D1 should be returned separately to the Authority as soon as it is completed and not retained until after the PD A11 claim has been assessed.

Fictitious Example 2

PD D1 previously claimed and assessed with award of 10% disablement. Further claim for change of circumstances is made at same time as a new claim for PD D9 and referred to The Health Assessment Advisory Service.

The claimant has a medical assessment and the D1 claim is determined without further x-ray evidence. The D9 claim is adjourned to await receipt of x-rays. (HCPs should discuss any case requiring adjournment with their local IIB lead or IIB helpdesk prior to adjourning the case.)

In these circumstances the BI8 for D1 claim has been completed, but the disablement assessment could well interact on any assessment for D9. In this case the two files should be held until the second case can be completed, then returned together to the Authority.

2.4 Disablement Assessment in Prescribed Diseases

A person has entitlement to disablement benefit if they suffer from a loss of physical or mental faculty resulting from an Industrial Accident or Prescribed Disease.

In circumstances where the claimant has the Prescribed Disease and causation is accepted then medical advice is required on the relevant loss of faculty and the degree and extent of any disablement which arises from that.

In cases where the claimant has a disease, e.g. carpal tunnel syndrome, then the advice must be that the PD is diagnosed. However if the disease is not caused by the relevant occupation, the HCP should advise accordingly, and should not give an opinion on the disablement questions as to do so may result in benefit being awarded inappropriately. It is important that the HCP gives a good explanation and justification for his/her opinion.

The disabling effects of Prescribed Diseases are assessed by those same principles, which are employed for Industrial Accidents, as described in IIB Handbook 1. However, there are important differences with special provisions relating to PDs. These are explained in general terms in the following paragraph. Specific reference to occupational deafness (PD A10), vibration damage to the nerves and/or blood vessels of the fingers (PD A11), conditions associated with bursitis/subcutaneous cellulitis (PD A5, 6 & 7), malignancy following radiation exposure (PD A1) and heat cataract (PD A2) is provided in later sections.

In PD claims, there are questions that the HCP must consider other than the disablement question. The HCP must first advise on the question of diagnosis

and decide whether the claimant is suffering or has suffered from a PD or any sequela.

A sequela is a condition, which has resulted from a PD, and it should be treated as if it were that disease, even though the condition itself may not be a PD.

For claims made on or after 06.04.83, Disablement Benefit is not payable until a period of 90 days (excluding Sundays) has elapsed. Different regulations were in place for earlier claims. The period starts from the day of the relevant Industrial Accident or the date from which the loss of faculty first existed in a PD case.

In addition, for claims made on or after the 01.10.86, payment of benefit will only occur when the extent of the resulting disablement amounts to:

- 14% or more present on the 91st day after an industrial injury or after date of onset for most PDs
- 20% or more in the case of occupational deafness (PD A10)
- 1% or more in cases of pneumoconiosis (PD D1) or byssinosis (PD D2)

Lung cancer related to asbestosis or asbestos exposure (PD D8 and PD D8A) and diffuse mesothelioma due to asbestos (PD D3), where diagnosed, are universally deemed to be assessed at 100% disablement.

With the exception of PD D1 and PD D2, single assessments of less than 14% do not qualify for payment. However if they can be aggregated with any other assessment, in respect of one or more Industrial Accidents or PDs over a common period, then payment can be awarded if the total amounts to 14% or more.

Aggregation applies to assessments of 1% and above and in the instance described above, a non-payable assessment is rendered payable by virtue of aggregation if the total amounts to 14% or more. Aggregation does not apply to assessments of less than 1%.

Other factors that apply are:

- An assessment of disablement in respect of pneumoconiosis, byssinosis or diffuse mesothelioma may be aggregated with other assessments for accidents or diseases. Aggregation only applies to assessments of 1% or above
- For occupational deafness, PD A10, there is no 91st day rule and the date of onset of the PD is taken to be the date of the claim
- For diffuse mesothelioma due to asbestos (PD D3) and primary carcinoma of the lung related to asbestosis or asbestos exposure (PD D8 and PD D8A), there is no 91st day rule and entitlement to benefit starts from the date of onset of loss of faculty

In summary the questions that need to be addressed by the HCP are:

- The diagnosis and date of onset questions
- Causation
- The disablement questions
- The recrudescence question if it arises (See section 3.6.3)

3. Providing medical advice to the Decision Maker in a Prescribed Disease report

3.1 The Diagnosis of Prescribed Diseases

Considered from a purely medical point of view, Prescribed Diseases are often indistinguishable from the same disease present in someone who has not been working in a Prescribed Occupation. There are many examples of this including Tuberculosis (B5), Carpal Tunnel Syndrome (A12) and Chronic Obstructive Pulmonary Disease (D12).

To deal with a PD claim the HCP requires:

- A broad understanding of the scope of the Prescribed Disease
- Sufficient medical history and clinical examination data to satisfy a diagnosis of the disease in the particular claim
- Sufficient occupational history to advise it is more probable than not, that factors in employment caused the disease

These steps assume greater significance when a disease is common in the population as a whole - such as Carpal Tunnel Syndrome.

Some Prescribed Diseases include causative factors in their definition. PD D4 is a good example of this. PD D4 is Allergic Rhinitis but the regulation states that the Allergic Rhinitis must be due to one of the sensitising agents listed in the legislation e.g. isocyanates; platinum salts; fumes or dusts arising from the manufacture, transport or use of hardening agents, etc. This is to mention just a few of the total list. It is therefore only possible to diagnose PD D4 when there is evidence of exposure to one or more of the listed sensitising agents. In other words while Allergic Rhinitis is common in the population as a whole, PD D4 can only be diagnosed when it is more probable than not that the Allergic Rhinitis is due to one or more of the factors listed in the regulations/legislation.

In some instances a disease may be present from a medical point of view, but not to the extent required to satisfy the diagnosis of the PD.

Examples of this include.

- PD D12 Chronic Obstructive Pulmonary Disease – there must be at least a 1 Litre drop in predicted FEV1 or an FEV1 of less than 1 Litre before PD D12 can be diagnosed

- PD A10 – the Regulations require that there is a 50 dB sensorineural hearing loss due to all causes in each ear and due in at least one ear to occupational noise. This is the minimum requirement to meet the diagnosis threshold for PD A10

- A medical diagnosis of vibration damage to the nerves and/or blood vessels of the fingers can be made when only a tip of one finger is affected by vascular changes in winter and with intermittent sensorineural symptoms. This however would not satisfy the criteria for PD A11 which requires a specific number of digits to be involved in both summer and winter months for vascular changes and constant sensorineural symptoms. (It should be noted that you must avoid the use of terms such as Vibration White Finger, VWF, or Hand Arm Vibration Syndrome, HAVS, in PD A11 reports, especially if the condition does not satisfy criteria for PD A11)

3.2 Taking the statement for a Prescribed Disease

In every Prescribed Disease medical assessment the HCP is required to:

- Understand the scope of the claimed Prescribed Disease. To have knowledge of the underlying medicine of that condition and the occupational factors which may have caused the disease.

- Take a full history of the symptoms, their date of onset, progress in relation to continuing exposure - or recovery in response to change of work or rests from the work place. To include the investigations undertaken and response to treatments provided. Any diagnosis quoted to the claimant by his/her medical carer or hospital staff. Consider the possible occurrence of sequela.

- Take a full and detailed occupational history, which includes a history of all occupational factors that may have resulted in a sufficient degree of exposure to cause the disease. Detail descriptions of the work undertaken. It is not sufficient to merely list the previous occupations. It may be useful to identify if there are any other workers with similar conditions in that work place. Describe any work place adjustments made by the employer since the onset of this condition - (extraction fans, personal protective equipment, ergonomic adjustments to desk arrangements or seating, work sharing practices, change of role etc) - and any change in the claimant's condition following such work place changes.

- Take an account of the functional effects of the condition, providing details of typical day activities to demonstrate any restrictions imposed by the effects of the PD.

- Take a detailed social history to exclude, for example, a hobby that could cause the disease, either wholly or partially.

- Take a detailed family history to exclude inherited causes of the disease, which cause the disease wholly or partially.

- Take a full personal past medical history and aim to show if the condition has occurred in the past, either in relation to occupation or otherwise. **It is imperative that in every assessment the past history is documented. This should include documentation of negative responses as well as positive**. In addition, enquiry should be made and documented regarding any subsequent problems developing after the onset of the Prescribed Disease being considered. This should include any subsequent injury or development of other medical conditions, whether or not this was related to the occupation.

- Perform a careful, appropriate formal and informal examination of the claimant. In those cases where a peak flow is recorded, in line with the European Union (EU) standard for peak flow meters it should be noted in brackets whether a "Wright" or "EU" meter was used.

HCPs should be aware that people who are entitled to be in attendance at the medical assessment are always entitled to take notes for their own purposes. These notes are not considered an official record of the process but it should be documented on the front page of the medical report, the fact that notes were being taken.

The form of words you must use has been clarified on legal advice and the following mandatory script has to be read out when a claimant / companion is taking notes during an assessment:

Please replace any copies of existing desk aids you hold with the one incorporating the following form of words:

"Where notes are taken by you, we consider it of assistance to both myself, as the assessing Healthcare Professional, and yourself to point out the following:

1. It is your right to take notes for your own use and benefit.

2. The notes will not be included in the Report I make save for the fact that notes were taken and further, they are not accepted by myself or the DWP as an official record of this assessment.

3. If the notes are subsequently produced at any time for any purpose, such as part of an appeal process, I the assessing Healthcare Professional, my employer and the Department for Work and Pensions reserve all rights to challenge anything in the notes in the event we are asked to comment on the content of the notes at a future time.

4. You are free to use your notes as you choose but if you chose to publicise the notes (other than in connection with correspondence with the DWP or under any appeal procedure) I would ask that you do not publicise my name."

HCPs should note that in the event of a medical assessment being conducted as a domiciliary visit, a smoking policy has been implemented. Please see Appendix 7.

3.3 Deciding the date of onset of the diagnosed Prescribed Disease

Medical advice is also required on the date of onset of the diagnosed Prescribed Disease.

The HCPs needs to ensure that appropriate detail is obtained in the history.

The date of onset of a PD is not necessarily the same as the date the disorder may have been diagnosed from a medical point of view:

- In some cases the date of onset of the PD can be readily determined. For example, for PD A3 where there is a history of rapid ascent or descent causing barotrauma. This is a single acute event and the date is clearly understood.

- In PD A10 (Occupational Deafness) the date of onset is taken to be the date of claim.

- In some PDs the date of onset has to be determined from a careful history. Where the Regulations define the severity of a condition before it can acquire the status of the PD it is often necessary to identify a time of transition from a medical diagnosis to the point in time when the criteria for the diagnosis of the PD are satisfied.

- This date may be a date before the scheduled employment began or a date before the PD was added to the schedule of PDs. For more details see next section.

3.3.1 Date of onset - Before the claimant's scheduled employment began

In this case, although the Prescribed Disease is diagnosed, the HCP should advise that the disease is not due to the nature of the scheduled employment and should go on to explain what factors, occupational or otherwise, are considered to have caused the disease. If appropriate the Decision Maker will then investigate whether the claimant was in a scheduled occupation at or before the date from which the disease was diagnosed.

3.3.2 Date of onset - Date before the PD was added to the schedule of Prescribed Diseases

In this case, HCPs are advised that the Prescribed Disease can be said to start at the earlier date but any assessment of disablement should not begin before the date the PD was added to the schedule.

3.4 Giving an opinion on Loss of Faculty

It is necessary to provide an answer on the Loss of Faculty question:

- When the claimed PD is diagnosed
- When the claimed PD is not diagnosed but another PD is diagnosed

Even if diagnosed from a legal point of view, a PD may not result in a loss of faculty, or the HCP may advise that the loss of faculty amounts to less than 1%. For example, a very small patch of dermatitis may be assessed at less than 1%. Following an Upper Tribunal Judge's Decision (R (I) 6/61) HCPs must distinguish between cases where there is no loss of faculty and those where there is a loss of faculty, but the resultant disablement does not amount to 1%.

The Decision Maker decides whether or not the claimant can be diagnosed as suffering from a specified PD, but this decision will require advice from an HCP on this question.

The following basic rules apply:

- If the HCP advises in the claimant's favour on the Diagnosis Question then advice on the Disablement Questions should be given.

- If the HCP does not find in favour of the claimant on the Diagnosis Question, then no advice should be given on the Disablement Questions.

- If the HCP advises that the claimant is suffering from a PD other than one referred by the Decision Maker, it is **very** important to make it clear to the Decision Maker that the advice relates to an alternative PD, and not to the PD which was referred to them. Hence the report should be annotated at Part 6 of the BI613 (i.e. where the advice on disability is given) and at Part 7 of the BI613 (or equivalent RD forms) that this advice refers to the **alternative** PD that has been diagnosed. The HCP should then give advice on the disablement questions. Note: the Decision Maker will need to investigate whether the alternative PD is prescribed in relation to the claimant.

3.5 Subsequent Claims for the Same Disease- previously not diagnosed

When a previous claim has been disallowed following advice given by an HCP to a Decision Maker or after the decision by a 1st Tier Tribunal, any new claim for the same PD is usually treated as a first claim.

If a claim for a PD fails on the diagnosis question, the records will be kept in line with document retention policies. Should there be a later claim for the same or a different PD, the records will be made available to the HCP if they have not been destroyed, for comparison of the history and the clinical findings.

Where a disallowance decision has been given, then a further claim to the same PD cannot result in acceptance of the diagnosis from a date earlier than the date of the first decision, except in certain circumstances during the period 5/7/99 to 18/3/2005.

3.6 Subsequent Claims for the Same Disease - previously diagnosed and assessed

3.6.1 Reassessment where there has been a provisional award which is nearing the review date

In this circumstance the appropriate action is to review the case and advise whether the level of assessment has changed. The HCP should ensure that the new assessment starts on the day after the existing assessment ends.

3.6.2 Reassessment where there has been a final award which has, or is about to, come to an end

In this case the claimant will in most cases apply for a review on grounds of change of circumstance. They may indicate that the condition has failed to improve, or indeed that it has worsened.

Where the decision has been given post DMA (Decision Making and Appeals) legislation of 05.07.99, and where finalised award has come to an end it ceases to apply, and cannot be superseded on the grounds of "change of circumstances". This means that any such application for change of circumstance which is received after the assessment period has ended has to be treated by the Decision Maker as a new claim. If, for example, you form the view that the injury or prescribed disease is no longer causing any effects, then you should advise that there is no loss of faculty rather than saying that no worsening of the relevant condition has occurred.

The Decision Maker is the arbiter regarding the type of referral and may still refer cases where a claim had been finalised post DMA legislation as change of circumstances rather than as new claims. The HCP should complete the assessment using the form which is present in the file and give advice accordingly.

A Change of Circumstance claim must be made within certain time limits. If the application is made too late, the only remedy the claimant may have is by way of review on grounds other than change of circumstances or by late appeal to a Tribunal.

Where a Change of Circumstance claim has been accepted, the HCP should consider whether there is any ongoing loss of faculty. If so then advice on an appropriate level of assessment should be given. In addition the HCP should consider when the change occurred, and should advise that the assessment starts from that date. It is worth bearing in mind that it should not be earlier than the date of the previous assessment.

The advice given to the Decision Maker must be clear as an upper tribunal judge has identified an area of potential confusion in previous guidance and practice. The confusion arose at a change of circumstance assessment where no change was advised.

In this particular case (CI/3779/2012), the claimant had an industrial accident on 4th May 2000 and made a claim to IIB in August 2003. The claimant had a final assessment of 11% disablement for the period 17th August 2000 to 16th August 2005 based on medical advice provided on 9th September 2004. The claimant made two further claims in 2008 and 2011.

At the later assessments medical advice was given that the claimant's condition had not changed since the last assessment. The Judge opined that this could have two meanings:

- The claimant was 11% disabled as per their original assessment
- There was no remaining loss of faculty due to the accident because the original HCP's prediction of no remaining functional effect due to the accident by 17th August 2005 was correct

The Judge determined that the advice given by the HCP that the claimant's condition had not changed since the last assessment was ambiguous and recommended tighter drafting of medical advice on disablement.

Therefore where no change is advised, the actual percentage or no remaining loss of faculty must be explicitly stated in the justification.

Auditors will continue to assess whether disablement is expressed appropriately or not bearing in mind this guidance. The current code of R55 (Disablement expressed appropriately) would be applicable.

Examples:

- Claimant has a 2 year provisional award at 20%. They are assessed for change of circumstances 12 months into the assessment period, stating they have got worse. The assessing practitioner carries out an assessment and is of the opinion that a 20% assessment remains appropriate as does the period.

  A suggested addition to the justification could be "The loss of faculty due to the original accident has not changed since the previous assessment of (enter date of previous assessment) and remains appropriately assessed at 20% for a period of two years from (enter the original date)".

  It remains a requirement to fully justify why the advised percentage is appropriate in your opinion.

- Claimant had a final assessment of 11% with a period that finished 3 years prior to your review. Your opinion following an assessment was that there was no remaining loss of faculty.

  A suggested addition to the justification could be "The assessment of the claimant's condition at the last assessment of (enter date of last assessment) is unchanged. There is no remaining loss of faculty due to the industrial accident of (enter accepted date of industrial accident)."

3.6.3 Recrudescence

This question can only arise when the claimant suffers a further attack of the same PD (other than pneumoconiosis, byssinosis, mesothelioma, occupational deafness, occupational asthma, primary carcinoma of the lung, bilateral diffuse pleural thickening or chronic obstructive pulmonary disease). The further attack is either within a period already covered by a previous assessment of disablement or, exceptionally, the period covered by compensation under the Workmen's Compensation Acts (the benefit which preceded IIB) for that disease.

If the HCP advises that the further attack commenced during such a period, the further attack will be treated as a recrudescence, unless the HCP decides that the PD has been contracted afresh.

The HCP may decide that the further attack is a fresh contraction beginning during the period of assessment in respect of the earlier attack.

In determining whether or not a further attack is a recrudescence or a fresh contraction, the HCP may take into account:

- Whether or not the disease is a chronic condition prone to relapse
- The extent to which further exposure to risk at work from the disease has occurred. **There must have been such further exposure for there to have been a fresh contraction**
- The time over which the symptoms have declined or resolved completely
- The presence of sensitisation

Therefore, a recrudescence is more likely to occur in chronic relapsing conditions, and a fresh contraction is more likely if there has been a prolonged period free of symptoms prior to the further attack.

If the HCP advises that the further attack began:

- Within the period of assessment in respect of the earlier attack, and it is a recrudescence, assessment will be made by way of a change of circumstances, if the claimant agrees.

- Within the period of assessment in respect of the earlier attack but it is a fresh contraction, the fresh contraction will begin on the date on which the claimant first suffered the loss of faculty from it. The commencing date of any assessment is subject to this date.

- Outside the period of assessment in respect of the earlier attack it must be treated as a fresh contraction commencing on the date on which the claimant first suffered a loss of faculty as a result of that attack.

The recrudescence question will be referred to an HCP where:

- The Decision Maker refers a diagnosis question and a recrudescence question arises.

- When a claimant, who has received disablement benefit or Workmen's Compensation for the same disease appeals against disallowance of the later claim by the Decision Maker, on the recrudescence question.

3.7 The Causation Question and the concept of Presumption

3.7.1 Causation

Many of the Prescribed Diseases are indistinguishable from the disease contracted outside of work. Judges of the Upper Tribunal (previously known as Commissioners) have advised that if the disease is diagnosed from a medical point of view, the PD must be diagnosed but may then be rebutted on the causation.

For example a person has Carpal Tunnel Syndrome, but the HCP is of the opinion that it is not due to the nature of the employment e.g. because he had used vibrating tools only for a short period of time before Carpal Tunnel Syndrome developed. The HCP should say "yes" A12 is diagnosed, but then go on to say "No" it is not due to the nature of the employment, with full justification of this advice.

3.7.2 Presumption

Regulation 4 is one of the most important of the Prescribed Diseases Regulations although the operation of the regulation is widely misunderstood. Some prescribed diseases are presumed to be due to a worker's occupation. The general rule for most diseases is to presume work caused the disease if the disease developed during the job or within a month of leaving it, unless contrary is proven. When the presumption rule applies the worker does not have to prove, in his or her case, that the job caused the disease. Where the date of onset is more than one month after leaving the employment then the onus of proving that the disease was caused by the employment lies with the claimant.

Presumption does not apply to A12a, C1, C2, C4, C5A/C5B, C6, C7, C12, C13, C16, C19, C20, C21, C22b, C23c/C23d, C25, C26, C27, C29, C30 and D5. For these PDs, the onus is on the claimant to establish, on the balance of probability that the disease was due to the nature of the employment.

IIAC reviewed this rule in 2015 for the first time since 1948 to ensure that it was in line with current medical thinking on the causation of work related diseases.

Although the presumption rule was altered for many prescribed diseases, very few claims were affected. For the more common diseases either no changes were proposed or claims were already managed in the way which was being recommended. The changes simply brought outdated law in line with current practice in the benefit. All the changes are detailed in Appendix 12.

Four types of changes were made to presumption:

- presumption was applied for the first time in some diseases
- the time rule for presumption was extended in many diseases
- presumption was removed from some workers in one disease
- some diseases were split so that different time rules can apply to different manifestations of disease

Healthcare Professionals will need to be aware of the current guidance in order to understand why new claims or change of circumstance requests have been made (in some cases).

These changes could have an effect on claim outcome in a small number of historical claims in specific circumstances, the diseases are A2, A3, B2, B6, B8, B13, C22A and the circumstances are detailed in Appendix 12. Change of circumstances requests or new claims could be received from people who previously were not diagnosed with these prescribed diseases when they claimed in the past. There is no effect on past claims for any other prescribed disease.

Healthcare Professionals should continue to give individual advice on the causation question in every case, whether presumption applies or not.

Overview of changes to presumption

Changes to the time rule in work related cancers:

For many of the work related cancers the rule that the disease is presumed to be due to the job has been extended from ‘in the job’ to ‘in the job and any time after leaving the job’. For most work related cancers the scheme has always accepted causation due to work, no matter how long ago the worker left the qualifying employment. For these diseases although the rule has changed there is no effect on any past claims or the handling of new claims. These diseases are: A1, A13, A14, C17, C18, C23(a), C23(b), C23(e), C24, C31, C32, D3, D6, D8, D8A, D9, D10, D11, D13.

Changes to the presumption rule in diseases due to biological agents B1, B2, B4, B5, B6, B7, B8 and B13:

In B2, B8 and B13 the presumption rule has been extended from 'in the job' to 'in the job and any time after leaving the job'. The effect in these diseases could be to enable the prescribed disease to be diagnosed as due to the nature of the employment when the claim had been refused previously due to the time that had elapsed between the employment and diagnosis of the condition. The presumption rule has been extended for these diseases because the medical literature shows or expert opinion believes that diagnosis can be delayed long after inoculation with the infectious agent. Healthcare Professionals should continue to advise on the causation question based on the balance of probabilities having weighed the medical evidence and considered the nature of the health condition claimed. For rare B diseases seek advice from the Clinical Project Lead / Clinical Policy and Projects Lead who can consult members of the Strategic Health & Science Directorate if necessary.
The presumption rule has been extended in B6 from 'in the job' to 'in the job and any time after leaving the job'.
B1 has been split into cutaneous (B1a) and pulmonary (B1b) forms of anthrax, the time rule for pulmonary anthrax has been extended to 2 months.
B4 has been split into B4a cutaneous larva migrans and B4b iron deficiency anaemia caused by gastrointestinal infection by hookworm. The presumption time rule has been extended to 12 months for anaemia due to hookworm infestation (B4b).
In B5 tuberculosis the benefit of presumption has been removed from workers who do not work in a hospital, laboratory or mortuary.
The time rule for B7 has been changed to 6 months to reflect the incubation period.

Changes to A2, A3 and A12, diseases due to physical agents:

A3 has been split into a (dysbarism) and b (osteonecrosis). The presumption rule has been extended so that long latency conditions cataract (A2) and osteonecrosis (A3b) are now covered by the rule if they occur 'in the job or any time after the job'. Presumption has been applied to A12b.

C22a nasal cancer, presumption has been applied.

Fictitious Example 1 – Presumption applies

Claimant employed as a typist from 1986 to date and makes a claim for PD A4. Medical opinion is that the claimant is suffering from the disease and the date of onset is given as 12.6.06.

As the claimant was in a prescribed occupation in employed earner's employment on that date and there is no evidence to the contrary, the Decision Maker accepts that the disease is due to the nature of employment.

Fictitious Example 2 – Presumption does not apply

Claimant employed as a typist from 1986 to 1999 and makes a claim for PD A4. Medical opinion is that the claimant is suffering from the disease and the date of onset is given as 12.06.06.

As the claimant was not in a prescribed occupation on the date of onset or within one month of that date, the disease cannot be presumed to be due to the nature of the employment and the onus of proof lies with the claimant.

Fictitious Example 3 – Contrary is proved

Claimant is employed as a part -time typist working 16 hours per week from 2003 to date and makes a claim for PD A4. Medical opinion is that the claimant is suffering from the disease and the date of onset is given as 12.6.06.

As the claimant is still employed in a prescribed employed earner's employment, the disease is presumed to be due to the nature of the employment unless the contrary is proved.

On the claim form the claimant states that she only works part – time because she is also an author. Investigations show that she spends between 4 and 5 hours per day writing short stories and also up to 8 hours per day at the weekend. Investigations also show that she has been writing since the early 1990's.

The Decision Maker weighs all the evidence and decides that the contrary is proved and decides that the disease is not due to the nature of the employed earner's employment.

Some exceptions to the general rule on presumption are Occupational Deafness (A10), and Tuberculosis (B5) where different rules apply; and Carpal Tunnel Syndrome (A12a) and Occupational Dermatitis (D5) where there is no presumption. There are further examples of this in the C Prescribed Diseases.

In the case of Occupational Deafness (A10), there is a wider presumption than for other PDs in that the disease is presumed to be due to the nature of the employment unless the contrary is proved.

For Tuberculosis (B5) the presumption applies where the date of onset of the disease is not less than 6 weeks after the date on which the claimant was first employed in the scheduled occupation and not more than 2 years after the date on which such employment terminated. The presumption rule applies for all types of workers working in hospitals, laboratories and mortuaries where post mortems are carried out. All other workers including healthcare workers who do not work in hospitals and mortuaries will no longer have the benefit of presumption. This reflects the evidence base which shows doubling of risk of tuberculosis in workers in hospitals, laboratories and mortuaries but little or no excess risk in community based health care work including general practice. Healthcare Professionals should give individual advice based on the circumstances of the exposure to tuberculosis.

In the case of Carpal Tunnel Syndrome due to use of vibrating tools (A12a) and Occupational Dermatitis (D5), because there is no presumption, the onus lies with the claimant to show that on the balance of probabilities his/her disease is due to the nature of the scheduled occupation. However presumption now applies for people making a claim under Carpal Tunnel Syndrome due to repeated palmar flexion and dorsiflexion for at least 20 hours per week (A12b)

The causation question is decided by Decision Makers but they are likely to need the benefit of medical advice for this in every case, whether presumption applies or not. Accordingly, when the diagnosis question is referred to the HCPs, they are also asked for this opinion.

If the HCP is of the opinion that the claimant is suffering from the medical condition but this is not due to the nature of his/her employment, then that opinion must be fully justified.

3.8 Disablement Questions

The disablement questions arising on Prescribed Disease cases are the same as those for accident cases (see Sections 1.3 and 1.4 in Handbook 1).

In assessing disablement the HCP should bear in mind that although signs of the Prescribed Disease may no longer be present there may still be a continuing loss of faculty. For example, an attack of non-infective dermatitis (D5) may be either a primary irritant contact dermatitis or an allergic contact dermatitis. If the latter is diagnosed then despite apparent recovery further reactions can be provoked by shorter or smaller exposures. Such sensitisation requires to be recognised in the duration of the disablement assessment.

In this example a small assessment of Life duration would be appropriate to identify an ongoing effect of the Prescribed Disease despite seeming recovery of the initial skin condition.

The disablement questions, on which an HCP gives advice, are:

- Has the accident or PD resulted in a relevant loss of faculty?
- What is the extent of disablement resulting from the loss of faculty to be assessed?
- What period is to be taken into account by the assessment?

Where disablement is assessed at less than 14% and the assessment does not aggregate with other assessments to give 14% or more, the assessment must be final – either as a ‘life final’ or as a ‘dated final’ award.

Where the assessment is 14% or more, or if less than 14% but aggregates with other assessments to give a total of 14% or more, the assessment can be provisional or final.

The advice that a disablement assessment is to be finalised does not necessarily mean that it must be for life. Finalised assessments can be advised for any period of time which is consistent with the likely prognosis of the injury or PD under consideration.

It can be noted here that not all disablement assessments follow the above rules. In PD A10 the rules differ - where there is a successful claim the assessment must be made life final at the initial assessment.

Cases of PD D3, PD D8 and PD D8A will be assessed at 100% disablement for life.

The principles of assessment are those outlined in IIB Handbook 1. Other effective causes of disablement are dealt with in the same way as in an Industrial Injuries claim. For details see IIB Handbook 1.

3.9 How to deal with uncertainty about an earlier Prescribed Disease Diagnosis

3.9.1 Assessment of Disablement after Diagnosis by the HM Courts and Tribunals Service

Judges for the Upper Tribunal held that a Tribunal hearing an appeal against an outcome decision need not make a new outcome decision where the appeal is allowed. This was a change to previous practice, where the tribunal would assess the level of disability in cases where they have diagnosed a Prescribed Disease.

This means that Tribunals may choose not to give an outcome decision on claims for Industrial Injuries Benefit. Appeal Submission writers may request that Tribunals give an outcome decision, but the Tribunal is not bound to do so.

As a result, cases may be referred where

- The Tribunal has diagnosed the PD, but not given an opinion on causation, and not assessed disablement.

- The Tribunal has diagnosed the PD, and given the opinion that it arises from the occupation, but not assessed disablement.

In both situations, if the HCP agrees with the Tribunal's opinion on diagnosis and causation, then he/she should advise the Decision Maker on the causation, loss of faculty disability, disablement etc.

There will also be cases where:

- The HCP disagrees with the diagnosis. In cases of this type the HCP should note at Part 4.1 'Diagnosis of PD* accepted by the Tribunal'.

- The HCP agrees the medical condition is diagnosed but is of the opinion

that it is not due to work. In cases of this type the HCP should note at Part 4.5 'Diagnosis of PD* and occupational causation accepted by the Tribunal'.

It should be noted that the HCP cannot say that the PD is not diagnosed, or that it is diagnosed but not due to the nature of the occupation. However, if the HCP disagrees with the Tribunal's opinion on diagnosis and/or causation, he/she should comment on this at part 6 of the BI613 (i.e. where the advice on disability is given) and at Part 7 of the BI613 (or equivalent RD forms) and must provide a clear explanation and detailed justification as to why their opinion is contrary to that of the HM Courts and Tribunals Service.

The HCP should then give advice on the loss of faculty, disability and level of disablement as appropriate.

3.9.2 Change of Circumstances and Renewal on previously diagnosed Prescribed Disease claim

Where the PD has been previously diagnosed and assessed, then a Change of Circumstance can be claimed, but when the HCP is of the opinion that the criteria for diagnosing the PD are not fulfilled, then it is in order to act accordingly. The HCP may advise that the PD was never diagnosed at all, or was not diagnosed from a particular date. A change of medical opinion is not itself grounds for supersession, as there has to be a material fact.

Thus there must be a full, clear explanation of the reasons for the opinion and the facts that have been considered in reaching it, and, if any dates are involved, the reason why that particular date has been chosen, for example, a date of recovery following an operation.

A similar approach should be followed when an HCP is considering a renewal and is of the opinion that diagnosis is not satisfied.

Although PD D12 (chronic obstructive pulmonary disease) is addressed in more detail in the specific respiratory diseases documentation, and has to be assessed by HCPs trained and approved in respiratory prescribed diseases, some information on the process of change of circumstances and renewals is provided here (UTS 14/2015). The prescription criteria for PD D12 were amended following advice from the IIAC so that the one litre loss of FEV1 would apply irrespective of any medical treatment taken by the claimant.

Previously various offsets were made to the FEV1 value to reflect the effects of treatment on the lung function. This could have resulted in a claimant being diagnosed with PD D12 in the past, who now no longer meets the threshold for diagnosis. In such a case the advice should be that 'the threshold for diagnosis of PD D12 is not met, PD D12 is no longer diagnosed.' The HCP could further explain that 'the claimant still has COPD but it is not severe enough to meet the diagnostic threshold for prescribed disease D12'.

3.10 Justification of Advice in Prescribed Disease

There are several tiers of advice in a PD report and the HCP should set out the logical evaluation of evidence which supports each answered question. Where a full history and a relevant clinical examination has taken place there will be adequate evidence to show the logical thought processes behind any opinion.

3.10.1 When a Prescribed Disease is not diagnosed

Summarise the salient points in support of that advice

This may be because:

- The medical features do not support the diagnosis and indicate an alternative diagnosis e.g.

  "PD A8 cannot be diagnosed because the history of elbow pain without thumb and hand pain and the absence of clinical findings in the wrist and forearm do not support a diagnosis of tenosynovitis."

  or

  "PD A6 cannot be diagnosed because there is no clinical evidence of skin thickening and inflammation or a bursa at the knee."

- A medical condition can be diagnosed but it is not of sufficient severity to be the PD e.g.

  "There is evidence of restricted airways disease, but as the FEV1 is less than 1 litre below predicted values PD D12 cannot be diagnosed"

  or

  "There is evidence of noise induced hearing loss but insufficient to meet the criteria defined for the diagnosis of PD A10".

3.10.2 Giving Adverse Causation advice

This can only be completed effectively if a detailed occupational history has been obtained

Factors, which may suggest that the necessary occupational exposure has not occurred, include:

- Vague job descriptions with no evidence that exposure/risk occurred e.g. descriptions of job tasks which are not repetitive in a PD A8 claim or work which does not involve contact with sensitisers or irritants in a D5 claim.

- Dates of onset of symptoms before or more than one month after any occupational exposure.

- Strong family history of a similar condition - i.e. others in family have this condition and they have had no occupational exposure.

- Previous medical history of the same condition and that was before employment in the Scheduled employment.

Occupational causation is difficult to refute, given the definition of a Prescribed Disease and the fact that the Decision Maker has accepted that the claimant works (or worked) in a Scheduled occupation. The advising HCP will need strong evidence of exposure elsewhere to support a view that the condition is not due to factors arising in the accepted occupation.

3.10.3 Justifying the level of disablement assessment

This should follow the principles that apply in the IIB Handbook 1 section 5.

3.10.4 The duration of the disablement assessment

Again this should follow the principles which apply in the IIB Handbook 1 section 5. The HCP needs to understand the probable prognosis in the occupational disease and to take into account the current treatment and its likely effect on outcome. These details should be set out to assist the Decision Maker.

3.10.5 Recording of Consideration of Further Medical Evidence

Where further medical evidence has been considered it is not sufficient to say, for example, “contents on file”, or “HCNs” (hospital case notes) as it will not be clear to future HCPs, HM Courts and Tribunals Services and Upper Tribunal Judges what has/has not been seen. The HCP must list the evidence with dates where relevant considered, and, where Hospital Case Notes have been obtained, complete an extract on form BI 127A.

4. Some of the more common Prescribed Diseases

This chapter provides basic details about some of the commoner Prescribed Diseases. Each section provides an account of common presentation of the disease and the usual occupational causation. Any available treatment and the likely prognosis are supplied wherever possible. It is intended that this summary should provide just enough information to be of first assistance to a HCP in the medical assessment setting. For fuller details of the medicine the reader is referred to relevant medical textbooks.

As medical understanding of some of the causes and effects of Prescribed Diseases, exposure levels of causative agents, etc, are constantly changing, it is impractical to attempt to keep up to date with changes in all the PDs.

In order to ensure that the advice they give is up to date medically, and in accordance with current legislation and case law, HCPs must seek guidance from the Clinical Project Lead or the Clinical Policy and Projects Lead in all cases requiring their advice on the Chemical (C) Diseases.

For any Prescribed Disease case where they are unfamiliar with the disease under consideration or on any aspect of an industrial injury Prescribed Disease case where assistance is needed, HCPs should seek advice from their local IIB lead, or the IIB helpdesk:

- **Birmingham IIB Helpdesk: 01216262279 (covering Birmingham, Nottingham, Cardiff, Bristol, Wembley and Croydon)**
- **Glasgow IIB Helpdesk: 01412493664 (covering Glasgow, Edinburgh, Newcastle, Leeds, Bootle and Manchester)**

4.1 Prescribed Disease A4

4.1.1 Summary of changes to PD A4

- The prescription of PD A4 has been changed to A4 – Task specific focal dystonia of the hand or forearm
- PD A4 is a task specific focal dystonia of the hand or forearm which is involuntary, sustained muscle contraction causing twisting movements and abnormal posture affecting only one part of the body
- References to “writer’s cramp” and “cramp of the forearm” should not be made
- The claimant should be asked to describe in detail the actions which trigger the abnormal posture

- As part of the clinical examination, the HCPs should not ask the claimant to demonstrate that they have dystonia by performing the action or actions claimed to trigger it. However, if the claimant volunteers to demonstrate the action then he/she should be allowed to do so.

4.1.2 Dystonias

Dystonias are sustained involuntary muscle contractions, often distorting body posture.

Aetiology

Dystonia may be:

- **Primary**

  Idiopathic

  Hereditary, usually as an autosomal dominant disorder with partial penetrance

- **Secondary**

  To degenerative or metabolic CNS disorders (e.g. Wilson's disease, stroke, Hallervorden-Spatz disease, various lipidoses, cerebral palsy)

  To drugs (most often phenothiazines, thioxanthenes, butyrophenones and antiemetics)

Types of dystonia

- **Generalized dystonia (dystonia musculorum deformans):**

  This rare dystonia is progressive and characterized by movements that result in sustained, often bizarre postures. It is often hereditary, usually as an autosomal dominant disorder with partial penetrance; siblings of affected individuals often have a mild form of the disorder. Symptoms usually begin in childhood with inversion and plantar fixation of the foot while walking. Individuals with the most severe form may become twisted into grotesque fixed postures. Mental function is usually preserved.

- **Focal dystonias:**

  These dystonias affect a single body part. They typically start in a person's 30s or 40s and affect women more often. Initially, spasms may be periodic, occurring randomly or during stress; they are triggered by certain movements of the affected body parts and disappear during rest. Over days, weeks or many years, spasms may progress; they may be triggered by movements of

unaffected body parts and may continue during rest. Symptoms vary depending on the specific muscles involved.

Meige's disease (blepharospasm-oromandibular dystonia) consists of involuntary blinking, jaw grinding, and grimacing, usually beginning in late middle age. It may mimic buccal-lingual-facial movements of tardive dyskinesia.

Occupational dystonia consists of focal dystonic spasms initiated by performing skilled acts (e.g. writing, typing).

Spastic dystonia consists of a strained, hoarse or creaky voice due to abnormal involuntary contraction of laryngeal muscles.

Torticollis begins with a pulling sensation followed by sustained torsion and deviation of the head and neck. In early stages, it can be voluntarily overcome.

Diagnosis

Diagnosis is clinical.

Treatment

This is in the first instance one of support and relief of symptoms.

For generalized dystonia, a high-dose anticholinergic drug or reserpine benefits a few individuals. For generalized dystonia treatment is with an anticholinergic drug.

For focal dystonia minimal changes in writing style may help to reduce the dystonia when writing. Use of a thickened pen or writing vertically on a board can help to minimise the problem.

Physiotherapy for the whole arm may relieve some symptoms.

For focal or segmental dystonias or for generalized dystonia that severely affects specific body parts, local injection of purified botulinum A toxin into the affected muscles by an experienced practitioner is the treatment of choice.

Botulinum toxin weakens muscular contractions but does not alter the abnormal neural stimulus. Toxin injection is particularly effective for blepharospasm and torticollis. Dosage varies greatly. Treatments must be repeated every 3 to 6 months.

Surgery, such as deep brain stimulation or selective peripheral denervation, may be performed on cases resistant to all other forms of treatment.

4.1.3 Prescribed Disease A4

Definition

Task -specific focal dystonia of the hand or forearm.

Scheduled occupation

Any occupation involving prolonged periods of handwriting, typing or other repetitive movements of fingers, hands or arms.

Description

Task-specific focal dystonia is an involuntary, sustained muscle contraction causing twisting movements and abnormal posture affecting only one part of the body. In the case of Prescribed Disease A4 the dystonia affects the fingers, hand or arm.

Symptoms arise when the person tries to write or to play a musical instrument or carry out repetitive finger or hand movements. The task activates the dystonia and postures of the hand impede both the quality and speed of execution of the task.

Aetiology

Dystonia is a neurological condition. The main symptoms are involuntary prolonged muscle spasms experienced as repetitive sometimes painful movements and postures. The movements are caused by abnormal function of the motor pathways in the central nervous system.

Symptoms

Symptoms usually begin between 30 and 50 years.

The condition is often described as starting after a period of excessive use, where there has been pressure to increase productivity or meet a target. There may be associated anxiety on the part or the claimant about meeting the extra demands.

Faults in techniques are thought to lead to a cycle of incoordination and even further disturbance in technique.

More commonly the sufferer complains of spasms and discomfort in the wrist, fingers and hand. The greater problem is the posture, which the arm/hand adopts as the task is attempted. The tool is held in strange and uncomfortable fashion, with excessive grip and flexion at the wrist. This exaggerated posture can extend up the arm and create elevation of the shoulder.

Tremor is not usually present.

Aching and pain are uncommon.

Clinical Examination

The clinical examination should include the cervical spine, shoulder and upper limb.

As part of their clinical examination HCPs should not ask the claimant to demonstrate that they have dystonia by performing the action or actions claimed to trigger it. However, if the claimant volunteers to demonstrate the action then he/she should be allowed to do so.

Diagnosis

The diagnosis is made on the basis of the history and exclusion of other possible causes of the symptoms (unless the claimant has demonstrated the dystonia). Thus the claimant should be asked to describe **in detail** the actions which trigger the abnormal posture.

Occupational factors

A history of repeated hand and arm movements within a job should be elicited. E.g. the need to write or type for long periods without rest.

Periods of pressure to speed up, tight deadlines or other factors should also be researched in the history taking.

Details of any work place adjustments should also be recorded.

Social history

Hobby details should be obtained – any finely dextrous activity should be described such as sewing, embroidery, rug making or playing a musical instrument. If such are carried out to excess, then they may prove to be a more reasonable cause than less strenuous finger/hand activities in an occupational setting.

Family history

A family history of dystonia could indicate a genetic causation.

Functional effects

This condition it is essentially a problem with the specific task. Thus overall disability may be minimal. **Note**: The ability to follow one's occupation is not a factor that can be considered when assessing disablement.

Differential Diagnosis

Primary dystonia.

Other causes of secondary dystonia.

Dupuytren's contracture of the hand.

Brachial plexus lesions.

RSI (Repetitive Strain Injury).

Tetany.

Other information

The term 'cramp' is confusing, as the condition does not involve the build–up of lactic acid which is the cause of 'cramp' in every day usage.

RSI **is not** Task-specific focal dystonia.

4.2 Prescribed Disease A6

PD A6 was formerly referred to as "beat knee". It was felt that the term "beat" was not widely used or understood in modern clinical practice and the term had caused confusion and misinterpretation. Thus the reference to "beat" has now been removed from the prescription.

Definition

Bursitis or subcutaneous cellulitis arising at or about the knee due to severe or prolonged external friction or pressure at or about the knee**.**

Occupation

Any occupation involving: Manual labour causing severe or prolonged external friction or pressure at or about the knee.

Background

'Beat Knee' is one of the oldest recognised industrial diseases.

The term 'beat' is a colloquial expression, and may have originated from the throbbing pain often associated with the acute stage of these conditions.

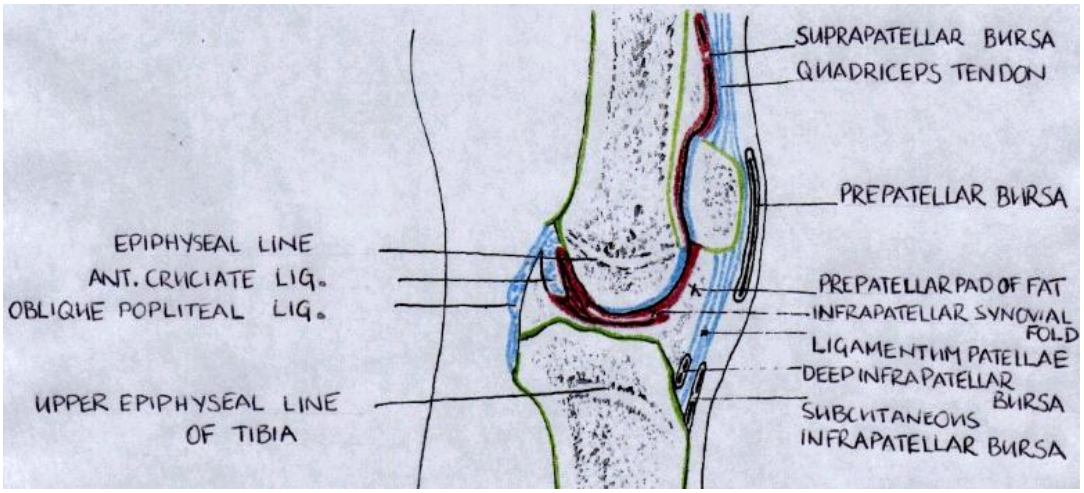
Once relatively common, the incidence of 'Beat knee' and other 'Beat diseases' has declined so that it is not often seen in clinical practice.

As a consequence the term 'beat' has dropped from common usage, and is not always understood by many clinicians. This has lead to 'beat disease' being erroneously diagnosed.

Thus the Industrial Injuries Advisory Council (IIAC) recommended references to the term should be removed from the legislation.

Occupational bursitis and/or cellulitis of the deeper layers of the skin and subcutaneous tissues occurs in any occupation, which involves direct pressure or friction, or repeated minor trauma over a subcutaneous bursa. Thus not all bursae are affected, only those which are close to the skin surface e.g. the subcutaneous prepatellar bursa (which is interposed between the lower part of the patella and the skin); the subcutaneous infrapatellar bursa (which is between the lower part of the tibial tuberosity and the skin. (See Fig 1).

Figure 1



Aetiology

Occupational bursitis and/or cellulitis occurs in occupations demanding much kneeling, and is more common in wet dirty conditions such as occur in coal mining. The condition also occurs in carpet fitters, clergy, and, historically, in house maids. The aetiological factors can be summarised as:-

- Hard manual work involving prolonged kneeling
- Direct pressure and/or repeated direct trauma to the knee.
- Wet, hot conditions leading to maceration (i.e. softening of the skin due to moisture leading to the skin having a reduced protective quality), direct pressure and friction to the skin.
- Occupational bursitis and/or cellulitis often occurs on return to the working conditions after a prolonged absence.

Clinical features

It can be classified into 3 groups

- Inflammatory lesions of the skin (cellulitis)
- Acute simple bursitis or chronic simple bursitis
- A combination of bursitis and cellulitis

Inflammatory lesions

These usually start with an infected hair follicle (folliculitis). In areas not subjected to pressure and friction an infected hair follicle will heal spontaneously. However if it occurs in an area subjected to prolonged direct friction or pressure as in kneeling this spontaneous healing cannot occur and an area of cellulitis develops. Swelling of the underlying bursa due to an effusion may occur as a sympathetic response. There has been a reduction in inflammatory causes over the years, probably as a result of better personal hygiene (e.g. due to the provision of pit head baths).

Acute simple bursitis

In a few cases there is a history of acute trauma in the form of a direct blow to the knee. Most cases develop spontaneously while kneeling at work or after returning home. The area is hot and tender with discolouration of the overlying skin.

Chronic simple bursitis

This is the most common variant. The person presents with a persistent fluctuant swelling. There is often a history of a previous acute episode.

Symptoms and signs

The dominant features are pain and swelling, and thickening of the skin over the affected bursa. Recurrences of the acute state are common.

Diagnosis

Pain and swelling at or around the knee is common, and there is often multiple pathology.

A careful evaluation of the history and clinical examination is required in order to establish causation and relevance to the occupation, and to establish whether all the signs and symptoms relate to bursitis and/or cellulitis or whether there are other effective causes of the symptoms and signs.

Differential diagnosis

A careful history of the condition and clinical examination is required to establish

the correct diagnosis, and the relevance to the occupation. Other causes of pain in the knee should be considered and may co-exist. The differential diagnosis includes:

- Osteoarthritis. The knee is a common site of osteoarthritis, which is often due to wear and tear, and/or previous trauma. Clinical examination will reveal crepitus in the knee (i.e. an audible and palpable grating of bone against bone due to the destruction of the normal joint anatomy), stiffness, pain and limitation of movement.

  (Note that if a person has worked for a definite period of time in certain specified occupations, the presence of osteoarthritis in the knees may fulfil criteria for PD A14 to be diagnosed)

- Inflammatory arthritis (e.g. gout, rheumatoid arthritis, chronic inflammatory polyarthritis)

- Meniscal damage. There will be a history of acute trauma, with subsequent pain and locking of the joint, and clinical examination will reveal instability of the joint, pain on movement and locking of the joint

- Musculo-tendinous lesions at the knee. Injury to the muscles and tendons around the knee can lead to acute pain at the time of the injury. In such injuries the muscles rapidly lose bulk if not used and this can lead to a self-perpetuating knee problem. Clinical examination will show instability of the knee

- Traumatic synovitis. Trauma to the knee can lead to an acute or chronic synovitis. On clinical examination there is an effusion in the knee joint

- Loose body within the knee joint. The loose body may be a piece of bone or meniscus. This can be due to trauma or due to osteoarthritis. They present with recurrent attacks of knee pain, and locking of the joint

- Patello-femoral pain. This is pain at the front of the knee. There are a variety of causes. In older people it is often due to osteoarthritis. In younger people it can be due to chondromalacia patellae

- Other potential causes of cellulitis (see section 4.4.1)

- Non-occupational trauma leading to bursitis

Treatment

- Acute bursitis requires rest
- Aspiration of the fluid may be required
- Chronic bursitis may need surgical excision of the sac
- Antibiotics are required if infection is present

4.2.1 Bursae and Bursitis

Bursae

A bursa is a fluid filled sac-like cavity or potential cavity that contains synovial fluid. Bursae occur in numerous parts of the body. Their function is to act as a buffer between structures to facilitate normal movement without friction between the two surfaces, for example they may lie between two muscles, or between muscle and bone, or between tendons and bone.

Bursae may be fairly constant anatomical structures, or they may be produced in response to external pressure – 'adventitious' bursae.

Some anatomical bursae communicate with the nearby joint and may be distended or diseased if pathology develops in the underlying joint.

Bursae at or around the knee

At, and around the knee, there are usually twelve or thirteen bursae, which are located as follows: four anteriorly; four on the medial aspect of the knee; and 4 or 5 on the lateral aspect of the knee. The actual location of the bursae at the knee are as follows:

a) **Anteriorly**

- The subcutaneous prepatellar bursa which lies between the lower part of the patella and the skin
- The infrapatellar bursa which lies between the upper part of the tibia and the ligamentum patellae
- The subcutaneous infrapatellar bursa which lies between the lower part of the tuberosity of the tibia and the skin
- The suprapatellar bursa which lies between the anterior surface of the lower part of the femur and the quadriceps muscle

b) **Medially** - there are four or five bursa between the bones, muscles and tendons. They do not have specific names.

c) **Laterally** - again there are four unnamed bursae between the various muscles, bones and tendons.

Bursitis

Bursitis can occur at many sites in the body, for example in the shoulder (subacromial or subdeltoid bursitis), the olecranon (miners' elbow), prepatellar (housemaid's knee) or suprapatellar, retrocalcaneal (Achilles), iliopectineal (iliopsoas), ischial (tailor's or weaver's bottom), greater trochanteric, and first metatarsal head (bunion).

Aetiology

Bursitis may be caused by:

- Trauma
- Chronic overuse
- Inflammatory arthritis (e.g. gout, Rheumatoid Arthritis)
- Acute or chronic infection e.g. pyogenic organisms, particularly *Staphylococcus aureus*; tubercule (which now rarely cause bursitis)

Symptoms and Signs

Acute bursitis causes pain, localised tenderness, and limited motion. Swelling and redness are frequent if the bursa is superficial (e.g. prepatellar, olecranon) because the bursal wall secretes a serous effusion when inflamed. Chemical (e.g. crystal-induced) or especially bacterial inflammation is particularly painful, red, and warm.

Chronic bursitis may follow previous attacks of bursitis or repeated trauma. Attacks may last a few days to several weeks, with multiple recurrences. Acute symptoms may follow unusual exercise or strain. The bursal wall is thickened, with proliferation of the synovial lining. The bursa may eventually develop adhesions, villus formation, tags, and calcareous deposits. Pain, swelling, and tenderness may lead to muscle atrophy and limited range of motion. X-ray of the shoulder may demonstrate subdeltoid calcific deposits, particularly in the supraspinatus tendon of the rotator cuff. X-ray of the elbow and the knee may also show calcific deposits.

In gout, crystals may be isolated in the olecranon and prepatellar bursae during acute inflammation.

Diagnosis

Localised tenderness over the particular bursa should be elicited, or swelling or synovial fluid from superficial bursae (e.g. olecranon, prepatellar) should be demonstrated. Infection should be excluded in cases of particularly painful, red, and warm swellings. Periarticular tendon or muscle tears, pyogenic bursitis, bleeding into the bursa, synovitis, osteomyelitis, and cellulitis must be ruled out. Pathological processes may simultaneously involve a communicating bursa and joint.

4.3 Prescribed Disease A7

Definition

Bursitis or subcutaneous cellulitis arising at or about the elbow due to severe or prolonged external friction or pressure at or about the elbow. (Previously referred to as “beat elbow”.)

Occupation

Any occupation involving: Manual labour causing severe or prolonged external friction or pressure at or about the elbow.

Aetiology

There may be a history of a single blow to the elbow, or a series of blows, or continuous pressure on the olecranon process as may occur when working in a confined space causing the person to rest for long periods on his elbows. The causative conditions are similar to those listed for Bursitis of the Knee.

Clinical features

The tissues around the elbow are swollen and painful and there may be signs of acute infection.

There is frequently an associated inflammatory reaction in the bursa over the olecranon process.

The condition is often associated with a history of injury in the olecranon area. Evidence of an abrasion of the skin may be seen.

There may be chronic painless enlargement of the olecranon bursa in miners who work in thin seams or workers who work in confined spaces and who therefore rest on their elbows. Although in these cases there is the potential to develop Bursitis of the Elbow, such swelling is not in itself Bursitis of the Elbow.

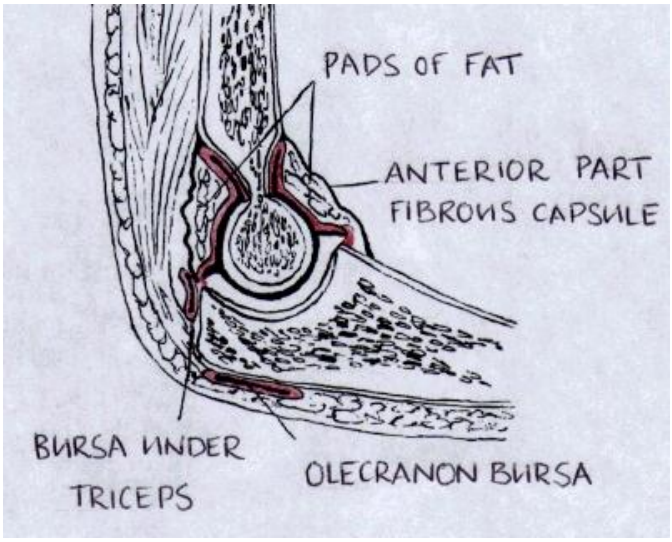


Figure 2

Differential Diagnosis

There are many causes of painful elbow, hence a careful history and clinical examination is required to diagnose Bursitis of the elbow, and to attribute the occupational causation.

Other causes of pain and swelling of the elbow should be considered and may co-exist with occupationally caused bursitis, and so a careful history of the condition and clinical examination is required to establish the correct diagnosis, and the relevance to the occupation.

Non-occupational trauma leading to bursitis.

Olecranon bursitis due to rheumatoid arthritis, gout and other forms of chronic inflammatory polyarthritis.

Musculotendinous lesions including lateral epicondylitis, medial epicondylitis, bicipital tendinitis etc.

Joint lesions, including arthritis, loose body, joint strain.

Nerve entrapments.

4.4 Prescribed Disease A5

Definition

Subcutaneous cellulitis of the hand (Previously referred to as “beat hand”).

Occupation

Any occupation involving: Manual labour causing severe prolonged friction or pressure on the hand.

Aetiology

Beat Hand is due to inflammation and/or infection of the subcutaneous tissues of the palmar aspects of the hand, thumb, or fingers due to a break in the integrity of the skin. Repeated friction and pressure may cause implantation of minerals or other particles. Recurrent bruising may cause devitalisation of the tissues. The condition often occurs when the employee has returned to work after a prolonged absence. Predisposition to the condition may be produced by any environmental factor, which reduces the resistance of healthy tissues to infection, which might include rough tool handles or wet conditions leading to maceration of the skin.

Clinical Features

Local cellulitis develops and may extend to the fascial spaces and tendon sheaths. The acute infection causes pain in the hand with tenderness, swelling, redness and reduced manual dexterity. With appropriate treatment the person should recover completely in a few weeks. However, without successful treatment, poor hand function, scarring and deformities, including contractures, may occur.

Note: Bursae and bursitis do not form part of Prescribed disease A5.

Differential Diagnosis

Non occupationally caused cellulitis.

Dupuytren's contracture.

Palmar ganglia.

Other conditions causing pain in the hands, including tendinitis, tenosynovitis, rheumatoid arthritis, chronic polyarthritis, de Quervain's disease etc.

4.4.1 Cellulitis

This is a diffuse inflammation and /or infection of connective tissue, especially in the loose connective tissue, which lies subcutaneously. There is diffuse, spreading, acute inflammation within solid tissues, characterised by hyperaemia (redness), white blood cell infiltration, and oedema (swelling) without cellular necrosis (cell death) or suppuration (formation of pus).

Aetiology

Cellulitis is caused by many micro-organisms, including

- *Streptococcus pyogenes* (group A haemolytic streptococcus) is the most common cause of superficial cellulitis
- *Staphylococcus aureus* occasionally produces a superficial cellulitis typically less extensive than that of streptococcal origin and usually only in association with an open wound or cutaneous abscess
- When associated with granulocytopenia, diabetic foot ulcers, or severe tissue ischaemia, aerobic gram-negative bacilli (e.g. *Escherichia coli, Pseudomonas aeruginosa*) may be responsible
- Cellulitis occurring after animal bites may be caused by unusual bacteria, especially *Pasteurella multocida* from dogs and cats
- Immersion injuries in fresh water may result in cellulitis caused by *Aeromonas hydrophila*
- Immersion injuries in warm salt water, *Vibrio vulnificus* may cause cellulitis

Symptoms and Signs

- Infection is most common in the lower extremities. A cutaneous abnormality (e.g. skin trauma, ulceration, tinea pedis, dermatitis) often precedes the infection; areas of lymphoedema or other oedema seem especially susceptible.
- Scars from saphenous vein removal for cardiac or vascular surgery are common sites for recurrent cellulitis, especially if tinea pedis is present.
- Frequently, however, no predisposing condition or site of entry is evident.
- The major findings are local erythema (redness) and tenderness, frequently with lymphangitis (inflammation of the lymphatic vessels) and regional lymphadenopathy (swelling of the lymph nodes).
- The skin is hot, red, and oedematous, often with an infiltrated surface resembling the skin of an orange (peau d'orange).
- The borders of the infection are usually indistinct, except in erysipelas, a type of cellulitis in which the raised margins are sharply demarcated.
- Petechiae are common.
- Systemic manifestations (fever, chills, tachycardia, headache, hypotension, delirium) may precede the cutaneous findings by several hours, but many individuals do not appear ill.

- Leukocytosis is common but not constant.

Diagnosis

The diagnosis usually depends on the clinical findings. The responsible organism often is difficult to isolate, even on aspiration or skin biopsy. Blood cultures are occasionally positive. Serological tests confirm a streptococcal cause but are usually unnecessary.

Course and Prognosis

Local abscesses form occasionally, requiring incision and drainage. Serious but rare complications include severe necrotizing subcutaneous infection and bacteraemia with metastatic foci of infection. Even without antibiotics, most cases of superficial cellulitis resolve spontaneously; however, recurrences in the same area are common, sometimes causing serious damage to the lymphatics, chronic lymphatic obstruction, marked oedema, and, rarely, elephantiasis. With antibiotics, such complications are uncommon. Symptoms and signs of superficial cellulitis usually resolve after a few days of antibiotic therapy.

4.5 Prescribed Disease A8

Definition

Traumatic inflammation of the tendons of the hands or forearms, or of the associated tendon sheaths.

Scheduled Occupations

Manual labour, or frequent or repeated movements of the hand or wrist

Description

Inflammation of the tendons or tendon sheaths at the forearm or wrists. The definition includes de Quervain's tenosynovitis which is inflammation affecting the tendons of the thumb - namely abductor pollicis longus and extensor pollicis brevis.

Symptoms

Sufferers complain of localised pain of a distinctive nature in the forearm or wrist. Movement aggravates the pain. Swelling may be present in which case this arises in the tendon sheath. Tenderness to palpation and crepitus may have been noticed.

Aetiology

Inflammation related to repetitive movements of the forearm and hand, particularly of a gripping or pulling or turning nature. Such actions increase friction within the tendon sheath.

Tenosynovitis is a distinct entity and should not be confused with non-specific "umbrella" terminology such as Repetitive Strain Injuries (RSI). The latter is a misnomer as there is often no demonstrable injury. More recently this group of non-specific upper limb complaints has been more suitably called Work Related Upper Limb Disorders. These include a variety of clinical entities, where the complaint is usually pain in an over use situation. Whilst this very broad definition can be considered as including 3 Prescribed Disease (PD A8, PD A4 or PD A12) most who complain of RSI do not have a Prescribed Disease.
HCPs considering PD A8 claims should avoid this terminology and concentrate on:

- Advising on the Diagnosis of Tenosynovitis as it has been described above (Inflammation of the tendons or tendon sheaths at the forearm or wrists. Including De Quervain's Tenosynovitis which is inflammation affecting the tendons of the thumb - namely abductor pollicis longus and extensor pollicis brevis)

and

- Confirmation of a causal link with the scheduled occupation

Diagnosis

The history should include an account of the symptoms and the presence and site of pain and any exacerbating or relieving factors. The pain is likely to improve at rest and during periods away from work. If the condition becomes chronic this relationship with work/rest becomes less clear cut. The time of onset is relevant and it should be discovered if this relates to a period of increased or changed work routine.

A past medical history of previous arm or neck pain may point to other diagnoses.

The HCP should also seek a detailed occupational history, looking for a clear connection between use of the arm/hand and the onset and exacerbation of forearm pain. The ergonomic nature of the specific work tasks should be outlined.

A functional approach to history taking is a valuable tool in approaching an assessment of disablement. A Typical Day account will show to what extent a claimant can use the upper limbs in routine daily tasks such as dressing, cooking shopping etc.

Informal observations of upper limb movements and hand use during the interview and clinical examination are also of considerable value, and should be recorded in the report. Spontaneous gripping, lifting and carrying actions will all point to a level of function which may be at odds with the claimant's own descriptions of limited functional ability.

Clinical examination may show local swelling and tenderness at palpation.
Crepitus may also be present, but its identification is not essential for the diagnosis to be confirmed. A fuller evaluation of motor skills and manual dexterity is also required.

A positive Finkelstein's test should be demonstrated - ulnar deviation of the wrist when the fingers are flexed over the thumb as it is rolled into the palm creates pain over the radial aspect of the wrist as the affected tendon is stretched.

Functional Effects

When PD A8 is diagnosed in the first instance problems usually arise with the specific work task, but any other similar movements will exacerbate the pain. This may affect the ability to prepare food, dress certain items of clothing, arrange the hair, sew, garden, DIY and other hobbies requiring grip and repeated hand action may no longer be possible.

Causation

Not all tenosynovitis is caused by work activity; some may arise from actions completed out of working hours. The Presumption is that PD A8 is caused by a Scheduled occupation. (Remember that presumption only applies when the person is in the prescribed occupation or within a month of leaving. Even if this applies, presumption may be rebutted if there is evidence to do so). If there is strong evidence of repetitive actions in a hobby or domestic task, and that is the more probable cause, then the HCP can advise that the tenosynovitis is not due to the accepted occupation.

Convincing evidence is required to support such advice and must confirm that the claimant has been at greater exposure outside the workplace than within it.

Treatment

The following are usual treatments for this condition. Rest alone can serve to correct the problem, but a rushed return to the causative activity can slow recovery.

- Avoidance of the identified causative movements
- Anti inflammatory drugs
- Rest of the limb

- Intra -synovial steroid injections
- Surgical release of any tethering in chronic conditions
- Occupational Health Physicians consider return to work after investigation of workplace practices, ergonomic assessments and implementation of appropriate adjustments. This may include job rotation, ensuring sufficient rest periods and automation of repetitive processes

Differential Diagnosis

The HCP should consider and exclude the following possible alternative diagnoses:

- Epicondylitis of elbow - Golfers and tennis elbow
- Osteoarthritis of the carpus
- Cervical spondylosis with root pain
- Carpal Tunnel syndrome

The HCP should keep in mind the possibility that these conditions may co-exist with a diagnosed PD A8, in which case they will constitute another effective cause of disablement in the upper limb.

The HCP should also consider other Prescribed Diseases such as PD A4 (Task specific focal dystonia) and PD A12 (Carpal Tunnel Syndrome).

4.6 Prescribed Disease A11

Date included in legislation: 01/04/1985
Date Regulations revised: 01/10/2007

Note: The terms "Vibration White Finger" and “Hand - Arm Vibration Syndrome" **must not be used when completing a report on PD A11**.

Definition

Vibration damage to the nerves and/or blood vessels of the fingers.

4.6.1 Scheduled Occupations

(a) The use of hand-held chain saws on wood; or

(b) The use of hand-held rotary tools in grinding or in the sanding or polishing of metal, or the holding of material being ground, or metal being sanded or polished, by rotary tools; or

(c) The use of hand-held percussive metalworking tools, or the holding of metal being worked upon by percussive tools, in riveting, caulking, chipping, hammering, fettling or swaging; or

(d) The use of hand-held powered percussive drills or hand-held powered percussive hammers in mining, quarrying, demolition, or on roads or footpaths, including road construction; or

(e) The holding of material being worked upon by pounding machines in shoe manufacture.

4.6.2 Description (Prescription) for PD A11

(a) Intense blanching of the skin, with a sharp demarcation line between affected and non-affected skin, where the blanching is cold-induced, episodic, occurs throughout the year and affects the skin of the distal with the middle and proximal phalanges, or distal with the middle phalanx (or in the case of a thumb the distal with the proximal phalanx) of:
(i) in the case of a person with 5 fingers (including thumb) on one hand, any 3 of those fingers, or
(ii) in the case of a person with only 4 such fingers, any 2 of those fingers, or
(iii) in the case of a person with less than 4 such fingers, any one of them, or as the case may be, the one remaining finger,
where none of the person's fingers was subject to any degree of cold induced, episodic blanching of the skin prior to the person's employment in an occupation described above, or

(b) Significant, demonstrable reduction in both sensory perception and manipulative dexterity with continuous numbness or continuous tingling all present at the same time in the distal phalanx of any finger (including thumb) where none of the person's fingers was subject to any degree of reduction in sensory perception, manipulative dexterity, numbness or tingling prior to the person's employment in an occupation described above,

where the symptoms in paragraph (a) or paragraph (b) were caused by vibration.

Background

In 2004, the Industrial Injuries Advisory Council conducted a review of PD A11 and recommended widening the terms of prescription of the disease to include severe sensorineural symptoms. Previously, only those with vascular symptoms at a level stated in the legislation were likely to be successful in their claim for Industrial Injuries Benefit.
These recommendations were accepted by the DWP and the regulations were made on 19th June 2007 and came into force on the 1st October 2007.

Aetiology

It is now recognised that vibration can affect the small sensory nerves of the fingers exposed to hand-held vibrating tools. The symptoms (i.e. tingling and numbness) arising from this damage usually pre-dates the onset of vascular symptoms, and may occur in the absence of vascular symptoms. The vascular symptoms are caused by vasospasm in the hands.

It should be noted that PD A11 is a distinct entity, which has been clearly defined in the regulations in both its nature and degree. Whilst medical specialists can look at the broad spectrum of vascular and sensorineural impairment, the HCP for PD A11 is confined to the very precise definition set out in the regulations. Therefore terms such as HAVS, VWF and Raynaud's must be avoided in any PD A11 report.

Some background information for readers is included in Appendix 3 relating to Raynaud's Disease, VWF and HAVS. This is strictly for background information for HCPs who may see references to these conditions submitted by the claimant.

4.6.3 Diagnosis

- **History taking**

In the IIAC report (CM6098), the Council stated that the gold standard for diagnosing PD A11 on the vascular criteria is a good clinical history.
History taking should be conducted in accordance with The Centre for Health and Disability Assessments' standards. As with all history taking, open questions and active listening techniques must be utilised.
It is essential in the PD A11 history that the claimant is encouraged to give a clear account of the chronology of changes in the hands over time. Therefore, the history must go back far enough to allow demonstration of the slow progression of the condition in conjunction with use of hand held vibrating tools or the holding of materials being worked upon by pounding machines. It is essential to establish whether any symptoms were present prior to exposure to vibration.

Detailed symptomatology is essential and the following information details essential points in the history that must be obtained in all cases.

- When and how symptoms started. The claimant should be encouraged to explain symptoms and asked what they mean by numbness or tingling or blanching if they use these terms.

- How the symptoms progressed over time. (Remember that PD A11 is a very slowly progressive condition). Demonstration of this is a convincing aspect of the history. A history of rapid onset disease, with involvement of all fingers in their entirety would not normally be consistent with vibration induced damage.

It is essential to try to obtain time scales on when the symptoms progressed to a level that would meet the criteria for the diagnosis of PD A11. As the disease is slowly progressive, it may be useful to encourage claimants to identify timescales using “life events” e.g. birth of children, weddings etc.

- What makes them better? As an example, sometimes immersion in warm water speeds recovery
- What makes them worse?
- Details of the manifestation of the colour change. In vibration induced blanching, there is usually clear demarcation between the normal and abnormal appearance of the skin. The abnormal appearance of the skin would be circumferential.
- How long does the blanching last?
- What happens on recovery? Do fingers become cyanotic of hyperaemic?
- Is there intermittent or persistent tingling and numbness in the tip of the fingers during an attack of blanching and independent of blanching
- Which symptoms arose first, the sensorineural or vascular?

- **Differential Diagnosis**

The possibility of an alternative diagnosis must always be considered, and the history must be adequate to cover this aspect.

Some areas, you may wish to specifically address include:

- The possibility of constitutional Raynaud’s disease or secondary causes of Raynaud’s phenomenon
- A detailed account of the dermatomal distribution of the numbness or tingling which may suggest peripheral nerve entrapment or damage from another cause. Examples include carpal tunnel syndrome, ulnar nerve entrapment at any level, cervical rib, and cervical spondylosis with nerve root involvement
- Consider whether symptoms appear consistent with a peripheral neuropathy of any cause (for example diabetic, alcohol, or drug induced)
- A history of previous trauma to the upper limbs should be elicited
- Rare causes of neurological disturbance due to poisoning should be considered e.g. ergot, nicotine
- Any history of connective tissue disorder must be explored

- History of vascular disease
- Work related upper limb disorders

- **Functional symptoms**

You must also ensure you obtain adequate information on the functional impact of their symptoms. This is best done through a typical day approach to ascertain if there are problems with everyday tasks involving fine dexterity. As experienced Healthcare Professionals, you should already be familiar with this concept.

- **Occupational History**

An occupational history must be obtained. This must be detailed and it is essential to cover the following:

- Full occupational history, including the nature of tools used, the percentage of the day spent using these tools, and the length of time in years using these tools.

  You should also ensure you enquire about progression of symptoms if there has been a period of time in the occupational history where exposure to vibration ceased

- It is also essential to enquire regarding progression of symptoms after ceasing the occupations involving exposure to vibration. Although the damage to blood vessels and nerves does not recover, once exposure to vibration ceases, there may be a slight improvement in the symptoms. Symptoms due to vibration do not worsen once exposure to vibration ceases. If there has been a worsening, some other cause of the symptoms must be explored

- Onset of symptoms once exposure to vibration has ceased must be carefully detailed. The consensus of opinion is that if symptoms develop more than a year or two after exposure to vibration has ceased, then the cause is not exposure to vibration, and thus there is some other cause of the symptoms

- If symptoms were present **before** exposure to vibration, then the symptoms are not due to vibration. However, care must be taken to ensure early “forgotten” exposure was **not** missed e.g. during an apprenticeship. It is therefore essential to cover a full occupational history to ensure you can accurately state there was indeed no exposure to vibration prior to the onset of symptoms

- **Past Medical History**

You must enquire about past medical history in considering possible other causes of sensory or vascular symptoms. Examples include:

- Previous upper limb fractures

- Connective tissue or rheumatological disorders
- Neck pain or injury
- Brachial plexus injuries
- Possible causes of neuropathy
- Vascular disorders

- **Family History**

A family history of Raynaud's phenomenon should be sought.

- **Social History**

In this area, you should enquire whether there may have been any potential for exposure to vibration as a result of hobbies pursued.

This may be adequately covered in the typical day history as long as the HCP is mindful of exploring past hobbies as well as current hobbies.

- **Clinical examination in PD A11**

As it is unlikely that an episode of blanching will be witnessed during an assessment, the clinical examination you perform must be directed towards the exclusion of other conditions.

The Department has provided a steer on the level of clinical examination thought to be essential in PD A11 claims.

Musculoskeletal System

The musculoskeletal system must be assessed using a functional examination approach. This should be approached in the normal MSO (musculoskeletal overview) manner and progress to a more focussed regional examination where abnormality is evident. You must carefully assess the cervical spine, shoulders, elbows and hands, looking for any evidence of arthritis, scarring (surgical or traumatic), circulatory problems, trophic changes and muscle wasting.

Central Nervous System (CNS)

The CNS must be examined with particular emphasis on sensory testing. Testing of power will usually have been performed as a part of the MSO. You may wish to consider testing reflexes if clinically indicated.

Sensory testing is of particular importance in assessing revised PD A11 claims.

Appropriate testing of sensation may include:

- Touch

- Position sense
- Vibration sense
- Two-point discrimination

HCPs should be reminded that testing of sensation to pain would not be considered appropriate in order to avoid claimant discomfort. They should also bear in mind that there are no appropriate facilities in the assessment centres for disposal of clinical sharps.

Vascular Examination

Examination for vascular disorders is also very important. You should check the extent of perfusion of the upper limbs and exclude a cervical rib.

Other Appropriate Examination

Testing should be performed to exclude other upper limb prescribed diseases such as PD A12 and PD A8. These may include Finkelstein's, Tinel's and Phalen's tests.

Adson's test may be used to detect possible thoracic outlet obstruction.

A brief outline of these tests is included in appendix 5.

- **Formal Examination Techniques**

Formal examination of dexterity should be performed using the Purdue Peg Board.

A formal assessment of grip should be made using the Jamar Dynamometer.

- **Observation**

As experienced Healthcare Professionals, you will be very aware of the value of observation.

Observation of fine dexterity is an essential part of the assessment, e.g. noting how the claimant copes with buttons, tying shoe laces, or the use of a pen.

4.6.4 Formal testing of Grip and Manual Dexterity

The Industrial Injuries Advisory Council recommended that the use of Purdue Peg Boards and Jamar Dynamometers be incorporated into the routine medical assessment of PD A11.

It should be remembered that these tests are non specific and they rely on claimant co-operation. The results may assist in confirming level of dexterity and grip strength, however must always be interpreted within the context of the history, observed behaviour and other clinical examination findings.

The Jamar Dynamometer

The Jamar hand Dynamometer has been used for many years in assessing grip strength.

The equipment will come with a manual provided with normal reference ranges and instructions on the use of the Jamar.

The Dynamometer provides a reading of grip force through the claimant squeezing a handle. The handle has almost no perceptible movement to enable more accurate results.

- The claimant should then be instructed to sit or stand comfortably, with their shoulder adducted and the elbow flexed to 90 degrees. The forearm and wrist should be in a neutral position
- The claimant should then be instructed to squeeze the handle with maximum strength. This is repeated 3 times. After each “squeeze”, the Jamar has to be reset to the zero reading
- Readings are taken for both hands and should be compared to the normal reference ranges provided in the manual. This should be interpreted in context with all other findings in the clinical examination

The Purdue Peg Board

The Purdue Peg Board was developed in 1948 by Joseph Tiffin, an industrial psychologist at the University of Purdue.

It has been used for many years as a method of assessing manual dexterity but can also give an indication of tactile sensibility.

It should be noted that the Purdue Peg board is a non specific test.

The pegboard is used in a standard manner.

- The pegboard is placed on a table of approximate height 760 mm (average desk height) directly in front of where the subject will be seated and orientated with the row of cups (which hold the pins) at the far end of the board
- The claimant should sit comfortably at the table
- An explanation of the process should be given to the claimant. They should be told this test is to see how quickly and accurately they can work with their hands
- The claimant should be instructed to begin with their dominant hand

- Instruct them to pick a pin up from the right cup and place it in the furthest away hole on the right side of the board. Tell them they will be expected to do this as quickly as possible

- Allow them to practice a few holes. When they appear to have grasped the concept, instruct them to remove the pins and place them back in the cup

- They will then proceed to the actual test. You should tell them to begin to fill the holes with the pegs as quickly as possible on your command and stop when you instruct them to do so. The claimant should be given exactly 30 seconds to fill as many holes as possible with the pins

- You should then count the number of pins inserted and record this

- The process should then be repeated with the non dominant hand, again allowing a few practice pins. The number of pins inserted with the left hand should then be recorded

- The process should then be repeated but on this occasion using both hands. The number of pins in this case is recorded as the number of pairs inserted. Again, you should allow a few practice pins to be inserted

- The scores for each method should then be compared to normative data. A reduction in the number of pegs inserted **may** suggest a reduction in manual dexterity and aid the HCP in deciding whether the claimant may fulfil the prescription for PD A11. (Remember that for sensorineural only PD A11, a diagnosis cannot be confirmed without further referral for sensory testing through Thermal Aesthesiometry and Vibrotactile testing)

4.6.5 Thermal Aesthesiometry & Vibrotactile Testing

Introduction

On the recommendation of IIAC, it has been decided that in cases where there is a history suggesting **severe sensorineural involvement** (i.e. significant, demonstrable reduction in sensory perception and manipulative and persistent numbness or persistent tingling – all present at the same time), thermal aesthesiometry and vibrotactile testing should be used to confirm the symptoms reported by the claimant.

Not all claimants will be referred for testing. **Only those claimants who do not fulfil the vascular prescription for PD A11 yet fulfil all the criteria listed in the legislation relating to sensorineural PD A11, should be referred.**

The HCP must **only** refer cases for Thermal Aesthesiometry and Vibrotactile Testing when the vascular criteria for diagnosis are not fulfilled but all sensorineural criteria appear to be present.

Testing will not be required where:

- PD A11 can be diagnosed on the basis of the vascular symptoms
- Sensorineural symptoms developed before the onset of exposure to vibration
- Sensorineural symptoms are to a lesser degree than detailed in the legislation, i.e. tingling and / or numbness is intermittent rather than continuous
- There is no demonstrable reduction in sensory perception
- There is no reduction in manipulative dexterity
- History and clinical examination findings suggest a non-vibration related cause for the sensory symptoms

Where testing is thought to be appropriate the HCP should adjourn the assessment, the claimant will be given a second appointment to return for assessment by another, specially trained HCP.

(HCPs should discuss any case requiring adjournment with their local IIB lead or IIB helpdesk prior to adjourning the case.)

Once this testing has been completed the file, with the result of testing, should be returned to the relevant BSC (Business Support Centre) for completion, of diagnosis advice and, where appropriate, assessment advice, with appropriate justification.

These tests have been devised to confirm that there is a significant reduction of sensory perception; however it should be noted that:

- The tests are not specific to vibration induced damage. Whilst they can show that there is some damage to the nerves, they do not indicate the cause of the damage.
- The tests do not show what effect the nerve damage has on function and are therefore of no assistance in the assessment of disablement in PD A11.

Some background information on the testing procedures is included in Appendix 4.

Interpretation of Tests

When interpreting the test results, it is essential that it is borne in mind that the tests only indicate the sensitivity of the claimant in detecting change in temperature or the ability to feel vibration. They are not specific tests but can help confirm whether neurological dysfunction has occurred.

A protocol has been developed between the DWP and Southampton University for the purposes of revised PD A11 to establish a consistent approach to testing and interpreting these tests.

The following data is extracted from the document written by Professor Michael J Griffin, Human Factors Research Unit, Institute of Sound and Vibration research, University of Southampton "The protocol for testing to confirm the severity of sensorineural damage in claims for the revised Prescribed Disease A11".

In normal persons unaffected by hand-transmitted vibration there is a wide range of thermal and vibrotactile thresholds. Prolonged exposure to severe hand - transmitted vibration causes the thresholds to be elevated sufficiently for them to be considered 'abnormal' and indicative of peripheral neurological disorder.

For the standardised tests, the distributions of normal thresholds for the perception of temperature and vibration are known. As a threshold becomes extreme, the probability of the threshold being present in normal persons reduces. The criterion for abnormality corresponds to the mean threshold of normal persons plus two standard deviations of the thresholds of normal persons. Only 2.5% of normal persons will have a threshold that reaches this criterion – so it is indicative of abnormality in all except 1 in 40 persons.

Criteria for abnormality

Abnormal thresholds are: hot thresholds greater than 48.5°C, cold thresholds lower than 19°C, 31.5 Hz vibration thresholds greater than 0.4 ms-2 r.m.s., and 125 Hz vibration thresholds greater than 1.0 ms-2 r.m.s. (see Table below).

It is not necessary that all thresholds reach the criteria – it is sufficient for one temperature threshold and one vibration threshold to indicate abnormality and provide confirmation of the symptoms.

Abnormal thresholds

Hot Thresholds	Greater than 48.5º C
Cold thresholds	Less than 19ºC
31.5 Hz vibration threshold	Greater than 0.4ms² r.m.s
125 Hz vibration threshold	Greater than 1.0ms² r.m.s

The information in this section of the training should allow the HCP to understand the basic principles of thermal aesthesiometry and vibrotactile testing but is not intended to explain the process in great technical detail. In the company, certain HCPs will be specifically trained in the operation of these machines and will be responsible for the testing and ensuring the tests are conducted in the appropriate manner.

For both Thermal Aesthesiometry and Vibrotactile testing, the test environment must be controlled. The ambient temperature of the room must be between 20-30º C. The finger temperature must also be measured prior to both tests and be in the range of 27 - 37ºC.

The fingers to be tested are as follows:

Thermal and vibrotactile thresholds should be measured on two fingers of both hands.

The fingers to be test on each hand are selected as follows:

- The index and little finger if the claimant has complained of symptoms in these fingers
- If either the index finger and/or the little finger are without symptoms, substitute one or two fingers in which the claimant has complained of symptoms
- If there are symptoms in only one finger, test that finger and the index finger
- If there are symptoms only on the index finger, test that finger and the little finger

4.6.6 Differential Diagnosis in PD A11

HCPs should be mindful of the many alternative causes for sensorineural impairment and vascular disturbance. Symptoms may arise from conditions such as a cervical rib, cervical spondylosis and other musculoskeletal disorders. Conditions such as Raynaud's Syndrome must also be considered along with other prescribed diseases such as PD A8 or PD A12. A careful history and clinical examination as detailed above should assist the HCP in the diagnosis of PD A11.

4.6.7 Assessment of Disablement in PD A11

The assessment of disablement in IIB is made on the basis of a comparison with a person of the same age and sex whose physical and mental condition is normal. The ability to follow the regular occupation, loss of earnings or additional expenses arising from the disease are irrelevant.

HCPs should apply the following principles when assessing PD A11:

- Does the PD result in personal injury?
- Is there a loss of faculty (LOF)? There is deemed to be no LOF if the resulting disablement is assessable at less than 1%. However, the Upper Tribunal Judges have pointed out that it is desirable to distinguish between cases where there is no LOF and those where there is one, but the resultant disablement is less than 1%

- What is the LOF? For example in PD A11 this could be intermittent loss of normal vascular function, with intermittent (or continual) neurological disturbance; persistent neurological disturbance

- What is the disability? The functional area affected by PD A11 is the performance of fine manipulative skills (i.e. manual dexterity), e.g. picking up small objects (pins, coins, etc); the ability to fasten/unfasten buttons; the ability to fasten/unfasten shoelaces; the ability to write etc.

  The disability in PD A11 should be cited as 'impaired manual dexterity'. (Note: 'upper limb dysfunction' covers a much broader range of potential disabilities, and thus should be avoided when describing the disability arising from PD A11)

- Are there other effective causes of the disability, either arising before the onset of the Prescribed Disease (O Pre conditions), or arising after the onset (O Post conditions)? Remember – pre-existing vascular symptoms, and pre-existing symptoms and signs of sensorineural damage exclude a diagnosis of PD A11, but there may be other pre-existing causes of disability e.g. amputation of a finger

- What is the disablement? HCPs are reminded that the assessment is a functional assessment, i.e. the effects of the medical condition not the medical condition itself. Hence the assessment of disablement is based on what the person cannot do in comparison to the norm for his/her age

- The disabling effects can be from vascular and sensorineural symptoms.

- The extent of vascular or sensorineural symptoms must be of sufficient severity to meet the requirements of prescription before PD A11 can be diagnosed. However once diagnosed the disabling effects of either types of symptoms should be included in the assessment – e.g. if diagnosed under vascular criteria the effects of any intermittent sensorineural symptoms should be included in the assessment. Likewise in cases diagnosed under sensorineural criteria assessment should include the disabling effects of any intermittent blanching

Note:

- The medical understanding of the effects of vibration have evolved since PD A11 was first prescribed in 1985. The disabling effects of the vascular component of PD A11 are now considered to be minimal, the disabling effects arise from the effects of sensorineural component

- The episodes of blanching may temporarily affect manual dexterity, but this is usually measured in minutes rather than hours and the assessment of disablement should therefore reflect this

- The Stockholm Scale is a clinical grading of HAVS which was proposed in the 1980s by an international meeting of experts. It consists of two scales - one for vascular and one for sensorineural symptoms. It has been used widely in epidemiological surveys, and clinical and medico-legal practice, although the scales do contain some ambiguities. (See appendix 6 for details of the scales and further information)

- Unless the sensorineural symptoms have progressed to 3Sn persistent i.e. the severest level of sensorineural involvement on the Stockholm Scale, the effects of the condition are unlikely to be more than an intermittent annoyance. The legislation reflects this for sensorineural only disease, but the same applies to cases which are diagnosed on the vascular criteria. The assessment of disablement should therefore reflect this

- If the informal and formal clinical examination has revealed little, if any, diminution in function when compared to a person of the same age and sex, then the assessment should be low, with a nominal award for the loss of an intact vascular and/or neurological system

- Even those with persistent symptoms must be assessed on what the person can/cannot do in comparison to the norm for their age. Dexterity and limb function deteriorate with age and this must be taken into account in the assessment

- The assessment of disablement takes into account the assessments laid down in the legislation, and all assessments should be related to the Scheduled assessments

The percentage assessment of disablement is extrapolated from the Scheduled assessments. In equating the loss of function resulting from PD A11 the following should be considered:

- What has the person said in his/her history regarding functional loss?
- How frequent are the attacks – the less frequent they are, the fewer times the function is abnormal, and the more frequent the person has normal function?
- How many fingers are involved?
- Which fingers are involved? The involvement of the index finger causes more disability than the involvement of the little finger
- How old is the person?
- Does the history equate with the formal clinical examination findings e.g. is there some demonstrable loss of sensation?
- Does that loss of sensation correlate with the distribution of the nerves

in the hand?

- Do the history and the formal clinical examination findings correlate with the informal observations that the HCP has made throughout the assessment. For example could he/she undress/dress unaided; could he/she tie shoelaces; could he/she pick up a pen; write their name; pick up a small object when not being formally examined

What assistance are the Scheduled assessments? For example, the loss of an index finger is 14%; the loss of the tip of the index finger with no loss of bone is 5%.

Thus, when assessing disablement always bear in mind the following

- How does the claimant equate to a normal person of the same age and sex? It is normal for an older person to have loss of dexterity in comparison to a young person

- How does the disablement in this case compare to those in the Scheduled assessments? For example, is there any justification for assessing disablement at say 25% when there is only an intermittent loss of manual dexterity, and non-disabling tingling? Compare this with the disablement resulting from the amputation of the tips of all the fingers of one hand. That would only attract an assessment in the order of 22 to 25%. (Note: the functional loss of all the tips of the fingers does not necessarily equate to the sum of the individual losses – such a case would require the combined loss of function to be assessed, and there has been an Upper Tribunal Judge's decision on this aspect of the assessment of disablement – R(I)39/61. The individual loss of the tips of the 4 fingers, not including the thumbs would result in an assessment of 13%)

The intermittent vascular symptoms (i.e. when the fingers are blanched) make the fingers clumsy on an intermittent basis. This intermittent clumsiness affects the fine manipulative skills (such as picking up a pin, fastening a button), but not on a continuous basis. Similarly the intermittent tingling and numbness due to the sensorineural involvement causes intermittent interference with fine manipulative skills (as above). The assessment of disablement in these cases will be low.

Even in cases with the persistent sensorineural symptoms (3Sn late) on Stockholm scale, the sensory loss is not equivalent to the loss of the finger tip as the sensory parameters are **not** lost, but there is diminution (numbness) and altered awareness (tingling). If **all** sensory perception were lost there would be evidence of injury and of trophic changes as the person would not be aware of damaging his finger tips. Trophic changes (i.e. gangrenous changes – stage 4 on the Stockholm Scale) would equate to the loss of finger tips, but such changes in PD A11 are **very** rare, and some authorities do not believe that vibration damage can lead to trophic changes.

The loss of dexterity caused by vascular and sensorineural symptoms of PD A11 rarely, if ever, equates to the loss of the tips of the fingers or the effects of the

loss of the index finger (NB it is rare for the thumb to be affected by vibration). Thus the assessment rarely exceeds 10% as the effects are less than the effects such as occur with the loss of an index finger and most assessments of disablement are in the order of 2 - 8% depending on the functional limitations. The assessment of disablement rarely exceeds 10% as it is rare for the effect on manual dexterity to equal or exceed the loss of an index finger. Only in cases where there is tissue loss would an assessment above 14% be warranted. **However**, the current consensus of medical opinion is that trophic changes are unlikely in vibration induced damage. Hence if trophic changes are present the HCP should re-consider his/her opinion on causation.

4.6.8 Date of Onset in PD A11

The onset of the disease is gradual and usually over a number of years. Only when the blanching and/or the sensorineural criteria are to the extent detailed in the legislation, can PD A11 be diagnosed, irrespective of when other symptoms first developed.

It is important to ascertain the date of onset of blanching and/or sensorineural symptoms to the extent they fulfil the criteria for the diagnosis of PD A11. Whilst only in very rare cases will the assessment of disablement be more than 14%, if the date of onset **pre-dates** October 1990 **and PD A11 is diagnosed using the vascular criteria**, then the claimant may be eligible for Reduced Earnings Allowance (REA).

As an extension to the Prescribed Disease, diagnosis of the disease using the sensorineural component does not fulfil the qualifying criteria for REA, irrespective of the date of onset.

When completing the justification of their assessment (in Part 7 of the BI613), to avoid causing any confusion or doubt for the DM, the HCP should clearly advise that it includes the effects of both the vascular and sensorineural symptoms.

4.7 Prescribed Disease A12

Definition

Carpal tunnel syndrome (CTS)

Date included in legislation: 19/04/1993
Date Regulations amended: 06/04/2007

Scheduled occupations

(a) The use, at the time the symptoms first develop, of hand-held powered tools whose internal parts vibrate so as to transmit that vibration to the hand, but excluding those tools which are solely powered by hand; or

(b) repeated palmar flexion and dorsiflexion of the wrist for at least 20 hours per week for a period or periods amounting in aggregate to at least 12 months in the 24 months prior to the onset of symptoms, where “repeated” means once or more often in every 30 seconds.

4.7.1 Carpal Tunnel Syndrome

The Median nerve enters the hand by passing through a tunnel formed by the convex anterior surface of the carpal bones and a ligament - the flexor retinaculum. As well as the Median nerve, the flexor tendons (i.e. the tendons which bend the fingers) of the fingers run through this tunnel. This is known as the Carpal Tunnel. The Ulnar and Radial nerves **do not** enter the hand via this tunnel.

Alterations in any of the anatomical structures in the tunnel can result in a loss of volume in the available space (which at best is limited) in the carpal tunnel and so produce compression of the median nerve: Carpal Tunnel Syndrome.

Aetiology

Carpal tunnel syndrome is caused by compression of the median nerve as it passes deep to the flexor retinaculum. However in relation to the use of vibrating tools, the pathology may be due to physical trauma to the nerve itself and/or blood vessels to the nerve, which may explain the poor results with carpal tunnel decompression in people with vibration induced Carpal Tunnel Syndrome.

Carpal Tunnel Syndrome is very common in the population as a whole, and it is more common in women than men. In women it occurs in 7 per 100 and in men 1 per 100.

Most cases of Carpal Tunnel Syndrome arise spontaneously (i.e. there appears to be no trigger causing it).

In 30% of cases there is an underlying medical condition:

- It may follow prolonged use of the wrist by arthritic people who need to use a walking stick
- Swelling of the tendons due to a tenosynovitis, e.g. Rheumatoid arthritis produces an inflammatory tenosynovitis of the flexor tendons or synovitis of the wrist joint. Carpal Tunnel Syndrome may also be associated with previous serious trauma, such as Colles' fracture or subluxation of the lunate (one of the carpal bones)
- Osteoarthritis of the wrist or ganglion formation
- It may also occur during the last trimester of pregnancy or as part of the premenstrual syndrome, presumably due to fluid retention
- Various metabolic diseases, including gout, hypothyroidism, acromegaly, fluid retention in renal disease or amyloidosis

- Exposure to vibration
- Overuse of the wrist, e.g. in actions which involve repeated flexion and extension of the wrist. In its recent report the Industrial Injuries Advisory Council (IIAC) reported that in order to develop Carpal Tunnel Syndrome the repeated flexion and extension should occur every 30 seconds or more often for at least 20 hours per week, and the duration of the qualifying employment should exceed a year

Because the condition is so common, and can have different causes each case needs careful evaluation to establish the actual cause (if any is identifiable) in an individual claimant.

If vibration is claimed as the cause of Carpal Tunnel Syndrome, a history of vibrating tool use within a particular occupation should be elicited. The following should be noted:

- Vibrating tool type
- Wrist posture whilst using the tool
- Weight of the tool. (The heavier the tool, the more likely)
- Frequency and duration of tool use
- Effects of change of job role/occupation to avoid the use of vibrating tools
- Relationship of onset of symptoms to use of tools

If repeated flexion and extension (palmar and dorsiflexion) is claimed as the cause of the Carpal Tunnel Syndrome the following should be noted:

- Wrist posture whilst performing work
- Frequency and duration of palmar and dorsiflexion whilst performing work
- Effects of change of job role/occupation to avoid the action
- Relationship of onset of symptoms to occupation
- Tool type and weight of the tool (if any involved in task)
- Wrist posture whilst using the tool
- Where possible the claimant should be asked to demonstrate the actions involved in their work

NB: IIAC considered the use of keyboards and mouse and advised that there was insufficient evidence to include keyboard and mouse use in the prescription of PD A12.

Symptoms of Carpal Tunnel Syndrome

- The affected person is usually a middle-aged female and one or both hands, but more commonly the dominant hand, may be involved

- Paraesthesia (tingling, numbness, pins and needles, burning) in the parts of the hands supplied by the Median nerve especially at night, and are often relieved by the person moving the hands about or by hanging them over the edge of the bed or above the shoulder

- The symptoms are characteristic and are most severe at night, when it commonly disturbs the person's sleep

- The person may also complain of an unpleasant sensation of fullness or stiffness in the fingers, especially in the morning, or that the fingers feel clumsy or weak

Clinical signs and investigations of Carpal Tunnel Syndrome

Clinical signs are:

- The symptoms may be reproduced by various manoeuvres that are designed to produce increased compression of the median nerve in the carpal tunnel

  This may be achieved by the HCP placing his/her thumb over the claimant's wrist and then fully flexing the claimant's hand for approximately 1 minute. Alternatively, both wrists may be held fully flexed by compressing the dorsal surfaces of the hands together (**Phalen's sign**). Phalen's test positive – paraesthesia in median nerve distribution within 60 seconds of full wrist flexion (sensitivity 75%, specificity 95%)

- Tapping (or percussion) over the Median nerve at the wrist (**Tinel's test** or sign) may also reproduce the claimant's symptoms. Tinel's test positive – paraesthesia in median nerve distribution when percussing over the carpal tunnel. Paraesthesia must be felt distal to the point of pressure for a positive test (sensitivity 64%, specificity 99%). (NB Tinel's test is also used to test other nerves, e.g. the ulnar nerve by tapping over the ulnar nerve as it enters the hand at the wrist)

- A neurological examination of the hand to determine any loss of sensation in the fingers and two-point discrimination

- Wasting of the thenar eminence, i.e. the part of the palm below the thumb

- Weakness in opposition or abduction of the thumb

- Clinical examination of the whole of the upper limb and neck is also necessary to exclude a more proximal cause of an entrapment neuropathy.

- Symptoms of the carpal-tunnel syndrome may be confused with a cervical spondylosis, or the two conditions may occur together

In clinical practice it may be necessary to exclude any underlying metabolic disorder or space-occupying lesions of the carpal tunnel by using appropriate investigations (e.g. blood tests, x-rays). Nerve conduction studies using sensory and motor latencies may be carried out and a comparison made with either the normal limb or with conduction in the ulnar nerve. Nerve conduction studies are not essential if the diagnosis can be made on the basis of the history and clinical findings.

Diagnosis

Diagnosis is often made clinically from characteristic symptoms and signs. Nerve conduction studies may have been performed.

Treatment

Treatment may include one or more of the following list. The effects of a particular treatment should be noted.

- Rest to the wrist. This may require a volar splint that is worn with the wrist held in the neutral position and includes the flexed fingers. It may be worn at night only, or continuously if symptoms are severe

- Injections. An injection of a long-acting corticosteroid into the carpal tunnel is a simple procedure that usually produces significant symptomatic relief within a day. This injection usually produces such good symptomatic relief that it may also be used as a diagnostic test

- Mobilization. Passive movements aimed at stretching the flexor retinaculum and improving the posteroanterior range of the intercarpal joints may be useful

- Decompression of the carpal tunnel. Surgery is indicated if:

  (a) Conservative methods fail (i.e. the methods described above);

  (b) Repeated injections are necessary at frequent intervals;

  (c) Severe motor impairment of the muscles of the thenar eminence is present

NB Anti-inflammatory drugs rarely provide any relief of symptoms. Physical methods – such as heat, exercise or massage – have no role in the treatment of this condition.

Prognosis

Most of the literature suggests that both surgical and non-surgical treatment for Carpal Tunnel syndrome relieves symptoms in approximately 90% of the cases. Therefore each case of PD A12 must be assessed on its own merits regarding prognosis.

Functional effects

Consider activities affected by reduced grip and dexterity within the typical day history such as dressing, shaving, hobbies, driving, playing a musical instrument. A clear description of the claimed effects and clinical findings to support the history of the functional effects is necessary to assess disablement.

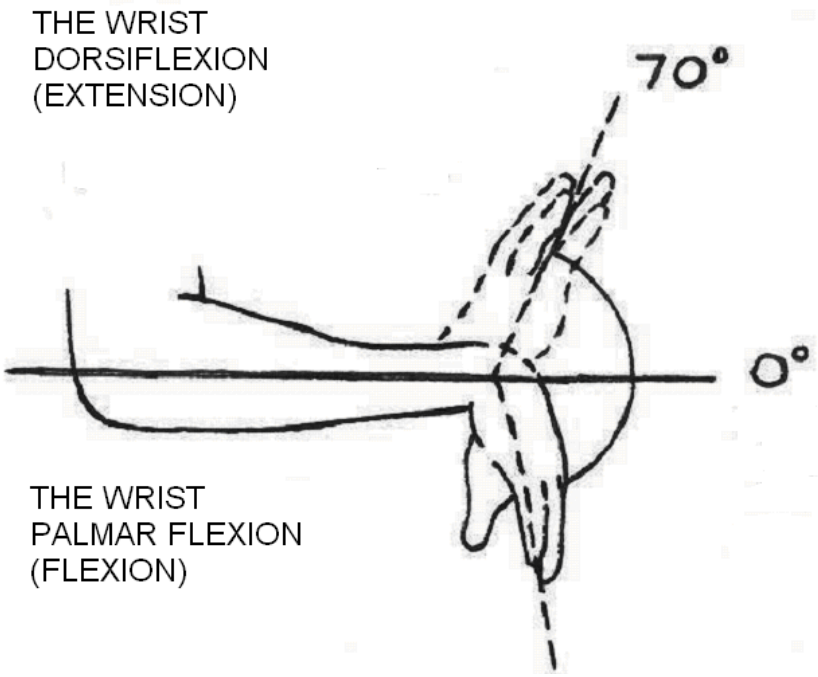
4.7.2 Due to the nature of

Unlike many other Prescribed Diseases, CTS due to the use of vibration tools is **not** presumed to be due to the nature of the occupation. Therefore once CTS has been diagnosed, an opinion must be given as to whether the disease is due to the nature of the occupation, on the balance of probability. Consider the following:

- The degree of the occupational factors outlined above
- Relationship of symptoms to vibrating tool use - the more time that has passed since exposure to vibration ceased, the less likely it is that exposure to vibration is the cause of the symptoms
- Relationship of symptoms to repeated palmar and dorsiflexion
- Other causes of Carpal Tunnel Syndrome independent of vibration and repetitive movements – pregnancy, oral contraception, diabetes mellitus, osteoarthritis of carpus, rheumatoid arthritis, hypothyroidism etc.
- Likely degree of contribution of other conditions (in whole or part)

CTS due to repeated palmar flexion and dorisflexion at the wrist is presumed to be due to the occupation if the prescription criteria are fulfilled unless the contrary is proven.

Figure 3



4.8 Prescribed Disease A14

Definition

Osteoarthritis of the Knee

Date included in legislation: 13/07/2009
Date regulations amended: 30/03/2012

Scheduled occupations

1. Work underground in a coal mine for a period of, or periods which amount in aggregate to, at least 10 years in any one or more of the following occupations:

   (a) Before 1st January 1986 as a coal miner; or

   (b) On or after 1st January 1986 as a -

   (i) Face worker working on a non-mechanised coal face.

   (ii) Development worker

(iii) Face-salvage worker

(iv) Conveyor belt cleaner

(v) Conveyor belt attendant

"A non-mechanised coal face" means a coal face without either powered roof supports or a power loader machine which simultaneously cuts and loads the coal or without both.

2. Work wholly or mainly fitting or laying carpets or floors (other than concrete floors) for a period of, or periods which amount in aggregate to, 20 years or more

Background

In its paper Cm 7440, IIAC recommended, and Minister accepted that osteoarthritis of the knee should be prescribed for coal miners who have worked underground for periods amounting to at least 10 yrs in total. The 10 years total includes any type of work as a miner underground prior to 1986, or, where service is from 1st January 1986 onwards, work only as a face worker on a non mechanised coal face, a development worker, a face-salvage worker, a conveyer belt cleaner, or conveyer belt attendant.

In November 2010, IIAC recommended that osteoarthritis of the knee be prescribed in relation to work wholly or mainly as a carpet fitter or carpet layer or floor layer for 20 years or more in aggregate. Work activities of qualifying claimants will include some or all of the following: installing linoleum, carpet or vinyl floorings; removal of old flooring; installing of underlay; installing of skirting board; and the associated preparatory work. Workers who lay parquet floors and wooden floors should also be covered by the terms of the prescription as should those whose main activity is laying floor tiles. Workers whose main activity is to lay concrete floors are not included.

There is no threshold for diagnosis. If there is x-ray evidence of osteoarthritis then diagnosis should be accepted.

Where x-ray evidence is not available then the diagnosis should be determined on clinical grounds such as crepitus, bony and soft tissue swelling, reduced range of movement and tenderness around the joint margin.

Being on a waiting list for knee replacement surgery or having a history of knee replacement surgery confirms the diagnosis of osteoarthritis of the knee. Arthritis may affect one or both knees.

Osteoarthritis of the knee is common in the general population, with a number of causes, risk factors and aggravating factors. These include age, heredity, female sex, obesity, and previous knee injury.

There is a long latent period for this condition; therefore development of arthritic changes many years after leaving the occupation is typical.

In claimants who fulfil the required degree of occupational exposure as above, osteoarthritis of the knee is likely due to the nature of the work. The medical literature suggests that the prevalence of osteoarthritis of the knee is at least doubled in miners who fulfil the occupational prescription for this prescribed disease. Occupational causation is likely whether the disease is confined to one knee or affects both knees, and whether or not there is a previous history of knee injury.

Assessment of PD A14 Claims

There are no specific diagnostic features which distinguish the Prescribed Disease osteoarthritis in knees in Miners or Carpet or Floor layers or Carpet fitters from osteoarthritis in knee due to other cause. In any one case it is possible that there is more than one cause.

Hence it is essential that the report records sufficient detail in the following areas to exclude/or identify other effective causes, such as obesity (65% of obese 65 yr olds will have radiographic changes of osteoarthritis), involvement in sports (30% of 45 – 59 yr olds who previously played soccer or lifted weights will have radiographic changes) or other knee problems. (There is no evidence to suggest an association with Chondromalacia Patellae).

The report should therefore include:

- Full details of all relevant symptoms e.g. onset and duration of pain, swelling, stiffness, reduced movement, activities of daily living

- Full relevant past medical history, including accidents and injuries to the knees. Where there has been an injury that caused disruption of the joint anatomy then it is highly likely that any arthritic change is partly due to the injury in addition to the occupational exposure. Consider other differential diagnoses of knee pain such as inflammatory arthropathies, gout or referred neuropathic pain from hip or leg.

  The HCP must also consider whether there is evidence of a history of PD A6 rather than osteoarthritis of knee. If there is no evidence of osteoarthritis but the history and clinical examination suggest PD A6 applies, this advice should be given on the report form. If you receive a file with a trailing file identifying previous final assessment for A6, now expired, you need to make sure in every case that there is no evidence of ongoing chronic pre-patellar bursitis.

  If there is none, then you include the condition in “any relevant past medical history, but since there is no longer any evidence of it, you would not include as an unconnected condition or consider it when advising about level of assessment. If there is evidence of ongoing PD A6 in addition to PD A14, then you need to consider whether there is any disablement. If there is then you need to source a second BI613 and complete for the PD A6 (remember that if the previous assessment has lapsed then it needs to be treated as a new claim).

- Full history of relevant investigations and treatment, and proposed treatment

- Relevant occupational history

  For those miners where the occupational exposure predates 1986, all that is required is that they have worked underground in a coal mine in any capacity. Where the occupational exposure spans or post dates 1st January 1986 then it is critically important that the history includes sufficient detail of what the work after that date entailed and dates worked. This is important because the prescription for work after 1st January 1986 demands exposure to work in specific jobs i.e. as a face worker on a non mechanised coal face, a development worker, a face-salvage worker, a conveyer belt cleaner, or conveyer belt attendant. Periods of work above ground and strikes need to be excluded.

  For Carpet or Floor Layers or Carpet Fitters, the length of time in the relevant occupation needs to be documented together with the type of flooring as concrete flooring is not included in the prescription of this condition.

- Relevant social history e.g. hobbies which may have contributed to OA knee

- Relevant clinical examination – both formal and informal examination findings

When PD A14 was introduced in 2009, the guidance indicated that 'wherever possible further medical evidence **must** be obtained.' Where the claimant has been to hospital and/or had an x-ray this should be documented in the BI100PD. The Centre for Health and Disability Assessments administrative staff will request the x-ray report and any clinic letters relevant to the knee problem prior to making the appointment. If these have not been made available or if the claimant has not had an x-ray you should proceed to come to a conclusion on diagnosis based on clinical evaluation. It is not appropriate in this circumstance to commission an x ray.

Note: IIAC opined that previous knee replacement was indicative that the claimant had had osteoarthritis knee to a disabling degree, and that evidence of Stage 3 to 4 on the Kellgren-Lawrence Scale (see below) was indicative of disabling OA knee.

Kellgren-Lawrence scale

Grade	Comments
0	No radiographic findings of osteoarthritis
1	Minute osteophytes of doubtful clinical significance
2	Definite osteophytes with unimpaired joint space
3	Definite osteophytes with moderate joint space narrowing
4	Definite osteophytes with severe joint space narrowing and subchondral sclerosis

The full IIAC report can be found at:

https://www.gov.uk/government/organisations/industrial-injuries-advisory-council

Assessment of disablement

HCPs are reminded that for Industrial Injuries purposes the principles of the assessment of disablement are:

- The assessment is made by reference to a person of the same age and sex whose physical and mental condition is normal. What is normal for a young male is not the same as a woman of the same age or an older man. Hence, the assessment of disablement in a sixty five year old male is made by comparing him to the norm for a sixty five year old male, where it would be reasonable to expect some reduction of general condition and mobility as compared to a younger person. IIAC noted that 'there could well be little or no assessable disablement in comparison to a person of the same age and sex in those above age 70'

- The special circumstances of the claimant other than age and sex are not taken into account. For example, inability to follow a particular occupation, loss of earnings or additional expense because of the effects of the accident does not affect the assessment of disablement

- The period of assessment is that period over which the average assessment of disablement is expected to remain constant. Should there be any significant fluctuation in the level of disablement (e.g. as a result of surgery), separate assessments should be made for the appropriate periods (split assessment). Disablement prior to surgery may be significant, in the immediate post operative period the level of disability would be expected to rise, but in the normal course of event the level of disablement after a period of recovery would be expected to decrease to a level below that which preceded the surgical treatment. The intention of surgical intervention is to restore near normal function

- The period of the assessment can be for an indefinite period. However, advice that a relevant loss of faculty will affect a person for an indefinite period requires very careful consideration particularly in the context of osteoarthritis of the knees. The relevance of disability is particularly likely to decrease with increasing age, and the likelihood of intervention where disability is significant is high. Disability which is considered to be fully relevant to occupational exposure in a 45 year old may with the passage of time become partly relevant or even not relevant

- If the date of any possible operative treatment is not known, then the advice on the period of assessment should be based on the foreseeable future. If there is worsening/improvement the claimant may apply for a change of circumstances review

HCPs should note:

- Osteoarthritic change should be bilateral and more or less symmetrical. If there is a significant difference between the L and R knees then it is likely that another effective cause is at least partly responsible.

However, where there is a strong history of occupational exposure – where the miner was crawling to low faces as a face worker for upwards of 20 years, for example, you need to be very careful before you attribute to something other than occupation. You may need to explore the precise nature of his work – is there anything that would account for the difference – did he, for example, always lead with the affected knee, or use the it to brace his equipment. If there is a credible rationale to do so you should accept relevance. Given the age profile of the claimants it seems highly likely that some background degenerative change is present. Be wary of indicating that the condition is unilateral only

- Claimants will probably be at least in their mid - 50s. The comparison for disablement is to the norm for the same age and sex. It is normal for older age groups to have some reduction of mobility. IIAC recognised that 'there could well be little or no assessable disablement in comparison to a person of the same age and sex in those above age 70'

  However remember that **severe osteoarthritis is not the norm**, even in quite elderly populations – only 18% of 70 year olds will have grade 3 or 4 changes on X ray.

- Claimants may have suffered accidental injuries to their knees, so careful consideration of relevance is necessary. Even if the accident was sustained in the course of their employment then it should be considered as another effective cause

- Research indicates that x-ray grading changes very little if at all over time. Therefore you should not assume that a claimant who has mild arthritis will necessarily progress to severe disease

- Where disablement is severe it is highly likely that operative treatment will occur, therefore it is probable that the assessment of disablement will vary over time and an indefinite award is not appropriate. The length of any award should reflect the likely timescales for operative treatment. However, where disablement is mild, an indefinite award may well be the correct outcome

HCPs are reminded that:

- In **all** cases there must be an explanation and justification that is understandable to the Decision Maker, of the advice being given. For example, if the occupational criteria are fulfilled but the advice is that the disability arising from the osteoarthritis of the knee is partially relevant, e.g. one knee is more affected than the other due to trauma

- The assessment of disablement in a non-scheduled assessment should be based upon the effects of the conditions listed in the Social Security General Benefit Regulations 1982. When advising on osteoarthritis of the knee, consider previous advice on the effects of ankylosed joints e.g. knee 30%. Remember that the disability is 'impaired lower limb function' and in most case both knees will be affected and need to be considered in the assessment.

Any pain or instability in the joint may result in a higher percentage assessment being advised when compared to a fully ankylosed joint, taking into account claimant's age and sex

- If the suggested assessment **does not** correlate with the relevant Scheduled or non scheduled assessments it is particularly important to explain the particular circumstances of the case which lead you to give the advice

- Where a split assessment has been given, an explanation of the reasoning should be provided, e.g. an increase in assessment for a period due to the effects of operative treatment

- HCPs must list and appraise any further medical evidence that has been presented, including any x-ray report or clinic letters

Note: Any explanation must be relevant to the case – it is NOT sufficient to say 'This advice falls within the consensus of medical opinion'. The decision making authorities need to know WHY it is within the consensus of medical opinion.

5. Some other Prescribed Diseases

5.1 Prescribed Disease A1

Definition

Leukaemia (other than chronic lymphatic leukaemia) or cancer of the bone, female breast, testis or thyroid.

Scheduled occupation

Any job involving: Exposure to electromagnetic radiations (other than radiant heat) or to ionising particles where the dose is sufficient to double the risk of the occurrence of the condition. For example, people working in the nuclear industry and hospital X-ray departments.

The regulation does not define the phrase “double the risk” therefore the Decision Maker should accept prescription based on the claimant’s occupation. A guide to the Decision Makers on the suggested occupations has been provided, but this list is not exhaustive. Decision Makers may ask for medical advice on the prescription question.

The National Radiological Protection Board (NRPB) has drawn up a table to show the minimum dose which it is estimated would double a person’s risk of each of these cancers:

Cancer	Minimum radiation dose which it is estimated would double a person’s risk of each of these cancers.
Leukaemia	0.23 Sieverts (Sv)
Bone	0.56 “”
Female breast	0.74 “”
Testis	0.81 “”
Thyroid	0.51 “”

Requirements

When giving advice to the Decision Makers, the HCPs use the prescription of the PD, and base their advice accordingly. Account should be taken of the information that has been given by the NRPB when formulating the advice.

The large majority of claimants will have dosimetry records as employers have a duty to monitor doses, and records must be kept for 50 years or until the age of 75. Hence the information on which to advise, on the balance of probabilities, that the work exposure was the cause of the disease should be available.

Due to the serious nature of the diseases involved confirmation of the disease is unlikely to pose problems, and in addition, information suggestive of causation, that it is linked to the occupation, may be available in those few cases where dosimetry is not recorded.

Care must be taken to take a full occupational, social, family history and history of the disease in order to establish relevance.

The time rule for presumption has been extended to apply any time after leaving the job as well as in the job. However it has always been accepted in the scheme that causation is due to work, no matter how long ago the worker left the qualifying employment.

Previously problems may have been encountered in obtaining advice on exposure levels. If assistance is required your local IIB lead or the IIB helpdesk may need to be contacted.

5.2 Prescribed Disease A2

Definition

Cataract

Scheduled occupation

Frequent or prolonged exposure to radiation from red-hot or white-hot material. For example, glass and metal workers, stokers.

Cataract is not prescribed unless the person was employed in employed earner's employment in a Scheduled occupation, for a period or periods amounting in aggregate to not less than 5 years.

When giving advice to the Decision Makers, the HCPs should take account of the prescription of the PD, and base their advice accordingly. When considering the prescription of the disease, the Industrial Injuries Advisory Council (IIAC) could find no convincing evidence that cataracts caused by infrared radiation can be distinguished medically from those that are not.

There is little information on how the risk of cataract varies accordingly to the intensity and duration of the occupational exposure to infrared radiation. Exposure of an individual worker will depend on several factors including temperature, the source of radiation, the distance of the eye from the source, whether eye protection is worn and how long the worker spends close to the source of radiation.

Hence IIAC suggested frequent and prolonged exposure over a period of five years in aggregate was reasonable when deciding prescription.
The time rule for presumption has been extended to apply any time after leaving the job as well as in the job. The medical literature has shown that the risk for cataract after exposure to heat, for example in glass blowers, persists for many years. In claimants with 5 years of exposure, doubling of risk of cataract can be considered to persist for life.

Care must be taken to take full occupational, social, family history and history of the disease to establish relevance. If the work that involves the prescribed activity is/was performed very infrequently, or for a short time, then it might be disregarded as being insufficient to be taken into account.

5.3 Prescribed Disease A13

Definition

Osteoarthritis of hip

Date included in legislation: 14/3/2005

Scheduled Occupation

Work in agriculture as a farmer or farm worker for a period of, or periods which amount in aggregate to, 10 years or more

Pathogenesis

Osteoarthritis of the hip is a degenerative condition of the hip joint characterised by radiographic changes, and pain and/or stiffness upon movement.

The disease is common in the population at large. However in one occupational group – farmers – there is a raised incidence of the disease sufficiently high that a clear association can be made between the occupation and the condition, even though there is still some uncertainty about exactly what aspect of farming is responsible. There are no distinctive clinical or radiographic features of the disease that help to distinguish between work-associated and non-occupational patterns of disease. The natural history of the disease varies, with a spectrum of severity.

Most people have mild, slowly progressive disease and cope without the need for surgery; but some individuals incur rapidly progressive joint damage. Total hip replacement is one of the most frequently undertaken surgical procedures in orthopaedic practice.

Risk factors for osteoarthritis of the hip

Various genetic and acquired factors may contribute to the occurrence of hip osteoarthritis.

- Congenital dislocation of the hip
- Perthes disease
- Acetabular dysplasia, and slipped femoral epiphysis
- Hip related fractures
- Generalised inflammatory disease of joints
- Ageing in isolation, or sometimes as part of a more widespread pattern of osteoarthritis that involves multiple joints
- Obesity
- Activities which increase the mechanical load on the joint and produce large compression forces (e.g. heavy regular lifting)

Clinical features

The typical symptoms of hip osteoarthritis are pain and stiffness. Pain is usually felt in the groin, but it may radiate to the knee, buttock, or inner thigh. It is mainly felt on weight bearing and aggravated by movement. Stiffness is most noticeable following a period of inactivity. Slowly over time, an effective shortening of the affected limb arises as contractures develop in the muscles around the hip joint. The affected individual finds it difficult to reach down to tie shoelaces, or put on socks, stockings and shoes. Loss of functional ability may progress from stiffness to difficulties in rising from a chair, walking, negotiating stairs, and dressing; a characteristic limp may develop, or a gait that is waddling. A walking stick is often needed for support. In severe disease, pain can also occur at night and can disturb sleep.

X-ray changes

Characteristic features of osteoarthritis – reduced joint space, subchondral sclerosis, development of bone cysts and osteophytes, irregularity of the joint surfaces, and deformity – may be apparent on hip radiographs. However such changes are common in the population at older ages.

Assessment of disability and disablement

Osteoarthritis of the hip is multifactorial in aetiology so other effective causes should be considered. Where Presumption is accepted, it does not follow that the disability has to be fully relevant. The time rule has been extended to cover any time after leaving the job as well as in the job, however decisions on PD A13 are

already made as if the longer time rule applies in view of the natural progression of the condition.

Prior to any hip replacement surgery there is likely to be limitation of mobility and pain (particularly at night). It is rare for mobility to be totally restricted. Hip replacement surgery is usually successful at reducing pain and restriction of movement such that any residual disablement is likely to be minimal. Many people who undergo hip replacement surgery are retired, but younger individuals would expect to return to work.

The assessment of disablement should be made by comparison to a person of the same age and sex whose condition is normal, using the scheduled and non scheduled assessments as a guide.

As a guide, the disablement arising from an ankylosed hip is in the order of 60%. Osteoarthritis of the hip rarely causes such a severe limitation of function.

The following are suggested guides to disablement:

- The disablement due to Osteoarthritis of the hip in those where surgical intervention is not indicated is unlikely to exceed 14%
- Immediately prior to hip replacement surgery disablement is unlikely to exceed 30%, and probably most will be in the region of 20%
- Modern hip replacement surgery normally leads to excellent functional recovery. Over 95% of people have a good long-term prognosis, but may not reach maximum improvement for 12 months. Hence an assessment in the region of 20% for 12 months post surgery should be considered as an appropriate assessment
- Any residual disablement after the immediate post-surgery recovery period of 12 months is unlikely to exceed 5% per hip

5.4 Prescribed Disease B14

Definition

Lyme disease

Date included in legislation 14/3/2005

Scheduled occupation

Exposure to deer or other mammals of a type liable to harbour ticks harbouring Borrelia bacteria.

Lyme disease is most commonly acquired in the following areas: New Forest, Exmoor, South Downs, Thetford Forest, woodland and heathland in Southern England, Lake District, North York moors, Scottish highlands and islands.

Occupational exposure may occur in those who work outdoors in high risk areas of the UK or who are in contact with animals in high risk areas of the UK. Occupations at risk include sheep farmers, particularly hill farmers from working dogs, deer farmers, game keepers, vets, agricultural workers, forestry workers, nature and outdoor pursuits workers.

Pathogenesis

Lyme borreliosis was first described in 1977 following an outbreak in Old Lyme, Connecticut. Lyme disease, as originally described, is caused by a spirochaete bacterium, Borrelia burgdorferi, carried by the tick, Ixodes ricinus. The reservoir hosts are deer and rodents, which are infested with ticks. Since the disease was first described several different strains of Borrelia have been identified which cause Lyme disease. The most common strains in Europe are Borrelia afzelii and Borrelia garinii.

The strain of spirochaete is significant in that it affects the way the disease manifests itself, and the severity and frequency of the manifestations. Lyme disease was originally reported as an infective arthritis, but Borrelia afzelii is associated with skin changes (acrodermatitis) and Borrelia garinii with nervous system effects. All the strains produce the characteristic sign of a tick bite with a red ring around it.

Humans contract the disease when bitten by infected ticks that attach themselves to skin and clothing as people walk through grasslands and forests where deer and other host mammals are living. Among those at risk are vets, deer farmers and forestry workers. Although the numbers of occupationally caused Lyme disease are low a relatively high incidence of the disease in deer hunters and forestry workers suggested a clear occupational risk.

Symptoms and signs

The infection can affect the skin, muscles, joints, nervous system and the heart. In about 50% of the cases a characteristic rash or lesion (erythema migrans) is seen. The rash begins a few days to a few weeks after the bite of an infected tick. The rash generally looks like an expanding red ring. It is often described as looking like a bull's-eye with alternating light and dark rings. However, it can vary from a reddish blotchy appearance to red throughout. The rash can be confused with an insect bite, or ringworm. At about the same time that the rash develops, flu-like symptoms may appear with headache, sore throat, stiff neck, fever, muscle aches, fatigue and general malaise.
Some people develop the flu-like illness without getting a rash. The early symptoms may disappear, and the condition may be self-limiting and can subside spontaneously. However, without treatment the disease may recur, worsen, or persist in a chronic form. The skin, muscles, joints, nervous system and heart may all be affected.

More serious problems can develop months to years later.

The later symptoms of Lyme disease can be quite severe and chronic. Muscle pain and arthritis, usually of the large joints is common. Neurological symptoms include meningitis, numbness, tingling, and burning sensations in the extremities, Bell's palsy (loss of control of one or both sides of the face), severe pain and fatigue (often extreme and incapacitating) and depression. Heart, eye, respiratory and gastrointestinal problems can develop. Symptoms are often intermittent lasting from a few days to several months and sometimes years. Chronic Lyme disease, because of its diverse symptoms, mimics many other diseases and can be difficult to diagnose.

In order for musculoskeletal and cardiac conditions to be attributed to Lyme borreliosis, there must be a high level of serum specific antibodies, and alternative diagnoses must be excluded. Mostly, the effects are transient, but chronic synovitis in one or more joints has been recorded, and episodes of cardiac rhythm disturbances, endomyocarditis and pericarditis also occur. It is reported that chronic cardiac conditions may be associated with Lyme borreliosis but no causal relation has been established. The neurological effects include meningitis, facial palsy and nerve root inflammation (meningo-radiculitis). The cerebrospinal fluid shows lymphocytic pleocytosis (presence of more cells than normal) with plasma cells. Specific IgM and IgG antibodies develop after some six weeks.

Chronic neurological involvement is reported as uncommon, and should not be diagnosed in the absence of lymphocytic pleocytosis and high levels of specific antibodies in the cerebrospinal fluid.

Treatment

Lyme disease is treated with antibiotics. Timely treatment increases chances of recovery and may lessen the severity of any later symptoms in both animals and man.

5.5 Prescribed Disease B15

Definition

Anaphylaxis

Date included in legislation: 14/3/05

Scheduled occupation

Employment as a health care worker having contact with products made with natural latex rubber.

The term health care worker should include anyone involved with human health care including home carers and care workers in residential homes. It does not include occupations where there is no health care involved - such as home helps and general staff in residential homes.

This Prescribed Disease ensures that that employed earners who became sensitised to natural rubber latex at work but who suffer an anaphylactic reaction following contact with the relevant allergen **outside** work, are covered by the Industrial Injuries provisions This Prescribed Disease relates only to allergic reactions to natural latex rubber. Secondary allergens (e.g. to kiwi fruit, banana etc) are **not** covered by the prescription.

Pathogenesis

Anaphylaxis is a life-threatening IgE-mediated allergic Type I hypersensitivity reaction due to contact of a sensitised individual with an allergenic protein. Common allergens which can provoke anaphylactic shock include drugs, insect stings, latex and certain food ingredients, such as nuts.

The commonest occupational cause of anaphylactic reactions is natural rubber latex. It is difficult to distinguish between occupational and non-occupational causes.

Initial exposure to the allergen induces specific IgE antibody. Subsequent contact can provoke an anaphylactic reaction, but an anaphylactic reaction does not occur inevitably after a person has developed IgE antibody. An anaphylactic-like (anaphylactoid) reaction can occur during first exposure to certain drugs; but these are a manifestation of a toxic, rather than an allergic, reaction to the drug.

Anaphylactic shock is due to a sudden massive release of histamine and other mediators from basophils into the bloodstream. These trigger constriction of the airways and swelling of tissue (angioedema), resulting in difficulty breathing, and dilatation of blood vessels, leading to shock and pulmonary oedema. Other symptoms of anaphylaxis include urticarial skin rash and gastrointestinal reactions, such as vomiting, abdominal cramps and diarrhoea. The onset of anaphylaxis can be very rapid, with symptoms occurring within minutes of contact with the allergen.

The clinical presentation of Anaphylaxis due to latex allergy is indistinguishable from that due to other sensitising agents. It is not appropriate to instigate invasive procedures in order to diagnose a condition such as this for benefit purposes.

Further medical evidence, may identify the sensitising agent, and corroborate any symptoms attributed to the anaphylactic reaction.

Points to remember when assessing disablement

Sensitisation in itself is non-disabling (Para 270 of Cm 5997 refers). In the absence of entitlement to Reduced Earnings Allowance (REA), there is no necessity to give a minimum 1% disablement though a less than 1% assessment will reflect the differentiation between the normal and the person with circulating antibodies.

Anaphylaxis is an acute condition with either full recovery or death occurring.

Thus in the absence of sequelae there is unlikely to be disablement after the 91st day. In a very small number of cases anoxia leads to brain damage, which may warrant an assessment of disablement.

A previous anaphylactic reaction is no indicator either to the likelihood of ever having a further attack, and nor is the severity of a previous attack an indicator of the severity of any further attack.

Reassurance and provision of an epinephrine pen for emergency use usually allows the person to lead a normal life.

A small minority may claim psychological sequelae. These cases will require careful evaluation of the cause and relevance of any psychological problems to a previous anaphylactic reaction. Most reactive psychological conditions improve with the passage of time from the causative condition, and are unlikely to be permanent. Hence only on rare occasions will a life award be justified.

Symptoms and signs

Symptoms vary, and rarely does any one person develop all the following possible symptoms. Symptoms usually start in 1 to 15 minutes (but rarely after as long as 2 hours) after exposure to the allergen.

The individual feels uneasy, becomes agitated and flushed, and complains of palpitations, paraesthesia, pruritus, throbbing in the ears, coughing, sneezing, urticaria and angioedema, and difficulty breathing owing to laryngeal oedema or bronchospasm. Nausea, vomiting, abdominal pain, and diarrhoea are less common. Shock may develop within another 1 or 2 minutes, and the individual may convulse, become incontinent, become unresponsive, and die. Primary cardiovascular collapse can occur without respiratory symptoms. Severe reactions can occur with massive angioedema and asthma but without evidence of cardiovascular involvement. By contrast some people only experience a mild reaction e.g. generalised pruritus, urticaria, angioedema, mild wheezing, nausea, and vomiting.

5.6 Prescribed Disease D3

Definition

Diffuse mesothelioma (primary neoplasm of the mesothelium of the pleura or of the pericardium or of the peritoneum)

Changes in legislation

From 29/7/02 the law provides that for a claimant who is suffering from PD D3 and who has loss of faculty which is prescribed as:

"***Impaired function of the pleura, pericardium or peritoneum caused by***

mesothelioma"

that the PD disablement shall be assessed as **100%.**

This allows fast tracking of such claims and ensures a rapid response without a routinely performed face to face examination of the claimant.

Scheduled occupations

Exposure to asbestos, asbestos dust or any admixture of asbestos at a level above that commonly found in the environment at large.

5.6.1 Background

Diffuse Mesothelioma cancer is linked to asbestos exposure, but is not the only cause of Diffuse Mesothelioma, and there are other forms of mesothelioma.

Mesothelioma is a tumour arising in the cells, which make up the lining of various body cavities. Most commonly the cells of the pleura are affected, but other sites in the body may be affected, for example the peritoneum, and the pericardium. Most cases of mesothelioma are malignant but there are benign types and not all cases of mesothelioma are related to asbestos exposure. The most commonly diagnosed form of mesothelioma is Diffuse Mesothelioma.

Other types of Mesothelioma include:

- Cystic mesothelioma. This is a rare peritoneal tumour which usually occurs in women of child - bearing age, but can also occur in men, e.g. cystic testicular mesothelioma. Most cases are benign but there is evidence that some cases develop into low-grade malignancy. Surgical removal is usually successful. Asbestos plays no role in the aetiology of these tumours

- Benign (solitary) mesothelioma, also known as pleural fibroma. Microscopically the tumour is predominantly composed of whorls of collagen fibres and reticulin interspersed with fibroblasts.

  Many of the tumours are asymptomatic and are detected as an incidental x-ray finding. Removal of the tumour is usually curative. Asbestos plays no role in the aetiology of these tumours

- Benign papillary mesothelioma. These can occur in men – arising from the tunica vaginalis, and in women in the peritoneum. Asbestos plays no role in the aetiology of these tumours

- Localised malignant mesothelioma. These are uncommon tumours. The microscopic appearance is like diffuse mesothelioma but without evidence of diffuse spread. The link between the tumour and exposure to asbestos has not been established. There is limited literature as the condition is very rare. What literature there is, points away from the cause being

asbestos. Localized malignant mesotheliomas should be differentiated from diffuse malignant mesotheliomas because of their localized presentation, quite different biologic behaviour, and far better prognosis than for diffuse mesothelioma - individuals may remain disease free for many years after surgical excision

It is essential therefore to be clear of the precise diagnosis when referring to 'mesothelioma'.

Causes of Diffuse Mesothelioma

Exposure to asbestos is linked to the development of malignant Diffuse Mesothelioma (often referred to as merely 'Mesothelioma'). Note, however about 20% of individuals do not have a documented history of asbestos exposure. The rise in malignant pleural Diffuse Mesothelioma during the last 3 decades is related to the increased use of asbestos since the 1930s. An increase in cases of Diffuse Mesothelioma was reported some years after the use of asbestos became widespread, and thus a link with asbestos exposure was made.

Asbestos exposure is **not** the only cause of Diffuse Mesothelioma. It is estimated that 1-2% of all cases of Diffuse Mesothelioma are not due to asbestos exposure. Before the widespread use of asbestos in the 20th century it was a rare tumour, but not unheard of, and there are references to it occurring throughout 19th century literature.

Non-asbestos causes of Diffuse Mesothelioma are:

- People with a history of previous radiotherapy have been reported as developing it at the site of the radiotherapy. (On average 15 years after the radiotherapy)
- It has been reported in association with chronic inflammation and scarring, as in cases of tuberculosis and emphysema
- Zeolite (erionite) is a non-asbestos mineral fibre that has been identified as the probable cause of an epidemic of malignant Diffuse Mesothelioma in Karain, a small village in the Anatolian region of Turkey where no asbestos was found in the village
- Heredity. Although occasional reports of familial cases of Diffuse Mesothelioma have been documented all have been associated with documented asbestos exposure. Because only 3%-8% of asbestos workers develop Diffuse Mesothelioma, the occurrence of this tumour in their families suggests that genetic factors may be important in the development of Diffuse Mesothelioma
- Certain strains of viruses have been shown to induce malignant Diffuse Mesothelioma in animals
- Other causes of Diffuse Mesothelioma have been suggested in experimental animal studies, e.g. Nickel and silica dust have been

reported to cause malignant Diffuse Mesothelioma in rats, but no causative relationship has been proved in humans; several chemical compounds, including polyurethane, ethylene oxide, and polysilicone plastics have also been shown to induce malignant Diffuse Mesothelioma in animals

Mesothelioma in children

Mesothelioma has been reported in children, though it is very rare, and the sporadic occurrence of mesothelioma in children has not been linked to asbestos exposure in the majority of cases, and these tumours may represent an entity entirely different from adult mesothelioma. In a study of 80 children with mesothelioma only 2 were known to be exposed to asbestos.

Cases reported in the literature include:

- A hospital in New York reported 6 cases of pleural mesothelioma and 1 of peritoneal mesothelioma over a 28-year period. When they wrote the report (in 1981) they only identified 42 other cases of malignant mesothelioma in children in the literature
- A case of malignant mesothelioma in a 16-day-old boy was reported in 1987
- A case of a benign peritoneal mesothelioma was reported in the UK in 1994

Diffuse Mesothelioma

About 75% of Diffuse Mesothelioma start in the chest cavity (pleural mesothelioma). 10% to 20% begin in the abdomen (peritoneal mesothelioma). Other very rare variants include pericardial mesothelioma, and malignant mesothelioma of the tunica vaginalis. (Note: some authors are of the opinion that asbestos is not a cause of malignant mesothelioma affecting the tunica vaginalis).

Diffuse Mesothelioma deaths are as follows:

- 1968: 153
- 2000: 1631
- 2001: 1848
- 2011- 2015: estimated at between 1950-2450 deaths per year

Summary

The rise in malignant Diffuse Mesothelioma in adults during the last 3 decades is related to the increased use of asbestos since the 1930s.

However asbestos is not the only cause of mesothelioma. Although very rare, both benign and malignant mesothelioma has been reported to occur which are not related to asbestos exposure.

Mesothelioma has been reported in children, but no link to asbestos exposure has been made in the vast majority of cases, thus suggesting that Diffuse Mesothelioma in children is a different entity to that in adults.

It is important to establish the precise diagnosis in claims for benefit in which the diagnosis is given as 'Mesothelioma'.

Most of those who develop mesothelioma, have worked in jobs or environments where they have been exposed to asbestos dust particles in the air. The exposure need not have been prolonged and this should be borne in mind when giving advice.

Asbestos use is now strictly controlled by law but up to and throughout the 1970's it was almost universally used in insulation - insulating pipes, boilers, roof tiles, fireproofing courses, clutches and brakes of cars and other vehicles including railway carriages. Relevant occupations can include people working in the construction, shipbuilding and engineering industry.

Suitable investigations are chest x-ray, CAT scan, pleural fluid aspiration and thorascopic biopsy. The main symptom is dyspnoea and may be accompanied by cough, haemoptysis and chest pain. The prognosis from diagnosis to death is usually a matter of months rather than years.

There is frequently a time delay of at least twenty years (and sometimes up to fifty years) between the exposure to asbestos and the development of mesothelioma. The time rule for presumption has been extended to cover any time after leaving the job as well as in the job, however for this condition it has always been accepted in the Scheme that causation was due to work no matter how long ago the worker left the qualifying employment.

Where the illness is as a result of occupational exposure to asbestos the person can make a claim for Prescribed Disease D3 (PD D3) i.e. under the Industrial Injuries Benefit Scheme and may receive payments under the 1979 Act, i.e. under the 1979 Scheme.

However there are a number of cases each year who have no occupational causation, who therefore cannot claim under the Scheme and the 1979 Scheme.

In March 2007 the Secretary of State announced his intention to extend the coverage for compensation to sufferers of Diffuse Mesothelioma who had been exposed to asbestos in the UK but were unable to claim compensation from other sources such as the IIB and the 1979 Scheme, for example, women who had washed their husband's clothes. The **2008 Diffuse Mesothelioma Scheme** seeks to compensate this group of people.

The Secretary of State announced that payments would be made within 6 weeks of application. The amount will be based on the age of the sufferer.

The start date for the 2008 Diffuse Mesothelioma Scheme was the 1st October 2008.

5.6.2 Handling the claim (Claimants with Work Exposure)

When the Decision Maker has obtained details of the claimant's working history and the asbestos exposure, the case is forwarded to The Health Assessment Advisory Service for advice to be given as a paper exercise.

There may be medical evidence on file although in the majority of cases referred; there will be no evidence or inadequate evidence. The role of the HCP is to interpret whatever evidence there is, or to obtain evidence to advise on the diagnosis of PD D3.

Sources of additional evidence are:

- The GP
- The hospital physician
- Other health care professionals such as Macmillan nurses etc.

If additional evidence is required there are several factors to be borne in mind.

A GP might not be fully aware of the diagnosis where hospital investigation is ongoing. Often the hospital might have the more up to date information, particularly when there are recent investigations such as scans or biopsies. Given the urgency of these cases the initial contact would most likely be by telephone and any discussions should be recorded. This record must be retained on file as part of the documentary evidence.

Where there is conflicting or inconclusive evidence several of the above professionals may need to be contacted before definitive advice can be given.
If the diagnosis of PD D3 can be confirmed, then the HCP should advise the Decision Maker with justification of their opinion on both diagnosis and date of onset on form D3/D8 Diagnosis form.

This opinion is based on consideration of the *balance of probability* in the case and the *consensus of medical opinion* in this condition.

It is important that a definite discrete date of onset is provided on the D3/D8 Diagnosis form.

It is likely that the illness could have been present for some months before initial presentation and the HCP should take this into account when advising a date of onset. This is because the Decision Maker may backdate payment to a date 3 months prior to the date of claim.

The level of assessment awarded by the Decision Maker would routinely be 100% for life.

If the diagnosis is not confirmed the HCP advises accordingly on form D3/D8 Diagnosis form - again with full justification and the file is returned to the district office.

Any advice to the Decision Maker must be fully justified with clear logical reasoning of evidence and a full explanation in lay terms.

In a very small number of cases it may be that medical evidence is not yet available, perhaps because of ongoing investigation. In this circumstance, the file should be returned to the District Office with details of the reason for delay. In such a case, provide a date when the evidence will be available and the file should be re-referred to The Health Assessment Advisory Service for further consideration of the diagnosis. The standard advice form for PD D3 has a dedicated section for entry of such detail.

PD D3 claims must be dealt with urgently - given the diagnosis the claimant must receive a swift response. They must also be handled sensitively. The HCP should be aware that some claims are posthumous and may require particularly sensitive handling. The approach to PD D3 should be as per DLA (Disability Living Allowance) or PIP (Personal Independence Payment) Special Rules claim handling.

Referral

The case should be referred to The Health Assessment Advisory Service in a **BI8** file with contact details of any GP or hospital physician (where relevant) and the claim pack **BI100-PN** with the consent form signed by the claimant or their appointee. Full details of working history and asbestos exposure should have been obtained.

Evidence

Suitable evidence might include copies of x-ray reports, hospital letters, biopsy results or scans. The aim is not to delay the processing of the claim by waiting for documentary evidence.

If no hard copy of evidence can be obtained, then a documented record of a telephone discussion with a medical professional would be acceptable. The details recorded must include the name of the person consulted and their professional status. However, documentary evidence should be obtained whenever possible, and given the urgency of the case FAX contact is suitable. (See Appendix 8 for the procedure to follow when faxing information.)

5.6.3 Handling the claim (Claimants with No Work Exposure)

Legislation

The Child Maintenance and Other Payments Act 2008 allows for a lump sum

compensation payment to be paid (2008 Diffuse Mesothelioma Scheme**)**.

The qualifying criteria are:

- The claimant must have been exposed to asbestos in the UK
- There must be evidence to show that the person suffers from Diffuse Mesothelioma

The claim must have been made within one year of diagnosis or, in the case of a claim being made by a dependant, dependants can claim within a year of the date of death. (Note: For the first 12 months of the 2008 Diffuse Mesothelioma Scheme the 'claim within one year of diagnosis' requirement will not apply to sufferers diagnosed before the introduction of the Scheme to ensure they have the opportunity to apply).

5.6.4 The Health Assessment Advisory Service involvement in the Scheme

The preferred approach is to avoid the need for the Health Assessment Advisory Service input if possible. The claim process has been made as simple as possible so claimants will not routinely be referred to the Health Assessment Advisory Service.

Claimants must provide evidence that they suffer from Diffuse Mesothelioma. If the claimant does not provide any evidence at all then the claim will fail. The Decision Maker would not immediately disallow the claim in these situations, rather they would advise the claimant what evidence they should provide and how they can obtain it. If they still provide no evidence then the claim would be disallowed.

However in a small number of cases the Decision Maker may request advice from the Health Assessment Advisory Service - this advice will be given as a paper exercise.

Thus there may be a small number of cases where the Health Assessment Advisory Service are asked to advise on the evidence provided by the claimant and/or seek information from the claimant's own health care professionals. For example:

- Where the evidence provided is not clear enough for the Decision Maker to make their decision on the claim
- Where issues relating to the diagnosis and/or causation are not clear, e.g. is cystic mesothelioma the same as diffuse mesothelioma? Is exposure to asbestos the cause of benign cystic mesothelioma?

Claimants will not usually be called for a medical assessment

In cases where the evidence provided is not clear enough for the Decision Maker to be able to decide on the claim, further clarification of the evidence and/or

additional evidence may be required.

The case should be referred to the Health Assessment Advisory Service with contact details of any GP or hospital physician (where relevant) and the claim pack PWC1 with the consent form signed by the claimant or their appointee. The medical evidence that the claimant has provided will be included in the file.

The role of the HCP is to advise on diagnosis of Diffuse Mesothelioma, to interpret evidence and to obtain evidence when required.

It is important that any advice given is justified and explained and is supported by medical evidence.

Sources of additional evidence could include:

- The GP
- The hospital physician
- Other health care professionals such as Macmillan nurses etc.

If additional evidence is required there are several factors to be borne in mind.

GPs may be the most easily contacted, but they might not be fully aware of the diagnosis where hospital investigation is ongoing. Often the hospital might have the more up to date information, particularly when there are recent investigations such as scans or biopsies. Therefore it may be necessary to contact more than one source of additional evidence. The HCP is not confined to seeking information from the sources listed in the claim form, but as the claim form asks for full contact details and if this is not provided the Decision Maker should have gathered the missing information before referring the case to the Health Assessment Advisory Service there should be no need for HCPs to delay the claim by making drawn out enquiries. However, in order to ensure appropriate advice is given a request for the name of the Consultant/ relevant hospital etc from the GP or vice versa may be needed in small number of cases.

Where there is conflicting or inconclusive evidence several of the above professionals may need to be contacted before definitive advice can be given.

Given the urgency of these cases the initial contact would most likely be by telephone and any discussions should be recorded on Form BI18 DM. Form BI18 DM should then be retained on file as part of the documentary evidence.

Any evidence obtained should be listed and included in the file. Telephone discussions should be tactfully and sensitively handled with an explanation that evidence is being sought to allow payment of benefit and that written consent is held on file from the claimant or their appointee.

The procedure is similar to the process of contacting medical professionals in DLA/PIP Special Rules claims and PD D3 claims where evidence is lacking.

Documentary evidence should be obtained wherever practical. In most cases because of the urgency of the case this would be by fax. (See Appendix 8)

Suitable evidence might include copies of x-ray reports, hospital letters, biopsy results or scans. However, you should not delay the provision of advice in order to await documentary evidence.

If no hard copy of evidence can be quickly obtained then a documented record of a telephone discussion with a medical professional would be acceptable.

The details recorded must include the name of the person consulted and their professional status.

In a very small number of cases it may be that medical evidence is not yet available because of, for example, ongoing investigations. In this circumstance you should return the file to **Barrow Benefits Delivery Office**, clearly stating the reason for delay and advising a date when the evidence will be available, when the file should be re-referred. The Decision Maker may then disallow the claim and, if appropriate, advise the claimant to make a fresh claim when the evidence to support their claim is available.

Form completion

As applicable to the case being referred advice should be given on:

- Where Diffuse Mesothelioma is diagnosed:
  - The date of diagnosis - this is based on the balance of probability and the consensus of medical opinion. It is important that a definite discrete date *dd/mm/yy* is given on the advice form if possible
  - The date of onset. It is important that a definite discrete date *dd/mm/yy* is given on the advice form if possible
  - Any other relevant information
- Where the evidence available does not support the diagnosis of Diffuse Mesothelioma:
  - Non-availability of evidence as referred to above
  - Any other relevant information which may assist the Decision Maker

Any advice to the Decision Maker must be fully justified with clear logical reasoning of evidence and a full explanation in lay terms.

The form should also include:

- All medical evidence considered should be listed
- The HCP's signature and the date

If HCPs need further information or clarification they are advised to speak to senior colleagues or their local Relevant Manager.

5.7 Prescribed Disease D13

Definition

Primary carcinoma of the nasopharynx

Date included in the legislation 07/04/2008

Scheduled Occupations

Exposure to wood dust in the course of the processing of wood or the manufacture or repair of wood products, for a period or periods which amount in aggregate to at least 10 years.

5.7.1 Background

A recommendation by the Industrial Injuries Advisory Council (IIAC) in its report "Nasopharyngeal cancer due to exposure to wood dust" (Cm 7162, published July 2007) to prescribe primary carcinoma of the nasopharynx due to exposure to wood dust has been accepted by Ministers and came into effect on 7 April 2008.

A review of the evidence indicated that exposure to wood dust in the course of processing wood or the manufacture of wood products for a period or periods which amount in aggregate to at least 10 years was associated with an increased risk of developing primary carcinoma of the nasopharynx.

The nasopharynx is an air space lying at the back of the nose above the soft palate. It connects the back of the nose to the back of the mouth.

Aetiology

Exposure to the Epstein-Barr virus is a known risk factor for cancer of the nasopharynx. Regardless of ethnic origin, most people with nasopharyngeal carcinoma show elevated titres of antibodies to the Epstein-Barr virus.

Ethnicity - they are one of the commonest malignancies seen in the Cantonese Chinese and their emigrants, who appear to be genetically susceptible to the oncogenic effects of the Epstein-Barr virus.

There is a recognised association with prolonged exposure to wood dust.

Incidence

Carcinoma of the Nasopharynx is rare in the UK, accounting for less than 0.25% of all cancers. Squamous cell carcinoma and its variant, lympho-epithelioma account for approximately 4 out of 5 malignant tumours of the nasopharynx. One in 5 individuals present under the age of 30 years.

Pathology

These tumours arise from the roof and lateral wall of the nasopharynx, especially the cleft posterior to the Eustachian tube cartilage (fossa of Rosenmuller). Rich sub-mucosal lymphatics produce early dissemination to retropharygeal and jugulodigastric nodes, sometimes contralaterally. Local invasion of the base of skull, sinuses and upper pharyngeal constrictors tends to occur later. Distant metastases are commoner than with any other head and neck primary, the commonest sites of spread being to the lungs, liver and bones.

Histologically, the majority of these tumours appear poorly differentiated or anaplastic, giving rise to problems of precise classification. Electron microscopy and immunoperoxidase staining may be helpful in distinguishing them from true lymphomas. The intimate association of small lymphocytes with epithelial cells in many of these tumours has led to the concept of a hybrid tumour of “lympho-epithelioma”.

Presentation

- **Lump in Neck** An enlarged neck node occurs in 75 per cent of people with nasopharyngeal carcinoma, and is the commonest presenting symptom. Such nodes are frequently painless, but some may be exquisitely tender. Clinical examination of the nasopharynx is mandatory in such people, often providing the diagnosis without resorting to neck node biopsy, which compromises the chance of cure. Hence the importance of a thorough otorhino-laryngological assessment prior to any open neck node biopsy.

- **Unilateral Deafness** Obstruction of the medial end of the Eustachian tube by tumour produces a middle ear effusion and associated unilateral conductive hearing loss. This finding in an adult should always prompt a thorough clinical examination of the nasopharynx, under general anaesthetic if necessary.

- **Cranial Nerve Paralysis** A nasopharyngeal tumour may invade the base of skull in two directions:

    - Invasion upwards through the foramen lacerum results in a lesion of the nerves in the lateral wall of the cavernous sinus – III,IV,VI and the ophthalmic division of V – resulting in ophthalmoplegia or pain in the face

    - Invasion laterally and backwards. Along the base of the skull destroys the nerves passing through the jugular foramen – IX,X,XI – causing a motor or sensory paralysis of the pharynx or larynx and occasionally severe pain in the ear

- **Nasal Symptoms** The tumour may present with epistaxis or with nasal obstruction if the tumour is large.

Investigations

The nasopharynx is examined under general anaesthetic and a specimen obtained for biopsy.

Radiographic views of the base of skull may be complemented with CT scan to show evidence of bone destruction.

Elevated levels of anti-Epstein-Barr antibodies occur in 90 per cent of individuals with nasopharyngeal carcinoma. These may be IgA, IgM or IgG antibodies to early and capsid EBV antigens. High titres fall during treatment and may be useful in monitoring the course of the disease.

Treatment

Radiotherapy is the primary treatment of choice in all cases, surgical removal of the primary being virtually impossible. These tumours, especially the lympho-epitheliomas, are radiosensitive at both primary and nodal sites. Good responses to induction chemotherapy have also been reported, but as yet no survival benefit has been shown conclusively. Radical neck dissection is usually reserved for neck recurrence following successful eradication of the primary tumour.

Prognosis

Prognosis has improved considerably over the past 30 years with the introduction of better treatment planning following accurate staging.

Prognosis depends on Staging at diagnosis. Early (stage 1) has a 5 year survival rate of 70 - 80%. Stage 4 has a 5 year survival rate between 0 and 20%.

5.7.2 Assessment of PD D13 Claims

Primary neoplasm of the nasopharynx due to wood dust is indistinguishable from nasopharyngeal carcinoma due to other causes. It is important therefore that the HCP takes a full occupational history.

HCPs should note that carcinoma of the nasal cavity or associated air sinuses is already prescribed (PD D6). The occupations involved in the prescription of PD D13 are not the same as those for PD D6, and the latter does not state a minimum exposure time.

The nasopharynx is an air space lying at the back of the nose above the soft palate. It connects the back of the nose to the back of the mouth. If the claim form, FME (Further Medical Evidence), etc does not give the precise anatomical site of the primary or the tumour has spread locally from the nose to the nasopharynx or vice versa consideration may have to be given to which PD applies. The two anatomical sites involved are in close association and local spread may make the identification of the primary site difficult so the PD which applies may hinge on the occupational exposure.

There may be cases where the prescription test is satisfied for both D6 and D13. It unlikely that there will be 2 primary tumours, but should a case arise the relevant disability and disablement will be the same.

In cases of doubt, advice may be sought from the Health & Benefits Division of the DWP.

The full IIAC report on Nasopharyngeal Cancer due to exposure to wood dust can be read at:

https://www.gov.uk/government/organisations/industrial-injuries-advisory-council

The time rule for presumption for PD D13 has been extended to cover any time after leaving the job as well as in the job, however for this condition it has always been accepted in the Scheme that causation was due to work no matter how long ago the worker left the qualifying employment.

5.8 Prescribed Disease C3

Change to PD C3

The change is an update to the terms of prescription to reflect advances in understanding of the mechanism and nature of the toxic effects of chronic exposure to phosphorus and its compounds. The change narrows down the prescription from the general 'poisoning by phosphorus…' to the specific named diseases that are consequences of occupational exposure to compounds of phosphorus.

The amendment was added to the schedule from 30/03/2012.

The main change was to split C3 into C3a and C3b with a separate occupational prescription for each component.

There are no changes to disability assessment in PD C3.

Immediate or ongoing effects due to high dose accidental exposure to phosphorus or its compounds should be administered as accidents.

The disease was reviewed as part of the Industrial Injuries Advisory Council's (IIAC) review of the 'C' diseases. The recommendation was first made in their

report: Cm 5395 'Conditions due to Chemical Agents'. 2002. Implementation was delayed until potentially contradictory research was completed. The research has since been published and has had no effect on IIAC's recommendations.

5.8.1 C3a - Phossy Jaw – Osteonecrosis of the jaw

C3 (a) is 'Phossy jaw' due to 'work involving the use or handling of, or exposure to, white phosphorus.'

Phossy jaw has long been recognised to be due to chronic exposure to white phosphorus. Although not previously named in the schedule it has been a prescribed disease since the start of the scheme in 1948. The symptoms and signs are of osteonecrosis of the jaw. This disorder should not occur with modern working methods and has not been diagnosed in the UK for many decades. However it remains a potential hazard of chronic exposure to white phosphorus and as such is to remain a prescribed disease.

Osteonecrosis of the jaw remains a rare disease but has had a renaissance in recent years due to the use of biphosphonate drugs. Biphosphonate induced osteonecrosis of the jaw is more common in cancer patients than those taking the drugs for osteoporosis.

5.8.2 C3b – Peripheral polyneuropathy or peripheral polyneuropathy with pyramidal involvement of the central nervous system, caused by organic compounds of phosphorus which inhibit the enzyme neuropathy target esterase.

It is now known that tri-orthocresyl phosphate (IUPAC name tri-o-tolyl phosphate) and a number of other organophosphorus compounds have the capacity to inhibit an enzyme known as neuropathy target esterase (NTE). Poisoning by such compounds can result in disabling injury to peripheral nerves; symptoms of this include weakness and tingling or burning in the arms and legs.

In some cases damage may affect part of the central nervous system called the pyramidal tracts. The pyramidal tracts can be subdivided into the anterior corticospinal tract, the lateral corticospinal tract and the anterolateral corticospinal tract. These tracts consist of upper motor neurones that carry impulses from the motor cortex, in the brain, via the spinal cord to lower motor neurons. Signs of damage to the pyramidal tracts include muscle weakness with upper motor neurone signs. Note that UMN signs are likely to co-exist with LMN signs in this condition. For example there may be muscle wasting with brisk reflexes.

Neurological effects are usually but not always completely reversible.

Toxicity of this sort is rare. The compounds known to produce toxic effects via this mechanism of action during ordinary use are not licensed for use in the UK. It is likely that if these neurological effects due to NTE inhibition occur at all, they will be in the context of a large accidental exposure that has caused severe cholinergic toxicity.

However as long as toxicity by NTE inhibition as a result of non-accidental exposure remains a remote possibility in the UK the disease will remain prescribed.

5.8.3 What has been removed from the prescription and why?

References to anticholinesterase and pseudoanticholinesterase action of organic compounds of phosphorus have been removed from the prescription. Organic compounds of phosphorus that inhibit the enzyme acetylcholinesterase including organophosphate insecticides used in agriculture and sheep dip cause well documented cholinergic side effects. These include chest tightness, wheezing, muscle weakness, abdominal cramps, vomiting, diarrhoea, increased sweating, salivation and watering eyes in people who have been recently exposed to unusually high doses. There is evidence that in some cases acute poisoning of this type can be followed by long term deficits in intellectual performance and by peripheral neuropathy. There is no evidence to suggest that these effects occur with small cumulative exposures. As effects by this mechanism always follow an acute poisoning, claims due to the anticholinesterase (acetylcholinesterase inhibiting) effects of organophosphates, can be managed under the accident provisions.

The term ‘pseudo anticholinesterase’ is no longer used in toxicology. This term was intended to describe the neurological effects of tri-orthocresyl phosphate which was classified as a ‘pseudo anticholinesterase’ inhibitor.
It is now known that these organophosphorus compounds act by inhibiting an enzyme called neuropathy target esterase (NTE). Toxic effects by this mechanism can be administered under the accident provisions or C3(b) disease provisions depending on the circumstances of exposure in the individual case.

5.9 Prescribed Disease C31 – Bronchiolitis Obliterans

In its report “Bronchiolitis Obliterans and Food Flavouring Agents” the Industrial Injuries Advisory Council (IIAC) recommended that Bronchiolitis Obliterans should be prescribed in relation to an occupation that involves work in the production of diacetyl, or the manufacture of food flavourings containing diacetyl, or the manufacture of food flavoured by diacetyl.

Ministers accepted IIAC’s recommendation, and on 18th July 2011, regulations came into force which state that bronchiolitis obliterans is prescribed in relation to any occupation involving the use or handling of, or exposure to, diacetyl (also called butanedione or 2,3 butanedione) in the manufacture of (a) diacetyl; or (b) food flavouring containing diacetyl; or food to which food flavouring containing diacetyl is added.

The time rule for presumption has been extended to cover any time after leaving the job as well as in the job, however for this condition it has always been accepted in the Scheme as work related if the diagnosis was made and the exposure conditions met.

Bronchiolitis Obliterans in relation to diacetyl exposure is a very rare disease. In the existing literature there has only been one reported case in the UK. However, evidence from the United States and Holland has identified a very significant increase

in the risk of Bronchiolitis Obliterans in those who work in occupations involving manufacture, use or handling of diacetyl.

HCPs should note that diagnosis of Bronchiolitis Obliterans is usually made by specialist respiratory physicians. Hence confirmatory evidence in the form of hospital case notes (HCNs), including x-ray reports, should be sought in all cases.

Due to the rarity of the condition there is little extra burden on the advice – giving process by obtaining HCNs etc in all claims. Cases must be assessed by HCPs trained in Respiratory Prescribed Diseases.

Bronchiolitis obliterans is an uncommon respiratory disease characterised by fixed airway obstruction, due to the bronchioles becoming narrowed or blocked by fibrous tissue. The disease is disabling and can be potentially severe, and can be life-threatening, leading to lung transplantation in some individuals.

In addition to the rare cases caused by exposure to diacetyl, Bronchiolitis obliterans may be caused occupationally by accidental, acute, high concentration exposure to industrial gases, such as nitrogen dioxide, sulphur dioxide, phosgene, ammonia, chlorine and methyl isocyanate (as occurred in the Bhopal disaster in 1984). Such exposures may be covered by the accident provisions of the II Scheme.
Non-occupational causes of bronchiolitis obliterans include viral infections, rheumatoid disease, organ transplant rejection and adverse drug reactions.

In the UK diacetyl is used in potato crisp production.

People with bronchiolitis obliterans have reduced lung function and typically have dry cough, shortness of breath upon exertion and, occasionally, wheezing. Symptoms are similar to asthma and chronic obstructive pulmonary disease. However, a characteristic distinction between bronchiolitis obliterans and chronic obstructive pulmonary disease is the far more rapid onset and time course of symptoms in the former (weeks or months and less than a year) in contrast to the slow and insidious onset of breathlessness in the latter.

Diagnosis should normally be based upon prior hospital investigations, and confirmatory evidence of diagnosis must be sought from the appropriate source(s) - usually the hospital.

Diagnosis of bronchiolitis obliterans is based on clinical features, lung function tests, chest radiographs and high resolution computed tomography scans (HRCT), and lung biopsy.

Affected individuals often have severe airways obstruction on spirometry that is unresponsive to treatment with bronchodilator medication (fixed airways obstruction). Lung volumes are typically reduced.

HRCT is considered the most helpful means of diagnosing the disease. Characteristically, this shows a mosaic pattern of pulmonary lobules during inspiration, air trapping (hyperinflation) during expiration, irreversible patchy fibrosis,

causing the walls of less involved bronchioles to be pulled outwards (called 'traction bronchiectasis'), mucus plugging, airway wall thickening and consolidation.

Note: Chest x-rays may show hyperinflation in some but not in all cases. Lung biopsies may also appear normal due to sampling errors.

Symptoms are similar to chronic bronchitis, emphysema and asthma.

Bronchiolitis obliterans is distinguished from them due to the relatively rapid onset of symptoms (within weeks or months) following diacetyl exposure, as compared to the much slower progression of the development of chronic airways disease.

5.10 Prescribed Disease C32 – Carcinoma of the Nasal Cavity or associated Air Sinuses (Nasal Carcinoma)

In its report "Chromium and sino-nasal cancer" (Cm 7740) the Industrial Injuries Advisory Council (IIAC) recommended that carcinoma of the nasal cavity or associated air sinuses (nasal carcinoma) should be prescribed in relation to any occupation that involves work in hexavalent chrome plating or the manufacture of inorganic chromates.

Ministers accepted IIAC's recommendation, and the Regulations came into force on 18th July 2011.

- Chromium is a metal element that is present naturally in the form of its ore: chromite. Chromite is processed into chromates (manufacture of inorganic chromates). Following processing chromium exists in several different oxidation states; these include metallic chromium (chromium 0), trivalent chromium (chromium III) and hexavalent chromium (chromium VI)

- Hexavalent chromium (chromium VI) is the only one of these with carcinogenic potential. Consequently exposure to hexavalent chrome plating should be confirmed as other forms of chrome plating are used e.g. trivalent chrome plating

You should note particularly the similarity of the condition to PD D6. This is also identified as carcinoma of the nasal cavity and air sinuses, but the prescription for D6 is defined by a different occupational exposure (to wood dust).

Although carcinoma of the nasal cavity or associated nasal sinuses is relatively common in some parts of the world, it is extremely rare in the UK. In 2007, the latest year for which data is available, fewer than 500 cases of any cause were diagnosed in the UK.

Presentation is usually late, with locally advanced disease. This is because the tumour grows within the bony confines of the local anatomy and is often asymptomatic until it erodes or invades adjacent structures.

Symptoms may include feelings of intranasal mass or obstruction, rhinorrhea, epistaxis, cranial neuropathies or pain. Long-standing lesions may alter facial features in a detectable manner by causing asymmetry or proptoses.

Visual disturbances and paraesthesia are common.

In advanced disease, there may be ulceration, necrosis, bone and/or soft-tissue invasion.

Carcinoma of the nasal cavity or associated air sinuses (nasal carcinoma) encompasses cancers of the nasal cavity and the paranasal air sinuses – these are the maxillary, ethmoid, sphenoid and frontal sinuses. Cancers arising from the nasopharynx are excluded from the definition.

The nasopharynx and nasal cavity are adjacent but anatomically distinct areas. A cancer arising in the nasal cavity may extend into the nasopharynx and vice versa. Imaging studies and reports are likely to be the most useful pieces of further evidence to differentiate the point of origin of a tumour in an individual case.

The most common type of sino-nasal cancer accounting for approximately 60% is squamous cell carcinoma. Adenocarcinomas account for approximately 10 to 20% of sino-nasal cancers and a range of rarer types account for the rest. Local invasion is present in a high proportion and around 20% will have or develop distant metastases. 5 year survival is around 50%.

Squamous cell carcinomas are associated with human papilloma virus (HPV) infection, and HPV-positive individuals may have a better prognosis than those who are HPV-negative. The role of HPV and its interaction with occupational risk factors in this disease is not known. HPV status, where known, will be irrelevant to the diagnosis of the prescribed disease.

The accepted method of treatment is radiotherapy and surgery. Since most treatment failures occur within 2 years, follow-up is likely to be frequent and meticulous during this period.

Medical evidence must always be sought to confirm the diagnosis. Hospital case notes will be the optimum source of information.

Because carcinoma of the nasal cavity and associated air sinuses caused by working in hexavalent chrome plating or the manufacture of inorganic chromates is indistinguishable clinically and pathologically from sino-nasal cancer due to any other cause, including those sino-nasal cancers related to exposure to wood dust it is important that a full occupational history is taken and recorded in every case, as well as a social history (e.g. details of hobbies etc) and family history.

The time rule for presumption has been extended to cover any time after leaving the job as well as in the job, however for this condition it has always been accepted in the Scheme that causation was due to work no matter how long ago the worker left the qualifying employment.

The effects may be functional or cosmetic or a combination of both.

5.11 Prescribed Disease C33 – Chloracne

Definition: Chloracne

Date included in the legislation: 16/03/15

The Industrial Injuries Advisory Council (IIAC) undertook a comparison of diseases listed as occupational by the European Union and the International Labour Organisation with those prescribed in IIB, chloracne was listed by both of those schemes. The evidence on chloracne was formally reviewed and IIAC's conclusion was that this disease is occupational by nature, could occur in the UK and should become a prescribed disease. As chloracne is a rare condition this section contains extensive background information.

Chloracne is a systemic disease caused by exposure to certain halogenated aromatic hydrocarbons (Chloracnegens). Skin disfigurement is the most prominent manifestation. Following exposure spots develop. The nature and distribution of the spots and other features are specific to chloracne and therefore it can be differentiated from ordinary adult acne on the clinical features alone (see Table 1 on the next page). The appearance of the skin condition improves over time and usually resolves.
Testing of blood and tissue for the causative chemicals can be done but it is not necessary for the diagnosis of chloracne under the scheme. In the past chloracne was a common disease in workers in the chemical industry; industrial hygiene has improved since then, with only 8 UK cases reported since 1993.

Assessment of disablement and award duration:

Functional effects will relate to the social disability caused by facial disfigurement and any mental health condition the claimant has developed as a result of their disfigurement. The mental health condition may or may not improve as the skin condition improves. Cosmetic improvement in the appearance of the skin condition is expected over 5 years.

Chloracne is associated with systemic effects including liver dysfunction and fatigue; these effects are acute and will have resolved by the 91st day.

Table 1. Differential diagnosis of acne vulgaris and chloracne

	Acne Vulgaris	Chloracne
Clinical features		
Age group	Adolescence and early adulthood	Working age, with no previous history of acne
Predilection site	Localised, including face, back, chest	Generalised, including the area behind the ear, the cheek, armpit, groin and extremities
Major lesions	Limited comedones, papules, pustules, cysts	Myriad comedones
Pathogenic factors		
Inflammation lesions	Common	Very rare
Sebum production	Increased	Decreased
Microflora	*Propionibacterium acnes* and *Propionibacterium granulosum*	No bacteria
Androgen	Dependent	The role of androgens in chloracne is unknown
Histopathology		
Sebaceous gland	Hypertrophic and atrophic scarring	Gradual replacement with keratinocytes
Sweat gland	Uninvolved	Palmoplantar hyperkeratotic lesions; acrosyringial plugging
Hair follicle	Thinning of the infundibular epithelial wall sebaceous gland duct	General hyperplasia of the infundibulum and significant thickening of the upper follicle
Therapy		
	Effective under treatment of antibiotics	Resistant to therapy retinoids and other treatment

Background:

Chloracne was first described in 1887 by Von Bettman and by Herxheimer in 1889. They suggested that the condition was caused by chlorine exposure and named it 'chloracne', owing to the similarity between its clinical features and those of the more common condition, acne vulgaris. Cases of chloracne result from occupational and environmental exposures.

Chloracne was once common among workers occupationally exposed to naphthalene and chlorinated biphenyls, including workers from the chemical industry exposed to pesticides. Since the 1960s synthetic resins have replaced these compounds and the incidence of chloracne has fallen dramatically. However, some workers are still being exposed occupationally to relevant chemicals and are at risk of developing chloracne.

Dioxins are the most potent of the environmental chloracnegens and, rarely, individuals may become exposed through contaminated industrial waste, contaminated food products or following an industrial accident. A widely publicised accident occurred at a chemical plant near Seveso, Italy in 1976 (Caramaschi *et al.*, 1981). Two kilograms of the most toxic dioxin, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) were discharged into the atmosphere during an explosion and subsequently 135 cases of chloracne were diagnosed among 2,000 inhabitants. Elsewhere, hundreds of veterans of the Vietnam War were exposed to dioxin via exposure to the herbicide Agent Orange, which was found to be contaminated with TCDD.

Epidemiology:

The prevalence of chloracne in the UK is unknown but there are thought to be fewer than 4,000 cases worldwide. In the UK, a total of eight new cases were reported to The Health and Occupation Reporting network (THOR http://www.medicine.manchester.ac.uk\coeh\thor) between 1993-2011, although not all cases would be known to dermatologists and not all dermatologists nationally participate in this reporting scheme.

Toxicology:

All chloracnegenic (chloracne-inducing) compounds are known to share structural features including two benzene rings with halogen atoms occupying at least three of the lateral ring positions. The position of the halogen substitutions appears to be critical to chloracnegenic activity.

Chloracnegens are absorbed into the body by direct contact (through the skin), inhalation, or ingestion. The average concentration of dioxin in a healthy individual with no prior history of occupational or environmental exposure is less than 10 parts per trillion, but it is over several hundreds of parts per trillion in patients with chloracne. There is no known lower limit of exposure to chloracnegens below which the disease is certain not to occur. Likewise there is no known exposure level above which it is certain to occur.

Clinical features

Chloracnegens are highly lipophilic (fat soluble) and can remain in body fat for long periods. Hence, chloracne may be long-lived. Clinical symptoms normally occur 2 to 4 weeks after exposure to chloracnegens, but are often still present 2 to 3 years following cessation of exposure and are sometimes still present after 15 to 30 years.

Following exposure, inflammatory comedones and straw-coloured cysts develop. Occasionally, pustules and non-infectious abscesses develop. The distribution of these lesions in chloracne is characteristic. Initially skin lesions appear on the face and neck, and later extend to trunk, extremities, genitalia or other areas. Comedones appear on the face and neck, especially below and to the outer side of the eye (malar crescent) and in the postauricular triangles (behind the ears). The ear lobes, suboccipital hairline and groin are often involved. The skin around the nose and above the eyes is usually spared. The cysts typically affect the neck, shoulders, chest, back, penis and scrotum. Other skin effects include decreased sebum secretion with skin xerosis, pigmentation, porphyrinopathy, hirsutism, skin thickening, palmoplantar hidrosis and palmoplantar hyperkeratotisis.

Rarely, individuals with chloracne may develop non-cutaneous systemic manifestations including fatigue, anorexia, liver dysfunction, hyperlipidaemia, anaemia, transient weight loss and delayed nerve conduction on testing. Such systemic features do not occur in the absence of the skin disorder and tend to resolve well before the skin lesions.

Chloracne, in contrast to ordinary acne, appears to be resistant to all tested forms of treatment. The only way to control the disease is to prevent exposure to chloracnegens.

Outbreaks of occupational chloracne have tended to mirror the technological advances of the twentieth century. In the 1920s, halogen waxes, chlorinated naphthalenes and biphenyls were recognized as causes of chloracne, whereas in the 1940s, chloronaphthalenes and polychlorinated biphenyls (as used in the shipbuilding industry) were identified. More recently, chlorinated phenols and benzenes used as herbicides and insecticides have been a source of chloracne. The Council found evidence that some workers continue to be exposed to relevant chemicals at work and that cases of chloracne are still arising. Contemporary occupations and exposures vary from shoemaking (halogenates in glue) (Passarini et al., 2010), to fire fighters occupationally exposed to polychlorinated biphenyls (Orris et al., 1986).

In addition, cases of chloracne have recently been reported in discovery chemists synthesising novel polycyclic halogenated chemicals, which were classified as triazoloquinoxalines. Seven male pharmaceutical chemists experienced the onset of a facial and truncal chloracne eruption during 1997. Six of the men worked in one laboratory; the other worked elsewhere but had visited the laboratory in question. The chemists were employed discovering novel organic compounds in the pharmaceutical industry (Gawkrodger et al., 2009). These chemicals had not previously been known to be chloracnegenic.

In instances where chloracne develops in an individual with no known exposure to a chloracnegen, environmental or occupational, but exposure to one or more novel substances at work, formal toxicological assessment may be indicated.

A study of former chlorophenol workers found high serum dioxin levels, both in those with chloracne and those without it (Collins et al., 2006). Therefore, occupational exposure to chloracnegens does not necessarily result in the development of chloracne in all individuals.

In a longitudinal cohort study of workers occupationally exposed to chloracnegens, the mean duration of residual chloracne was 26 years and in some workers it had been present for 30 years (Moses et al., 1984). Skin examination in 288 veterans of the Vietnam War, 17 to 22 years after exposure to the herbicide Agent Orange, indicates that chloracne can persistent in the long term: 11.5% of subjects had the disease (Panteleyev et al., 1991).

There is no epidemiological evidence base to suggest fatigue as a result of Chloracne persists past the 90th day.

Action:

Chloracne cases must be discussed prior to completion with the Clinical Project Lead or Clinical Policy and Projects Lead as per existing C case guidance.

Specialist training is required to make the clinical diagnosis and within the IIB scheme evidence from someone with appropriate skills must be sought. This will usually be the consultant dermatologist treating the claimant. Absence of this level of specialist confirmation will result in advice that the diagnostic criteria are not met.
This does not require a face to face assessment.

Each case must be considered individually and skin disfigurement can persist for many years but an initial term of 5 years will enable significant improvement to occur in many cases. It is anticipated that any mental health condition related to the skin appearance will resolve with the skin condition, however the mental health condition may persist even if the skin condition completely resolves.

As a standard C case presumption does not apply.

5.12 Prescribed Disease D10 – Lung Cancer In Coke Oven Workers

The change is the addition of a new occupational group to D10 Primary carcinoma of the lung. The change follows recommendations made in the Industrial Injuries Advisory Council's (IIAC) report: 'Lung cancer in coke oven workers' Cm 8163 published September 2011. The prescription changed on 01/08/2012.

IIAC last considered lung cancer in coke oven workers in 1986. At that time it was known that coke oven emissions contained carcinogens such as polycyclic aromatic hydrocarbons, formaldehyde and phenol. Workers were exposed to these substances by inhalation and lung cancer was a plausible risk of such exposures. At the time there was some evidence of a higher incidence of lung cancer in some exposed workers but the dose required to double risk and whether the risk was consistent across all coke oven works was unknown. They concluded that there was not the evidence to recommend prescription at that time.

IIAC has reported on the updated literature in 2011, this mainly consists of long term follow up data from workers exposed to coke oven work before the 1980s.

Exposure levels to carcinogenic agents in coke oven emissions are likely to have reduced substantially since the 1970s following better plant design and personal safety controls. The updated findings suggest a more than doubled risk of lung cancer among coke oven workers but not amongst workers in the coke plant who are not directly engaged in oven work. The risk is highest in those exposed to top oven work. Industrial hygiene monitoring shows that carcinogen exposure is higher in the top oven role.

The disease is biologically plausible, the incidence of the disease shows a dose response to the putative carcinogen and the effect is seen in more than one population of exposed workers. Consequently IIAC has recommended prescription of lung cancer in coke oven workers. There is not enough evidence to suggest a cut off date when the risk of lung cancer became less than doubled in these workers but exposures in the industry are much reduced.

Primary lung cancer of any histological type is accepted. The diagnosis of D10 will be based on the occupational history alone as the carcinomas associated with the exposure have no features that distinguish them from lung cancers seen in the non-exposed population.

5.12.1 About Coke Ovens and the Coking Process

Coke plants convert coal into coke which is a dense fuel mainly used in blast furnaces at steel works. Coke ovens are massive structures that are built in rows known as batteries. Typically there are 25 to 66 ovens in each battery. Each oven has several chambers for heating, coking and regenerating heat. The ovens are sequentially filled, heated and emptied and cleaned so ensuring smooth processing of coal into coke and efficient use of skilled oven workers time.

The coal is charged through holes in the top of the oven and discharged as coke through doors on the sides of the oven. Usable by-products of the coking process are extracted during coking including ammonia, benzene, toluene, tar, oil and methane.

5.12.2 Job titles and job locations at coke works

The coke oven has many different components and there are many job titles associated with work on the various parts of the coke oven that can differ on a regional basis. In addition there are jobs in the coke works that are not related directly to oven work. Work in a coke works working on the coke oven is prescribed for D10, any other job that happens to be in the works is not prescribed. Work on the coke oven for the purposes of IIB can be divided into two categories.

The two categories relate to the scientific evidence of risk of lung cancer and they are:

- Top oven work
- Other oven work

Top oven work job titles include lidsman, car man (charger man), valveman or tarman, top oven maintenance worker.

All the other areas of the oven can be considered other oven work. In the original research a worker was counted as a top oven worker if he worked on the top oven the majority of the time and the same principle should be used for benefit assessment.

For example a man who occasionally covered other jobs or sometimes rotated to cover sickness absence who mainly worked on the top oven should be classified as a top oven worker for the period on which he mainly worked on the top oven. If he worked on different parts of the oven in the course of his career you will need to calculate how many years of top and other oven work he did and use these figures to aggregate his exposure to meet the prescription. The prescription differentiates between top oven work and other oven work because of the higher risk of lung cancer seen in top oven workers. The disease is prescribed after five years exposure in top oven workers and 15 years exposure in other oven workers. Exposures to top and other oven work in the course of a career can be aggregated. A diagram of a coke oven is provided at Appendix 9.

If a worker has carried out some top oven work, time served on the top oven is equivalent to three times as long working on other oven work. For example if a man has worked for 2 years on the top oven this would count for 6 years worth of other coke oven work and he would need to work for another 9 years in 'other' oven work to meet the prescribed period of 15 years exposure in aggregate. It may be that occupational histories will consist of periods working in the coke plant but not on the ovens, periods of other oven work and of top oven work.

The time rule for presumption has been extended to cover any time after leaving the job as well as in the job, however for this condition it has always been accepted in the Scheme that causation was due to work no matter how long ago the worker left the qualifying employment.

5.12.3 Diagnosis and Treatment of Lung Cancer

Evidence of diagnosis should be available from the claimant's doctor if it has not already been provided by the claimant with their claim form. Many of these claimants will be being fast tracked as priority claims because they are terminally ill. You may need to contact the claimant's GP by telephone to confirm the diagnosis so that the assessment can be completed without delay. Any other information such as stage and proposed treatment will be useful in the assessment of award duration and disablement assessment; however detailed further medical evidence is not required. It will be clear from the history in most cases whether the claimant is having palliative or curative treatment.

5.12.4 Disablement Assessment

The loss of faculty is lung impairment caused by primary carcinoma of the lung. Lung cancer usually presents at an advanced stage and treatment is generally palliative. Many of these claimants will be terminally ill.

In most cases a disablement assessment of 100% for life will be appropriate.

Those claimants undergoing potentially curative treatment are not terminally ill, the effects of their disease are likely to change over time, however for many in this group treatment outcomes are also poor. In many of these cases a fixed term award of 100% is appropriate with review at a later stage when prognosis has become clearer.

5.13 Prescribed Disease D9 – Unilateral or bilateral diffuse pleural thickening with obliteration of the costophrenic angle

The previous regulation detailing the diagnostic criteria for D9 diffuse pleural thickening relied on measurements to be taken on the standard PA chest x-ray. The increasing use of non-standard x-rays has meant that a standard PA chest x-ray is unlikely to be available. The legislation therefore changed following a recommendation from the Industrial Injuries Advisory Council (IIAC). The prescription of PD D9 was amended on 6th April 2006.

- The International Labour Office (ILO) has periodically published guidelines on how to classify radiographs for the pneumoconioses. The definition and guidance within the ILO system regarding the Costophrenic Angle Obliteration is as follows:

  'Costophrenic angle obliteration is recorded as either present or absent, separately for the right and for the left.

  The lower limit for recording costophrenic angle obliteration is defined by the standard Radiograph I / I , t / t .

  If the pleural thickening extends up the lateral chest wall from the obliterated costophrenic angle, as it does in I / I, t / t, the thickening should be classified as diffuse pleural thickening.

  Costophrenic angle obliteration may occur without diffuse pleural thickening'.

- The costophrenic angle may look abnormal but it may not be 'obliterated' as defined. There are other causes of abnormalities of the costophrenic angle. Particular consideration should be given to this possibility when there are no other changes attributable to pleural thickening.

- The ILO system does not use or clarify the term **blunting**, but this non-specific term is used by Radiologists to describe abnormal appearances of the area between the diaphragm and the lateral chest wall.

The following are necessary for the diagnosing of PD D9:

(a) An x-ray which shows obliteration of the costophrenic angle. At Appendix 10 there is an illustration of the minimum extent of 'obliteration'. (Note: this is for illustration purposes only and not comparison purposes). Lesser degrees would not constitute 'obliteration and would not fulfil the criteria for diagnosing

PD D9.
and
(b) There must be pleural thickening extending up the lateral chest wall as demonstrated in Appendix 10.

Diffuse Pleural Thickening causes restriction resulting in a decrement in the FVC or the TLC rather than FEV1. Thus claimants fulfilling the prescription for PD D9 are likely to have a reduction in lung function confirming a restrictive defect.

HCPs should note that:

- **The diagnosis of PD D9 requires both diffuse pleural thickening and obliteration, not just one of the criteria**. There must therefore be evidence of pleural thickening affecting the pleura of the chest wall associated with obliteration of the costophrenic angle.

- The term blunting is a non specific term used by Radiologists, whereas obliteration is more frequently likely to be used in reference to ILO classification so care must be taken in those few cases where x-rays are not available for the HCP to view to be confident that incorrect terminology is not being used. It is always important to consider whether associated pleural thickening is also described. e.g. if the report says “blunting”, is there also mention of thickening extending up the chest wall?

- It is therefore best practice to make every effort to obtain actual x-rays rather than relying on reports, as the latter may not have sufficient information regarding the presence of diffuse pleural thickening; or if there is reference to the costophrenic angle, whether the ‘changes are sufficient to fulfil the criteria for the diagnosis of PD D9.

- Diffuse Pleural thickening is not the only cause of radiological changes at the costophrenic angle. Other conditions such as previous pneumonia and effusion, pyothorax or chest trauma with haemothorax can cause obliteration or blunting of the costophrenic angle but are more likely to result in unilateral disease. Hence a careful evaluation is required when unilateral disease is present and it should not be assumed that the cause is definitely asbestos exposure. Pleural plaques and connective tissue disorders may also cause pleural abnormalities.

- In all cases (whether the actual x-ray is available to view or not), the HCP must explain and justify in their report why he/she is advising that D9 is or is not diagnosed. The claimant may have “diffuse pleural thickening” from a clinical point of view, and may have been given this diagnosis by his/her own medical practitioners so it is important to explain the distinction between the diagnosis from a clinical point of view, and the diagnosis of a prescribed disease. For the latter to be diagnosed the disease must have progressed to a degree where it fulfils the criteria laid down in the legislation, whereas the former may be diagnosed before the legal criteria are fulfilled.

- It is particularly important in all cases, but especially so if the x-ray is not available, to explain the facts of the case that leads the HCP to diagnose or not diagnose PD D9.

Summary

The diagnosis of PD D9 therefore needs a history of exposure to asbestos and obliteration of at least one costophrenic angle to the minimum degree illustrated and a clinical diagnosis of pleural thickening.

As asbestos related disease normally affects both angles, it is important to exclude other more likely causes of unilateral changes.

Where x-ray changes are in doubt or unclear in exposed individuals it is important to remember that obliteration with diffuse pleural thickening is likely be associated with a reduction in the FVC and TLC and a functional loss that reflects this. The FVC, is however, not a specific diagnostic criterion listed in the Regulations.

The time rule for presumption has been extended to cover any time after leaving the job as well as in the job, however for this condition it has always been accepted in the Scheme that causation was due to work no matter how long ago the worker left the qualifying employment.

6. Occupational Deafness PD A10

Occupational deafness is defined as:

"Sensorineural hearing loss amounting to at least 50 dB in each ear, being the average of hearing losses at 1, 2 and 3 kHz frequencies, and being due in the case of at least one ear to occupational noise (occupational deafness)".

In practice, the 50 dB average includes ageing effects. The overall hearing loss is first measured by air conduction audiometry. If the average of this over the 1, 2 and 3 kHz frequencies is 50 dB or more, bone conduction audiometry should have been carried out to measure the sensorineural loss. If the air/bone gap averaged over the 1, 2 and 3 kHz frequencies exceeds 10 dB then the bone conduction averages should be used to decide the diagnosis question. If the air/bone gap is 10 dB or less the air conduction averages should be used.

HCPs deal with claims broadly on the same lines as claims for other PDs but there are special provisions that are unique to this disease.

6.1 Diagnosis

In a case in which the occupational tests are satisfied, the claimant is referred for pure tone audiometry. The claimant's hearing is tested by pure tone audiometry using air and, if appropriate, bone conduction, and further tests as necessary may be carried out in order to determine the level of the sensorineural hearing threshold due to occupational noise and age and due to all causes.

The diagnosis question is referred to an HCP for advice on form BI 118 (OD) where:

- The audiometric report indicates that the claimant's loss of hearing satisfies the conditions detailed above, or
- The claimant appeals against the DM's decision that the diagnosis conditions are not satisfied

A recrudescence question (See Section 3.6.3) cannot arise on claims for occupational deafness because it is specifically excluded by the Regulations.

Procedural matters on diagnosis

In occupational deafness cases, the HCP will have an audiometric report. This report will incorporate pure tone audiograms, which must as a minimum include recordings at the 1, 2 and 3 kHz frequencies. An occupational deafness case will never be put to the HCP for a diagnosis advice without a report. On any report that records the audiometric findings, the HCP should always check these against the audiogram.

6.2 Assessment of disablement

General

Regulations provide that a 50 dB sensorineural hearing loss due to all causes in each ear and due in at least one ear to occupational noise, which is the minimum which would meet the diagnosis threshold. Such a case is assessed at 20 % and a 106 dB or greater loss in each ear is assessed at 100%. There are scheduled assessments for other intermediate levels of hearing loss and these are shown in the table in Appendix 2.

The practical effect of the above regulation is that a claimant who obtains a favourable answer to the diagnosis question cannot have his/her disablement initially assessed at less than 20%.

Specific Criteria related to a PD A10 claim

To meet the criteria for Occupational Deafness the claimant must have worked in employed earner's employment, in one or more of the prescribed occupations for a total of at least 10 years

The claim must be made within 5 years of leaving the employment which caused the deafness. Further claims can be made if an initial claim is turned down but this must be after a period of 3 years (unless by waiting for 3 years the claimant is excluded by the 5-year rule).

Method of assessment

Every case coming before an HCP for advice on assessment will contain one or more audiograms and an audiometric report. This will include details of the level of the sensorineural hearing loss due to occupational noise and age in each ear as well as information about any other otological conditions found.

The method of assessing the percentage binaural disablement due to occupational deafness is shown in regulations. For ease of assessing the percentage binaural disablement, the table in Appendix 2 should be used. Any fraction of an average hearing loss, where the average is over 50 dB must be rounded down to the next whole figure.

For the purpose of the calculation of the binaural disablement, once diagnosis has been satisfied, the better ear will be that ear in which the hearing loss from all causes is less. An HCP should give an exact assessment and should not round up or down. If the claimant has a sensorineural hearing loss of 50 dB or more in each ear due to all causes, and in at least one ear due to occupational noise and age averaged over 1, 2 and 3 kHz frequencies, then additional deafness from other causes must be taken into account in the assessment. In other words, the assessment will be based on the overall hearing loss due to all causes.

The assessment in respect of occupational deafness cannot start from a date earlier than the date of the claim in respect of which the occupational and diagnosis conditions are satisfied. This date is known as the date of onset. This assessment for PD A10 must be final.

Appeals

PD A10 claimants have the same rights of Appeal as in any other claim.

More Complex Presentations

The diagnosis and assessment of PDA10 is not always as outlined above. Claims can include irregularities within the audiometric evidence, inconsistencies at clinical examination, the presence of other hearing and ear disorders, or tinnitus. The reporting HCP can only respond to these variations in presentation through a full understanding of NIHL (Noise Induced Hearing Loss) and other ear disease together with a working knowledge of the various methods used to test hearing loss. For more details about testing see Appendix 2.

Appendix 1 - The Prescribed Diseases

Social Security (Industrial Injuries)

PD Regulations, Schedule 1, Regulations 2 and 4, Part 1

List of Prescribed Diseases and the occupations for which they are prescribed

A. CONDITIONS DUE TO PHYSICAL AGENTS	
Prescribed Disease	**Prescribed Occupation - any occupation involving:**
A1 Leukaemia (other than chronic lymphatic leukaemia) or cancer of the bone, female breast, testis or thyroid.	Exposure to electromagnetic radiations (other than radiant heat) or to ionising particles where the dose is sufficient to double the risk of the occurrence of the condition.
A2 Cataract.	Frequent or prolonged exposure to radiation from red-hot or white-hot material.
A3 A3(a) Dysbarism, including decompression sickness, barotrauma. A3(b) Osteonecrosis	Subjection to compressed or rarefied air or other respirable gases or gaseous mixtures.
A4 Task-specific focal dystonia of the hand or forearm.	Prolonged periods of handwriting, typing or other repetitive movements of the fingers, hand or arm.
A5 Subcutaneous cellulitis of the hand.	Manual labour causing severe or prolonged friction or pressure on the hand.
A6 Bursitis or subcutaneous cellulitis arising at or about the knee due to severe or prolonged external friction or pressure at or about the knee.	Manual labour causing severe or prolonged external friction or pressure at or about the knee.
A7 Bursitis or subcutaneous cellulitis arising at or about the elbow due to severe or prolonged external friction or pressure at or about the elbow.	Manual labour causing severe or prolonged external friction or pressure at or about the elbow.
A8 Traumatic inflammation of the tendons of the hand or forearm, or of the associated tendon sheaths.	Manual labour, or frequent or repeated movements of the hand or wrist.

A10 Sensorineural hearing loss amounting to at least 50dB in each ear, being the average of hearing losses at 1, 2 and 3 kHz frequencies, and being due in the case of at least one ear to occupational noise.(occupational deafness)	The use of, or work wholly or mainly in the immediate vicinity of the use of, a: (a) band saw, circular saw or cutting disc to cut metal in the metal founding or forging industries, circular saw to cut products in the manufacture of steel, powered (other than hand powered) grinding tool on metal (other than sheet metal or plate metal), pneumatic percussive tool on metal, pressurised air arc tool to gouge metal, burner or torch to cut or dress steel based products, skid transfer bank, knock out and shake out grid in a foundry, machine (other than a power press machine) to forge metal including a machine used to drop stamp metal by means of closed or open dies or drop hammers, machine to cut or shape or clean metal nails, or plasma spray gun to spray molten metal; (b) pneumatic percussive tool:- to drill rock in a quarry, on stone in a quarry works, underground, for mining coal, for sinking a shaft, or for tunnelling in civil engineering works; (c) vibrating metal moulding box in the concrete products industry, or circular saw to cut concrete masonry blocks; (d) machine in the manufacture of textiles for:- weaving man-made or natural fibres (including mineral fibres), high speed false twisting of fibres, or the mechanical cleaning of bobbins (e) multi-cutter moulding machine on wood, planing machine on wood, automatic or semiautomatic lathe on wood, multiple cross-cut machine on wood, automatic shaping machine on wood, double-end tenoning machine on wood, vertical spindle moulding machine (including a high speed routing machine) on wood, edge banding machine on wood, bandsawing machine (with a blade width of not less than 75 millimetres) on wood, circular sawing machine on wood including one operated by moving the blade towards the material being cut, or chain saw on wood; (f) jet of water (or mixture of water and abrasive material) at a pressure above 680 bar, or jet channelling process to burn stone in a quarry; (g) machine in a ship's engine room, or gas turbine for:- performance testing on a test bed, installation testing of a replacement engine in an aircraft, or acceptance testing of an Armed Service fixed wing combat aircraft; (h) machine in the manufacture of glass containers or hollow ware for:- automatic moulding, automatic blow moulding, or automatic glass pressing and forming; (i) spinning machine using compressed air to produce glass wool or mineral wool;

	(j) continuous glass toughening furnace; (k) firearm by a police firearms training officer; or (l) shot-blaster to carry abrasives in air for cleaning.
A11 (a) Intense blanching of the skin, with a sharp demarcation line between affected and non-affected skin, where the blanching is cold-induced, episodic, occurs throughout the year and affects the skin of the distal with the middle and proximal phalanges, or distal with the middle phalanx (or in the case of a thumb the distal with the proximal phalanx) of (i) in the case of a person with 5 fingers (including thumb) on one hand, any 3 of those fingers, or (ii) in the case of a person with only 4 such fingers, any 2 of those fingers, or (iii) in the case of a person with less than 4 such fingers, any one of them, or as the case may be, the one remaining finger, where none of the person's fingers was subject to any degree of cold induced, episodic blanching of the skin prior to the person's employment in an occupation described in the second column in relation to this paragraph, or (b) Significant, demonstrable reduction in both sensory perception and manipulative dexterity with continuous numbness or continuous tingling all present at the same time in the distal phalanx of any finger (including thumb) where none of the person's fingers was subject to any degree of reduction in sensory perception, manipulative dexterity, numbness or tingling prior to the person's employment in an occupation described in the second column in relation to this paragraph, where the symptoms in paragraph (a) or paragraph (b) were caused by vibration	(a) the use of hand-held chain saws on wood; (b) the use of hand-held rotary tools in grinding or in the sanding or polishing of metal, or the holding of material being ground, or metal being sanded or polished, by rotary tools; (c) the use of hand-held percussive metal-working tools, or the holding of metal being worked upon by percussive tools, in riveting, caulking, chipping, hammering, fettling or swaging; (d) the use of hand-held powered percussive drills or hand-held powered percussive hammers in mining, quarrying, demolition, or on roads or footpaths, including road construction; (e) the holding of material being worked upon by pounding machines in shoe manufacture.

A12 Carpal tunnel syndrome	(a) The use, at the time the symptoms first develop, of hand-held powered tools whose internal parts vibrate so as to transmit that vibration to the hand, but excluding those tools which are solely powered by hand; (b) repeated palmar flexion and dorsiflexion of the wrist for at least 20 hours per week for a period or periods amounting in aggregate to at least 12 months in the 24 months prior to the onset of symptoms where repeated means once or more often in every 30 seconds.
A13 Osteoarthritis of the hip	Work in agriculture as a farmer or farm worker for a period of, or periods which amount in aggregate to, 10 years or more.
A14 Osteoarthritis of the knee	1. Work underground in a coal mine for a period of, or periods which amount in aggregate to, at least 10 years in any one or more of the following occupations: (a) before 1st January 1986 as a coal miner; or (b) on or after 1st January 1986 as a – (i) face worker working on a non-mechanised coal face; (ii) development worker; (iii) face-salvage worker; (iv) conveyor belt cleaner; or (v) conveyor belt attendant "A non-mechanised coal face" means a coal face without either powered roof supports or a power loader machine which simultaneously cuts and loads the coal or without both." 2. Work wholly or mainly fitting or laying carpets or floors, (other than concrete floors) for a period of, or periods which amount in aggregate to, 20 years or more.

B: CONDITIONS DUE TO BIOLOGICAL AGENTS	
Prescribed Disease	**Prescribed Occupation - any occupation involving:**
B1 B1(a) – Cutaneous anthrax B1(b) – Pulmonary anthrax	(a) Contact with anthrax spores, including contact with animals infected by anthrax; or (b) Handling, loading, unloading or transport of animals of a type susceptible to infection with anthrax or of the products or residues of such animals
B2 Glanders	Contact with equine animals or their carcasses.
B3 Infection by leptospira.	a) Work in places which are, or are liable to be, infested by rats, field mice or voles, or other small mammals; or b) Work at dog kennels or the care or handling of dogs; or c) Contact with bovine animals or their meat products or pigs or their meat products.
B4 B4(a) - Cutaneous larva migrans B4(b) – Iron deficiency anaemia caused by gastrointestinal infection by hookworm	Contact with a source of ankylostomiasis
B5 Tuberculosis.	Contact with a source of tuberculosis while undertaking: a) work in a hospital, mortuary in which post mortems are conducted or laboratory, or b) work in any other workplace
B6 Extrinsic allergic alveolitis (including farmer's lung)	Exposure to moulds or fungal spores or heterologous proteins by reason of employment: a) agriculture, horticulture, forestry, cultivation of edible fungi or malt-working; or b) loading or unloading or handling in storage mouldy vegetable matter or edible fungi; or c) caring for or handling birds; or d) handling bagasse e) work involving exposure to metalworking fluids mists.

B7 Infection by organisms of the genus brucella.	Contact with– a) animals infected by brucella, or their carcasses or parts thereof, or their untreated products; or b) laboratory specimens or vaccines of, or containing brucella.
B8A Infection by hepatitis A virus	Contact with raw sewage
B8B Infection by hepatitis B or C virus	Contact with a) human blood or human blood products; or b) any other source of hepatitis B or C virus
B9 Infection by Streptococcus suis	Contact with pigs infected by Streptococcus suis, or with the carcasses, products or residues of pigs so infected.
B10(a) Avian chlamydiosis	Contact with birds infected with chlamydia psittaci, or with the remains or untreated products of such birds.
B10(b) Ovine chlamydiosis	Contact with sheep infected with chlamydia psittaci, or with the remains or untreated products of such sheep.
B11 Q fever	Contact with animals, their remains or their untreated products.
B12 Orf	Contact with sheep, goats, or with the carcasses of sheep or goats.
B13 Hydatidosis	Contact with dogs.
B14 Lyme Disease	Exposure to deer or other mammals of a type liable to harbour ticks harbouring Borrelia bacteria
B15 Anaphylaxis	Employment as a healthcare worker having contact with products made with natural rubber latex

C: CONDITIONS DUE TO CHEMICAL AGENTS	
Prescribed Disease or Injury	**Prescribed Occupation - any occupation involving:**
C1 C1(a) Anaemia with a haemoglobin concentration of 9g/dL or less, and a blood film showing punctate basophilia; C1(b) peripheral neuropathy; C1(c) central nervous system toxicity.	The use or handling of, or exposure to the fumes, dust or vapour of, lead or a compound of lead, or a substance containing lead.
C2 Central nervous system toxicity characterised by parkinsonism.	The use or handling of, or exposure to the fumes, dust or vapour of, manganese or a compound of manganese, or a substance containing manganese.
C3 (a) Phossy Jaw (b) Peripheral polyneuropathy or peripheral polyneuropathy with pyramidal involvement of the central nervous system, caused by organic compounds of phosphorous which inhibit the enzyme neuropathy target esterase.	a) Work involving the use or handling of, or exposure to, white phosphorous. b) Work involving the use or handling of, or exposure to, organic compounds of phosphorous.
C4 Primary carcinoma of the bronchus or lung.	Exposure to the fumes, dust or vapour of arsenic, a compound of arsenic or a substance containing arsenic.
C5A Central nervous system toxicity characterised by tremor and neuropsychiatric disease	Exposure to mercury or inorganic compounds of mercury for a period of, or periods which amount in aggregate to, 10 years or more.
C5B Central nervous system toxicity characterised by combined cerebellar and cortical degeneration.	Exposure to methylmercury.
C6 Peripheral neuropathy.	The use or handling of, or exposure to, carbon disulphide (also called carbon disulfide)
C7 Acute non-lymphatic leukaemia.	Exposure to benzene.
C8, C9, C10 and C11 removed from schedule.	

C12 (a) Peripheral neuropathy; (b) Central nervous system toxicity.	Exposure to methyl bromide (also called bromomethane).
C13 Cirrhosis of the liver.	Exposure to chlorinated naphthalenes.
C14 and C15 removed from schedule	
C16 (a) Neurotoxicity; (b) Cardiotoxicity.	Exposure to the dust of gonioma kamassi.
C17 Chronic beryllium disease.	Inhalation of beryllium or a beryllium compound.
C18 Emphysema.	Inhalation of cadmium fumes for a period of, or periods which amount in aggregate to, 20 years or more.
C19 (a) Peripheral neuropathy; (b) Central nervous system toxicity.	Exposure to acrylamide.
C20 Dystrophy of the cornea	Exposure to quinone or hydroquinone
C21 Primary carcinoma of the skin.	Exposure to arsenic or arsenic compounds, tar, pitch, bitumen, mineral oil (including paraffin) or soot
C22 (a) Primary carcinoma of the mucous membrane of the nose or paranasal sinuses; (b) Primary Carcinoma of the bronchus or lung.	Work before 1950 in the refining of nickel involving exposure to oxides, sulphides or water-soluble compounds of nickel.
C23 Primary neoplasm of the epithelial lining of the urinary tract.	(a) The manufacture of 1-naphthylamine, 2-naphthylamine, benzidine, auramine, magenta or 4-aminobiphenyl (also called biphenyl-4-ylamine); (b) work in the process of manufacturing methylene-bisorthochloroaniline (also called MbOCA) for a period of, or periods which amount in aggregate to, 12 months or more; (c) exposure to 2-naphthylamine, benzidine, 4-aminobiphenyl (also called biphenyl-4-ylamine) or salts of those compounds otherwise than in the manufacture of those compounds; (d) exposure to orthotoluidine, 4-chloro-2-methylaniline or salts of those compounds; or (e) exposure for a period of, or periods which amount in aggregate to, 5 years or more, to coal tar pitch volatiles produced in aluminium smelting

	involving the Soderberg process (that is to say, the method of producing aluminium by electrolysis in which the anode consists of a paste of petroleum coke and mineral oil which is baked in situ).
C24 (a) Angiosarcoma of the liver; or (b) Osteolysis of the terminal phalanges of the fingers; or (c) Sclerodermatous thickening of the skin of the hand; or (d) Liver fibrosis due to exposure to vinyl chloride monomer	Exposure to vinyl chloride monomer in the manufacture of polyvinyl chloride
C24A Raynaud's phenomenon due to exposure to vinyl chloride monomer	Exposure to vinyl chloride monomer in the manufacture of polyvinyl chloride before 1 January 1984
C25 Vitiligo.	The use or handling of, or exposure to, paratertiarybutylphenol (also called 4-tertbutylphenol), paratertiarybutylcatechol (also called 4-tertbutylcatechol), para-amylphenol (also called p-pentyl phenol isomers), hydroquinone, monobenzyl ether of hydroquinone (also called 4-benzyloxyphenol) or mono-butyl ether of hydroquinone (also called 4-butoxyphenol).
C26 (a) Liver toxicity; (b) Kidney toxicity.	The use or handling of, or exposure to, carbon tetrachloride (also called tetrachloromethane).
C27 Liver toxicity.	The use or handling of, or exposure to, trichloromethane (also called chloroform).
C28 removed from schedule.	
C29 Peripheral neuropathy.	The use or handling of, or exposure to, n-hexane or n-butyl methyl ketone
C30 (a) Dermatitis; (b) Ulceration of the mucous membrane or the epidermis.	The use or handling of, or exposure to, chromic acid, chromates or dichromates.

C31 Bronchiolitis obliterans	The use or handling of, or exposure to, diacetyl (also called butanedione or 2,3-butanedione) in the manufacture of: (a) diacetyl; or (b) food flavouring containing diacetyl; or (c) food to which flavouring containing diacetyl is added.
C32 Carcinoma of the nasal cavity or associated air sinuses	(a) the manufacture of inorganic chromates; or (b) work in hexavalent chrome plating.
C33 Chloracne	Exposure to a substance causing chloracne

D. MISCELLANEOUS CONDITIONS

Prescribed Disease or Injury	Prescribed Occupation- any occupation involving:
D1 Pneumoconiosis	(1)(a) The mining, quarrying or working of silica rock or the working of dried quartzose sand or any dry deposit or dry residue of silica or any dry admixture containing such materials (including any occupation in which any of the aforesaid operations are carried out incidentally to the mining or quarrying of other minerals or to the manufacture of articles containing crushed or ground silica rock); (b) the handling of any of the materials specified in the foregoing subparagraph in or incidental to any of the operations mentioned therein, or substantial exposure to the dust arising from such operations. (2) The breaking, crushing or grinding of flint or the working or handling of broken, crushed or ground flint or materials containing such flint, or substantial exposure to the dust arising from any such operations (3) Sand blasting by means of compressed air with the use of quartzose sand or crushed silica rock or flint, or substantial exposure to the dust arising from sand and blasting. (4) Work in a foundry or the performance of, or substantial exposure to the dust arising from, any of the following operations:- (a) the freeing of steel castings from adherent siliceous substance; (b) the freeing of metal castings from adherent siliceous substance: (i) by blasting with an abrasive propelled by compressed air, by steam or by a wheel, or (ii) by the use of power-driven tools. (5) The manufacture of china or earthenware (including sanitary earthenware, electrical earthenware and earthenware tiles), and any occupation involving substantial exposure to the dust arising there from. (6) The grinding of mineral graphite, or substantial exposure to the dust arising from such grinding. (7) The dressing of granite or any igneous rock by masons or the crushing of such materials, or substantial exposure to the dust arising from such operations. (8) The use, or preparation for use, of a grindstone, or substantial exposure to the dust arising there from. (9) (a) The working or handling of asbestos or any admixture of asbestos;

	(b) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos; (c) the cleaning of any machinery or plant used in any foregoing operations and of any chambers, fixtures and appliances for the collection of asbestos dust; (d) substantial exposure to the dust arising from any of the foregoing operations. (10)(a) Work underground in any mine in which one of the objects of the mining operations is the getting of any mineral; (b) the working or handling above ground at any coal or tin mine of any minerals extracted there from, or any operation incidental thereto; (c) the trimming of coal in any ship, barge, or lighter, or in any dock or harbour or at any wharf or quay; (d) the sawing, splitting or dressing of slate, or any operation incidental thereto. (11) The manufacture of carbon electrodes by an industrial undertaking for use in the electrolytic extraction of aluminium from aluminium oxide, and any occupation involving substantial exposure to the dust arising there from. (12) Boiler scaling or substantial exposure to the dust arising there from. (13) Exposure to dust if the person employed in it has never at any time worked in any of the other occupations listed.
D2 Byssinosis.	Work in any room where any process up to and including the weaving process is performed in a factory in which the spinning or manipulation of raw or waste cotton or of flax, or the weaving of cotton or flax, is carried on.
D3 Diffuse mesothelioma (primary neoplasm of the mesothelium of the pleura or of the pericardium or of the peritoneum).	Exposure to asbestos, asbestos dust or any admixture of asbestos at a level above that commonly found in the environment at large.

D4 Allergic rhinitis which is due to exposure to any of the following agents – (a) isocyanates; (b) platinum salts; (c) fumes of dusts arising from the manufacture, transport or use of hardening agents (including epoxy resin curing agents) based on phthalic anhydride, tetrachlorophthalic anhydride, trimellitic anhydride or triethylenetetramine (d) fumes arising from the use of rosin as a soldering flux; (e) proteolytic enzymes; (f) animals including insects and other arthropods used for the purposes of research or education or in laboratories; (g) dusts arising from the sowing, cultivation, harvesting, drying, handling, milling, transport or storage of barley, oats, rye, wheat or maize, or the handling, milling, transport or storage of meal or flour made there from (h) antibiotics (i) cimetidine; (j) wood dust; (k) ispaghula; (l) castor bean dust; (m) ipecacuanha; (n) azodicarbonamide; (o) animals including insects and other arthropods or their larval forms, used for the purposes of pest control or fruit cultivation, or the larval forms of animals used for the purposes of research, education or in laboratories (p) glutaraldehyde; (q) persulphate salts or henna; (r) crustaceans or fish or products arising from these in the food processing industry; (s) reactive dyes; (t) soya bean; (u) tea dust; (v) green coffee bean dust; (w) fumes from stainless steel welding. (x) products made with natural rubber latex	Exposure to any of the agents set out in column 1 of this paragraph
D5 Non-infective dermatitis of external origin (excluding dermatitis due to ionising particles or electromagnetic radiations other than radiant heat).	Exposure to dust, liquid or vapour or any other external agent except chromic acid, chromates or bi-chromates, capable of irritating the skin (including friction or heat but excluding ionising particles or electromagnetic radiations other than radiant heat).

D6 Carcinoma of the nasal cavity or associated air sinuses (nasal carcinoma).	(a) Attendance for work in or about a building where wooden goods are manufactured or repaired; or (b) attendance for work in a building used for the manufacture of footwear or components of footwear made wholly or partly of leather or fibreboard; or (c) attendance for work at a place used wholly or mainly for the repair of footwear made wholly or partly of leather or fibreboard.
D7 Asthma which is due to exposure to any of the following agents – (a) isocyanates; (b) platinum salts; (c) fumes or dusts arising from the manufacture, transport or use of hardening agents (including epoxy resin curing agents) based on phthalic anhydride, tetrachlorophthalic anhydride, trimellitic anhydride or triethylenetetramine (d) fumes arising from the use of rosin as a soldering flux; (e) proteolytic enzymes; (f) animals including insects and other arthropods used for the purposes of research or education or in laboratories; (g) dusts arising from the sowing, cultivation, harvesting, drying, handling, milling, transport or storage of barley, oats, rye, wheat or maize, or the handling, milling, transport or storage of meal or flour made there from: (h) antibiotics; (i) cimetidine; (j) wood dust; (k) ispaghula; (l) castor bean dust; (m) ipecacuanha; (n) azodicarbonamide; (o) animals including insects and other arthropods or their larval forms, used for the purposes of pest control or fruit cultivation, or the larval forms of animals used for the purposes of research, education or in laboratories (p) glutaraldehyde; (q) persulphate salts or henna; (r) crustaceans or fish or products arising from these in the food processing industry;	Exposure to any of the agents set out in column 1 of this paragraph

(s) reactive dyes; (t) soya bean; (u) tea dust; (v) green coffee bean dust; (w) fumes from stainless steel welding; (wa) products made with natural rubber latex (x) any other sensitising agent (occupational asthma).	
D8 Primary carcinoma of the lung where there is accompanying evidence of asbestosis.	(a) The working or handling of asbestos or any admixture of asbestos; or (b) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos; or (c) the cleaning of any machinery or plant used in any of the foregoing operations and of any chambers, fixtures and appliances for the collection of asbestos dust; or (d) substantial exposure to the dust arising from any of the foregoing operations.
D8A Primary carcinoma of the lung	Exposure to asbestos, in the course of:- (a) The manufacture of asbestos textiles; or (b) spraying asbestos; or (c) asbestos insulation work; or (d) applying or removing materials containing asbestos in the course of ship building, where all or any of the exposure occurs before 1 January 1975, for a period of, or periods which amount in aggregate to, five years or more, or otherwise, for a period of, or periods which amount in aggregate to, ten years or more.
D9 Unilateral or bilateral diffuse pleural thickening with obliteration of the costophrenic angle	(a) The working or handling of asbestos or any admixture of asbestos; or (b) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos; or (c) the cleaning of any machinery or plant used in any of the foregoing operations and of any chambers, fixtures and appliances for the collection of asbestos dust; or (d) substantial exposure to the dust arising from any of the foregoing operations.

D10 Primary carcinoma of the lung.	(a) Work underground in a tin mine; or (b) exposure to bis (chloromethyl) ether produced during the manufacture of chloromethyl methyl ether; or (c) exposure to zinc chromate, calcium chromate or strontium chromate in their pure forms. d) employment wholly or mainly as a coke oven worker: (i) for a period of, or periods which amount in aggregate to, 15 years or more; or (ii) in top oven work, for a period of, or periods which amount in aggregate to, 5 years or more; or (iii) in a combination of top oven work and other coke oven work for a total aggregate period of 15 years or more, where one year working in top oven work is treated as equivalent to 3 years in other coke oven work.
D11 Primary carcinoma of the lung where there is accompanying evidence of silicosis	Exposure to silica dust in the course of: (a) the manufacture of glass or pottery; (b) tunnelling in or quarrying sandstone or granite; (c) mining metal ores; (d) slate quarrying or the manufacture of artefacts from slate; (e) mining clay; (f) using siliceous materials as abrasives; (g) cutting stone; (h) stonemasonry; or (i) work in a foundry.
D12 Chronic obstructive pulmonary disease where there is evidence of a forced expiratory volume in one second (measured from the position of maximum inspiration with the claimant making maximum effort) which is: (i) at least one litre below the appropriate mean value predicted, obtained from the following prediction formulae which give the mean values predicted in litres: • For a man, where the measurement is made without back-extrapolation, (3.62 x Height in metres) - (0.031 x Age in years) -1.41; or, where the measurement is made with back-extrapolation, (3.71 x Height in metres) - (0.032 x Age in years) -	Exposure to coal dust (whether before or after 5th July 1948) by reason of working – (a) underground in a coal mine for a period or periods amounting in aggregate to at least 20 years (b) on the surface of a coal mine as a screen worker for a period or periods amounting in aggregate to at least 40 years before 1st January 1983; or (c) both underground in a coal mine, and on the surface as a screen worker before 1st January 1983, where 2 years working as a surface screen worker is equivalent to 1 year working underground, amounting in aggregate to at least the equivalent of 20 years underground. Any such period or periods shall include a period or periods of incapacity while engaged in such an occupation.

1.44; ▪ For a woman, where the measurement is made without back-extrapolation, (3.29 x Height in metres) -(0.029 x Age in years) -1.42; or, where the measurement is made with back-extrapolation, (3.37 x Height in metres) -(0.030 x Age in years) -1.46; or (ii) less than one litre The value of one litre in (i) and (ii) above shall be construed as fixed, and shall not vary by virtue of any treatment or treatments.	
D13 Primary carcinoma of the nasopharynx	Exposure to wood dust in the course of the processing of wood or the manufacture or repair of wood products, for a period or periods which amount in aggregate to at least 10 years.

Appendix 2 - Information to assist with a claim to Occupational Deafness

Structure of the Ear, Causes of Deafness, and Assessment of Hearing

Structure of the ear

The ear is in 3 parts: -

- The external ear - the pinna, and the external auditory canal, which ends at the Tympanic Membrane
- The middle ear – which starts at the inner surface of the tympanic membrane and contains the ossicles, (or ossicular chain), the last of which connects to the oval window. The middle ear is connected to the throat by the Eustachian tube and therefore contains air
- The inner ear, which as well as having the Cochlea, (the organ of hearing), contains the organ which controls balance. The cochlea is lined with hair cells that have small hairs (cilia) which move in response to sound waves. Different cilia respond to different frequencies of sound. The cilia, which respond to high frequency sound, are much more fragile than those responding to low frequency sound, and hence are more susceptible to damage. When sound is transmitted to the inner ear, the cilia move. This movement is transferred, as an electrical impulse, to the brain. The louder the noise, the more the cilia move. (See below with regard to TTS and PTS)

Hearing

The loudness of a sound is measured in decibels (dB). The decibel scale is a logarithmic scale. O dB does not mean that there is no sound. A doubling of loudness is not reflected in a doubling of the dB reading.

The minimum point at which a sound can be heard is known as the threshold.

This hearing threshold may be temporarily increased. For example following a rock concert, performers and fans, notice that the loudness of a noise they can hear has to be increased above the usual level for them due to temporary damage to the ear. This phenomenon is known as Temporary Threshold Shift (TTS), and is due to the cilia in the cochlea being temporarily 'stunned' and unable to move. With continued noise exposure, over time these cilia die causing permanent damage to hearing. Once permanent damage occurs there needs to be a louder noise before one can hear. This is known as Permanent Threshold Shift (PTS). This permanent shift may be as a result of several different causes, including exposure to excessive noise.

Sound waves enter the external auditory canal and cause the tympanic membrane to vibrate.

This in turn causes the ossicles to vibrate and oscillate the oval window, thereby moving the fluid in the cochlea, which in turn causes the cilia to move. The cilia convert the movement into an electrical impulse, which is transmitted via the auditory nerve to the brain, which then recognises that there has been a noise, which it then interprets.

The transmission of sound described above is known as Air Conduction (AC).

In addition to sound being transmitted by air conduction, it can be transmitted through the bones of the skull, bypassing the outer and middle ear, directly to the inner ear. This is known as Bone Conduction (BC). If there are any problems in the outer and middle ears, then bone conduction hearing will be better than air conduction.

Whilst AC can be worse than BC it can never be better than BC because both routes of sound transmission go to the same place, that is the inner ear.

NB. Occasionally the audiogram may show the AC to be a few dB better than BC, but this is due to subject error and/or limitations in audiometry and cannot reflect a true difference.

Causes of deafness

Loss of conduction of sound to the inner ear due to impairment of transmission through the external and/or middle ear is a conductive hearing loss which may be caused by:

- Blockage in the external canal, for example by wax or a foreign body
- Perforation of the tympanic membrane
- Fluid in the middle ear, as a result of infection, or blockage of the Eustachian Tube
- Damage to the ossicles, due to trauma or otosclerosis

Loss of sensorineural detection of sound, due to disorder of the cochlea in the inner ear or the auditory nerve and its central connections is sensorineural hearing loss which may be caused by:

Congenital and Hereditary causes such as:

- Familial deafness
- Pre-natal infections such as Rubella

Acquired causes:

- Infections such as mumps, meningitis

- Trauma such as a severe head injury
- Ototoxic drugs including streptomycin, quinine, and high doses of aspirin
- Degenerative causes – the effect of ageing on the cochlea and auditory nerve causing deafness
- Explosions
- Tumour, such as an acoustic neuroma
- Noise

Noise induced deafness

The louder the noise, the more the fluid in the cochlea moves, and hence the more the cilia move. The cilia are fragile and with prolonged exposure to noise, are destroyed and no longer transmit sound. The cilia that respond to high frequency noise are more fragile than those responding to low frequency sound and are thus more susceptible to damage, and hence noise induced deafness affects the higher frequencies before it affects the lower frequencies.

Noise induced deafness:

- Is slowly progressive if exposure to noise continues
- Takes many years to become appreciably deaf
- Is unlikely to be the cause of rapid onset and/or rapidly progressive deafness, when another cause should be sought
- Will not progress once exposure to noise ceases
- Other factors such as ageing may cause greater deterioration
- Is usually bilaterally symmetrical, with very similar patterns in both ears
- Does not cause a profound deafness
- Has a low frequency maximum loss at 40 dB, and a high frequency maximum loss is about 75 dB

In an individual hearing loss (deafness) may be due to a conductive cause, a sensorineural cause, or a combination. In addition, occasionally the claimant may exaggerate the symptoms, or have a psychogenic deafness.

In this case the cause is not within the hearing mechanism, but due to a psychiatric cause, often described as Non-organic hearing loss (NOHL), in other words a feigned hearing loss.

An experienced audiometrician will recognise audiometric inconsistencies indicative of NOHL. The more experienced the tester, the more likely the detection of feigned hearing loss. Where there is evidence of inconsistencies on audiometric testing individuals should be referred for objective hearing assessment (See below).

Audiometric inconsistencies that suggest NOHL are: -

- Variable Audiometric Responses - Some individuals, particularly those feigning hearing loss, often give varying and inconsistent responses

- Flat Audiometric Configuration - Individuals exhibiting bilateral non-organic hearing loss often present with pure tone thresholds that have a relatively flat audiometric configuration. This is due to the individual adopting an imaginary supra threshold “target loudness level”. A pure tone stimulus having a loudness below the individual’s chosen “criterion” will not elicit a response whilst a stimulus that approaches it will. In some cases there will be an underlying hearing impairment, with the additional non-organic element superimposed

- Informal observations of Hearing Ability - The degree of hearing impairment recorded on the audiogram should be a reasonable reflection of an individual’s hearing ability. For example, if the audiogram suggests a loss of 90 dB bilaterally but it is possible to converse with the individual in a reasonably quiet voice without visual cues, then there is strong reason to doubt the audiometric validity

- Low Frequency Loss - Noise exposure and age related changes generally cause sensorineural hearing loss over the frequencies from 3kHz to 8kHz. Therefore if the audiogram has a significant loss at the lower frequencies, consideration should be given to performing objective testing, that is ERA

- Exaggerated hearing loss on Pure Tone Audiometry - It is relatively easy to feign PTA to show a worse hearing loss than is actually present. (See 2nd bullet point above). However it is very difficult to do the reverse, that is, make one’s hearing appear to be better than it actually is, as this would require consistently guessing when a sound that you could not hear was being presented. The tester does not present the sounds in an orderly manner and out of view of the person being tested. Hence it is not possible to predict when a sound is being presented in order that a response can be made. On some occasions the response will correlate with the presentation of a sound, but this will be purely by chance, and show an inconsistent and unreliable audiometric pattern

Hearing Testing

General

There are several methods of testing hearing. In some circumstances only a crude test of hearing may be required, in others detailed analysis of the hearing may be essential. Much depends on the reasons for undertaking testing. For example, pre-employment screening may only require an indication that hearing is normal, whereas in the diagnosis for treatment purposes there may be a need for more accurate testing. The assessment of hearing for benefits other than for PD A10 may only require a rough estimate of the hearing loss. Crude tests can help corroborate the results of more refined hearing tests in the therapeutic setting and in the assessment of deafness (both for PD A10 and accidental damage).

The measurement of hearing thresholds requires a procedure, which is not too complex for the individual being tested, provides a reasonable level of accuracy and can be completed in a reasonably short period of time by a trained operator. In most individuals, pure tone audiometry (PTA) fulfils these criteria, but in a person who, for whatever reason, is uncooperative, more objective testing is required, such as evoked response audiometry.

Crude Tests of Hearing

- Free field speech testing, also referred to as the Conversational Voice (CV) test will give a rough guide to hearing loss. It requires the person's response to quiet voice, and conversational voice. (Testing by whisper is not recommended). Ideally if this method of testing is used the decibel level of the voice should be tested using a sound level meter. The person being tested should not be able to pick up visual clues, by lip-reading. This is not recommended as the only test used, but it can give an indication as to whether formal testing has given an accurate picture of the hearing loss. (See notes below)

- Tuning fork tests differentiate between a fault in conduction through the external and middle ear (conductive deafness) and a defect in sound detection by the inner ear or higher centres (sensorineural deafness). In the healthy ear, sound is heard better through air (via the ear canal and middle ear conduction) than sound introduced directly to the inner ear through bone conduction. Tuning fork testing can usually give an indication as to whether formal testing has given an accurate picture of the type of hearing loss present.

- The Rinne Test uses a vibrating 512 Hz tuning fork held close to the external auditory meatus and then on the mastoid process. The person is asked to compare the loudness of the two sounds. If air conduction is better than bone conduction, then the Rinne test is positive. This occurs in normal ears and where there is a sensorineural loss only. When bone conduction is better than air conduction, it is termed Rinne negative.

A false negative test can be obtained when there is a profound or total absence of hearing in the test ear, and the sound is heard by bone conduction through to the other, good ear.

- The Weber Test uses a vibrating tuning fork placed on the forehead. If one cochlea is under-functioning the sound appears louder in the good ear. If both cochlea are healthy, but there is a unilateral conductive loss, then the sound will be louder in the ear with the conductive loss.

Notes about the Conversational Voice Test

The following is a very rough guide to the noise level of speech:

- It is normal to hear a quiet voice at 60 cms from the ear
- Conversational voice not over 4 metres – loss approximates to 30dB in both ears
- Conversational voice not over 3 metres - loss approximates to less than 40dB in both ears
- Conversational voice not over 2 metres - loss approximates to 50 – 53 dB in both ears
- Conversational voice not over 1 metre - loss approximates to 61- 66 dB in both ears
- Conversational voice not over 30cms – loss approximates to 73 - 79 dB in both ears
- Shout from not beyond 1 metre away- loss approximates to 80dB

More Refined Tests of Hearing

Speech Audiometry – This is a skilled technique that requires both training and practice by a specialist in this field.

The aim of this test is to find out under optimum conditions how well a person can identify speech sounds, that is how well spoken words can be discriminated. The test involves presenting lists of monosyllabic words, via an earphone, at differing sound levels. For each list, a percentage score of correct replies is made and this is plotted against sound level. There are two derived figures obtained from this graph. The optimum discrimination score (ODS) is the highest score obtained on the graph. Speech reception threshold or half-peak level (HPL) is considered to be level at which the person hears correctly half of what is presented to him, which is the level given on the graph by a discrimination score of ODS/2. The difference between the HPL for a normal person and the derived HPL for the person produces a figure of half peak level elevation and acts as a guide for threshold elevation and can be used as a cross check.

There are inherent problems with speech audiometry as there is no agreed procedure, no real calibration, and is an expensive test in terms of time. Calculation of masking levels is also difficult. (See below for masking). Variations of spoken word and dialects mean that interpretation of the test has inherent difficulties.

Where a speech test shows hearing to be worse than expected from pure tone threshold the possibility of a neural cause, such as an acoustic neuroma must be considered.

Audiometry

General

There are several methods of performing audiometry, including self-recording (Bekesey), Tympanometry / Impedance Audiometry, Pure Tone Audiometry (PTA), Electric (or Evoked) Response Audiometry (ERA).

Pure Tone Audiometry

A pure tone is a single frequency stimulus that is presented to each ear through the earphones for approximately a second until the threshold of hearing, that is the minimum intensity of noise audible, is acknowledged in at least 50 % of presentations.

The conductive hearing loss is measured by Air Conduction (AC), which tests the transmission of sound via the external and middle ear to the inner ear. The test requires pure tones to be presented through the earphones to the external auditory canal.

To measure the sensitivity at the cochlea, which is the sensorineural loss, requires a stimulus bypassing the outer and middle ear and stimulating the cochlea directly. This is achieved by presenting a vibration stimulus to the skull, usually the mastoid bone behind the ear, and is referred to as bone conduction (BC). It is often necessary to use masking noise in the non-test ear to ensure that the results are purely from the ear under test. Audiometers cannot record BC hearing above 60 dB.

The response to the same frequency is tested several times. The test requires a response from the individual, usually by pressing a button when they hear the sound, so it is a subjective test. Provided the person is co-operative, PTA is a very good method. However, PTA is subjective, as the person gives a voluntary conditioned response to an acoustic stimulus. Audiometric tests should be performed by appropriately trained audiometricians experienced in the assessment of individuals exhibiting traits of non-organic or feigned hearing loss. Otherwise the “true” hearing level of a person is elevated during such conventional tests and is often undetected.

If the hearing loss is due to a sensorineural hearing impairment, and affecting the cochlea or auditory nerve, then the AC and BC thresholds should be similar.

If a blockage occurs in the external ear, such as a perforation of the tympanic membrane, or a defect within the middle ear, perhaps due to an infection, then there will be a difference between the AC and BC thresholds.

Then an Air Bone Gap (ABG) develops and the responses to BC will be better than to AC. The greater the conductive hearing loss the greater will be the ABG.

Various other factors including extrinsic and intrinsic variables affect the reliability and accuracy of this test.

- The extrinsic variables can be controlled, particularly those of the physical environment, including temperature, humidity, light, and ambient background noise. All equipment should function in accordance to relevant appropriate International Standards Procedures and be regularly calibrated to ensure continuing accuracy

- The intrinsic variables which potentially affect the measurements include; intelligence, motivation, attention, fatigue, judgement criteria, familiarity with procedure, comprehension of instructions, financial motivation, state of health, real fluctuation in hearing sensitivity etc.

The results of the PTA testing are plotted on an audiogram. This has as its reference point a 'normal' value determined by testing young healthy adults. This is a biological norm as opposed to a scientific absolute. The vertical axis is a logarithmic scale for sound level measured in decibels (dB) – where O dB is the normal sound threshold (0.00021 dynes/cm^2), 10dB has 10 times this intensity, 20 dB has 100 times the intensity. A threshold of 20 dB or better is acknowledged as within 'normal' limits by most practitioners. The horizontal axis gives the frequency of each tone tested.

The audiogram is not a mathematical graph. Hence, it cannot be assumed that a frequency, which has not been tested, falls on the line connecting two other measurements that were tested, because different cilia respond to different frequencies. Unless each frequency is tested there is no way of knowing what the hearing ability will be at any particular frequency. For example, the cilia responding to frequency Y may be damaged, but those responding to X or Z may be normal.

There is a characteristic pattern of audiogram in cases of hearing loss due to the exposure to noise. The cilia that respond to high frequency sound are more susceptible to damage than those that respond to low frequency sounds. Thus the hearing loss is worse in the higher frequencies than the lower frequencies. A 'flat' audiogram where the hearing loss is much the same across all the frequencies is not characteristic of noise induced hearing loss (NIHL).

Masking

Although headphones allow sound to be presented to each ear separately (monaurally), it should not be assumed that the test ear is the one actually responding.

When the threshold of hearing is very different between the two ears (asymmetrical loss) it is possible that when testing the worse ear, the better ear detects the signal. This is termed cross hearing or cross over and the recorded hearing levels for the worse ear may be a “shadow” of the better ear and not the true hearing level. The attenuation is referred to as transcranial transmission loss.

When the difference in thresholds is greater than the transcranial transmission loss, the involvement of the better ear must be excluded by “masking” the cochlea of the better ear. Masking is achieved by presenting a masking noise (narrow band noise) to the better ear.

Tympanometry / Impedance Audiometry

Tympanometry is an important test of middle ear function in which the compliance (mobility) of the tympanic membrane and the ossicular chain are measured. Modern automatic devices produce an indication of normality or pathology in a matter of seconds. They may be used as a screening method for the common childhood condition, otitis media, or to confirm a clinical diagnosis of middle ear abnormality.

The basis of this test is the ability of the tympanic membrane and ossicular chain to absorb energy from sound introduced into the external ear canal. This is maximal when the middle ear and external ear pressure are equal. With a discrepancy of pressure across the tympanic membrane less sound is absorbed and more reflected up the ear canal.

Electric (or Evoked) Response Audiometry (ERA)

ERA is a well-established method for the objective assessment of hearing acuity for people who are unable or unwilling to perform the subjective tests, such as PTA. The types of cases include:

- Young children and/or those with multiple handicaps suspected of hearing loss
- Where it is suspected that the PTA does not reflect the true hearing loss, when the response to CV suggests a better level of hearing indicated by the PTA
- When accurate thresholds need to be established, as in cases of litigation and in relation to claims for noise induced deafness

Therefore, the major advantage of this test for medico-legal assessment is that is it possible to produce an objective audiogram, assessing the loss at each frequency for AC and BC, and not relying on the subjective responses of the individual, as in conventional audiometric assessments.

The term ‘ERA’ is an umbrella term covering a variety of different responses, which can be recorded from various levels along the auditory pathways using appropriate stimulation and recording conditions. These responses can be measured at brainstem (brainstem ERA) or cerebral cortex level (cortical ERA or CERA).

The method chosen depends upon a variety of factors such as maturation of the brain, age and the type of information required.

For example, brain stem ERA is often used to test children, as the response is resistant to anaesthetic and sedative agents.

The most appropriate form of ERA testing in adults is the Cortical Evoked Response Audiogram (CERA) also known as the Vertex Response (SVR). For this test, short duration tones, similar to those used in PTA are used to elicit a response.

The level of auditory stimulus is reduced until no response is obtained and hence the threshold of hearing is established objectively.

Electrical potentials in response to repeated noise stimulation are recorded, and using a computer, added together sequentially on the same time basis relative to the stimulus (summated) and then averaged to give a 'smooth' record.

The ERA can therefore indicate whether an individual's hearing loss is genuine or not and if not, can give an estimate of the true auditory threshold. At present, the commonest and most reliable procedure is direct determination of response presence or absence at various intensities until the response is just detectable in the averaged record. Clinically this parallels the conventional audiometric procedure, substituting response detection of the evoked response for the individual's behavioural response.

ERA is not superior to PTA in all respects, as is sometimes suggested. Both methods have benefits. PTA is more sensitive for identifying hearing thresholds than ERA, so it remains the method of choice when assessing the threshold of hearing loss. Towards the hearing threshold the ERA signal becomes submerged in background signals and the tracing can only be read to within 20 to 30 dB of the threshold. Mathematical techniques are then used to give the definitive readings.

If the difference between objective (ERA) and subjective (PTA) techniques is less than ± 10 dB, then the subjective thresholds should be used, as the ERA test has verified the behavioural responses.

If the ERA results are better than the subjective results by more than 10 dB then there is a strong possibility of Non-organic hearing loss (NOHL) and the ERA results should be used for the assessment of hearing threshold.

The Clinical Assessment of Noise Induced Hearing Loss, in relation to Prescribed Disease A10

Introduction

In any individual case, the diagnosis of NIHL is based on a combination of history, clinical examination and pure tone audiometric findings.

In many cases because of the latent period between noise exposure and assessment, the age of the individual, the co-existence of other ear pathology, the unknown factor of socio-acusis and the varying individual susceptibility to noise damage, the diagnosis is one of probability rather than certainty. In the assessment of a person with hearing loss, it is important to consider all possible causes of the damage. It is important to consider such causes as ear infections, trauma, ototoxic drugs, genetic causes, ageing and other diseases as the cause of hearing loss.

The history must establish that the individual has been exposed to the damaging effects of noise, exceeding the established damage risk criteria.

There are techniques available for apportioning hearing loss to different episodes of noise exposure, but these depend on a full occupational and social history and may require sound exposure measurements to be taken in the workplace(s), and so for most individuals, cannot be used as the data is not available.

The following topics should be covered in the history:

Hearing Loss

- Has the individual noticed any hearing difficulty?
- Is the hearing loss present in one or both ears? If so, which ear is worse?
- When was the hearing loss first noticed?
- How did the individual become aware of the hearing loss?
- Was the onset of hearing loss gradual or sudden?
- When is the individual especially affected by the hearing loss, one to one conversations, in a group, listening to TV, on the telephone etc?
- How has it affected the individual's performance of their occupation, or access to employment?
- The presence of tinnitus (See below)

Past Medical History of Ear Disease

- Is there any history of a previous ear disease or ear surgery?
- Are any aural symptoms present, including ear discharge, vertigo, fluctuating hearing loss, aural fullness, pain in the ear?
- History of head injury
- Exposure to ototoxic drugs, e.g. aminoglycoside antibiotics

- Treatment for Tuberculosis (possible use of ototoxic drugs)
- Frequent episodes of otitis media in childhood
- Other relevant diseases
- Is the hearing loss symmetrical or worse in one ear compared to the other?

Family history of hearing loss

A familial deafness may not become apparent until adult life.

Social history

- History of recreational shooting, motorcycling, playing in a band
- Continuous wearing of “Walkman” type headphones, or listening to very loud music, etc
- Use of noisy DIY tools such as drills, grinders, mechanical hammers

Occupational History

All periods of employment to present day.

Some assessment should be made of the noise levels on a daily basis, the number of hours per day exposed to noise, whether any hearing protection or other noise abatement measures were employed, whether workers had to shout loudly to communicate.

Because the inner ear may recover during quiet periods, it may be necessary to record the noise-free intervals during an average week, taking into account social as well as industrial noise exposure.

When exposed to weapons, it is important to record the length of service in the organisation, the frequency of firing, the type of weapons discharged and the approximate number of rounds fired. When was ear protection provided, what type, and when was it used? Was it worn at all times?

Any measures implemented to reduce noise exposure.

Symptoms of temporary hearing loss and/or tinnitus.

Clinical Examination

Clinical examination of the individual should include full examination of the pinna and skin over the mastoid process to exclude scars from previous surgery.

The entire external auditory canal should be visualised, the full tympanic membrane should be visualised using an auroscope. The middle ear should be examined to establish if there is any evidence of acute or chronic middle ear disease.

The nose and throat should be examined to rule out any co-existing pathology, which could potentially affect the middle ear, such as Eustachian tube dysfunction secondary to nasal polyps, which may exacerbate the hearing loss and tinnitus.

If the tinnitus is described as pulsatile, the major vessels in the neck should be auscultated to rule out a transmitted bruit. Unilateral pulsatile tinnitus should always be appropriately investigated.

Audiological Findings

The pure tone audiometric findings must be correlated with:

- The type and duration of noise exposure
- The age of the individual
- Co-existence of other ear pathology

The classic audiometric evidence of early noise induced damage to the inner ear structures is the appearance of a notch on the audiogram between 3 kHz and 6 kHz with the maximum notch usually at 4 kHz. The hearing is relatively normal at the lower frequencies, the loss is maximum at 4 kHz, with slight improvement at 8 kHz. With continued exposure, the lower frequencies become affected so the audiogram flattens, but does not become totally horizontal. Involvement of the lower frequencies may take several decades of noise exposure.

Summary of Diagnosis of Noise Induced Hearing Loss (NIHL)

A diagnosis of NIHL does not necessarily equate to a diagnosis of PD A10. NIHL may be present but the severity is insufficient to fulfil the criteria of PD A10.

The prescription of PD A10 specifically refers to the average losses over 1, 2 and 3 kHz. Although the hearing is worse in the higher frequencies (4kHz and above), most of the speech falls within the 1,2 and 3 kHz frequencies so it is only when those frequencies are affected to a significant degree that there is any disablement from the effects of noise.

When evaluating NIHL, and in particular PD A10, the following should be considered:

- A history of noise exposure
- Absence of other pathology causing hearing loss
- Hearing loss is always sensorineural, affecting the hair cells in the inner ear
- Hearing loss is usually bilateral, but may be asymmetrical if due to asymmetric noise exposure, for example when shooting. Audiometric patterns are usually similar bilaterally
- Noise almost never produces a profound hearing loss. Usual low frequency limits are about 40 dB and high frequency limits about 75 dB
- Once the exposure to noise is discontinued, there is no significant further progression of hearing loss as a result of the noise exposure
- Previous NIHL does not make the ear more sensitive to further noise exposure. As the hearing threshold increases, the rate of loss decreases
- Given stable exposure conditions, losses at 3 kHz, 4 kHz and 6 kHz will usually reach a maximum level in about 10-15 years
- Continuous noise exposure over the years is more damaging than interrupted exposure to noise, which permits the ear to have a rest period

Occupational Deafness (PD A10) Disablement

The Binaural disablement may be read directly off the table below.

The pure tone hearing levels in the table refer to the average values of the 1, 2, 3 kHz Hearing Loss (HL), measured in dB.

1, 2 , 3 kHz average	Pure Tone HL	WORSE EAR								
Pure Tone HL	dB	50-53	54-60	61-66	67-72	73-79	80-86	87-95	96-105	106+
BETTER EAR	50-53	20	22	24	26	28	30	32	34	36
	54-60	22	30	32	34	36	38	40	42	44
	61-66	24	32	40	42	44	46	48	50	52
	67-72	26	34	42	50	52	54	56	58	60
	73-79	28	36	44	52	60	62	64	66	68
	80-86	30	38	46	54	62	70	72	74	76
	87-95	32	40	48	56	64	72	80	82	84
	96-105	34	42	50	58	66	74	82	90	92
	106	36	44	52	60	68	76	84	92	100

Tinnitus

General

It is important to note that the prevalence of tinnitus in the normal population is considerable. Estimates of tinnitus prevalence in industrialised countries are as follows:

- Approximately 35% of the population studied have experienced tinnitus
- Approximately 10% have experienced spontaneous tinnitus lasting over 5 minutes
- At least 5% experience tinnitus that interferes with getting to sleep and/or causes moderate or severe annoyance
- Only 0.5% experience tinnitus that has a severe effect on the ability to lead a normal life
- Probably the most useful statistic is that 7% of adults in the UK have at

some time seen their family doctor about tinnitus

- Tinnitus occurs in 11% of people under 40 years, in 13% of people aged between 40 and 60 years and 18% of people over 60 years of age
- Over 1/3 of adults with auditory complaints complained of tinnitus

Causes of Tinnitus

In view of the high prevalence of tinnitus in normal populations, tinnitus due to causes other than noise should be considered:

1. Physiological Tinnitus

All people experience this. It usually consists of short tonal noises that last a few seconds. An awareness of tinnitus may occur in soundproof rooms or when the level of ambient noise is low.

2. Local Causes

Tinnitus may be associated with any otological disorder and with any form of deafness. Local causes include:

- Wax
- Chronic suppurative otitis media
- Meniere's disease
- Otosclerosis
- Acoustic neuroma
- Ototoxic drugs
- Aneurysm
- Vascular intracranial tumour
- Muscle tics, affecting tensor tympani, stapedius, and muscles at opening of Eustachian tube
- Presbycusis
- Noise induced hearing loss that is the most common identifiable pathological cause

3. General causes

Tinnitus is often a feature of general ill health:

- Fever (due to any cause)
- CVS disorders, such as hypertension, atheroma
- Haematological disorders including anaemia, hyperviscosity
- Neurological disease, such as Multiple Sclerosis
- Metabolic disease for example diabetes, hypothyroidism
- Alcohol

4. Noise Induced Tinnitus (NIT)

May follow an episode of severe acoustic trauma but more commonly is the result of continuous noise exposure.

The main determinant predicting the likelihood of tinnitus appears to be the hearing threshold level.

The greater the reduction in hearing threshold level, the more likely the presence of tinnitus. NIT should be closely associated with the period of noise exposure. However, tinnitus may occur years following exposure to noise due simply to age related hearing loss.

6. Noise Induced Permanent Tinnitus (NIPT)

NIPT may occur immediately following a single episode of acoustic trauma, but noise induced hearing loss (NIHL) normally occurs before NIPT. In the majority the onset is usually insidious and many individuals cannot give a clear date of onset.

NIPT is a subjective symptom, and currently there exists no objective test that can prove its existence. Essentially, if an individual gives reliable and consistent information about health, degree of noise exposure and if consistent in repeated audiometric testing, it is reasonable to believe that person is also “honest” concerning the complaint of tinnitus.

Noise induced permanent tinnitus should start no later than within 1 year of the cessation of exposure. Tinnitus that starts more than 1 year after the removal from a noisy environment is unlikely to be related to noise.

7. Incidence of Tinnitus

In a noise-exposed population the incidence is approximately:

- 35% among those who had been exposed for up to 10 years
- 50% for those who had worked with noise for 11 and 30 years

8. Descriptions of the Tinnitus associated with NIHL

The quality of tinnitus is described as ringing or buzzing by the majority, though a minority describes whistling or pulsing. When individuals with tinnitus are asked to compare the sound/character of the tinnitus with a pure tone, a good (but not invariable) correlation is noted between the individual's hearing loss frequencies and the pitch of the tinnitus.

9. Categories of Tinnitus

Tinnitus can be categorised as slight, mild, moderate or severe on the basis of the history. A complete history using open ended, non-leading questions is essential in deciding the severity of an individual's tinnitus. Tinnitus causes fewer problems over time due to the effects of habituation.

The following should be covered in the history:

- Is tinnitus present?
- If present, how does it trouble the individual?
- Is it constant or intermittent; and if intermittent how often does it occur and how long does each bout last?
- How long has it existed?
- It is unilateral, bilateral or central?
- How severe? Is it slight, mild, moderate or severe?
- What was the relationship between the onset of tinnitus and the history of noise exposure?
- Is it noticeable in the presence of background noise, or when quiet?
- Does it interfere with sleep?
- Has the individual sought medical advice?
- Has the individual had any treatment?
- Does it interfere with normal lifestyle activities?

Classification of Tinnitus

For an individual to be assigned to a severity category, they must meet all the criteria in the said category.

Slight Tinnitus

- Noise heard in the head or either ear occurring occasionally, about once or twice per month
- Does not cause any interference with sleep or concentration and does not interfere with lifestyle.

Mild Tinnitus

- Noise may occasionally interfere with sleep or concentration
- Does not interfere with normal lifestyle activities
- Noise in the head or either ear, not heard in the presence of background noise

Moderate Tinnitus

- This is defined as intermittent or continuous sounds heard in the head or either ear occurring for a period of at least two years
- The noise is heard during waking hours in the presence of background noise and frequently interferes with the ability to get to sleep, on a monthly basis
- The individual should have documented evidence of attending a primary care physician for advice and management about tinnitus

Severe Tinnitus

- This is defined as noise localised to the head or ears present during waking hours and interfering with the ability to get to sleep
- It causes disturbance of sleep pattern and interferes with the ability to carry out normal occupational and social activities
- The tinnitus should have been constantly present in such a manner for two years
- There should be documented evidence of the claimant seeking primary physician care and referral for specialist opinion
- There must be documented evidence of the claimant requiring treatment including tinnitus maskers and medication for control and to improve concentration

Percentage Disablement Due to Tinnitus

Slight/Mild tinnitus

- 0% added as the assessment of disablement for NIHL will cover any

disablement due to tinnitus

Moderate tinnitus

- 1 - 2% additional disablement to that due to NIHL

Severe tinnitus

- 4-5% additional disablement to that due to NIHL

Appendix 3 - **Raynaud's Disease, VWF and HAVS**

The following information is from a document written by Dr S Reed and may be of interest to HCPs to revise terminology around these conditions.

Raynaud's Disease is caused by intense vasospasm of the micro-arterial supply to the fingers on exposure to cooling.

The causes of Raynaud's Disease are:

Primary Raynaud's Disease: otherwise called constitutional, or idiopathic white finger, the cause of which is unknown. It often affects other family members. It may affect the toes and nose. It affects about 5% of men and 25% of women.

Secondary Raynaud's Disease: causes of which include:

- connective tissue disorder, e.g. Scleroderma
- trauma to the extremities or to proximal blood vessels (e.g. thoracic outlet syndrome)
- occlusive vascular disease (e.g. thromboangiitis obliterans, arterial emboli)
- dysglobinaemias
- intoxication (e.g. ergot, nicotine)
- neurogenic (e.g. poliomyelitis)
- vibration (referred to as Vibration White Finger - VWF)

Thus vibration is only one of many causes of secondary Raynaud's Disease.

The onset of vibration-related Raynaud's Disease is gradual over a number of years, with attacks being rare and in winter only. In the vast majority of cases only the fingers are affected, (but it can affect the toes and, more rarely, the nose, ears, cheeks and chin),

An episode begins with tingling in the tips of the finger/s. This is followed by an intensely white, clearly demarcated area of blanching that initially affects only the distal parts of the fingers, but, as the disease progresses, moves proximally to involve the middle, then proximal phalanges. Whilst there is blanching, the fingers are numb. An episode can be triggered by quite gentle cooling, but in the early years they are more likely to occur in winter, becoming all year round as time goes on.

Blanching is circumferential affecting the front, back and sides of the fingers and always involves the nail-bed.

As the blanching wears off (after anything from 5 to 60, or more minutes), there may be significant, burning discomfort that some miners call “the hot aches”.

The colour changes of Raynaud’s Disease are characteristic. The blanching is an intense whiteness with a well defined demarcation from the unaffected skin. The blanching may last a few minutes or last up to an hour or two. Sometimes immersion in warm water speeds up recovery. On recovery the fingers may become cyanotic (i.e. turn a greyish –blue colour) or become hyperaemic (i.e. very red in colour). Blanching is not just a paleness, it is an intense whitening. In severe cases, with the passage of time, there may be trophic changes leading to gangrene of the tips of the fingers. However trophic changes rarely, if ever, occur in vibration- induced Raynaud’s Disease. Thus if there are trophic changes it is probable that the cause is **not** vibration. Trophic indicates tissue death. The tissue death may occur due to interrupted blood flow (e.g. in gangrene) or/and interrupted nerve supply so the person in unaware of damage having occurred (as in frost bite). This degree of damage is associated with the connective tissue diseases.

As the symptoms are intermittent, and only occur in colder conditions, it is rare for the HCP to witness an attack. Thus, in a specific case, it is important to obtain an accurate description of the symptoms and their evolution both in an individual episode and through time, to identify the cause of the symptoms and to establish an accurate diagnosis to ensure that more serious causes of the symptoms (e.g. diabetes) are not overlooked. This history should also make it clear whether the claimant has actually experienced the condition, or simply read about it.

The HCP should take a detailed clinical history from the person to diagnose any medical condition present and to establish the cause, including causes of Raynaud’s Disease (see above).

Note: The history is the individual’s account of his/her symptoms, the progression of the symptoms over time, how the symptoms affect them etc, and its interpretation requires disability analysis skills.

Experts agree that the ‘gold standard’ for diagnosing Raynaud’s Disease is the clinical history that the person gives. Although there are many tests that may aid the diagnosis none are reliable, accurate or objective (i.e. do not rely on person cooperation), and thus are not recommended for use when considering vascular damage. If performed no reliance should be placed on the results of these tests if they do not support the information obtained in the history. Tests for vascular symptoms **are not** recommended for use in the Industrial Injuries Scheme.

As it is rare for the HCP to witness an attack of blanching, the diagnosis and causes of Raynaud’s Disease is based primarily on the history of the symptoms as given by the individual. It is therefore vitally important that the HCP is satisfied that the claimant’s account is accurate. Particular care needs to be taken in taking a detailed history in order to establish whether the cause of the symptoms is primary Raynaud’s Disease or one of the other causes of secondary Raynaud’s Disease or one of the other many causes of sensorineural problems.

Vibration White Finger (VWF)

Vibration White Finger (VWF) is one example of a secondary form of Raynaud's Disease. Primary Raynaud's Disease is common in the community. Hence it does not automatically follow that the exposure to vibration **caused** the problem. However having Primary Raynaud's Disease does not preclude also having PD A11. Indeed it probably makes it more likely to arise if there is exposure to vibrating tools.

Careful consideration has to be given as to the occupational causation. There must have been considerable exposure to hand transmitted vibration, but extent and magnitude together with individual susceptibility are factors in the development of VWF. The damaging effects of the many different types of vibrating tools and the different individual susceptibilities are so variable and complex that a very detailed occupational history doesn't add much value to the benefit assessment. Detailed occupational histories are essential to epidemiological studies. For IIB purposes, Raynaud's Disease can be caused by just a few years regular exposure to the wrong tool in a susceptible individual.

Sensori-neural effects of vibration

It is now recognised that vibration can affect the small sensory nerves of the fingers exposed to hand-held vibrating tools. The symptoms (tingling and numbness) arising from this damage usually pre-dates the onset of vascular symptoms, and may occur in the absence of vascular symptoms.

The symptoms are not unique to vibration damage and are a feature of many other medical conditions (e.g. diabetic peripheral neuropathy; carpal tunnel syndrome, cervical radiculopathy).

Prescribed disease A11 (PD A11) – vascular component

Early in the development of the VWF and HAVS there is little if any functional disability. Hence IIAC have recommended that only when the condition has progressed to a certain level should it be considered to be a prescribed disease.

The diagnosis of the medical condition (HAVS and/or Raynaud's Disease) does not equate to the diagnosis of the Prescribed disease (PD A11) as the symptoms and signs may not be of a sufficient severity or extent to fulfil the strict qualifying criteria required for the diagnosis of PD A11 and the cause may not be vibration.

The prescription of PD A11 includes reference to blanching as an indicator of when the condition has progressed to a stage where there is a possibility that there is some loss of function. Secondary Raynaud's Disease due to vibration begins as a minor, intermittent, non-disabling problem with the tips of the fingers, which over time and continuing exposure to vibration may progress to involve more of the fingers that just the tips. Only when it has progressed to the level listed in the Regulations would we expect any loss of normal function.

The prescription does not include all the possible symptoms, but in order to diagnose the condition and exclude other possible conditions, the HCP must consider all the symptoms and signs.

Prescribed disease A11 (PD A11) – sensorineural component

The prescription of the sensorineural component of PD A11 has been set to reflect a degree of severity where one would expect there to be a disability (This is defined as the continual presence of sensory symptoms and a continuous loss of grip and dexterity). Lesser severity of symptoms does not result in disability.

Hand Arm Vibration Syndrome (HAVS).

HAVS is a much broader condition than Raynaud's Disease and VWF. It includes elements of Raynaud's Disease, i.e. blanching, but also includes symptoms attributable to nerve damage, and musculoskeletal problems in the upper limbs. It is important to note that HAVS is not a Prescribed Disease.

There is no universal description of HAVS, so different authors attribute different entities to HAVS. For example:

Some authors include Carpal Tunnel Syndrome as an element of HAVS, whereas others do not (in Industrial Injuries Benefit carpal tunnel syndrome is a separate prescribed disease - Prescribed Disease A12), other authors include musculoskeletal problems affecting the upper limb.

In its recent report IIAC avoid the concept of HAVS for Industrial Injuries purposes. They limited the PDs to A11, requiring either continuous sensory and grip/dexterity disability (2007 rules) and/or sufficiently extensive and perennial blanching (1984 rules). A12 (1994) is a more specific and intermittent neuropathy associated with vibrating tool use and certain defined heavy uses of the hands and wrists.

The DTI compensation scheme (now closed) defined HAVS as 'exposure to vibration causing peripheral circulatory disturbances that manifest as vibration induced white finger, peripheral neurological disturbances such as numbness, tingling and paraesthesia and musculo- skeletal disturbances in the hand and the arm'.

Consequently, to avoid confusion the term Hand-arm vibration Syndrome (HAVS) is to be avoided when referring to and giving advice in relation to Prescribed Diseases A11 or A12.

The terms vibration white finger (VWF), Hand arm vibration syndrome (HAVS) and Prescribed disease A11 (PD A11) are **not** interchangeable.

- VWF is a secondary form of Raynaud's Disease where there is intermittent intense whitening (referred to as Blanching) of the fingers due to vibration damage to the micro-blood supply to the fingers. A clinical diagnosis of VWF can be made on the basis of only minimal symptoms, for example blanching of the tip of one finger in winter months only.

- HAVS is a syndrome which includes blanching of the fingers, peripheral nerve damage (referred to as sensorineural problems), and musculoskeletal problems in the hands and arms. Not all elements are required for the diagnosis of HAVS to be made, for example, some people would diagnose HAVS in a person with tingling of the fingers; or a person with blanching of the tip of one finger. Nor is there one, universal definition of HAVS. For example some authors include carpal tunnel syndrome whilst others do not.

- The vascular component of PD A11 is only diagnosed when the symptoms of VWF have progressed to a level of severity listed in the legislation. PD A11 can only be diagnosed when symptoms are present throughout the year and affect the middle or proximal phalanges of at least three fingers of a hand (or less when one or more of the fingers is missing). The criteria for the diagnosis of PD A11 are indicative of the disease having progressed to a level of severity which is much more severe than is required to clinically diagnose vibration white finger or the vascular component of HAVS.

Whilst the laboratory tests may help to confirm the presence of nerve damage they are of no assistance in the assessment of disablement for Industrial Injuries Benefit purposes as it is a functional assessment of what the person can or cannot do in comparison to a person of the same age and sex whose physical and mental condition is normal. Thus the assessment is not of the medical condition diagnosed, but the effects the medical condition has on the person. The same medical condition can have very different effects in different people. For example different people have different sensitivity to pain; some people may have another medical condition which interacts with the condition under consideration thereby worsening it etc. Thus whilst these tests may assist in confirming the severity of the nerve damage they:

- Give no indication of the cause of the nerve damage

- Do not assist in the assessment of the effects. In order to assess function the HCP needs specialist training in disability assessment medicine

In conclusion:

- A clinical diagnosis of Raynaud's Disease does not equate to a diagnosis of VWF or PD A11

- A diagnosis of HAVS does not equate to a diagnosis of VWF

- A clinical diagnosis of VWF does not equate to a diagnosis of PD A11 as only limited blanching is needed for a clinical diagnosis of VWF, whereas PD A11 needs a defined (prescribed) level of progression of symptoms

- A diagnosis of HAVS does not equate to diagnosis of A11

Appendix 4 - Background Information on Thermal Aesthesiometry & Vibrotactile Testing

This information in this section of the training should allow the HCP to understand the basic principles of thermal aesthesiometry and vibrotactile testing but is not intended to explain the process in great technical detail. In the company, HCPs will be specifically trained in the operation of these machines and will be responsible for the testing and ensuring the tests are conducted in the appropriate manner. This training will cover the interpretation of the results as the HCP will be expected to review the output from the testing to provide advice on the diagnosis of PD A11.

For both Thermal Aesthesiometry and Vibrotactile testing, the test environment must be controlled. The ambient temperature of the room must be between 20-30º C. The finger temperature must also be measured prior to both tests and be in the range of 27-37ºC.

The fingers to be tested are as follows:

Thermal and vibrotactile thresholds should be measured on two fingers of both hands.

The fingers to be test on each hand are selected as follows:

- The index and little finger if the claimant has complained of symptoms in these fingers
- If either the index finger and/or the little finger are without symptoms, substitute one or two fingers in which the claimant has complained of symptoms
- If there are symptoms in only one finger, test that finger and the index finger
- If there are symptoms only on the index finger, test that finger and the little finger

Thermal Aesthesiometry

"The thermal aesthesiometer provides measurement of tactile perception thresholds for thermal stimuli (i.e. the minimum change in temperature which can be perceived)".[1] This may be expressed in simpler terms as measuring the threshold of feeling hot or cold.

Testing can be performed to determine warm and cool thresholds. During testing, the Thermal Aesthesiometer is controlled by a personal computer. The person places a finger on a contact plate which is heated via thermocouples and the temperature of the contact plate is varied through the programming software of the P.C.

The person being tested will press a response button when they detect a change in temperature.

The machine is programmed to repeat a series of temperature changes a pre-set number of times to establish threshold levels for perception of warmth and cool sensation.

A mean threshold for hot and cold perception is then calculated. These results will be assessed in the context of agreed protocols devised by Southampton University and the DWP.

A sample of the printout of results from this testing is provided overleaf. You will have to interpret these results within the context of the other information from the assessment.

The operator of the testing equipment will be responsible for setting the parameters and conducting the test appropriately. The software records and produces a printout of the mean values of the testing.

[1] HV Lab. University of Southampton Thermal Aesthesiometer, Instructions for use

HVLaB Thermotactile Threshold Test

PERSONAL DETAILS

Surname: Date of Birth:

Forenames: Reference Number:

TEST RESULTS

Test Date: Room Temperature: 21.8°C

Tester: Finger skin temp. (left) 32.9°C

Finger skin temp. (right) 31.8°C

Measurements selected for diagnostic comparison	Measurement Site	Test	Abnormality	Threshold °C	Standard deviation °C	Criteria for abnormality (°C)
yes	Left index	Hot	o	44.14	0.55	>48.5
yes	Left index	Cold	o	25.96	0.66	<19
yes	Left little	Hot	o	40.58	0.65	>48.5
yes	Left little	Cold	o	25.29	1.29	<19
yes	Right index	Hot	o	40.38	0.84	>48.5
yes	Right index	Cold	o	26.92	1.28	<19
yes	Right little	Hot	o	45.59	2.26	>48.5
yes	Right little	Cold	xx	18.09	2.03	<19

O = No abnormality found

xx= Abnormality

NOTES

TEST PARAMETERS

Number of measurements: 4 Temperature increment: 1.0° C

Test order: Hot first Temperature decrement: 1.0°C

Reference temperature: 32.5° C Number of reversals: 6

Vibrotactile Threshold testing

"The Vibrotactile Perception Meter is intended for measuring vibrotactile perception thresholds (i.e. the vibration magnitude at which a vibration stimulus can be just perceived). It may be used for the assessment of sensory changes associated with neurological dysfunction".[2]

The testing is supervised by a trained operator (HCP) and they are responsible for setting the appropriate test parameters.

The claimant is instructed to place the fleshy part of their fingertip over the centre of a vibrating probe at a constant push force of 2N. (There is a gauge on the machine to let them know what force they are exerting). There should be no exposure to vibration from tools or other machines in a 1-hour period prior to the measurement of vibrotactile thresholds.

Vibration is assessed at a low frequency setting and a higher frequency setting. The vibration amplitude is increased slowly and the claimant is instructed to depress the response button as soon as they are able to feel the vibration. The vibration amplitude is then decreased and the subject releases the button when they can no longer feel the vibration. This process is repeated several times and the results recorded by the P.C. linked to the machine.

The software processes the results and produces a printed report with the mean values achieved in the testing.

A sample of the output is shown overleaf. You will be expected to interpret these results in context with all other findings at the assessment.

[2] HV Lab University of Southampton. Vibrotactile Perception Meter. Instructions for use

HVLaB Vibrotactile Threshold Test

PERSONAL DETAILS

Surname: Date of Birth:

Forenames: Reference Number:

TEST RESULTS

Test Date: Room Temperature: 21.8°C

Tester: Finger skin temp. (left) 32.9°C

Finger skin temp. (right) 31.8°C

Measurements selected for diagnostic comparison	Measurement Site	Frequency (Hz)	Abnormality	Threshold (m/s^2)	Standard deviation (m/s^2)	Criteria for Abnormality (m/s^2)
yes	Left index	31.5	xx	0.41	0.08	>0.4
yes	Left index	125	o	0.54	0.07	>1.0
yes	Left little	31.5	o	0.26	0.02	>0.4
yes	Left little	125	xx	1.26	0.25	>1.0
yes	Right index	31.5	o	0.27	0.05	>0.4
yes	Right index	125	o	0.55	0.09	>1.0
yes	Right little	31.5	o	0.39	0.09	>0.4
yes	Right little	125	xx	1.72	0.38	>1.0

O = No abnormality found

xx= Abnormality

NOTES

TEST PARAMETERS

Number of measurements: 8 Initial amplification rate: 5.0dB/sec

Measurement Duration: 45 sec. Final amplification rate A: 3.0dB/sec

Number of reversals: 6 Final amplification rate B: 3.0dB/sec

Appendix 5 - Upper Limb Examination Tests

Adson's test

The person is seated and the head extended and rotated to the affected side with their arm hanging by their side and abducted to 45 degrees. The person is asked to take a deep breath. A reduced or absent radial pulse on the affected side, may suggest thoracic outlet obstruction.

Finkelstein's Test

A positive Finkelstein's test may be found in De Quervain's tenosynovitis.

The person is asked to flex the thumb across the palm. The wrist should be flexed and gently moved into ulnar deviation. If positive, pain is felt on the radial aspect of the wrist over the tendons.

Tinel's Sign

A positive Tinel's sign may be found in carpal tunnel syndrome. The median nerve is tapped just proximal to the distal radius. If positive, tingling is felt in the appropriate dermatomal distribution.

Phalen's test

A positive Phalen's test may be found in carpal tunnel syndrome. This test is performed with the elbow resting on the desk and the forearm vertical. The wrist is then flexed. The test is considered positive if tingling or numbness is felt in the appropriate dermatomal distribution within 60 seconds.

Modified Allen's Test

The individual is asked to clench his/her fist for 30 seconds while the hand is elevated above the level of the heart. Pressure is applied over the ulnar and radial arteries to occlude both of them. The individual is asked to open the hand while this is still elevated and the hand should be blanched/pale. The ulnar pressure should be released and colour should normally return in 5 – 15 seconds. Any prolonged blanching/pallor would suggest an abnormality in the circulation of the hands.

Appendix 6 - **The Stockholm Scales**

This material has been extracted from a document written by Dr S Reed.

1. The Stockholm Scale is a clinical grading of HAVS which was proposed in the 1980s by an international meeting of experts.

2. It consists of two scales - one for vascular and one for sensorineural symptoms.

3. These scales are used widely in epidemiological surveys, and clinical and medico-legal practice, although they do contain some ambiguities:

Expressions such as “occasional attacks”, “frequent attacks” and “most fingers” in the vascular scale and terms “intermittent” and “persistent” in the sensorineural scale, have not been strictly defined and are open to different interpretations.

The positioning on the Scales is based on the individual’s history of symptoms, and hence is subjective. Appropriate tests and reference standards for confirming the grading; the reduced sensory perception; and the reduced tactile discrimination in the sensory scale are not defined.

4. The Stockholm Scales can be a useful basis for grading the frequency, and extent of the symptoms of HAVS, but the positioning on the scales does not correlate with the functional disability the condition cause and are not useful in determining the % disablement for IIB purposes.

5. Thus the Stockholm Scale may be a useful tool in considering whether the symptoms have progressed to a stage where PD A11 could be diagnosed and assessed. **But note the Stockholm Scale has limitations in its usefulness:**

- The Stockholm Scale is based on self-reported symptoms
- There is **no direct correlation** between the positioning on the Scale and disability
- There are limitations to the Scale as there is no definition of the terms used

6. As a rough guide:

Vascular component Stages 0 – 2 inclusive on the Stockholm scale would not be sufficient to diagnose PD A11.

Vascular component symptoms fall between Stages 2 and 3 - PD A11 would be diagnosed if attacks occurred throughout the year. The functional limitations are likely to be of a very minor nature.

Vascular component Stage 4 PD A11 would be diagnosed and the assessment of disablement should reflect the tissue loss as per the

Scheduled assessments. However, the current consensus of medical opinion is that trophic changes are unlikely in vibration induced damage. Hence if trophic changes are present the HCP should re-consider his/her opinion on causation.

Sensorineural component Stage 1SN and Stage 2SN are unlikely to result in any functional impairment.

IIAC considered that only when the sensorineural component equated to Stage 3SN persistent, i.e. continuously present, would there be any functional impairment.

The Stockholm Scales

Vascular Component

Stage	Grade	Description
0		No attacks
1	Mild	Occasional attacks affecting only the Tips of one or more fingers.
2	Moderate	Occasional attacks affecting distal and middle (rarely also proximal) phalanges of one or more fingers
3	Severe	Frequent attacks affecting all phalanges of most fingers.
4	Very Severe	As in stage 3, with trophic changes in the fingertips.

Neurological component

Stage	Description
0SN	Vibration-exposed but no symptoms
1SN	Intermittent numbness with or without tingling
2SN	Intermittent or persistent numbness, reduced sensory perception
3SN	Intermittent or persistent numbness, reduced tactile discrimination and/or reduced tactile discrimination and/or manipulative dexterity

Appendix 7 - **Policy on Passive Smoking**

The HCP should take note of the following guidance:

Due to potential health concerns associated when Healthcare Professional (HCPs) undertake domiciliary medical assessments within a passive smoking environment, guidance from the DWP policy on passive smoking is as follows:

“When visiting claimants:

- Employees can politely request that they don’t smoke during the interview
- Should they refuse the employee may terminate the interview
- Should the claimant still need to be interviewed attempts should be made to re schedule the visit or as a last resort they will be required to attend a DWP office

It is good practice to advise the claimant of this request prior to the visit”.

If the claimant refuses to stop smoking during the medical assessment, the HCP can terminate the medical assessment. The claimant should then be offered an appointment to attend an Assessment Centre (AC) (even if they fall under the category that would normally not be required to attend an AC). It can then be shown that you have offered them an alternative. If they refuse or can't attend because of their health, they should be offered another home visit, but warned that this will only take place if the claimant agrees not to smoke during the visit. If at the next home visit, the claimant again refuses to stop smoking, the HCP should terminate the visit

It should be noted that it is a personal choice of the HCP whether to proceed with the medical assessment if this is to take place in a smoke filled environment.

Appendix 8 - Faxing of Information and Dealing with Unexpected Findings

Introduction

As part of DWP Security Accreditation*, The Centre for Health and Disability Assessments needs to ensure that any process where faxing documents, containing claimant data, is required, that a secure fax solution is followed.

**Security Accreditation is about putting increased security measures in to ensure our claimant's data is better protected.*

This guidance has been written to explain that faxing personal data can continue, however this is to be made a more secure process.

Also within this guidance is a revised process for the following:

- Unexpected Findings
- Missing Information
- AC3s

PROCESS ON FAXING DOCUMENTS

NOTE: Also see revised process for Unexpected Findings, Missing Information and AC3 below '*Guides Affected'* within this guidance.

Sending a Fax

The following process must be followed each time a document, containing multiple claimant data items is faxed:

- **Telephone the recipient to inform them that you are sending them a fax**
- **Confirm the recipient's fax number**
- **Ask the recipient to ensure they wait by the fax machine to receive it**
- **Dial the number carefully**

You will also ask the recipient to call you to confirm receipt, **or**, you can call them to confirm receipt. Whichever way, **receipt must be confirmed verbally**.

Receiving a Fax

The following process must be followed when receiving a fax, containing multiple claimant data items:

- **You will receive a telephone call from the sender of the fax**

- **Confirm the fax number with the sender**
- **You must wait by the fax machine to receive the fax**
- **Call the sender to confirm receipt**

NOTE: All fax/cover sheets **MUST** clearly state the Receiver and Sender's Name/Section **and** Fax Number.

Fax received without Prior Notification

Where potential recipients are not informed prior to a fax, containing claimant data, being sent, **it is a breach of claimant confidentiality.**

If you receive a fax from the DWP, containing claimant data, without being informed beforehand, you must bring this to the attention of the Service Delivery Lead. The Service Delivery Lead should then investigate further and contact the sender, reminding them of the correct process.

If the problem persists, this should be escalated to their local Relevant Manager, who will raise the issue with the appropriate DWP Lead / Relevant Manager.

If Fax is sent to Wrong Fax Number

If the sender realises they have sent the fax to the wrong number, they must inform the receiver as soon as possible. Where it is possible to retrieve the fax, you should do so.

Where a receiver recognises that the fax has been sent to the wrong fax machine, they should inform the sender and ensure they have the correct number for future reference. Where possible, the fax should be retrieved.

Where either of these occurs, a security incident should be raised - refer to the Centre for Health and Disability Assessments Security Incident Reporting Procedures (MED-AHSIRP01) for further information.

Veterans UK

When there is a requirement to fax a request/any information to/from the Veterans UK, **any reference to regiments must be removed.** If there is a need to urgently fax previous medical documents or service medical documents that the Centre for Health and Disability Assessments may require, approval to fax must be obtained from the Service Delivery Lead or by Veterans UK, this is to ensure that the data it not put at any risk on the public network.

Faxing must only be used as a last resort and not as a daily medium to transfer personal information. If faxing is to be used, the volume of personal information must be kept to the minimum.

The above process on *Sending a Fax* and *Receiving a Fax* must be followed.

Due to revisions to the Ministry of Defence (MoD) security procedures there is a need to change the usage of faxing when processing Veterans UK (VUK) information.

Previously as part of Security Accreditation VUK information could be faxed over providing approval was sought from VUK or from a Service Delivery Lead and any reference to regiments was removed. Whilst these principles should still be followed there are some specific changes to certain VUK referrals.

Medicals Direct Group (MDG)

Confirmation of receipt of the Exchange Template will be made by a telephone call to confirm all the cases sent are received. MDG will follow this by returning a signed copy in the post.

In arranging appointments MDG will make contact with the Specialist required via telephone initially and follow up with a redacted fax containing no claimant information confirming the request.

Confirmation of appointments made by MDG will be given to The Centre for Health and Disability Assessments via telephone call. Details will then be confirmed by post.

Protected Population Referrals

Under no circumstances must any information relating to Protected Population referrals be faxed. All information should double-bagged and transferred via the TNT fully tracked 'track and trace' courier service.

Unexpected Findings Process

Situations arise when HCPs carrying out disability assessments may come across information that they feel should be reported to the claimant's General Practitioner. Detailed guidance on dealing with unexpected findings is given in Appendix C of the Industrial Injuries Benefit Handbook 1.

GMC Guidelines have made it clear that Registered Medical Practitioners who have contractual obligations to third parties should not pass on information to the claimant's GP without claimant consent for such action, unless there were exceptional circumstances. The GMC recommend that practitioners make every effort to explain to individuals why information should be passed on to those responsible for their medical care.

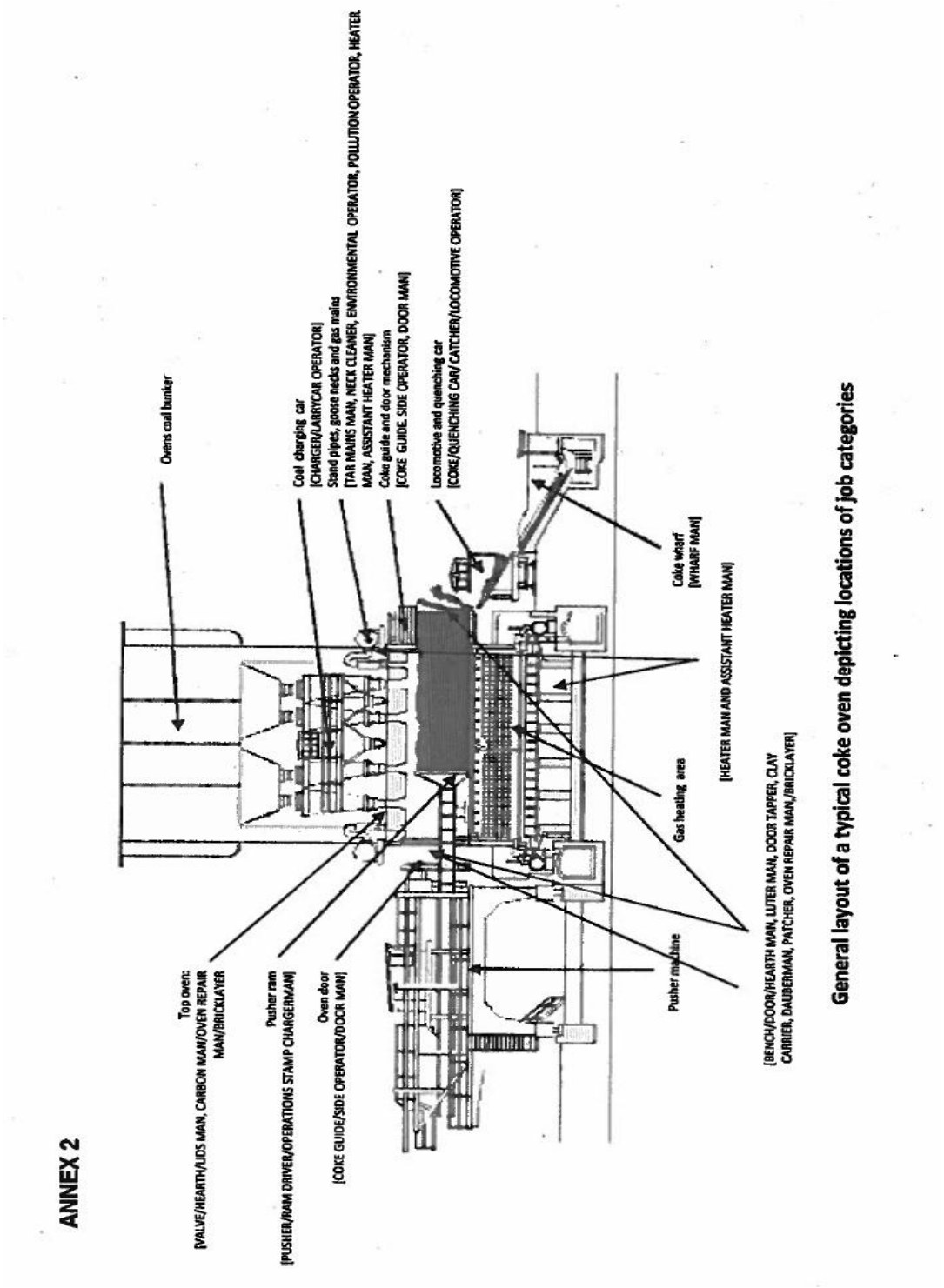
There may be rare occasions when despite the claimant's inability or refusal to give informed consent, the practitioner may, in his/her professional judgement pass on information about that individual.

This discretion must be exercised within the GMC guidelines, and practitioners must be prepared to justify their decision to take such action. The types of circumstances when unauthorised disclosure by practitioners would be justified include:

- When the release of that information is necessary to protect others from risk of death or serious harm;
- Or when the claimant requires urgent medical treatment, but cannot be contacted within a suitably rapid period of time.
- Or when the individual is not competent to give consent.

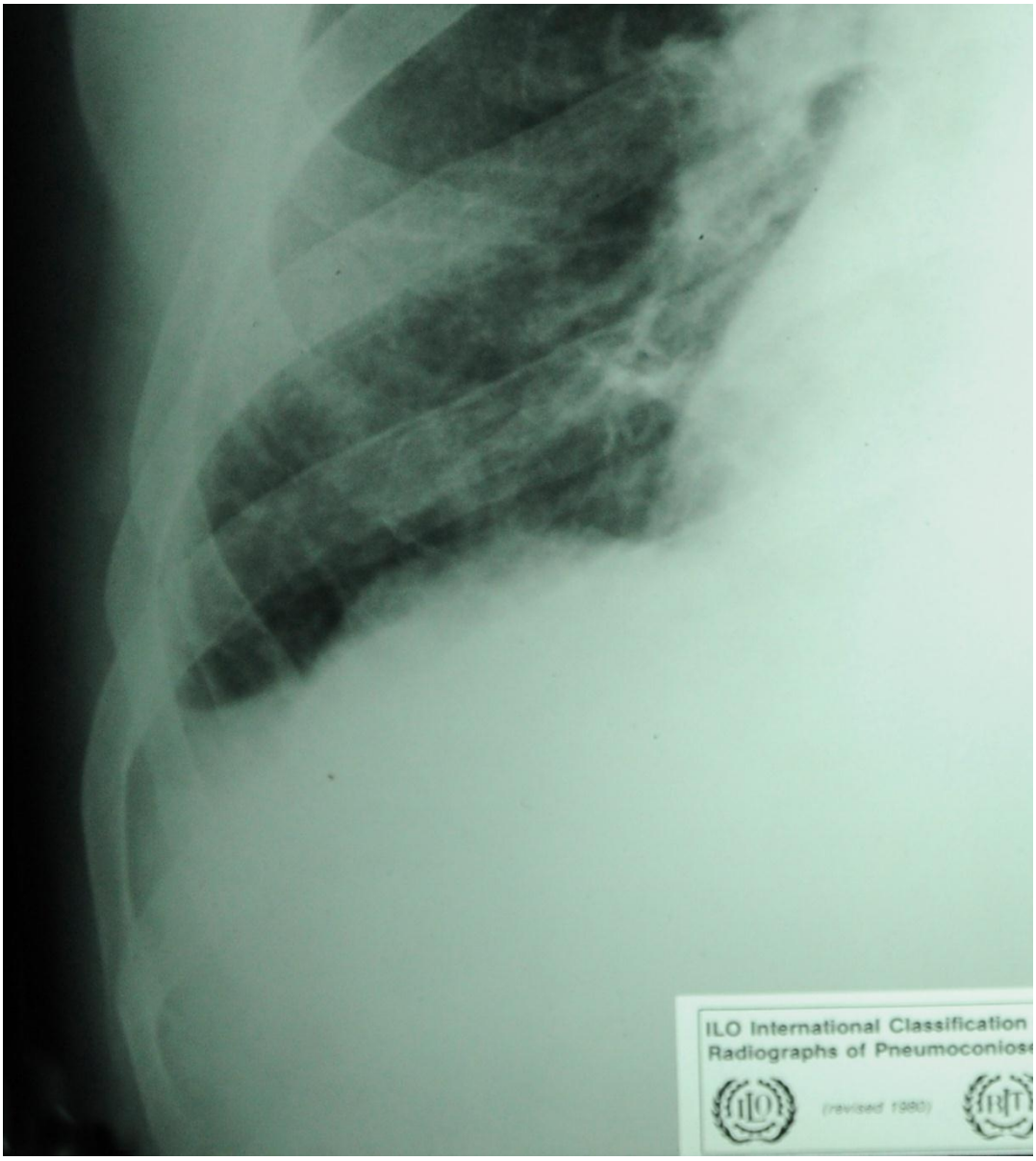
All practitioners are strongly advised to read these guidance notes from the GMC. If any practitioner does not have a copy then s/he should contact the GMC at 178 Great Portland St, London W1 W5JE (tel: 0845 357 8001).

Appendix 9 **- Diagram of a Coke Oven**



Appendix 10 - PD D9 X-ray changes

Illustration of the minimum extent of 'obliteration' and pleural thickening extending up lateral chest wall. (Note: this is for illustration purposes only and not comparison purposes). Detailed information on PD D9 is available in the Respiratory Prescribed Diseases Handbook.



Appendix 11 – List of Prescribed Diseases which require assessment by RD-trained HCP

This is the full list of Prescribed Diseases that require assessment by a Respiratory Prescribed Disease trained Healthcare Professional.

B5	Tuberculosis
B6	Extrinsic allergic alveolitis (including farmer's lung)
C4	Primary carcinoma of the bronchus or lung
C17	Chronic beryllium disease
C18	Emphysema
C22B	Primary carcinoma of the bronchus or lung
C31	Bronchiolitis Obliterans
D1	Pneumoconiosis
D2	Byssinosis
D3	Diffuse mesothelioma (primary neoplasm of the mesothelium of the pleura or of the pericardium or of the peritoneum)
D4	Allergic Rhinitis (due to specific occupational agent exposure)
D7	Asthma (due to specific occupational agent exposure)
D8	Primary carcinoma of the lung where there is accompanying evidence of asbestosis
D8A	Primary carcinoma of the lung
D9	Unilateral or bilateral diffuse pleural thickening with obliteration of the costophrenic angle
D10	Primary carcinoma of the lung
D11	Primary carcinoma of the lung where there is accompanying evidence of silicosis
D12	Chronic obstructive pulmonary disease

Appendix 12 - Changes to Presumption rule in IIB

Prescribed disease	Changes to prescribed disease
A1 Leukaemia, bone, breast, testicular and thyroid cancer	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted in the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.
A2 Cataract	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. The effect of this change will be to allow diagnosis of A2 in people who left the industry many years ago. The medical literature shows that doubling of risk for cataract after exposure to heat e.g. in glass blowing persists for many years. It is thought there may be some claims where diagnosis was refused because of the length of time between the end of employment in the industry and diagnosis of the disease. In claimants with 5 years of exposure doubling of risk of cataract can be considered to persist for life.
A3 Dysbarism	A3 has been split into A3a dysbarism and A3b osteonecrosis. The time rule for A3b osteonecrosis has been extended so that presumption applies any time after leaving the job as well as in the job. It is possible that cases of osteonecrosis have been refused because of the length of time between relevant employment and the diagnosis of osteonecrosis. The risk of osteonecrosis persists in the long term. The expert view is that osteonecrosis is a disease of professional divers and is not seen in recreational divers.
A4 Task-specific focal dystonia	The rules for this disease have not changed.
A5 Subcutaneous cellulitis of the hand	The rules for this disease have not changed.
A6 Bursitis of the knee	The rules for this disease have not changed.
A7 Bursitis of the elbow	The rules for this disease have not changed.
A8 Tendonitis of the hand or forearm	The rules for this disease have not changed.
A10 Hearing loss	The rules for this disease have not changed.

A11 Blanching	The rules for this disease have not changed.
A12 Carpal tunnel syndrome	The rule has not changed for people making a claim under (a), the use of hand held powered tools whose internal parts vibrate. The rule has changed for people making a claim under (b), repeated palmar flexion and dorsiflexion for at least 20 hours per week. The standard presumption rule now applies but the new rule reflects how claims have been managed under (b) since it was introduced.
A13 Osteoarthritis of the hip	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. Decisions about this disease are already made as if the new longer time rule applies.
A14 Osteoarthritis of the knee	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. Decisions about this disease are already made as if the new longer time rule applies.
B1 Anthrax	B1 has been split into a) cutaneous anthrax and (b) pulmonary anthrax. This disease usually takes the form of a skin infection (cutaneous anthrax), but very rarely it affects the lungs and other organs (pulmonary anthrax). The time rule for the commoner skin disease has not changed. The time rule for pulmonary anthrax has been extended to 2 months. Decisions about this disease are already made as if the new longer time rule applies.
B2 Glanders	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. This disease is no longer endemic in the UK; contact Clinical Project Lead/Clinical Policy and Projects Lead for latest advice.
B3 Leptospirosis	The rules for this disease have not changed.
B4 Ankylostomiasis	This disease has been split into (a) cutaneous larva migrans and (b) iron deficiency anaemia caused by gastrointestinal infection by hookworm. This disease usually takes the form of a skin infection, but rarely it affects the gut and causes blood loss (anaemia caused by hookworm). The time rule for the commoner skin disease (B4a) has not changed. The time rule for anaemia due to hookworm infestation (B4b) has been extended to 12 months. Decisions about this disease are already made as if the new longer time rule applies.
B5 Tuberculosis	The presumption rule remains the same for all types of workers working in hospitals, laboratories and mortuaries where post mortems are carried out. All other workers including healthcare workers who do not work in hospitals and mortuaries will no longer have the benefit of presumption. This reflects the evidence base which shows doubling of risk of TB in workers in hospitals, laboratories and mortuaries but little or no excess risk in community based health care work including general practice. Healthcare Professionals should give individual advice based on the circumstances of the exposure to TB.
B6 Extrinsic allergic alveolitis	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. Healthcare Professionals should give individual advice on causation based on the circumstances of the exposure at work.

B7 Brucellosis	The time rule for presumption has been changed to reflect the incubation period of this disease. Decisions about this disease have always been made based on the incubation period rather than the presumption rule. Brucellosis has been eradicated from the UK since 1979. Sporadic cases have occurred here since as a result of infected cattle imports.
B8 Infectious hepatitis	B8A hepatitis A the time rule for presumption has been increased to 2 months to reflect the incubation period of this disease. Decisions about this disease have always been made based on the incubation period rather than the presumption rule. B8B hepatitis B or hepatitis C the time rule has been extended so that presumption applies any time after leaving the job as well as in the job. It is possible that a claim was turned down due to length of time between the employment and the diagnosis of the disease so there may be new claims or change of circumstances cases for this disease. Healthcare Professionals should give individual advice on causation based on the medical evidence.
B9 Streptococcus suis	The rules for this disease have not changed.
B10 Avian and ovine chlamydiosis	The rules for this disease have not changed.
B11 Q fever	The rules for this disease have not changed.
B12 Orf	The rules for this disease have not changed.
B13 Hydatidosis	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. It is possible a claim was refused due to the length of time between the end of employment with dogs and diagnosis of the disease. Hydatidosis may not be diagnosed until many years later.
B14 Lyme disease	The rules for this disease have not changed.
B15 Anaphylaxis	The rules for this disease have not changed.
C1 Effects of lead	The rules for this disease have not changed.
C2 Effects of manganese	The rules for this disease have not changed.
C3 Effects of phosphorus	The rules for this disease have not changed.
C4 Effects of arsenic	The rules for this disease have not changed.
C5 Effects of mercury	The rules for this disease have not changed.

C6 Effects of carbon bisulphide	The rules for this disease have not changed.
C7 Effects of benzene	The rules for this disease have not changed.
C12 Effects of methyl bromide	The rules for this disease have not changed.
C13 Effects of chlorinated napthalenes	The rules for this disease have not changed.
C16 Effects of gonioma kamassi	The rules for this disease have not changed.
C17 Berylliosis	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. This disease has always been assessed as work related even if the employment was many years ago.
C18 Effects of cadmium fume	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. This disease is always assessed as work related if the exposure conditions are met.
C19 Effects of acrylamide	The rules for this disease have not changed.
C20 Effects of quinone	The rules for this disease have not changed.
C21 Skin cancer	The rules for this disease have not changed.
C22 Cancers of the nose and lung	Presumption will now be applied to claims for cancer of the nose and paranasal sinuses under C22a. This is because relative risk for this disease is very high even if employment was short. Employment of 1 year or more could be expected to double the risk even though the last exposure was in the 1950s. (expert opinion) The rules for C22b lung cancer have not changed, presumption does not apply.
C23 Bladder cancer	The time rule for presumption under C23 a, b and e has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted by the scheme that causation is due to work no matter how long ago the worker left the qualifying employment. The rules for C23 c and d have not changed.
C24 Effects of vinyl chloride monomer	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted by the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.

C25 Vitiligo	The rules for this disease have not changed.
C26 Effects of carbon tetrachloride	The rules for this disease have not changed.
C27 Effects of chloroform	The rules for this disease have not changed.
C29 Peripheral neuropathy	The rules for this disease have not changed.
C30 Chromate dermatitis	The rules for this disease have not changed.
C31 Bronchiolitis obliterans	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. This disease has always been assessed as work related if the diagnosis is made and the exposure conditions are met.
C32 Nasal cancer	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted in the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.
D1 Pneumoconiosis	The rules for this disease have not changed.
D2 Byssinosis	The rules for this disease have not changed.
D3 Diffuse mesothelioma	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted in the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.
D4 Allergic rhinitis	The rules for this disease have not changed.
D5 Dermatitis	The rules for this disease have not changed.
D6 Nasal cancer	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted by the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.
D7 Asthma	The rules for this disease have not changed.
D8 and D8A Lung cancer	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted by the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.
D9 Diffuse pleural thickening	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted by the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.

D10 Lung cancer	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted by the scheme that causation is due to work no matter how long ago the worker left the qualifying employment. Healthcare Professionals should advise based on the medical evidence and the individual circumstances of the case.
D11 Lung cancer	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted by the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.
D12 COPD	The rules for this disease have not changed.
D13 Nasopharyngeal cancer	The time rule has been extended so that presumption applies any time after leaving the job as well as in the job. For this disease it has always been accepted by the scheme that causation is due to work no matter how long ago the worker left the qualifying employment.

Observation form

Please photocopy this page and use it for any comments and observations on this document, its contents, or layout, or your experience of using it. If you are aware of other standards to which this document should refer, or a better standard, you are requested to indicate this on the form. Your comments will be taken into account at the next scheduled review.

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