

A GUIDE TO THE MANAGEMENT OF OCCUPATIONAL ASTHMA

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Foreword

The Occupational Safety and Health Service of the Department of Labour (OSH) is committed to providing health and safety information so that managers and their employees are able to control the risks arising in the workplace to their health and wellbeing.

In New Zealand 90 percent of businesses employ less than ten people. This size of enterprise is unable to afford an occupational health nurse or doctor and local general practitioners act as the default occupational health advisers for this size of industry. General practitioners play a crucial role in the diagnosis and treatment of all occupational illnesses including asthma.

This booklet is the fourth of a series of publications by OSH, highlighting important occupational health problems. It is aimed at providing general practitioners and other primary health care practitioners with diagnostic information and practical solutions to the occupational health problems they face in their day-to-day practice. It is not intended to act as a textbook but as a concise and practical aid to the general practitioner in his or her everyday practice.

Asthma is recognised as a major health problem in New Zealand. What is unknown is the extent of occupational influences on the incidence of asthma amongst the New Zealand working population. Because of this lack of knowledge and because occupational influences are often overlooked, occupational asthma has been included in the OSH Notifiable Occupational Disease System (NODS).

I hope that this information complements the other clinical material available on diagnosing and treating asthma, makes the general practitioner's task easier and contributes towards preserving the health of all those at work.

R Hill
General Manager

Summary

- The exact incidence of occupational asthma is unknown but probably affects about 10 percent of adult asthmatics.
 - An occupational origin must be considered in any case of asthma arising for the first time in an adult.
 - The majority of occupational asthmatics suffer considerable ongoing clinical and financial problems because of their asthma — early recognition and control is vital for them and their families.
 - The diagnosis can be inexact but relies on:
 - a clinical history consistent with asthma,
 - an exposure history to some potential causative agent,
 - evidence of reversible airways obstruction associated with the workplace exposure.
 - The best measure of the occupational origin of a patient's asthmatic symptoms is obtained by peak flows.

A minimum of *four* peak flow measurements a day for *two* weeks is recommended.
 - OSH expertise is available to help make the diagnosis by elucidating the workplace factors and advising on control measures.
 - Recognition and control of the causative agent is the most important step in controlling the patient's symptoms.
 - Treatment of symptoms is no different from other types of asthma but will often be unsuccessful if the exposure is not identified and controlled.
 - In the experience of the panel, the four most common agents to date causing occupational asthma in New Zealand are:

Isocyanate paints, foams and plastics	Car and furniture painters using two-pot paints, foam and plastic manufacturers.
Animal fur and proteins	Laboratory workers, veterinary workers, etc.
Flour and grain dusts	Farmers, grain workers, bakers.
Epoxy resins and other plastics	Boat builders, mould manufacturers, plastic manufacturing processors.
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The History and Incidence of Occupational Asthma

Asthma arising from occupational exposures has long been recognised. It is now clear that developing such an asthma can be serious medically and financially for the patient.

Occupational factors have long been recognised as causing asthma and in aggravating the symptoms of people with pre-existing asthma. In 1713 Ramazzini, an Italian physician who interested himself in the occupational causes of illness, noted urticaria and shortness of breath in grain sifters exposed to organic dusts.¹

In the 1920s Hunter² first published his classical textbook *The Diseases of Occupations* and dealt with occupational asthma under the category of pneumoconiosis (dust diseases of the lungs). He listed numerous individual occupations where asthma-like conditions have arisen. With the industrial development that has occurred this century many of these occupations no longer exist but have been superseded by other agents in new occupations.

Occupations well recognised as being at risk of causing asthma include spray painting with isocyanate paints (two-pot paints used extensively by car painters), carpenters working with Western red cedar and aluminium smelting (potroom asthma at Ti Wai Point Aluminium Smelter).

Both overseas and in New Zealand it is clear that developing an occupational asthma has a profound impact on the wellbeing of that person.

Gannon and Burge³ in the UK and Garrett⁴ in NZ have shown that people in whom the diagnosis of occupational asthma has been made suffer ongoing problems. Some continue to be exposed and suffer continuing symptoms, others change jobs but suffer a financial loss (Gannon reports a median loss of 54 percent of annual income), while other investigators have found that statutory financial compensation awarded to sufferers did not match their loss of income.

Up to 50 percent of people who have suffered from occupational asthma are left with persisting asthma after removal from exposure and this may be severe and disabling⁵. Occasionally asthma attacks, precipitated by occupational exposures, are fatal even though the preceding disease may have been considered quite mild.

The frequency of occupational asthma is unknown in New Zealand. Overseas incident rates vary, but Tarlo⁶ estimates 5 percent of asthma being caused by occupational factors while Gannon and Burge, reporting on the SHIELD scheme in the West Midlands region of the United Kingdom⁷, quote an incidence (the number of new cases in one year) of 43 per million employed. In New Zealand terms this means that some 77 new asthmatics each year suffer a totally preventable illness attributable to their occupation.

The UK Guidance Note¹⁴ on occupational asthma quotes incidence figures ranging from 2 to 10 percent of all adult asthma as being occupational in origin.

Why Has Occupational Asthma Been Included in the NODS System ?

What NODS is trying to achieve

NODS has been established for three principal reasons:

1. To obtain statistical information about the trends in occupational diseases in New Zealand. This allows OSH to fulfil its requirements to Government, professional agencies and the appropriate public health agencies.
2. To act as a source of information for employers, employees and those professionals involved in providing occupational safety and health information and health care — hence this series of guidelines.
3. To focus interventions back into workplaces so as to reduce the incidence of occupational disease in the future.

Notification to this system requires patient consent which ensures compliance with medical ethics and the Privacy Act 1992.

For certain diseases, including occupational asthma, validation panels have been formed and these panels gather any further information required (workplace data, patient factors, etc.) to validate the diagnosis. If necessary, this can include an investigation of the workplace by OSH (which does not require the patient's identity to be revealed).

The diagnostic criteria for occupational asthma, like a number of diseases, are the subject of debate and definitions differ from country to country and, indeed, amongst specialists within any one country.

As a consequence, the advisory panel for this booklet has made some arbitrary decisions concerning diagnostic criteria, recommended methods of investigating the illness and the possible range of specialist investigations. These decisions will be reviewed as the panel gains experience with the cases reported to OSH, but are intended to aid statistical clarity.

What is Occupational Asthma?

A brief background to the complexities of occupational asthma

What seems a neat diagnostic category of occupational asthma becomes increasingly blurred as experience is gained with the condition. The term can be used to encompass pre-existing asthma exacerbated by the work environment, while other definitions refer only to asthma causally related to exposure in the work environment, i.e. “de novo” asthma.

Diagnostic definitions are often complicated by the parallel issue of compensation.

The mechanisms that underlie the development of occupational asthma are not fully understood. Some definitions require sensitisation to be proven before the diagnosis can be accepted* while other definitions take a much broader approach.

There appear to be two causal pathways in the aetiology of occupational asthma¹³.

1. A sensitising agent (either a high molecular weight agent by itself or a low molecular weight hapten conjugated with body proteins) can cause asthma.

Subsequent exposure may cause early bronchoconstrictive reactions occurring within minutes, or later reactions of longer duration. There may be a combination of dual early and late reactions.

However, the nature and timing of laboratory-induced responses described above do not necessarily reflect the pattern of asthma arising from constant exposure at work.¹⁴

2. A heavy, sudden exposure to an irritating agent can give rise to asthma. The latter experience has been called “Reactive Airways Dysfunction Syndrome” (RADS) and may be the mechanism for such occupational asthma as the “potroom asthma” experience in aluminium smelting.

The late asthmatic response or the after effects of a sudden heavy exposure to an irritating agent are associated with “bronchial hyper-responsiveness” where the airways react to a variety of stimuli such as cold, exercise, smoke and dusts as well as the original provoking agent. Many of these stimuli are encountered outside the workplace and this pattern of complaint can make the diagnosis of occupational asthma very difficult.

The diagnosis of occupational asthma, like that of all occupational diseases, is built upon the foundation of a good occupational and clinical history, a framework of knowledge of what the patient is exposed to and, to complete the metaphor, the roof of clinical findings.

Smith proposes six criteria for establishing the diagnosis of occupational asthma⁸ and these criteria are useful to use as a check when considering the diagnosis.

They are:

1. Clinically demonstrable variable airways obstruction.
2. A known provoking agent is present in the workplace.
3. There must be exposure to this agent for a sufficient time.

* Industrial Injuries Advisory Council (UK) — Asthma that develops after a variable period of symptomless exposure to a sensitising agent at work.

4. Tests of pulmonary function show airways obstruction after exposure to a specific agent. (This can be demonstrated by getting the patient to note their exposures on the peak flow chart as they challenge themselves in the workplace by working with the suspected exposure.)
5. The asthmatic condition usually develops when inhaling low concentrations of the specific agent.
6. Symptomatic improvement occurs in many cases when the patient is away from work, and symptoms are aggravated during the work day or working week.

These criteria can be used as a guide when making the diagnosis of occupational asthma.

Occupational and Clinical History

Steps in making the diagnosis

A careful history is the most important tool to making a diagnosis of occupational asthma. The main point of the history is to establish the temporal link between the work and the asthma.⁹

Occupational History

The occupational history attempts to establish the likely agent and the length and degree of exposure.

The occupational history should record the work that the patient has done since leaving school and the exposures that the patient had in these occupations. The intention is to identify the causative agent and whether the symptoms are totally new or an aggravation of longstanding problems.

Particular attention should be paid to what these exposures (e.g. dusts, chemicals) consisted of. For example, “lots of dust” needs some clarification as dusts may be inert as in soil dusts, contain wood and chemicals as in wood dust, biological material (animal protein and spores) as in dust from a poultry shed, or dried chemicals (powder coating plants or timber treatment plants).

Chemicals are often only known by their commercial names or are a mixture of other chemicals. OSH occupational hygienists are available to offer advice as to the potential of materials or products to cause asthma or to research the constituent parts of a product. They may be contacted at the OSH branch offices listed in this guide.

A useful tool when trying to assess a product is to obtain the Material Safety Data Sheet usually referred to as the MSDS. This can be obtained from the manufacturer of the chemical product free of charge or from the Poisons Centre in Dunedin. The MSDS presents information concerning the product

in a standard way and is useful as a shorthand, quick reference when assessing the risk to patients.

An occupational asthma investigation form used by the OSH occupational health nurses is included in Appendix 2 for use if desired.

Note should be made of:

1. Latency Period

There should be a time lapse between the first exposure and the development of symptoms. This can vary according to the specific agent and the degree of exposure. For example, a large spill of a substance or a fire involving isocyanates can cause immediate symptoms.

However, it is usual for a longer period of exposure to occur before symptoms develop. (It should be remembered that a person may have been sensitised in a previous occupation and develop symptoms very quickly on being rechallenged by the exposure in a new workplace.)

Commonly, occupational asthma begins within one or two months of initial exposure and will usually have occurred within one to two years of exposure. Very uncommonly, the sensitising period can be a matter of days or weeks.⁵

2. Exposure History

Exposure means taking a guess at the dose of a substance the person received. It is essentially a dose (or concentration of the substance) time calculation and takes into account the following:

■ *How messy was the workplace ?*

Sometimes you must rely on anecdotal estimates (“I couldn’t see across the workshop because of the dust”). Occasionally environmental monitoring (i.e. dust concentration measurements) has been performed and can be used. (This sort of information may have been collected by OSH or the employer.)

■ *How many hours a week was the patient exposed ?*

Try to rough out a time estimate for the exposure — so many hours per week for so many years. This is essentially similar to the calculation of pack years used for smokers.

■ *What protection was in place and was it working ?*

Protection can range from sophisticated exhaust booths with their own ventilation through to the personal protection offered by a respirator.

It is important to assess how well this protection worked. In many workplaces there is no protection available, or the ventilation was switched off or inoperative because of faulty maintenance.

Personal protection (e.g. respiratory masks) is often ineffective because people find such protection uncomfortable and won't wear them and no effort is made by management to ensure the protection is used.

Clinical History

The key questions to ask your patient

The clinical history is vital to making the diagnosis and is composed of several parts. Usually, it is the clinical history that enables the diagnosis of occupational asthma to be made. There are four key screening questions to ask. Although the actual pattern of asthma varies according to the substance and the patient, these questions will help identify the majority of sufferers:

■ *Does your asthma vary during the working week ?*

Usually, occupational diseases including asthma worsen during the working week so that by the end of the shift period there has been a steady deterioration in symptoms and clinical measurements.

Often questioning reveals certain days and tasks where symptoms are more pronounced helping to identify the cause of the asthma.

■ *Is your asthma better or worse on days away from work ?*

Patients may note a continued improvement away from work, especially on long weekends or holidays. Identifying work/non-work periods on peak flow charts gives a vital clue to occupational asthma.

■ *Is your asthma better or worse on holidays ?*

For those patients with severe asthma and who have a slow recovery, the two- or three-day break may not be sufficient to allow a return to the non-asthmatic state. Patients often report a dramatic improvement of symptoms after several days away from the workplace and a relapse of symptoms within 48 hours of returning to work.

In complex cases it is necessary to differentiate between holidays at home or away from home because of the compounding effect of irritants.

■ *Is your sleep disturbed by cough or breathing problems ?*

Occupational asthma often causes "night" cough or episodes of wheezing or shortness of breath waking the patient from sleep. These symptoms can be quite mild, are often initially misinterpreted because they do not occur at work (e.g. the shift worker will blame the light and noise outside for waking him or her or attribute the symptoms to a recurrent cold).

Smoking History

Burge¹² notes that the prevalence of occupational asthma is higher in smokers and smoking enhances the production of specific IgE in an occupational setting. A variety of mechanisms have been postulated to explain this increased prevalence including an increase in exposure to an occupational agent by smoking in the at risk environment.

Smoking history is always relevant. Some employers or insurance companies will attempt to blame smoking as the cause of the symptoms rather than accepting that work can be a causative agent. It will be impossible to control an occupational asthma while your patient continues to smoke in the workplace in the presence of the causative exposure. Without a smoking history you cannot properly advise the patient or their employer.

The Role of Atopy

Obtaining a history of atopy is important when considering whether an asthma is the result of becoming sensitised or when trying to assess whether the work situation has aggravated a pre-existing asthma. Positive skin prick tests to a variety of common allergens and an increased total serum IgE help support the diagnosis of atopy.

Clinical Findings

Measurements used to make the diagnosis

The NODS Asthma Panel tries to obtain the following information when assessing a case:

- Clinical evidence of reversible airways obstruction (peak flow variability, response to inhaled beta-agonist such as an increased peak flow or FEV₁).
- A peak flow pattern demonstrating a increased peak flow variability associated with exposure. To measure this adequately the panel recommends:

A minimum of four peak flows a day for two weeks.

The best of three (to ensure a consistent result) peak flows is recorded. Before work, during work (mid shift), immediately after work and as the patient goes to bed are the suggested times. A peak flow chart is available in Appendix 2 and pads of this peak flow chart are available from the local OSH branch office.

The patient must mark the time at work and not at work and, if possible, periods when they were exposed to the suspected aetiological agent, presuming the exposure was not continuous. They should also

try and record symptoms such as cough, wheeze and shortness of breath on the chart.

Ideally, these peak flows should be performed over a week at the end of the patient's holidays and the beginning of their work period. The peak flow chart should always include time away from work, even weekends or the long shift break.

- Lung function tests (FEV_1 and FEV_1/FVC) both before and after administration of a bronchodilator.

To gain the maximum benefit these tests should be arranged for a time when the patient is in the middle of their working cycle and after a medication-free period of at least 4 hours.

This might be the counsel of perfection for some general practitioners, but many practices now have spirometers and will have the ability to follow this protocol.

- In specialist centres the measurement of non-specific bronchial responsiveness or irritability is a useful tool in assessing the patient. This involves measurement of the provocative concentration of histamine or methacholine that causes a 20 percent fall in FEV_1 . This concentration is called the PC_{20} . A positive methacholine or histamine challenge supports a clinical diagnosis of asthma when baseline pulmonary function is normal. A negative study result does not, however, rule out occupational asthma.¹⁰

In some industries serial measurements of bronchial responsiveness are used to:

- (a) Monitor at risk employees;
- (b) Assess suspected cases of occupational asthma;
- (c) Assess response to treatment; and
- (d) Monitor a person's wellbeing after re-introduction to the workplace.

Other specialised tests are available overseas. These include skin testing, radioallergosorbent testing (RAST assays) and specific inhalation challenges.

Skin testing with common allergens (house dust, grass and tree pollens) can be useful in determining the atopic status of a patient. These tests do not predict the reaction of the airways to these allergens. Atopy may point to an occupational exacerbation of a pre-existing asthma.

In a few cases, appropriate extracts of potential occupational allergens are available for skin testing, but it must be remembered that both skin testing and serologic testing document exposure and sensitisation but may not necessarily be associated with symptoms of asthma¹⁰. Such tests are not performed routinely in New Zealand to the knowledge of the NODS panel and it is often not clear what the tests mean when they are performed.

Specific inhalation challenges are not performed in New Zealand. They have been described as the final arbiter of proof⁹ but they are exceedingly expensive and are not without considerable risk to the patient. Furthermore, problems arise in estimating the actual inhaled dose encountered in the workplace, often of a mixture of substances and, in evaluating the results of artificial doses given, in an artificial environment.

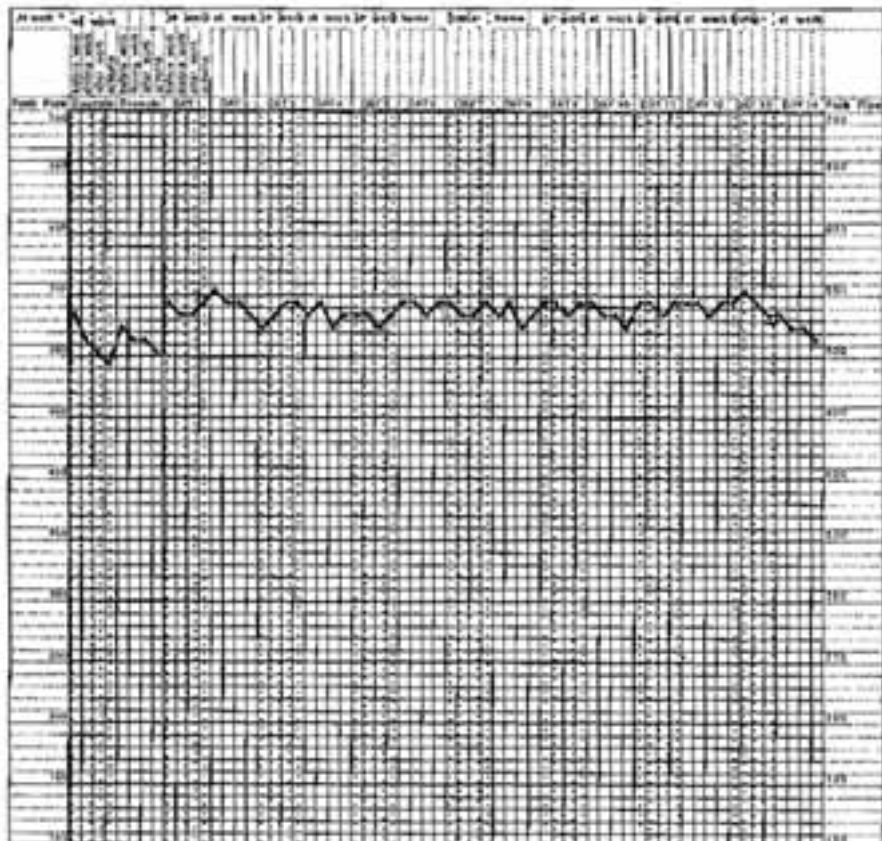
Interpretation of Peak Flow Recordings

The common patterns of peak flow recording in occupational asthma

The peak flows seek to establish the relationship between work and symptoms. Once occupational asthma is occurring the symptoms usually worsen during the working week and recover, to some extent, during the weekend or shift break.

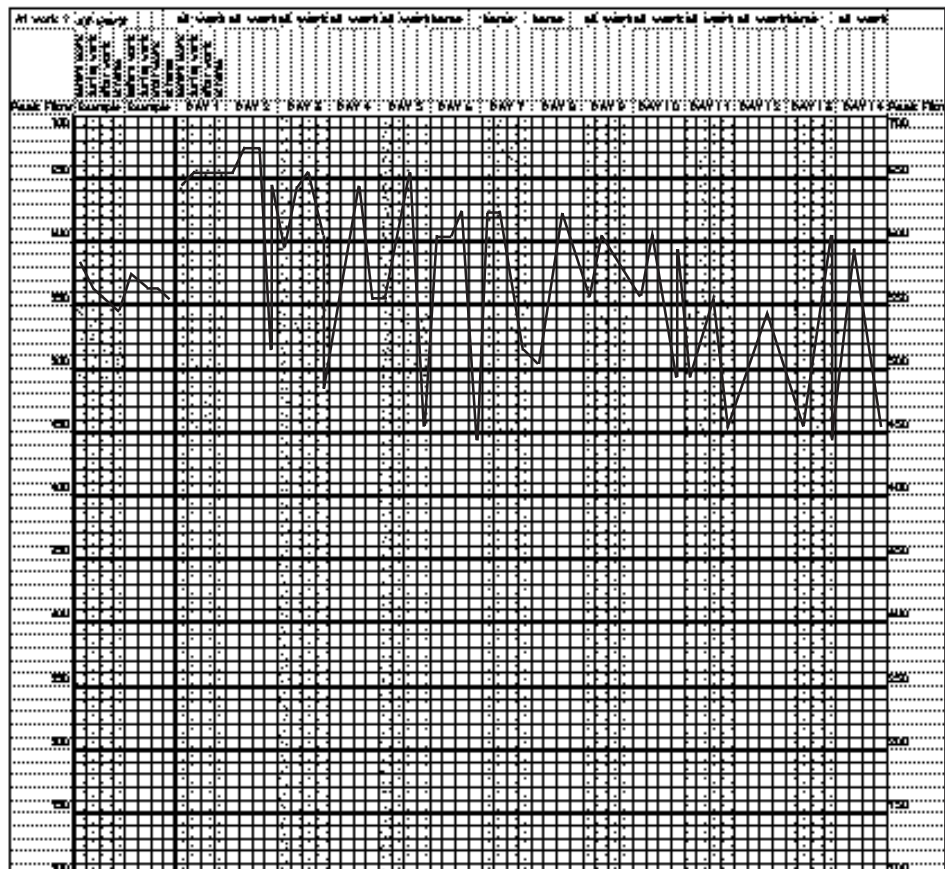
Burge reports the following patterns of peak flow response⁹:

- A normal record with a peak flow in the normal range (age and height) with a diurnal variation of < 15 percent demonstrated by the following graph.



- Patients with a reduced mean peak flow or an increased diurnal variation that fail to show an improvement over weekends. Occupational asthma may be missed because of the long period needed for some people to recover and, in these cases, peak flows over a period of two weeks away from work may be necessary.
- Patients showing clear evidence of occupational asthma where peak flow records show an increased diurnal variation and deteriorating peak flows when at work and improvement when away from work.
- Patients who show a deterioration at work and improvement away from work where the diurnal variation is less than 15 percent. This situation can occur where there is severe occupational asthma with low peak flow values, or where there is an additional workplace irritant on top of unrelated chronic airflow obstruction.

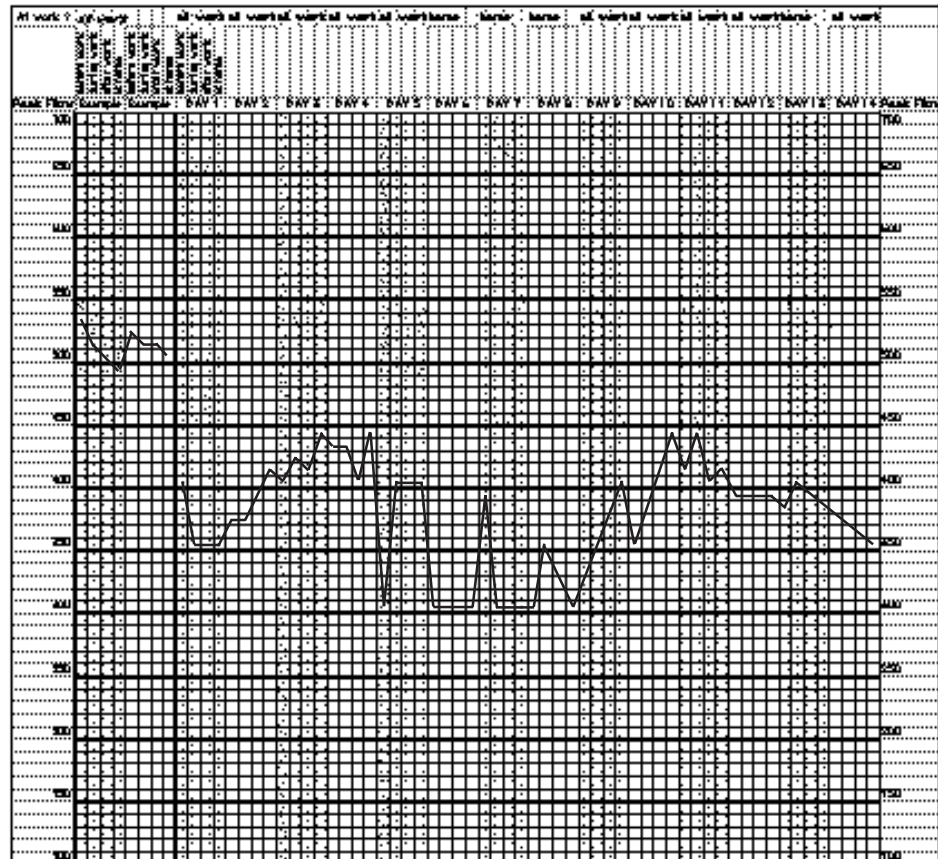
Discussion of these various patterns can be confusing and the following peak flow chart measured by a welder working in the ship repair industry is a good example of a positive peak flow chart. It shows an excessive diurnal variation with a steady deterioration over the working period. Extra peak flows taken in the middle of the night show dramatic falls in peak flow values. The peak flows at the beginning of the chart represent the only time the welder wasn't working and give a baseline of his non-exposed lung function.



A number of exposures are the possible culprit(s) and include the welding fumes composed of oxides of nitrogen, sulphur dioxide, metal fumes from the metal, and the products of combustion from cutting isocyanate foam insulation and ozone produced by the welding process.

This welder was using little in the way of personal protection and the welding often took place in a confined space where the oxygen content of the inhaled air could be reduced because of its consumption in the welding process.

A second peak flow chart is from an electronic worker referred by her general practitioner with persistent and poorly controlled asthma. This patient was exposed to solder fumes containing colophony and her peak flow values show a steady deterioration during the working week and a recovery over the weekend.



This person's symptoms were controlled by the installation of some efficient local exhaust ventilation and remains well and in continued employment.

Getting the Information to Make the Diagnosis

When you suspect one of your patients has occupational asthma, but you are unsure of the possible causes or the condition of the workplace, OSH is able to provide information or have its officers inspect the workplace if it is warranted.

The Departmental Medical Practitioners, members of the OSH Asthma Panel and the local OSH office are listed at the back of this booklet and can be contacted for advice or assistance.

Treatment of Occupational Asthma

How can you help your patient?

The pharmacological treatment of occupational asthma is no different from any other form of asthma. The only important difference between the treatment of occupational asthma compared with non-occupational asthma is the necessity of identifying the causative exposure and controlling it.

Too often it seems that advice given to sufferers consists of telling the sufferer to “wear a mask” or alternatively to “find another job”. Neither of these two options are necessarily the best way of dealing with the problem.

There is evidence that the early recognition of occupational asthma and control of the causative exposure lessens the severity of the condition.¹⁵

What advice you can give to control the problem?

The Health and Safety in Employment Act (1992) requires that employers identify hazards and then control them by elimination, isolation and minimisation.

This hierarchy of control measures is a useful tool to use when advising your patients or their employers of the steps to consider.

Elimination

In today's industrial climate substituting the hazard with another product or material is often feasible. For example, the employer of the electronic worker, whose peak flows are represented above, was able to purchase a riveting machine which did away with the need to use solder in some of the circuit-board manufacturing processes. OSH occupational hygienists can advise doctors if alternative products or processes are available.

Isolation

By restricting the agent to some part of the factory or to specific times, it may be possible to remove your patient from exposure to the asthma-causing agent and relieve their symptoms. It should be remembered that for the sensitised patient very small exposures will be sufficient to precipitate symptoms.

Minimisation

The minimisation of the risk from the asthma-causing agent can be achieved by a number of measures. The first control measure to consider is adequate ventilation (both local and environmental) and the last measure is personal protection by means of respirators.

Ventilation is a complex solution and the employer is well advised to seek some expert advice before purchasing equipment or approaching the problem on a piecemeal basis.

Personal protection (a respirator) is not a cheap option. To be used successfully, the respirator must be appropriate for the agent or mixture in question, be individually fitted, maintained correctly and, most importantly, worn whenever the person is exposed to the agent or mixture. It should only be used in conjunction with other measures and where substitution of the offending agent is not practicable.

Most people find wearing a respirator a nuisance and compliance with rules to wear a respirator is often poor because of a number of factors. Such protection may be suitable where the exposures are not heavy and are for only a small portion of the working shift.

Respiratory protection is a subject in its own right and rather than cover it inadequately in this booklet a copy of *A Guide to Respirators and Breathing Apparatus* may be obtained from any OSH branch office.

Only as a last resort, and after consultation with a respiratory or occupational medical specialist, should someone be advised to abandon their occupation. The decision is obviously of great importance to the individual and has implications for compensation issues as well as the more obvious treatment issues.

Compliance with the Health and Safety in Employment Act 1992

Occupational asthma is defined in the Health and Safety in Employment Act as “serious harm”. As such there is a duty on the employer to inform OSH “as soon as is possible” when an employee is diagnosed as suffering occupational asthma. The obligation is not on the doctor, although the doctor is able to inform OSH via the NODS scheme if the patient consents.

A doctor would have fulfilled any obligation under law by giving the patient a letter to hand to their employer stating that the patient was suffering from occupational asthma.

Conclusion

Occupational asthma is relatively uncommon, but when it does occur it has a profound impact on the individual’s wellbeing. It should be considered in any adult who is suffering asthma for the first time in their lives.

Many other asthma sufferers have their condition made worse by workplace factors, and the suggestions about control measures apply to them as well as the “pure” occupational asthmas.

The most important difference in the treatment of occupational asthma is to try and identify the causative exposure and control the patient’s exposure to it.

As with most medical problems, this requires a team approach in which the OSH health and technical services are able to play a part in protecting your patients.

If you have any comments about this booklet, or suggestions for future information booklets, please write to:

OSH Health Services
Occupational Safety and Health Service
Department of Labour
PO Box 3705
Wellington

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Appendix 1: Materials Recognised as Causing Occupational Asthma (adapted to New Zealand conditions)

1. High Molecular Weight Compounds

Material	Industry and At Risk Occupations
Animal products, insects, other laboratory animals Guinea pig Mouse Rabbit Rat	Laboratory workers Veterinarians Animal handlers
Birds Pigeon Chicken Budgerigar	Pigeon breeders Poultry workers Bird breeders
Insects Grain mite Cockroach Cricket Moth/butterfly	Grain workers Research laboratory Laboratory workers Field contact Fish bait breeder Entomologists
Plants Wheat.rye/flour Buckwheat Coffee beans Castor beans Tea Tobacco leaf Hops	Grain handler Bakers, millers Food processing Oil processing Food processing Tobacco industry Brewing industry
Biologic enzymes B subtilis Trypsin Pancreatin Papain Pepsin Flaviastase Bromelin Fungal amylase	Detergent industry Plastic, pharmaceutical Pharmaceutical Laboratory Pharmaceutical Pharmaceutical Pharmaceutical Manufacturing, bakers
Vegetables Gum acacia Gum tragacanth	Printers Gum manufacturing
Other Crab Prawn Hoya	Fish processing Fish processing Oyster farming

2. Low Molecular Weight Compounds

Material	Industry and At Risk Occupations
Diisocyanates Toluene diisocyanate (TDI) Diphenyl methane diisocyanate (MDI) Hexamethylene diisocyanate (HDI)	Polyurethane foam industry, plastics, varnish Foundries, plastics Automobile spray painting
Anhydrides Phthalic anhydride Trimellitic anhydride Tetrachlorophthalic anhydride Maleic anhydride	Epoxy resins, plastics (boat building)
Wood dust Western red cedar California redwood Cedar of Lebanon Cocoboila Iroko Oak Mahogany Abiruana African maple Tanganyika aningre Central African walnut Kejaat African zebra wood NZ rimu Particle board (formaldehyde)	Carpentry, construction, cabinet making, sawmilling
Metals Platinum Nickel Chromium Cobalt, vanadium, tungsten carbide	Platinum refining, jewellery manufacture, photography Metal plating, stainless steel welding Hard metal industries (steel making, tool hardening, etc)
Fluxes Aminoethyl ethanolamine Colophony	Aluminium soldering Electronic industries, welding
Drugs Penicillins Cephalosporins Phenylglycine acid chloride Piperazine HCL Psyllium Methyl dopa Spiramycin Salbutamol Amprolium HCL Tetracycline Sulphone chloramides	Pharmaceutical Pharmaceutical Pharmaceutical Chemist Pharmaceutical Pharmaceutical Pharmaceutical Pharmaceutical Poultry feed mixer Pharmaceutical Manufacturer, brewer

2. Low Molecular Weight Compounds (Continued)

Material	Industry and At Risk Occupations
Other chemicals Dimethyl ethanolamine Persulphate salts and henna Ethylene diamine Azodicarbonamide Dioazonium salt Hexachlorophene Formalin Urea formaldehyde Freon Glutaraldehyde Paraphenylene diamine Furfuryl alcohol (furan bases resin)	Spray painting Hairdressing Photography Plastics and rubber Photocopying and dyes Hospital staff Hospital staff Insulation, resins, particle boards Refrigeration Health care workers, radiographic processors Fur dyeing Foundry mould making
Aliphatic polyamines Ethylene diamine Diethylene triamine Triethylene tetramine	Hardeners, particle board

Appendix 2: OSH Occupational Asthma Investigation Form

Questions 1-41 of this form should be completed by the Occupational Health Nurse while interviewing the person notified as a possible case. Questions 42-53 may be completed later.

Please return this form to the Regional Departmental Medical Practitioner when complete.

Personal details of person investigated

1. Surname:
2. Given names:
3. Home address:
4. Telephone numbers:
5. Date of birth:
6. Gender:
Male Female
7. Ethnic origin?
European
Maori
Pacific Island
Maori
Other
If other please specify below:

8. Name and address of family doctor?

Smoking

9. Have you ever smoked for as long as a year?
Never **Go to question 11**
Used to smoke **Go to question 10**
Smokes now **Go to question 10**

10. At what age did you begin smoking?

On average how many cigarettes did you/do you smoke each day?

If you no longer smoke how old were you when you stopped smoking?

Occupational History

11. Current employment status?
Employed **Go to question 12**
Unemployed **Go to question 14**
Retired **Go to question 14**
On a sickness benefit **Go to question 14**

12. Current job:
Job title:
Description:

13. Current employer's name and address:

14. Please list your past employers and the number of years worked for each employer (use additional paper if needed).

Job title and employer	Duration from 19.. to 19..	Please describe the nature of the work performed in this job

Symptoms

15. Have you had wheezing or whistling in your chest in the last twelve months?

No Yes

16. In the last 12 months have you been breathless when the wheezing was present?

No Yes

17. Have you had this wheezing or whistling when you did not have a cold?

No Yes

18. Have you had wheezing or tightness in your chest during or straight after work?

No **Go to question 20**

Yes **Go to question 19**

19. Does the wheezing or tightness in your chest improve when you are away from work such as holidays or weekends?

No Yes

20. Have you woken up with a feeling of tightness in your chest at any time in the last twelve months?

No Yes

21. Do you have a persistent cough?

No **Go to question 23**

Yes **Go to question 22**

22. Do you tend to cough up phlegm on most days?

No Yes

23. Do you get short of breath walking on the flat?

No Yes

24. Do you get short of breath walking up a slight incline?

No Yes

25. Do you get short of breath more than other people of the same age?

No Yes

26. Do you notice that the wheeze or cough gets worse during the working week?

No Yes

27. Do you get short of breath when you are away from work on a holiday or a long break?

No Yes

28. Do you wake up at night short of breath?

No Yes

29. Do you wake up at night because of persistent coughing?

No Yes

30. Do you know what makes you wheeze?

No **Go to question 33**

Yes **Go to question 31**

31. If you answered "yes" to **Question 30**, what are all the things that make you wheeze? (Mark the appropriate boxes)

Cold

Dusts

Cigarette smoke

Chemicals such as solvent or isocyanate fumes

Go to question 32

Other

Go to question 32

32. If you answered yes to "chemicals" or "other" please specify.

33. Are you currently taking any medicines for asthma (e.g. inhalers, aerosols or pills)?

No **Go to question 34**

Yes **Please specify below**

Medication

34. Have you been seen by a respiratory specialist?

No Yes

If yes, please enter the name of the specialist below.

35. Have you been seen admitted to a hospital because of asthma?

No Yes

If yes, please enter the name of the hospital below.

Medical history

36. Did you get asthma when you were a child under 16?

No Yes

37. Have you ever suffered from eczema?

No Yes

38. Have you ever suffered from hayfever?

No Yes

39. Do you have a family history of asthma?

No Yes

40. Do you have a family history of eczema?

No Yes

41. Do you have a family history of hayfever?

No Yes

42. Please complete the following table indicating: substances used in the workplace that could be implicated in this patient's problems; length of exposure; intensity of exposure. Use extra paper if necessary.

In order to assess whether this person's exposure was heavy or moderate, take into account factors such as the percentage of the day that the person was exposed and whether any protection was used by the person.

Substance	Months of exposure	Exposure estimate (e.g. in your opinion was this person's exposure heavy, moderate or light)

Workplace environmental monitoring

43. Has environmental monitoring for the suspected workplace substances been carried out?

No

Yes **If yes, please attach results.**

44. Does environmental monitoring need to be carried out?

No Yes

45. What environmental monitoring have you and/or the occupational hygienist done? Tests ordered (attach a copy of the results to this form).

46. Is there a problem in the place of work?

No Yes

Biological monitoring

47. Patient clinical data

Age	Height

	Predicted	Sample 1	Sample 2	Sample 3	Post bronchodilator
FEV1					
FEC					
FEV1%					

Attach the peak flow diary to this form. Peak flow forms must record time away from work and time since last dose of bronchodilator was taken by the patient. (Peak flow forms are compulsory for this diagnosis.)

48. When was the last dose of bronchodilator taken by the patient?

Details of investigating nurse

49. Investigated by

50. Branch office

51. Date investigation commenced

52. Date investigation concluded

53. Which of the following actions were initiated as a result of this notification?

- Discussion with management re safety systems
- Workplace health and safety assessment
- Investigatory biological tests
- Education
- Recommendation for consultation with private sector health and safety providers
- Investigation of other workers
- Workplace improvements recommended
- Workplace improvements raised
- Enforcement action required
- Environmental monitoring

Please use additional paper if you would like to make any comments (e.g. justification for your exposure estimate), and return with this form to your regional DMP.

Signature (OHN)

Date

