INDUSTRIAL DERMATITIS

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Industrial dermatitis is the commonest of all occupational diseases. The range of occupational skin diseases is indicated and the mechanisms of contact dermatitis, the largest single group of occupational dermatoses, explained. The methods available for the prevention of occupational dermatoses at the earliest possible stage are reviewed.

INTRODUCTION

Industrial dermatitis is skin disease primarily caused by the industrial environment. It is the commonest occupational disease in industrialised countries. Of all industrial injury benefits paid out in this country for occupational diseases prescribed under the 1965 National Insurance (Industrial Injuries) Act, a little more than 65% are for industrial dermatitis. Occupational skin disease has recently been estimated to cost the United States at least 34 million dollars annually, with the possibility of a true figure 10 times this estimate. The real cost of occupational skin disease, however, is to the quality of the lives of working men and women.

The skin can react in a variety of ways to industrial substances and the term industrial dermatoses is now preferred to the term industrial dermatitis. The overall term occupational dermatoses allows the inclusion of the skin problems of the non-industrial workforce.

Occupational Dermatoses

The largest single group of occupational dermatoses are eczematous reactions of the skin due to contact between it and external chemical agents. This type of skin disease is known as contact eczema or contact dermatitis. In this context the word "dermatitis" has a specific meaning synonymous with eczema, rather than the more general meaning it has in the term "industrial dermatitis". Eczema or dermatitis is an inflammatory skin reaction, recognisable by trained physicians by its characteristic combination or redness, swelling, blistering, oozing of fluid, flaking, cracking, thickening up of the

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skin and slight bleeding.

The skin's repertoire of appearances in disease is, however, somewhat limited and not all eczematous skin rashes are caused by contact with external chemical agents. It is possible for other forms of eczema to appear at any time from infancy to old age. The causes of these other forms of eczema are largely unknown but are generally regarded as being determined by the individual's constitutional or hereditary make-up rather than by any external factors. It can sometimes be difficult even for physicians to distinguish between constitutional (endogenous) and contact (exogenous) forms of eczema. The skin's range of reactions to external stimuli may be restricted, but it is not totally limited to eczema. Mineral oil and coal tar pitch can cause acne and skin cancer, certain polychlorinated aromatic hydrocarbons another form of acne known as chloracne, glass fibre an intense itching, vinyl chloride monomer a special type of skin thickening termed scleroderma, platinum salts "nettle-rash", and alkyl phenols white patches in the skin called leucoderma. All these conditions are examples of non-eczematous occupational dermatoses.

Not all external stimuli causing occupational dermatoses are chemicals. Responsibility may lie with biological agents such as bacteria, viruses, fungi, yeasts and biting arthropods or physical agents like heat, cold, high and low humidity, mechanical trauma, vibration and both ionising and non-ionising radiation. Skin diseases removed far more troops from jungle warfare in Vietnam than the enemy.

There are many toxic chemicals, like organophosphorous pesticides, which are absorbed through the skin and attack internal organs without necessarily causing any kind of skin rash. Other chemicals, such as polychlorinated aromatic hydrocarbons, can cause both skin problems and systemic or generalised toxicity. Some chemicals, such as wood rosin (colophony), can cause asthmatic lung disease as well as eczematous skin disease.

Contact Dermatitis

Having indicated the variety of occupational dermatoses and some of the interfaces between occupational dermatology and other areas of occupational medicine, occupational contact dermatitis remains the most important skin problem in industry. Chemicals capable of causing contact dermatitis can do so in two main ways. Contact irritants are substances which directly damage the skin if in contact with it in sufficient concentration for sufficient time. The more obvious industrial irritants are acid of pH less than 2, alkalis of pH more than 11 and many organic solvents. However, weak or marginal irritants are also an important cause of industrial contact dermatitis and require prolonged and repeated contacts in order to provoke an inflammatory effect. Water-based metalworking fluids ("soluble oils") are a prime example of marginal irritancy. A marginal irritant may not cause any obvious burning and stinging on the skin and yet can be insidiously damaging to it.

The other way that a chemical can cause contact dermatitis is by a process known as sensitization. Contact sensitizers are substances which can penetrate the outer layers of the skin and induce a specific type of allergic reaction involving the body's immune system. Such chemicals usually have a molecular weight of less than 1000. They may cause no direct skin damage at all. Sensitization may occur after only one contact or after many years of repeated contact. Once initiated the process of sensitization takes 7-14 days to be completed. Individuals vary in their ability to be sensitized. Once an individual has become sensitized to a substance, further skin contacts with

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that substance, even in very small quantities, will elicit an allergic contact dermatitis. Chromates, epoxy resin and hardeners and rubber processing chemicals are examples of common industrial contact sensitizers. Some substances, such as epoxy hardeners, can be both irritant and sensitizing. Some potential irritants and sensitizers only cause an effect together with the additional stimulus of ultraviolet light in sunlight or industrial light sources. These substances are described as phototoxic and photoallergic respectively. Polychlorinated salicylanilides are an example of the latter.

Contact sensitization, once established in an individual, can be regarded for practical purposes as permanent. Whether or not that individual continues to have allergic contact dermatitis as a result of their allergy will depend upon whether or not contact with the specific contact sensitizer is sustained. Because of the inherent toxicity of most industrial allergens if introduced into the body, desensitization injections of the type familiar in the treatment of hayfever or asthma are not normally a practical possibility. Contact sensitization is variously estimated to account for 20 to 50% of occupational contact dermatitis.

Patch Testing

While the physician has to rely largely on circumstantial evidence and his background knowledge of contact irritants in diagnosing irritant contact dermatitis, a powerful weapon has been developed in the diagnosis of allergic contact dermatitis, patch testing. The principle of a patch test is as follows: if the skin of an individual previously sensitized to a substance is presented with that substance at an appropriate concentration, a visible skin reaction will occur usually between one and 3 days later. Patch test substances are applied to the skin under occlusion by adhesive tape for 1-2 days. Crucial to the validity of the test is the phrase "appropriate concentration". Too high a concentration may irritate the skin of most nonsensitized and sensitized individuals alike and cause "false positive" reactions. Too low a concentration may fail to provoke a reaction at all even in a previously sensitized individual, a "false negative" result.

The use of samples of substances direct from the workplace is capable of providing both false positive and false negative patch test results. A small concentration of a stabilising chemical in an organic solvent, for example, may be capable of sensitizing certain individuals routinely exposed to the solvent. If the solvent is put on as a patch test undiluted it may simply provoke a false positive irritant reaction or no reaction at all and there may be too little stabilising chemical there to provoke a reaction under the conditions of a patch test, even in a person previously sensitized to it. If the solvent is diluted, say 25%, it may not now cause a false positive irritant reaction but the stabilising chemical will be even more likely to give a false negative reaction. If sensitivity to the stabilising chemical is to show up, it may have to be patch tested alone at a concentration say 10 times higher than its product concentration in order to give a skin reaction under the conditions of the patch test. Too high a concentration of stabilising chemical and it too may begin to give false positive irritant reactions.

There is much more to patch testing, therefore, than simply applying every substance an employee works with to his skin for 48 hours. Specialised training and experience are required for patch testing to have any reliability.

PREVENTION

Equipped with what is known about the causes of occupational detmatoses, what can be done to prevent them happening? Medical surveillance of the workforce, provided that it results in accurate diagnoses, is a vital source of secondary prevention. Surveillance is especially important at research and development, pilot plant and early production stages involving the introduction of any new chemical. There have been occasional instances of chemicals causing skin rashes at an early stage in the development of a process but these rashes having been overlooked until skin problems arose later in general usage. Such optimism may sometimes be justified, if for example usage concentrations are intended to be much lower than research laboratory levels, but early warning signs should always be intensively investigated if mistakes are to be avoided.

The main input of the dermatologist, therefore, is in the early and accurate diagnosis of dermatoses as they occur and the subsequent advice he can provide, as a result, on the prevention of further similar dermatoses. Techniques and principles have been derived in this way which should all now be playing their part in the primary prevention of occupational dermatoses.

Toxicity Assessment

The best possible way of secondarily preventing occupational dermatoses is to substitute toxic with non-toxic chemicals. The equivalent technique is primary prevention is the prediction of whether a chemical is going to be toxic to the skin. The central problems here are the facts that no chemical is absolutely non-toxic and that, while some idea of the inherent toxicity of a chemical may be gained from laboratory testing, this information needs to be converted into an estimation of actual hazard in the workplace before it is useful.

Assessments of chemicals as potential skin hazards should start with an examination of the chemical similarities of the molecule with other substances about which skin toxicity date already exists. Given that paraphenylenediamine is a contact sensitizer it is not surprising perhaps that benzocaine and sulphanilamide also are (Fig. 1).

The next step is to use such standardised laboratory test procedures as have been devised and are possibly relevant to the chemical in question. Laboratory test procedures have been developed for the assessment of skin irritancy, sensitizing potential, photosensitizing potential, acnegenecity, depigmentation, hair loss, carcinogenicity and other parameters. Most of these tests depend upon the use of laboratory animals such as the rabbit and the guinea pig. The reliability of all of them depends upon the excellence of the experimenter. For both these reasons toxicity data obtained in these ways is still fallible. For example, a comparison of the results of 25 different laboratories, using the same standard rabbit eve and skin irritant tests for the same series of chemicals, showed very little agreement (Weil and Scala (1)). The more experience that is gained with such tests, however, the better predictivity is likely to become. The guinea bit maximization test (Magnusson and Kligman (2)) for sensitizing potential is capable of producing reasonably repeatable results, provided that the precise test details are strictly adhered to. Better test procedures for irritancy still require to be developed.

It must always be remembered that some knowledge of the skin exposure expected to a chemical in use is essential for its hazard to be finally estimated. Details of usage concentration and degree of exposure are crucial: the smaller each is, the less likely is a toxic chemical to be hazardous. In general, sensitizers are hazardous down to lower levels than irritants.

It is less likely that skin toxicity problems of materials will be missed if individual constituents are tested independently than if they are tested mixed together in a product. The primary concern in toxicity testing should be not to mimic real life exposure but to provide rigidly standardised test conditions so that results are truly comparative. It may also be necessary sometimes to fractionate a substance in order to isolate a sensitizing chemical from a mixture of chemically related but non-sensitizing chemicals. A good example of this is the recent work of Fregert and Thorgeirsson (3) on epoxy resin. They have shown that the contact sensitizing ability of bisphenol A-epichlorhydrin based epoxy resin resides almost entirely in resin of molecular weight (MW) 340, with perhaps some contribution from MW 624, but that all higher molecular weights in the resin are non-sensitizing. There are technical difficulties in excluding MW 340 from commercial resins, because at present it is the chemical backbone of most of them, but this investigative work has raised the possibility of definitive primary prevention of epoxy resin dermatitis, one of the commonest forms of allergic contact dermatitis in modern industry.

Predictive testing of chemicals is never likely to be the complete answer to prevention for several reasons. Firstly, laboratory animals do not always react to chemicals in the same way as humans. Secondly, chemicals with a predicted toxicity may still have to be used in industry, because there is no known safer substitute available. Thirdly, the precise chemical nature of industrial products can vary between batches. For example, the rubber chemical dithiodimorpholine has been found to be responsible allergic contact dermatitis in certain rubber factories, including one in Sweden (Fregert (4)). Batches of this chemical from 6 manufacturers showed several shades of colour. No difference in chemical composition could be found by thin layer chromatography. Nevertheless, when the batch of dithiodimorpholine was changed in the Swedish factory, 4 workers sensitized to the original batch were able to continue in their jobs without any further recurrence of their dermatitis. More sensitive analytical techniques might be capable of demonstrating a chemical difference between batches of dithiodimorpholine.

Work Process

Because predictive testing has its limitations, other levels of prevention have important roles to play. Mechanization or automation of the work process can theoretically eliminate the hazard of a known skin irritant or sensitizer. A tape-controlled machine tool, for example, might be able to use safely a metalworking fluid that on a manually-operated machine tool could cause skin irritation. Skin problems can still arise, however, from processes which might be regarded as fully automatic, because of handling stages at the beginning or end of the process, routine cleaning procedures or the necessity for intervention following breakdowns.

When complete automation is not possible, it may be possible to segregate a stage of the work process with a particularly high skin hazard. Where this too is not practicable, a reduction in the concentration or duration of a toxic substance in contact with the skin may be preventive. Biological variability among the human population makes it difficult to lay down entirely safe levels of skin irritants and sensitizers, but laboratory animal testing and previous experience can give some guidance. Formaldehyde, for example, rarely appears to sensitize at the very low levels at which it is present as a preservative in products such as shampoos or industrial hand cleansers, whereas repeated exposures to 10% formaldehyde solutions would be expected to carry a significant rate of sensitization.

Industrial Hygiene

The safest possible process design can founder on the rocks of poor industrial hygiene. Lack of regular cleaning and maintenance programmes can result in entirely unnecessary outbreaks of occupational dermatoses. In addition, if employees find themselves working in poor conditions, it is extremely unlikely that any programme of protective clothing, personal hygiene and good working method will be given their fullest cooperation.

Protective Clothing

If potentially toxic levels of chemicals genuinely cannot be avoided, then protective clothing can play its part. It is striking, however, how often clothing is not truly protective because it in fact allows penetration. It has been demonstrated, for example, how rapidly certain organic solvents can penetrate rubber gloves (Sansone and Tewari (5)). Rubber gloves can also be rapidly penetrated by sensitizers. Methyl methacrylate in bone cement was able to sensitize orthopaedic surgeons carrying out joint-replacement surgery who, of course, wear surgical gloves throughout the procedure.

Working Method

Working method and protective clothing are closely interrelated. If protective gloves are to be taken on and off during work, it is essential that contamination of the hands from touching the outside of the gloves is avoided. It is widely acknowledged throughout industry that working method alone is sometimes able to prevent dermatitis. I recently observed two women carrying out the same manual agitation of a small jig in a tank of mineral acid solution. In one case there was considerable splashing of solution onto the gloved hand, in the other there was virtually none. Efficiency of agitation was identical. The difference resided in the relative speed and angles of arm, wrist and hand movements.

Personnel Selection

Personnel selection has a limited role at present in the prevention of occupational dermatoses. This is simply because of our current lack of knowledge about how to detect individual predisposition to the development of dermatoses. The presence or past history of eczema or dermatitis in an individual may make it statistically more likely that future exposure to an irritant will result in dermatitis, but there is a considerable degree of uncertainty when applying this generalization to any individual. We have no way at all of predicting whether or not a prospective employee will be more or less likely than average to become sensitized to chemicals during his employment. Although many tests have been proposed for determining the susceptibility of skin contact irritants, none has yet proved sufficiently reliable and practical to carry out to warrant widespread adoption. Pre-employment medical screening nevertheless allows at least the current condition of the skin to be established and the process of education, discussion and supervision of skin care to be initiated.

Personal Hygiene

Personal hygiene can never be disregarded as a preventive measure though its contribution can sometimes be exaggerated. It has a particularly important part to play in the prevention of contact irritation and conditions such as oil folliculitis. Sometimes skin cleansers themselves can be aggressive to the skin and skin washing can then promote rather than prevent dermatitis. Barrier creams can make the removal of potential irritants from the skin easier so that less aggressive skin cleansing is necessary. Barrier creams are much less effective in the prevention of allergic contact dermatitis.

Personal hygiene is not simply a matter of providing good washing facilities, though this is of course the first essential. A strong commitment must exist on the part of the management to the proper maintenance of facilities and a strong commitment on the part of the employees to their proper use. Personal hygiene measures need to be explained to new employees and then discussed again after they have been doing their work for a few weeks and can better appreciate their relevance.

Education, Discussion and Supervision

Education, discussion and supervision are the keys to the success of all programmes of personal hygiene, protective clothing and good working method. The best preventive programmes, however, are those that do not depend too heavily on these latter measures. The long term effectiveness of a preventive programme depends upon the measures discussed earlier. The chemicals used in industry should be tested for potential skin toxicity and processes designed so as to minimise the hazard from potential toxic effects so discovered. The effectiveness of prevention must be monitored by the early detection and accurate diagnosis of occupational dermatoses. This means a great deal of hard work, but the current cost to industry of occupational dermatoses in economic and human terms justifies a considerable investment of resources in their prevention.

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Figure 1 Para-compounds: all contact sensitizers