# Occupational Skin Disease

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# Foreword

This book contains the most up-to-date material available in any text on occupational skin disease. Not only does it include complete descriptions of the dermatoses affecting such workers as butchers and bakers, it also includes those affecting workers involved in the manufacture of the most recent technological products.

The information in this expertly organized book will guide dermatologists, allergists, and industrial physicians in the successful management of occupational dermatoses. The practical advice given for prophylaxis will enable workers, preventive medicine and rehabilitation experts, industrial nurses and hygienists, and plant superintendents to plan and implement methods that should help lower the incidence of such disease. Others concerned with industry, such as lawyers, insurance carriers, safety engineers, compensation court referees, and labor and union leaders, should consult this text for information that will aid them in their decision making about occupational dermatoses.

Currently there is an urgent need for dermatologists to become more directly involved in the battle against occupational skin disease. Although dermatitis is one of the most prevalent of occupational diseases, the field heretofore has been neglected and is now in its infancy. Thus the opportunities for young physicians are numerous.

I feel certain this book will inspire many of these dermatologists to care, and I know of no better way for dedicated young physicians to begin to grasp these opportunities than to study this book. Such study is surely the first essential step in acquiring expertise in occupational dermatology.

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# Preface

I wrote this book in the hope of providing the reader with as complete and concise a picture of occupational skin disease as is possible with today's information. Because contact dermatitis comprises approximately 95 percent of occupational skin disease, I have allotted it the most space. The list of allergens in Appendix C of Chapter 8 is one of the most comprehensive to be found anywhere; others are undoubtedly present in the work environment, but discovering them and estimating their proper patch test concentrations will require future investigation. The patch test concentrations recommended are generally accepted; a few are debatable and may provoke some controversy. I have attempted, however, to give the lowest concentrations known to elicit allergic reactions without causing the irritant reactions so easily misinterpreted as allergic, especially by those inexperienced in patch testing.

The final section of the book describes work processes in a large number of occupations, along with their common irritants and allergens. This information will be of great practical value to dermatologists and allergists, and will, I hope, stimulate them to use the patch test more frequently and effectively in their daily practices.

Most of the chapters have been reviewed by one or more of my colleagues, themselves prominent in this or related fields. I wish to acknowledge with gratitude the contributions of my contributing authors, Drs. Geo von Krogh, Howard I. Maibach, Robert L. Baran, and Charles W. Whitmore. I am also greatly indebted to the following for their help with individual chapters: Drs. Alexander A. Fisher, Marion B. Sulzberger, Howard I. Maibach, Joseph LaDou, Jud Scholz, Desmond Burrows, Darrell S. Wilkinson, James S. Taylor, James E. Weaver, Frances J. Storrs, Edward A. Emmett, Steven R. Cohen, James R. Nethercott, C. G. Toby Mathias, John C. Mitchell, and Martin Goldner as well as Herbert Stanek, LL.B, who reviewed the chapter on medicolegal aspects.

I also wish to thank Gail Mowen for the considerable care, time, and attention she gave to the word processing, and the personnel of the Lane Medical Library at Stanford University for their help and patience. The editorial staff of Grune & Stratton deserves special commendation for performing a gigantic task with diligence and thoroughness, meticulously checking everything in every way possible for accuracy, consistency, and style. My thanks to Leonard Winograd of the Department of Dermatology, Stanford Medical Center, who prepared the photographs with such care.

Finally, to Dr. Eugene Farber, other members of the Stanford dermatology faculty, and especially to the dermatology residents at Stanford, my heartfelt thanks for their encouragement and support.

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# Contents

CHAPTER 1

# Contact Dermatitis Due to Irritation and Allergic Sensitization

The skin's basic and vital function is protection, which it achieves through a variety of properties. Having remarkable tensile strength and resiliency, the skin provides a defense against physical injury, especially shearing stress. The armor of keratin acts as a barrier against irritating and allergenic chemicals, systemic poisons, and a multitude of microorganisms, while the presence of melanin furnishes protection against the damaging effects of ultraviolet light. The unremitting upward movement of the cellular epidermis provides continual renewal and at the same time discourages the colonization of bacteria and fungi. Highly integrated, complex biochemical functions go on every minute to make these activities function smoothly and efficiently.

Yet in spite of the ability of human skin to withstand the assaults of a frequently hostile environment, the skin is still the most commonly injured organ in industry today. According to 1978 statistics of the U.S. Department of Labor, skin disorders comprise more than 45 percent of all occupationally related diseases, annually affecting approximately one worker per thousand in the private sector. Including numerous cases that are never reported, the total number of workers with occupational skin disease must exceed several hundred thousand each year, resulting in much suffering and great financial loss to workers and employers alike.

Most occupational skin disease results from contact with a chemical substance, of which more than 33,000 are in common use in the United States. The 1978 *Registry of Toxic Effects of Chemical Substances* categorizes 2674 of these as irritants; brief and incomplete toxicity data are available on about 1000 (NIOSH, 1978). Unfortunately, useful information regarding cutaneous toxicity is lacking for most of them.

The cutaneous reactions to these chemicals are almost as varied as the chemicals themselves. All are irritants to some degree, yet relatively few are known to be contact allergens. Of the 50 most important chemicals in U.S. production today (Table 1-1), each has some potential for skin irritancy; but only three—formaldehyde and, rarely, ethyl and isopropyl alcohol—are also contact allergens. It is therefore easy to understand why irritant contact dermatitis comprises more than 75 percent of all occupational skin disease (Schwartz et al., 1957). A knowledge of the mechanisms of skin irritation and allergic sensitization is obviously very important to physicians.

TABLE 1-1 Top 50 Chemicals (Production Volume) Produced in the United States in 1980

| | | |
|---|---|---|
| 1. Sulfuric acid | 18. Ethylbenzene | 35. Sodium sulfate |
| 2. Ammonia | 19. Carbon dioxide | 36. Phenol |
| 3. Lime | 20. Methanol | 37. Aluminum sulfate |
| 4. Oxygen | 21. Styrene | 38. Acetone |
| 5. Nitrogen | 22. Vinyl chloride | 39. Cyclohexane |
| 6. Ethylene | 23. Xylene | 40. Calcium chloride |
| 7. Sodium hydroxide | 24. Terephthalic acid | 41. Vinyl acetate |
| 8. Chlorine | 25. Formaldehyde* | 42. Acrylonitrile |
| 9. Phosphoric acid | 26. Hydrochloric acid | 43. Isopropyl alcohol* |
| 10. Ammonium nitrate | 27. Ethylene oxide | 44. Propylene oxide |
| 11. Nitric acid | 28. Ethylene glycol | 45. Sodium silicate |
| 12. Sodium carbonate | 29. Ammonium sulfate | 46. Acetic anhydride |
| 13. Urea | 30. *p*-Xylene | 47. Sodium tripolyphosphate |
| 14. Propylene | 31. Cumene | 48. Titanium dioxide |
| 15. Toluene | 32. Butadiene | 49. Ethanol* |
| 16. Benzene | 33. Acetic acid | 50. Adipic acid |
| 17. Ethylene dichloride | 34. Carbon black | |

From Chemical and Engineering News' top fifty chemical products. June 8, 1981, p 33.
*Possible contact allergen.

## Irritation

### SKIN IRRITANTS

It is difficult to devise an all-inclusive definition of an irritant because the term comprises so many different substances with a wide range of reactions. Actually any substance can be an irritant under certain circumstances, as Kligman (1980) has emphasized. For years an irritant was considered to be any substance that causes irritation on any person's skin, provided the concentration and duration of contact are sufficient. Yet numerous substances cause irritation on only a small percentage of persons, who react because of individual local skin factors present at the site and time of contact. Examples are certain oils, alcohols, and glycols, especially propylene glycol (Warshaw and Herrmann, 1952). At the opposite end of the spectrum are such powerful irritants as sodium hydroxide and hydrofluoric acid, which in 100% concentration immediately produce a third-degree burn on anyone's skin, sometimes with fatal results.

The diagnosis of irritation is often made by exclusion of other possible causes, and therefore a definition of an irritant must be rather broad: a skin irritant is any substance that, acting directly, damages the skin at the site of application through a nonimmune mechanism. This definition excludes the possibility of participation by allergic sensitization, although irritation predisposes the skin to development of contact allergic sensitization.

While most irritants are chemicals, morphologically similar reactions can be produced by certain microorganisms, ultraviolet and ionizing radiation, and thermal injury. Because of this, physicians sometimes experience difficulty differentiating reactions due to chemical exposure from fungal infections, miliaria, photosensitivity reactions, and others.

Skin irritants range from strong to mild. *Strong irritants* are intrinsically damaging, corrosive substances that rapidly injure anyone's skin immediately following contact. Examples are strong alkalies (Fig. 1-1) and acids, certain metallic substances and their salts, and many organic compounds. Although the response varies somewhat among different strong irritants, the reaction to irritants of the same class is similar, and the chief factors of importance, in addition to the intrinsic nature of the chemical, are the concentration and duration of contact. Thus the substance and its ability to damage the skin are of primary concern, not the individual, because almost everybody responds with a similar reaction (Rostenberg, 1957).

*Mild (or moderate) irritants* are less toxic substances that in normal usage cause irritation in only a small percentage of exposed persons. Following repeated or prolonged contact, how-

ever, every individual will develop a reaction. Examples are detergents and soaps, and a large number of organic compounds including many organic solvents (Figs. 1-2A and B).

The chief factors contributing to irritation are shown in Table 1-2. Perhaps in no other disease is evaluation of the contributing factors more important than in dermatitis due to contact with irritants. Early and exact diagnosis is important because therapeutic success depends on avoiding contact with the irritant and eliminating, if possible, the contributing factors. In the case of mild irritants, however, workers can resume contact once the skin is completely healed, unless there are other factors present that prohibit continued exposure, such as atopic dermatitis.

The most important environmental factors leading to the development of irritant dermatitis are

- *Low relative humidity*, which may occur in the general workroom (Rycroft and Smith, 1980), or in a localized space such as a workbox into which the worker inserts hands and arms only, as while polishing crystals (Malten, 1981)
- *Friction*, such as occurs while operating grinding machines and other equipment
- *Occlusion*, especially of clothing contaminated with solvents and other irritants
- *Lacerations*, even minor ones, into which irritant materials can enter
- *Excessive environmental heat*, inducing sweating, which in turn brings irritant substances (e.g., cement dust) into solution

The major genetic condition leading to occupational irritant dermatitis is atopy.

It is common for physicians to make a diagnosis of "allergy" when treating workers with irritant dermatitis. This diagnosis frequently results in unnecessary job changes for the employees as well as unwarranted expense for employers and insurance companies. When an outbreak of dermatitis occurs in a factory and many workers are involved, it usually, but not always, signifies that an irritant rather than an allergic sensitizer is responsible. It frequently means that adequate protective measures are not being used and/or that the chemical substance, especially if recently introduced, possesses unusual or unrecognized irritant properties. Only occasionally does the outbreak indicate the existence of a potent allergic sensitizer.

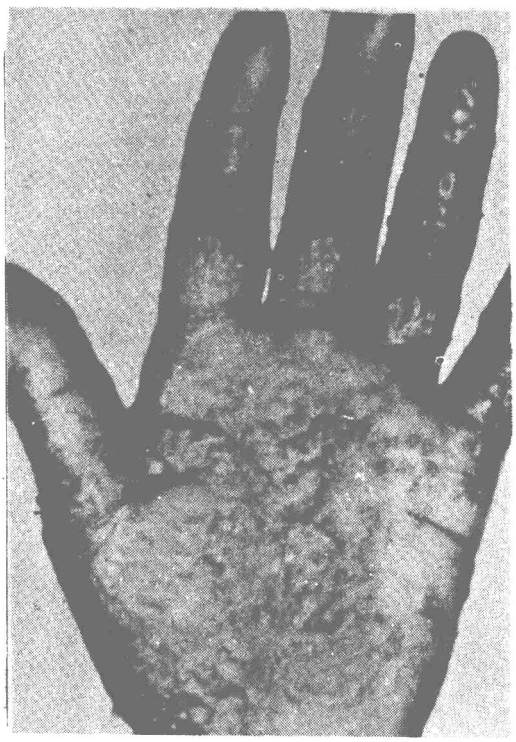

FIGURE 1-1 A chemical burn due to sodium hydroxide in an electroplater, showing marked vesiculation with dissolution of surface keratin.

## CLINICAL APPEARANCE

The clinical appearance of dermatitis due to irritation varies considerably, ranging from slight erythema at the point of contact to large bullae with necrosis and ulceration (Fig. 1-3). Bullous, destructive, ulcerative lesions suggest a sudden and often accidental contact with a strong primary irritant such as a caustic alkali or a strong acid (Malten, 1981). Such lesions immediately follow contact with the irritant, and the relationship is obvious.

Most mild irritants, however, produce a clinical picture described as *eczema*, a term derived from a Greek word meaning "boiling over." An eczematous dermatitis shows vesiculation and oozing of the skin surface sometime during its development. This clinical appearance is not

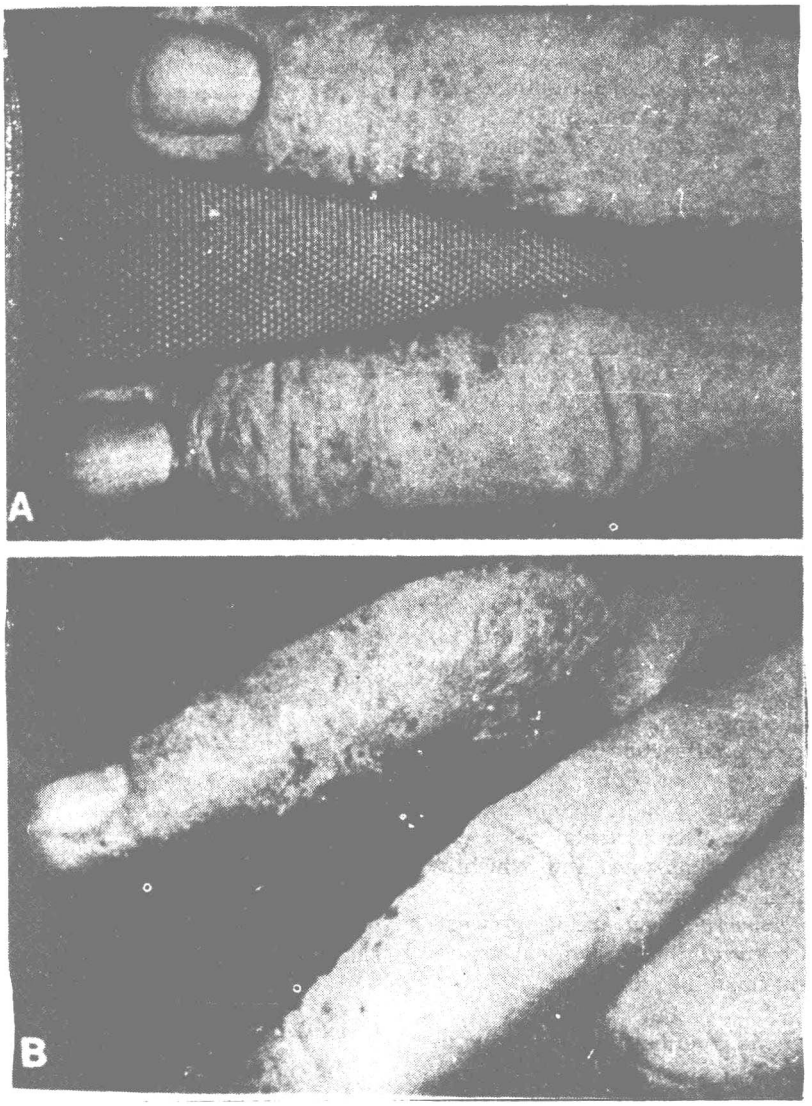


FIGURE 1-2 Irritant dermatitis due to mild irritants in a male hairdresser. Soaps, detergents, and the thioglycolates are the chief irritants in this occupation.

much different from that of many other skin diseases, and differentiation from allergic sensitization is generally impossible from gross inspection alone.

The earliest change produced by mild irritants is erythema, which is usually limited to the area of contact. If irritation continues, edema develops, followed by the appearance of various-sized vesicles and papules on the reddened area. After several days crusts and scales form, and if contact with the irritant ceases, the process is completed in one to three weeks. Pruritus is often associated to a variable degree but is usually not as marked as in early stages of dermatitis due to allergic sensitization. Stinging and burning may be present especially with moderate and strong irritants.

In subacute and chronic irritant dermatitis,

TABLE 1-2 Factors Contributing to the Development of Cutaneous Irritation

- Factors related to the substance
  - Chemical nature of the substance
    - pH
    - Solubility in water and fats
    - Detergent action
  - Physical state
    - Gas
    - Volatile liquid
    - Heavy liquid
    - Semisolid
    - Solid
  - Concentration
    - Amount
    - Contact with skin
- Host factors
  - Surface area affected
  - Region of skin
  - Length of exposure
  - Presence or absence of occlusion
  - Dryness
  - Sweating
- Host factors (continued)
  - Pigmentation
  - Presence of hair
  - Sebaceous activity
  - Concurrent and preexisting skin disease
  - Pruritogenic threshold
- General host factors
  - Age
  - Sex
  - Race
  - Genetic background
- Environmental factors
  - Temperature
    - Heat
    - Cold
  - Humidity and moisture
  - Friction
  - Pressure
  - Occlusion
  - Lacerations

the skin becomes thickened after several days or weeks of continuing mild irritation; has a firm, "infiltrated" feel; and later shows lichenification with spotty areas of hyperpigmentation. Painful fissuring is one of the most uncomfortable aspects of subacute dermatitis (Fig. 1-4).

The affected sites are those areas actually exposed. In most occupations the hands and forearms have the greatest contact with irritants, particularly the dorsal and lateral aspects of the hands and fingers. The palms and soles are partially protected by the presence of a thick stratum corneum.

An irritant in clothing produces dermatitis at the region of greatest contact, such as the anterior thighs, the upper back, axillary areas, and feet. Dusts collect in flexural areas, under the collar and belt, and at the tops of the shoes.

The scalp is rarely involved with contact dermatitis, not just because of the protective mantle of hair, for even a hairless scalp is somewhat resistant to irritants and allergens, for unknown reasons.

The male genitalia are a common site of involvement from transfer of the irritant by the hands. Contact dermatitis tends to appear in areas not always thoroughly cleaned, such as

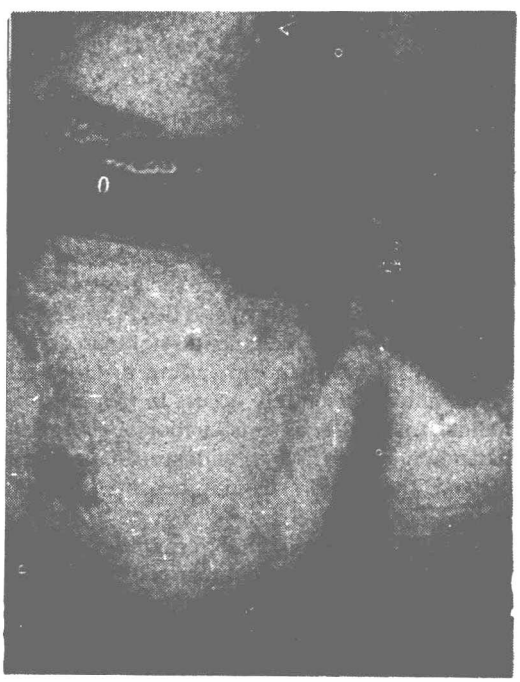

FIGURE 1-3 A gardener accidently sprayed himself with a pesticide/kerosene combination, soaking his clothing, which he failed to remove for several hours. The blistering was so deep that third-degree burn scars remained.

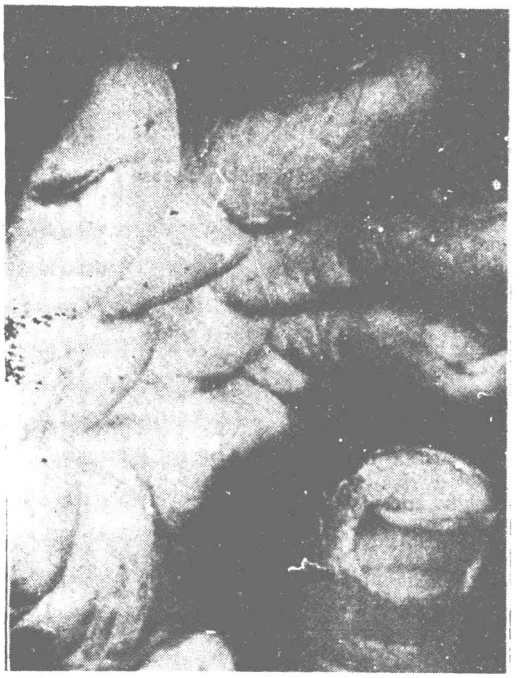

FIGURE 1-4 Fissuring in acute and subacute irritant dermatitis is painful and unfortunately common.

under rings, between fingers, and in intertriginous areas.

The initial appearance of occupational dermatitis is always at the site of contact with the irritant, and disappearance usually occurs following its removal, provided the treatment is not irritating or sensitizing and home and other nonoccupational contactants do not increase or maintain the irritation. Spreading to other regions of the body rarely occurs in irritant dermatitis, unless allergic sensitization or autoeczematization ensues. When widespread areas are affected, the entire skin may become hyperirritable (Björnberg, 1968).

Each recurrence of irritant dermatitis requires approximately the same number of contacts and identical, or almost identical, conditions as were necessary for the development of the original dermatitis, provided the other conditions remain the same. This is an important point in differentiation from dermatitis due to allergic sensitization, which requires single and often trivial exposure to the allergen for the disease to recur in a fully sensitized person.

Occasionally certain features are present that strongly suggest the nature of the irritant. Some of these are shown in Table 1-3. Changes of skin color as markers of occupational exposure are listed in Table 1-4. Heavy-metal-induced hyperpigmentation may present especially characteristic features (Granstein and Sober, 1981).

## HISTOLOGIC APPEARANCE

Experimental studies have shown the following changes to be fairly characteristic of strong irritant reactions.

- There is epidermal necrosis with separation of the epidermis from the dermis and vesicle formation.
- Predominantly polymorphonuclear leukocytes are present in the vesicle fluid.
- The intraepidermal vesicles and bullae are usually located high in the epidermis and contain a mixture of neutrophils and lymphocytes.

With mild and most moderate irritants it is almost impossible to differentiate irritant reactions from those due to allergic sensitization by histologic means alone. Bandmann (1962) studied cutaneous reactions to numerous irritants and found many differences in the histologic appearance of the dermatitis caused by various mild and moderate irritants. Some reactions were follicular, while others were petechial, and some could easily be falsely interpreted as allergic reactions. Björnberg (1968) also stressed the difficulty in differentiation between moderate and mild irritants.

Using the electron microscope, Nagoo et al. (1972) demonstrated the irritant effects of sodium hydroxide and hydrochloric acid on human epidermis. Specimens were obtained from the forearms of seven volunteers, 15–180 minutes after application of 1N sodium hydroxide or 1N hydrochloric acid. The two irritants evoked different changes in the stratum corneum: the sodium hydroxide produced dissolution of the contents of horny cells, whereas the hydrochloric acid was usually associated with preservation of these cells, although their cytoplasm showed a porous pattern. Penetration of the living epidermis was more rapid with sodium hydroxide, with disappearance of tonofilament-desmosome complexes in sodium hydroxide–treated sites, but not with hydrochloric acid.

TABLE 1-3 Clinical Features that May Suggest the Etiology of Irritant Contact Dermatitis

Ulcerations
- Strong acids, especially chromic (Samitz, 1955), hydrofluoric, nitric, hydrochloric, sulfuric
- Strong alkalies, especially calcium oxide (Zackheim and Pinkus, 1957)
- Calcium hydroxide, sodium hydroxide, sodium metasilicate, sodium silicate, potassium cyanide, trisodium phosphate
- Salts, especially arsenic trioxide (Birmingham et al., 1965), dichromates
- Solvents, especially acrylonitrile, carbon bisulfide
- Gases, especially ethylene oxide, acrylonitrile (Radimer et al., 1974)

Folliculitis and acneiform
- Arsenic trioxide
- Glass fibers
- Oils and greases
- Tar
- Asphalt
- Chlorinated naphthalenes
- Polyhalogenated biphenyls and others (see Chapter 4)

Miliaria
- Occlusive clothing and dressing
- Adhesive tape
- Ultraviolet
- Infrared
- Aluminum chloride (Shelley and Horvath, 1960)

Pigmentary alterations
- Hyperpigmentation
  - Any irritant or allergen, especially phototoxic agents such as psoralens, tar, asphalt, phototoxic plants, and others
- Hyperpigmentation (continued)
  - Metals: inorganic arsenic (systemic), silver, gold, bismuth, mercury
  - Radiation: ultraviolet, infrared, microwave, ionizing
- Hypopigmentation
  - *p-tert*-Amylphenol (Kahn, 1970)
  - *p-tert*-Butylphenol (Gellin et al., 1970; Kahn, 1970)
  - Hydroquinone (Oettel, 1936)
  - Monobenzyl ether of hydroquinone (Oliver et al., 1939)
  - Monomethyl ether of hydroquinone (Brun, 1967)
  - *p-tert*-Catechol (Gellin et al., 1970)
  - *p*-Cresol (Shelley, 1974)
  - 3-Hydroxyanisole (Brun, 1967)
  - Butylated hydroxyanisole (Vollum, 1971)*
  - 1-Isopropyl-3,4-catechol (Bleehen, 1968)
  - 1-*tert*-Butyl-3,4-catechol (Bleehen, 1968)
  - 4-Hydroxypropriophenone (Bleehen, 1968)

Alopecia
- Borax (Tan, 1970)
- Chloroprene dimers (Irish, 1963)

Urticaria
- Chemicals, cosmetics, animals, foods, plants, textiles, woods (see Chapter 3)

Granulomas
- Keratin (Meneghini and Gianotti, 1964)
- Silica (Epstein, 1950)
- Beryllium (Grier et al., 1948)
- Talc
- Cotton
- Bacteria
- Fungi
- Parasites

*Questionable (see text)

They suggested that their results could establish a baseline for comparison with other concentrations of acids and alkalies and thus lead to a better understanding of the pathology of irritation.

Lupulescu et al. (1973), using the same methods as Nagoo et al., studied the effects of kerosene and acetone. Kerosene had greater biologic activity as a skin irritant than acetone, causing cytolysis of epidermal cells, while acetone induced a peculiar and less destructive type of vacuolization. Since both are lipid solvents, it appears that their damaging effects resulted mainly from the removal of lipid components present within the stratum corneum. Seventy-two hours after application, the epidermal pattern was restored to normal. These findings are similar to those seen clinically.

## HARDENING DUE TO IRRITATION

After daily exposure to irritants the normal skin of most workers becomes tough and resistant, permitting continued contact with the substance without further irritation. This process is known as *hardening:* a purely individual adaptive phenomenon that occurs following contact with irritants (McOsker and Beck, 1967).

TABLE 1-4 Changes of Skin Color as Markers of Occupational Exposure

*Orange*
- Tetryl (trinitrophenylmethylnitramine) in explosives workers
- Chlorine gas in chemical workers, bleachers, laundry workers, swimming pool maintenance workers, and others
- Phenothiazine in insecticide workers, dye and pharmaceutical workers, nurses

*Yellow*
- Picric acid and picrates in explosives and dye workers (Schwartz, 1944)
- Dinobuton (2-(1-methyl-2-propyl)-4,6-dinitrophenyl isopropylcarbonate), Acrex, Dessin, Dinofen, and others—acaricides and fungicides (Wahlberg, 1974)—in agricultural workers
- 4,4'-Methylenedianiline—catalyst for epoxy and urethane resins (Cohen, 1981)—in plastics workers
- Bichromate in electroplaters, leather tanners, lithographers, and others
- Fluorescein dye in machinists and metal flaw detectors
- Glutaraldehyde in nurses, dental personnel, and hemodialysis technicians
- Sodium nitrite (etching electrolyte) in machinists (Fregert, 1980)
- Nitric acid

*Green*
- Copper dust in electrical workers, machinists, and copper smelters

*Blue*
- Silver nitrate in photographers (Buckley et al., 1965)
- Sulfadiazine silver (Silvadene) in pharmaceutical workers and nurses in burn units (Pariser, 1978)
- Oxalic acid in automobile radiator cleaners, dye makers and dryers, metal cleaners, and others
- Bismuth, gold, and lead salts, which induce a blue-gray pigmentation

*Brown*
- Chrysarobin and anthralin in pharmacists and nurses
- Arsenic, which induces a bronze pigmentation on the trunk
- *p*-Phenylenediamine in dye manufacturers and photograph developers (color usually)
- Permanganates in bleach and dye makers, water purifiers, and paper pulp bleachers
- Phenothiazines in agricultural workers, veterinarians, and nurses

*Black*
- Osmium trioxide in histology technicians, incandescent lamp makers, organic chemical synthesizers, and platinum hardeners
- Mercury in cosmetologists and veterinarians produces a slate gray pigmentation that is more pronounced in skin folds
- Resin of *Toxicodendron radicans*, which produces a black lacquerlike deposit on the skin (Guin, 1980; Mallory et al., 1982)

Repeated daily contact is necessary, and even short periods away from work effect some decrease in resistance. The protection is entirely local, occurring only at the site of contact.

Clinically, hardened skin is somewhat coarse and thickened, usually showing slight scaling and increased pigmentation. On histologic examination the most striking feature is an increased thickness of the stratum corneum without significant change in the underlying epidermis.

Suskind (1967) believes that hardening is not only an adaptive phenomenon due to thickening of the stratum corneum but also an enzymatic "adaptation" of the epidermal cells and the underlying dermal vasculature, without the necessary development of an increased horny layer.

The hardening phenomenon is common and widely accepted among industrial personnel. Physicians usually see hardening after it has happened. Deliberate attempts to induce it are inadvisable because one cannot predict who will successfully develop it and because allergic sensitization can result from such attempts.

## OCCUPATIONAL MARKS

Occupational marks represent the effects of a particular occupation on a worker's skin. Usually considered to be calluses or corns that develop in locations subjected to repeated friction, pressure, or other trauma, they include discolorations, telangiectases, tattoos, odors, deformities, and other changes.

At one time such marks were very common among workers and served to clearly indicate many occupations. Today, with increasing automation, less frequent manual operation of tools, better protective clothing, and a shorter work

FIGURE 1-5 Hydrofluoric acid readily penetrates tissue, especially in the periungual regions, and if not treated promptly, may cause necrosis of bone and loss of the distal phalanx. (Courtesy of George Wilson, M.D.)

week, occupational marks have become less frequent, almost disappearing from many industries. The interested reader is referred to Ronchese's valuable book (1948).

Occupational marks must be distinguished from so-called pseudo-occupational marks, such as knuckle pads, knuckle biting, nail biting, cuticle pulling, and trichotillomania.

## SPECIAL TYPES OF IRRITATION

### *Hydrofluoric Acid Burns*

Hydrofluoric acid (HF) is one of the most caustic and corrosive of inorganic acids (Wetherhold and Shepherd, 1965). The fluoride ion readily penetrates the skin, especially through small abrasions and cracks or under the proximal nail fold to the deeper tissues, causing necrosis of soft tissues and rapid decalcification of bone. The acid is hygroscopic and does not induce blistering, as do other acids. Intense, excruciating pain accompanies the tissue destruction and is often difficult to control with anesthetic agents (Dibbell et al., 1970).

With concentrations of acid above 70% the symptoms of irritation are immediately noted, and the burn is usually a third-degree destruction of tissue. With concentrations below 30%, however, there may be a latent period of up to 24 hours before the patient becomes aware of the effects. Because of the insidious nature of HF, the risk of burns is very great. The acid readily diffuses through pinholes in rubber gloves and often causes delayed burns around the nail plate that heal slowly and may lead to loss of the nail. Untreated, HF burns in this area can result in loss of the distal phalanx (Fig. 1-5).

When large areas of the body are burned, there is a substantial risk of systemic poisoning. But even when relatively small areas are burned, death can occur, not only from inhalation of HF fumes but also from the burns themselves (Dieffenbacher and Thompson, 1962; Tepperman, 1980). Because percutaneous absorption is immediate and nearly complete following massive exposure, prompt hospitalization is necessary when large areas are burned. One of the chief causes of death is reduction of serum calcium, which occurs rapidly because of the affinity of the fluoride ion for body calcium. (Treatment of HF burns is discussed in Chapter 9.)

NIOSH estimates that 350,000 workers are potentially exposed to fluorides. The most common uses for HF are given in Table 1-5.