Clinical Algorithms

Dermatitis

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Dermatitis may be caused by endogenous factors as in atopic dermatitis or by exogenous factors as in contact dermatitis. Both types of factors may coexist in the same patient, and determination of aetiological factors is often difficult as the skin changes associated with atopic dermatitis, allergic contact dermatitis, irritant dermatitis, and fungal infections can be very similar.

Allergic contact dermatitis usually causes itching and is characterised by erythematous papules or vesicles. Oedema in the upper dermis and epidermis may cause large vesicles or small bullae resulting in weeping and, later, crusting. If the dermatitis becomes chronic the skin will become dry and scaly with thickening and exaggerated markings and may crack. The distribution of the dermatitis may provide important clues, with areas of dermatitis corresponding to areas in contact with the sensitising agent. Allergic contact dermatitis tends to be more severe and vesicular than irritant dermatitis and is more likely to spread to sites distant from the primary site.

Irritant dermatitis is commoner than the allergic type and results from repeated contact with irritants such as oils, alkalis, soaps, solvents, and detergent. It tends to begin with dryness and cracking of the skin and is limited to areas in contact with the irritant. Sites commonly affected are the backs of the hands, the webs of the fingers, and the forearms. Soap and detergent may accumulate under a ring and produce an irritant reaction mimicking allergy to metal.

The patient's history is important in the search for likely sensitisers or irritants. The primary site affected should be established together with the time course and spread of the dermatitis. Exacerbations may show a temporal relation with specific activities or tasks at work or at home or may follow contact with substances handled or applied —such as, toiletries, cosmetics, or medicaments. More than one substance may be implicated, and a knowledge of common sensitisers is required (table). In addition to these common sensitisers, plants, particularly *Primula obconica*, may cause allergic reactions. Topical antihistamines bought without prescription and phosphorus sesquisulphide, which is present in "strike anywhere" matches, also deserve consideration as possible sensitisers. If the cause of suspected allergic contact dermatitis is not clear or the dermatitis persists despite simple measures, patch tests to a standard series of allergens (table) together with selected suspect allergens should be performed.[1] These cannot be done during an acute exacerbation or if the dermatitis is extensive. Patch testing with irritants is not valuable and is not carried out. It is helpful to explain to patients that a sensitiser could be something previously handled for years with impunity and to encourage mindfulness of anything coming into contact with the affected skin.

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Atopic dermatitis forms a distinct group. It develops predominantly in association with asthma, hay fever, or a family history of atopic disease. It often begins in infancy but rarely before the age of 3 months. The itchy papules, which first develop on the face, arms, and legs are soon excoriated and often become secondarily infected. After infancy the distribution changes to affect the flexures of the elbows, knees, wrists, and ankles, and lichenification commonly develops. Atopic dermatitis tends to improve throughout childhood with over half of the patients clear by the age of 13, but an appreciable number of cases continue into adulthood. Patients who have had atopic dermatitis are more likely to develop an irritant dermatitis in early adulthood when exposed to housework or occupations such as hairdressing. Atopic dermatitis can develop later in childhood, but if itchy dermatitis develops after the second year signs of scabies should be sought. A slowly spreading asymmetrical dermatitis with scaling should arouse suspicion of fungal infection.

After elimination of any remediable factors in the aetiology of the dermatitis initial treatment depends on whether the dermatitis is acute or chronic. (The algorithm indicates where treatment deviates from the following guidelines.) In the acute, wet, exudative stage soaks of normal saline or 1/4-10 000 solution of potassium permanganate are used. If infection is suggested by local heat, pus, weeping, a yellow crust, or fever systemic antibiotics are given. Infection is usually caused by *Staphylococcus aureus* or *Streptococcus haemolyticus* and should be confirmed by culture. Suitable antibiotics would be flucloxacillin or erythromycin. As an acute episode subsides a regimen similar to that used for chronic dermatitis can be followed.

In chronic dermatitis excessive bathing should be avoided and use of irritants such as soap minimised. Bland emollients are the mainstay of treatment together with the judicious use of the weakest steroid preparation that will control the dermatitis. Potent steroids should be used only rarely in infants and young children and never on the face. More potent steroids may be necessary to produce an effect on the hands and feet; in resistant cases penetration of the steroid can be greatly enhanced by short term occlusion with polythene. Systemic antihistamines are of value in reducing itching and scratching and are often given at night. Sedative antihistamines are preferable as sedation may be their mode of action.[2] For chronic lichenified eczema topical tar preparations are helpful and may be used together with occlusive dressings to prevent scratching. These combinations may be messy and may cause sensitisation; but these disadvantages need to be weighed against the advantages of reducing secondary infection. Traditionally creams have been used in the exudative phase and ointments in the dry phase, but there is no good evidence to support this approach. Creams are more likely to contain preservatives and are more easily washed off the skin. Treatment with stanozolol may be beneficial in patients with stasis dermatitis and lipodermatosclerosis.[3] The benefits of preparations combining a topical steroid with an antibiotic or antiseptic are debatable.

Some patients with atopic dermatitis will not respond to these treatments, and for them many other modes of treatment are now being evaluated with some evidence for their effectiveness. These include evening primrose oil,[4] sodium cromoglycate,[5] and oral

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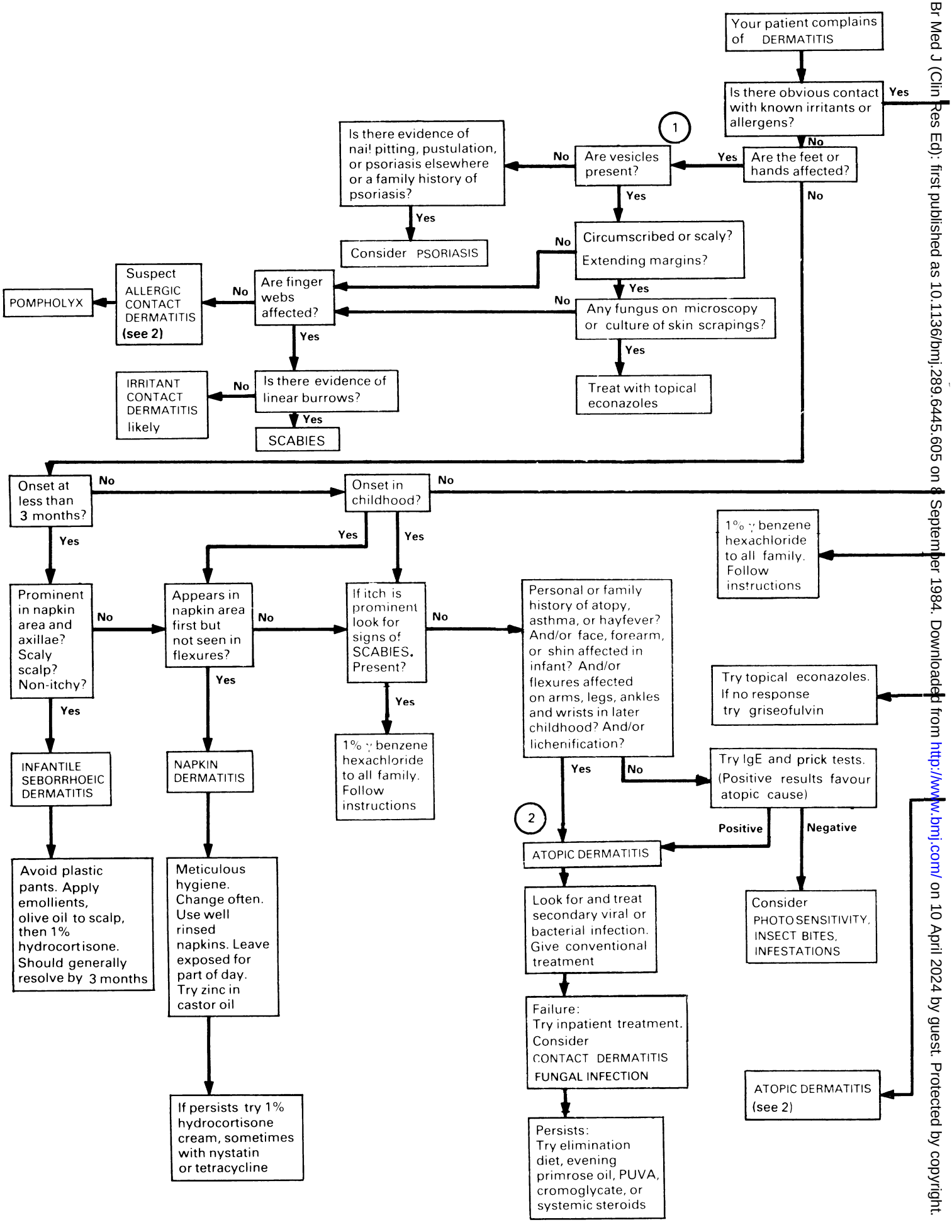

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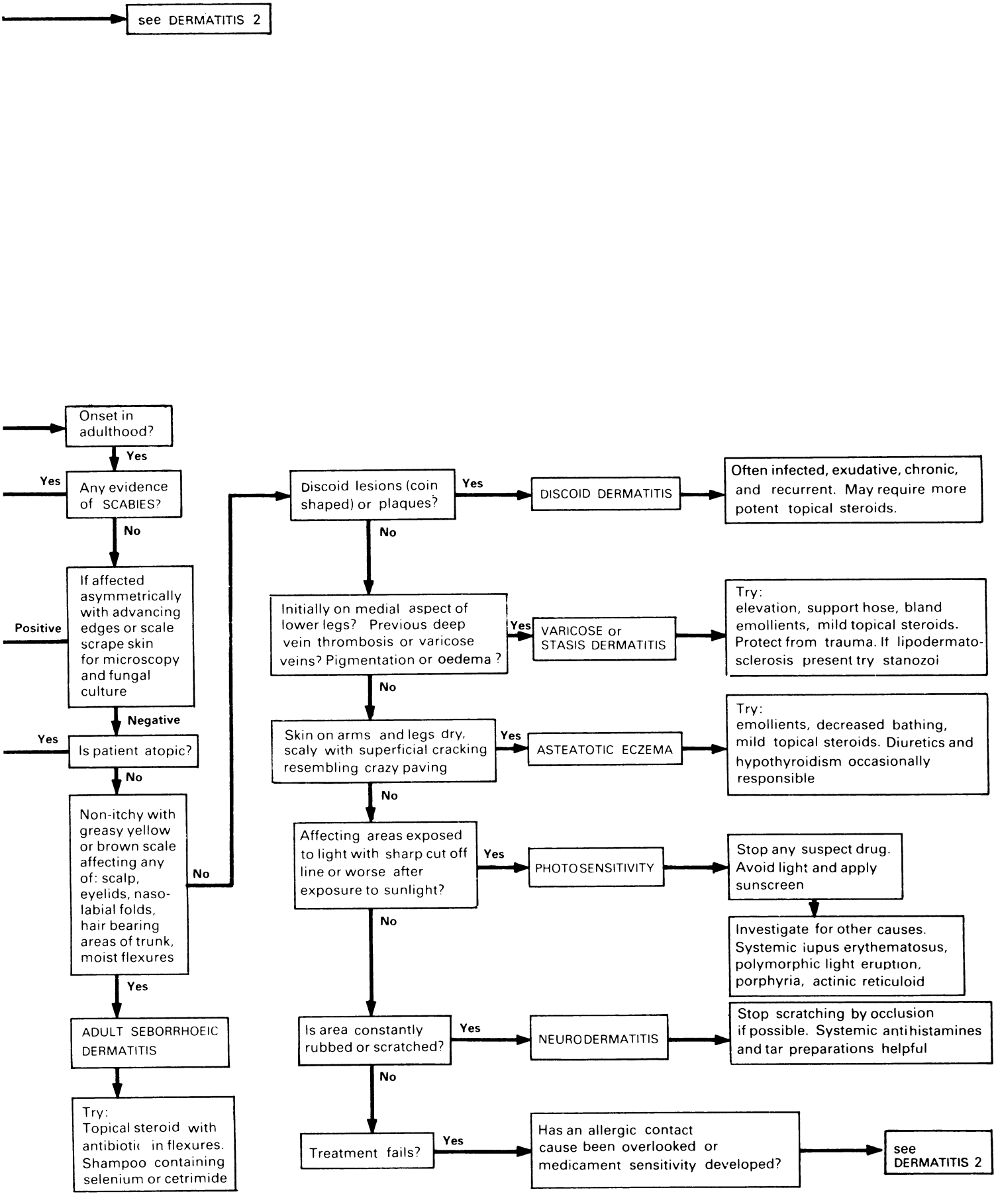
Dermatitis 1



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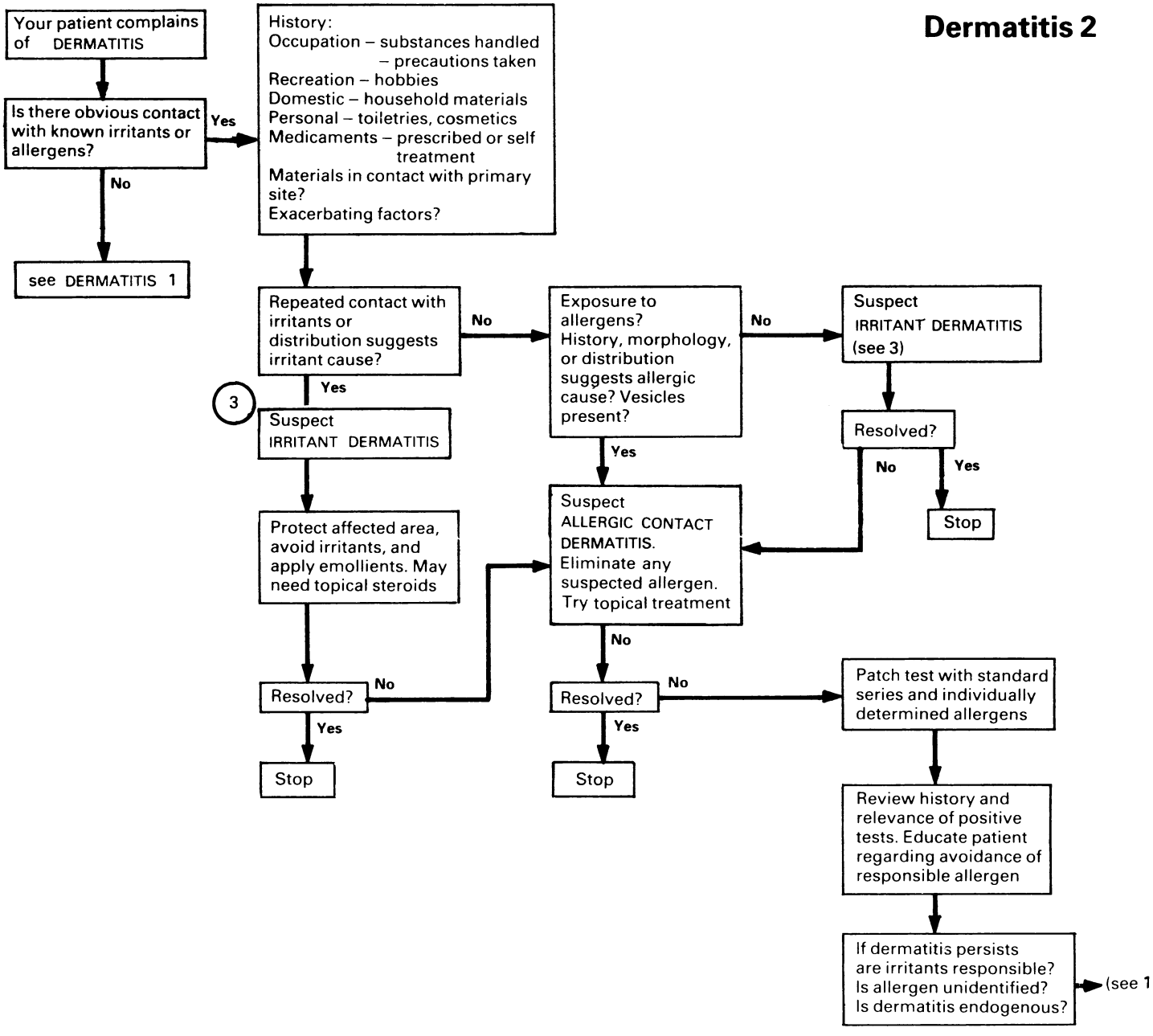

psoralen photochemotherapy (PUVA).[6] Elimination diets are more effective in young children and only benefit particular patients.[7] A diet excluding eggs, milk, chicken, food colour, and preservatives has been found useful in children not responding to topical steroids.[8]

Standard test series of the international contact dermatitis research group

Test substance	Materials in which sensitiser is commonly found
Potassium dichromate, 0·5% petrolatum	Cement, leather
P-phenylene diamine, 1% petrolatum	Black dye
Thiuram rubber mixture, petrolatum	Rubber, pesticides, disulfiram
Neomycin, 20% petrolatum	Topical medicaments
Cobalt chloride, 1% petrolatum	Metal alloys, paints
Caine mixture, petrolatum	Topical local anaesthetics
Nickel sulphate, 2·5% petrolatum	Plated metals, jean studs
Chinoform, 5% petrolatum	Topical antibacterial agent
Colophony (rosin), 20% petrolatum	Adhesive tape, varnish, polish
Parabens, petrolatum	Preservative in cosmetics and topical medicaments
p-Phenylenediamine derivatves rubber mixture, petrolatum	Rubber (gloves, shoes, etc)
Wool alcohols, 30% petrolatum	Lanolin (ointment base)
Mercaptobenzothiazole rubber mixture, petrolatum	Rubber (gloves, shoes, etc)
Epoxy resin, 1% petrolatum	Rubber, antifreeze, cutting oil
Balsam of Peru, 25% petrolatum	Electrical insulation, glues, plastics
Butyl formaldehyde resin, 1% petrolatum	Perfume (in cosmetics, soaps, etc)
Carba rubber mixture	Leather adhesive in shoes
Formaldehyde, 2% water	Domestic rubber, elastic, contraceptives
Ethylenediamine, 1% petrolatum	Black plastic, preservative in shampoo, cosmetics
Perfume mixture, 2% each, petrolatum	Industrial use, stabiliser in Tri-Adcortyl cream
	Perfumed products

Occasionally short term treatment with systemic steroids will be required. Desensitisation injections have a minor part to play in patients with frequent exacerbations, demonstrable reactions to prick tests, and IgE reactions to an unavoidable antigen.[9] If used low doses should be given, and caution is necessary to avoid an initial exacerbation.

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