Chapter 36

Dental Materials

36

Tuula Estlander, Kristiina Alanko, Riitta Jolanki

Contents

36.1 Introduction

Dental care personnel and dental laboratory workers are exposed daily to several allergens or irritants that cause delayed- or immediate-type allergic or irritant contact dermatitis. In addition, the hands of workers are repeatedly exposed at short intervals to water and cleansing agents. Even the use of plastic or rubber protective gloves is not without problems. Water- and air-tight gloves may hydrate and irritate the skin. In the worst case, allergy to glove materials may develop. Gloves permeable to chemicals used at work may even promote the development of allergy to these chemicals.

The same dental care products may also elicit allergic or irritant reactions in dental patients. They usually cause delayed-type allergic contact stomatitis (ACS) reactions, but immediate reactions are also possible. Patch testing is essential in distinguishing delayed allergic reactions from irritant ones. A biopsy may sometimes be necessary to exclude other oral diseases.

36.2 Dental Care Personnel and Dental Laboratory Workers

Nowadays, it is known worldwide that dentists, dental nurses, and other dental workers are at considerable risk of developing occupational contact dermatitis from the materials used in their work, whereas their patients only rarely develop contact stomatitis from dental materials [1–17]. Other occupational diseases may also develop, for instance bronchial asthma, rhinitis, conjunctivitis, pharyngitis, and laryngitis [18–23].

During the past decades, the frequency of occupational contact dermatitis has increased steadily in dental care personnel and was considered to be about 40% in the first part of the 1990s [11]. In the 1990s, three times as many occupational diseases of dentists and dental nurses compared to earlier were reported to the Finnish Register of Occupational Diseases

(FROD). In the same period, plastic materials had taken the place of amalgam in dental restoration, and the use of protective gloves [usually natural rubber latex (NRL) gloves] had become common practice because of the increased risk of HIV (human immunodeficiency virus) and hepatitis virus infections. In particular, allergic contact dermatitis (ACD) caused by methacrylates and NRL gloves, as well as contact urticaria due to NRL gloves had increased [14–15].

According to the information obtained from the FROD in 1990–2000, and from the study on dental nurses, two-thirds of all occupational diseases of dental care personnel were cases of contact dermatitis, with ACD being the most common of them. Typically, the number of cases of ACD is more than two-thirds, and the cases of irritant contact dermatitis is less than one-third of all the cases of contact dermatitis. Dental care workers belong to the eight most risky occupations concerning occupational allergic contact dermatitis. The risk of developing ACD is six times more common in dental care work compared to the average risk in all occupations [14–15].

Similar results concerning the sensitization of dental care personnel have been obtained in other studies as well [6–7, 17]. The frequency of type-IV sensitivity in dentists, dental hygienists, dental assistants, or dental students attending annual health screenings in 1997, 1998, 2000, 2002, and 2003 arranged by the American Dental Association (ADA) Annual Sessions, held in various major American cities, was determined by patch testing. One hundred and seventy eight (178) dentists and 51 non-dentists participated, and 49% of the patch-tested participants displayed positive reactions to at least one allergen. The most prominent allergens derived from dental materials were rubber chemicals and methacrylates [17].

In 1990–2000, a total of 151 cases of dermatosis among dentists and cases among 302 dental nurses were reported to the FROD. The number of cases of contact dermatitis was 349, of which more than two-thirds (255 cases) were cases of allergic contact dermatitis (type-IV allergy). Ninety-four (94) were cases of contact urticaria or protein contact dermatitis (type-I allergy) [14].

Dental laboratory workers have a similar risk of developing hand eczema as other dental personnel [24–26]. At the beginning of the 1980s, a Finnish questionnaire study of dental technicians [24] revealed the frequency of their hand dermatitis, both present and previous, to be about 30%. These technicians were mainly men (80%). In a Danish cross-sectional questionnaire study among dental technicians [27], the 1-year prevalence of skin problems of the hands was 43%, which was eight times as high as among the general population. More than half (60%) of the study participants were women. Another study with the same group showed that there was a rapid increase in the skin problems of dental technician trainees [28].

In a Swedish retrospective cohort study [26] of former dental technician students (n=2,139), a postal questionnaire inquired about the factors for the occurrence of hand eczema, including age of onset, occupational exposure, and use of protective gloves. In dental technicians, the incidence of hand eczema was 7.1 cases/1,000 person years among men and 10.8 among women during the time that they were exposed to acrylates. Based on the results, the risk of hand eczema was stated to be more than doubled in dental technicians.

36.3 Allergic Contact Dermatitis

36.3.1 Clinical Picture

The hands, and especially the fingers of dentists, dental nurses, orthodontists, and dental technicians, are most exposed to hazardous chemicals, which are often both allergens and irritants. Allergic contact dermatitis (ACD) caused by chemicals is typically located on the fingertips, which may become very dry, hyperkeratotic, chapped, smarting or itching, and reddish (pulpitis). There may also be vesicles or scaling. Stinging or burning sensations are also quite common, especially in cases caused by acrylics. ACD caused by methacrylates can be followed by mild paresthesia. The symptoms of paresthesia may last for weeks, even for half a year after the dermatitis has disappeared. These symptoms can also appear without previous development of contact allergy [29]. Also, the nail folds may become inflamed and red, swollen, and chapped [30]. Dermatitis may also appear in other locations, including the face, eyelids, and other exposed skin areas by airborne contact or by contaminated hands or gloves [5, 11].

ACD caused by rubber or plastic gloves appears typically on the skin areas covered by the gloves. Separate, itching eczematous areas may occur on the backs of the hands or on the wrists, or there may be reddish, swollen, and scaling dermatitis throughout the glove-covered areas. The dermatitis is usually the worst in skin areas that are in close contact with the gloves. Sometimes edema of the face and eyelids may be associated [31].

After contact with the causative agent has stopped, the dermatitis will disappear in different ways, de-

pending on its location. Even very mild pulpitis may take 2 or 3 weeks to cure, whereas dermatitis on the backs of the hands may disappear in a week. The skin usually remains symptomless if it is not in contact with the causative allergen, but one contact a week with the allergen makes dermatitis recur and may induce chronic dermatitis.

36.3.2 Causative Agents

36.3.2.1 Acrylics and Other Plastic Chemicals

Methacrylates and Acrylates

The methacrylates and acrylates contained in uncured dental plastic materials are the most common causes of ACD in dental personnel, including dentists, orthodontists, and dental nurses, and in dental laboratory workers, such as dental technicians and dental laboratory assistants [1–5, 7, 11, 14–15, 17, 25–26].

Up until the 1980s, knowledge of the irritant and sensitizing effects of dental materials was scarce. However, hypersensitivity to methylmethacrylate (MMA) in the manufacture of prostheses was reported as early as in 1941 [32, 33]. In 1954, Fisher and Woodside [34] described two dentists and two dental mechanics (technicians) who were occupationally sensitized to methylmethacrylate and who also had positive patch test reactions. The number of reports on dental personnel occupationally sensitized to acrylics was still small in the 1970s. Knowledge about the sensitizing capacity of acrylics was lacking and MMA was widely used as a standard allergen in revealing sensitization to dental acrylics. Better possibilities for investigating and understanding allergies associated with dental materials were not discovered until the beginning of the 1980s. New information was obtained about the sensitizing capacity of methacrylate and acrylate compounds in two theses using guinea pig maximization tests [35, 36]. At the same time, Tony Axell, together with Bert Björkner, Sigfrid Fregert, and Bo Niklasson in co-operation with the Scandinavian Institute of Dental Materials (NIOM), began to develop a patch test series suitable for examining stomatitis patients as well as dental care personnel [37]. The series contained 21 patch test substances, which were selected based on reported cases of contact allergy to dental materials, and the most frequently used components with documented or suspected potential contact allergens in dental practice. Most of these substances are still included in commercially available dental screening series, e.g., Chemotechnique Diagnostics, Malmö, Sweden and Trolab, Hermal, Reinbeck, Germany. In the 1990s, several clinical studies showed that MMA is a rather poor screening allergen for acrylics, and confirmed previous guinea pig sensitization studies on acrylics indicating that especially low-molecular-weight (LMW) methacrylates, such as 2-hydroxyethylmethacrylate (2-HEMA), triethylene glycol dimethacrylate (TREGDMA), and ethyleneglycol dimethacrylate (EGDMA) in the dental materials are stronger contact sensitizers than MMA, and also need to be used for patch testing [1–2, 4, 38–44] (see also Table 1).

Dental Composite Resins

The plastic products used in dental restoration can be dental composite resins (DCR), glass ionomers, glass-ionomer cements with plastic reinforcement, and compomers (glass ionomer added to DCR). DCR based on bisphenol A and methacrylates, e.g., 2,2-bis[4-(2-Hydroxy-3-methacryloxypropoxy)phenyl]-propane (Bisphenol A glycidyl methacrylate or bis-GMA), have been used since 1962 [46]. bis-GMA is the most extensively used hardening binder of DCR, which can also be replaced by urethane dimethacrylate (UEDMA), which has similar properties. bis-GMA and epoxy diacrylate sensitized four out of eight dental patients having occupational ACD in the 1980s [38]. Thereafter, new cases have not been reported from dental practice. High-molecular-weight (HMW) bis-GMA and UEDMA are probably not as common a sensitizer as LMW methacrylates. Allergy to UEDMA may be even less common than allergy to epoxy dimethacrylate [5].

Because HMW methacrylates have high viscosity, LMW methacrylates, including TREGDMA and EGDMA, are added to dilute these HMW monomers. The most commonly used dimethacrylate is TREGDMA. When water solubility is necessary, 2-HEMA is added, e.g., in glass ionomers and compomers. DCR may also contain chemically reactive prepolymers as allergens, usually methacrylated epoxies and urethanes, methacrylates, and dimethacrylates.

Concomitant positive patch test reactions to sensitizing methacrylates are common [2, 4, 25], which can be ascribed to multiple sensitization or cross-reactions based on animal tests [2, 47, 48]. Patch test results of sensitized workers with large (meth)acrylate series of Chemotechnique Diagnostics indicate that inter-patient cross-reactions to acrylics vary. During their work, career dental personnel are exposed to various DCR products, which may differ in composition from one batch to another [4–5].

DCR also contain inorganic fillers, e.g., fine particles of glass or quartz, pigments, nonreactive inert

Table 1. Allergic (meth)acrylate reactions in a 10-year study (October 1994 – September 2004) at the Finnish Institute of Occupational Health. At least one allergic patch test reaction was shown by 53 patients. The results have been compared to the sensitizing capacity of (meth)acrylates, based on animal studies, according to Björkner [45]. Source of (met)acrylates: *C* Chemotechnique Diagnostics AB, Malmö, Sweden; *T* Trolab, Hermal, Reinbeck, Germany; *O* manufactured by the Finnish Institute of Occupational Health (FIOH). Sensitizing capacity: *NG* not given; *I* weak; *II* mild; *III* moderate; *IV* strong; *V* extreme

(Meth)acrylate series	Abbreviation	Source	Patch test concentration (%, w/w)	Allergic/ tested	Allergic (%)	Rank order	Sensitizing capacity
Ethyl acrylate	EA	C	0.1	13/404	3.2	7	NG
Butyl acrylate	BA	C	0.1	4/403	1.0	20	NG
2-Ethylhexyl acrylate	2-EHA	C	0.1	0/403	0	–	V
Methyl methacrylate	MMA	C/T	2	7+4/258+305	2.0	10	NG
Ethyl methacrylate	EMA	C	2	14/402	3.5	6	NG
n-Butyl methacrylate	BMA	C	2	3/402	0.7	24	NG
2-Hydroxyethyl methacrylate	2-HEMA	C/T	2/1	12+18/93+304	7.6	2	I
2-Hydroxypropyl methacrylate	2-HPMA	C	2	33/403	18.2	1	I
Ethyleneglycol dimethacrylate	EGDMA	C/T	2	11+16/91+302	6.9	3	I
Triethyleneglycol dimethacrylate	TREGDMA	C/T	2	3+7/92+306	2.5	8	I
1,4-Butanediol dimethacrylate	BUDMA	C	2	7/397	1.8	12	I
Urethane dimethacrylate	UDMA	C	2	3/397	0.8	22	II
2,2-bis[4-(2-Methacryloxyethoxy)-phenyl]propane	bis-EMA	C	1	3/403	0.7	24	IV
2,2-bis[4-(Methacryloxy)phenyl]propane	bis-MA	C	2	0/332	0	–	V
2,2-bis[4-(2-Hydroxy-3-methacryl-oxypropoxy) phenyl]propane	bis-GMA	C/T	2	1+2/90+303	0.8	22	I
1,4-Butanediol diacrylate	BUDA	C	0.1	8/402	2.0	10	III-V
1,6-Hexanediol diacrylate	HDDA	C	0.1	5/396	1.3	16	III-V
Diethyleneglycol diacrylate	DEGDA	C	0.1	16/403	4.0	4	II
Dipropyleneglycol diacrylate	DPGDA	O	0.1	2/174	1.1	18	NG
Tripropyleneglycol diacrylate	TPGDA	C	0.1	6/403	1.5	14	IV
Triethyleneglycol diacrylate	TREGDA	C	0.1	3/77	3.9	5	I
Trimethylolpropane triacrylate	TMPTA	C	0.1	5/403	1.2	17	IV
Pentaerythritol triacrylate	PETA	C	0.1	7/403	0.5	13	V
Oligotriacrylate 480	OTA 480	C	0.1	2/403	0.5	28	III
Epoxy diacrylate {2,2-bis[4-(2-hydroxy-3-acryloxypropyl)phenyl]propane}	bis-GA	C	0.5	3/401	0.7	24	III–V
Urethane diacrylate (aliphatic)	al-UDA	C	0.1	1/403	0.2	31	V
Urethane diacrylate (aromatic)	ar-UDA	C	0.05	4/403	1.0	20	II
N, *N*-Methylenebisacrylamide	MBAA	C	1	1/403	0.2	31	NG
Tetrahydrofurfuryl methacrylate	THFMA	C	2	10/396	2.5	8	NG
N, *N*-Dimethylaminoethyl methacrylate	DMAEMA	C	0.2	0/66	0	–	NG
Glycidyl methacrylate	GMA	O	0.1	5/324	1.5	14	NG
Ethoxylated bisphenol-A dimethacrylate	EBADMA	O	0.2	2/175	1.1	18	NG
Ethoxyethyl acrylate	EEA	O	0.1	1/324	0.3	29	NG
2-Phenoxyethyl acrylate	PEA	O	0.1	2/338	0.6	27	NG
Isobornyl acrylate	IBA	O	0.1	0/338	0	–	NG
Ethyl cyanoacrylate	ECA	O	10	1/341	0.3	29	NG

polymers, and polymeric waxes that necessarily are not allergens [38]. When these additional substances are missing, the product is called a resin [4–5, 14].

Polymerization of the DCR mixture may take place by using chemicals or visible light. Double curing materials are cured using both chemicals and light. DCR may, therefore, contain various additives, such as photoinitiators (e.g., camphoroquinone), other initiators [e.g., (di)benzoylperoxide], activators (e.g., tertiary aromatic amines), and inhibitors (e.g., hydroquinone or methylhydroquinone), which may also sensitize [5, 11, 14].

Light-cured glass ionomers contain similar sensitizing methacrylates as DCR, and may cause allergy. A dental nurse had daily handled light-cured hybrid glass ionomers and developed occupational fingertip dermatitis, typical of ACD caused by acrylate compounds. On patch testing, she reacted to several acrylics, including 2-HEMA. Her hybrid glass ionomer primer and liquid also provoked an allergic patch test reaction [49].

Dentin bonding systems are needed to ensure firm adhesion of the DCR to the tooth. The first dentin-resin bonding agent was N-phenyl glycine glycidyl methacrylate, developed by Bowen in 1962 [46]. Since then, a large number of new dentin bonding compounds have been developed [51]. In 1978, a bonding system with a hydrophobic resin (methacryloxyethyl phenyl phosphate), phenyl P, mixed with a water-soluble form of methacrylate resin, i.e., 2-HEMA, was marketed in Japan. In 1983, the 3M Company introduced a bonding system using a phosphate ester of bis-GMA, and in 1988 saw a new system based on maleic acid and 2-HEMA. Eleven patients were sensitized to acrylics in dentin bonding systems. Four dental nurses and five dentists developed ACD, one dentist had pharyngitis, but no skin symptoms, and one dental nurse was probably sensitized from patch testing with her own undiluted acrylate products [51]. Concentrations of methacrylates identified in dental restorative materials are given in Table 2. However, the composition may change and new products are continuously being developed.

Before the restorative material (e.g., DCR) is applied into the teeth, the cavity is treated with an acidic etching agent and a bonding system, e.g., with a primer followed by an adhesive. The curing takes place with visible light. 2-HEMA is commonly used in both primers and adhesives. Nowadays more often one-component bonding systems are used [4, 5].

Table 2. Concentrations of methacrylates identified in dental restorative materials [142]. For the abbreviations of (meth)acrylates, see Table 1

Identified methacrylate	Concentration (%, w/w)	
	Range	Median
Bonding materials (seven products)		
2-HEMA	0.3–28	17
bis-GMA	21–40	27
EGDMA	0.05–0.4	<0.3
TREGDMA	4–46	
UDMA	2–29	
Diethyleneglycol dimethacrylate	0.05–5	
Trimethylolpropane trimethacrylate	3–7	
EMA	1	
Glycerine dimethacrylate	4–8	
Methacrylic acid	Not quantified	
2-HPMA	0.3	
1-Chloromethyl-2-hydroxyethyl methacrylate	Not quantified	
Composite resins (8 products)		
bis-GMA	6–21	10
TREGDMA	3–7	6
EGDMA	0.05–5	
UDMA	8–15	11
bis-EMA	6–8	
Decamethylene dimethacrylate	0.05–1	
2-HEMA	7	
DMAEMA	2	
bis-MA	5	
Diethyleneglycol dimethacrylate	Not quantified	
Methacrylic acid	Not quantified	
Glass ionomers (2 products)		
2-HEMA	0.2–23	
EGDMA	0.1–0.2	
Methacrylic acid	Not quantified	
TMPTMA	9	
2-HPMA	0.3	

Prostheses

The composition of the basement sheets of dental prostheses has been almost the same for decades. They are prepared from a mixture of polymethyl methacrylate (PMMA) powder and liquid MMA, and the mass is molded manually or mechanically. The components of the powder and liquid of an acrylic denture base material are shown in Table 3. The powder may also contain copolymers with different acrylates, e.g., polybutyl acrylate, or methacrylates, e.g., polyethyl methacrylate and polyisobutyl methacrylate or polystyrene, initiators [(di)benzoylperoxide, barbiturates], colors, pigments (salts of cadmium, calcium, and zinc), butyl hydroxytoluene (BHT), and various filling agents, such as silicone dioxide [4, 5, 11, 25, 52].

Table 3. Components of the powder and liquid of an acrylic denture base material [5]

Powder	Liquid
Polymethyl methacrylate or polymer	Methyl methacrylate or monomer
Organic peroxide initiator	Hydroquinone inhibitor
Titanium dioxide for control translucency	Dimethacrylate or cross-linking agent[a]
Inorganic pigments for color	Organic amine accelerator[b]
Dyed synthetic fibers for appearance	

[a] A cross-linking agent is present if the manufacturer indicates that the material is a cross-linked acrylic
[b] The amine is present only if the material is labeled as a product to be processed at room temperature. Some manufacturers list them as cold-curing or self-curing materials

When PMMA powders melted at lower temperatures than usual are used, they can contain, in addition, copolymers of MMA, e.g., ethylmethacrylate (EMA). The powders may also contain small amounts of monomer impurities, e.g., MMA and ethylacrylate (EA). *N*-Butyl methacrylate, isobutyl methacrylate, and other methacrylates can be used to replace liquid MMA. After molding, the acrylate mixture polymerizes. The reaction is based on the use of heat, chemicals, light (UV or visible), or microwaves. (di)Benzoylperoxide is used in mixtures that polymerize at room temperature (cold-cured or self-cured) or at higher temperatures (heat-cured) to initiate the hardening process [4, 5, 11, 25].

If the manufacturer indicates that the material is a cross-linked acrylic, then 1,4-butanediol dimethacrylate (BUDMA), EGDMA, or ethylene glycol methacrylate can be used., e.g., the monomer liquid in heat-polymerizable products contains 1,4-butanediol dimethacrylate or EGDMA as cross-linkers. Monomer liquids may also contain 2-HEMA and other dimethacrylates. Cold-curable liquids may contain allergenic activators such as *N*, *N*-dimethyl-*p*-toluidine, 4-tolyldiethanolamine, and diethanol-*p*-toluidine. Liquids may also contain stabilizers (polymerization inhibitors), such as (methyl)hydroquinone, *p*-methoxyphenol or butylated cresols (BHT). Other components include ethyl alcohol and plasticizers, such as phthalates (dibutyl phthalate). In addition, benzophenones or benzotriazoles, phenyl salicylate, methyl salicylate, resorcinol monobenzoate, or stilbene can be added as UV-absorbers [3–5, 24, 25, 52–55].

Also, complex light-curable (UV or visible) acrylics similar in composition to DCR are used by dental technicians, and this may mean an increased risk of sensitization to these materials. According to some safety data sheets, these products may contain urethaneacrylates, e.g., polyester urethaneacrylate, dimethacrylates, e.g., diurethane dimethacrylate, 1,6-hexanedioldimethacrylate, methacrylates, e.g., MMA, dimethylaminoethyl methacrylate, acrylates, e.g., 3-dimethyaminoneopentylacrylate, and accelerators, photoinitiators, e.g., camphoroquinone, and other compounds, including fillers, pigments, and BHT [4, 5].

Crown and bridge materials can contain, in addition to MMA, e.g., tetrafurfuryl methacrylate, EGDMA, TREGDMA, I, 4-BUDMA and UEDMA [5, 25, 53].

Fasting cements for prostheses usually contain similar acrylate compounds as DCR.

Dental technicians and other dental laboratory workers also use daily many other materials, such as glues, plasters, waxes, dental alloys, polishing pastes, and enamel. Molding plasters can contain melamine-formaldehyde resin, which has sensitized dental technicians [25].

Cyanoacrylate glues are used almost daily in dental laboratories. They are usually used to repair cracks in plaster and stone (hard) plaster models, but can also be used to glue together broken acrylic prostheses. Cyanoacrylate glues seldom sensitize. Cases have been reported from glueing artificial nails [56–58] and attaching false hair with the glues [59]. The allergens in these cases were ethyl cyanoacrylate and MMA. Cyanoacrylate glues may also irritate the skin and induce chemical burns as a result of accidental exposure [57]. Nail dystrophy has been described after the use of artificial nails [58]. Asthma caused by cyanoacrylate exposure is also well known [60].

In a German study, out of 55 patch tested dental technicians, 16% reacted to MMA, 33% to 2-HEMA, and 27% to EGDMA. Positive reactions to other methacrylates, e.g., to EMA (11%) and TREGDMA (4%), and to acrylates, e.g., EA (6%) and pentaerythritol triacrylate (PETA) (4%), were less common. The study also demonstrated high cross-reactivity between 2-HEMA and EGDMA and moderate cross-reactivity between MMA and 2-HEMA. Only two positive reactions to TREGDMA were observed and one to BUDMA, suggesting that these are less sensitizing compounds than MMA, 2-HEMA, and EGDMA [25]. MMA, 2-HEMA, and EGDMA were also the most common reactors in a previous German study of dental technicians [55].

Additives in Dental Acrylics

Additives used in dental restoration and prosthetic materials are seldom the cause of ACD.

(di)Benzoyl peroxide is an essential part of these materials. An appreciable amount of benzoylperoxide can be present in dentures [61]. However, only solitary cases of sensitization have been reported. Two cases of ACD from manufacturing dental prostheses were reported by Calnan [62]. Kanerva et al. 1994 [63] described a dentist who was sensitized to mercury and benzoyl peroxide. Benzoyl peroxide has also caused sensitization in other exposures, including acrylic bone cement, an arm prosthesis, acne treatment preparations, baking additives, and the treatment of leg ulcers [64–66]. It has also caused airborne ACD [67]. On patch testing, benzoyl peroxide is an irritant that easily causes false-positive reactions. The frequency of 11% positive reactions of the participants in an American patch test study of dental personnel suggests several irritant reactions [17].

Also *N*,*N*-dimethyl-*p*-toluidine and 4-tolyl diethanolamine are very rare sensitizers in dental personnel. A dentist with occupational ACD had positive patch tests reactions to 4-tolyl diethanolamine, as well as to coconut diethanolamide and *N*-ethyl-4-toluene sulfonamide [68].

Methylhydroquinone and hydroquinone are used to prevent unintended polymerization, but sensitization in dental personnel has not been reported. Other stabilizers, *p*-methoxy phenol and BHT, are also very rare sensitizers [5, 24].

Plasticizers are added to improve the flexibility, softness, and pliability of plastics. Dibutyl phthalate has been added as a plasticizer at various times to denture base resins by the manufacturer or by the dental technician. In general, allergy to dibutylphthalate is very rare [5].

Camphoroquinone is used as an initiator for visible-light-cured DCR materials and primers. It has been considered as a nonsensitizer, but one case of active sensitization from patch testing has been reported [69].

Various UV-absorbers are incorporated in DCR products, other plastics, textiles, and sunscreens. They include, e.g., 2-hydroxy-4-methoxy-benzophenone (Eusolex 4360), 2-(2-hydroxy-5-methylphenyl)benzotriazole (Tinuvin P), phenyl salicylate, methyl salicylate, resorcinol monobenzoate, and stilbene. Sporadic cases of sensitization have been reported, but not in dental personnel [4, 5, 53].

Epoxy Resin and Bisphenol A

Sensitizing diglycidylether-of-bisphenol-A epoxy resin (DGEBA-ER) is, nowadays, used as a component of a root canal sealant. Some of the dental workers sensitized to DCR also reacted to DGEBA-ER on patch testing [45]. DCR may contain DGEBA-ER as an impurity. Possibly, DGEBA-ER and bis-GMA cross-react in some individuals, although there is also evidence that they do not cross-react [5, 45, 70].

Bisphenol A is a raw material in the production of epoxy and acrylic resins. A dental nurse was sensitized to bisphenol A, possibly as an impurity in the DCR products that she had handled at work [71].

Bisphenol A can also be used as an additive in the manufacture of PVC plastics. A dentist and an oral hygienist were sensitized from the use of PVC gloves of the same trademark. However, it cannot be ruled out that the DCR which they had handled during their restorative work may have contributed to their sensitization. After these cases, the disposable PVC gloves on the Finnish markets were analyzed, but bisphenol A could no longer be found [72].

36.3.2.2 Rubber Chemicals

Protective gloves are the most common source of occupational allergic contact dermatitis from rubber chemicals [31]. They are among the most common causes of ACD in dental care personnel, in addition to methacrylate and acrylate compounds [6, 14, 15, 17]. They have to be taken into account also in dental laboratory work, even though gloves are not used as consistently as in dental restoration work [24–26, 53]. There may also be other causes of rubber sensitization among dentists, e.g., dams and polishing discs made of rubber [38].

Rubber gloves are usually manufactured using various automated processes. The primary ingredient is rubber polymer, which is blended with 15–20 additives, including vulcanizing agents, accelerators, antioxidants, pigments, fillers, and oils. Rubber polymer can be a natural product made from milky liquid (natural latex) of the rubber tree, or it can be manufactured synthetically. Whether a rubber glove is called natural (NRL) or synthetic depends on the origin of the polymer used in its manufacture [31].

Sensitizing chemicals are contained in gloves made of both natural and synthetic rubbers. However, natural rubber gloves are commonly used gloves, and are, therefore, probably the main cause. The three most important allergenic causative chemicals include thiuram, dithiocarbamate, and benzothiazole accelerators [31]. In an American patch test study of

dental personnel, 10% reacted to thiurams and 12% to carbamates [17].

In 1990–2000, a total of 61 (18%) out of 255 cases of allergic contact dermatitis in dental personnel reported to the FROD were caused by rubber chemicals [14, 15].

36.3.2.3 Antimicrobials

Antimicrobials are also an important group of sensitizers among dental care and dental laboratory workers [5, 14, 15, 17]. They can be components of disinfectants and cleansing agents, e.g., glutaraldehyde, formaldehyde and formaldehyde-releasing agents, glyoxal, chloramine-T, and persulfates. They can also be used as components of tooth bleaching agents (persulfates), and they may be present in medicines used to cure gingivitis, disinfectant liquids of implants, and mouth and hand washes (chlorhexidine). All antimicrobials used in hand cleansing agents and hand creams can also be causes of contact allergy in dental work (e.g., isothiazolinones, methyldibromo glutaronitrile, and formaldehyde liberators).

Glutaraldehyde is widely used as an antimicrobial agent in the cold sterilization of dental equipment and in hospitals, e.g., for disinfecting metal parts of beds and in hospital laboratories. It may be present in dental acrylic adhesives and bonding agents at concentrations of 0.7–5%. It is also an irritant, and has previously been considered to be a weak sensitizer [73]. However, more recent reports suggest it to be a stronger sensitizer [74–76]. Glutaraldehyde, in addition to other antimicrobials, has induced sensitization in dental nurses [38, 76, 77]. Glutaraldehyde and formaldehyde do not cross-react, but concomitant sensitization is common [3, 64]. In an American patch test study, glutaraldehyde and formaldehyde each produced 3.5% positive reactions with no evidence of cross-reactivity [17].

Formaldehyde is a commonly used chemical and a frequent sensitizer in many countries [78]. Paraformaldehyde, previously commonly used to treat root canals, has been an important source of formaldehyde allergy in dentistry [15, 38]. Formaldehyde as such is possibly no longer a component in disinfecting and cold-sterilizing liquids in dental practice. Various formaldehyde-releasing agents are probably the source of formaldehyde in some cleansing agents and soaps. According to Flyvholm's investigation in Danish markets [79], bromonitropropanediol, bromonitrodioxane, and trihydroxyethylhexahydro s-triazine were the most common formaldehyde releasers in cleansing agents, and bromonitrodioxane, imidazolidinyl urea, and bromonitropropanediol in soaps and other skin care products. Patients allergic to formaldehyde will benefit from information on exposure to formaldehyde releasers. In addition, some formaldehyde releasers can act as allergens themselves [14, 15, 79].

Minimal amounts of formaldehyde which possibly leach from acrylics are not important in the development of allergy to the chemical.

Glyoxal (ethanediol) is a dialdehyde, which can be a component in many disinfectants used to disinfect equipment and rooms in hospitals and in dental practices. Elsner et al. [80] reported on seven health care workers sensitized to the chemical. Two of these seven also reacted to formaldehyde and three of six to glutaraldehyde. One report describes a dental nurse who had developed occupational ACD from glyoxal, glutaraldehyde, and neomycin sulfate [77].

TEGO, the commercial name of certain disinfectants sold in many countries under various trade names, has been the cause of several cases of ACD [38, 81, 82]. The active ingredient of TEGO is dodecyldi-(aminoethyl)glycine (DDAG), but is not present in all TEGO products. It has been widely used in Europe as an antiseptic for instruments in hospitals and in dental practices [38]. From the 1970s to the 1990s, it was the most common antimicrobial agent causing ACD in Finnish dental personnel [15, 38, 83]. Since 1991, TEGO products have not been available in Finland.

Chlorhexidine, 1,6-di-(4-chlorophenyldiguanido)-hexane was introduced in the 1950s. It is a guanidin disinfectant. Chlorhexidine diacetate, digluconate, or hydrochloride can be used as an antimicrobial, e.g., in topical antiseptics, for disinfectants, e.g., in ointments, mouth, and hand washes. It is also used to cure gingivitis. Despite its widespread use, delayed allergic reactions, as well as photoallergic reactions can be considered rare. Immediate reactions due to exposure to the chemical are more important [5, 84].

Quaternary ammonium compounds classified in cationic detergents are, nowadays, increasingly used as disinfectants for various dental instruments and equipment. They are irritants that can also cause delayed irritation reactions. A delayed irritant reaction may be difficult to distinguish from true allergic reactions on patch testing.

Benzalkonium chloride is the most extensively used quaternary ammonium compound in medical use. It has been classified as weak allergens in animal experiments [85]. It has been concluded that, in the average population, benzalkonium chloride is not a relevant allergen, whereas in medical professionals and in ophthalmological patients, it is possibly a relevant one, but only some cases of allergy to the compound have been reported. A dental nurse having

contact allergy to benzalkonium chloride from a sterilizing solution has been reported. [5, 76, 84].

Placucci et al. [86] reported hand dermatitis in a dental nurse from *N*-benzyl-*N*, *N*-dihydroxyethyl-*N*-cocosalkyl-ammonium chloride, present in disinfectant wipes used in dentistry.

ACD from polyvinylpyrrolidone-iodine (*povidone-iodine*, Betadine) has rarely been reported. It is used at a concentration of 4% as a skin cleanser. Those sensitized to povidone-iodine are usually not allergic to iodine [87, 88]. The chemical is possibly a weak sensitizer. Occupational ACD is rare [89–91], but has been reported in a dentist and in an operating room nurse [92].

Products containing *potassium persulfate* are used to disinfect surfaces, but not instruments in dental practice. It is also used in toothpastes and other bleaching agents of teeth. The chemical may irritate the skin and cause delayed and immediate allergic reactions, as well as asthma [5, 93–95].

36.3.2.4 Metals

Metallic *mercury* has been used, e.g., in dental amalgams, thermometers, pharmaceuticals, antifouling agents, and agricultural chemicals. Mercury unites with many metals to form an amalgam. Amalgams prepared from zinc, tin, and mercury have been used as dental cements, and amalgams of mercury with gold, silver, or copper have been used as fillings for teeth [3]. The composition of amalgams used in different countries varies and there has also been variation over time [96]. Occupational amalgam allergy is relatively rare, but has been reported in dentists and dental nurses [2, 5, 97, 98].

Gold in dentistry is used in the form of alloys with silver, copper, palladium, platinum, and zinc to make, e.g., crowns and bridges. Previously, contact allergy to metallic gold and gold salts was considered to be low, but during the past 10 years, a high frequency of positive reactions among dermatitis patients patch tested with gold sodium thiosulfate have been reported [99–107]. Patch testing with the salt may cause a long-lasting patch test reaction [108]. Hypersensitivity to gold may be seen, together with contact allergy to other metals, including mercury, nickel, and palladium [109–111].

Several sporadic cases of ACD from both metallic gold and gold salts have been reported. Occupational gold allergy has been reported in the electronics and gold-plating industry [111]. A dental nurse working in a special dental laboratory polished gold crowns and bridges in periods of 2 weeks, and was exposed to fine metal dust. She developed itching dermatitis on her hands and face when polishing the pieces, and the dermatitis faded soon after she had stopped the work. On patch testing, she showed a positive reaction to gold sodium thiosulfate, probably from exposure to the dust containing gold (unpublished).

Orthodontists are increasingly applying braces to children and adults. Since at least 10% of women are allergic to *nickel*, a nickel-allergic orthodontist may get hand dermatitis when bending the metal parts of braces. Dental technicians may use instruments releasing nickel, and they can also be exposed to materials containing nickel, resulting in sensitization and ACD to nickel, or their pre-existing nickel allergy may worsen [3, 5, 11, 14].

Palladium is a metal found most commonly in ore combined with platinum, gold, and copper. It is used in varying amounts (4–82%) in cast dental restorations. It has also been used instead of amalgam in dental fillings to avoid the possible toxicity of mercury. Dermatitis from palladium was previously considered rare, but nowadays, about one-third of the patients allergic to nickel sulfate also show positive patch test reactions to palladium, possibly as a sign of cross-reactivity. There are no convincing reports on occupational dermatitis caused by palladium [3, 5, 112, 113].

Cobalt–chromium alloys, which form the framework of partly metal dentures, and base metal alloys contain about 60% cobalt. Dental technicians may have a risk of developing sensitivity to *cobalt*, e.g., when exposed to the polishing dust of these alloys. However, none of the 55 dental technicians in a German study reacted to cobalt [25]. It is often not clear whether *chromium* or other metals or metal salts have caused the allergic reactions elicited by dental metals [5, 25].

Aluminum is used as pure metal or as an alloy, e.g., in dental materials. Aluminum salts can be used in dental ceramics. Allergy is very rare, and has not been reported from dental aluminum [5, 113].

Dental amalgam may also contain *copper*, but allergic reactions to copper are rarely reported. Many of the patients who are patch test positive to copper are concomitantly positive to nickel sulfate, and the question of cross-reactivity has, therefore, been raised. On the other hand, the copper patch test substance may contain nickel, and the positive reaction may represent allergy to nickel. However, copper allergy has been reported [5, 113–115]. Metallic platinum is also used in dentistry, but it rarely causes ACD [113].

Dental amalgam also contains *silver* and *tin*. Metallic silver has not been reported to cause ACD.

There is no convincing evidence of sensitization caused by tin [113].

Titanium frameworks with removable partial dentures have been recommended for use in patients allergic to other metals. Titanium is also used in dental implants. Some reports indicate that the metal can act as an allergen. Its use in these applications is still recommended [5, 113].

36.3.2.5 Colophony, Eugenol, and Balsam of Peru

Colophony or rosin is a resin obtained from different species of coniferous trees. There are three types of rosin, depending on the method of recovery. Colophony is a complex mixture of resin acids (about 90%) and neutral substances. The major acids are abietic acid and dehydroabietic acid. As a result of exposure to air, oxidized components are present in colophony. The oxidized components are important sensitizers. The major allergen is the primary oxidation product, 15-hydroperoxyabietic acid. Patients with positive patch test reactions to colophony often also react to balsam of Peru and fragrance mix [116]. Colophony is present in dental materials, e.g., in periodontal dressings, impression materials, cavity varnishes (cements), and temporary filling materials. Zinc-oxide-eugenol (ZOE) cements may also contain colophony. Even more than 30% colophony may be present in Duraphat, a fluoride varnish. Occupational dermatitis caused by colophony has been reported in dental nurses [117, 118] and in a dental technician [119].

In dentistry, essential oils are chiefly used as pharmaceutical aids and mild antiseptics. Eugenol is an important chemical constituent of clove oil. It is also present in many other products, including cinnamon oil, perfumes, soaps, bay rum, pimento oil (allspice), flower oils, food spices, and flavoring agents [64]. It is one of the eight components in the fragrance mixture of the standard patch series used to detect fragrance allergy. In dentistry, eugenol is mixed with zinc oxide to form ZOE cement. It can also be used in toothache drops, antiseptics, and mouth washes. ZOE has beneficial physical and therapeutic effects, making it suitable for use as a provisional restorative material, base material, and root canal filling material. Eugenol can also be combined with colophony and used as an intermediate two-component restorative material with polymethylmethacrylate powder, e.g., in IRM liquid. The two components of IRM are mixed before use. Also, eugenol-free IRM liquid is available. When eugenol is used in dental preparations, including impression pastes, surgical packing, and cements and provisional restorative fillings, it may also be the cause of occupational ACD in dental personnel [120, 121].

Other sensitizing oils can also be constituents of dental products, e.g., cinnamon, peppermint, anise, and spearmint oil. Balsam of Peru can be present in liquids mixed in surgical and impression pastes. Also, other balsams, e.g., Canada balsam, can be used [14, 122].

36.3.2.6 Impression Compounds and Resin Carriers

Silicon-based materials, alginate, and beeswax are commonly used as impression compounds. Silicon-based materials have probably not caused sensitization in dental personnel. Two cases of contact allergy have been reported, caused by a catalyst in a silicon-based material [123]. Alginates have not caused any definite cases of sensitization [124]. Beeswax is a sensitizer, and occupational dermatitis has been reported [125]. Dental modeling waxes may contain at least 17% beeswax [5].

Resin carriers are used to isolate cavities under restorations, e.g., *N*-ethyl-4-toluene-sulphonamide. A dentist with multiple sensitivities to materials that she had used in her dental practice also displayed a positive reaction to the chemical [68]. In a Swedish multicenter study, 9 of 1,657 patch-tested patients with oral symptoms reacted to *N*-ethyl-4-toluene-sulphonamide [5].

36.3.2.7 Local Anesthetics

Local anesthetics can be divided into two groups, amides and esters, based on their structure. Allergies to local anesthetics were common earlier, when the ester group of anesthetics, e.g., benzocaine was used, but allergy from amides is rare. Up to 1991, only 18 cases had been reported since the 1940s, when amide anesthetics were more extensively used [126]. Cross-reactions may occur between structurally related ester anesthetics, but not between structurally unrelated groups. Cross-reactions between amide anesthetics are not well known.

Dentists' sensitization to local anesthetics was rather common earlier [64]; nowadays, sensitization to these products is probably unusual. Benzocaine, tetracaine, and procaine used to be the sensitizers in these cases [64]. Lidocaine (xylocaine, lignocaine) is an amide anesthetic and does not cross-react with benzocaine or tetracaine. It is safe to both dentists and their patients because allergic reactions are rare

[127]. Mepivacaine and prilocaine have caused a few solitary cases of sensitization [127, 128].

36.4 Contact Urticaria, Protein Contact Dermatitis, and Other Immediate Reactions

Contact urticaria (CU) may be an immunological (allergic) or a nonimmunological reaction. IgE-mediated (type-I) allergic reactions are usually caused by proteins, but certain LMW chemicals may also elicit similar immediate hypersensitivity reactions caused by both allergic and unknown mechanisms [5].

36.4.1 Clinical Picture

Contact urticaria reaction as a result of type-I allergy develops in minutes, usually in less than half an hour, after the skin of the hands, especially the back of the hands and fingers and wrists and forearms, has come into contact with the causative allergen. Sometimes, the eyelids can be the worst affected, probably by airborne contact or by the hands. Typically, there is redness and whealing on the skin of the contact areas, which may also be swollen and itching or smarting. A contact urticaria reaction also disappears quickly, usually in the course of a few hours, leaving the affected skin completely symptomless. Sometimes, a local contact urticaria reaction may elicit generalized urticaria. Other symptoms of type-I allergy are also common, including itching and running of the eyes or nose, conjunctivitis, rhinitis, coughing, dyspnea, or asthma. In the worst case, a life-threatening anaphylactic reaction may develop.

Type-I allergy may also lead to so-called protein contact dermatitis. When the skin is repeatedly in contact with proteinaceous causative agents, whealing may no longer be seen on the skin. The appearance of dermatitis resembles that of eczema and cannot be distinguished from allergic or irritant contact eczema caused by chemicals.

36.4.2 Causative Agents

36.4.2.1 Protective Gloves

Proteins in *natural rubber latex* (NRL) are the most important cause of contact urticaria in general, especially in dental personnel [129], and NRL gloves are the most important source. Tarlo et al. [130] reported that 10% of dental students and staff had NRL sensitivity. Safadi et al. [131] reported that 12% of oral health care workers had positive skin prick tests to latex protein. Heese et al. [132] reported positive prick tests to NRL in 8.7% of 296 dental students. Lindberg and Silverdahl [13], in a study of 527 dental professionals (192 dentists, 269 nurses, 64 hygienists, 2 in administrative work), tested 389 participants with CAP-RAST (Pharmacia Upjohn Diagnostics, Uppsala, Sweden) to estimate the prevalence of NRL allergy: 7.2% were found positive in the test. There was a significant difference among the three professions: 10.2% positive dentists (13 of 128 tested), 6.0% positive nurses (13 of 216 tested), and 4.4% positive hygienists (2 of 45 tested). In Finland, dentists and dental nurses have been estimated to have the greatest risk of all occupations investigated of getting immediate allergy to latex proteins. Based on the cases reported to the FROD in 1991–1996, the incidence rate of NRL allergy in dental nurses was 11.8 cases/10,000 workers and in dentists 6.0 cases/10,000 workers. Dental nurses had 50 times as much contact urticaria and protein contact dermatitis caused by NRL proteins as all the occupations on average [129]. Also, occupational asthma caused by NRL is possible. In a study based on the cases reported to the FROD in 1990–1998, 62 cases of occupational respiratory hypersensitivity were observed in dental personnel. NRL caused ten cases of occupational rhinitis and two cases of asthma [23].

The *cornstarch powder* in NRL gloves has very seldom been reported as a cause of contact urticaria [133, 134]. On rare occasions, *chemicals* have been reported to cause contact urticaria from rubber products. A case of contact urticaria caused by latex-free nitrile gloves has been reported [5].

36.4.2.2 Low-Molecular-Weight Chemicals

Haptens may also cause IgE-mediated reactions. The hapten binds to protein or another macromolecule, and the resulting hapten–carrier conjugate acts as an allergen [135].

Chloramine-T (sodium-*N*-chlorine-*p*-toluene sulfonamide), used in dental work as a disinfectant of instruments, boxes, and surfaces, can cause occupational contact urticaria, as can persulfates used for the same purposes. In addition, they can be the cause of occupational rhinitis and asthma. At the Finnish Institute of Occupational Health (FIOH) in 1990–1998, three cases of asthma and one case of rhinitis in dental personnel were diagnosed as being caused by chloramine-T [23].

Chlorhexidine can be present in agents used to cure gingivitis and as a constituent of hand washes.

As an acetate or gluconate salt, it is used for topical application, on skin or mucous membranes, wounds, burns, surgical instruments, and surfaces. It can cause contact urticaria and asthma [136]. It can also be the cause of photosensitivity and fixed drug eruptions [136].

Colophony and eugenol have also caused immediate-type hypersensitivity reactions. In a Finnish study of dental personnel [23], one case of occupational rhinitis caused by Nobetec containing colophony was diagnosed. Contact urticaria from eugenol has been considered to be a nonimmunological reaction, but recently, it has been reported to cause type-I sensitivity and contact urticaria in a dental patient [137].

Acrylics may cause immediate hypersensitivity as well. Contact urticaria, conjunctivitis, rhinitis, pharyngitis, and asthma from cyanoacrylates, MMA, acrylic acid, and nonspecified acrylics have been reported [18–22], but the mechanism of the reactions is not known. According to the FROD, a total of 64 cases of occupational respiratory diseases were diagnosed in dental personnel in Finland; two cases were diagnosed in 1975–1989 and 62 in 1990–1998. There were 28 cases of occupational asthma (18 caused by methacrylates), 28 occupational rhinitis (6 caused by methacrylates), 7 allergic alveolitis, and 1 organic toxic syndrome. This study shows the increasing frequency of respiratory hypersensitivity in dental personnel [23].

36.5 Irritant Contact Dermatitis

Dental workers are exposed to many skin irritants. The most common irritants include cleaning agents (detergents) and disinfectants used for hands, as well as for surfaces and instruments, wet work, hydrating effect of protective gloves, and dental acrylics.

Occupational irritant contact dermatitis is, in general, more common than allergic contact dermatitis. However, according to information obtained from the FROD concerning occupational dermatoses of dentists and dental nurses in 1990–2000, only 19% of the reported 86 cases were due to irritation [14]. Detergents were reported as the main causes of irritant contact dermatitis in half of the cases (51%), wet work in 19%, and methacrylates in 10% of the cases. In a study on Finnish dental nurses, frequent hand washing was considered to be the main cause of irritant dermatitis. Half of the nurses reporting work-related hand dermatitis said that using protective gloves aggravated their hand dermatitis [15].

Corresponding results were obtained in a study of 55 patch tested dental technicians; 13 (24%) had irritant contact dermatitis and 2 had allergic/irritant contact dermatitis [25]. The causative agents were metals and plastics (acrylics), plasters, and ceramics. The most important agents causing irritant contact dermatitis to dental technicians have been wet work, work with plaster, grinding, and physical irritation, as caused by polishing metal and plastic materials. Hand washing up to 100 times a day was considered to contribute as well [24, 25]. Mürer et al. [27] studied Danish dental technicians and found acrylates to be the most important cause of their hand problems. Of the 69 having hand dermatitis at the time of the questionnaire study, 64 reported using MMA or cyanoacrylate glue daily or almost daily. Three reported allergy to MMA. A study on dental technician trainees [28] showed that, shortly after beginning their education, the trainees had the same high proportion of skin problems as the dental technicians at work.

36.6 Photo-Related Reactions

Phototoxic or photoallergic reactions may represent a new problem in dentistry as a result of extensive powerful light sources in the curing of dental resins. Many substances, including sulfonamides present in some cavity liners, phenothiazines, griseofulvin, and tetracyclines, used in dentistry may have phototoxic properties. Photoallergic compounds in dentistry include eugenol, chlorhexidine, derivatives of 4-aminobenzoic acid (PABA), sulfonamides, and phenothiazines. A generalized erythematous eruption of the face and submental area in a dental hygienist was caused by trimethoprim medication and exposure to a photocuring unit [5, 138].

36.7 Investigations

In investigations, the determination of exposure to chemicals, explanation of the work techniques used, as well as skin tests (patch and prick test) are the most important tasks, supplemented by clinical examination of the skin (localization and type of eruption), and follow-up of the course of dermatitis during working days and weekends, as well as during holiday periods and sick leave. Sometimes, the determination IgE-specific antibodies in the serum of the patient will be added to examinations.

Safety data sheets (SDSs) may be helpful in detecting exposing chemicals, but it should be remembered that not all components are given in the sheets [139–142]. In a recent study [142], acetone-soluble methacrylates in commercial dental restorative ma-

Table 4. Dental screening series of Chemotechnique Diagnostics (Malmö, Sweden) (*C*), Hermal (Trolab, Reinbeck/Hamburg, Germany) (*T*), and the Finnish Contact Dermatitis Group (*F*). For the abbreviations of (meth)acrylates, see Table 1. Chemotechnique Diagnostics has three dental screening series: a broad series (*B*), and specific series for patients (*P*), and for staff (*S*) (*NI* not included)

Test substance	Concentration in petrolatum or in water (aq.) (%)		
	C	T	F
(Meth)acrylates			
MMA	2 (B, P, S)	2	2
TREGDMA	2 (B, P, S)	2	2
UDMA	2 (B)	NI	2
EGDMA	2 (B, P, S)	2	2
bis-GMA	2 (B, P, S)	2	2
bis-EMA	2 (P)	NI	NI
BUDMA	2 (B, P, S)	NI	2
bis-MA	2 (B)	NI	NI
2-HEMA	2 (B, P, S)	1	2
DMAEMA	0.2 (B, P)	NI	0.2
HDDA	0.1 (B, P)	NI	NI
THFMA	2 (B, P, S)	NI	2
Diurethane dimethacrylate	NI	2	NI
Epoxy resin compounds			
Bisphenol A	NI	1	1
Epoxy resin	0.1 (P)	NI	NI
Acrylate activators, inhibitors, UV filters			
N,*N*-Dimethyl-4-toluidine	5 (B)	2	NI
2-Hydroxy-4-methoxy-benzophenone	10 (B)	NI	NI
N-Ethyl-4-toluenesulphonamide	0.1 (B, P)	NI	0.1
4-Tolyldiethanolamide	2 (B)	NI	2
Methylhydroquinone	1 (B)	NI	1
Hydroquinone	NI	1	1
Camphoroquinone	1 (B)	NI	NI
2(2-Hydroxy-5-methylphenyl)benzotriazol	1 (B, P)	NI	NI
Benzoyl peroxide	NI	1	NI
Metals			
Potassium dichromate	0.5 (B, P)	NI	0.5
Cobalt chloride	1 (B, P)	NI	1
Gold sodium thiosulfate	2 (B, P)	0.25	NI
Potassium dicyanoaurate	NI	0.002 aq.	NI
Nickel sulfate	5 (B, P)	NI	5
Copper sulfate	2 (B)	NI	NI
Palladium chloride	2 (B, P)	1	1
Aluminum chloride hexahydrate	2 (B)	NI	NI
Tin	50 (B)	NI	NI
Mercury	0.5 (B, P, S)	NI	0.5
Ammoniated mercury	NI	1	NI
Mercuric chloride	NI	NI	0.1
Mercury ammonium chloride	NI	NI	1
Amalgam	NI	5	NI
Amalgam alloying metals	NI	20	NI
Ammonium tetrachloroplatinate	NI	0.25	NI
Fragrances, colophony			
Eugenol	2 (B, P, S)	1	2
Colophony	20 (B, P)	NI	20
Balsam of Peru	25 (P)	NI	NI
Menthol	NI	1	NI
Peppermint oil	NI	2	NI
R-(L)-Carvone	5 (P)	NI	NI
Antimicrobials			
Formaldehyde	1 aq. (B)	NI	1 aq.
Glutaraldehyde	0.2 (S)	NI	0.2
Chlorohexidine digluconate	NI	NI	0.5 aq.
Ammonium persulfate	NI	NI	2.5
Anesthetics			
Caine mix III (benzocaine, dibucaine, tetracaine)	NI	NI	10
Rubber chemicals			
Thiuram Mix	NI	NI	1

terials – seven bonding materials, eight DCRs, and two glass ionomers – were identified by gas chromatography with mass-selective detection, and were quantified with liquid chromatography with ultraviolet detection. Information about methacrylates was given in the SDSs for only about half of the products that, according to the analysis, contained methacrylates. This result and corresponding previous results indicate that SDSs need to be improved.

If available, a special data base for dental materials, e.g., the German Info-Dent, would give more detailed information about the products. All the information in Info-Dent about the ingredients of the product was obtained from the manufacturer, mostly in confidence [11].

The clinical diagnosis of occupational ACD is confirmed by patch testing. The dental screening series of Chemotechnique Diagnostics, Trolab, and the Finnish Contact Dermatitis Group are shown in Table 4. These series contain the most common sensitizers in dental materials. If dental acrylics allergy is suspected, but methacrylate compounds in a dental screening series have, nevertheless, displayed negative results, an extensive methacrylate series (one example in Table 1) may give more information about the causative agent. Patch testing with a rubber chemical series (Chemotechnique or Trolab) may also be decisive in some cases [31].

Skin prick tests with or without determination of IgE-specific antibodies in the patient's serum are necessary when type-I allergy is suspected.

Some cases of active sensitization caused by commercial (meth)acrylate patch test substances and the patient's own acrylic products have been reported [143–147]. Previously, three patients at the FIOH were sensitized when higher patch test concentrations of certain acrylate patch test substances were used.

Despite excellent screening series, patch testing with suspected materials, such as dental acrylics and rubber and plastic glove materials, may be necessary. This is because minor components or impurities, and not the main components, may be the cause of sensitization, and also it may reveal new allergenic components. An analysis of the suspected product may also be necessary to detect special impurities possibly left in the manufacturing processes of separate acrylate compounds, e.g., epoxy resin or bisphenol A in the production of epoxy acrylates.

Patch testing with suspected acrylic products is a difficult task because too low a concentration may cause a false-negative patch test result, and too high a concentration may sensitize. It has been suggested that DCRs should be tested at 1–2% petrolatum [148]. In possible further tests with the products, the concentration should not exceed that of any acrylics. Patch tests or use tests with undiluted acrylic products should never be performed, as even a single exposure with undiluted allergen may sensitize [146, 147]. Reports include a patient who had been sensitized from patch testing with undiluted dentin bonding acrylics, and another patient with contact leucoderma from undiluted DCR [5, 147]. In patch testing materials other than those containing acrylics, the recommendations of Jolanki et al. [148] should be followed.

36.8 Hand Protection

In the prevention of occupational contact dermatitis in dental care and laboratory work, it is of essential importance to ensure the cleanliness of the work environment, and to use technical aids that lessen the handling of reactive chemicals and encourage the use of nontouch techniques. Highly sensitizing DCRs containing various methacrylates and other products in dental restoration and monomer liquids containing MMA and other acrylate compounds should never be handled with the bare hands. However, it is difficult to select disposable gloves to protect against chemicals. Many chemicals permeate thick industrial gloves, and thin gloves made basically from the same material are permeated even more rapidly. Thin gloves also break more easily under chemical or mechanical stress and, similar to thick gloves, they may also have holes or defects. Many acrylics quickly penetrate all disposable gloves [149–155].

Permeation studies of NRL and PVC disposable examination gloves showed that these gloves do not give sufficient protection against methacrylates, such as 2-HEMA contained in primers used in dental restoration. Solvents in materials, e.g., acetone or ethyl alcohol, markedly worsen the protection given by the glove [153]. Acetone should be omitted from the dentin bonding materials, as it can penetrate even thick industrial gloves in less than 5 min. It would, therefore, be better to use ethyl alcohol instead, if possible [149–151, 153, 155, 156].

At least double gloving with PVC or NRL gloves should be used for a 15-min task. For tasks lasting 15 min to 30 min, good quality nitrile rubber gloves should be used, preferably as a double layer with other gloves. A simple PE (polyethene) glove under another glove may improve the protection considerably when performing longer tasks. Double-gloving becomes easier if the inner gloves are of a larger size. Against MMA in liquids used in the manufacture of basement sheets of prostheses or bridges, there are hardly any protective glove alternatives available, except laminated gloves, e.g., PE/EVAL(ethylene vinyl alcohol)/PE at present [155]. Gloves contaminated

with uncured acrylic materials should be removed immediately, and the hands washed with water and cleansing agents.

Common protective glove materials usually give sufficient protection against cleansing agents and X-ray developers. Recent studies of the permeation of common hospital chemicals through surgical single-layered and double-layered NRL gloves and single-layered chloroprene (neoprene) gloves showed that potassium hydroxide (45%), sodium hypochlorite (13%), or hydrogen peroxide (30%) did not permeate the gloves. Furthermore, none of glutaraldehyde, chlorhexidine digluconate, or povidone-iodine in the commercial disinfectant solutions studied permeated the gloves [157].

Based on permeation studies, disposable gloves made of NRL or PVC, for example, provide sufficient protection against occasional splashes of disinfectants. Alcohols and formaldehyde permeate these gloves rapidly, and contaminated gloves must be replaced quickly and the hands must be washed. However, in continuous contact, even diluted glutaraldehyde and concentrated hydrogen peroxide permeate thin examination gloves. Chlorhexidine digluconate or povidone are not likely to permeate intact gloves [155].

To prevent NRL allergy, PVC gloves, synthetic rubber gloves, or NRL gloves with a low protein content are recommended. PE gloves under NRL gloves increase the protection and prevent sensitization to glove proteins and chemicals [15, 31, 158].

36.9 Patients

36.9.1 Oral Mucosa

The oral mucosa, like the skin, is exposed to irritants and sensitizers. The allergic reactions can be immediate, type-I reactions, e.g., from contact with NRL, or delayed, type-IV reactions, e.g., from contact with dental metals or acrylics. The term mucosal contact dermatitis has been used for delayed reactions. There is a lesser tendency to sensitization through the mucous membrane than through the skin. A chronic irritant reaction may develop due to repeated or constant exposure to irritant or toxic agents at low concentrations over long periods. Chronic irritant reactions can be seen in areas of the oral mucosa that are in close contact with amalgam or other fillings, possibly from mechanical causes. The clinical appearance of these lesions may be difficult to distinguish from those caused by contact allergy. The diagnosis is based on the exclusion of contact allergy with negative patch tests [5].

The mucosa is considered to be more resistant to irritants than the skin. The reactions to contactants are lessened by saliva, buffers, and possibly yeasts, which can modify the appearance of stomatitis. Regions with inflammation with or without ulcerations beneath removable partial dentures have caused problems for prosthodontists. Potential factors include microbial infection, obstructive sialadenitis, and allergic or irritant reactions to metal frameworks [159].

Contact allergy has been described as a factor in oral lichenoid reactions and recurrent oral ulceration. Some investigators have suggested that allergic factors are involved in patients with the burning mouth syndrome [160], while others have not [161]. Allergic factors are probably of minor importance in most cases of burning mouth syndrome, but may have contributed to the symptoms of some patients [5].

36.9.2 Allergic Contact Stomatitis and Cheilitis

36.9.2.1 Clinical Picture and Symptoms

The subjective symptoms of patients with allergic contact stomatitis (ACS) are often more prominent than the clinical signs. The complaints include burning and stinging sensations, numbness, soreness, and loss of taste. The clinical appearance varies from barely visible changes to mild or severe erythema and edema. Lingual papillae may disappear and the mucosa may look smooth, waxy and glazy, and show edema. If vesicles appear, they rupture quickly and form erosions [162].

In allergic reactions to base materials of dentures, there is a clear border between the reddish inflamed mucosa covered by the denture and the adjacent uninvolved area. The clinical appearance due to an ill-fitting plate may be similar, and patch testing is, therefore, necessary. Similarly, ACS or allergic contact cheilitis from dental metals or acrylics often shows a distinct border just around the treated tooth, but lichenoid reactions without allergy are also possible. ACS may also mimic oral changes caused by vitamin deficiency and some systemic diseases. ACS is often accompanied by cheilitis [163]. The clinical appearance includes dryness, scaling, fissuring, and angular cheilitis. It can also be caused by contactants applied to the lips. Lips rarely show edema or vesiculation. Allergic contact cheilitis does not have a boundary of normal skin immediately adjacent to the vermilion border, in contrast to perioral dermatitis, which is an endogenous skin disease. Exogenous

perioral dermatitis, on the other hand, can develop from allergy to dental products [5, 164].

36.9.2.2 Causative Agents

Acrylics and Other Plastic Chemicals

Dental patients are exposed to uncured acrylic monomers for only short periods. Therefore, they are at much less risk of developing allergy than the dentists or dental nurses. Accordingly, sensitization of patients from dental acrylics other than prosthetic devices is rare [164, 165].

In the manufacture of removable dental prostheses, polymerization may remain incomplete and leave, e.g., MMA monomer in the denture, possibly causing sensitization. The heat-cured method of dentures induces more complete polymerization than the cheaper cold-cured methods, which may leave more residual monomer in the acrylate-based denture. In a German study, 0.3–4.4% residual MMA monomer was identified in all of the dental plastics investigated [54, 166].

Fisher showed that the sensitizing agent of acrylic prostheses was MMA monomer, but thought that heat-cured dentures were not allergenic [34]. Later, Crissey [167] reported allergic denture sore mouth or stomatitis from heat-cured prostheses. Kaaber [168] has reported 18 cases of MMA-induced prosthesis stomatitis. Aphthous ulcerations have been reported from TREGDMA [169]. Edema and burning sensations in both lips have been reported from a prosthesis, which, according to the manufacturer, contained in its powder component polymethylmethacrylate, benzoyl peroxide, cadmium, and ferric salts, and in the liquid MMA, EGDMA, and hydroquinone. On patch testing, the patient reacted to MMA, 2-HEMA, 2-HPMA, and EGDMA. When she started to use a dental prosthesis made of nickel and chromium, the edema of the lips resolved [170]. Edema and ulceration of the lips from 2-HEMA and TREGDMA was reported by Agner and Menné [171], and vesiculation of the lips and perioral skin from TREGDMA and bis-GMA was reported by Niinimäki et al. [172]. Also, more generalized reactions from the use of prostheses have been described, i.e., chronic urticaria without mucosal or perioral symptoms [173], and stomatitis and edema of the tongue, lips, eyelids, and hands [174]. Dental prostheses with 5–11 times higher content of residual monomer than in heat-cured dentures are also in general use. Allergic denture stomatitis may be encountered more often than previously believed [5]. Several other case reports have been published [96, 169, 175–179].

A female patient displaying a positive patch test to MMA first developed contact stomatitis from one prosthesis, but became symptomless when she used a prosthesis made of Vulcanite rubber. After more teeth were removed, a new complete upper and lower prosthesis was needed. The new prosthesis gradually began to cause worsening stomatitis with burning, itching, and erythema of the oral mucous membrane. The patient also had itching on her lips and on a small skin area around the mouth. The oral symptoms were accompanied by generalized itching and occasional whealing on her lower elbows. On patch testing, she reacted to MMA, EGDMA, 1,4-butanediol diacrylate, and 2-HEMA. In addition, her prostheses also gave positive reactions. The patient's prosthesis was coated with LPH Lack, and UV-light curing was performed for 7 h. She was able to use her prosthesis for half a year without any symptoms of stomatitis, and after relaquering for at least 8 months more [54].

Another female patient developed gingivitis, stomatitis, and perioral dermatitis after insertion of a temporary crown made of restorative two-component material. The base paste and catalyst of the crown contained three methacrylates, i.e., a proacrylate, which is a modification of bis-GMA; a triacrylate, which is saturated aliphatic tricyclic methacrylate; and urethane methacrylate. On patch testing, she reacted to bis-GMA, and other epoxy diacrylates and methacrylates, as well as to the base paste and catalyst of the crown. Allergic reactions were probably elicited by bis-GMA, a cross-reacting methacrylate or other methacrylates in the temporary crown [165].

Only two cases of extra-oral manifestations of delayed allergy ascribed to bis-GMA have been reported. One patient, who developed a measles-like rash, itching, open blisters, and mild respiratory distress but not stomatitis, was reported at the end of the 1970s. After the allergen was removed, complete recovery occurred in 6 months [180]. A recent report described a 12-year-old boy with itchy, relapsing dermatitis on his limbs, trunk, and face. A few days after remodeling of the connections of his orthodontic device, a new, more severe vesicular eruption appeared. He had worn this appliance for over 1 year without any changes in the oral mucosa. On patch testing, he reacted to bis-GMA and *p-tert*-butyl-phenolformaldehyde resin. He also reacted to the bonding paste, which contained bis-GMA. After removal of the orthodontic prosthesis, the dermatitis disappeared within 2 months [181].

Mucosal symptoms caused by *additives* in dental plastics are even rarer than those caused by dental acrylics. Kaaber et al. [182] reported one positive patch test reaction to *N*, *N*-dimethyl-4 toluidine

among 53 denture wearers. Tosti et al. [183] and Verschueren and Bryunzeel [184] reported on patients who had denture sore mouth syndrome from the same chemical. (di)Benzoylperoxide has also been described as a cause of stomatitis [64]. Hydroquinone has been reported on rare occasions to cause gingivostomatitis [185].

Bisphenol A has been reported to cause burning mouth syndrome in a patient. The denture used was of unknown composition, but the patient showed a positive patch reaction to bisphenol A and epoxy resin. It was hypothesized that the epoxy resin used for repairing the denture caused the sensitization [186].

A patient possibly sensitized to epoxy resin at the age of 15 developed painful swelling of oral mucosa for half a day following root canal treatment with product AH 26 (Dentsply De Trey, Germany), which, according to the manufacturer, contains DGEBA-epoxy resin, but not bis-GMA. Two years later, she had developed chronic stomatitis, beginning a few hours after insertion of provisional dental bondings, which were subsequently removed. Patch testing in two sessions showed positive reactions to bis-GMA, and epoxy resin, bisphenol F epoxy resin, and a weak reaction to diphenylmethane-4,4´-diisocyanate. Dental restorations free of plastic materials and new amalgam fillings were inserted, and these were tolerated without any side-effects [187]. Allergic contact dermatitis caused by bis-GMA and associated with sensitivity to epoxy resin has been reported in dental patients by Carmichael et al. [188].

Metals

Mercury amalgam allergy has aroused a great deal of controversy. Previously, it was considered to be a rare sensitizer, but later several studies have shown it to be much more common [189–193]. Many patients with allergic ACS or oral lichen planus (OLP) have become symptomless after the removal of their amalgam restoration [189–193]. The role of dental amalgam in the etiology of OLP or oral lichenoid lesions (OLLs) remains controversial. Some authors have reported that two-thirds of the patients with OLP or OLL have allergy to mercury, whereas other studies show much lower figures [194, 195]. Martin et al. [196] suggest that the corrosion of amalgams and the presence of a galvanic effect from dissimilar metals in continuous contact (bimetallism) are associated with an increased risk of OLL. Amalgam may induce OLL without an allergic mechanism too. OLL may be one disease or a number of similar immunologic or other responses to various stimuli, such as mercury from corroding amalgam fillings [197].

In a study by Athavale et al. [198], 55 patients with OLL were referred for patch testing due to suspected allergy to dental metals (ammoniated and metallic mercury, salts of gold, platinum, palladium, zinc, and copper). Of these 55 patients, 25 (45%) had a relevant positive reaction. Allergy to mercury, and to a lesser extent to gold, was potentially relevant to OLL. Compared with other studies [109], the proportion of patients who were patch test positive to mercury was lower, but more patients reacted to gold. On follow-up, eight of the nine who had their dental metals removed improved after 1 year. The possibility also remained that the replacement of the amalgam removed a physical agent that was causing OLL by an irritant mechanism. The authors concluded that type-IV allergy to mercury in dental amalgam, or to a lesser extent to gold in dental restorations seems to be relevant to the causation of OLL in some patients, but would not be the only mechanism for inducing the condition [198].

In a previous study [199] of 84 patients with typical OLL lesions adjacent to amalgam fillings, encouraging results were obtained. The patch tested metals or metal salts included metallic mercury, ammoniated mercury, mercuric chloride, in some cases phenyl mercuric nitrate, and amalgam discs. Of 84 patients, 33 (39%) had positive patch test findings. Of the 33 patch tested patients, 30 underwent replacement of their amalgam fillings, and 28 (87%) patients experienced improvement of their symptoms and signs within 3 months. The authors concluded that, in some cases, mercury allergy is a factor in the pathogenesis of OLL. It has also been suggested that the removal of dental amalgam is an important therapeutic procedure, even if OLLs are not adjacent to the dental amalgam fillings [200].

Gold salts can be strong sensitizers, but allergy to metallic gold has been considered to be rare. In a study by Ahlgren et al. [201], 102 patients referred for patch testing due to suspected contact dermatitis showed that there was a positive relationship between contact allergy to gold and the presence and amount of dental gold alloys. Metallic gold in dental crowns and restorations has been reported to cause stomatitis and gingivitis [108, 202]. Patch tests for allergy to gold should include gold sodium thiosulfate, GSTS [203], but not gold trichloride [204]. Instead, gold leaf, metallic gold, or gold scrapings may give false-negative results [64]. Metals other than gold may also be the cause of gold jewelry dermatitis or stomatitis, because gold alloys contain variable amounts of other metals as well, including nickel, copper, zinc, silver, or palladium. In a Finnish study [105], 12.4% of patients were positive to GSTS; 25%

had symptoms from jewelry or dental restorations. As in the above-mentioned study, dental gold was concluded to be able to cause OLL [198] and possibly to contribute to burning mouth syndrome in some patients. However, mechanisms other than allergy are often involved in OLL and burning mouth syndrome. Despite this fact, it may not be wise to use golden dental restorations for patients with allergic patch test reactions to GSTS, or to remove restorations from symptomless GSTS allergic patients [5].

In general, *nickel*-sensitive persons have been found to tolerate orthodontic treatment with nickel-containing devices without symptoms. However, stomatitis and systemically induced contact dermatitis from metal wire in orthodontic devices have been reported [191, 205]. Stainless steel tools have very seldom been reported to cause allergic contact dermatitis, although intraoral stainless steel appliances may, in even rarer cases, induce systemic contact dermatitis without stomatitis [206, 207]. On the other hand, nickel allergy may be local and appear only as mucosal inflammation.

Palladium is being used increasingly in industry, jewelry making, and dentistry, and is becoming more common after the EU directive restricted the use of nickel in all products that are in direct contact with the skin. In a study [208] of 4,446 patients patch tested during 1991–2000, 2.3% of the men and 6.7% of the women showed a positive reaction to palladium. Simultaneous sensitization to nickel was common, and the number of those sensitized only to palladium was small. Patch test reactions to palladium chloride may reflect cross-reactivity to nickel sulfate [112, 209]. Patients allergic to palladium chloride tolerate skin contact and, apparently, also mucosal contact with metallic palladium [210]. It is, therefore, uncertain whether metallic palladium in the mouth could be dissolved into its salts and induce stomatitis in patients with dental devices containing palladium. Relatively few cases of relevant palladium-induced allergy have been reported [209, 211]. Koch and Baum [212] reported a patient with ACS due to combined allergy to palladium and platinum from a dental alloy. In addition, contact stomatitis, urticarial, and lichenoid reactions have also been reported [213, 214].

Cobalt and *chromium* allergy seldom originate from dental devices. Fisher reported on a patient whose chrome-cobalt pins used to fasten porcelain teeth to acrylic dentures induced extensive stomatitis and cheilitis [64]. A patient allergic to cobalt in a metal denture developed hand dermatitis [215]. A few cases of systemic contact dermatitis from dental products containing chromium have been reported [205, 216, 217].

Although allergy to *copper* can be considered rare [114, 115], sensitization to copper may have contributed to OLL at least in some cases [64, 218–222]. Koch and Baum [212] reported on a patient who had ACS due to concomitant sensitization to palladium and *platinum*. Some reports suggest that *titanium* may act as an allergen [223, 224]. *Indium* and *iridium* can be used in dental amalgams, as well as in white gold, onto which porcelain is fused in making dental crowns and bridges. Marcusson et al. [225] reported several patients with suspected sensitivity to dental materials, and who, on patch testing, reacted to indium and iridium. Indium isotopes used medically have been reported to cause anaphylactoid reactions.

Vilaplana et al. [96] reported allergic patch test reactions to various *rare metals*, such as rhodium, beryllium, copper, and zinc, in addition to allergic reactions to nickel and mercury. A report on two patients indicates that beryllium may cause ACS and gingivitis [226]. It has also been suggested that beryllium should not be used in dental alloys [227]. Müller-Quernheim et al. [228] reported on a dental technician who was thought to have developed berylliosis from occupational exposure to beryllium.

Manganese will, in future, be increasingly used in the manufacture of dental prostheses [229]. Although manganese has been suggested to have limited potential to cause sensitization [230], sensitization to manganese should, nevertheless, be remembered as a cause of stomatitis in patients wearing dental prostheses. Recently [231], a patient with ACS probably from sensitization to manganese has been reported. The prosthesis was made of chromium-cobalt alloy, which contained 64.8% cobalt, 28.5% chromium, 5.3% molybdenum, 0.5% silica, 0.5% manganese, and 0.4% carbon. On extensive patch testing, the patient reacted only to manganese chloride at 5% in pet. and 15 controls were negative to manganese. She was fitted with a manganese-free denture and remained symptomless thereafter.

Other Compounds

Impression compounds are rare agents that cause oral mucosal symptoms. Two cases of contact allergy have been reported caused by a catalyst in a silicon-based material [123]. Beyer and Belsito [232] reported allergic gingival hyperplasia from silicon tetrachloride used as curing cement in a porcelain crown. Alginates have not caused any definite cases of sensitization [124].

Propolis, made by bees to build, protect, and repair hives, is used in cosmetic and medicinal preparations because of its antiseptic, anti-inflammatory, and an-

esthetic properties. Its therapeutic qualities have been well documented for intraoral treatment [233]. A patient treating her recurrent oral ulcerations with an alcoholic solution of propolis 25% as a mouthwash twice daily has been reported. Two days after starting the treatment, she developed labial edema, oral pain and swelling, dysphonia, and mild dyspnea. On patch testing, propolis as well as 25% mouthwash produced a positive reaction. A few cases of cheilitis and other intraoral conditions have been reported. As a result of its possibly increased use in oral preparations, propolis should be taken into consideration as a possible cause of intraoral allergic symptoms [234].

When *eugenol* is used in dental preparations, including impression pastes, surgical packing, and cements, it may cause contact urticaria, gingivitis, and stomatitis [120, 121, 235, 236]. Three cases of eugenol allergy have been reported; in one of the patients, a eugenol impression paste produced allergic cheilitis and ACS [120].

Colophony or rosin may also be included in various dental materials (see Sect. 36.3.2, Causative Agents). A patient with contact stomatitis from colophony has been reported [117], as well as a case of systemically induced contact dermatitis caused by dental rosin [236].

Rubber chemicals in dentists' rubber gloves coming into contact with the skin of rubber-chemical-allergic patients during operations or restorative treatment may induce relatively long-lasting swollen dermatitis on the contact areas on the face.

Allergenic compounds in *toothpastes* may also cause cheilitis [237, 238].

36.9.3 Immediate Reactions

36.9.3.1 Proteins in Natural Rubber Latex

NRL gloves are, generally, the most common cause of type-I allergy and contact urticaria on the skin, especially in health care workers and dental personnel [129]. Because immediate allergy to NRL is quite common in the general population, dental patients are also a special risk group when one remembers that mucosal contact usually gives a stronger reaction than skin contact. Dental patients should always be asked about their possible NRL allergy. No other NRL rubber materials, e.g., dams, should be used if latex allergy is present.

36.9.3.2 Gutta-Percha

Boxer et al. [239] reported on an NRL-allergic dental hygienist who underwent root canal surgery. During the operation, gutta-percha points were inserted into a maxillary molar. Despite of the avoidance of NRL gloves, the patient reported immediate discomfort, lip and gum swelling, a throbbing sensation around the tooth, and diffuse urticaria. Persistent oral discomfort and urticaria followed. The gutta-percha was removed 4 weeks later, and the patient experienced immediate relief of her oral discomfort. Urticarial lesions disappeared in a few hours. The authors were not able to demonstrate an allergic prick test or IgE antibodies to gutta-percha. NRL and gutta-percha represent examples of isomerism. Both are HMW polymers and are structured from the same basic units [240]. They are derived from trees of the same botanical family, and may, thus, have potential for cross-reactivity [239].

36.9.3.3 Fibrin Tissue

A patient who developed urticaria and shortness of breath 1 h after dental examination and tooth extraction has been reported [241]. The patient's extraction socket had been filled with a commercial fibrin tissue to stop bleeding. The cause was believed to be the bovine protein of the fibrin tissue. Another similar case has also been reported [242].

36.9.3.4 Metals

Nickel and cobalt are not common causes of contact urticaria. In rare cases, nickel has caused both delayed and immediate allergy with contact urticaria, rhinitis, asthma, and contact dermatitis [243]. A case of chronic urticaria has been reported from a nickel-containing dental prosthesis [5]. Platinum is a strong type-I allergen [244, 245]. Iridium, another metal of the platinum group, has been reported to induce respiratory allergy and contact urticaria [246]. Also, other metals of the platinum group, such as ruthenium, rhodium, and palladium, have caused immediate allergy [247, 248]. Mercury salts [249] and sodium fluoride [250] present in 31% of the toothpastes sold in Finland [237] have caused contact urticaria.

36.9.3.5 Formaldehyde

Formaldehyde is a rather rare cause of immediate allergy [251], but has caused anaphylaxis after the application of formaldehyde-containing tooth fillings [252]. The patient also had specific serum IgE antibodies to formaldehyde, but prick and patch tests were negative. At least 15 patients [253] have been reported to have developed urticaria or anaphylaxis

from formaldehyde released from root-canal disinfectants, and most of these cases were due to paraformaldehyde-containing root canal fillings. Of the 15 reported cases, 11 displayed anaphylaxis to formaldehyde, suggesting that type-I allergy caused by formaldehyde in tooth fillings tends to provoke life-threatening symptoms. Specific IgE to formaldehyde in the patients' sera was clearly elevated in all six cases tested, and three other patients showed positive formaldehyde prick tests. A characteristic feature of the type-I allergic response was that at least 7 of the 15 reported patients presented with allergic symptoms 2–12 h after dental treatment with paraformaldehyde. This is probably because formaldehyde is gradually released from water-soluble paraformaldehyde, and gradually penetrates the dentin, and is, thus, increasingly being present in the circulating blood, finally in amounts able to trigger symptoms. Of 13 tested patients, 7 also showed positive reactions to formaldehyde, indicating they had combined type-I and type-IV allergy to formaldehyde. The authors also suggest that direct mucous membrane contact or direct infusion into the blood plays an important role in the development of type-I allergy [253]. Paraformaldehyde-containing root canal medications have not been used in Finland, for example, for about 15 years.

36.9.3.6 Chlorhexidine

The potential risk of anaphylactic reactions from the application of chlorhexidine has been well known since the 1980s [254, 255]. In 1986, Ohtoshi et al. [256] demonstrated IgE antibodies in the sera of eight patients with anaphylaxis caused by chlorhexidine. Today, there are numerous reports of anaphylaxis due to the chemical (reviewed by Krautheim et al. [136]). Chlorhexidine has caused severe anaphylactic reactions in two dental patients [257, 258]. Both were healthy and unaware of their sensitivity. The first patient developed anaphylaxis when chlorhexidine liquid was sprayed into the cavity after the extraction of a wisdom tooth, the other one suffered from pericoronitis and developed anaphylaxis when Hibitane Dental Gel 1% (chlorhexidine) was applied to the gingival pocket. Krautheim et al. [136] analyzed the reported previous anaphylactic reactions caused by chlorhexidine and suggested that patients with previous sensitization to chlorhexidine and with relatively mild contact dermatitis are at risk of severe immediate-type reactions during their following contacts with the chemical. Chlorhexidine may cause anaphylaxis through the mucosal route at a much lower concentration than elsewhere, generally as low as 0.05%. The Japanese Ministry of Health recommended avoiding the use of chlorhexidine on mucous membranes in 1984.

36.9.4 Investigations

The investigations have focused on the same work tasks as in the cases of suspected occupational dermatoses of dental care and dental laboratory personnel. In addition to patch and prick tests, as well as determinations of specific IgE antibodies in the sera, the examination and follow-up of the mucous membranes of the mouth is important. In some cases, biopsies are necessary to exclude other diseases of the mucous membranes.

References

1. Estlander T (1990) Occupational skin disease in Finland. Observations made during 1974–1988 at the Institute of Occupational Health, Helsinki (thesis). Acta Derm Venereol Suppl (Stockh) 155:1–263
2. Kanerva L, Estlander T, Jolanki R (1994) Occupational skin allergy in dental profession. Dermatol Clin 12: 517–532
3. Kanerva L, Estlander T, Jolanki R (1995) Dental problems. In: Guin JD (ed) Practical contact dermatitis: a handbook for the practitioner. McGraw-Hill, New York, pp 397–432
4. Kanerva L, Estlander T, Jolanki R, Alanko K (2000) Dermatitis from acrylate compounds in dental personnel. In: Menné T, Maibach HI (eds) Hand eczema, 2nd edn. CRC Press, Boca Raton, Fla., pp 251–274
5. Kanerva L (2001) Skin disease from dental Materials. In: Rycroft RJG, Menné T, Frosch PJ, Lepoittevin J-P (eds) Textbook of contact dermatitis. Springer, Berlin Heidelberg New York, pp 841–881
6. Wrangsjö K, Österman K, van Hage-Hamsten M (1994) Glove-related skin symptoms among operating theatre and dental care unit personnel (II). Clinical examinations, tests and laboratory findings indicating latex allergy. Contact Dermatitis 30:139–143
7. Wrangsjö K, Swartling C, Meding B (2001) Occupational dermatitis in dental personnel: contact dermatitis with special reference to (meth)acrylates in 174 patients. Contact Dermatitis 45:158–161
8. Munksgaard EC, Hansen EK, Engen T, Holm U (1996) Self reported occupational dermatological reactions among Danish dentists. Eur J Oral Sci 104:396–402
9. Hill JV, Grimwood RE, Hermesch CB, Marks JG Jr (1998) Prevalence of occupationally related hand dermatitis in dental workers. J Am Dent Assoc 129:212–217
10. Lönnroth EC, Shahnavaz H (1998) Adverse health reactions in skin, eyes, and respiratory tract among dental personnel in Sweden. Swed Dent J 22:33–45
11. Rustemeyer T, Frosch PJ (2000) Occupational contact dermatitis in dental personnel. In: Kanerva L, Elsner P, Wahlberg JE, Maibach HI (eds) Handbook of occupational dermatology. Springer, Berlin Heidelberg New York, pp 899–905

12. Wallenhammar LM, Örtengren U, Andreasson H, Barregard L, Bjorkner B, Karlsson S, Wrangsjo K, Meding B (2000) Contact allergy and hand eczema in Swedish dentists. Contact Dermatitis 43:192–199
13. Lindberg M, Silverdahl M (2000) The use of protective gloves and the prevalence of hand eczema, skin complaints and allergy to natural rubber latex among dental personnel in the county of Uppsala, Sweden. Contact Dermatitis 43:4–8
14. Alanko K, Estlander T, Jolanki R (2003) Allergologia hammashoidossa (Allergology in dental care, in Finnish). In: Autti H, LeBell Y, Meurman JH, Murtomaa H (eds) Therapia odontologica. Academica, Helsinki, pp 288–296
15. Alanko K, Susitaival P, Jolanki R, Kanerva L (2004) Occupational skin disease among dental nurses. Contact Dermatitis 50:77–82
16. Rao R, Shenoi SD (2004) Dermatological problems in dental health personnel. Contact Dermatitis 50:252
17. Hogan D, Hamman C, Rodgers P, Siew C (2004) Occupationally based type IV hypersensitivity in dental professionals. Contact Dermatitis 50:141
18. Kanerva L, Estlander T, Jolanki R, Pekkarinen E (1992) Occupational pharyngitis associated with allergic patch test reactions from acrylics. Allergy 47:571–573
19. Estlander T, Kanerva L, Kari O, Jolanki R, Mölsa K (1996) Occupational conjunctivitis associated with type IV allergy to methacrylates. Allergy 51:56–59
20. Sala E, Hytönen M, Tupasela O, Estlander T (1996) Occupational laryngitis with immediate or immediate type specific chemical hypersensitivity. Clin Otolaryngol Allied Sci 21:1404–1411
21. Savonius B, Keskinen H, Tuppurainen M, Kanerva L (1993) Occupational respiratory disease caused by acrylics. Clin Exp Allergy 23:416–424
22. Piirilä P, Kanerva L, Keskinen H, Estlander T, Hytönen M, Tuppurainen M, Nordman H (1998) Occupational respiratory hypersensitivity caused by preparations containing acrylates in dental personnel. Clin Exp Allergy 28:1404–1411
23. Piirilä P, Hodgson U, Estlander T, Keskinen H, Saalo A, Voutilainen R, Kanerva L (2002) Occupational respiratory hypersensitivity in dental personnel. Int Arch Occup Environ Health 75:209–216
24. Estlander T, Rajaniemi R, Jolanki R (1984) Hand dermatitis in dental technicians. Contact Dermatitis 10:201–205
25. Rustemeyer T, Frosch PJ (1996) Occupational skin disease in dental laboratory workers. (I) Clinical picture and causative factors. Contact Dermatitis 34:123–133
26. Meding B, Hosseiny S, Wrangsjö K, Andersson E, Hagberg S, Wass U, Toren K, Brisman J (2004) Hand eczema, skin exposure and glove use in dental technicians. Contact Dermatitis 50:203
27. Mürer AJL, Poulsen OM, Roed-Petersen J, Tüchsen F (1995) Skin problems among Danish dental technicians. A cross-sectional study. Contact Dermatitis 33:42–47
28. Mürer AJL, Poulsen OM, Tüchsen F, Roed-Petersen J (1995) Rapid increase in skin problems among dental technician trainees working with acrylates. Contact Dermatitis 33:106–111
29. Kanerva L, Mikola H, Henriks-Eckerman M-L, Jolanki R, Estlander T (1998) Fingertip paresthesia and occupational allergic contact dermatitis caused by acrylics in a dental nurse. Contact Dermatitis 38:114–116
30. Kanerva L, Henriks-Eckerman M-L, Estlander T, Jolanki R (1997) Dentist's occupational allergic paronychia and contact dermatitis caused by acrylics. Eur J Dermatol 7:177–180
31. Estlander T, Jolanki R (2005) Allergic contact dermatitis from rubber and plastic gloves In: Boman A, Estlander T, Wahlberg JE, Maibach HI (eds) Protective gloves for occupational use, 2nd edn. CRC Press, Boca Raton, Fla., pp 27–144
32. Stevenson WJ (1941) Methyl-methacrylate dermatitis. Contact Point 18:171–173
33. Moody WL (1941) Severe reaction from acrylic liquid. Dent Digest 47:305
34. Fisher AA (1954) Allergic sensitization of skin and oral mucosa to acrylic denture materials. J Am Med Assoc 156:238–242
35. van der Walle HB (1982) Sensitizing potential of acrylic monomers in guinea pigs. Thesis, Katholieke Universiteit Nijmegen, Krips Repro, Meppel, The Netherlands, pp 1–112
36. Björkner B (1984) Sensitizing capacity of ultraviolet curable acrylic compounds. Thesis, Lund, Sweden, pp 1–78
37. Axell T, Björkner B, Fregert S, Niklasson B (1983) Standard patch test series for screening contact allergy dental materials. Contact Dermatitis 9:82
38. Kanerva L, Estlander T, Jolanki R (1989) Allergic contact dermatitis from dental composite resins due to aromatic epoxy acrylates and aliphatic acrylates. Contact Dermatitis 20:201–211
39. Kanerva L, Turjanmaa K, Estlander T, Jolanki R (1991) Occupational allergic contact dermatitis from 2-hydroxyethyl methacrylate (2-HEMA) in a new dentin adhesive. Am J Contact Dermat 2:24–30
40. Kanerva L, Henriks-Eckerman M-L, Estlander T, Jolanki R, Tarvainen K (1994) Occupational allergic contact dermatitis and composition of acrylates in denting bonding systems. J Eur Acad Derm Venereol 3:157–169
41. Guerra L, Vincenzi C, Peluso AM, Tosti A (1993) Prevalence and sources of occupational contact sensitization to acrylates in Italy. Contact Dermatitis 28:101–103
42. Tosti A, Guerra L, Vincenzi C, Peluso AM (1993) Occupational health hazards from synthetic plastics. Toxicol Ind Health 9:493–502
43. Cleenewerk MB (1997) Current features of allergic occupational dermatoses. Rev Fr Allergol Immunol Clin 37:617–633
44. Kanerva L Jolanki R, Estlander T (1997) 10 years of patch testing with the (meth)acrylate series. Contact Dermatitis 37:255–258
45. Björkner B (1989) Kontaktallergi för ultraviolett härdande akrylatprodukter I färger och lacker (in Swedish). Arbete och Hälsa 20:1–39
46. Bowen RL (1962) Dental filling material comprising vinyl silane treated fused silica and a binder consisting of the reaction product of bisphenol A and glycidyl acrylate. US Patent 3,066,112
47. Rustemeyer T, de Groot A, von Blomberg BME, Frosch PJ, Scheper RJ (1998) Cross-reactivity patterns of contact sensitizing methacrylates. Toxicol Appl Pharmacol 148:83–90
48. van der Walle HB, Bensink T (1982) Cross reaction pattern of 26 acrylic monomers in guinea pig skin. Contact Dermatitis 8:376–382
49. Kanerva L, Estlander T, Jolanki R (1997) Occupational allergic contact dermatitis of dental nurse caused by acrylic tri-cure glass ionomer. Contact Dermatitis 37:49–50
50. ADEPT report (1990) Pertinent information on cosmetic, adhesive, and restorative dentistry. ADEPT Institute, Santa Rosa, Calif., 1:33–44

51. Kanerva L, Henriks-Eckerman M-L, Estlander T, Jolanki R, Tarvainen K (1994) Occupational allergic contact dermatitis and composition of acrylates in dentin bonding systems. J Eur Acad Derm Verereol 3:157–168
52. Finnish Advisory Board of Chemicals (1992) Acrylate compounds: uses and evaluation of health effects. Government Printing Centre, Helsinki, Finland, pp 1–60
53. Kanerva L, Estlander T, Jolanki R, Tarvainen K (1993) Occupational contact dermatitis caused by exposure to acrylates during work with dental prostheses. Contact Dermatitis 28:268–275
54. Kanerva l, Tarvainen K, Jolanki R, Estlander T (1995) Successful coating of an allergenic acrylate-based dental prosthesis. Am J Contact Dermat 6:24–27
55. Schnuch A, Geier J (1994) Kontaktallergene bei Dentalberufen. Dermatosen 42:253–255
56. Belsito DV (1987) Contact dermatitis to ethyl-cyanoacrylate-containing glue. Contact Dermatitis 17:234–236
57. White IR (1990) Adhesives. In: Adams RM (ed) Occupational skin disease, 2nd edn. Saunders, Philadelphia, Pa., pp 395–407
58. Guin JD, Baas K, Nelson-Adesokan P (1998) Contact sensitization to cyanoacrylate adhesive as a cause of severe onychodystrophy. Int J Dermatol 37:31–36
59. Tomb RR, Lepoittevin J-P, Durepaire F, Grosshans E (1993) Ectopic contact dermatitis from ethyl cyanoacrylate instant adhesives. Contact Dermatitis 28:206–208
60. Lozewicz S, Davison AG, Hopkirk A, Burge PS, Boldy DA, Riordan JF, McGivern DV, Platts BW, Davies D, Newman Taylor JA (1985) Occupational asthma due to methyl methacrylate and cyanoacrylates. Thorax 40:836–839
61. Smith DC (1959) The acrylic denture base – the peroxide concentration in dental polymers. Br Dent J 107:62–67
62. Calnan CD, Stevenson CJ (1963) Studies in contact dermatitis XV: dental materials. Trans St John's Hosp Dermatol Soc 49:9–26
63. Kanerva L, Tarvainen K, Estlander T, Jolanki R (1994) Occupational allergic contact dermatitis caused by mercury and benzoyl peroxide. Eur J Dermatol 4:359–361
64. Fisher AA (1986) Contact dermatitis, 3rd edn. Lea and Febiger, Philadelphia, Pa.
65. Jager M, Balda BR (1979) Loosening of hip prosthesis at contact allergy to benzoyl peroxide. Arch Orthop Trauma Surg 94:175–8
66. Vincenzi G, Cameli N, Vassipoulou A, Tosti A (1991) Allergic contact dermatitis due to benzoyl peroxide in arm prosthesis. Contact Dermatitis 24:66–67
67. Quirce S, Olaguibel JM, Garcia BE, Tabar AI (1993) Occupational airborne contact dermatitis due to benzoylperoxide. Contact Dermatitis 29:165
68. Kanerva L, Jolanki R, Estlander T (1993) Dentist's occupational allergic contact dermatitis caused by coconut diethanolamide, N-ethyl-4-toluene sulfonamide and 4-tolyldiethanolamine. Acta Derm Venereol (Stockh) 73:126–129
69. Malanin K (1993) Active sensitization to camphoroquinone and double active sensitization to acrylics with long-lasting patch test reactions. Contact Dermatitis 29:284–285
70. Jolanki R (1991) Occupational skin diseases from epoxy compounds. Epoxy resin compounds, epoxy acrylates and 2,3-epoxypropyl trimethyl ammonium chloride (thesis). Acta Derm Venereol Suppl (Stockh) 169:1–80
71. Jolanki R, Kanerva L, Estlander T (1995) Occupational allergic contact dermatitis caused by epoxy diacrylate in ultraviolet-light-cured paint, and bisphenol A in dental composite resin. Contact Dermatitis 33:94–99
72. Aalto-Korte K, Alanko K, Henriks-Eckerman M-L, Estlander T, Jolanki R (2003) Allergic contact dermatitis from bisphenol A in PVC gloves. Contact Dermatitis 49:202–205
73. Cronin E (1980) Contact dermatitis. Churchill Livingstone, Edinburgh, pp 1–915
74. Bardazzi F, Melino M, Alagna G, Veronesi S (1986) Glutaraldehyde dermatitis in nurses. Contact Dermatitis 14:319–320
75. Nethercott JR, Holness DL, Page E (1988) Occupational contact dermatitis due to glutaraldehyde in health care workers. Contact Dermatitis 18:193–196
76. Cusano F, Luciano S (1993) Contact allergy to benzalkonium chloride and glutaraldehyde in a dental nurse. Contact Dermatitis 28:127–127
77. Kanerva L Miettinen P, Alanko K, Estlander T, Tupasela O, Jolanki R (2000) occupational allergic contact dermatitis from glyoxal, glutaraldehyde and neomycin sulfate in a dental nurse. Contact Dermatitis 42:116–117
78. Flyvholm M-A (1997) Formaldehyde exposure at the workplace and in the environment. Allergologie 5:225–231
79. Flyvholm M-A (2000) Formaldehyde and formaldehyde releasers. In: Kanerva L, Elsner P, Wahlberg JE, Maibach HI (eds) Handbook of occupational dermatology. Springer, Berlin Heidelberg New York, pp 474–478
80. Elsner P, Pevny I, Burg G (1990) Occupational contact dermatitis due to glyoxal in health care workers. Am J Contact Dermat 1:250–253
81. Suhonen R (1980) Contact allergy to dodecyl-di-(aminoethyl)glycine (Desimex I). Contact dermatitis 6:290–291
82. Foussereau J, Samsoen M, Hecht MT (1983) Occupational dermatitis to Ampholyt G in hospital personnel. Contact Dermatitis 9:233–234
83. Estlander T, Kanerva L, Jolanki R (1989) Occupational skin sensitization to the antimicrobials ortho-benzyl para-chlorophenol and Ampholyte 103G. In: Frosch P, Dooms-Goossens A, Lachapelle J-M, Rycroft RJG, Scheper RJ (eds) Current topics in contact dermatitis. Springer, Berlin Heidelberg New York, pp 88–91
84. Timmer C (2000) Antimicrobials and disinfectants. In: Kanerva L, Elsner P, Wahlberg JE, Maibach HI (eds) Handbook of occupational dermatology. Springer, Berlin Heidelberg New York, pp 462–473
85. Snuch A (1997) Benzalkonium chloride. Dermatosen 45:179–180
86. Placucci F, Benini A, Guerra L, Tosti A (1996) Occupational allergic contact dermatitis from disinfectant wipes used in dentistry. Contact Dermatitis 35:306–306
87. Lachapelle J-M (1984) Occupational allergic contact dermatitis to povidone-iodine. Contact Dermatitis 11:189–190
88. Ancona A, de la Torre RS, Macotela E (1985) Allergic contact dermatitis from povidone-iodine. Contact Dermatitis 13:66–68
89. Kudo H, Takahashi K, Suzuki Y, Tanaka T, Miyachi Y, Imamura S (1988) Contact dermatitis from a compound mixture of sugar and povidone-iodine. Contact Dermatitis 18:155–157
90. van Ketel WG, van der Berg WH (1990) Sensitization to povidone-iodine. Dermatol Clin 8:107–109
91. Tosti A, Vincenzi C, Bardazzi F, Mariani R (1990) Allergic contact dermatitis to povidone-iodine. Contact Dermatitis 23:197–198
92. Kanerva L Estlander T (1999) Occupational allergic contact dermatitis caused by povidone-iodine (Betadine). Environ Dermatol 6:101–104

93. White IR, Catchpole HE, Rycroft RJG (1982) Rashes amongst persulphate workers. Contact Dermatitis 8: 168–172
94. van Joost T, Roesyanto ID (1991) Sensitization to persulphates in occupational and non-occupational hand dermatitis. Contact Dermatitis 24: 376–378
95. Kanerva L, Alanko K, Jolanki R, Aalto-Korte K, Estlander T (1999) Occupational allergic contact dermatitis from potassium persulfate. Contact Dermatitis 40: 116–117
96. Vilaplana J, Romaguera C, Cornellana F (1994) Contact dermatitis and adverse oral mucous membrane reactions related to use of dental prostheses. Contact Dermatitis 30: 80–84
97. Ancona A, Ramos M, Suarez R, Macotela E (1982) Mercury sensitivity in a dentist. Contact Dermatitis 8: 218
98. Kanerva L, Komulainen M, Estlander T, Jolanki R (1993) Occupational allergic contact dermatitis from mercury. Contact Dermatitis 28: 26–28
99. Björkner B, Bruze M, Möller H (1994) High frequency of contact allergy to gold sodium thiosulfate. An indication of gold allergy? Contact Dermatitis 30: 144–151
100. Bruze M, Edman B, Björkner B, Möller H (1994) Clinical relevance of contact allergy to gold sodium thiosulfate. J Am Acad Dermatol 31: 579–583
101. Möller H, Larsson Å, Björkner B, Bruze M (1994) The histological and immunohistochemical pattern of positive patch test reactions to gold sodium thiosulfate. Acta Derm Venereol (Stockh) 74: 417–423
102. Bruze M, Hedman H, Björkner B, Möller H (1995) The development and course of test reactions to gold sodium thiosulfate. Contact Dermatitis 33: 386–391
103. McKenna KE, Dolan O, Walsh MY, Burrows D (1995) Contact allergy to gold sodium thiosulfate. Contact Dermatitis 32: 143–146
104. Sabroe RA, Sharp LA, Peachey RDG (1996) Contact allergy to gold sodium thiosulfate. Contact Dermatitis 34: 345–348
105. Räsänen LR, Kalimo K, Laine J, Vainio O, Kotiranta J, Pesola I (1996) Contact allergy to gold in dental patients. Br J Dermatol 134: 673–677
106. Fleming C, Forsyth A, MacKie R (1997) Prevalence of gold contact hypersensitivity in the west of Scotland. Contact Dermatitis 36: 302–304
107. Leow Y-H, Ng S-K, Goh C-L (1998) A preliminary study of gold sensitization in Singapore. Contact Dermatitis 38: 69–70
108. Aro T, Kanerva L, Häyrinen-Immonen R, Silvennoinen-Kassinen S, Konttinen YT, Jolanki R, Estlander T (1993) Long-lasting allergic patch test reaction caused by gold. Contact Dermatitis 28: 276–281
109. Koch P, Bahmer FA (1995) Oral lichenoid lesions, mercury hypersensitivity and combined hypersensitivity to mercury and other metals: histologically-proven reproduction of the reaction by patch testing with metal salts. Contact Dermatitis 33: 323–328
110. Heise H, Beyer H, Rauschenbach D (1997) Ergebnisse der Epikutantestung auf Goldsalze versus Amalgam. Z Dermatol 183: 68–73
111. Isaksson M, Bruze M (2000) Gold. In: Kanerva L, Elsner P, Wahlberg JE, Maibach HI (eds) Handbook of occupational dermatology. Springer, Berlin Heidelberg New York, pp 544–550
112. Liden C (2000) Nickel. In: Kanerva L, Elsner P, Wahlberg JE, Maibach HI (eds) Handbook of occupational dermatology. Springer, Berlin Heidelberg New York, pp 524–533
113. Wahlberg JE (2000) Other metals. In: Kanerva L, Elsner P, Wahlberg JE, Maibach HI (eds) Handbook of occupational dermatology. Springer, Berlin Heidelberg New York, pp 551–555
114. Förström L, Kiistala U, Tarvainen K (1977) Hypersensitivity to copper verified by test with 0.1% $CuSO_4$. Contact Dermatitis 3: 280–281
115. Karlberg A, Boman A, Wahlberg JE (1983) Copper – a rare senstitizer. Contact Dermatitis 9: 134–139
116. Karlberg A-T (2004) Colophony. In: Kanerva L, Elsner P, Wahlberg JE, Maibach HI (eds) Condensed handbook of occupational dermatology. Springer, Berlin Heidelberg New York, pp 312–330
117. Isaksson M, Bruze M, Björkner B, Niklasson B (1993) Contact allergy to Duraphat. Scand Dent Res 101: 49–61
118. Kanerva L, Estlander T (1999) Occupational allergic contact dermatitis from colophony in 2 dental nurses. Contact Dermatitis 41: 342–343
119. Cockayne SE, Murphy R, Gawkrodger DJ (2001) Occupational contact dermatitis from colophonium in dental technician. Contact Dermatitis 44: 42–43
120. Göransson K, Karltorp N, Ask H, Smedberg O (1967) Några fall av eugenolöverkänsligheter (Some cases of eugenol hypersensitivity; in Swedish with English summary). Svensk Tandläk Tidskr 60: 545–549
121. Kanerva L, Estlander T, Jolanki R (1998) Dental nurse's occupational allergic contact dermatitis from eugenol used as a restorative dental material with polymethylmethacrylate. Contact Dermatitis 38: 339–340
122. Yli-Urpo et al (2003) Hammashoidon materiaalit (Dental materials, in Finnish). In: Autti H, LeBell Y, Meurman JH, Murtomaa H (eds) Therapia odontologica. Academica, Helsinki, Finland, pp 1019–1037
123. Ölveti E, Hegedus C (1994) Contact allergy reactions to Silodent impression material (in Hungarian). Fogorv Sz 87: 115–119
124. Rice CD, Barker BF, Kestenbaum T, Dykstra MA, Lumpkin D (1992) Intraoral vesicles occurring after alginate impressions. Oral Surg Oral Med Oral Pathol 74: 698–704
125. Camarasa G (1975) Occupational dermatitis from beeswax. Contact Dermatitis 1: 124
126. Klein CE, Gall H (1991) Type IV allergy to amide-type local anesthetics. Contact Dermatitis 25: 45–48
127. Curley RK, Macfarlane AW, King CM (1986) Contact sensitivity to amide anesthetics lidocaine, prilocaine, and mepivacaine. Arch Dermatol 122: 924–926
128. Suhonen R, Kanerva L (1997) Contact allergy and cross-reactions to prilocaine. Am J Contact Dermat 8: 231–235
129. Jolanki R, Estlander T, Alanko K, Savela A, Kanerva L (1999) Incidence rates of occupational contact urticaria caused by natural rubber latex. Contact Dermatitis 40: 329–331
130. Tarlo SM, Sussman GL, Holness DL (1997) Latex sensitivity in dental students and staff: a cross-sectional study. J Allergy Clin Immunol 99: 396–401
131. Safadi GS, Safadi TJ, Terezhalmy GT, Taylor JS, Battisto JR, Melton Al Jr (1996) Latex hypersensitivity: its prevalence among dental professionals. J Am Dent Assoc 127: 83–88
132. Heese A, Peters KP, Koch HU, Hornstein OP (1995) Häufigkeit und Zunahme von Typ I-Allergien gegen Gummihandschuhe bei Zahnmedicinstudenten (Incidence and increase in type I allergies to rubber gloves in dental medicine students, in German). Hautarzt 46: 15–21
133. Hamann C (1993) Hold the talc, pass the cornstarch. J Am Dent Assoc 124: 14–16
134. Seggev JS, Mawhinney TP, Yunginger JW, Braun SR (1990) Anaphylaxis due to cornstarch surgical glove powder. Ann Allergy 65: 152–155

135. Kanerva L (1997) Contact urticaria from dental products. In: Amin S, Lahti A, Maibach HI (eds) Contact urticaria syndrome. CRC Press/LLC, Boca Raton, Fla., pp 119–128
136. Krautheim AB, Jermann THM, Bircher AJ (2004) Chlorhexidine anaphylaxis: case report and review of the literature. Contact Dermatitis 50:113–116
137. Bhalla M, Thami GP (2003) Acute urticaria due to dental eugenol. Allergy 58:158
138. Hudson LD (1987) Phototoxic reactions triggered by a new dental instrument. J Am Acad Dermatol 17:508–509
139. Foussereau J (1991) Guide de dermato-allergologie professionnelle. Masson, Paris, France
140. Kanerva L, Henriks-Eckerman M-L, Jolanki R, Estlander T (1997) Plastics/acrylics: material safety data sheets need to be improved. Clin Dermatol 15:533–546
141. Henriks-Eckerman M-L, Kanerva L (1997) Product analysis of acrylic resins compared to information given in material safety sheets. Contact Dermatitis 36:164–165
142. Henriks-Eckerman M-L, Suuronen K, Jolanki R, Alanko K (2004) Methacrylates in dental restorative materials. Contact Dermatitis 50:233–237
143. Kanerva L, Estlander T, Jolanki R (1988) Sensitization to patch test acrylates. Contact Dermatitis 18:10–15
144. Kanerva L, Estlander T, Jolanki R (1992) Double active sensitization caused by acrylics. Am J Contact Dermat 3:23–26
145. Kanerva L, Estlander T, Jolanki R (1992) Active sensitization caused by 2-hydroxyethyl methacrylate, 2-hydroxypropyl methacrylate, ethyleneglycol dimethacrylate and N,N-dimethylaminoethyl dimethacrylate. J Eur Acad Derm Venereol 1:165–169
146. Kanerva L, Lauerma A (1998) Iatrogenic acrylate allergy complicating amalgam allergy. Contact Dermatitis 38:58–59
147. Kanerva L, Turjanmaa K, Estlander T, Jolanki R (1991) Occupational allergic contact dermatitis from iatrogenic sensitization by a new acrylate dentin adhesive. Eur J Dermatol 1:25–28
148. Jolanki R, Estlander T, Alanko K, Kanerva L (2004) Patch testing with patient's own work materials. In: Kanerva L, Elsner P, Wahlberg JE, Maibach HI (eds) Condensed handbook of occupational dermatology. Springer, Berlin Heidelberg New York, pp 195–205
149. Pegum JS, Medhurst FA (1971) Contact dermatitis from penetration of rubber gloves by acrylic monomer. Br Med J 2:141–143
150. Waegemaekers THJM, Seutter E, den Arend JACJ, Malten KE (1983) Permeability of surgeons' gloves to methyl methacrylate. Acta Orthop Scand 54:790–795
151. Darre E, Vedel P, Jensen JS (1987) Skin protection against methylmethacrylate. Acta Orthop Scand 58:236–238
152. Munksgaard EC (1992) Permeability of protective gloves to (di)methacrylates in resinous dental materials. Scand J Dent Res 100:189–192
153. Munksgaard EC (2000) Permeability of protective gloves by 2-HEMA and TEGDMA in the presence of solvents. Acta Odontol Scand 58:57–62
154. Mäkelä EA, Väänänen V, Alanko K, Jolanki R, Estlander T, Kanerva L (1999) Resistance of disposable gloves to permeation by 2-hydroxyethyl methacrylate and triethyleneglycol dimethacrylate. Occup Hyg 5:121–129
155. Mäkelä EA, Jolanki R (2005) Chemical permeation through disposable gloves. In: Boman A, Estlander T, Wahlberg JE, Maibach HI (eds) protective gloves for occupational used, 2nd edn. CRC Press, Boca Raton, Fla., pp 299–314
156. Mäkelä EA, Vainiotalo S, Peltonen K (2003) Permeation of 70% isopropyl alcohol through surgical gloves: comparison of standard methods ASTM F 739 and EN 374. Ann Occup Hyg 47:305–312
157. Mäkelä EA, Vainiotalo S, Peltonen K (2003) The permeability of surgical gloves to seven chemicals commonly used in hospitals. Ann Occup Hyg 47:313–323
158. Estlander T, Jolanki R, Kanerva L (2000) Protective gloves. In: Menné T, Maibach HI (eds) Hand eczema, 2nd edn. CRC Press, Boca Raton, Fla., pp 309–322
159. Taylor DT, Morton TH (1991) Ulcerative lesion of the palate associated with removable partial denture castings. J Prosthet Dent 66:213–221
160. Shah M, Lewis FM, Gawkrodger DJ (1996) Contact allergy in patients with oral symptoms: a study of 47 patients. Am J Contact Dermat 7:146–151
161. Helton J, Storrs F (1994) The burning mouth syndrome: lack of role for contact urticaria and contact dermatitis. J Am Acad Dermatol 31:201–205
162. Rietschell RL, Fowler JF Jr (1995) Fisher's contact dermatitis, 4th edn. Williams and Watkins, Baltimore, Md.
163. Ophaswongse S, Maibach HI (1995) Allergic contact cheilitis. Contact Dermatitis 33:365–370
164. Kanerva L, Alanko K (1998) Stomatitis and perioral dermatitis caused by epoxy diacrylates in dental composite resins. J Am Acad Derm 38:116–120
165. Kanerva L, Alanko K, Estlander T (1999) Allergic contact gingivostomatitis from a temporary crown made of methacrylates and epoxy diacrylates. Allergy 54:1316–1321
166. Marx H, Fukui M, Stender E (1983) Zur Frage der Restmonomer-Untersuchung von Prosthesenkunststoffen. Dtsch Zahnärtzl Z 38:550–553
167. Crissey JT (1965) Stomatitis, dermatitis, and denture materials. Arch Dermatol 92:45–48
168. Kaaber S (1990) Allergy to dental materials with special reference to the use of amalgam and polymethylmethacrylate. Int Dent J 40:359–365
169. Guerra L, Vincenzi C, Peluso AM, Tosti A (1993) Role of contact sensitizers in the burning mouth syndrome. Am J Contact Dermat 4:154–157
170. Ruiz-Genao DP, Moreno de Vega MJ, Sánchez Pérez J, Garcia-Diez A (2003) Labial edema due to an acrylic dental prosthesis. Contact Dermatitis 48:273–274
171. Agner T, Menné T (1994) Sensitization to acrylates in a dental patient. Contact Dermatitis 30:249–250
172. Niinimäki A, Rosberg J, Saari S (1983) Allergic stomatitis from acrylic compounds. Contact Dermatitis 9:148
173. Lunder T, Rogi-Butina M (2000) Chronic urticaria from acrylic dental prosthesis. Contact Dermatitis 43:232–233
174. Bauer A, Wollina U (1998) Denture-induced local and systemic reactions to acrylate. Allergy 53:722–723
175. Kanzaki T, Kabasawa Y, Jinno T, Isayama K (1989) Contact stomatitis due to methyl methacrylate monomer. Contact Dermatitis 20:146–148
176. Ölveti E (1991) Contact dermatitis from an acrylic-metal dental prosthesis. Contact Dermatitis 24:57
177. Corazza M, Virgili A, Martina S (1992) Allergic contact stomatitis from methyl methacrylate in a dental prosthesis, with persistent patch test reaction. Contact Dermatitis 26:210–211
178. Fowler JF Jr (1992) Burning mouth caused by dentures. Am J Contact Dermat 3:3–4
179. Kobayashi T, Sakuraoka K, Hasegawa Y, Konohana A, Kurihara S (1996) Contact dermatitis to an acrylic dental prosthesis. Contact Dermatitis 35:370–371

180. Nathanson D, Lockhart P (1979) Delayed extraoral hypersensitivity to dental composite material. Oral Surg Oral Med Oral Pathol 47:329–333
181. Menni S, Lodi A, Coassini D, Boccardi P, Rossini P, Crosti C (2003) Unusual widespread vesicular eruption related dental composite resin sensitization. Contact Dermatitis 48:174
182. Kaaber S, Thulin H, Nielsen E (1979) Skin sensitivity to denture base materials in the burning mouth syndrome. Contact Dermatitis 5:90–96
183. Tosti A, Bardazzi F, Piancastelli E, Brasile GP (1990) Contact stomatitis due to N, N-dimethyl-paratoluidine. Contact Dermatitis 22:113
184. Verschueren GLA, Bryunzeel DP (1991) Allergy to N, N-dimethyl-p-toluidine in dental materials. Contact Dermatitis 24:149
185. Torres V, Mano-Azul AC, Correia T, Soares AP (1993) Allergic cheilitis and stomatitis from hydroquinone in an acrylic dental prosthesis. Contact Dermatitis 29:102–103
186. van Joost TH, van Ulsen J, van Loon LAJ (1988) Contact allergy to denture materials in the burning mouth syndrome. Contact Dermatitis 18:97
187. Koch P (2003) Allergic contact stomatitis from BIS-GMA and epoxy resins in dental bonding agents. Contact Dermatitis 49:104–105
188. Carmichael AJ, Gibson JJ, Walls AWG (1997) Allergic contact dermatitis to bisphenol-A-glycidyldimethacrylate (BIS-GMA) dental resin associated with sensitivity to epoxy resin. Br Dent J 183:297–298
189. Pang PK, Freeman S (1996) Oral lichenoid lesions caused by allergy to mercury in amalgam fillings. Contact Dermatitis 33:423–427
190. Smart ER, Macleod RI, Lawrence CM (1995) Resolution of lichen planus following removal of amalgam restorations in patients proven allergy to mercury salts: a pilot study. Br Dent J 178:108–112
191. Alanko K, Kanerva L, Jolanki R, Kannas L, Estlander T (1996). Oral mucosal diseases investigated by patch testing with a dental screening series. Contact Dermatitis 34:263–267
192. Laine J, Kalimo K, Forssell H, Happonen R-P (1992) Resolution of oral lichenoid lesions after replacement of amalgam restorations in patients allergic to mercury compounds. Br J Dermatol 126:10–15
193. Laine J, Kalimo K, Happonen R-P (1997) Contact allergy to dental restorative materials in patients with oral lichenoid lesions. Contact Dermatitis 36:141–146
194. Koch P, Bahmer F (1999) Oral lesions and symptoms related to metals used in dental restorations: clinical, allergological, and histologic study. Am J Acad Dermatol 41:422–430
195. Scalf LA, Fowler JF Jr, Morgan KW, Looney SW (2001) Dental metal allergy in patients with oral, cutaneous and genital lichenoid lesions. Am J Contact Dermat 12:146–150
196. Martin MD, Broughton S, Drangsholt M (2003) Oral lichen planus and dental materials: a case-control study. Contact Dermatitis 48:331–336
197. Östman PO, Anneroth G, Skoglund A (1994) Oral lichen planus lesions in contact with amalgam fillings: a clinical, histologic, and immunohistochemical study. Scand J Dent Res 102:172–179
198. Athavale PN, Shum KW, Yeoman CM, Gawkrodger DJ (2003) Oral lichenoid lesions and contact allergy to dental mercury and gold. Contact Dermatitis 49:264–265
199. Wong L, Freeman S (2003) Oral lichenoid lesions (OLP) and mercury in amalgam fillings. Contact Dermatitis 48:74–79
200. Pigatto P, Passoni E, Crippa R, Tanzi C, Zerboni R, Brambilla L, Guzzi G, Muratori S (2004) Oral lichenoid reactions and amalgams: no topographical relationship. Contact Dermatitis 50:176
201. Ahlgren C, Ahnlide C, Björkner B, Bruze M, Liedholm R, Möller H, Nilner C (2002) Contact allergy to gold is correlated to dental gold. Acta Derm Venereol (Stockh) 82:41–44
202. Laeijendecker R, van Joost T (1994) Oral manifestations of gold allergy. J Am Acad Dermatol 30:205–209
203. Fowler JF Jr (1987) Selection of patch test materials for gold allergy. Contact Dermatitis 17:23–25
204. Möller H, Ahnlide I, Gruvberger B, Bruze M (2004) Gold trichloride as a marker of contact allergy to gold. Contact Dermatitis 50:176
205. Veien NK, Borchorst E, Hattel T, Laurberg G (1994) Stomatitis or systemically-induced contact dermatitis from metal wire in orthodontic materials. Contact Dermatitis 30:210–213
206. Kerosuo H, Kanerva L (1997) Systemic contact dermatitis caused by nickel in a stainless steel orthodontic appliance. Contact Dermatitis 36:112–113
207. Lyzak WA, Flaitz CM, McGuckin RS, Eichmiller F, Brown RS (1994) Diagnosis and treatment of an oral base-metal contact lesion following negative dermatologic patch test. Ann Allergy 73:161–165
208. Larese Filon F, Uderzo D, Bagnato E (2003). Sensitization to palladium chloride: a 10-year evaluation. Am J Contact Dermat 14:78–81
209. Kanerva L, Kerosuo H, Kullaa A, Kerosuo E (1996) Allergic patch test reactions to palladium chloride in schoolchildren. Contact Dermatitis 34:39–42
210. de Fine Olivarius F, Menné T (1992) Contact dermatitis from metallic palladium in patients reacting to palladium chloride. Contact Dermatitis 27:71–73
211. Vincenzi C, Tosti A, Guerra L, Kokelj F, Nobile C, Rivara G, Zangrando E (1995) Contact dermatitis to palladium: a study of 2,300 patients. Am J Contact Dermat 6:110–112
212. Koch P, Baum HP (1996) Contact stomatitis due to palladium and platinum in dental alloys. Contact Dermatitis 34:253–257
213. Camarasa JG, Serra-Baldrich E, Lluch M, Malet A, Garcia Calderon G (1989) Recent unexplained patch test reactions to palladium. Contact Dermatitis 20:388–389
214. Nakayama H, Nogi N, Kasahara N, Matsuo S (1990) Allergen control. An indispensable treatment for allergic contact dermatitis. Dermatol Clin 8:197–204
215. Glendenning WE (1971) Allergy to cobalt in metal denture as cause of hand dermatitis. Contact Dermatitis Newslett 10:255–226
216. Hubler WR Jr, Hubler WR Sr (1983) Dermatitis from chromium dental plate. Contact Dermatitis 9:377–398
217. Guimaraens D, Gonzalez MA, Conde-Salazar L (1994) Systemic contact dermatitis from dental crowns. Contact Dermatitis 30:124–125
218. Frykholm K, Frithiof L, Fernström B, Moberger G, Blohm G, Björn E (1969) Allergy to copper derived from dental alloys as a possible cause of oral lesions of lichen planus. Acta Derm Venereol (Stockh) 49:268–281
219. Santosh V, Ranjith K, Shenoi S, Sachin V, Balachandran C (1999) Results of patch testing with dental materials. Contact Dermatitis 40:50–51

220. Wöhrl S, Hemmer W, Focke M, Götz M, Jarish R (2001) Copper allergy revisited. J Am Acad Dermatol 45: 863–870
221. Gerhardsson L, Björkner B, Karlsteen M, Schutz A (2002) Copper allergy from dental copper amalgam? Sci Total Environ 290: 41–46
222. Vergara G, Silvestre JF, Botella R, Albares MP, Pascual JC (2004) Oral lichen planus and sensitization to copper sulfate. Contact Dermatitis 50: 374
223. Dunlap CL, Vincent SK, Barber BF (1989) Allergic reaction to orthodontic wire: report of case. J Am Dent Assoc 118: 449–450
224. Schweitzer A (1997) Erstfeststellung einer Titan-Allergie. Dermatosen 45: 190
225. Marcusson JA, Cederbrant K, Heilborn J (1998) Indium and iridium allergy in patients exposed to dental alloys. Contact Dermatitis 38: 297–298
226. Haberman AL, Pratt M, Storrs FJ (1993) Contact dermatitis from beryllium in dental alloys. Contact Dermatitis 28: 157–162
227. Klotzer WT (1991) Metalle und Legierungen – Korrosion, Toxikologie, sensibiliserande Wirkung (Teil) (Metals and alloys – corrosion, toxicology, sensitivity reactions 1, in German). ZWR 100: 300–304
228. Müller-Quernheim J, Zissel G, Schopf R, Vollmer E, Schlaak M (1996) Differential diagnosis of berylliosis/sarcoidosis in a dental technician (in German). Dtsch Med Wochenschr 121: 1462–1466
229. Vilaplana J, Romaguera C (1998) New developments in jewellery and dental materials. Contact Dermatitis 39: 55–57
230. Motolese A, Truzzi M, Giannini A, Seidenari S (1993) Contact dermatitis and contact sensitization among enamellers and decorators in the ceramic industry. Contact Dermatitis 28: 59–62
231. Pardo J, Rodriguez-Serna M, De La Cuadra J, Fortea JM (2004) Allergic contact stomatitis due to manganese in a dental prosthesis. Contact Dermatitis 50: 41
232. Beyer DJ, Belsito DV (1997) Delayed hypersensitivity to silicon causing gingival hyperplasia. Contact Dermatitis 27: 234
233. Magro-Filho O, de Carvalho AC (1994) Topical effect of propolis in the repair of sulcoplasties by the modified Kazanjian technique. Cytological and clinical evaluation. J Nihon Univ Sch Dent 36: 102–111
234. Garrido Fernández S, Lasa Luaces E, Echechipia Madoz S, Arroabarren Aléman E, Anda Apiñániz M, Tabar Purroy AI (2004) Allergic contact stomatitis due to therapeutic propolis. Contact Dermatitis 50: 321
235. Hensten-Pettersen A, Östavil D, Wennberg A (1985) Allergic potential of root canal sealers. Endod Dent Traumatol 1: 61–65
236. Bruze M (1994) Systemically induced contact dermatitis from dental rosin. Scand J Dent Res 101: 376–378
237. Sainio EL, Kanerva L (1995) Contact allergens in toothpastes and a review of their hypersensitivity. Contact Dermatitis 33: 100–105
238. Veien NK, Hattel T, Laurberg G (1993) Systemically aggravated contact dermatitis by aluminium in toothpaste. Contact Dermatitis 28: 199–200
239. Boxer MB, Grammer LC, Orfan N (1994) Gutta-percha allergy in a health care worker with latex allergy. J Allergy Clin Immunol 93: 943–944
240. Fisher AA (1994) The safety of gutta-percha from root canal use in rubber sensitive individuals. Am J Contact Dermat 5: 188
241. Ockenfels HM, Seemann U, Goos M (1995) Allergy to fibrin tissue in dental medicine. Contact Dermatitis 32: 363–364
242. Wüthrich B, Bianchi-Kusch E, Johansson SG (1996) Allergic urticaria and angioedema caused by a hemostatic sponge of bovine fibrin used in tooth extraction. Allergy 51: 49–51
243. Estlander T, Kanerva L, Tupasela O, Keskinen H, Jolanki R (1993) Immediate and delayed allergy to nickel with contact urticaria, rhinitis, asthma and contact dermatitis. Clin Exp Allergy 23: 306–310
244. Baker DB, Gann PH, Brooks SM, Gallagher J, Bernstein IL (1990) Cross-sectional study of platinum salts sensitization among precious metals refinery workers. Am J Ind Med 18: 653–664
245. Schena D, Barba A, Costa G (1996) Occupational contact urticaria to cisplatin. Contact Dermatitis 34: 220–221
246. Bergman A, Svedberg U, Nilsson E (1995) Contact urticaria and anaphylactic reactions caused by occupational exposure to iridium salt. Contact Dermatitis 32: 14–17
247. Murdoch RD, Pepys J, Hughes EG (1986) IgE antibody responses to platinum group metals: a large scale refinery survey. Br J Ind Med 43: 37–43
248. Murdoch RD, Pepys J (1987) Platinum group metal sensitivity: reactivity to platinum group metal salts in platinum halide salt-sensitive workers. Ann Allergy 59: 464–469
249. Torresani C, Caprari E, Manara GC (1993) Contact urticaria syndrome due to phenylmercuric acetate. Contact Dermatitis 29: 282–283
250. Camarasa JG, Serra-Baldrich E, Lluch M, Malet A (1993) Contact urticaria from sodium fluoride. Contact Dermatitis 28: 294
251. Dykewicz MS, Patterson RP, Cugell DW, Harris KE, Wu AF (1991) Serum IgE and IgG to formaldehyde-human serum albumin: lack of relation to gaseous formaldehyde exposure and symptoms. J Allergy Clin Immunol 87: 48–57
252. Wantke F, Hemmer W, Haglmuller T, Gotz M, Jarisch R (1995) Anaphylaxis after dental treatment with formaldehyde-containing tooth-filling material. Allergy 50: 274–276
253. Kunisada M, Adachi A, Asano H, Horikawa T (2002) Anaphylaxis due to formaldehyde released from root-canal disinfectant. Contact Dermatitis 47: 215–218
254. Nomura M, Okano M, Okada N et al (1983) Four cases with anaphylaxis induced by chlorhexidine. Skin Res 25: 306
255. Nishioka K, Doi T, Katayama I (1984) Histamin release in contact urticaria. Contact Dermatitis 11: 191
256. Ohtoshi T, Yamauchi N, Takodoro K, Miyachi S, Suzuki S, Miyamoto T (1986) IgE antibody-mediated shock reaction caused by topical application of chlorhexidine. Clin Allergy 16: 155–161
257. Petersen JK (1994) Et tilfaelde af akut anafylaktisk shock efter mundskylning med klorheksidinoplosning (in Danish). Tandlaegebladet 98: 335–338
258. Petersen JK, Heiden M (1995) Nyt tilfaelde af anafylaktisk shock over for klorheksidin (in Danish). Tandlaegebladet 99: 733