Neoprene Splinting: Dermatological Issues

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Occupational therapists are expanding their use of custom and commercial soft splints fabricated from neoprene (polychloroprene), but little has been written regarding dermatological issues associated with this material. Skin contact with neoprene poses two dermatological risks: allergic contact dermatitis (ACD) and miliaria rubra (i.e., prickly heat). Allergic reaction to neoprene is generally ascribed to the accelerants used to manufacture the man-made rubber, specifically thiourea compounds and mercaptobenzothiazole (MBT). Symptoms of neoprene-related ACD include itching, skin eruptions, swelling, and hemorrhages into the skin. Miliaria rubra creates small, red, elevated, inflammatory papules and a tingling, burning sensation.

Although neoprene hypersensitivity is rare, its incidence may grow as neoprene becomes a more commonly used material. It is recommended that therapists screen patients for a history of dermatological reactions to neoprene or other materials containing thiourea compounds or MBT and educate patients to discontinue splint use if dermatological symptoms develop. Therapists are also encouraged to notify splint manufacturers regarding all ACD reactions.

In addition to understanding hand anatomy, kinesiology, and function, successful splinting requires a thorough knowledge of the properties, benefits, and drawbacks of splinting materials (Breger-Lee & Buford, 1991). Rigid or semirigid thermoplastics remain the most commonly used splinting materials, but an increasing number of occupational therapists are also using soft custom and commercial splints (Collins, 1996). There are several soft splinting materials available for clinical use, including flexible thermoplastic materials, felt, Terry cloth, and polyurethane foam (Breger-Lee & Buford, 1991). Because of its soft texture, hook-and-loop-responsive finish, and excellent cosmesis, neoprene is one of the most popular soft materials, especially for soft splints used by pediatric (Casey & Kratz, 1988; Kennedy, 1996; Uditsky & Hogan, 1996) and geriatric patients (Berger, Cavanaugh, Coppard, & Lohman, 1996). Neoprene's controlled stretch makes it a logical material when slight tissue compression or dynamic force is desirable (Casey & Kratz, 1988; Clark, 1997). The 1997-1998 catalogs of three national manufacturers of orthoses offer more than 30 styles of commercial neoprene splints as well as neoprene sheet and strapping materials for custom splinting.

Developed in 1931, neoprene (also known as polychloroprene) is one of the earliest of the synthetic rubbers (Smith, 1985). In addition to its use in upper-extremity and lower-extremity splints, neoprene is used in a wide range of products, including wet suits, swimming goggles, shoe insoles and orthopedic inserts, “warm garments” (i.e., clothing or “warmers” used to retain joint or body heat),

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protective gloves used to handle toxic materials, automobile weather stripping, and as a replacement for latex. Typically, these products are used without adverse event, but occasionally the neoprene in these items produces allergic contact dermatitis (ACD), miliaria, or other dermatological problems (Adams, 1982; Alomar & Vilatella, 1985; Fowler & Callen, 1988; Fowler & Clark, 1991; Goette, 1984; Grimalt & Romaguera, 1975; Kanerva, Estlander, & Jolanki, 1994; Masmoudi & Lachapelle, 1987; Meding, Baum, Bruze, Roupe, & Trulsson, 1990; Reid, van Grutten, & Rycroft, 1993; Roberts & Hanifin, 1979, 1980; White & Vickers, 1970).

This article was initiated when a participant in a neoprene splint study developed ACD and the authors were unable to find discussion of ACD or other adverse dermatological reactions in the occupational therapy literature. The article describes two dermatological risks associated with neoprene splinting and offers precautions for the safe use of this splinting material.

**Patient Case**

A 41-year-old man participating in a splint research project did not wear the splint while sleeping. When the patient reported that he wore the splint for 2 hours after leaving the clinic (Day 1) and for 2 hours the next morning while at work mowing grass, operating a tractor, and driving a truck. The weather was hot, and he reported that he had perspired freely when working but showed no immediate adverse dermatological reaction to the splint. The patient wore the splint for an additional 4 hours the evening of Day 2. He did not wear the splint while sleeping. When the patient awoke the morning of Day 3 (Saturday), small isolated itching welts (i.e., hives, urticaria) covered the area that had been in contact with the splint. By late Day 3, the hand was swollen from fingertips to mid-forearm, including areas that had not had direct contact with the splint. By evening, bony landmarks of the finger joints were obscured and he was unable to form a fist. The patient self-treated his condition with continuous ice packs to reduce the itching. On Day 4 (Sunday), he contacted the authors to inform us of his condition. He reported that he had been unable to sleep due to the severity of pain and itching. The patient was self-employed and hesitant to take on the financial burden of an emergency room visit. We discussed options with the patient and, it was agreed that he would continue to use ice, would visit an emergency room if his condition worsened, and would come to the physician’s office on Monday (Day 5) if his symptoms did not resolve spontaneously. The patient was seen by his physician on Day 5, diagnosed with ACD, and placed on a course of oral corticosteroids and diphenhydramine. This treatment had reduced his symptoms when he was seen in the study clinic on Day 7, but the patient still presented with observable swelling of the right hand, causing end-range tightness of composite finger flexion. Small welts and erosions (where the patient had scratched open the skin) covered the area that had had direct contact with the splint. On exit interview, the patient reported no known allergy to neoprene before this episode but did indicate a history of allergy to self-adhesive bandages. The patient lost 1 week of work because of the ACD.

**Allergic Contact Dermatitis**

There are two types of allergic contact reactions. Type 1 hypersensitivity (traditionally referred to as immediate hypersensitivity) is mediated by an interaction between antigen and IgE antibodies (Benjamini & Leskowitz, 1991). Its signs and symptoms range from localized swelling to systemic anaphylaxis. It is rapidly occurring, developing within minutes to hours of contact. Type 4 hypersensitivity (traditionally known as delayed hypersensitivity) results when an antigen interacts with T-lymphocytes and the resulting cytokines attract inflammatory cells that accumulate at the site of contact. Type 4 reactions generally take 18 to 24 hours to fully manifest (Benjamini & Leskowitz, 1991) and produce various forms of localized dermatitis, including redness, scaling, blistering, and swelling (Marks & DeLeo, 1992). The typical allergic reactions that result from neoprene contact appear to be of the delayed hypersensitivity variety, though we cannot exclude the possibility that neoprene could produce immediate hypersensitivity reactions as well.

Regardless of type, allergic contact reactions occur in two phases: the induction phase and the elicitation phase (Dahl, 1996). In the induction phase, initial contact with an allergenic substance prepares the immune system to recognize the allergen. In this stage, no allergic reaction is apparent, but the system is primed to trigger a reaction when the allergen is encountered at a later date. In the elicitation phase, reexposure to the allergen triggers allergic symptoms. Subsequent reexposures tend to intensify the reaction.

Several of the chemical compounds used in neoprene manufacture have been associated with ACD and toxic reactions. Mercaptobenzothiazole (MTB) and its derivatives and formaldehyde resins are known allergens present in neoprene (Emmett et al., 1994; Foussereau, Cavelier, & Selig, 1976; Grimalt & Romaguera, 1975; Malten, 1967). However, it is thiourea compounds that are thought to be the allergens responsible for much of the neoprene hypersensitivity (Masmoudi & Lachapelle, 1987).

Thiourea compounds are used to accelerate the curing process that creates neoprene and to provide the water resistance prized in many neoprene products. Thiourea compounds may be found in several other products known to cause ACD, including polyvinylchloride (PVC) and PVC adhesive tape, fungicides and pesticides, paint and glue removers, silver polish dips, and diazo papers (used in photography and textile patterns) (Bruze & Fregert, 1983; Dooms-Goossens, Debusschère, Morren, Roelandts, &
Trulson, surfaces (Foussereau, Muslmani, Cavelier, Coopman, 1988; Dooms-Goossens et al., 1987; Fregert, Trulson, & Zimerson, 1982; Geier & Fuchs, 1993). Thiourea compounds and MBT are also used in the neoprene adhesives that bind neoprene to its nylon lining and other surfaces (Foussereau, Muslmani, Cavelier, & Herve-Bazin, 1986).

There are several thiourea compounds that may be used in neoprene (see Appendix). Individuals may react to none of the compounds, one compound, or several compounds. Because a patient may not cross-react to the different compounds, a person who is hypersensitive to one neoprene item may show no response to another neoprene item that uses a different thiourea compound as an accelerator. Most splint companies purchase neoprene from external suppliers and are unlikely to know the chemical composition of the material. Neoprene manufacturers generally do not divulge the composition of their neoprene unless there is an immediate medical need (R. Cash, Rubatex Corp., personal communication, September 5, 1997). This means that it is difficult to ascertain the chemicals that may be present in a specific neoprene material, splint, or adhesive.

A person may use a neoprene object for days, weeks, months, or even years without incident before developing allergic hypersensitivity to the material (Kanerva et al., 1994). However, once the induction phase is complete, subsequent exposure to neoprene generally produces symptoms in the allergic person within a few hours to 2 weeks of contact (Adams, 1982; Kanerva et al., 1994, Meding et al., 1990).

Symptoms and signs of neoprene-associated ACD are usually localized to the area of skin that had direct contact with the material. Severe itching is often the main complaint, though swelling both at the contact area and distally is common. Physical findings include eczematous skin eruptions, urticaria, and hemorrhages into the skin (i.e., purpura) that produce first red, then purple patches of discoloration. Less typically, neoprene-related ACD has produced hypopigmentation, where the skin becomes pale or white (Fowler & Callen, 1988; Goette, 1984). It has been suggested that this symptom may be due to a toxic reaction rather than a true allergic reaction (Goette, 1984).

Because purpura does not occur during patch testing, it has been suggested that the purple discolorations seen in neoprene-related ACD may be caused by tissue compression resulting from neoprene’s characteristic tight fit rather than as a true part of the allergic constellation (Meding et al., 1990). This suspicion is supported by the fact that purpura has been produced by tight-fitting jeans in the absence of ACD (Petrozzi & Lockshin, 1974).

Treatment for neoprene-related ACD relies on topical corticosteroids and oral antihistamines. Oral corticosteroids are used in cases of severe reaction. Oral or topical antibiotics may be used in cases where scratching has opened the skin and resulted in secondary infection. Postinflammatory hyperpigmentation of the affected limb may continue even 10 weeks to 15 weeks after the dermatitis has been resolved (Meding et al., 1990). The allergic reaction can require hospitalization (Adams, 1982), but, unlike allergic reactions to latex, the authors could find no published case of life-threatening anaphylaxis resulting from neoprene exposure. Indeed, neoprene is often suggested as a safe alternative when patients demonstrate latex hypersensitivity.

It is estimated that 3.4% of dermatology clinic patients are hypersensitive to MBT (Emmert et al., 1994) and that 0.4% to 1.1% are hypersensitive to at least one thiourea compound (Fowler & Clark, 1991; Fregert, Dahlquist, & Trulson, 1983; Fregert et al., 1982; Kanerva et al., 1994). Because of incomplete cross-reactivity, screening studies would have to include several thiourea compounds in order to establish an accurate estimate of the compounds’ allergic incidence in the general population. There are no such large-scale studies in the literature, although Swedish researchers estimated that .01% of that country’s general population could be allergic to the single thiourea compound, diphenylthiourea (Meding et al., 1990). To put these incidences into perspective, it is estimated that less than 1% of the general population (Sussman & Beezhold, 1995) and 2-60% to 67% of patients with spina bifida (Kelly, Kurup, Reijula, & Fink, 1994) are allergic to latex.

As more persons are exposed to neoprene through its use in common objects, it is likely that allergies to that material will be seen more regularly. Therefore, although the small numbers of persons currently at risk for ACD do not warrant rejection of this generally beneficial splinting material, therapists need to become more informed about the allergic risks associated with the material, to screen and monitor the use of neoprene splints, and to educate patients so that the effects of any ACD are limited.

Miliaria

Miliaria is a second dermatological problem encountered with neoprene splinting. The most common form of miliaria is miliaria rubra, also known as prickly heat. Prickly heat develops when sweat ducts are blocked by dirt, dust, creams, powders, or skin debris and sweat is retained within the lower layers of the epidermis (McKay, 1981). Prickly heat is characterized by small red, elevated, inflammatory papules that produce a tingling, burning, or pricking sensation (Feng & Janniger, 1995; Lobitz & Dobson, 1965). It is most commonly associated with the high temperatures that accompany summer but can be induced in any situation where excessive perspiration collects on the skin. Prickly heat can occur in even cool temperatures when occlusive materials, especially materials that are tight against the skin, create hot and humid microenvironments that trap perspiration and block sweat ducts. Neither the nylon lining that contacts the skin nor the neoprene that forms the body of a splint absorb or wick perspiration away from the skin. As a result, a snugly fitting neoprene splint provides an ideal environment for miliaria. This is especially so...
when splints are worn in hot weather or during tasks that produce excessive perspiration. Prickly heat may also occur when neoprene splints become wet either through immersion during functional tasks (e.g., washing dishes) or when “mouthed” by pediatric patients. The authors know of several cases of prickly heat resulting from neoprene splints, including 2 that occurred during the aforementioned neoprene splint study of 20 participants.

Prickly heat is treated by altering the microenvironment or macroenvironment that caused the localized reaction. This includes removing any item or material that contributed to the blockage and cooling and drying the skin. For symptomatic relief, patients may use wet-to-dry soaks (i.e., cool, saline-soaked towels permitted to air dry on the skin) and solutions that absorb sweat and cool the affected skin surface (Feng & Janniger, 1995). Typically, patients improve in 2 to 3 days, with symptoms resolving in 1 to 2 weeks. In situations where patients have open blisters, antibacterial medication may be needed to prevent or control infection.

Recommendations

The current incidence of dermatological reactions to neoprene is small. However, to ensure the safety of our clientele, therapists should follow some simple precautions when using neoprene splinting materials.

Screen Patients

Therapists should ask patients about their history of allergies and should consider alternative splinting materials when patients are at greater risk for allergic or other adverse reaction to neoprene splints. This includes patients who are prone to miliaria or who plan to use the splints in warm, wet environments or during tasks likely to increase sweating. In addition, neoprene alternatives should be considered if patients have a history of allergic reaction to neoprene or to thiourea compounds in other materials or have a strong history of dermatological allergies in general. Few people will know that they are allergic to neoprene. In most of the published case studies of neoprene-related ACD, patients had no prior indication of sensitivity to neoprene or other rubber products. Therefore, if a patient reports a history of hypersensitivity to any of the neoprene or thiourea items mentioned earlier in this article, it may be prudent to avoid neoprene in favor of other soft splinting materials.

Even multiple washings of neoprene are unlikely to reduce a splint’s levels of thiourea compounds or MBT allergens significantly enough to permit someone who is hypersensitive to continue use of the item (T. H. Risby, Johns Hopkins University, personal communication, September 5, 1997). Such washing might reduce the levels of allergens on the surface of a splint but is less able to affect allergen levels in deeper portions of the material (E. A. Emmett, Jefferson University, personal communication, September 8, 1997). Thus, allergens are likely to continue leaching from the splint, especially if the patient perspires during wear. After persons have demonstrated ACD to neoprene, they are likely to react to contact with even trace residues of the allergens (Roberts & Hanifin, 1979).

Educate Patients Regarding Risk:

Thiourea compounds and MBT leach from neoprene and neoprene adhesives when in the presence of human sweat, human plasma, or saltwater (Emmett et al., 1994). Hot and humid environments, typified by the work conditions of the case patient, increase the likelihood of both miliaria and allergic sensitization because they clog sweat ducts and encourage leaching of the allergens from the neoprene onto the skin. Plasma produces the highest levels of leaching of the thiourea compounds and is thought to actually bind with MBT (Emmett et al., 1994). As a result, patient information sheets should warn against wearing neoprene splints over open wounds or during excessively hot weather or tasks that are likely to induce sweating. Some commercial neoprene splints’ instruction sheets caution against splint wear by patients with existing contact dermatitis, open wounds, or known neoprene allergy and urge discontinuation if any dermatological symptoms develop associated with neoprene splint wear. Other instruction sheets do not mention dermatological risks or suggest any action and therefore need to be supplemented by the treating therapist.

Though stockinette helps absorb sweat, anecdotal accounts conflict regarding its efficacy in preventing or controlling miliaria when used with neoprene splints. Stockinette does not act as a barrier to leaching allergens and therefore will not prevent or control ACD in a situation where perspiration is present as a conducting medium. However, stockinette may act as a sufficient barrier in cases where the patient wears the splint in a cooler climate and reports only a mild reaction. The authors could find no study of the leaching action of human saliva or of mucosal reactions to neoprene. However, it seems prudent to warn patients or caregivers against extensive mouthing by children wearing neoprene splints.

Discontinue Splint Use at the First Sign of Allergy or Miliaria

As with many allergies, ACD is likely to become more severe with repeated exposure to the allergen. Therefore, all patients should be told to discontinue splint use at the first signs of any allergic reaction and to inform the therapist immediately.

Being unable to easily identify which thiourea compounds have been used in a specific neoprene material, patients who demonstrate neoprene-related ACD should be warned to avoid future exposure to neoprene in any form. If occupation or avocation requires continued exposure, the patient will need to undergo patch testing to iden-
tify the specific allergen or allergens responsible for the reaction. The therapist, physician, or patient may then contact the neoprene manufacturer to determine whether there are options that do not use the specific compound or compounds believed responsible for the ACD (Rietschel & Fowler, 1995). One patient, after being diagnosed with allergy to diethylthiourea in his neoprene wet suit, wore it without incident, only to require hospitalization after he used a friend’s wet suit containing that thiourea compound (Adams, 1982).

Limit Your Own Exposure to Neoprene Adhesives

Though relatively few occupational therapists fabricate custom neoprene splints, those who do should take care to limit their vocational exposure to neoprene and neoprene glue. To this end, therapists should wear protective gloves when fabricating neoprene splints and work in well-ventilated areas, especially when sealing seams with heat. Though direct contact is generally needed to produce ACD, Dooms-Goossens, Boyden, Ceuterick, and Degreef (1979) reported a dermatological reaction to airborne vapor released when paper containing dimethylthiourea was heated with an iron. At the earliest signs of ACD, a therapist should discontinue contact with the provocative material and seek medical care.

Inform Manufacturers and the Professional Community of Allergic Reactions

In addition to referring patients for proper medical care, therapists should inform the manufacturer and supplier of the splint or splinting material about the allergic reaction. In the absence of a central data bank for such information, manufacturers will wish to track the incidence of ACD and seek less-reactive alternatives if incidence increases. Therapists should also consider reporting adverse reactions in professional forums so that clinicians are kept informed of the risks that may be associated with the burgeoning numbers of splinting materials.

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Appendix

Common Thiourea Compounds Known To Cause Allergic Contact Dermatitis

- Dimethylthiourea (Note: This thiourea compound is less likely to be found in neoprene [R. Cash, Rubatex Corp., personal communication, September 5, 1997].)
- Ethylborylthiourea
- Ethylthiourea
- Dimethylborylthiourea
- Dimethylthiourea
- Diburylthiourea
- Ethylthiourea
- Diburylthiourea
- Dimethylborylthiourea

References


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