Skin Therapy Letter ©

Volume 11 • Number 7 • September 2006

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EDITOR: DR. STUART MADDIN

Practical Management Strategies for Diaper Dermatitis

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ABSTRACT

Common diaper dermatitis is an irritant contact diaper dermatitis (IDD) created by the combined influence of moisture, warmth, urine, feces, friction, and secondary infection. It is difficult to completely eradicate these predisposing factors in a diapered child. Thus, IDD presents an ongoing therapeutic challenge for parents, family physicians, pediatricians, and dermatologists. This article will focus on practical management strategies for IDD.

Key Words: *diaper dermatitis, IDD*

IDD is a common inflammatory eruption of the skin in the diaper area created by the presence of moisture, warmth, urine, feces, and friction, and is seen in 25% of children wearing diapers.[1]

Pathogenesis

Four key factors contribute to the development of IDD:[2]

Wetness

- Wet diapers result in excessive hydration and maceration of the stratum corneum[3] leading to impaired barrier function, enhanced epidermal penetration by irritants and microbes, and susceptibility to frictional trauma.[4]

Friction

- IDD is most commonly distributed in areas with the greatest skin-to-diaper contact.[5]
- Mechanical trauma disrupts the macerated stratum corneum, exacerbating barrier dysfunction.

Urine and feces

- The interaction of urine and feces is key to the pathogenesis of IDD. Bacterial ureases in the stool degrade the urea that is found in urine, releasing ammonia and increasing local pH.[6]
- Fecal lipases and proteases are activated by the increased pH. They cause skin irritation and disruption of the epidermal barrier.[7]
- Ammonia does not irritate intact skin; it is thought to mediate irritation by contributing to the high local pH.[6]

Microorganisms

- *Candida albicans (C. albicans)* and, less commonly, *Staphylococcus aureus (S. aureus)* infections are associated with IDD.[8]
- The warm, humid, and high pH environment in the diaper provides the ideal milieu for microbial proliferation.
- Innate antimicrobial microflora cannot survive in a high pH environment.[9]
- There is a positive correlation between the clinical severity of IDD and the presence and level of *C. albicans* in the diaper, mouth, and anus of infants.[8]

Clinical Features

IDD initially presents with localized asymptomatic erythema, and can progress to widespread painful, confluent erythema with maceration, erosions, and frank ulceration.[10] IDD commonly spares the skin folds, and affects the convex skin surfaces in close contact with the diaper including the buttocks, genitalia, lower abdomen, and upper thighs. IDD complicated by Candida presents with beefy red intertriginous plaques and satellite papules and pustules in the diaper area. IDD complicated by *S. aureus* appears impetiginized, with erosions, honey-colored crust, and lymphadenopathy.

Granuloma gluteale infantum and Jacquet erosive diaper dermatitis are distinctive, severe variants of IDD. Granuloma gluteale infantum presents in the setting of IDD with violaceous papules and nodules on the buttocks and in the groin. The pathogenesis of granuloma gluteale infantum is not clear. Potential risk factors include treatment with topical steroids,[11] candida infection, and occlusive plastic diaper covers.[12] Granuloma gluteale infantum follows a self-limited course, resolving in weeks to months, often with residual scarring.[5,11] The presence of punched-out erosions or ulcerations with heaped-up borders characterizes Jacquet erosive diaper dermatitis. This uncommon and severe presentation of IDD typically occurs in the context of frequent liquid stools, poor hygiene, infrequent diaper changes, or occlusive plastic diapers.[12]

Dermatosis	Clinical Features
IDD	• erythema, maceration, erosions, ulcerations • localized to convex skin surfaces in contact with the diaper, while sparing the folds
Candidiasis	• beefy, red plaques with satellite papules and pustules • can affect entire diapered skin and does not spare the folds
Bacterial infection	• impetigo: flaccid bullae, superficial erosions, and honey-colored crust • folliculitis: erythematous follicular papules and pustules
Granuloma gluteale infantum	• asymptomatic 0.5–3cm erythematous, violaceous papules and nodules
Jacquet erosive diaper dermatitis	• punched-out ulcers or erosions with elevated margins
Psoriasis	• well-circumscribed pink-red plaques in diaper area and inguinal folds • silvery scale usually absent
Allergic contact dermatitis	• acute, subacute, or chronic eczematous eruption localized to area of contact with allergen • pruritic
Langerhans cell histiocytosis	• erythematous infiltrated papules, pustules, and nonhealing erosions or ulcerations, with foci of hemorrhage, in diaper area • seborrheic dermatitis-like eruption on scalp and postauricular area • systemic involvement including anemia, diarrhea, organomegaly, lymphadenopathy, and bony involvement
Acrodermatitis enteropathica	• eczematous eruption may evolve into crusted and eroded vesiculobullous and pustular lesions • acral, periorificial, and anogenital distribution • triad of dermatitis, alopecia, and diarrhea presents upon weaning from breast milk

Table 1: Clinical features of diaper dermatoses

It is imperative to consider other conditions that may occur in the diaper area. Several excellent references are available that outline the differential diagnosis of IDD.[5,13] Please see Table 1 for a review of the clinical features of relevant diaper dermatoses.

Risk Factors

Fecal incontinence and diarrhea are risk factors for severe IDD because of prolonged and frequent skin contact with stool. Examples of at-risk children include those with Hirschsprung's disease, fecal impaction and overflow, and anogenital malformations.[14] Fecal proteases and lipases are also upregulated when gastrointestinal transit time is low.[9] Increased bile acids in stool, seen in short gut syndrome and conjugated hyperbilirubinemia, also increase protease activity.[12] Children with atopic dermatitis are prone to IDD because of their sensitivity to irritants and a greater susceptibility to secondary infection.

Treatment

Diapers

The continuous use of diapers is at the root of IDD. Maximizing "diaper-free" time is a widely recommended preventative strategy, but is not very practical. Frequent diaper changes are essential for maintaining dryness and keeping urine and feces separated. Diapers should be changed as soon as they are wet or soiled, at least every 3–4 hours and more frequently in the neonate due to increased skin fragility.[15] Parents should forego tight-fitting diapers and consider a diaper slightly larger than the infant to minimize the contact between skin and urine or feces.[5] Common IDD should resolve when children become toilet trained.

The advent of disposable diapers and the ongoing development of new diaper technology has radically changed the face of IDD. Early cellulose-core containing disposable diapers were dramatically improved by the addition of cross-linked sodium polyacrylate polymers to the diaper core.[10,16,17] These polymers, also called absorbent gelling materials, bind water in a gel matrix when hydrated.[16,17] This gel effectively traps moisture away from the skin surface. It controls pH through its buffering capacity, and by separating urine from feces.[17] These diapers are referred to as superabsorbent diapers.[16] In a study of 1,614 infants, superabsorbent diapers were associated with reduced skin wetness, superior pH control, and less diaper dermatitis compared with cellulose-core disposable and cloth diapers.[17] Originally, these diapers were developed with an impenetrable backsheet (outer cover) to prevent leaks, but this led to increased humidity and skin maceration. A "breathable" diaper was subsequently developed with a backsheet that is permeable to air and vapor but still impenetrable to leaks.[16] This backsheet is readily identified by its cloth-like, rather than plastic, texture. The "breathable" superabsorbent diaper has been shown to reduce the prevalence of severe IDD by up to 50%.[10] Nearly all commercially available disposable diapers in North America now use polyacrylate gel-core technology, and many use the breathable backsheet (e.g., Pampers®, Procter & Gamble; Huggies®, Kimberly-Clark). A novel diaper has recently been developed that transfers a petrolatum and zinc oxide-based formula to the child's skin.[18] In a double-blinded, randomized trial, infants using this diaper had consistently less skin erythema and diaper rash compared with those using a superabsorbent diaper alone over a 4-week period of use.[18]

Cloth diapers are not recommended for patients with IDD. They increase skin wetness, promote mixing of urine and feces, and are associated with Jacquet erosive diaper dermatitis.[12]

Barrier

Application of a suitable barrier preparation is the cornerstone of prevention and treatment of IDD. There is a notable absence of controlled trials to support and guide the use of barrier preparations for IDD. Anecdotal evidence is abundant and suggests a barrier preparation should be applied to the diaper area after every diaper change and bath. A suitable barrier preparation should minimize transepidermal water loss (TEWL) and decrease permeability to irritants.[9] The barrier corrects these deficits by forming a lipid barrier over the skin surface, or by penetrating the stratum corneum and assuming the role of endogenous intercellular lipids.[5,19] The barrier also minimizes cutaneous friction. The barrier must be lipid-rich, long-lasting and adherent to the macerated and eroded diapered skin.

Pastes are the most hardy and desirable barriers, followed by ointments. Ointments are superior to creams and lotions, which are poorly adherent, minimally occlusive, and contain preservatives. Diaper pastes are tenacious semisolid compounds containing a high proportion (usually >10%)[9] of a fine powder such as zinc oxide, titanium dioxide, and starch or talc.[20] Pastes should be applied thickly, like "icing on a cake", and can be covered by petroleum jelly to avoid sticking to the diaper.[14] Products containing fragrance, preservatives, and other ingredients with irritant or allergic potential

should be avoided. Products containing boric acid, camphor, phenol, and salicylates should be avoided due to potential systemic toxicity.[21] The local ostomy nurse may also be a valuable resource in identifying suitable barrier preparations in severe IDD.

Cleansing

Children predisposed to IDD should be bathed daily in a lukewarm bath using an irritant-free and fragrance-free soap or cleanser followed by liberal application of a barrier preparation to the diaper area. The diaper area should be cleaned gently and dried by patting with a towel to avoid any undue friction. Aggressive wiping at diaper changes should be avoided. Residual adherent barrier paste does not need to be wiped off along with the urine and stool at each diaper change. Mineral oil can help facilitate paste removal, if required.[5,14]

It is a commonly held belief that baby wipes contribute to IDD; however an investigator-blinded, parallel-comparison study of 102 infants found no difference between skin cleaned with an alcohol-free, nonwoven disposable wipe, and skin cleaned with water and a cleanser.[22] Moreover, skin cleaned with wipes had statistically better rash scores in the intertriginous areas, suggesting that wipes may help parents access hard-to-reach areas. These wipes were found to be safe and well tolerated in infants with atopic dermatitis. Baby wipes can cause an allergic contact hand dermatitis in caregivers, in a "grip-like" distribution.[23] It is prudent to choose wipes without fragrance and preservatives to avoid allergic sensitization.

Infection

Candida infection is often associated with moderate-severe cases of IDD. *C. albicans* is present in the mouth, inguinal and perianal skin more frequently in patients with IDD.[8] The azoles, nystatin, and ciclopirox are all appropriate topical anticandidal agents,[5,24] but few well-designed comparative trials are available to guide clinical practice. Twice-daily application is recommended until resolution. In a National Ambulatory Medical Care Survey (NAMCS), more than 200,000 visits for diaper dermatitis in the US were reviewed; nystatin and clotrimazole were the most commonly prescribed topical antifungals (27% and 16% respectively).[1] A prospective, randomized study compared topical nystatin with mupirocin in the treatment of IDD complicated by *C. albicans* infection. Treatment with both agents resulted in mycological cure; however, resolution of IDD was observed in all patients treated with mupirocin compared with only 30% treated with nystatin.[25] Application of miconazole nitrate 0.25% in a zinc oxide and petrolatum base has demonstrated efficacy and safety in vehicle-controlled, randomized, double-blinded trials.[26,27] In an open trial,

Dermatosis	Clinical Features
Antimicrobial/ anti-inflammatory	• A topical antifungal may be required for cases exhibiting features of candidiasis (beefy, red erythema, satellite papules, and pustules). • Topical or systemic antibiotics can be considered for cases with secondary bacterial infection (erosions, honey-coloured crust, lymphadenopathy, fever). • For marked inflammation, use a mild topical corticosteroid. Mid-to-high potency corticosteroids should be avoided in the diaper area of infants.
Barrier preparation	• Select a thick, irritant and fragrance-free barrier paste or ointment to be applied liberally after each diaper change and bath.
Cleansing	• Daily bathing using a fragrance-free soap is recommended. • Diaper area should be cleaned gently to minimize additional friction. • Unscented and alcohol-free baby wipes are suitable for cleaning the diaper area.
Diaper	• Maximize "diaper-free" time. • Select breathable, superabsorbent, polyacrylate gel-core disposable diapers. • Increase frequency of diaper changes to every 3–4 hours or immediately when soiled/ wet (more frequently in neonates).
Education	• The best strategy for treatment of IDD is PREVENTION. • Review the aggravating factors for IDD with parents: wetness, friction, urine/feces, microorganisms. • Emphasize preventative modifications to current diaper regimen.

Table 2: The **ABCDE**s of diaper dermatitis: a practical approach

ciclopirox 0.77% topical suspension demonstrated significant improvement in rash severity and superior mycological cure by 7 days in patients with IDD and *C. albicans* infection.[28] There is little evidence to support the addition of an oral antifungal to topical therapy in IDD.[29] Patients with concomitant oral thrush, however, may benefit from a course of systemic antifungal therapy.[5]

Corticosteroids

A short course of a mild topical corticosteroid is frequently necessary in moderate-to-severe IDD. Hydrocortisone 1% ointment can be applied to affected areas twice daily for a limited duration. Mid-to-high potency corticosteroids should never be used in the diaper area. The NAMCS documented a surprisingly high rate of moderate-to-high potency halogenated topical corticosteroid use in IDD. Triamcinolone acetonide or betamethasone dipropionate use, either alone or in combination with antifungals, was documented in a staggering 24.3% of visits for diaper dermatitis.[1] Atrophy, systemic absorption, candidiasis, and granuloma gluteale infantum are all associated with mid-to-high potency corticosteroid use in the diaper area. The topical calcineurin inhibitors, tacrolimus and pimecrolimus, have not been studied for the treatment of IDD. These agents have been studied for efficacy and safety as a steroid-sparing treatment for atopic dermatitis in infants <2 years old.[30] Although they are not approved for use in this age group, they may be a useful off-label alternative for IDD in the appropriate clinical setting.

Other Agents

A number of other agents have been reported to be efficacious in the treatment of IDD. A recent pilot study found clinical and mycological benefits using a 1:1:1 mixture of honey: olive oil: beeswax to treat IDD.[31] Eosin, an orange-red dye derived from coal tar, is a common agent used for IDD in Europe. It was found to have a greater rate of clearance of IDD within 5 days compared with zinc oxide and a moderate-potency topical corticosteroid ointment.[32] In a randomized, vehicle-controlled study, topical vitamin A cream did not improve the outcome of IDD.[33]

Conclusion

IDD is a common dermatosis afflicting diapered children. It is caused by wetness, friction, urine, stool, and microorganisms. A proactive approach targeting predisposing factors is the best defence against IDD.

References

1. Ward DB, Fleischer AB Jr, Feldman SR, Krowchuk DP. Characterization of diaper dermatitis in the United States. *Arch Pediatr Adolesc Med.* 154(9):943-6 (2000 Sep).
2. Atherton DJ. The aetiology and management of irritant diaper dermatitis. *J Eur Acad Dermatol Venereol* 15 Suppl 1:1-4 (2001).
3. Willis I. The effects of prolonged water exposure on human skin. *J Invest Dermatol* 60(3):166-71 (1973 Mar).
4. Zimmerer RE, Lawson KD, Calvert CJ. The effects of wearing diapers on skin. *Pediatr Dermatol* 3(2):95-101 (1986 Feb).
5. Shin HT. Diaper dermatitis that does not quit. *Dermatol Ther* 18(2):124-35 (2005 Mar-Apr).
6. Berg RW, Buckingham KW, Stewart RL. Etiologic factors in diaper dermatitis: the role of urine. *Pediatr Dermatol* 3(2):102-6 (1986 Feb).
7. Andersen PH, Bucher AP, Saeed I, Lee PC, Davis JA, Maibach HI. Faecal enzymes: *in vivo* human skin irritation. *Contact Dermatitis* 30(3):152-8 (1994 Mar).
8. Ferrazzini G, Kaiser RR, Hirsig Cheng SK, et al. Microbiological aspects of diaper dermatitis. *Dermatology* 206(2):136-41 (2003).
9. Atherton DJ. A review of the pathophysiology, prevention and treatment of irritant diaper dermatitis. *Curr Med Res Opin* 20(5):645-9 (2004 May).
10. Akin F, Spraker M, Aly R, Leyden J, Raynor W, Landin W. Effects of breathable disposable diapers: reduced prevalence of Candida and common diaper dermatitis. *Pediatr Dermatol* 18(4):282-90 (2001 Jul-Aug).
11. Bluestein J, Furner BB, Phillips D. Granuloma gluteale infantum: case report and review of the literature. *Pediatr Dermatol* 7(3):196-8 (1990 Sep).
12. Fiorillo L. Therapy of pediatric genital diseases. *Dermatol Ther* 17(1):117-28 (2004).
13. Kazaks EL, Lane AT. Diaper dermatitis. *Pediatr Clin North Am* 47(4):909-19 (2000 Aug).
14. Borkowski S. Diaper rash care and management. *Pediatr Nurs* 30(6):467-70 (2004 Nov-Dec).

15. Lane AT, Rehder PA, Helm K. Evaluations of diapers containing absorbent gelling material with conventional disposable diapers in newborn infants. *Am J Dis Child* 144(3):315-8 (1990 Mar).

16. Odio M, Friedlander SF. Diaper dermatitis and advances in diaper technology. *Curr Opin Pediatr* 12(4):342-6 (2000 Aug).

17. Campbell RL, Seymour JL, Stone LC, Milligan MC. Clinical studies with disposable diapers containing absorbent gelling materials: evaluation of effects on infant skin condition. *J Am Acad Dermatol* 17(6):978-87 (1987 Dec).

18. Baldwin S, Odio MR, Haines SL, O'Connor RJ, Englehart JS, Lane AT. Skin benefits from continuous topical administration of a zinc oxide/petrolatum formulation by a novel disposable diaper. *J Eur Acad Dermatol Venereol* 15 Suppl 1:5-11 (2001 Sep).

19. Clark C, Hoare C. Making the most of emollients. *Pharm J* 266:227-229 (2001).

20. Juch RD, Rufli T, Surber C. Pastes: what do they contain? How do they work? *Dermatology* 189(4):373-7 (1994).

21. Farrington E. Diaper dermatitis. *Pediatr Nurs* 18(1):81-2 (1992 Jan-Feb).

22. Ehretsmann C, Schaefer P, Adam R. Cutaneous tolerance of baby wipes by infants with atopic dermatitis, and comparison of the mildness of baby wipe and water in infant skin. *J Eur Acad Dermatol Venereol* 15 Suppl 1:16-21 (2001 Sep).

23. Guin JD, Kincannon J, Church FL. Baby-wipe dermatitis: preservative-induced hand eczema in parents and persons using moist towelettes. *Am J Contact Dermat* 12(4):189-92 (2001 Dec).

24. Gupta AK, Skinner AR. Management of diaper dermatitis. *Int J Dermatol* 43(11):830-4 (2004 Nov).

25. de Wet PM, Rode H, van Dyk A, Millar AJ. Perianal candidosis—a comparative study with mupirocin and nystatin. *Int J Dermatol* 38(8):618-22 (1999 Aug).

26. Concannon P, Gisoldi E, Phillips S, Grossman R. Diaper dermatitis: a therapeutic dilemma. Results of a double-blind placebo controlled trial of miconazole nitrate 0.25%. *Pediatr Dermatol* 18(2):149-55 (2001 Mar-Apr).

27. Spraker MK, Gisoldi EM, Siegfried EC, et al. Topical miconazole nitrate ointment in the treatment of diaper dermatitis complicated by candidiasis. *Cutis* 77(2):113-20 (2006 Feb).

28. Gallup E, Plott T, Ciclopirox TS Investigators. A multicenter, open-label study to assess the safety and efficacy of ciclopirox topical suspension 0.77% in the treatment of diaper dermatitis due to *Candida albicans*. *J Drugs Dermatol* 4(1):29-34 (2005 Jan-Feb).

29. Hoppe JE. Treatment of oropharyngeal candidiasis and candidal diaper dermatitis in neonates and infants: review and reappraisal. *Pediatr Infect Dis J* 16(9):885-94 (1997 Sep).

30. Patel RR, Vander Straten MR, Korman NJ. The safety and efficacy of tacrolimus therapy in patients younger than 2 years with atopic dermatitis. *Arch Dermatol* 139(9):1184-6 (2003 Sep).

31. Al-Waili NS. Clinical and mycological benefits of topical application of honey, olive oil and beeswax in diaper dermatitis. *Clin Microbiol Infect* 11(2):160-3 (2005 Feb).

32. Arad A, Mimouni D, Ben-Amitai D, Zeharia A, Mimouni M. Efficacy of topical application of eosin compared with zinc oxide paste and corticosteroid cream for diaper dermatitis. *Dermatology* 199(4):319-22 (1999).

33. Bosch-Banyeras JM, Catala M, Mas P, Simon JL, Puig A. Diaper dermatitis. Value of vitamin A topically applied. *Clin Pediatr (Phila)* 27(9):448-50 (1988 Sep).

Plasma Skin Regeneration Technology

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ABSTRACT

Plasma skin regeneration (PSR) technology uses energy delivered from plasma rather than light or radiofrequency. Plasma is the fourth state of matter in which electrons are stripped from atoms to form an ionized gas. The plasma is emitted in a millisecond pulse to deliver energy to target tissue upon contact without reliance on skin chromophores. The technology can be used at varying energies for different depths of effect, from superficial epidermal sloughing to deeper dermal heating. With the Portrait® PSR device (Rhytec, Inc.) there are three treatment guidelines termed PSR1, PSR2, and PSR3. The PSR1 protocol uses a series of low-energy treatments (1.0–1.2 Joules) spaced 3 weeks apart. The PSR2 protocol uses one high-energy pass (3.0–4.0 Joules) performed in a single treatment, and the PSR3 protocol uses two high-energy passes (3.0–4.0 Joules) performed in a single treatment. All protocols improve fine lines, textural irregularities, and dyspigmentation; however, skin tightening is probably more pronounced with the high-energy treatments.

Key Words: *plasma skin regeneration, PSR*

Plasma skin regeneration (PSR) technology can be used at varying energies for different depths of effect, from superficial epidermal sloughing to deeper dermal heating. In a pilot study evaluating the use of a single full-facial treatment at high energy (3–4 Joules), Kilmer, et al. demonstrated a mean improvement in overall facial rejuvenation of 50% by 1 month.[1] Potter, et al. used silicone molding to demonstrate a 39% reduction in fine-line depth 6 months after one high-energy, full-face treatment.[2] Bogle, et al. evaluated a series of three low-energy (1.2–1.8 Joules), full-face treatments for facial rejuvenation and found a 37% improvement in facial rhytids at 3-month follow-up.[3] In the same study, participants rated themselves as having a 68% improvement in overall facial rejuvenation at 3-month follow-up.[3] Histologic analysis of post-treatment samples revealed a decrease in solar elastosis with significant new interdigitating collagen and thickening of the collagen band at the dermal-epidermal junction.[3] The mean depth of new collagen formation was 72.3µm.[3] Epidermal thickness was not changed by the treatment.[3]

The Plasma Skin Regeneration Device

The PSR device consists of an ultra-high-frequency (UHF) radiofrequency generator that excites a tuned resonator and imparts energy to a flow of inert nitrogen gas within the handpiece. The activated, ionized gas is termed plasma. Nitrogen is used for the gaseous source because it is able to purge oxygen from the surface of the skin, minimizing the risk of unpredictable hot spots, charring, and scar formation. Upon formation, the plasma is directed through a quartz nozzle out of the tip of the handpiece and onto the skin. The plasma appears as a characteristic lilac glow that transitions to a yellowish light called a Lewis-Raleigh afterglow.

As the plasma hits the skin, energy is rapidly transferred to the skin surface, causing instantaneous heating in a controlled, uniform manner, without an explosive effect on tissue or epidermal removal. The depth and area of thermal effect are determined by the energy setting and spot size of the handpiece. The energy can be adjusted from 1–4 Joules per pulse. The intended spot size of 6mm is reached when the device is held approximately 5mm from the surface of the skin; however, the thermal effect can be increased or decreased by defocusing the handpiece either closer or farther away from the skin surface. High temperatures during each pulse erode the tungsten resonator in the handpiece, so the handpiece must be replaced after each use.

Treatment Guidelines

There are three recommended treatment guidelines, PSR1, PSR2, and PSR3. The PSR1 protocol uses a series of low-energy treatments spaced 3 weeks apart. The first treatment is performed at 1.0–1.2 Joules, and

fluences are increased as tolerated at subsequent visits. Recovery time is 3–4 days. The PSR2 protocol uses one high-energy pass (3.0–4.0 Joules) with a recovery time of 5–7 days, and the PSR3 protocol uses two high-energy passes (3.0–4.0 Joules) with a recovery time of 6–10 days. A series of treatments in the mid-energy group (1.5–3.0 Joules) have produced good results in improving skin texture and discoloration, but they have only slightly less recovery time than a single high-energy treatment, and less skin tightening. Thus, most practitioners prefer to use the suggested PSR1, 2, or 3 protocols.

Clinical Protocol

The first step in treatment is to assess the patient and determine the goals of treatment. Low-energy PSR1 treatments can normally be performed under local anesthesia with a topical agent. For mid-to-high energies, patients will require adjunctive oral anesthesia such as meperidine or a codeine derivative in addition to a topical agent. Patients should arrive at least 1 hour beforehand so that the topical anesthetic cream can be applied and left on for approximately 1 hour. Oral anesthesia should be administered 30–45 minutes before the procedure begins. To avoid unexpected downtime, it is important for the physician to develop a standard protocol for removal of topical anesthesia and delay time before starting the procedure. Hydration of the epidermis influences the amount of energy that is absorbed and the depth of thermal insult achieved, with drier tissue absorbing more energy.[4]

Generally, it is a good idea to work in aesthetic segments of the face (i.e., forehead, nose, cheek, chin, etc.), removing the anesthetic cream for each area immediately before treating that area rather than removing the cream for the entire face all at once. This helps to standardize the delay time between anesthetic removal and treatment. Anesthetic should be gently wiped off with dry gauze. Again, it is not necessary to use water or alcohol-soaked gauze as this will change the hydration properties of the skin.

Once a facial segment is ready for treatment, the tip of the handpiece should be held approximately 5mm from the skin's surface. (Figure 1) The pulses are delivered in a paintbrush fashion in one direction across the treatment area. The pulses should be delivered in rows of one direction (either all right to left, or all left to right) because a zig-zag pattern has been found to cause heat build-up at the corners where one changes direction to start the subsequent row. Pulses should not be overlapped more than about 10%. To avoid lines of demarcation in the high-energy protocol, the borders of the treatment area should be feathered by increasing the distance of the nozzle from the surface of the skin to about 1cm. Feathering can also be achieved by holding the handpiece nozzle at an angle with respect to the skin surface or reducing the power setting. There is no need for feathering in the low-energy PSR1 protocol.

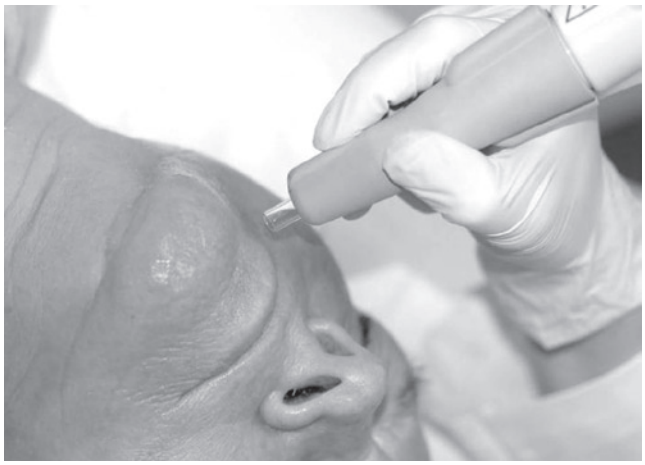

Figure 1: Plasma treatment at low energy, holding the handpiece approximately 5mm from the surface of the skin. Note there is no denudation from the energy application to the chin.

Postprocedure Care

Patients should be instructed to avoid sun exposure and apply a bland ointment to the face at frequent intervals after the procedure while the skin is healing. Low-energy PSR1 treatments may cause only erythema for 2–3 days. High-energy treatments will cause erythema and a "dirty" look to the skin, which will resolve in 5–10 days as re-epithelialization occurs and the photodamaged epidermis sloughs off. It is important for patients not to manually pick at the peeling skin to avoid prolonged erythema or scarring.

There have been no major side-effects reported in studies to date. As in all procedures utilizing heat energy, side-effects that could occur include erythema, edema, epidermal de-epithelialization, scarring, and hyperpigmentation. There have been no reported instances of hypopigmentation. Erythema and edema are common postprocedure, usually resolving in several days. Edema can be decreased by the application of ice following the procedure. Epidermal de-epithelialization is a risk at higher energies and should be treated with appropriate wound care and liberal application of a bland ointment. Temporary hyperpigmentation has been reported at mid-to-high energy treatments. Scarring is rare.

Conclusion

Plasma skin regeneration technology is a novel method to rejuvenate the skin and has shown good results in the improvement of fine lines, dyspigmentation, and textural irregularities. High-energy protocols can offer the added benefit of increased tissue tightening. The treatments are safe and no major side-effects have yet been reported.

References

1. Kilmer S, Fitzpatrick R, Bernstein E, Brown D. Long term follow-up on the use of plasma skin regeneration (PSR) in full facial rejuvenation procedures. *Lasers Surg Med* 36(Suppl 17):22 (2005).
2. Potter M, Harrison R, Ramsden A, Andrews P, Gault D. Facial acne and fine lines: transforming patient outcomes with plasma skin resurfacing. *Lasers Surg Med* 36(Suppl 17):23 (2005).
3. Bogle MA, Arndt KA, Dover JS. Evaluation of plasma skin regeneration technology in low fluence full-facial rejuvenation. *Arch Dermatol* (In Press, 2006).
4. Penny K, Sibbons P, Andrews P, Southgate A. A histopathologic assessment of the effects of hydration on the absorption of plasma skin regeneration energy (PSR) in an animal model. *Lasers Surg Med* (In Press, 2006).

Class	Name/Company	Approval Dates and Comments
Vaccine	**Quadrivalent Human Papillomavirus Recombinant Vaccine** *GARDASIL®* Merck	The Committee for Medicinal Products for Human Use in Europe recommended approval of this vaccine in July 2006 for the immunization of children and adolescents aged 9–15 years and of adult females aged 16–26 years for the prevention of cervical cancer, high-grade cervical dysplasia (CIN 2/3), high-grade vulvar dysplastic lesions (VIN 2/3) and external genital warts caused by human papillomavirus types 6, 11, 16, and 18. In June 2006 the US Centers for Disease Control and Prevention's (CDC's) Advisory Committee on Immunization Practices voted unanimously to recommend that girls and women 11–26 years of age be vaccinated with this product to prevent cervical cancer, precancerous and low-grade lesions, and genital warts caused by the human papillomavirus types listed above. GARDASIL® is a ready-to-use, three dose, intramuscular vaccine.
Antipsoriatic Agent	**Infliximab** *Remicade®* Centocor	The US FDA accepted a supplemental Biologics License Application in June 2006 for this product for inhibiting the progression of structural damage and improving physical function in patients with active psoriatic arthritis.
Medical Device	*Humira® Pen* Abbott Pharmaceuticals	The US FDA approved a new device for administering Humira® in June 2006. Humira® is approved for the treatment of moderate-to-severe rheumatoid arthritis and psoriatic arthritis. This new device offers improved ease of use with its one-touch activation and easy-to-grasp size and shape.

Drug News

Malignant Melanoma Research	Researchers from the Dana-Farber Cancer Institute have identified a novel gene that facilitates the spread of malignant melanoma using a technique that they believe can speed the discovery of hard-to-find cancer genes. Recently reported in *Cell**, the gene, NEDD9, is abnormally abundant in more than a third of melanomas that have metastasized, but not in primary melanomas that have not spread. The investigators used genome-scanning methods, such as array-CGH (comparative genomic hybridization), to uncover structural abnormalities of the chromosomes of cancer cells. *Kim M, Gans JD, Nogueira C, et al. *Cell* 125(7):1269-81 (2006 Jun).
Scleroderma Research	In a bold attempt to control scleroderma, physicians at Duke University Medical Center are leading a national study to test whether stem cell transplants can reconstruct defective immune systems. At this time, the predominant therapy for this disease is cyclophosphamide; however, about 50% of patients with severe organ involvement die within 5 years of diagnosis. This study (Scleroderma Cyclophosphamide or Transplantation – SCOT) will increase the dose and duration of cyclophosphamide and compare this regimen against stem cell transplants, using purified cells derived from a patient's own blood. This 7-year randomized clinical trial will enroll 226 patients at 36 institutions throughout the US. If successful, the therapy would represent the first therapy ever to treat and potentially reverse the disease itself, not just alleviate its symptoms.

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Subscription Information. Annual subscription: Canadian $94 individual; $171 institutional (plus GST); US $66 individual; $121 institutional. Outside North America: US$88 individual; $143 institutional. We sell reprints in bulk (100 copies or more of the same article). For individual reprints, we sell photocopies of the articles. The cost is $20 to fax and $15 to mail. Prepayment is required. Student rates available upon request. Sales inquiries: business@skincareguide.com

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