CLINICAL MANAGEMENT extra

Incontinence and Incontinence-Associated Dermatitis

CME
CATEGORY 1
1 Credit

CE
ANCC
3.5 Contact Hours

Diane Langemo, PhD, RN, FAAN • Distinguished Professor Emeritus & Adjunct Professor • University of North Dakota College of Nursing • Grand Forks, North Dakota
Darlene Hanson, MS, BSN, PhD(c) • Clinical Associate Professor • University of North Dakota College of Nursing • Grand Forks, North Dakota
Susan Hunter, MSN, RN • Associate Professor • University of North Dakota College of Nursing • Grand Forks, North Dakota
Patricia Thompson, MS, RN • Clinical Assistant Professor • University of North Dakota College of Nursing • Grand Forks, North Dakota
In Eui Oh, BSN, MS(c) • University of North Dakota College of Nursing • Grand Forks, North Dakota

Dr Langemo has disclosed that she is/was a recipient of grant/research funding from Medline Industries and DM Systems and is/was a member of the speakers' bureau for Posey and DM Systems. Ms Hanson has disclosed that she is a recipient of grant/research funding from Medline Industries. Ms Hunter and Ms Thompson have disclosed that they have no significant relationship with or financial interest in any commercial companies that pertain to this educational activity. Ms Oh has disclosed that she is/was a recipient of grant/research funding from Medline Industries and DM Systems.

All staff, including spouses/partners (if any), in a position to control the content of this CME activity have disclosed that they have no financial relationships with, or financial interests in, any commercial companies pertaining to this educational activity.

To earn CME credit, you must read the CME article and complete the quiz and evaluation on the enclosed answer form, answering at least 13 of the 18 questions correctly.

This continuing educational activity will expire for physicians on March 31, 2012.

PURPOSE:

To enhance the learner's competence in prevention and treatment of incontinence-associated dermatitis (IAD).

TARGET AUDIENCE:

This continuing education activity is intended for physicians and nurses with an interest in skin and wound care.

OBJECTIVES:

After participating in this educational activity, the participant should be better able to:

1. Relate the pathophysiology of IAD to the development of signs and symptoms.
2. Use assessment and intervention tools in the care of patients with IAD.
3. Construct an effective plan of care for patients with IAD.

ABSTRACT

Incontinence is a prevalent problem and can lead to many complications. Both urinary and fecal incontinence can result in tissue breakdown, now commonly referred to as incontinence-associated dermatitis. This article addresses the types of incontinence, its etiology and pathophysiology, assessment, prevention and treatment, and the latest research.

KEYWORDS: urinary incontinence, incontinence-associated dermatitis, bowel incontinence, fecal incontinence

ADV SKIN WOUND CARE 2011;24:126-40; quiz 141-2.

Incontinence is a prevalent problem, particularly among individuals in acute and long-term care, often predisposing them to complications that result in increased morbidity and, for acute-care individuals, longer length of stay.[1,2] Incontinence can lead to tissue irritation and breakdown, commonly referred to as incontinence-associated dermatitis (IAD). In the United States, the Centers for Medicare & Medicaid Services addressed the problem of urinary incontinence in long-term care in Tag F315.[3] After reading this article, clinicians will be better able to evaluate the types of incontinence; to define IAD and assess its prevalence, etiology, and pathophysiology; to apply the appropriate assessment, prevention, and treatment strategies for IAD; and interpret the latest research on IAD.

URINARY INCONTINENCE

Urinary incontinence is an involuntary loss of urine. In some instances of urinary incontinence, there is a single causative factor; however, in the majority of cases, there are multiple complex factors at play, yet not well understood. Although some risk factors for urinary incontinence are modifiable, there is some evidence that preventive measures can help reduce the likelihood of developing urinary incontinence.[4] Studies have noted an association between urinary incontinence and type 2 diabetes mellitus related to neuropathic and microvascular changes within the urinary system.[5–7]

BOWEL INCONTINENCE

Bowel incontinence is a condition of unintentional loss of liquid or solid stool.[4] Continence of stool is dependent on competent muscles and nerves in and around the rectum and anus; thus, stool incontinence occurs with functional or structural impairment to these muscles and nerves.[8] Although bowel incontinence can affect individuals at any age, it more commonly is seen in women and older individuals. As feces contain more than 500 different microorganisms, the potential for infection in the presence of fecal incontinence and IAD is significant.[9]

There are 4 factors that maintain control over bowel function, and impairment in any one of them can result in bowel incontinence. The 4 factors are rectal sensation, rectal storage capacity, sphincter pressure, and the established bowel habits of the individual.[4] Impairment of the anal sphincter is the most common cause of fecal incontinence.[10] Fecal incontinence is actually a sign or symptom of another disease or condition, rather than a distinct diagnosis in and of itself. Therefore, it is important to establish the cause of the incontinence so appropriate treatment can be implemented.[10,11] Risk factors for fecal incontinence include frailty in older individuals, female sex, impaired mobility, and cognitive impairment.[8]

DOUBLE INCONTINENCE

Double incontinence is the involuntary loss of both urine and stool and can result in a breakdown of tissue in the affected area. Historically, a variety of terms have been utilized to describe IAD. These include diaper rash, perineal dermatitis, contact dermatitis, irritant dermatitis, moisture maceration injury, and heat rash.

INCONTINENCE-ASSOCIATED DERMATITIS (PERINEAL DERMATITIS)

In a 2007 consensus statement, IAD was defined as ''an inflammation of the skin that occurs when urine or stool comes into contact with perineal or perigenital skin.''[12] The panel chose the term *incontinence-associated dermatitis* as most reflective of the damage to the skin that involves more than the perineum. The perineal region includes the areas between the vulva/scrotum and anus, buttocks, and perianal, coccyx, and upper/inner thigh regions.[13] Individuals in any age group can experience IAD. Skin can be damaged by moisture and irritants that can arise from a variety of sources. One source is urinary and/or fecal incontinence. Urine and feces contain a variety of chemical irritants as well as moisture, all of which can result in IAD.

IAD is characterized by epidermal erosion and a macerated appearance of the skin.[12] IAD may present with some subjective signs, such as complaints of tingling, itching, burning, or pain, or objective signs that may also include erythema, swelling and/or oozing of the area, the formation of vesicles, and crusting and/or scaling.[13] In individuals with darker skin tones, the inflammation usually appears a different color than surrounding skin, possibly whitish, dark red/purple, or even yellow, but not necessarily red. In these individuals, palpation can be most helpful, as it reveals the presence of induration or firmness of the area compared with surrounding tissue (Figure 1).[12,14,15]

PREVALENCE

Incontinence

Statistics on incontinence are a bit unclear and difficult to compare, as some report just urinary or fecal incontinence, some report the two separately, and some studies report them together. In addition, some studies are of institutionalized individuals, whereas others report incontinence in community-living individuals. In community-living individuals who self-report, it has been reported that modesty and shame can impact the accuracy of results. Urinary incontinence prevalence rates range from 7.7% to 78.6% in long-term care and 22% to 46% in acute-care settings; fecal incontinence ranges from 7% to 42%, and double incontinence ranges from 20% to 78%.

Bliss et al[16] utilized cross-sectional assessment data from the minimum data set of 59,558 residents in long-term-care facilities and reported an incontinence prevalence of 59.8%. Of those with incontinence, 7.7% of residents had only urinary incontinence, 12.4% had only fecal incontinence, and 39.7% had double incontinence (both urinary and fecal incontinence). In another study of 981 incontinent long-term-care residents without perineal skin damage, 78.6% had both urinary and fecal incontinence, whereas 19.6% had only urinary incontinence, and 1.8% had only fecal incontinence. Women comprised more than 80% of the sample, with 98% being 65 years or older.[17,18] It would appear that the statistics for urinary incontinence have not changed significantly over time, as the Centers for Disease Control and Prevention in 1991 reported that it affected between 15% and 34% of hospitalized patients older than 6 years. A report by Newman et al[19] of long-term-care residents noted a rate of up to 50% for urinary incontinence and between 23% and 66% for fecal incontinence.

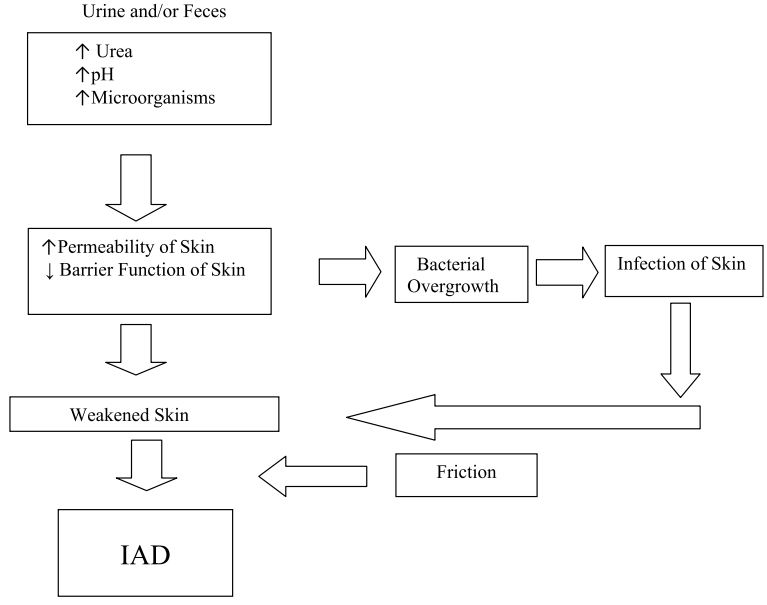
Urinary incontinence in acute-care settings has ranged from 22% to 46.6%,[20–23] whereas the range for fecal incontinence is 7% to 42%.[2,23–25] The overall prevalence of double incontinence varies in the literature from 19.7% to 32.4%.[26–29] Four reports note that double incontinence is more common (50%–70%) than either urinary or fecal incontinence alone.[26–29]

Acute and Long-term Care

In combined studies, urinary and/or fecal incontinence in acute care was found in 19.7% of patients,[30] whereas in long-term-care facilities, it has been reported to range from 48%[31] to 78.6% of residents.[17,32] Up to 42.7% of the incontinent acute-care patients had some perineal skin injury.[30] In the Bliss et al[17] study of 3405 long-term-care residents, 73% had incontinence. Of this group, 10% had only urinary incontinence, 15% had only fecal incontinence, and 48% had double incontinence. IAD was found in 5.7% of residents (n = 3405).

A study of 608 patients 4 years or older in 2 acute-care hospitals assessed the prevalence of incontinence and associated skin injury. This study identified an overall incontinence prevalence of

Figure 1.
CONCEPTUAL MODEL OF IAD



19.7%. Fecal incontinence occurred in 13.6% compared with 6.1% of those who experienced urinary incontinence. The lower incontinence prevalence rate could be explained in that 33% of the sample had an indwelling catheter and were considered continent for purposes of the study. In this sample, those 80 years or older had the highest prevalence of incontinence.[30]

ETIOLOGY/PATHOPHYSIOLOGY

Knowledge of skin layers and their associated function is helpful for accurate assessment and treatment of skin conditions. The skin is the largest organ in the body and the body's first line of defense. The skin's 2 primary layers are the epidermis and the dermis, which lie over the subcutaneous layer. Sebum, an oily substance produced in the dermal layer, is secreted through the sebaceous glands and functions to maintain skin suppleness and a waterproof barrier.[33] The protective secretions of the skin, for example, sebum, enable it to maintain the naturally acidic pH between 4 and 6.8 (mean, 5.5), which is often referred to as the acid mantle.[34,35] The acid mantle, described as a fine film, along with the stratum corneum, serves as a protective barrier, preventing transepidermal water loss and aiding in the formation and maturation of epidermal lipids. This protective barrier enhances the ability of the skin to remain intact.[36,37] Transepidermal water loss occurs passively as small amounts of water cross through the stratum corneum, the outermost layer of the epidermis. It is thus an indicator of the barrier function of the stratum corneum.[38,39]

The acid mantle of the skin can be harmed in a variety of ways. Washing the skin with harsh alkaline soaps or cleansers can disrupt and damage the acid mantle, requiring up to 14 hours to be restored.[36] Unfortunately, when the skin comes into contact with ammonia produced from the breakdown of urea in urine, the pH of the skin increases, leaving it more permeable and thus vulnerable. This permeability is further increased by the presence of excess moisture. Damage to the acid mantle impairs the skin's protective abilities to withstand bacterial invasion and IAD. Ongoing insults only result in more damage and increased chance for bacterial and/or fungal infection. IAD tends to be seen more frequently in individuals with diabetes mellitus, chronic renal disease, and cardiovascular disease, as these conditions tend to cause a higher pH of the skin, impairing the acid mantle.

Although the precise pathophysiology of perineal dermatitis and IAD is not entirely clear, it is well known that skin that is overhydrated is more permeable to irritants, at greater risk for breakdown, and more vulnerable to bacterial growth and fungal invasion. In addition, when both urinary incontinence and fecal incontinence are present in combination, the result is a higher pH.[40–43] In IAD, the liquid content of the urine overhydrates the skin, and the urea and ammonia present in the breakdown of urine lead to an alkaline pH, which is exacerbated further by fecal urase. These combine to alter the acid mantle and open up the skin portal to microorganism invasion. The skin barrier function is compromised after 2 or more days of prolonged exposure to water,[44] whereas urine or stool damages the skin in a much shorter time.[30] The prolonged effect of this is local dermatitis, tissue excoriation, and pain.[45,46] A conceptual model of IAD is presented in Figure 1.

Feces contain both proteolytic and lipolytic digestive enzymes, specifically protease and lipase, which are of an alkaline pH and normally inactivated as the stool passes through the digestive tract. However, in certain conditions, when the pH of the skin has increased, the digestive enzymes can once again be activated, causing greater skin irritation.[34] Skin that is frequently in contact with feces can become erythematous, taut, and painful, leading to IAD.[35] Skin with an impaired barrier function is more easily invaded by bacteria,[47] resulting in moisture lesions or IAD.[35] Soft or formed stool can injure the skin, particularly when it is not removed promptly. In the presence of diarrhea or frequent, loose, watery stools, the skin becomes more irritated from digestive enzymes, overhydrated from the liquid stool content, more permeable to water, and more vulnerable to IAD, as well as friction and shear forces. In the older individual with drier skin, the breakdown can occur more quickly.[35] When skin exposed to incontinence is also exposed to mechanical forces, such as friction and shear, the skin can more quickly become excoriated.

RISK FACTORS

A number of risk factors and variables contribute to altered skin integrity, which can lead to IAD or a pressure ulcer (PrU). Some of the PrU risk factors are similar to IAD, whereas others are more specific to it. An earlier conceptual model by Brown and Sears[13] was based on an integrative review of 16 articles. The model delineated 3 factors that contribute to IAD: (1) tolerance of the tissue, (2) the perineal environment, and (3) toileting ability of the individual. More recently, Gray[48] identified 6 possible risk factors for IAD. These include "(1) chronic exposure to moisture, (2) fecal and urinary incontinence, (3) use of a containment device, (4) alkaline pH, (5) overgrowth or infection with pathogens, and (6) friction."[48] Other risk factors associated with IAD include poor skin condition (eg, aging or steroid use), pain, inadequate oxygenation of skin, fever, and decreased mobility.[12,15,49,50]

Moisture

The source of excessive moisture in an incontinent patient can arise from urine, perspiration, or liquid stool.[14] The risk for skin

damage is directly related to the frequency and amount of incontinent urine and the containment device (eg, pad or brief) used to capture moisture next to the perineal skin and the resultant perspiration. The ongoing presence of moisture leads to overhydration or maceration of the skin, thereby compromising its barrier function to unwanted water, chemicals, and microorganisms. Normal, as well as pathogenic, bacterial flora increase when the moisture barrier is compromised, increasing the chance of secondary infection. A secondary infection is more likely in fecal or both fecal and urinary incontinence.[43]

Skin pH

Normal skin pH is slightly acidic; however, it becomes alkaline when exposed to urinary or fecal incontinence. Both the urea and the ammonia in the urine increase the pH. Normal bacterial overgrowth also leads to urea production. Any prolonged exposure of the perineal skin to moisture from perspiration, particularly under an incontinence brief, results in an alkaline pH. In the presence of feces, the pH can rise to 8 or higher,[43] resulting in a greater risk of damage and susceptibility for bacterial growth.[36]

Bacterial Colonization

There is an overgrowth of bacteria in the perineal area with prolonged exposure to urine, feces, or perspiration. Again, this is increased when an incontinence brief, pad, or containment device is used. With the overgrowth of bacteria, infection is more likely. Patients more prone to skin infections include those with diabetes, obese patients with skin folds within the perineum, and immunocompromised patients. The most common infecting pathogen is *Candida albicans*, probably related to it being a normal inhabitant of the gastrointestinal tract,[43,51] as well as the fact that it begins multiplying on the skin at a skin pH of 6 to 7.[52] Another bacterial condition that is being seen more frequently is caused by *Clostridium difficile*, a Gram-positive, spore-forming anaerobic bacteria that is a normal inhabitant of the large intestine, but ''kept in check'' by the bacteria inherent to the area. In some individuals, particularly the frail and/or acutely ill older adults on antibiotics, the normal intestinal bacteria are removed, allowing the *C difficile* to multiply, resulting in acute diarrhea.[53]

Friction

Friction is defined by the National Pressure Ulcer Advisory Panel (NPUAP)[54] as ''the resistance to motion in a parallel direction relative to the common boundary of 2 surfaces.'' The NPUAP added that ''successful pressure ulcer prevention will address contributing factors such as friction and moisture.'' Friction is increased when perineal skin rubs over containment materials, clothing, or surfaces such as a bed or chair.[19] Friction also occurs with vigorous rubbing of the perineal skin during cleansing. Overhydrated skin is more easily abraded or blistered in the presence of friction than is dry skin, so minimizing or eliminating skin exposure to friction is important in preventing IAD, as well as PrUs. Overall, friction damage tends to be more superficial than PrUs.[43]

Aging

The skin changes that accompany aging are an additional risk factor that can increase the chances of perineal dermatitis changes. As individuals age, the skin becomes thinner, drier, and less elastic, and without the skin's fat emulsion, the ability of the epidermis to serve as a water and protective barrier is compromised.[55] Although the stratum corneum normally contains about 15% water, in older individuals it decreases to less than 10%.[56] In addition, with aging, the cells are replenished less rapidly, resulting in an increased healing time when damage does occur. Sensory perception, immune function, and thermoregulation also are decreased.[57] When the skin contains excess moisture, it is more vulnerable to friction and shear injuries.[35] In a survey of 608 subjects, the group older than 80 years had the highest prevalence of incontinence.[30]

Other risk factors include mechanical chafing/friction and use of topical cleansers or topical agents that may alter the skin's own protection. Steroid use can alter skin condition as can fever, continual exposure to moisture, immobility, decreased sensory perception, and decreased cognitive awareness.[13,15] Of all of the risk factors, fecal incontinence appears to be associated most strongly with IAD.[30]

ASSESSING PERINEAL SKIN INJURY AND RISK FOR IAD

A variety of tools are available to assess risk for perineal skin breakdown. The Perineal Assessment Tool is a 4-item instrument based conceptually on the 4 determinants in perineal skin breakdown: intensity and duration of irritant, perineal skin condition, and contributing factors. Subscales are rated from 1 (least risk) to 3 (most risk), with a total score range of 4 to 12.[58] The tool is widely used, and Nix[58] reported that nurse wound care specialists have tested the tool and concurred that it is a valid measurement of IAD risk (Figure 2).

The Perirectal Skin Assessment Tool (PSAT) by Brown and Sears[13,59] uses descriptions of skin color and integrity as an estimate of the amount of actual skin damage resulting from the incontinence. In addition, the size of the affected area and patient symptoms are also rated. The Skin Assessment Tool (SAT) was proposed by Kennedy et al[60] and categorizes IAD based on a cumulative score from 3 categories (Figure 2). The

Figure 2.
2 INSTRUMENTS FOR EVALUATING INCONTINENCE AND INCONTINENCE-ASSOCIATED DERMATITIS (IAD)

Perineal Assessment Tool[58]

Factors	Scoring
I. Intensity of irritant	Cumulative score is calculated; higher the score, higher the risk for IAD
1. Formed stool and/or urine	
2. Soft stool with or without urine	
3. Liquid stool with or without urine	
II. Duration of irritant	
1. Linen/pad changes at least every 8 hours	
2. Linen/pad changes at least every 4 hours	
3. Linen-pad changes at least every 2 hours	
III. Perineal skin condition	
1. Clear and intact	
2. Erythema/dermatitis with or without candidiasis	
3. Denuded/eroded with or without dermatitis	
IV. Contributing factors	
1. 0-1 contributing factors	
2. 2 contributing factors	
3. 3 or more contributing factors	

Skin Assessment Tool (SAT)[60]

I. Area of skin breakdown	Cumulative score is calculated; higher score indicates more severe IAD
0. None	
1. Small area (<20 cm^2)	
2. Moderate area (20-50 cm^2)	
3. Large area (>50 cm^2)	
II. Skin redness	
0. No redness	
1. Mild redness (blotchy and non-uniform)	
2. Moderate redness (severe in spots but not uniform in appearance)	
3. Severe redness (uniformly severe in appearance)	
III. Erosion.	
0. None	
1. Mild erosion involving epidermis only	
2. Moderate erosion of epidermis with moderate involvement of dermis with low volume or no exudates	
3. Extreme erosion of epidermis and dermis with moderate volume and persistent exudate	

Adapted with permission.[50-60]

categories include the area of skin breakdown, skin redness, and skin erosion. Because the PSAT and the SAT tools use precise measurements, they are used more often in clinical research than in clinical practice.

A recently published tool, the Incontinence-Associated Dermatitis and Its Severity Instrument, is used to identify IAD and its severity.[61] The tool contains the 4 constructs of location, redness, skin loss, and rash; is valid and reliable; can be used by nurses expert in assessing IAD, as well as nursing care assistants; and allows for serial evaluations over time.[61]

Although further research is needed to evaluate the effectiveness of each assessment tool, it is paramount that the individual who is experiencing incontinence has a comprehensive assessment to determine the incontinence etiology and rule out related factors or conditions that contribute to the incontinence. Once this is accomplished, a thorough perineal, perirectal, and IAD assessment can be completed. The assessments should be individualized and ongoing, and should include a thorough pain assessment and interventions to manage the incontinence, as well as the moisture-associated skin damage. Guidelines for how often assessments are done have not been definitively established; however, a literature review clearly indicates that when clear, consistent prevention and treatment protocols have been implemented, it has been beneficial to incontinent patients who had experienced skin breakdown. Whichever tool an agency chooses to use, the agency should clearly define the frequency of assessment and establish reliability between caregivers who use the tools.

ASSESSMENT, PREVENTION, AND TREATMENT OF IAD

Can Incontinence Be Prevented?

It is essential that the cause of the urinary incontinence be identified and treatment interventions directed to modifying or eliminating the cause. Muscles and ligaments of the pelvic floor serve to maintain continence. Whereas pelvic floor muscle training and biofeedback are effective in younger women, research has demonstrated only short-term effectiveness in older women.[4] Although the 1992 Urinary Incontinence Guideline Panel of the Agency for Health Care Policy and Research[62] delineated recommendations for diagnosis and treatment of urinary incontinence, they are considered not relevant for clinical practice today.

When Incontinence Occurs, How Can the Associated Dermatitis Be Prevented?

Two review articles on IAD were located following an extensive literature search.[14,63] The literature was consistent in delineating that prevention of IAD includes routine inspecting, cleansing, moisturizing, protecting the skin, and incontinence training as the core of a perineal skin care program. The plan of care must be individualized and include assessment and management of the etiology, assessment of risk and of the perineal skin, gentle cleansing and moisturizing, skin barrier/protectant application, and use of containment devices when indicated.[50,64] For everyone, regularly and promptly removing soil and irritants from the skin is important along with preventing or minimizing exposure of the skin to additional damaging irritants.[43]

How Do Clinicians Distinguish Between IAD and a PrU?

It is important to distinguish IAD from Stage I or Stage II PrUs, which usually occur over a bony prominence such as the coccyx. As opposed to a moisture lesion, a PrU results from pressure and/or shear and tends most often to be located over a bony prominence, whereas a moisture lesion is generally located over

natal clefts (eg, gluteal cleft) and/or in the perianal area, and not over a bony prominence. Nonblanchable erythema can be seen with both a Stage I PrU and IAD, but with IAD the erythema is more irregular in its borders and dispersed throughout the area exposed to the urinary or fecal incontinence.[65] A PrU tends to be a discrete lesion that is more circular in shape with distinct edges and can be of full thickness, whereas IAD is diffuse with irregular edges, is superficial, is found more so in the skin folds, may cause pruritus, and when on the buttocks may have a ''kissing'' shape. There is no necrosis in a moisture lesion, where there can be in a PrU. A full-thickness injury is usually not seen with IAD unless there is infection.[15] If the caregiver is cognizant of the etiology, signs, and symptoms, he/she is better able to differentiate between IAD and a PrU.[66] Differentiating between the 2 conditions is important for research, reporting, appropriate treatment, and reimbursement.[15,66]

Maklebust and Magnan[67] demonstrated that individuals who were both incontinent and immobile had a 37.5% greater risk of developing a PrU, making clear the connection between IAD and PrUs. As injury to the skin (PrU) is now a quality indicator in acute care, accurately distinguishing between IAD and a PrU is most important.

Where Would One Begin in Prevention?

For any individual at risk for IAD, a preventive regimen needs to be implemented that includes inspection, gentle cleansing, routine use of moisturizers, and a moisture barrier/skin protectant to minimize skin contact with urine and feces.

Inspecting: Assessment begins with a thorough inspection of the perineal skin. Assessment of the perineal skin needs to be done on admission to the healthcare treatment setting so that the prevention and treatment plan can be individualized and periodically reviewed throughout the stay to determine the effectiveness of the interventions. Regular and thorough inspection of the skin and, in particular, noting skin color changes over time are important. Skin assessment with each turn or repositioning and each time the skin is cleansed is helpful.

Normally the perineal skin resembles that of the lower abdomen. IAD will initially result in erythema of the skin, which will progress to a dark red appearance that can be accompanied by erosion, blistering, and a serous exudate. Incontinence resulting from urine tends to affect the labial folds in females and the inferior of the scrotum in males, whereas fecal incontinence tends to be evidenced in the perianal space and eventually the posterior thighs.[48]

Inspect the perineal skin, paying particular attention to skin folds as the opposing surfaces trap and harbor moisture. Should a secondary infection be present, it can be identified and treated as well. The warm, moist environment of the skin folds, as well as the entire perineal area, encourages bacterial and fungal colonization, overgrowth, and infection. IAD and candidiasis often coexist and present as a maculopapular rash with central confluence and satellite lesions surrounding the area. A study of 976 acute-care inpatients reported that candidiasis was seen 18% of the time.[30] In addition, friction occurs when the skin folds lie or rub against one another, contributing to tissue breakdown. Once incontinence occurs, the overall goals are to keep the skin dry and free of irritants. A wide array of techniques exists to control exposure of the skin to moisture; however, it is beyond the scope of this article to discuss all of the techniques.

Cleansing: Intact epithelium serves as the barrier to protect normal skin. Normal skin pH is acidic and in healthy states varies from 4 to 6.8, but more commonly from 4.9 to 5.5 to maintain the acid mantle.[48] The ideal skin cleanser should have a pH as close as possible to that of normal skin. Although the packaging on most products does not note the pH, it can be ascertained by checking the Material Safety Data Sheet or checking with the manufacturer.

The cleanser should lift the urine and feces from the skin without altering the acid mantle or causing an excess removal of lipids.[48] In contrast, perineal skin cleansers combine a surfactant and detergent to loosen and remove residue and irritants. A surfactant can act as a detergent or emulsifier. Some also contain emollients, moisturizers, or humectants to restore or preserve the skin barrier functions.[68] Also available are no-rinse cleansers and perineal cleansing products that incorporate a cleanser, moisturizer, and moisture barrier within a disposable towelette (Table 1).[48]

Soap is made from a mixture of fatty acids and alkali and has a higher (alkaline) pH.[52] Soap also requires application with water and a washcloth, which remove important oils from the skin, compromising the barrier function.[48] Generally, soap is to be avoided because of its high alkaline content. Skin cleansers are a better choice to cleanse the skin.

Cleanse skin gently, using care to avoid rigorous scrubbing or friction to minimize the risk of compromising the skin barrier function.[43,48,68] In situations of dried or difficult-to-remove feces, gently applying a moistened cloth on the area and softly patting around if needed can be most helpful. In 4 different studies, a variety of combinations of a perineal skin cleanser and skin protectant resulted in less erythema.[69–72] Three studies documented that staff costs associated with using soap and water were greater than costs associated with a no-rinse and combination products.[73–75]

Moisturizing: Maintenance of skin moisture is important for all patients.[14] A moisturizer is a hydrator or lubricant used to preserve suppleness and enhance the skin barrier function.[57] Moisturizers contain humectants (eg, glycerine, lanolin, methyl

Table 1.
SKIN CLEANSER TYPES AND DEFINITIONS

Cleanser Category[a]	Cleanser Definition
Emollient	A substance that softens and soothes the skin
Humectant	A substance that preserves the moisture or water content of the skin
Moisturizer	Complex mixtures of chemical agents to make the external layers softer and more pliable; product works by increasing water content through reduction or evaporation
Surfactant	Substance that acts to lower surface tension of a liquid, allowing easier spreading, as well as lowering the tension between 2 liquids, or between a liquid and a solid

[a]Printed with permission, Nix and Ermer-Seltun.[68]

glucose, or mineral oil) or emollients (eg, lanolin). Avoid products with strong concentrations of humectants (chemicals such as glycerine, a-hydroxyl acids, urea, and lactic acid that act to retain moisture in the skin).[15] Moisturizers can be applied separately or as part of a specially designed cleansing system.

Protective garments and padding: Use superabsorbent adult disposable briefs designed to absorb and wick moisture from incontinence away from the skin when the individual is ambulating. Using an underpad in bed and a brief when out of bed is generally recommended.[76] Underpads are not tight or impermeable as are briefs, making them more desirable in most situations.[41,76] Check the brief and underpad at least every 2 hours to ensure it is dry. The area of the skin lying on an underpad is often moist. Thus, when turning the individual, it is important to expose this area to air for evaporation of moisture and drying of the skin surface. As possible, implement strategies to reduce, eliminate, or contain the urinary and/or fecal incontinence, including ongoing vigilance on the part of the caregiver.

Protecting the skin: It is essential to protect the skin from contact with feces to the greatest extent possible to prevent IAD and related PrU risk. The skin can be protected by frequent cleansing following urine or feces release. Skin protectants or barrier products are very helpful in protecting the skin. Lastly, urinary or fecal containment devices are available.[1,77,78]

A topical skin protectant/barrier is a product that isolates and protects the skin from the effects of excessive moisture, urine, and/or stool.[14] Common active ingredients in a protectant or barrier include dimethicone, petrolatum, or zinc oxide, either alone or in some combination.[48] Dimethicone is easier to apply and less greasy than the other 2 mentioned, but consequently becomes less occlusive to moisture. Petrolatum is quite thick but easily removed from the skin and is more effective against urine than stool. Zinc oxide is a white powder and is usually mixed in a petrolatum base, making it a bit more difficult to remove from the skin. Zinc oxide has mild antiseptic and astringent properties and is effective for both urine and stool.[48] Application of a skin protectant/barrier is recommended for all patients at risk of IAD. Once the skin becomes denuded, there is less success in getting the product to adhere. Most skin protectants can also be incorporated into a 1-step cleansing system. Protectants are packaged as creams or ointments, with ointments containing less water and more oil, whereas creams are oil in a water base. The oil in the products can be either lanolin or petrolatum.[48] The addition of powder to an ointment results in a paste, which is thicker and more durable than ointment without powder.[79] An important principle to bear in mind is that the easier the product is removed with water, the less durable barrier it will be for urine, stool, or exudates.[76]

Another type of product available is a liquid skin sealant, which is a polymer in combination with a solvent. Upon application to the skin, the solvent evaporates and the polymer dries, forming a moisture barrier to protect the underlying skin. Because alcohol is a component, it is contraindicated when perineal irritation is present.[48] However, products are available without alcohol.

For individuals who require frequent cleansing, skin sealants would not be appropriate because they are easily removed during cleansing.

Although a variety of products are on the market for preventing IAD, the best approach is always to treat the cause of the incontinence and stop or minimize the exposure of the skin to it.[1,12,34]

Reducing friction: Friction damage tends to be superficial.[43] Preventing or minimizing friction is important to maintain healthy tissue and minimize vulnerability to IAD, as tissue injured by friction is more vulnerable to damage from IAD. Measures to reduce friction include using a turn sheet and lifts, not scrubbing the skin in the perineal area, and using a barrier/protectant.

Development and treatment of IAD: The best treatment is prevention. Once IAD occurs, intensified efforts to protect and treat the IAD are a must. Following an assessment of the perineal area skin and an evaluation of the IAD causative factors, duration, and previous interventions and results, an individualized plan of care can be developed. At the very least, these individuals require cleansing after each incontinent episode, followed by application of a moisturizer/moisture protectant/barrier product.

When fecal incontinence is present, application of a product that is effective against stool penetration, such as zinc oxide,

would be advised.[48] When the individual is experiencing liquid stool, the interventions must become more aggressive. Again, a zinc oxide–containing barrier product will help protect the skin from the highly irritating stool enzymes. It is not necessary to remove the entirety of the product at each cleansing, so caution and avoidance of vigorous scrubbing are important. When it is indicated to remove all the zinc oxide, mineral oil is helpful.[48]

Recently, a tool was proposed to classify and treat IAD that uses descriptions of the current skin condition to determine if IAD is present. The tool is called the IAD Intervention Tool (IAD-IT) (Figure 3).[15] Three factors comprise the tool and include (1) change in skin color as compared with surrounding skin (or for darkly pigmented skin individuals, induration or hardness), (2) presence or absence of blisters or weeping, and (3) presence or absence of symptoms in areas exposed to the incontinence. The assessments are used to determine if the individual has intact at-risk skin or mild, moderate, or severe IAD. The unique aspect of this scale is that it includes suggested interventions for each category. The tool can be used by both licensed and unlicensed staff.[15]

Use of containment devices: A variety of containment devices are available, with the most common being underpads or absorbent briefs that wick moisture away from the skin, as previously discussed. Indwelling urinary catheters can be used when deemed appropriate; however, there is a higher incidence of urinary tract infections when these are used.[80] External urinary catheters can help contain the urine.

Fecal containment devices are used to divert incontinent stool away from the perianal area and to aid in preventing fecal contamination of area wounds and dressings. Devices noted in the literature also include closed external collection systems, colostomy bags, and fecal pouches.[30] In addition to diverting stool, the device helps decrease the embarrassment of loose diarrhea. One system provides access for administration of rectal medication delivery and retention. Although the tube-and-pouch device generally works well, it is difficult to achieve and maintain a seal when the tissue is excoriated around the anus. It is important to ensure that the patient has an intact anal sphincter that is able to hold the device in place.[35] These are not appropriate for patients who are ambulatory, are agitated, or who slide up and down in bed because the device can be dislodged. These devices are not recommended for chronic incontinence[81] and are not recommended for longer than 29 days.

Bowel management systems were found to reduce the number of PrUs in an intensive care unit,[82] whereas in both long-term care and a burn unit, the result was a reduction in the incidence of perianal excoriation and fewer bed linen changes.[83] Although few adverse effects have been encountered, a decrease in sphincter tone can occur with prolonged use.[84]

PAIN ASSESSMENT AND MANAGEMENT

A vicious cycle of pain is most often associated with IAD, given the skin damage that is present and the often ongoing exposure to further irritants, as well as the clean-up necessitated with each subsequent episode of incontinence. IAD pain is often ''likened to that of burns on an individual's buttocks and thighs.''[15] Pain obviously increases morbidity and in some cases length of stay related to the decreased mobility and lack of cooperation with some interventions.[15]

SUGGESTED INTERVENTIONS/PROTOCOL

In addition to the already described prevention and treatment interventions, additional suggested protocols for addressing IAD are provided in Figure 4. The IADIT, depicted in Figure 3, targets interventions to the classification of IAD. The figure has 3 columns. The far left column contains the classification of IAD along with a picture to illustrate the 5 categories (high risk, early IAD, moderate IAD, severe IAD, and fungal appearing rash). The definitions for each category are in the middle column. The far right column contains the appropriate interventions for each of the categories. The tool is quick and simple to use and does not require the clinician to do any measurements, nor does it require extensive training, making it appropriate for use by both licensed and unlicensed staff. The uniqueness of this tool is the interventions for each category, which guide care plan development.[15] Figure 5 is yet another recommended protocol for prevention and treatment of IAD developed by Gray et al.[12] The first column addresses the condition of the skin, the middle column the treatment goals based on skin condition, and the right column delineates specific interventions that can be implemented.

The authors of this article recommend a systematic approach to the management of incontinence and associated dermatitis, using a comprehensive tool that addresses etiology, assessment, prevention, treatment, and evaluation. Several tools have been developed with this purpose in mind. The Wet-to-Dry Algorithm (Figure 6), designed by the authors, is an example of a tool that reminds caregivers to further explore the incontinence, recommends a thorough evaluation of the incontinence and skin, and directs the healthcare professional to strategies that are intended to prevent further skin breakdown. The tool refers the caregiver to treatment options, includes client and family education, and challenges the caregiver to revise and reevaluate the care if initial efforts are not successful.

STAFF EDUCATION: INITIAL AND ONGOING

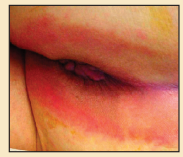
Education of healthcare providers and patients is essential to effective management of IAD. At the outset, it is important to establish whether the problem is IAD or a split-thickness PrU.

Figure 3.
INCONTINENCE-ASSOCIATED DERMATITIS INTERVENTION TOOL (IADIT)

Skin Care for Incontinent Persons

The #1 priority is to address the cause of incontinence. Use this tool until incontinence is resolved.

1. **Cleanse incontinence ASAP and apply barrier.**
2. **Document condition of skin at least once every shift in nurse's notes or per organization's policy for documenting skin breakdown.**
3. **Notify primary care provider when skin injury occurs and collaborate on the plan of care.**
4. **Consider use of external catheter or fecal collector.**
5. **Consider short term use of urinary catheter only in cases of IAD complicated by secondary infection.**

	Definition	Intervention
HIGH-RISK	Skin is not erythematous or warmer than nearby skin but may show scars or color changes from previous IAD episodes and/or healed pressure ulcer(s). Person not able to adequately care for self or communicate need and is incontinent of liquid stool at least 3 times in 24 hours.[1]	1. Use a disposable barrier cloth containing cleanser, moisturizer, and protectant.[2,3] 2. If barrier cloths not available, use acidic cleanser (6.5 or lower), **not soap** (soap is too alkaline); cleanse gently (soak for a minute or two **– no scrubbing**); and apply a protectant (ie: dimethicone, liquid skin barrier or petrolatum). 3. If briefs or underpads are used, allow skin to be exposed to air for 30 minutes twice a day by positioning semi-prone. Use containment briefs only for sitting in chair or ambulating – not while in bed. 4. Manage the cause of incontinence: a) Determine why the person is incontinent. Check for urinary tract infection, b) Consider timed toileting or a bladder or bowel program, c) Refer to incontinence specialist if no success.[4]
EARLY IAD 	Skin exposed to stool and/or urine is dry, intact, and not blistered, but is pink or red with diffuse (not sharply defined), often irregular borders. In darker skin tones, it might be more difficult to visualize color changes (white, yellow, very dark red/purple) and palpation may be more useful. Palpation may reveal a warmer temperature compared to skin not exposed. People with adequate sensation and the ability to communicate may complain of burning, stinging, or other pain.	
MODERATE IAD 	Affected skin is bright or angry red – in darker skin tones, it may appear white, yellow, or very dark red/purple. Skin usually appears shiny and moist with weeping or pinpoint areas of bleeding. Raised areas or small blisters may be noted. Small areas of skin loss (dime size) if any. This is painful whether or not the person can communicate the pain.	↑ ***Include treatments from box above plus:*** 5. Consider applying a zinc oxide-based product for weepy or bleeding areas 3 times a day and whenever stooling occurs. 6. Apply the ointment to a non-adherent dressing (such as anorectal dressing for cleft, perforated film absorbent dressing for flat areas, or ABD pad for larger areas) and gently place on injured skin to avoid rubbing. Do not use tape or other adhesive dressings. 7. If using zinc oxide paste, **do not scrub the paste completely off** with the next cleaning. Gently soak stool off top then apply new paste covered dressing to area. 8. If denuded areas remain to be healed after inflammation is reduced, consider BTC ointment (balsam of peru, trypsin, castor oil) but remember balsam of peru is pro-inflammatory. 9. Consult WOCN if available.
SEVERE IAD 	Affected skin is red with areas of denudement (partial-thickness skin loss) and oozing/bleeding. In dark-skinned persons, the skin tones may be white, yellow, or very dark red/purple. Skin layers may be stripped off as the oozing protein is sticky and adheres to any dry surface.	↑ ***Include treatments from box above plus:*** 10. Position the person semiprone for 30 minutes twice a day to expose affected skin to air. 11. Consider treatments that reduce moisture: low air loss mattress/overlay, more frequent turning, or astringent of acetic acid/aluminum acetate. 12. Consider the air flow type underpads (without plastic backing).
FUNGAL-APPEARING RASH 	This may occur in addition to any level of IAD skin injury. Usually spots are noted near edges of red areas (white, yellow, or very dark red/purple areas in dark-skinned patients) that may appear as pimples or just flat red (white or yellow) spots. Person may report itching which may be intense.	Ask primary care provider to order an anti-fungal powder or ointment. Avoid creams in the case of IAD because they add moisture to a moisture damaged area (main ingredient is water). In order to avoid resistant fungus, use zinc oxide and exposure to air as the first intervention for fungal-appearing rashes. If this is not successful after a few days, or if the person is severely immunocompromised, then proceed with the following: 1. If using powder, lightly dust powder to affected areas. Seal with ointment or liquid skin barrier to prevent caking. 2. Continue the treatments based on the level of IAD. 3. Assess for thrush (oral fungal infection) and ask for treatment if present. 4. For women with fungal rash, ask healthcare provider to evaluate for vaginal fungal infection and ask for treatment if needed. 5. Assess skin folds, including under breasts, under pannus, and in groin. 6. If no improvement, culture area for possible bacterial infection.

1. Bliss DZ, Zehrer C, Savik K, et al. Incontinence-associated skin damage in nursing home residents: a secondary analysis of a prospective, multicenter study. *Ostomy Wound Manage*. 2006;52:46–55.
2. Institute for Healthcare Improvement. Prevent Pressure Ulcers: How-To Guide. May 2007. Available at: http://www.ihi.org/nr/rdonlyres/5ababb51-93b3-4d88-ae19-be88b7d96858/0/pressureulcerhowtoguide.doc, accessed 10/21/07.
3. Gray M, Bliss DB, Ermer-Seltun J, et al. Incontinence-associated dermatitis: a consensus. *J Wound Ostomy Continence Nurs*. 2007;34:45-54.
4. Junkin J, Selekof JL. Prevalence of incontinence and associated skin injury in the acute care inpatient. *J Wound Ostomy Continence Nurs*. 2007;34:260-269.

Figure 4.
EXAMPLE OF INTERVENTIONS FOR INCONTINENCE SKIN CARE PROTOCOL[18,68,94]

- Assess the perineal area skin daily in high-risk and incontinent individuals
- Document the perineal assessment
- Gently cleanse the skin during bathing and at the time of soiling with warm water and a mild pH-balanced perineal cleanser using minimal friction
- Use high-absorbency underpads to wick moisture away from the skin
- Avoid using diapers and adult briefs in bed; they should be used only when the patient is out of bed
- Minimize friction and shear forces via proper positioning, repositioning, and transfer techniques
- Avoid the use of antibacterial and perfumed products that may further irritate or injure the skin
- In the presence of mild irritation and intact skin, apply a skin protectant moisture barrier after each cleansing, per physician's order if needed
- In the presence of severe irritation and nonintact skin, apply a zinc oxide–based skin protectant moisture barrier, per physician's order. This can be removed with mineral oil.
- In the incontinent individual who has itching or burning in the perineal area, assess for a secondary yeast infection; apply a skin protectant moisture barrier containing an antifungal as indicated per physician's order.

Periodic education on the agency's IAD protocol, adherence to it, and areas for improvement are needed. Education on care of perineal-rectal area skin is needed, along with what to assess, how to develop and evaluate an individualized treatment plan, and what conditions to report (eg, increased inflammation, fever, pain, edema, etc).[85] As new IAD products or protocols are initiated, education for nursing care staff is mandatory.[86] Accurate documentation is a must.

RESEARCH RELATED TO IAD

Lyder et al[87] studied 15 patients free of IAD at baseline and compared a nonstructured regimen for 4 weeks and then a structured regimen for 4 weeks. The structured regimen consisted of use of a cleanser, moisturizer, and moisture repellant to the perineal area skin following each incontinence episode. There was no difference in the incidence of IAD in the 2 groups, as each had a 23% incidence of IAD. Another study of 32 LTC facility residents occurred over 3 weeks where residents were randomly assigned to 1 of 2 skin care regimens. Subjects in group 1 had their perineal skin cleansed with soap and water after each incontinence episode, followed by application of a moisturizing lotion, whereas those in group 2 had the perineal area cleansed with a no-rinse skin cleanser after each episode, followed by application of a barrier cream after the first incontinence episode of each shift. There was no significant difference in maintenance of skin integrity between the 2 groups (69% vs 72%); however, there was a significant reduction in staff time for perineal skin care when the no-rinse cleanser was used (mean reduction in time = 79 minutes per day).[88]

Another 6-week prospective study compared the efficacy of 4 IAD skin regimens in a national study of 1918 LTC facility residents in multiple LTC facilities. Skin care regimens included (1) an acrylate polymer-based film applied 3 times per week, (2) a 43% petrolatum-based ointment applied after every incontinent episode, (3) a cream consisting of 12% zinc oxide and 1% dimethicone applied after every incontinent episode, and (4) an ointment of 98% petrolatum applied after each incontinent episode. There was no significant difference in IAD between regimens. The incidence of IAD overall was 3.4%, whereas the incidence of perineal damage (did include PrUs) was 4.6%. The use of a defined skin care regimen and quality skin care products was felt to contribute to a low incidence of IAD in a high-risk population.[86] Two other studies[5,89] compared soap and water to no-rinse perineal cleansers in 10 older women over 3 weeks. The regimens studied were (1) soap and water alone, (2) no-rinse cleanser alone, (3) soap and water and skin protectant, and (4) no-rinse skin cleanser and moisturizer. For those treated with soap and water alone, erythema, transepidermal water loss, and altered skin pH were most severe, whereas these were least severe for those women treated with the lower pH cleanser and moisturizer. Unfortunately, the study time was short, and the sample was small. In a 12-week preintervention and 12-week postintervention study with 34 subjects, a thick, disposable washcloth was used that contained a no-rinse cleanser, moisturizer, and skin protectant (3% dimethicone). There was a statistically significant difference between the preintervention and postintervention incidence of IAD (15% vs 0%).[74]

Three other studies examined the efficacy of structured perineal skin care regimens, including IAD, skin tears, and/or PrUs.[71,73,74] All 3 studies documented that the implementation of a structured skin care regimen over a 12-week period resulted in a statistically significant reduction in the incidence of perineal skin breakdown and/or sacral skin breakdown.

A 77-bed LTC facility was the setting for a study trialing an incontinence management protocol.[90] The protocol was based on the 1992 and 1996 Agency for Health Care Policy and Research (now the Agency for Healthcare Research and Quality) clinical practice guidelines on incontinence and PrU prevention (US Department of Health and Human Services).[62] The protocol included a comprehensive urinary function assessment and potential etiologies of incontinence; scheduled, individualized toileting or bladder training; every-2-hour wetness assessments; and cleansing and barrier cream application following every incontinent episode. There were 63 residents followed up for a total of 10,285

Figure 5.
RECOMMENDATIONS FOR PREVENTION AND TREATMENT OF INCONTINENCE-ASSOCIATED DERMATITIS

Condition of Skin	Treatment Goals	Interventions
Intact skin in person w/urinary or fecal incontinence	Prevent IAD Minimize contact w/irritants (urine, stool, and excessive moisture) Maintain skin protection Reduce barriers to appropriate care	Begin a structured skin care regimen 1) Cleanse perineal skin daily and after each major incontinence episode using no-rinse cleanser 2) Avoid scrubbing skin; use soft or disposable washcloth 3) Apply appropriate moisturizer (often a cream product containing humectants and emollient) 4) Apply skin protectant to minimize contact between urine &/or stool (ointment containing petrolatum, zinc oxide, dimethicone, or combination of these products), or apply a copolymer film product (skin sealant) in patients judged to be at high risk for developing IAD (high-volume/high-frequency urinary or fecal incontinence, double fecal and urinary incontinence, and fecal incontinence w/liquid stool). 5) Combine steps using a product containing a cleanser plus a moisturizer with or without a skin protectant. 6) Educate caregivers to apply structured skin regimen and routinely assess for IAD. 7) Begin aggressive treatment program for underlying incontinence.
Mild-to-moderate IAD (skin remains intact but erythema present, with or without candidiasis)	Minimize contact with irritants (urine, stool, and excessive moisture) Maintain skin protection Eradicate cutaneous Candidiasis	1) Combine a structured skin care program with active treatment of IAD 2) Routinely cleanse and moisturize the skin using above noted steps 3) Routinely apply skin protectant, options include: Ointment containing petrolatum, zinc oxide, dimethicone, or combination of these products. a) A copolymer film product (skin sealant) b) Skin protectant ointment with active ingredients designed to promote wound healing (Balsam-Peru, castor oil, and trypsin [BCT] ointment or gel) 4) Treat cutaneous candidiasis when present 5) Apply moisturizer or moisture-barrier combination product with antifungal agents (azole or allylamine) 6) Educate caregivers to apply structured skin regimen and routinely assess for resolution or progression of IAD 7) Evaluate or begin management program for underlying incontinence

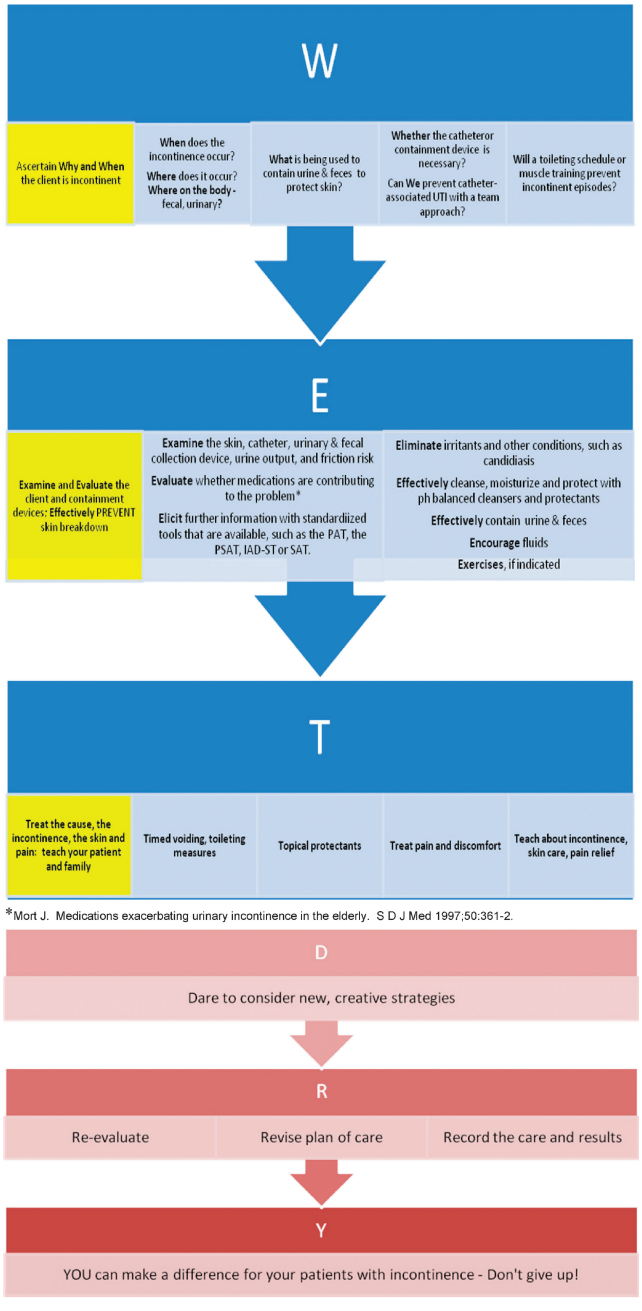
Reprinted with permission from Gray et al.[12]

days. Of these 63 residents, 34 (54%) received incontinence management treatments, resulting in 14 becoming dry with the toileting program (no more than 2 wet episodes/24 hours).

SUMMARY

IAD is a very prevalent problem that demands serious attention. Incontinence and IAD are significant issues that cause costly consequences. And, incontinence ranks as the second-leading reason for long-term-care facility placement in the United States.[24,91–93] Frequent and ongoing assessment is crucial. Although routine assessments on admission in long-term care include incontinence, this is not always true in acute care. Identifying the cause of the problem is one of the first steps in treatment, and this can be done using a validated and reliable assessment tool. To the fullest extent possible, identify interventions to eliminate the cause of the incontinence if

Figure 6.
WET-TO-DRY ALGORITHM



possible, and when it is not possible to eliminate the cause, implement interventions to minimize it. Early preventive intervention is a must, along with treatment individualized to the patient. It is essential to identify interventions to eliminate the causes of incontinence or minimize its effects and take extreme measures to protect the skin of patients at risk for or with IAD.

As a result of reading this article, clinicians should able to evaluate the types of incontinence; to define IAD and assess its prevalence, etiology, and pathophysiology; to apply the appropriate assessment, prevention, and treatment strategies for IAD; and interpret the latest research on IAD. ●

PRACTICE PEARLS

- Risk factors associated with incontinence-associated dermatitis (IAD) include urinary and fecal incontinence, poor skin condition, pain, inadequate oxygen of skin, fever, and decreased mobility.
- A variety of available tools can be helpful in assessing for perineal skin breakdown.
- Identify the cause of urinary incontinence and direct treatment interventions to modify or eliminate the cause.
- Preventive regimens for individuals at risk for IAD should include inspection, gentle cleansing, moisturizers, and skin protectants.
- Education of healthcare providers and patients is essential to effective management of IAD.

REFERENCES

1. Wishin J, Gallagher TJ, McCann E. Emerging options for the management of fecal incontinence in hospitalized patients. J Wound Ostomy Continence Nurs 2008;35(1):104-10.
2. Fonda D, Nickless R, Roth R. A prospective study of the incidence of urinary incontinence in an acute care teaching hospital and its implications on future service development. Aust Clin Rev 1988;8(30):102-7.
3. Centers for Disease Control. Urinary incontinence among hospitalized persons aged 65 years and older—United States, 1984-1987. MMWR Morb Mortal Wkly Rep 1991;40:433-6.
4. Landefeld CS, Bowers BJ, Feld AD, et al. National Institutes of Health state-of-the-conference statement: prevention of fecal and urinary incontinence in adults. Ann Intern Med 2008;148:449-58.
5. Lewis-Byers K, Thayer D. An evaluation of two incontinence skin care protocols in a long-term care setting. Ostomy Wound Manage 2002;48(12):44-51.
6. Jackson R, Vittinghoff E, Kanaya A, et al. Urinary incontinence in elderly women: findings from the Health, Aging, and Body Composition Study. Obstet Gynecol 2004;104:301-7.
7. Lifford KL, Curhan GC, Hu FB, Barbieri RL, Grodstein F. Type 2 diabetes mellitus and risk of developing urinary incontinence. J Am Geriatr Soc 2005;53(11):1851-7.
8. Norton C, Chelvanayagam S, eds. In: Bowel Continence in Nursing 2004. Oxford, UK: Beaconsfield Publishing; 2004;248-50.
9. Whitman DH, ed. In: Intra-abdominal Infections: Pathophysiology and Treatment. Hoechst: Frankfurt, Germany; 1991:20-1.
10. Ness W. Faecal incontinence: what influences care and management options? Br J Nurs 2008;17(18):1148-52.
11. NICE. Faecal incontinence: the management of faecal incontinence in adults. http:guidance.nice.org.uk/CG49/quickrefguide/pdr/English. Last accessed September 14, 2010.
12. Gray M, Bliss DZ, Doughty DB, Ermer-Seltun J, Kennedy-Evans KL, Palmer MH. Incontinence-associated dermatitis: a consensus. J Wound Ostomy Continence Nurs 2007;34:45-54.
13. Brown D, Sears M. Perineal dermatitis: a conceptual framework. Ostomy Wound Manage 1993;39(7):20-5.
14. Gray M. Incontinence-related skin damage: essential knowledge. Ostomy Wound Manage 2007;53(12):28-32.

15. Junkin J, Selekof JL. Beyond ''diaper rash'': incontinence-associated dermatitis: does it have you seeing red? Nursing 2008;38(11 Suppl):56hn1-10.
16. Bliss DZ, Savik K, Harms S, Fan Q, Wyman JF. Prevalence and correlates of perineal dermatitis in nursing home residents. Nurs Res 2006;55:243-51.
17. Bliss DZ, Zehrer C, Savik K, Smith G, Hedblom EC. An economic evaluation of four skin damage prevention regimens in nursing home residents with incontinence: economics of skin damage prevention. J Wound Ostomy Continence Nurs 2007;34(2):143-52.
18. Wound, Ostomy, and Continence Nurses Society. Guideline for prevention and management of pressure ulcers. WOCN Clinical Practice Guideline Series. Glenview, IL: WOCN Society; 2003:14.
19. Newman K, Wallace D, Wallace J. Moisture control and incontinence management. In: Krasner DL, Rodeheaver GT, Sibbald RG, eds. Chronic Wound Care: A Clinical Source Book for Healthcare Professionals. 3rd ed. Wayne, PA: HMP Communications; 2001.
20. Nair B, O'Dea I, Lim L, Thakkinstian A. Prevalence of geriatric syndromes in a tertiary hospital. Aust J Ageing 2000;19(2):81-4.
21. daSilva AP, Santos VL. Prevalence of urinary incontinence in hospitalized patients. Rev Esc Enferm USP 2005;39(1):36-45.
22. Mecocci P, von Strauss E, Cherubini A, et al. Cognitive impairment is the major risk factor for development of geriatric syndromes during hospitalization: results from the GIFA study. Dement Geriatr Cogn Disord 2005;20:260-2.
23. Sgadari A, Topinkova E, Bjrnson J, Bernabei R. Urinary incontinence in nursing home residents: a cross-national comparison. Age Ageing 1997;26(Suppl 2):49-54.
24. Bliss DZ, Johnson S, Savik K, Clabots CR, Gerding DN. Fecal incontinence in hospitalized patients who are acutely ill. Nurs Res 2000;49:101-8.
25. Schultz A, Dickey G, Skoner M. Self-report of incontinence in acute care. Urol Nurs 1997;17:23-8.
26. Nelson R, Norton N, Cautley E, Furner S. Community-based prevalence of anal incontinence. JAMA 1995;274(7):559-61.
27. Nelson R, Furner S, Jesudason V. Fecal incontinence in Wisconsin nursing homes: prevalence and associations. Dis Colon Rectum 1998;41:1226-9.
28. Ouslander JG, Palmer MH, Rovner BW, German PS. Urinary incontinence in nursing homes: incidence, remission and associated factors. J Am Geriatr Soc 1993;41: 1083-9.
29. Ouslander JG, Kane RL, Abrass IB. Urinary incontinence in elderly nursing home patients. JAMA 1982;248:1149-98.
30. Junkin J, Selekof JL, Lerner J. Prevalence of incontinence and associated skin injury in the acute care patient. J Wound Ostomy Continence Nurs 2007;34(3):260-9.
31. Gabrel CS. An Overview of Nursing Home Facilities: Data From the 1997 National Nursing Home Survey. Advance Data From Vital and Health Statistics; no. 312. Hyattsville, MD: National Center for Health Statistics; 2000.
32. Newman D, Preston A, Salazar S. Moisture control, urinary and fecal incontinence, and perineal skin management. In: Krasner D, Rodeheaver G, Sibbald RG, eds. Chronic Wound Care: A Clinical Source Book for Healthcare Professionals. 4th ed. Malvern, PA: HMP Communications; 2007:609-27.
33. Mairis E. Four senses for a full skin assessment. Observation and assessment of the skin. Prof Nurse 1992;7:376-80.
34. Berg RW. Etiology and pathophysiology of diaper dermatitis. Adv Dermatol 1988;3:75-98.
35. Beldon P. Moisture lesions: the effect of urine and faeces on the skin. Wound Essentials 2008;3:82-4, 86-7.
36. Yosipovitch G, Hu J. The importance of skin pH. Skin Aging 2003;11:88-93.
37. Rippke F, Schreiner V, Schwanitz H. The acidic milieu of the horny layer: new findings on the physiology and pathophysiology of skin pH. Am J Clin Dermatol 2002;3:261-72.
38. Van Sam V, Passet J, Maillols H, Guillot B, Guilhou J. TEWL measurement standardization: kinetic and topographic aspects. Acta Derm Venereol 1994;74:168-70.
39. Lyder CH. Perineal dermatitis in the elderly. A critical review of the literature. J Gerontol Nurs 1997;23:5-10.
40. Neander KD. Dermatological aspects of incontinence [in German]. Krankenpflege J 1990;28:509-13.
41. Mayrovitz HN, Sims N. Biophysical effects of water and synthetic urine on skin. Adv Skin Wound Care 2001;14:302-8.
42. Fiers S, Thayer D. Management of intractable incontinence: In: Doughty DV, ed. Urinary and Fecal Incontinence: Nursing Management. 2nd ed. St Louis, MO: Mosby; 2000.
43. Gray M, Ratliff C, Donovan A. Perineal skin care for the incontinent patient. Adv Skin Wound Care 2002;15:170-5.
44. Tsai TF, Maibach HI. How irritant is water? An overview. Contact Dermatitis 1999;41: 311-4.
45. Barbenel JC, Jordan MM, Nicol SM, Clark MO. Incidence of pressure sores in the greater Glasgow Health Board area. Lancet 1997;8037:548-50.
46. Cooper P, Gray D. Comparison of two skin care regimes for incontinence. Br J Nurs 2001; 10:S6, S8, S10.
47. Keller PA, Sinkovic SP, Miles SJ. Skin dryness: a major factor in reducing incontinence dermatitis. Ostomy Wound Manage 1990;30:60-4.
48. Gray M. Preventing and managing perineal dermatitis: a shared goal for wound and continence care. J Wound Ostomy Continence Nurs 2004;31(1 Suppl):S2-9.
49. Palmer MH, Baumgarten M, Langenberg P, Carson JL. Risk factors for hospitalized acquired incontinence in elderly female hip fracture patients. J Gerontol 2002;57(10): M672-7.
50. Zulkowski K, Kindsfater D. Examination of care planning needs for elderly newly admitted to an acute care setting. Ostomy Wound Manage 2000;46:32-6.
51. Faria D, Shwayder T, Krull EA. Perineal skin injury: extrinsic risk factors. Ostomy Wound Manage 1996;42(7):28-37.
52. Nix DH. Factors to consider when selecting skin cleansing products. J Wound Ostomy Continence Nurs 2000;27:260-8.
53. Adams SD, Mercer DW. Fulminant *Clostridium difficile* colitis. Curr Opin Crit Care 2007;13: 450-5.
54. National Pressure Ulcer Advisory Panel. National Pressure Ulcer Advisory Panel Support Surface Standards Initiative. http://www.npuap.org/NPUAP_S3I_TD.pdf. Last accessed September 10, 2010.
55. Richey M, Richey H, Fenski N. Age-related skin changes: development and clinical meaning. Geriatrics 1988;43(4):49-64.
56. Lekan-Rutledge D. Management of urinary incontinence: skin care, containment devices, catheters, absorptive products. In: Doughty D, ed. Urinary and Faecal Incontinence: Current Management Concepts. 3rd ed. St Louis, MO: Mosby Elsevier; 2006.
57. Sibbald RG, Campbell K, Coutts P, Queen D. Intact skin—integrity not to be lost. Ostomy Wound Manage 2003;49(6):27-41.
58. Nix DH. Validity and reliability of the Perineal Assessment Tool. Ostomy Wound Manage 2002;48(2):43-9.
59. Brown DS. Perineal dermatitis: can we measure it? Ostomy Wound Manage 1993;39(7): 28-31.
60. Kennedy K, Leighton B, Lutz J. Cost-effectiveness evaluation of a new alcohol-free, film-forming incontinence skin protectant. White paper. St Paul, MN: 3M Healthcare; 1996.
61. Borchert K, Bliss DZ, Savik K, Radosevich DM. The incontinence-associated dermatitis and its severity instrument: development and validation. J Wound Ostomy Continence Nurs 2010;37(5):527-35.
62. Urinary Incontinence Guideline Panel. Urinary Incontinence in Adults: Clinical Practice Guideline. Rockville, MD: Agency for Health Care Policy and Research; 1992. AHCPR Publication 92-0038.
63. Beeckman D, Schoonhoven L, Verhaeghe S, Heyneman A, Defloor T. Prevention and treatment of incontinence-associated dermatitis: literature review. J Adv Nurs 2009;65: 1141-54.
64. Nix D. Prevention and treatment of perineal skin breakdown due to incontinence. Ostomy Wound Manage 2006;52(4):26-8.
65. Evans J, Stephen-Haynes J. Identification of superficial pressure ulcers. J Wound Care 2007;16(2):54-6.
66. Zulkowski K, Ratliff C. Perineal dermatitis or pressure ulcer: how can you tell? Nursing 2008;36(12 Pt 1):22-3.
67. Maklebust J, Magnan M. Risk factors associated with having a pressure ulcer: a secondary data analysis. Adv Skin Wound Care 1994;7(6):25, 27-8, 31-4.
68. Nix D, Ermer-Seltun J. A review of perineal skin care protocols and skin barrier product use. Ostomy Wound Manage 2004;50(12):59-67.
69. Warshaw E, Nix D, Kula J, Markon CE. Clinical and cost effectiveness of a cleanser protectant lotion for treatment of perineal skin breakdown in low-risk patients with incontinence. Ostomy Wound Manage 2002;48:44-51.
70. Whittington K, May S. Cleansing regimens for continence care. Prof Nurse 1998;14: 167-72.
71. Hunter S, Anderson J, Hanson D, Thompson P, Langemo D, Klug M. Clinical trial of a prevention and treatment protocol for skin breakdown in two nursing homes. J Wound Ostomy Continence Nurs 2003;30:250-8.
72. Dieter L, Drolshagen C, Blum K. The development of cost-effective care for the patients with incontinence. Research poster presented at the 38th Annual Conference of the WOCN Society; June 24-8, 2006; Minneapolis, MN.

73. Bale S, Tebble N, Jones V, Price P. The benefits of implementing a new skin care protocol in nursing homes. J Tissue Viability 2004;14(2):44-50.
74. Clever K, Smith G, Browser C, Munroe K. Evaluating the efficacy of a uniquely delivered skin protectant and its effect on the formation of sacral/buttock pressure ulcers. Ostomy Wound Manage 2002;48(12):60-2, 64-7.
75. Narayanan S, Van Vleet J, Strunk B, Ross RN, Gray M. Pressure ulcer treatments in long term care facilities. J Wound Ostomy Continence Nurs 2005;32:163-70.
76. Kemp MG. Protecting the skin from moisture and associated irritants. J Gerontol Nurs 1997;20(9):8-14.
77. Brazzelli M, Shirran E, Vale L. Absorbent products for containing urinary and/or fecal incontinence in adults. J Wound Ostomy Continence Nurs 2002;29:45-54.
78. Hughes S. Do continence aids help to maintain skin integrity? J Wound Care 2002;11:235-9.
79. Spraker M. Diaper dermatitis. Pediatric Basics 1991;56:2-5, 8.
80. Nix DH. Prevention and treatment of perineal skin breakdown. In: Milne C, Corbett L, Dubuc D, eds. Wound, Ostomy, Continence Nursing Secrets. Philadelphia, PA: Hanley & Belfus, Inc; 2002:373-7.
81. Cottenden A, Bliss D, Fader M, et al. Management with continence products. In: Abrams P, Cardozo L, Khoury S, Wein A, eds. Incontinence Basics and Evaluation. Vol 1. Paris, France: Health Publications; 2005:151-253.
82. Benoit RA Jr, Watts C. The effect of a pressure ulcer prevention program and the Bowel Management System in reducing pressure ulcer prevalence in an ICU setting. J Wound Ostomy Continence Nurs 2007;34:163-75.
83. Keshava A, Renwick A, Stewart P, Pilley A. A nonsurgical means of fecal diversion: the Zassi Bowel Management System. Dis Colon Rectum 2007;50:1017-22.
84. Kim J, Shim MC, Choi BY, Ahn SH, Jang SH, Shin HJ. Clinical application of continent anal plug in bedridden patients with intractable diarrhea. Dis Colon Rectum 2001;44:1162–7.
85. Haisfield-Wolfe ME, Rund C. A nursing protocol for the management of perineal-rectal skin alterations. Clin J Oncol Nurs 2000;4(1):15-21.
86. Bliss DZ, Zehrer C, Savik K, Thayer D, Smith G. Incontinence-associated skin damage in nursing home residents: a secondary analysis of a prospective, multicenter study. Ostomy Wound Manage 2006;52(12):46-55.
87. Lyder CH, Clemes-Lowrance C, Davis A, Sullivan L, Zucker A. Structured skin care regimen to prevent perineal dermatitis in the elderly. J ET Nurs 1992;19:12-6.
88. Bliss DZ, Zehrer C, Ding L, Hedblom EC. An economic evaluation of skin damage prevention regimens among nursing home residents with incontinence: labor costs. J Wound Ostomy Continence Nurs 2005;32(Suppl 3):51.
89. Byers PH, Ryan PA, Regan MB, Shields A, Carta SG. Effects of incontinence care cleansing regimens on skin integrity. J Wound Ostomy Continence Nurs 1995;22:187-92.
90. Frantz RA, Xakellis GC Jr, Harvey PC, Lewis AR. Implementing an incontinence management protocol in long-term care. Clinical outcomes and costs. J Gerontol Nurs 2003;29(8):46-53.
91. Campbell AJ, Reinken J, McCosh L. Incontinence in the elderly: prevalence and prognosis. Age Ageing 1985;14(2):65-70.
92. Johanson JF, Lafferty J. Epidemiology of fecal incontinence: the silent affliction. Am J Gastroenterol 1996;91:33-6.
93. Thomas TM, Ruff C, Karran O, Mellows S, Meade TW. Study of the prevalence and management of patient with faecal incontinence in old people's homes. Community Med 1987;9:232-7.
94. Cuzzell JZ. Practical tips for incontinence. Home Care Provid 1997;2:286-7.

For more than 59 additional continuing education articles related to Skin and Wound Care topics, go to NursingCenter.com/CE.

CE Connection

CONTINUING MEDICAL EDUCATION INFORMATION FOR PHYSICIANS

Lippincott Continuing Medical Education Institute, Inc. is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Lippincott Continuing Medical Education Institute, Inc. designates this journal-based CME activity for a maximum of 1 *AMA PRA Category 1 Credit™*. Physicians should only claim credit commensurate with the extent of their participation in the activity.

PROVIDER ACCREDITATION INFORMATION FOR NURSES

Lippincott Williams & Wilkins, publisher of the *Advances in Skin & Wound Care* journal, will award 3.5 contact hours for this continuing nursing education activity.

LWW is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 3.5 contact hours. Lippincott Williams & Wilkins is also an approved provider of continuing nursing education by the District of Columbia and Florida #FBN2454.

Your certificate is valid in all states.

The ANCC's accreditation status of Lippincott Williams & Wilkins Department of Continuing Education refers only to its continuing nursing education activities and does not imply Commission on Accreditation approval or endorsement of any commercial product.

CONTINUING EDUCATION INSTRUCTIONS

- Read the article beginning on page 126.
- Take the test, recording your answers in the test answers section (Section B) of the CE enrollment form. Each question has only one correct answer.
- Complete registration information (Section A) and course evaluation (Section C).
- Mail completed test with registration fee to: Lippincott Williams & Wilkins, CE Group, 333 7th Avenue, 19th Floor, New York, NY 10001.
- Within 3 to 4 weeks after your CE enrollment form is received, you will be notified of your test results.
- If you pass, you will receive a certificate of earned contact hours and an answer key. Nurses who fail have the option of taking the test again at no additional cost. Only the first entry sent by physicians will be accepted for credit.
- A passing score for this test is 13 correct answers.
- Nurses: Need CE STAT? Visit **http://www.nursingcenter.com** for immediate results, other CE activities, and your personalized CE planner tool. No Internet access? Call 1-800-787-8985 for other rush service options.
- Questions? Contact Lippincott Williams & Wilkins: 1-800-787-8985.

Registration Deadline: March 31, 2013 (nurses); March 31, 2012 (physicians)

PAYMENT AND DISCOUNTS

- The registration fee for this test is $29.95 for nurses; $22 for physicians.
- Nurses: If you take two or more tests in any nursing journal published by LWW and send in your CE enrollment forms together by mail, you may deduct $0.95 from the price of each test. We offer special discounts for as few as six tests and institutional bulk discounts for multiple tests. Call 1-800-787-8985 for more information.