Understanding Risk Factors in Pressure Ulcer Development and Wound Healing

Educating your staff

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Pressure ulcers affect hundreds of millions of people worldwide, complicate the delivery of patient care, and contribute to patient deaths and disability. In fact, nearly 60,000 U.S. hospital patients are estimated to die from complications due to hospital-acquired pressure ulcers each year. It is estimated that 2.5 million patients are treated each year in U.S. acute-care facilities for pressure ulcers. Incidence rates vary by clinical settings, ranging from 0.4 percent to 38 percent in acute care and 2.2 percent to 23.9 percent in long-term care.

The impact of pressure ulcers on healthcare cost is tremendous. The total cost of treatment of pressure ulcers in the United States is estimated at $11 billion per year. According to CMS, 257,412 preventable pressure ulcers were reported as secondary diagnoses in hospitals in 2007. The average cost of each of those pressure ulcers is estimated to be $43,180 per hospital stay.

Based on the risk for pressure ulcers, a holistic care plan should be developed to help prevent pressure ulcers. There is no cookie-cutter skincare plan that would be right for every patient. True, there are certain components that apply to every skincare plan (cleansing, moisturizing, positioning, nutrition and so on) but the specifics will vary depending on the individual’s risk factors and circumstances. However, avoiding or taking steps to counteract known risk factors for pressure ulcers can go a long way toward preventing them.

Here are 11 contributing factors for pressure ulcers. These do not necessarily “cause” pressure ulcers, but they can contribute to a pressure ulcer developing. Some of them are beyond our control; some cannot be eliminated, but we can implement strategies to decrease them. The good news is that some of these factors are things that we definitely can control.

1. Circulation
Pressure ulcers can occur when skin breaks down because of decreased circulation. Blood supplies our tissues with oxygen and other nutrients needed to survive. When blood flow is blocked or reduced, the tissue can literally “starve” for oxygen and nutrients. The result is that the tissue dies. Poor circulation allows a pressure ulcer to develop.

2. Mechanical stress
The skin is the body’s largest organ and it can be stressed whenever we rub, scratch, or cut it, put pressure on it or create friction. For example, sliding a patient along a bed sheet can create friction on the skin. While this friction would not necessarily cause a pressure ulcer, it can weaken already-stressed skin and set the stage for further damage.

3. Temperature
Science is just beginning to appreciate the role temperature plays in the wound healing process. If a patient has a wound, it will heal best if the wound temperature is kept as close to normal (homeostasis) as possible. The wound temperature should be the same as the temperature of the tissue around it. Changing the dressing can affect wound temperature. For example, it can take up to four hours for the wound temperature to return to homeostasis after one dressing change.

4. Too wet/too dry
Healthy skin cells divide and thrive when they live in an optimal moist environment. Keeping the skin’s moisture balance just right can help prevent a pressure ulcer. Skin that is too wet (maceration) or too dry (desiccation) makes it easier for the skin to break down or for a wound to not heal.

5. Infection
Microscopic bacteria are one of the biggest enemies for any patient and they are all around us while we try to keep the skin and wounds free of bacteria. When it comes to pressure ulcers, infection can unnecessarily prolong inflammation, the first phase of wound healing.

6. Chemical stress on wounds
In an effort to clean the wound and kill bacteria, it is often recommended that chemical products be applied to the wound. Such chemicals might include povidone-iodine, hydrogen peroxide, hypochlorite (Dakin’s), alcohol, acetic acid, iodophors and Tripsin (“Balsam of Peru”). It is true that these solutions will kill bacteria, but they can also kill healthy tissue as well. The chemicals can do more harm than good, so choosing an appropriate cleanser is important. Bacteria must be reduced in the wound, but in a way that does not destroy the optimal healing environment.

7. Medications
Patients with pressure ulcers typically take
medications, and many common medications can actually slow or affect wound healing. The best known of these drugs are anticoagulants (warfarin and other so-called “blood thinners”), anti-inflammatory drugs (such as NSAIDs like ibuprofen), chemotherapeutic agents and antibiotics. The need for such drugs must be balanced against the potential risk to the patient in terms of slowing the healing of pressure ulcers. Medications that alter alertness, activity and mobility might also make a person less able to respond to pressure.

8. Disease
The better a person’s overall health, the less risk he or she has for pressure ulcers. Many diseases, such as diabetes and vascular disease, are risk factors for developing a pressure ulcer and can also make it much harder for the body to heal a pressure ulcer that might occur. This is of particular concern for patients with diabetes or any disease that suppresses the immune system.

9. Nutrition
Good nutrition is essential to healthy skin. Patients who are well nourished and continue to get good nutrition in the hospital are at less risk for pressure ulcers than patients with nutritional deficits. When evaluating nutrition, be sure that you get laboratory values. Do not depend on just asking the patient about eating habits or observing what the patient eats from his or her hospital tray.

10. Age
Advanced age is a risk factor in pressure ulcer development because older skin tends to be drier, breaks down more easily and forms new cells more slowly. This is obviously not a controllable risk factor, but one that needs to be taken into account.

11. Body build
In particular, two body types are at increased risk for pressure ulcer development. Obese patients and extremely thin patients are both at higher risk for pressure ulcers.

Obese patients are at higher risk because circulation to fatty tissue is not as good as circulation to leaner muscular tissue. Poor circulation means less oxygen, less nutrition and more risk for pressure ulcers. Very thin patients run a risk as well because there is less fatty tissue to “cushion” them. “Thin skin” over bony prominences around knees, heels and hips are high-risk zones for pressure ulcers.

Areas at Risk
Certain parts of the body are at greater risk for pressure ulcers than others. In general, the areas with the “thinnest skin” are most at risk, places where there is a bony prominence and not much “cushion” to protect it. These areas include:
• The backs of the heels;
• The back of the head (occiput);
• Knees;
• Elbows;
• Buttocks and tailbone (coccyx, sacrum);
• Hipbone when lying on side (Greater Trochanter).

Any area of the body that is covered (by a cast, boot, restraint, tubing, collar, etc.) is also vulnerable to a pressure ulcer.

The Stages
If a pressure ulcer is forming (Stage I), it will likely show up first as a darkened area of skin. On patients with light skin tones, it may show up as brown, reddish-brown, red or purple. On patients with dark skin tones, it is more likely to appear purple or black. This discolored area will have a distinct (but not necessarily regular) boundary. If you put a little manual pressure on the discolored area, it does not turn lighter (non-blanchable). This discolored area is usually surrounded by reddened, discolored or swollen skin. The skin around the discolored area may feel hardened.

In a deep tissue injury (DTI), the upper layer of skin is intact but there is an area of dark red, purple or blue-black that indicates that the tissue underneath the epidermis has been damaged. A DTI can also be present if there is a blood-filled blister on the skin’s surface. A DTI may progress to a full-thickness Stage 3 or Stage 4 pressure ulcer.

When you do see pressure ulcers, you will probably be asked to describe them. The National Pressure Ulcer Advisory Panel (NPUAP) has set up a system to help define pressure ulcers by stages.

Deep Tissue Injury (DTI) May appear in one of two ways, either a reddish, purplish, or dark area of discolored skin that is still intact and may be hard to the touch or a blood-filled blister.
Stage I Redness or discoloration (may show up as a darkened area in patients with dark skin tones) on an area of otherwise intact skin. If you press it, it does not lighten in color. The discolored area has a distinct (but possibly irregular) boundary and the area around it can be swollen, red or hardened.

Stage II Shallow but open wound with a pink wound bed or an open or ruptured pus-filled blister.

Stage III A deep open wound in which you may be able to see the subcutaneous tissue.

Stage IV Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar might be present on some parts of the wound bed. Undermining and tunneling are often present.

Unstageable Full thickness or tissue loss in which the base of the ulcer is covered by slough and/or eschar in the wound bed.

Risk Factors that Prevent Healing

The risk factors associated with developing pressure ulcers are similar to the risk factors that can prevent an existing pressure ulcer from healing properly. To understand these risk factors, we should review how the body heals itself when the skin is wounded.

Skin is the body’s largest organ. It covers and protects our entire body and regenerates itself constantly. It must be strong enough to protect us, yet flexible enough to move with us. Damage to the skin can take many forms.

There are three distinct layers of the skin:

1. Epidermis

The outer layer of skin is called the epidermis. The epidermis does not contain any blood vessels. The epidermis itself is made up of five sub-layers (sometimes called substrata). Those layers (from most to least superficial) are called:
1. Stratum corneum
2. Stratum lucidum
3. Stratum granulosum
4. Stratum spinosum
5. Stratum basale

The body builds epidermal cells from the bottom up. They start out in the basale layer. As they work their way to the top, they change a bit in composition (adding keratin) and they change shape. When they finally reach the upper layer (stratum corneum), they die and get sloughed off. This “shedding” of old skin cells is called desquamation. This process of new cell growth, cell migration and desquamation goes on throughout the lifetime.

2. Dermis

Below the epidermis is a thick layer of skin that includes blood capillaries and connective tissue called the dermis. The dermis acts like a cushion to protect the body from normal stress and strain. Nerve endings that respond to pressure and heat are found in this skin layer, which also contains hair follicles and sweat glands. The dermis is further divided into two layers:
1. Papillary region (closest to the epidermis)
2. Reticular region (contains a lot of collagen and elastic fibers)

3. Subcutaneous

The innermost layer of skin is the hypodermis, which is sometimes also called the subcutis. When a wound occurs, the body has the ability to regenerate or recreate epidermal and dermal tissue (skin at both the outer and middle layer). A cascade of biochemical events takes place in three phases:
1. Inflammatory
2. Proliferative
3. Remodeling

Risk factors for pressure ulcers are actually the same things that would impede or interfere with the body’s cascade of repair responses. In other words, the risks for getting a pressure ulcer are the same things that make it hard for a pressure ulcer, once developed, to heal.

The first phase in wound healing involves inflammation. The wound and surrounding area may change color, increase in temperature and swell as the body mobilizes to destroy bacteria in and around the wound and to try to neutralize any debris in the wound. The inflammatory stage is the body’s acute response to a wound and is typically of short duration. Anything that prolongs or interferes with the inflammatory stage (medication, infection) can interfere with healing.

The inflammatory phase sets the body up for the proliferative phase. In this phase, new blood capillaries are formed (angiogenesis), collagen is replaced and new skin cells grow and move into place (epithelization). In epithelization, specialized cells called myofibroblasts grow along the wound edges and spread out over the surface of the wound, contracting themselves to help close the wound.

In the final phase of remodeling, the body adjusts collagen in the dermis layer and cells that are no longer needed die off in a process called apoptosis.

The best way to deal with the 11 risk factors in wound development and wound healing is to provide the body’s healing cascade with the most optimal environment in which to “do its thing.”

It is estimated that 2.5 million patients are treated each year in U.S. acute-care facilities for pressure ulcers.

Here are 11 tips for creating a healing skin environment:
1. Do whatever is possible to improve the patient’s circulation.
2. Do not drag or slide the patient when moving him; reduce mechanical stress to the skin.
3. Keep the wound temperature constant and be alert to temperature changes, which can be warning signals.
4. Keep the moisture balance stable and don’t let the skin get too wet or too dry. This is good advice for all parts of the body, not just the wound area.
5. Help fight the bacterial invasion with appropriate weapons.
6. Don’t go overboard with chemical weapons, since some of them (including old standbys like povidone-iodine, hydrogen peroxide, alcohol and others) kill healthy cells as well and may do more harm than good.
7. Consider the role medications can play in slowing healing: the physician may have to weigh the risks and benefits of certain drugs, such as blood thinners and antibiotics.
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8. The patient's underlying and co-morbid conditions play a part in healing. We can't necessarily control these factors, but we should account for them in the healing process. Pain may be a factor in delayed wound healing.

9. Make sure the patient is adequately nourished by checking lab reports (don't rely on the patient's self-reports of what they are eating).

10. Older patients heal more slowly than younger ones; we can't do anything about it, but we need to adjust our expectations appropriately. Give extra effort to protect elderly patients from getting a pressure ulcer in the first place, since they are much less resilient and heal more slowly.

11. Obese patients and the very thin are most at risk for pressure ulcers. Be extra diligent with such patients to prevent pressure ulcers from developing.

The development of pressure ulcers presents a difficult challenge to the healthcare provider who must stay well versed on how to prevent pressure ulcers as well as how to effectively treat them once they do develop.

What's the Difference Between Incidence and Prevalence of Pressure Ulcers?

Two words you commonly hear and read about with reference to pressure ulcers and other medical conditions are prevalence and incidence. They are often used together. You might see the terms used interchangeably, which is incorrect.

What is Prevalence?
Prevalence is defined as a cross-sectional count of the number of cases of a medical condition at a specific point in time. Prevalence measures all cases of a condition (e.g., pressure ulcers) among those at risk for developing the condition. Measures of prevalence are made at one point in time (e.g., a specific day). This number includes all residents that have a pressure ulcer, no matter when it occurred.

What is Incidence?
Incidence is the number of new cases of pressure ulcers in a specific period of time (e.g., 3 months, 6 months or 1 year) in relation to the total number of persons in the population who are "at risk" at the beginning of the time period. This number reflects the new pressure ulcers that have occurred after admission to a facility and before discharge.

A prevalence and incidence study is a combination of two studies that, when done consecutively, provides a facility with key statistics on patients with existing pressure ulcers and on those who acquire them during their stay.

Connie Yuska, RN, MS, CORLN is vice president of Clinical Services for Medline Industries Inc. and hosts a series of free educational webinars on pressure ulcer prevention for both acute and long-term care professionals (www.medline.com/pressureulcerprevention/webinar. A graduate of the J&J/Wharton Nurse Executive Program, she is a member of the Board of the Illinois Organization of Nurse Leaders and a member of the American Organization of Nurse Executives. Most recently, she served as the Chief Nursing Officer of a large community hospital in Chicago and as a consultant for Joint Commission Resources in the Quality and Patient Safety Solutions division.

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