

Pressure ulcer risk factors: There is no higher priority than prevention

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Abstract: Pressure ulcers, or PRUs, have affected humans for ages, and addressing the overall management of pressure ulcers is now a prominent national healthcare issue. Despite current interest and advances in medicine, surgery, nursing care, and self-care education, pressure ulcers remain a major cause of morbidity and mortality. This is particularly true for persons with impaired sensation, prolonged immobility, or advanced age. Research in the area of pressure ulcers, specifically in characterization, prevention, and treatment of pressure ulcers, is important in preventing secondary complications in persons with disabilities. As the standards of acute, post traumatic, and rehabilitation care improve, the population of persons with lifelong functional impairments continues to grow. Consequently, the prevention of secondary complications has become an increasingly prominent concern.

(Key words: pressure ulcer; risk factors)

I. Introduction

Webster's New Riverside University Dictionary defines an ulcer as "an inflammatory, often suppurating lesion on the skin or an internal mucosal surface of the body, as in the duodenum, resulting in necrosis of the tissue." Dorland's Medical Dictionary describes an ulcer (Latin, *ulcus*; Greek, *heliosis*) as "a local defect or excavation on the surface of an organ or tissue which is produced by sloughing of inflammatory necrotic tissue¹."

The National Pressure Ulcer Advisory Panel (NPUAP) is an independent nonprofit organization formed in 1987 and dedicated to the prevention, management, treatment, and research of pressure ulcers. The NPUAP defines a pressure ulcer as an area of unrelieved pressure over a defined area, usually over a bony prominence, resulting in ischemia, cell death, and tissue necrosis².

Common sites of involvement

Several studies reveal varying sites of occurrence with different diagnoses. These sites of occurrence include the ischium (28%), the sacrum (17-27%), the trochanter (12-19%), and the heel (9-18%)^{3,4}. Pressure ulcers commonly develop on the occiput of geriatric and pediatric patients who spend extended amounts of time lying supine. Patients with the secondary manifestations of osteoporosis and associated thoracic kyphosis can develop pressure ulcers over the spinous processes. Elderly patients and patients with diabetes often have pressure ulcers on the heel^{3,5}.

Epidemiology

The incidence in hospitalized patients ranges from 2.7% to 29%, and the prevalence in hospitalized patients is 3.5% to 69%. Patients in critical care units have an increased risk of pressure ulcers, as evidenced by a 33% incidence and 41% prevalence. Elderly patients admitted to acute care hospitals for nonelective orthopedic procedures, such as hip replacement and treatment of long bone fractures, are at even greater risk, with a 66% incidence^{1,4}.

In the nursing home environment, the prevalence of pressure ulcers is in the range of 2.6-24%. The incidence is 25% in residents admitted from an acute care hospital. Patients with preexisting pressure ulcers demonstrate a 26% incidence of additional pressure ulcer formation over a 6-month period. The incidence in chronic care hospitals is reported to be 10.8%, whereas 33% of those admitted to a chronic care hospital have pressure ulcers. Long-term follow-up demonstrates that most ulcers healed within a year⁶.

Morbidity and mortality

Patients predisposed to pressure ulcers are at higher risk of morbidity and mortality. Infection is the most common major complication of pressure ulcers. The offending pathologic organisms in pressure ulcers can be anaerobic or aerobic. Aerobic pathogens commonly are present in all pressure ulcers, whereas anaerobes tend to be present more often in larger wounds (65% in grade III and above)⁷.

The most common organisms isolated from pressure ulcers are *Proteus mirabilis*, group D streptococci, *Escherichia coli*, *Staphylococcus* species, *Pseudomonas* species, and *Corynebacterium* organisms. Patients with bacteremia are more likely to have *Bacteroides* species in their pressure ulcers. These wounds do not need to be cultured routinely unless systemic signs of infection are present (eg, malodorous drainage, leukocytosis, fever, hypotension, increased heart rate, changes in mental status)⁸.

Clinical alertness is needed because the signs commonly associated with impending or fulminating infection are frequently absent in elderly patients or in patients who are immunocompromised. In geriatric patients with pressure ulcers, bacteremia is reported to occur at the rate of 3.5 per 10,000 hospital discharges. Because the mortality rate in this population approaches 50%, antibiotic treatment for wound infection or secondary bacteremia provides the appropriate spectrum of coverage specific to the offending organisms. Because indiscriminate use of antibiotics leads to resistant organisms and because the specific drugs of choice and antimicrobial agents change rapidly, physiatrists typically need to consult an infectious disease specialist to facilitate the management of these complex problems⁹.

Approximately 60,000 people die each year from complications of pressure ulcers. Development of pressure ulcers has been associated with a 4.5-times greater risk of death than that for persons with the risk factors but without pressure ulcers. A secondary complication, wound-related bacteremia, can increase the risk of mortality to 55%⁶.

Risk Factors

Numerous diverse etiopathologic factors interact to cause pressure ulcers. These factors can be classified as pathomechanical or pathophysiologic.

Pathomechanical Factors (Extrinsic or Primary)

Prolonged pressure

The most important factor in the development of pressure ulcers is unrelieved pressure. Pressure ulcers arise from prolonged tissue ischemia caused by pressure that exceeds the tissue capillary pressure. Prolonged pressure deprives tissues of oxygen and essential nutrients, owing to ischemia and hypoxia, which then causes the necrosis and ulceration¹⁰.

Several critical questions at the heart of pressure ulcer research still remain unanswered: How much pressure is required to produce predictable ulceration? How long must pressure be sustained to produce predictable ulceration? Which tissues are at greatest risk of ulceration?

In 1930, Landis used a microinjection method to cannulate the arteriolar limb of capillaries in human fingernail beds to study capillary blood pressure. He reported an average pressure of 32 mm Hg in the arteriolar limb, 22 mm Hg in the midcapillary bed, and 12 mm Hg on the venous side. Decades later, these findings were reproduced by utilizing laser Doppler methods in an animal model in which the mean capillary pressure, at 45 mm Hg, approximated human capillary pressure. This finding is useful for future studies of this component of the pressure ulcer cascade¹¹.

Interface pressure

Interface pressure remains an ambiguous factor in the development of pressure ulcers. Defined as "perpendicular force per unit area between the body and support surface" by the NPUAP, it is commonly believed that interface pressure is affected by the stiffness and composition of the body tissue and by the geometric shape of the body being supported. While interface pressures less than 32 mm Hg are assumed by many clinicians to be safe, pressures in excess of 32 mm Hg are thought to lead to closure of capillary beds and tissue ischemia. However, these perceptions remain to be challenged¹⁰.

Products aimed at reducing or relieving pressure have tended to use interface pressure as the standard for judging product efficacy. Further investigation of the application of the criterion standard of 32 mm Hg that resulted from Landis' work to pressure ulcer pathology is needed, particularly since the transmission of load on tissue and muscle can decrease or increase based on characteristics of the tissue at different sites. Tissue can be more or less able to withstand pressure, depending on other patient characteristics¹¹.

The area of pressure as the primary factor in the development of pressure ulcers also requires more thorough study. For example, what helps persons with able bodies to withstand extreme tissue loads in areas at risk and avoid developing pressure ulcers? Why are persons with certain types of disease processes typically free from these ulcers? A few articles have noted that persons with amyotrophic lateral sclerosis are at relatively low risk for developing pressure ulcers because of the dermal thickening and increased collagen secondary to the increased density of the collagen fibrils associated with amyotrophic lateral sclerosis¹².

One hypothesis asserts that pressure overcoming capillary closing pressure leads to ischemia and reperfusion injury, notably in the muscle, which develops the lesion and eventually ulcerates. This ischemia-reperfusion

mechanism ultimately leads to neutrophil-mediated inflammatory tissue destruction, most likely a free radical injury, that eventually causes pressure ulceration⁹.

Shear

Shear is mechanical stress directed parallel to the plane of interest. Shearing forces have been implicated as pathomechanical contributors in the development of pressure ulcers, especially those on the sacrum. Though scientific evidence is lacking, it is logical to conclude that the angular and vertical force that occurs downward when patients are in the semi-upright position in bed tends to distort the tissues and blood vessels near the sacrum, placing this region at risk for tissue breakdown. Furthermore, in a research project focused on assessing the pressure-reducing effects of operating-table mattresses, Defloor et al concluded that elevation of the top end of the operating table at 30° might cause high pressure and shearing force on the sacrum during head and neck surgery. The synergy of shear and pressure leads to the formation of pressure ulcers¹³.

Friction

Friction is the force of 2 surfaces moving across one another. Friction and the increased drag coefficient that occurs when moving patients across bedsheets and other support surfaces can cause microscopic or macroscopic tissue trauma. Moisture, maceration, and tissue breakdown increase the surface tension of the skin and the support surface. Moisture from sensible fluid loss or incontinence leads to maceration of tissues, which in turn makes the tissues more susceptible to pressure, shear, and friction damage¹⁴.

Immobility

Immobility is a major extrinsic factor associated with the risk and formation of pressure ulcers. Immobility in bed tends to cause pressure ulcers on the occiput, sacrum, heels, malleoli, and trochanteric regions, whereas patients who use wheelchairs for mobility tend to develop pressure ulcers over the ischial tuberosity¹³.

In patients who are paralyzed or who are neurologically compromised or who underwent prolonged periods of sedation or anesthesia, the afferent nerves are unable to engage the sensorimotor feedback system. As a result, early warnings of prolonged ischemia, such as discomfort, do not produce the normal adjustments in body position that intermittently relieve pressure on areas at risk. Such adjustments occur instinctively in persons who are neurologically intact, even during sleep. While asleep, healthy subjects show some movement and change of position every 15 minutes. Making fewer than 20 movements per night increases the risk of developing a pressure ulcer¹².

In surgical patients, the duration of the immobility is even longer than the duration of the surgery because patients are already immobile during the preoperative period and remain so until arrival in the recovery room. Research has shown that the chance of developing pressure ulcers at least doubles in operations lasting longer than 4 hours¹⁵.

Studies using diverse measurement techniques have documented pressures at specific anatomic sites that are at risk for pressure ulcers. In 1991, Schubert et al utilized a laser Doppler technique to measure local blood flow in geriatric patients at risk for pressure ulcers. Their study demonstrated that geriatric patients have a delayed hyperemic response to increased regional blood flow as a result of pressure applied to areas at risk. Tissue at risk and ulcerous tissue were slower to exhibit increased blood flow as a result of a controlled local hyperthermic stimulation¹².

Pathophysiologic Factors (Intrinsic or Secondary)

Pathophysiologic factors underlying pressure ulcers include fever, anemia, infection, ischemia, hypoxemia, hypotension, malnutrition, SCI, neurologic disease, decreased lean body mass, and increased metabolic demands. Nutrition and anemia are important factors in the healing and prevention of pressure ulcers⁵. Individuals with pressure ulcers should have adequate nutrition, especially good protein intake and stores. Decreased lean body mass is a particular risk factor. Clinical parameters indicating good nutrition and immunocompetence are adequate calorie intake and maintenance of ideal body weight. The indirect measurements of these parameters are total lymphocyte count, transferrin level, and total albumin and protein. Anemia is not an intrinsic risk factor; in addition, patients whose hemoglobin value is below 10 g/dL have difficulty healing¹¹.

Hypotension is an additional intrinsic risk factor for the formation of pressure ulcers. Treating the medical condition that predisposes a patient to these ulcers is imperative. If possible, the pathophysiologic factors should be controlled in conjunction with eliminating the pathomechanical factors. If any problem is treated in isolation, the multifactorial problem is not resolved¹⁵.

II. Conclusion

Pathomechanical factors (extrinsic or primary) include the following:

- Compression
- Maceration
- Immobility

- Pressure
- Friction
- Shear

Pathophysiologic factors (intrinsic or secondary) include the following:

- Fever
- Anemia
- Infection
- Ischemia
- Hypoxemia
- Malnutrition
- Spinal cord injury
- Neurologic disease
- Decreased lean body mass
- Increased metabolic demands

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