Pressure Ulcers: A Critical Review of Definitions and Classifications

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Abstract
Pressure ulcers are serious health problems. Although a vast amount of literature addresses prevention and treatment strategies, conceptual difficulties persist regarding pressure ulcer definitions, classifications, and distinction from other tissue lesions. Based on a review of terminologies as well as current state of knowledge on pathophysiology and etiology, questions as to what pressure ulcers are and what they are not are addressed. Because pressure forces seem to play a minor role in the development of superficial ulcers, the authors suggest these types of wounds no longer be termed pressure ulcers. A more general term such as decubitus ulcer may be a more appropriate way to characterize wounds that emerge as a result of compressive forces, shearing forces, and/or friction in patients dependent on skilled care. For clinical practice, a two-category classification is proposed: superficial ulcers predominantly caused by friction and deep ulcers predominantly caused by pressure. This simple classification could enhance diagnostic accuracy and reliability. Multidisciplinary communication and research is needed to develop valid and reliable definitions and classifications for pressure ulcer-like wounds.

Key Words: pressure ulcers, definitions, concepts, diagnosis, validity


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Pressure ulcers are common and their development is complex. Even though terms to describe these wounds and the systems to classify them have undergone numerous revisions, there are still conceptual difficulties. It is important to clarify the concept of pressure ulcers to ensure accurate diagnosis, communication, prevention, and treatment decisions. The purpose of this review is to examine commonly used pressure ulcer definitions and classifications and to propose a simple solution to overcome diagnostic problems and uncertainty in clinical practice.

What are Pressure Ulcers?
The term pressure ulcer became popular in the early 1970s.1,2 Previous terms for skin and tissue lesions of this type included bedsore3,4 or decubitus ulcers.5,6 Use of the newer term is reflected in the US National Library of Medicine’s controlled vocabulary used for indexing articles for MEDLINE and PubMed. Terms such as bedsore or decubitus are synonym entry terms that refer to the medical subject heading (MeSH) pressure ulcer that was not introduced until 2006. The Library of Medicine7 describes a pressure ulcer as “an ulceration caused by prolonged pressure on the skin and tissues when one stays in one position for a long period of time, such as lying in bed. The bony areas of the body are the most frequently affected sites which become ischemic under sustained and constant pressure.”

The Excerpta Medica database (EMBASE) produced by Elsevier uses terms such as pressure ulcer, bedsore, or pressure sore synonymously as key words referring to the subject heading decubitus, introduced in 1979. In EMBASE, decubitus belongs to the category skin ulcers. Similarly, in the International Statistical Classification of Diseases and Related Health Problems (ICD-10), the term pressure ulcer refers to the diagnosis decubitus ulcer (L89), which also belongs to the category diseases.
of the skin and subcutaneous tissue. Further explanations or definitions are not given.

Two more detailed definitions that have been widely adopted in clinical practice and research were provided by the European Pressure Ulcer Advisory Panel (EPUAP) and the National Pressure Ulcer Advisory Panel (NPUAP). The EPUAP\(^9\) defines a pressure ulcer as “...an area of localized damage to the skin and underlying tissue caused by pressure, shear, friction, and/or a combination of these.” According to the NPUAP,\(^2\) a pressure ulcer is “...localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear and/or friction.”

According to the presented definitions, the factor pressure seems to play a key role in pressure ulcer development; whereas, the factors shear and friction are included only in the EPUAP and NPUAP definitions.

**Pressure.** The relationship between long-enduring pressure and tissue damage is well established.\(^6,11,12\) In the sitting or lying position, tissue is compressed between the underlying surface and bony prominences, which explains why most pressure ulcers are located at these body sites. However, the process of tissue breakdown is not yet fully understood. Reviews of the literature\(^13,14\) consolidate pressure ulcer causation into four main theories:

1. **Ischemia caused by capillary occlusion.** Applied pressure causes capillary occlusion, leading to ischemia. Cellular metabolism is interrupted and metabolic waste products are accumulated; these actions change capillary permeability, leading to edema and cellular infiltration. Increasing the degree and duration of ischemia not only increases changes in membrane permeability and extravasation, but also enhances cellular necrosis and inflammatory reactions.\(^6,15\)

2. **Reperfusion injury.** During the ischemic period, affected tissue reduces metabolism in order to reduce hypoxic and ischemic damage and to preserve tissue function. When reperfusion occurs, liberated free radicals cause inflammatory responses and advance cell damage.\(^16-19\)

3. **Lymphatic function is impaired.** Pressure reduces the blood supply, which leads to hypoxia. Hypoxia damages the lymphatic vessels; subsequently, lymphatic motility is lost and lymph flow impaired. Metabolic waste products are accumulated, leading to tissue necrosis.\(^20\) Small amounts of pressure seem to increase the lymphatic drainage but when a critical point is exceeded, lymph flow is reduced.\(^21\)

4. **Mechanical deformation of tissue cells.** Pressure leads to large deformation of tissue and cells, consequently triggering volume changes and cytoskeletal reorganization that cause initial damage and tissue breakdown.\(^12,13\) Cell membrane rupture due to cell-to-cell contact also was considered to cause irreversible tissue damage.\(^20\)

Regardless of the relevance of these individual theories, it is highly probable that all four processes contribute to pressure ulcer formation.\(^14,22\) However, pressure-induced tissue damage is related not only to body sites, but also to the type of tissue involved. Muscle and/or subcutaneous fat have been found to be more sensitive to compression than skin and fascia.\(^11,14,16,25-27\)

The skin (epidermis and dermis) survives longer periods of ischemia without irreversible damage as compared to muscle tissue.\(^29\) Therefore, pressure-induced tissue injuries always start in the muscle and/or subcutaneous fat; visible skin damage occurs later.\(^11,14,16,17,19,26-28\)

**Shear.** Shear distorts and compresses tissue. Pressure is thought to have a perpendicular effect on tissue; shear exerts diagonal force. However, a firm distinction between pressure and shear is hardly possible because pressure always causes shear and shear causes compressive forces within the strained tissue.\(^12,17,29\) Comparable to pressure forces, deeper structures like muscle and subcutaneous fat are more sensitive and vulnerable than the skin to shear\(^21\) because the skin contains collagen and elastic fibers that provide tensile strength.

**Friction.** Friction inflicts parallel forces on the skin, found to be especially damaging to superficial layers.\(^23,26,30\) Additional factors (eg, moisture due to incontinence) also contribute to the development of superficial skin lesions. However, although the skin can become macerated and inflamed, neither friction nor moisture has been found to lead to deeper tissue layer damage,\(^4,17,31\) an observation recently supported by high-resolution ultrasound scans that show that while friction affects the dermis, deep tissue layers remain intact.\(^32,33\)

In summary, according to current pathogenetic knowledge, both compressive and shearing forces seem to primarily affect deeper tissue layers; whereas, friction appears to solely contribute to superficial lesions. Taking these different etiological mechanisms into account, the term pressure ulcer, as defined to date, covers a broad range of types of wounds, but not all of them are necessarily caused by pressure or shear. Therefore, the question remains: Are all pressure ulcers actually pressure ulcers?

**How are Pressure Ulcers Classified?**

Pressure ulcers are classified according to their visible clinical appearance. The first well-documented pressure classification system was proposed by Shea in 1975.\(^14\) Shea differentiated five categories: the category “Closed Pressure Sore,” indicating deep tissue injury under intact skin, and grades 1 to 4 where increasing numbers indicate more severe...
tissue damage. In subsequent years, this classification was modified several times and new classification systems with varying numbers of categories have been introduced. Most classifications demonstrate increasing numbers of grades or stages to indicate more extensive damage to deeper tissue layers. However, there are glaring distinctions among the definitions and the number of categories comprising the two most popular classifications (EPUAP and NPUAP) (see Table 1). This causes serious problems for international communication and research — issues that are being addressed by a joint NPUAP/EPUAP initiative.

The degree of reliability of these classifications is contradictory. At present, no empirical evidence recommending a specific pressure ulcer classification system for use in daily practice has been found. In general, it seems that practitioners most often disagree regarding 1) the diagnosis of non-blanchable erythema (grade 1/Stage I pressure ulcers) and 2) the differentiation between superficial pressure ulcers (grade 2/Stage II pressure ulcers) and moisture-related skin lesions. Furthermore, the validity of present pressure ulcer classifications has to be questioned, because, as stated previously, different etiologies lead to different clinical appearances. Tissue breakdown due to pressure and/or shear always starts in the muscle and/or subcutaneous fat, while superficial skin ulcers are caused by friction in combination with other factors detrimental to the skin. In other words, superficial skin lesions possibly located over bony prominences and caused by friction and/or moisture must not be classified as pressure ulcers, because pressure played no role. Although this issue is the topic of frequent discussion, consensus has not yet been achieved. Also, from a histological point of view, distinguishing between superficial pressure ulcers and moisture lesions is impossible. On the other hand, empirical evidence exists that superficial skin lesions (grade 2/Stage II pressure ulcers) can be caused by pressure; however, cited studies focused on the skin only. Arao et al described complete decline of epidermis and dermal papillae in a grade 2/Stage II pressure ulcer, but neither the main cause (pressure, shear, or friction) was known nor deeper tissue beneath the superficial skin lesion examined. Investigations that include all tissue layers (ie, superficial skin to subcutaneous fat, fascia, and muscle) are rare. Based on the existing literature, one conclusion appears obvious: When superficial skin lesions due to compression are visible, muscle or subcutaneous fat damage must have taken place because deeper tissue is more vulnerable to pressure forces.

Although the distinction between superficial pressure ulcers and moisture-related lesions seems to be difficult for both etiological and clinical reasons, some authors argue that a correct differentiation is important because pressure ulcers should be recorded as clinical incidents and are interpreted as an adverse event in patient care, which has regulatory consequences. This triggers further questions: First, does this mean that other skin problems such as incontinence-related dermatitis or damages due to tape removal are not incidents? Assuming this is true, how should suspected large proportions of false-positive and false-negative judgments of superficial pressure ulcers (as discussed in the systematic review and four empirical studies, mentioned previously) be handled? Regardless of the liability implications of both questions, conceptual definitions and diagnostic instruments that take the actual complexity of superficial skin lesion development into account but do not cause additional uncertainty are needed.

Extensive destruction and necrosis of deeper tissue due to long-enduring unrelieved pressure is a well-known phenomenon. A clinically relevant concern is that hours or days after that injury, skin alterations may or may not be visible. Therefore, classifying these lesions correctly hardly seems possible until the full extent of tissue damage can be seen. After all, is it really possible to distinguish between grade 1/Stage I pressure ulcers and deep tissue injuries when the skin is intact? Although the clinical relevance and course of grade 1/Stage I pressure ulcers are yet not fully understood, it can be assumed that deeper tissue layers already are involved when grade 1/Stage I pressure ulcers are visible. Furthermore, not all deep tissue injuries progress to full-thickness defects.

**How Can Conceptual Problems Be Solved?**

Pressure ulcer development is a complex phenomenon; it is apparent that superficial and deep ulcers have different etiologies. Unequivocal definitions as well as classifications of pressure ulcers should clearly characterize these kinds of lesions.

One possible solution would be to exclude grade 2/Stage II pressure ulcers from the current classifications to overcome the conceptual problems regarding superficial skin lesions. Instead, they should be labelled as friction or moisture lesions. In that case, only grade 3/Stage III and Stage IV ulcers would be real pressure ulcers caused by pressure. However, this provides only a theoretical solution because in clinical practice, pressure, shear, and friction forces usually coexist. For example, when patients are highly care dependent — ie, persons with restricted mobility and activity and moist skin — develop tissue lesions, it is difficult to determine the main etiology with certainty. According to the available EPUAP and NPUAP definitions, without knowing the exact etiology, it is impossible to decide whether the lesion is a pressure ulcer. Consequently, when clinicians are not sure whether such tissue damage is caused by pressure, the lesion should not be termed pressure ulcer. A more general term such as decubitus ulcer, as provided by the ICD-10, would be more appropriate.

The authors’ experience has shown that in clinical practice it often is difficult to establish the distinct causes when decubitus ulcer-like wounds occur. Therefore, it would be logical to exclude etiology from future definitions. Focusing on clinical signs and symptoms may be more promising in enhancing diagnostic accuracy, as long as the treatment remains independent of the etiology. The authors suggest classifying decubitus
ulcers into two broad categories: 1) superficial ulcers and 2) deep ulcers. This distinction would have advantages: it is simple and simplicity enhances diagnostic accuracy and reliability.\(^{35}\) It avoids any kind of assumptions regarding etiology, especially the difficult distinction between pressure and moisture lesions. Avoiding the terms stages or grades clarifies that both categories (superficial and deep) can develop independently from each other and that deep ulcers are not necessarily a progression from superficial ulcers. Additionally, the category unstageable, proposed by the NPUAP, becomes redundant, because the ulcer can be classified as deep. Finally, the authors recommend use of the term nonblanchable erythema (grade 1/Stage I pressure ulcers) and suspected deep tissue injuries as important clinical warning signs for subsequent tissue breakdown. Occurrence of these signs should indicate an urgent need for preventive measures; however, such skin impairments should not be classified as ulcers until tissue damage is visible.

While the proposed solution might be too simple for some circumstances, such as research, the importance of pressure ulcer classifications in clinical practice may be overestimated. Grading tissue lesions according to the available pressure ulcer classifications provides only minor guidance for treatment — general wound management principles including assessment of the presence of devitalized or necrotic tissue, inflammation or infection, moisture, characteristics of the wound edge, and measurement of the wound size are much more important for guiding care.\(^{52,53}\) Specification of the anatomical depth of the tissue damage according to currently used pressure ulcer classifications seems to be more suitable for outcome measures or endpoints in clinical trials.

**Conclusion**

The recommendations made in this article may provide a means to overcome diagnostic problems and uncertainty in
clinical practice when caring for patients with lesions and wounds that present within the context of skilled care. However, some questions require further investigation. The term *decubitus ulcer* needs more conceptual clarity — ie, what is a decubitus ulcer? What are the etiologic factors, clinical course, and prognosis for lesions in the different categories? Which differentiation is most relevant for clinical decision-making? Answers to these questions are necessary for the development and evaluation of evidence-based diagnostic instruments that facilitate choice of effective, individually targeted prevention and treatment strategies. Validity of taxonomies used in databases or in the ICD-10 could be improved; obviously, decubitus ulcers are not the only diseases of the skin. Answers are forthcoming only through multidisciplinary communication and research.

### References


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### Table 1. Description of pressure ulcer classifications continued

**Shea**

| Grade 4: Clinical presentation resembles that of a Grade 3 except that bone can be identified in the base of the ulceration, which is more extensively undermined with profuse drainage and necrosis. Anatomic limit: no limit |
| **Open pressure sores:** Ischemic necrosis in the subcutaneous fat without skin ulceration leading to the development of a bursa-like cavity filled with necrotic debris. Resembling a Grade 3 sore in extent and depth. Overlying pigmented, thickened, and fibrotic skin eventually ruptures, creating a small skin defect draining a large base. Anatomic limit: deep fascia |
| **Closed pressure sores:** Ischemic necrosis in the subcutaneous fat without skin ulceration leading to the development of a bursa-like cavity filled with necrotic debris. Resembling a Grade 3 sore in extent and depth. Overlying pigmented, thickened, and fibrotic skin eventually ruptures, creating a small skin defect draining a large base. Anatomic limit: deep fascia |

**EPUAP**

| Grade 4: Extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures with or without full-thickness skin loss |

**NPUAP**

| Stage IV: Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. Often includes undermining and tunneling. Further description: The depth of a Stage IV pressure ulcer varies by anatomical location. The bridge of the nose, ear, occiput, and malleolus do not have subcutaneous tissue and these ulcers can be shallow. Stage IV ulcers can extend into muscle and/or supporting structures (eg, fascia, tendon, or joint capsule) making osteomyelitis possible. Exposed bone/tendon is visible or directly palpable |

| Suspected deep tissue injury: Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer, or cooler as compared to adjacent tissue. Further description: Deep tissue injury may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid, exposing additional layers of tissue even with optimal treatment |

| Unstageable: Full-thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green, or brown) and/or eschar (tan, brown or black) in the wound bed. Further description: Until enough slough and/or eschar is removed to expose the base of the wound, the true depth, and therefore stage, cannot be determined. Stable (dry, adherent, intact without erythema or fluctuance) eschar on the heels serves as “the body’s natural (biological) cover” and should not be removed |