Egyptian Journal of Chemistry
http://ejchem.journals.ekb.eg/

Pressure Ulcer: An Updated Review for Healthcare Professionals

Mohammed Naif Almutairi*, Mahfoudh Saad Alghamdi, Khalid Assaf Mohammed Almutairi, Ali Ali Alotaibi, Awadh Hamid Alhafi, Muteb Marshad Alanazi, Naief Qalit Almutairi, Ahmed Nijr Owaidh Alrahimi, Yousef Mesfer Almutairi, Ali Abdullah Almntshre, Eidan Abdullah Saleh Aleidan, Alqahtani Mohammed Saeed, Majed Nasser Mohammad Maashi, Sultan Mesfer Alotaibi, Abdulrahman Saad Marzouq Albagami

Ministry of Defense, Saudi Arabia

Abstract

Background: Pressure injuries are preventable lesions of skin and soft tissues driven by sustained pressure and shear at bony prominences; device-related variants are increasingly recognized and cluster at the sacrum, ischium, and trochanter.

Aim: To synthesize current concepts on etiology, epidemiology, mechanisms, histopathology, evaluation, staging, management, prognosis, complications, and rehabilitation for healthcare professionals.

Methods: Narrative synthesis of frameworks and findings summarized in this review, including NPIAP/ICD-11 classification, risk instruments, and outcome reports.

Results: Immobility, moisture, malnutrition, anemia, and endothelial dysfunction are principal risks; shear magnifies deep tissue failure, and as little as two hours of uninterrupted loading can initiate injury. Histology progresses from papillary dermal vascular change to full-thickness necrosis; chronic ulcers harbor extracellular-matrix bacterial aggregates, whereas osteomyelitis beneath exposed sacral bone is uncommon and typically superficial. In the United States, about three million adults are affected annually; hospital-acquired pressure injury costs may exceed \$26.8 billion, with incidences near 8.3 per 100 acutely ill patients and early onset within five days of admission. Healing at six months is >70% for stage 2, ~50% for stage 3, and ~30% for stage 4; sacral recurrence is common.

Conclusion: Standardized terminology, early risk stratification, dependable off-loading and microclimate control, prudent debridement and dressings, nutritional optimization, and multidisciplinary coordination reduce incidence, accelerate healing, and limit recurrence.

Keywords: pressure injury; decubitus ulcer; NPIAP; staging; Braden scale; prevention; debridement; negative pressure wound therapy; osteomyelitis; cytokines..

1. Introduction

Pressure injuries—historically referred to as bedsores, decubitus ulcers, or pressure ulcers—are focal lesions of the skin and underlying soft tissues that arise when sustained mechanical loading impairs tissue integrity. The precipitating forces are chiefly prolonged pressure and shear, which compromise perfusion and deform tissue planes, and these stresses characteristically act over areas where bone lies close to the surface.[1][2] Epidemiologically, the majority of lesions cluster at a limited set of anatomical sites; approximately seventy percent involve the sacrum, ischial tuberosities, or greater trochanters, reflecting both skeletal prominences and common patient postures. Nonetheless, the distribution is broader and includes the occiput, scapula, elbow, heel, lateral malleolus, shoulder, and external ear, all of which present physiologic vulnerabilities under sustained loading.[3] Beyond classical bony-pressure interfaces, iatrogenic and non-medical sources of localized force can precipitate comparable harm. Contemporary practice recognizes device-related pressure injuries linked to equipment such as oxygen masks, cervical collars, casts, or even personal electronics—illustrating that any persistent, poorly distributed contact pressure may initiate the pathogenic cascade.[4] Clinically, pressure injuries span a continuum from subtle, early tissue compromise to extensive, full-thickness destruction involving muscle, fascia, or bone. Their outward appearance reflects a complex interplay among force magnitude and duration, shear deformation, tissue tolerance, and microclimatic conditions at the skin interface. This heterogeneity necessitates precise descriptive frameworks so that multidisciplinary teams can communicate with clarity, align on severity, and stratify management strategies. Over time, multiple classification schemes have emerged in response to these needs. The most widely endorsed contemporary frameworks are those advanced by the National Pressure Injury Advisory Panel (NPIAP) and the International Statistical Classification of Diseases and Related Health Problems, Eleventh Revision (ICD-11). The NPIAP taxonomy, updated to reflect evolving insights into deep tissue involvement and device-related patterns, was disseminated in 2019, while ICD-11 codifications, finalized in 2018, established a global standard for reporting and surveillance.[4] Together, these systems facilitate consistent documentation,

*Corresponding author e-mail: (Mohammed Naif Almutairi).
Receive Date 08 August 2025, Revise Date: 28 September 2025, Accept Date: 02 October 2025
DOI: 10.21608/ejchem.2025.412132.12156

enable epidemiologic comparisons across settings, and support quality improvement efforts by anchoring clinical observations to shared definitions.

Macroeconomic and public health perspectives further underscore the importance of this condition. During the COVID-19 pandemic, pressures on health systems, altered patterns of hospitalization and intensive care utilization, and the logistical challenges of infection control coincided with a measurable rise in pressure-ulcer occurrence. The resulting escalation in direct treatment costs and indirect resource utilization amplified the national economic burden and drew renewed attention to prevention and early intervention. These trends reinforced longstanding lessons: attentive risk assessment, judicious repositioning protocols, optimization of support surfaces, and vigilant device fitment are essential pillars of harm reduction in both acute and long-term care environments. The historical roots of medical understanding in this area reach back to seminal clinical observers. In the nineteenth century, the French physician Jean-Martin Charcot documented a troubling association between necrotic lesions over the buttocks and sacrum and subsequent mortality. He termed this entity “decubitus ominous,” conveying his conclusion that, once such eschar developed, death frequently ensued.[5] Although modern supportive care and targeted prevention have transformed the prognosis for many at-risk patients, Charcot’s formulation endures as a cautionary reminder that pressure injury can signal profound systemic vulnerability and that prevention is invariably preferable to belated treatment.

Nomenclature has likewise evolved in response to expanding pathophysiologic insight. In 2016, the National Pressure Ulcer Advisory Panel (NPUAP) promulgated updated terminology, advocating the term “pressure injury” in place of “pressure ulcer.” The stated aim was to encompass the full spectrum of tissue damage—including presentations in which the skin surface remains intact but deeper tissues are compromised—so that classification and clinical discourse would better reflect the underlying biology rather than a single morphologic endpoint. Concurrently, the organization announced its own rebranding from NPUAP to the National Pressure Injury Advisory Panel (NPIAP), aligning the body’s name with the revised conceptual framework and emphasizing a broadened remit that spans prevention, identification of early tissue damage, and management of established lesions. In aggregate, contemporary understanding portrays pressure injury as a preventable, multifactorial condition rooted in mechanical loading that exceeds tissue tolerance. Pathogenesis unfolds when capillary perfusion is occluded and cells are subjected to deformation and ischemia, with reperfusion injury and inflammatory cascades further exacerbating damage upon relief of pressure. Shear forces magnify harm by distorting microvasculature and accelerating deep tissue compromise. These dynamics explain the predilection for skeletal prominences and the heightened risk observed in individuals with impaired mobility, diminished sensation, or systemic conditions that reduce tissue resilience. The same principles elucidate device-related injuries, where focal compression, rigid edges, or poorly cushioned interfaces concentrate stress on small surface areas and precipitate tissue failure.[4]

From a systems perspective, classification by NPIAP and codification in ICD-11 function as the lingua franca for clinical documentation and research.[4] Structured staging enables consistent triage, guides resource allocation, and informs outcome tracking. It also undergirds educational initiatives aimed at interprofessional teams, ensuring that nurses, therapists, physicians, and administrators employ mutually intelligible descriptors when auditing care processes or implementing prevention bundles. In practice, the adoption of harmonized terminology has tangible benefits: it reduces ambiguity in handoffs, supports accurate billing and epidemiologic reporting, and enhances the fidelity of multicenter studies assessing prevention technologies, support surfaces, repositioning schedules, or risk-assessment tools. The surge in cases observed alongside COVID-19 made these organizational imperatives even more salient. Heightened reliance on invasive devices, extended immobilization, and the clinical complexities of critical illness increased exposure to the mechanical and physiologic determinants of tissue injury. At the same time, constraints on staffing and personal protective equipment challenged routine skin assessment and repositioning protocols. Health systems that weathered these pressures most effectively tended to be those that had already embedded robust preventive infrastructures: reliable risk screening on admission, standardized offloading practices, routine device skin checks, and a culture of early escalation when tissue compromise was suspected. These experiences, while born of crisis, have catalyzed durable process improvements with benefits that extend far beyond pandemic conditions. In summary, pressure injuries represent localized tissue damage driven by sustained pressure and shear at or near bony prominences, with a characteristic distribution and increasingly recognized device-related forms.[1][2][3][4] Modern classification systems—principally those promulgated by NPIAP and encoded in ICD-11—provide the conceptual and operational scaffolding for accurate description, surveillance, and quality improvement.[4] The historical observations of Charcot capture the gravity of advanced lesions, while contemporary terminology emphasizes the broader continuum of injury that precedes overt ulceration.[5] As demonstrated during COVID-19, the clinical and economic stakes are substantial, making prevention, early detection, and standardized communication foundational to high-quality care.

Etiology

The genesis of pressure injuries is inherently multifactorial, arising from the convergence of extrinsic mechanical forces and intrinsic patient vulnerabilities that together overwhelm tissue tolerance. Externally, sustained perpendicular loading (pressure), tangential forces (shear), repetitive surface abrasion (friction), and an unfavorable microclimate characterized by excess moisture act in concert to deform skin and subcutaneous tissues and to impede perfusion, thereby initiating ischemic cascades. Internally, systemic factors—most notably malnutrition, anemia, and endothelial dysfunction, diminish cellular resilience and microvascular responsiveness, accelerating the transition from reversible injury to sustained tissue damage.[6][7] In practical terms, these influences rarely operate in isolation; rather, they potentiate one another. For example, moisture macerates the stratum corneum and reduces its coefficient of friction threshold, thereby magnifying the impact of shear and friction. Simultaneously, compromised nutritional and hematologic status narrows the margin of safety by reducing oxygen delivery and impairing reparative capacity. The cumulative burden is a biomechanical and physiologic milieu in which even routine positional loading can precipitate progressive tissue harm.[6][7]

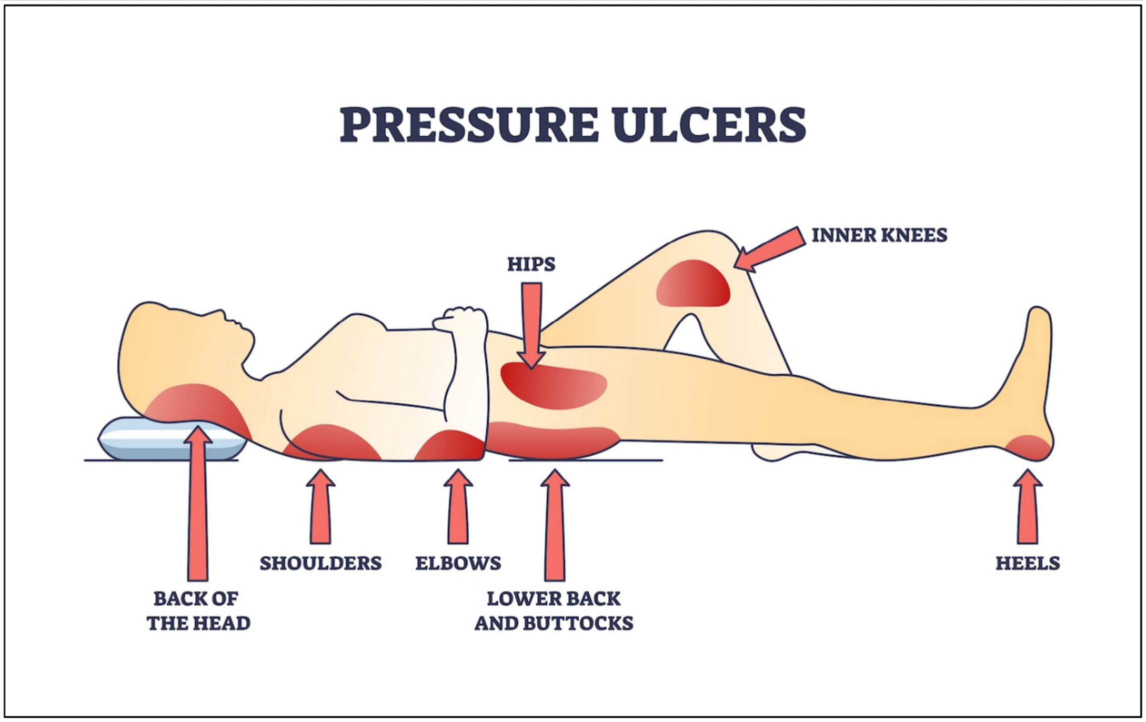

Figure 1: Pressure Ulcer Areas.

Risk stratification consistently highlights several determinants that recur across care settings. Diminished mobility limits natural periodic offloading and deprives tissues of intermittent reperfusion, rendering them susceptible to sustained capillary occlusion. Elevated skin moisture—whether from perspiration, incontinence, or exudate—weakens barrier integrity and increases frictional injury at the skin–support interface. Poor nutritional status undermines collagen synthesis, immune function, and overall tissue repair. Loss of sensory perception removes the patient's intrinsic alarm system that ordinarily triggers repositioning when discomfort signals excessive local stress. In addition to these core risks, investigators have identified the compounding effects of advanced age, cognitive impairment, and coexisting conditions that retard wound healing; these factors diminish physiologic reserve and reduce the likelihood of timely recognition and mitigation of early tissue compromise.[1] Taken together, these attributes delineate a vulnerable phenotype in which minor mechanical loads can have outsized consequences.

At the microcirculatory level, the pathophysiologic sequence begins when prolonged external compression collapses the capillary network within compressed tissues. This occlusion lowers local oxygen tension and curtails nutrient delivery, setting the stage for cellular energy failure. Over time, ischemic tissue accumulates metabolic byproducts and other toxic intermediates that cannot be adequately cleared. If unrelieved, this environment inexorably progresses to cellular apoptosis and necrosis, which clinically declare themselves as ulceration. Importantly, the temporal threshold for harm is shorter than often appreciated: in immobilized individuals—such as those confined to bed or undergoing operative procedures—only two hours of uninterrupted pressure can suffice to establish the substrate for a decubitus lesion.[7] This observation underscores why prevention protocols emphasize frequent repositioning, appropriate support surfaces, and vigilant intraoperative pressure management. Beyond purely mechanical occlusion, dysregulation within neural control circuits that govern local blood flow contributes to lesion formation. Normally, neurovascular reflexes orchestrate dynamic, pressure-induced vasodilation and redistribute perfusion to protect threatened tissues. When these regulatory mechanisms are impaired—whether by neuropathy, spinal cord pathology, or other disorders of autonomic function—the buffering capacity that would otherwise counter episodic pressure is blunted. As a result, tissues remain ischemic under loads that a healthy neurovascular system might accommodate or swiftly correct, thereby lowering the threshold for injury and hastening progression once damage has begun.[8] This neurovascular dimension helps explain why individuals with neurological disease or anesthesia-induced autonomic suppression appear especially susceptible under otherwise comparable conditions.

Although the above mechanisms are well established, their clinical expression is profoundly context dependent. In the intensive care unit, for example, sedation, vasoactive medications, and invasive devices create a nexus of risk: immobility increases duration of loading; hemodynamic lability narrows perfusion margins; and tubes or masks introduce rigid pressure points. In long-term care, cognitive decline and continence issues amplify moisture exposure and delay self-initiated repositioning. Across settings, the common denominator remains the imbalance between extrinsic forces and intrinsic resilience. When the cumulative mechanical load (including pressure, shear, and friction) exceeds the tissue's biologic capacity to adapt and recover—especially in the presence of malnutrition, anemia, or endothelial dysfunction—injury ensues.[6][7] To translate these principles into operational risk assessment, clinicians pay particular attention to patients with prominent mobility limitations, altered sensation, and compromised nutritional or vascular status. The importance of mobility merits emphasis: spontaneous micro-movements and posture shifts ordinarily disperse focal loads, restore capillary patency, and prevent the sustained deformation that damages cytoskeletal and membrane structures. When such movements are absent or markedly reduced, as in paralysis, deep sedation, or profound deconditioning, the interval to harm shrinks dramatically. Moisture management is similarly pivotal. Excessive humidity and liquid exposure soften keratin, heighten frictional coefficients, and increase susceptibility to shear-induced separation within the dermis–epidermis junction, creating an entry point for deeper structural failure. Nutritional deficits, particularly protein–energy malnutrition and micronutrient insufficiencies, undermine fibroblast activity and angiogenesis, slowing recovery from even subclinical insults. Superimposed anemia further reduces oxygen-carrying capacity, compounding ischemic stress at pressure sites.[6][7]

The two-hour window cited for the establishment of a pathological foundation in bedridden or surgical patients is not a rigid boundary but rather a clinically meaningful benchmark that integrates the combined effects of load magnitude, tissue tolerance, and systemic reserve.[7] In the intraoperative setting, prolonged anesthesia attenuates protective reflexes and abolishes pain-mediated repositioning; concurrently, controlled hypotension or intraoperative blood loss may curtail perfusion, shrinking the margin before ischemia becomes injurious. Postoperatively, residual sedation and analgesia can prolong immobility and dampen sensory feedback, perpetuating risk. These dynamics argue for structured intraoperative padding, scheduled repositioning, and early postoperative mobilization whenever feasible. The role of neural regulatory dysfunction extends beyond classic neuropathies. Any condition that interferes with autonomic integration or local neurogenic vasodilation can undermine protective hyperemia after transient loading. In diabetes mellitus with peripheral neuropathy, for instance, afferent deficits and small-fiber dysfunction impede both detection of noxious stimuli and the microvascular adjustments needed to sustain perfusion under pressure. Analogous challenges arise in spinal cord injuries, multiple sclerosis, or degenerative neurologic conditions, where impaired motor control and sensory loss conspire with altered vascular reactivity to elevate risk. Even transient states—such as deep anesthesia—can reproduce aspects of this vulnerability by silencing feedback loops that would otherwise prompt pressure relief.[8]

Patients particularly predisposed to developing decubitus ulcers commonly present with one or more of the following conditions or circumstances, which often cluster and reinforce each other:

- Neurologic disease
- Cardiovascular disease
- Prolonged anesthesia
- Dehydration
- Malnutrition
- Hypotension
- Surgical patients

In aggregate, the etiological landscape of pressure injuries is best understood as a dynamic interplay in which external mechanical stresses and microclimatic conditions intersect with intrinsic physiologic and neurologic reserves. Where that intersection tilts toward sustained deformation and inadequate perfusion—especially in the setting of malnutrition, anemia, or endothelial dysfunction—tissue breakdown becomes not only possible but probable.[6][7][1][7][8] Accordingly, effective prevention hinges on simultaneously reducing extrinsic loads (through repositioning, support surfaces, and moisture control) and bolstering intrinsic resilience (through nutritional optimization, hemodynamic stability, and management of neurologic or vascular comorbidities).

Table 1. Risk factors, mechanistic links, bedside indicators, and immediate preventive actions

Domain / Risk factor	Mechanistic link to injury	Bedside indicators	Primary preventive actions
Immobility / reduced activity	Sustained capillary occlusion; prolonged deformation	Limited turns, sedation, restraints	Turning schedules; micro-turns; appropriate support surfaces
Shear & friction (transfers, slides)	Distorts microvasculature; deep tissue failure	Sliding in bed/chair; shear marks	Draw-sheets, friction-reducing devices; transfer training
Moisture (incontinence, sweat, exudate)	Maceration; barrier failure; ↑ friction	Moist perineum, soggy linens	Timed toileting; barrier creams; wicking dressings; microclimate control
Malnutrition / protein-energy deficit	Impaired collagen synthesis, immunity	Weight loss, low prealbumin	Dietitian referral; protein/energy supplements
Anemia	Reduced oxygen delivery	Low Hb; pallor; fatigue	Treat causes; optimize Hb where appropriate
Endothelial/vascular disease	Poor autoregulation; ischemia	PVD, diabetes	Off-loading vigilance; perfusion assessment
Sensory loss / neuropathy	Absent pain-mediated repositioning	Diminished protective sensation	Timed turns; caregiver checks; cushions
Devices (masks, collars, casts)	Focal compression; edge effects	Imprints, non-blanchable erythema	Fit checks; padding; rotation/relief of device pressure
Cognitive impairment	Missed cues; poor adherence	Delirium, dementia	Staff/caregiver prompts; simplified protocols

Epidemiology

Pressure ulcers constitute a pervasive global health challenge, affecting large numbers of patients across care settings each year and imposing substantial clinical and systems-level consequences.[7] In the United States alone, contemporary estimates indicate that as many as 3 million adults experience a pressure injury annually, underscoring the magnitude of this problem within a single high-resource healthcare market.[3] The attendant economic impact is likewise formidable. In 2019, Padula and Delarmente projected that hospital-acquired pressure injury (HAPI) expenditures could surpass $26.8 billion when direct treatment costs, extended hospitalization, and related resource utilization were considered.[9] In their analysis, the modeled incidence was 8.3 HAPI per 100 patients with acute illness—an estimate that aligns closely with prior observations reporting that 7.9% of individuals deemed at risk ultimately developed a HAPI.[10] Together, these figures illustrate not only the clinical prevalence of pressure injuries but also the degree to which they strain healthcare budgets, staff time, and quality metrics.

Epidemiologic patterns reflect the interplay between intrinsic vulnerability and extrinsic mechanical loading. Sacral decubitus ulcers, the prototypical manifestation, are observed most frequently among older adults, a population in which multimorbidity, frailty, and reduced physiologic reserve converge to elevate risk. Individuals who are incontinent, paralyzed, or generally debilitated demonstrate a particularly high susceptibility because moisture, immobility, and diminished muscular support amplify the intensity and duration of pressure and shear at bony prominences. By contrast, persons with intact sensory perception, preserved mobility, and normal cognition exhibit a lower incidence; their intrinsic feedback mechanisms—pain, discomfort, and proprioceptive cues—prompt frequent position changes that interrupt ischemic intervals and prevent sustained tissue deformation. Age remains a dominant demographic correlate: approximately two-thirds of pressure ulcers occur in patients older than 70 years, a statistic that reflects both biologic aging of skin and vasculature and the greater likelihood of hospitalization, immobility, and device exposure in this cohort.[7]

Temporal analyses within acute-care environments highlight the rapidity with which risk converts to overt injury once susceptible patients are admitted. Some reports indicate that 83% of hospitalized patients who develop pressure ulcers do so within the first five days of their stay, a window that coincides with the period of greatest immobilization, hemodynamic instability, and exposure to invasive monitoring or therapeutic devices.[7] This early-onset pattern has operational significance for prevention programs: admission and peri-admission intervals warrant intensified surveillance, immediate risk stratification, and the prompt deployment of off-loading surfaces, moisture management, and scheduled repositioning. It also clarifies why HAPI rates serve as sentinel indicators of nursing workload, staffing adequacy, and the reliability of institutional skin-care protocols. In sum, the epidemiology of pressure ulcers is characterized by high prevalence, concentration among elderly and functionally impaired populations, and a marked propensity for early development during hospitalization.[7][3][9][10] The consonance between modeled incidence and observational data reinforces the robustness of current estimates, while cost projections emphasize the necessity of primary prevention as a cost-containment and patient-safety strategy.[9][10] Targeted interventions in the first days of care, particularly for older adults with immobility, incontinence, or neurologic deficits, remain central to bending the curve of both incidence and economic burden.[7]

Pathophysiology

The formation of decubitus ulcers reflects a convergent pathway culminating in tissue hypoperfusion, ischemia, and ultimately necrosis, despite the fact that multiple initiating factors may be involved. Although soft tissues can accommodate transient elevations in external load, the principal driver of injury is persistent, unrelieved compression over time. Hemodynamically, pathogenic pressure first impairs venous outflow once it surpasses the venous capillary closing pressure of approximately 8 to 12 mm Hg, and then further compromises arterial inflow when it exceeds the arterial capillary pressure of roughly 32 mm Hg. When these thresholds are sustained, local blood delivery is curtailed, cellular metabolism shifts toward energy failure, and a cascade of structural and inflammatory degeneration ensues, progressing to ulceration and tissue death.[11] In clinical settings, such deleterious pressure may arise from contact with rigid or minimally compliant interfaces, including firm mattresses, hospital bed rails, or any hard surface that concentrates load over small areas, particularly at bony prominences.[11] The microenvironment and systemic inflammatory state modulate this mechanobiologic process. Moisture alters the skin's barrier function and mechanical properties, promoting maceration and weakening the epidermal-dermal junction; in this context, even modest stress can precipitate breakdown or deepen existing lesions. Friction—generated when skin repetitively rubs against bedding, clothing, or other surfaces—abrades the superficial layers, producing micro-injuries that increase susceptibility to deeper ischemic damage. Over time, these local perturbations interact with the hemodynamic effects of sustained pressure to accelerate progression from reversible compromise to overt necrosis.[11]

Recent observations during the COVID-19 era highlight an additional, disease-specific dimension to pathophysiology. The European Pressure Ulcer Advisory Panel (EPUAP) has underscored the role of systemic inflammation in intensifying tissue vulnerability among patients with COVID-19. Elevated levels of pro-inflammatory cytokines—particularly interleukin-6 and tumor necrosis factor-alpha—appear to exacerbate and perpetuate inflammatory signaling following cellular injury. This cytokine milieu can amplify local tissue damage and impede resolution, thereby facilitating the onset and maintenance of pressure ulcers in affected individuals.[11] In sum, chronic external loading initiates perfusion failure, while microclimatic factors such as moisture and friction degrade cutaneous defenses, and systemic inflammation—exemplified in COVID-19—further entrenches the inflammatory state, together driving a final common pathway of ischemia and necrosis.[11]

Histopathology

Histopathological examination of pressure injuries delineates a progressive, dynamic continuum that corresponds closely to the clinical evolution of these lesions. The earliest alterations manifest as blanchable erythema, advance to non-blanchable erythema, and then progress through decubitus dermatitis to overt decubitus ulceration, culminating in black scab or gangrene when tissue loss becomes full thickness. Morphologically, the initial events are centered in the superficial microvasculature: the first detectable change occurs within the vessels of the papillary dermis, after which structural necrosis extends to cutaneous components. The terminal appearance—scab or gangrene—represents a full-thickness defect arising either from persistent ischemia and anoxemia or from abrupt vascular compromise, such as sudden occlusion of larger vessels precipitated by shear forces. This sequence underscores a final common pathway in which prolonged perfusion failure and mechanical deformation converge to produce irreversible tissue death.[12] The distinction between blanchable and non-blanchable erythema has practical histologic implications. Blanchable erythema reflects an early, potentially reversible perfusion disturbance, whereas non-blanchability indicates more advanced, fixed microvascular injury with evolving cellular demise. As perfusion deficits persist, cutaneous structures—including the epidermis, adnexal units, and dermal connective matrix—undergo progressive necrosis. This transformation from superficial vascular perturbation to architectural collapse explains the stepwise clinical staging, linking microcirculatory compromise in the papillary dermis to macroscopic ulceration and, ultimately, to the dry necrosis typified by black scab or gangrenous change when ischemia remains unrelieved or is acutely exacerbated by shear-induced vessel occlusion.[12]

Chronicity further imprints a distinct histopathological signature. Detailed analyses of long-standing pressure ulcers consistently reveal densely aggregated bacterial colonies embedded within the extracellular matrix. These microbial aggregates are characteristic of chronic lesions and are notably absent in acutely developed ulcers. Their localization within matrix compartments suggests structural protection from host defenses and contributes to the persistent inflammatory milieu that impedes orderly wound repair. The presence of such concentrated colonies helps differentiate chronic pressure injuries from newly formed ulcers on histologic grounds and highlights the role of sustained bioburden—rather than transient contamination—in perpetuating tissue breakdown and delaying re-epithelialization.[13] In practical terms, this distinction emphasizes that the histology of chronic ulcers reflects not only the sequelae of ischemia and mechanical stress but also the stabilizing influence of entrenched microbial communities within the wound microenvironment.[13] Skeletal involvement, particularly in sacral lesions where bone exposure is common, has been the subject of targeted histopathologic inquiry. Contrary to prevalent clinical assumptions, examination of exposed bone underlying sacral pressure ulcers demonstrates a relatively low incidence of true osteomyelitis. When infection of bone is identified, it tends to be focal and confined to superficial layers of the cortical plate rather than diffusely invasive. This pattern indicates that direct exposure of bone does not equate to deep osseous infection and that cortical changes, when present, are often limited in extent. Recognizing this nuance is essential for avoiding overdiagnosis and overtreatment: histologic confirmation remains the reference standard for differentiating superficial cortical involvement from bona fide medullary osteomyelitis in the setting of sacral pressure injuries.[14] These findings also align with the broader pathophysiologic narrative, wherein cutaneous and subcutaneous ischemia predominates, while deeper structures exhibit variable and frequently circumscribed participation.[14]

Taken together, the histopathology of pressure injuries can be conceptualized as a staged cascade initiated by superficial microvascular dysfunction and culminating, under sustained ischemic and mechanical stress, in full-thickness necrosis. The early papillary dermal vascular changes provide the substrate for progressive tissue loss; chronic wounds accrue entrenched bacterial aggregates within the extracellular matrix that perpetuate inflammation and hinder healing; and even where bone is exposed, osteomyelitic involvement is often absent or limited to shallow cortical foci. This integrative perspective connects microscopic events to clinical appearance and trajectory, reinforcing the centrality of timely off-loading and perfusion restoration to interrupt the march from reversible erythema to ulcer and gangrene, and informing judicious interpretation of microbial and osseous findings in chronic sacral lesions.[12][13][14]

History and Physical

In individuals with intact sensation, preserved mobility, and normal cognition, sustained external loading provokes discomfort or pain that naturally prompts a postural adjustment. This intrinsic feedback loop interrupts ischemic intervals and protects tissues from sustained deformation. By contrast, most patients who present with pressure injuries lack one or more of these protective attributes. Frequently, lesions are first recognized by a caregiver or bedside attendant rather than the patient, as sensory loss at the affected site can blunt awareness of early injury. Practical clues include staining of clothing or bed linens with serosanguinous fluid or frank purulence. Clinical appearance varies by anatomic location because skin thickness, subcutaneous padding, and muscle bulk modulate how tissues transmit and withstand force. The sacrum, ischial tuberosities, and greater trochanters are most commonly involved; other high-risk areas include the occiput, scapula, elbows, heels, lateral malleoli, shoulders, and ears.[3] Importantly, deep tissues—especially muscle—may become ischemic and necrotic before visible cutaneous breakdown emerges, creating a deceptive "tip-of-the-iceberg" phenomenon in which superficial changes underestimate the true depth and extent of damage. A meticulous history and holistic assessment are therefore indispensable. Any disorder of the nervous system or cardiovascular apparatus heightens predicted risk and often slows subsequent healing. Equally crucial are nutritional and hydration status, cognitive function, and baseline and current mobility; each influences tissue tolerance, the capacity to redistribute load, and the likelihood that patients will detect and respond to nociceptive cues.[15] The clinician should also review continence, pain, medications (e.g., sedatives, vasopressors, anticholinergics), and the presence and fit of medical devices, since masks, collars, tubing, and casts can generate focal, device-related pressure.

The initial evaluation should capture a clear timeline and context for the lesion's emergence and evolution. Core historical elements include:

- **Duration of immobility or bed confinement.** Specify typical positions, turning schedules, and use of support surfaces.
- **Total length of hospitalization to date.** Early hospital days often coincide with peak risk and may anchor the onset window.
- **Precipitating medical causes of immobility** (e.g., paraplegia, quadriplegia, stroke, major trauma leading to prolonged recumbency).
- **Natural history of the wound.** Identify the first site involved, the sequence of spread if any, changes in dimensions, morphology, exudate, odor, and pain profile over time.
- **Comorbid systemic disease.** Conditions such as diabetes mellitus, peripheral arterial disease, and malignancy commonly impede or delay tissue repair and must be documented.
- **Sensory awareness.** Determine whether the patient can localize the lesion or associated pain, or is unaware due to neuropathy, spinal cord injury, or altered mental status.

- **Evidence of discharge or malodor.** Note character (serous, purulent, sanguinous), volume, and odor, as these features inform staging, infection risk, and dressing selection.

The physical examination begins with a general survey: level of alertness, comfort, body habitus, hydration, and hemodynamic stability. Inspect all typical pressure points systematically, including those obscured by devices or bedding. For each lesion, document anatomic location, dimensions (length × width × depth), and the presence of undermining or tunneling. Describe wound bed composition (granulation, slough, eschar), margin configuration, and the condition of periwound skin (erythema—blanchable vs non-blanchable—maceration, induration, warmth, fluctuance). Assess quantity and quality of exudate and any odor after cleansing, recognizing that malodor may reflect bioburden but can also arise from necrotic tissue independent of invasive infection. Evaluate pain carefully; discordance between advanced tissue injury and minimal pain should prompt consideration of sensory impairment. Palpate bony prominences to appraise stability and detect crepitus or fluctuance suggestive of deeper involvement. Where feasible, perform a focused neurologic assessment of protective sensation and motor function, and screen distal pulses and capillary refill to gauge perfusion.

Staging should align with accepted frameworks and be complemented by clinical judgment about deeper, occult injury when surface findings are minimal yet risk factors are pronounced. Because muscle and fascia can fail before epidermal compromise is evident, an apparently shallow defect over a prominent bone may conceal substantial subdermal necrosis. This reality underscores the need for diligent off-loading even when the skin appears only mildly discolored. Concomitantly, examine for device-related pressure points at mask straps, tubing anchor sites, cervical collars, casts, and splints; early indentation or non-blanchable erythema in these zones warrants immediate modification of fit or padding. Historical data regarding nutrition, hydration, cognition, and mobility should be integrated with bedside findings to stratify risk and tailor early management.[15] Patients with neurologic or cardiovascular disease merit especially aggressive prevention and surveillance, given their impaired autoregulation and diminished reserve. Finally, clear documentation—photographs where permitted, standardized measurements, and consistent terminology—facilitates interdisciplinary communication and enables objective tracking of response to off-loading, moisture control, debridement, and adjunctive therapies. In sum, careful history taking and a structured, head-to-toe physical examination transform a deceptively simple skin finding into a comprehensive appraisal of systemic vulnerability, mechanical exposure, and healing potential, guiding timely intervention while averting underestimation of disease burden.[3][15]

Evaluation

A comprehensive evaluation of a patient with a pressure injury (PI) should integrate a meticulous wound assessment with a structured appraisal of the patient's overall risk profile. The aim is twofold: to characterize the current lesion precisely—so that staging, therapeutic planning, and monitoring are reliable—and to quantify future risk using validated instruments, enabling timely preventive interventions. The elements below outline a pragmatic, reproducible approach suitable for bedside practice and longitudinal follow-up. Begin with the ulcer history, documenting the presumed etiology, the duration since onset, and all previous treatments attempted. Note the patient's response to dressings, off-loading strategies, debridement, adjunctive modalities, antibiotics, and any tolerance or adherence issues. Clarify precipitating and perpetuating factors (e.g., immobility, device pressure, moisture exposure), as these guide immediate risk mitigation and inform differential diagnosis when evolution is atypical.

Proceed to staging, which requires a careful examination of the depth of the wound in keeping with accepted staging systems (detailed separately under "staging"). Correlate visible tissue layers with clinical signs of deeper involvement, recognizing that superficial appearances may underestimate subcutaneous or muscular damage. Accurate staging at baseline establishes the reference point for evaluating trajectory and treatment efficacy. Record the size of the affected area using consistent methodology (e.g., greatest length × greatest width × depth in centimeters). Photographs and measurement tools (paper rulers or single-use depth probes) improve reproducibility; always measure after wound cleansing to reduce error. Tracking absolute dimensions and percentage change over time helps distinguish true healing from transient fluctuations due to edema or slough burden. Actively search for sinus tracts, undermining, and tunneling. Gentle probing can delineate hidden extensions that increase infection risk, complicate dressing selection, and may necessitate advanced imaging or surgical consultation. Document clock-face orientation (e.g., undermining from 3 to 6 o'clock) and maximal extent in centimeters to facilitate consistent re-examination by different team members.

Evaluate the presence of drainage. Describe volume (none, scant, moderate, heavy) and character (serous, sanguineous, serosanguinous, purulent) and note any malodor after cleansing. Exudate quality and quantity influence dressing choice and may signal bioburden, retained necrotic tissue, or a change in microbial milieu that warrants culture or antimicrobial strategies. Identify and quantify necrotic tissue. Distinguish adherent eschar from slough, estimate the percentage of the wound bed occupied, and note consistency (soft, fibrous, leathery) and adherence (loose versus firmly attached). The burden and type of devitalized tissue guide the debridement plan and help anticipate bleeding risk, pain needs, and frequency of reassessment. While characterizing wound features, incorporate risk assessment instruments to reduce the incidence of new or worsening PIs and to prioritize resource allocation. Three scales are commonly cited: Waterlow, Norton, and Braden, with the Braden scale being the most widely used and best supported in contemporary literature. The Braden tool comprises six subscales, five scored from 1 to 4 and one from 1 to 3; the sum of the points yields a risk score. Lower totals reflect greater vulnerability to pressure injury and should trigger proportionate preventive measures (e.g., specialized support surfaces, turning schedules, moisture management, nutritional optimization).

Braden Scale Subscales

- **Sensory perception (1–4)** completely limited, very limited, slightly limited, or no impairment Assesses the patient's ability to detect and respond to pressure-related discomfort.
- **Mobility (1–4)** completely immobile, very limited, slightly limited, or no limitations Captures the capacity to change and control body position independently.

- **Moisture (1–4)** constantly moist, very moist, occasionally moist, rarely moist
  Reflects microclimate at the skin surface; persistent moisture elevates maceration and friction risk.
- **Nutrition (1–4)** very poor, probably inadequate, adequate, excellent
  Screens protein-energy intake and overall nutritional adequacy, which modulate tissue resilience and repair.
- **Activity (1–4)** bedfast, chairfast, walks occasionally, walks frequently
  Gauges habitual ambulation and time spent upright versus recumbent, a proxy for pressure exposure patterns.
- **Friction and Shear (1–3)** problem, potential problem, no apparent problem
  Estimates risk from sliding forces and repeated micro-abrasion during transfers or repositioning.

Interpretation of Scores

- Mild Risk = 15 to 18
- Moderate Risk = 13 to 14
- High Risk = 10 to 12
- Very High Risk = 9 or below

In practice, the Braden score should be integrated with clinical judgment and contextual factors such as acute illness severity, device use, hemodynamic status, and staffing realities. Reassess at clinically meaningful intervals—on admission/transfer, after operative procedures, with any change in medical status, and at least daily in high-risk settings—so that prevention bundles can be intensified or de-escalated responsively. Align the risk score with actionable protocols: for example, very high-risk patients may require low-air-loss or alternating-pressure surfaces, two-hourly repositioning (or more frequent micro-turns), moisture-wicking barriers, friction-minimizing transfer techniques, and early nutrition intervention. Document findings in a standardized format to support interdisciplinary communication, facilitate auditing of preventive reliability, and enable outcome tracking over time. By coupling rigorous lesion characterization with validated risk stratification—particularly via the Braden scale—clinicians can direct resources to those most likely to benefit and reduce the likelihood of PI progression or recurrence.

Treatment / Management

An overarching principle governs the care of pressure injuries: rigorous prevention is superior to any downstream therapeutic strategy. The preventive agenda has two complementary aims—first, to maintain or augment the tissue's intrinsic tolerance to mechanical load, and second, to ensure consistent off-loading of vulnerable areas. In practical terms, this translates into meticulous skin care (keeping the integument clean and dry), judicious hydration and nutrition (including supplementation for patients with limited oral intake), and the routine use of pressure-redistributing supports across the care continuum. Cushions and mattresses that disperse load away from bony prominences, structured turning schedules, and the thoughtful selection of support surfaces constitute the core of this approach. Notably, rotation every four hours on a viscoelastic foam surface yields fewer pressure injuries than two-hourly turning on a standard mattress, underscoring how material properties of the interface can be as influential as the turning interval itself. Support technologies should be individualized by risk level, ulcer stage, mobility, comfort needs, and whether microclimate control (temperature and moisture modulation at the skin–device interface) is required.

Once a pressure ulcer has developed, management should proceed systematically, prioritizing measures that stabilize the local environment and rectify contributing forces. Key early actions include active off-loading of the involved anatomical site, control of any infection with appropriate drainage, debridement of devitalized tissue, and evidence-based wound care tailored to exudate and bioburden.[16] Pressure redistribution remains central even after ulcer formation: frequent, assisted repositioning, use of immersive or alternating-pressure beds, specialized dressings that reduce shear and maintain moisture balance, and—when relevant—refitting or modifying prosthetics and orthoses to remove focal pressure mirrors the strategies used in prevention.[16] If a localized collection is suspected or confirmed, incision and drainage should be performed. Antiseptic agents such as iodine, silver sulfadiazine, hydrogen peroxide, or Dakin solution may be used temporarily when infection is a concern; however, their prolonged application can impede wound healing and should be avoided once sepsis control has been achieved. Systemic antibiotics are reserved for patients with significant cellulitis or systemic signs of infection rather than being prescribed reflexively. Importantly, even in sacral ulcers with exposed bone, contemporary evidence indicates a low incidence of osteomyelitis; when present, osseous infection tends to be focal and superficial, a nuance that should temper assumptions and limit unnecessary antibiotic exposure.[14]

Debridement is a cornerstone intervention to convert a chronic, colonized wound into one physiologically poised to heal. Mechanical debridement effectively removes necrotic tissue and biofilm that otherwise perpetuate inflammation and block granulation. A critical caveat is the stable, dry eschar without purulence or fluctuance, for which debridement is not recommended, because that eschar can function as a protective cover until perfusion and local conditions improve.[16] Debridement strategy should be aligned with the overall plan for moisture control, bacterial balance, and tissue stimulation. Dressings should then be selected according to ulcer stage, exudate level, and infection status, with the overarching goals of maintaining a moist, not macerating wound bed, protecting periwound skin, and minimizing shear at the surface. Guidance from the National Pressure Injury Advisory Panel (NPIAP) supports silicone foam dressings as a preferred default because of their favorable moisture-handling, cushioning, and atraumatic removal characteristics. In a comprehensive hospital program aimed at reducing pressure-injury incidence, Swafford and colleagues reported strong results using Allevyn Life silicone adhesive dressings for any patient with a Braden score ≤14, operationalizing a risk-linked prophylactic dressing protocol.[10] A practical, exudate-stratified dressing algorithm can streamline bedside decisions and support consistent care across teams:[16]

<u>If infection is present, first determine exudate volume and manage accordingly:</u>

- None: consider silver, honey, or foam dressings.
- Some: consider alginate, negative pressure wound therapy (NPWT), silver, gauze, or foam.

- Copious: consider alginate, NPWT, gauze, or foam.

If infection is not present, categorize by exudate volume:

- None: consider hydrogel or transparent film to preserve moisture and protect.
- Some: consider alginate, hydrocolloid, NPWT, gauze, or foam.
- Copious: consider alginate, hydrocolloid, NPWT, gauze, or foam.

These recommendations emphasize matching a dressing's absorptive capacity and occlusivity to the wound's bioburden and exudative behavior, while protecting the periwound from maceration and frictional trauma. Contamination control may require adjunctive measures. For ulcers in regions prone to urinary or fecal soiling, diversion strategies (ranging from catheters and fecal management systems to, in selected cases, diversion procedures) can reduce bacterial load and enzymatic injury, protecting both wound bed and periwound tissue. Whether to advance to operative management depends on depth, extent, and response to optimal conservative care. Foundational steps—thorough cleaning, debridement, and drainage—should precede any attempt at closure, and NPWT can serve as a preoperative bridge, promoting granulation, reducing edema, and preparing a more favorable wound bed for reconstruction.[17]

The goals of reconstructive surgery are twofold: (1) obliterate dead space to eliminate cavities where fluid and bacteria can persist, and (2) provide durable, well-vascularized coverage that tolerates pressure and shear in day-to-day activities. Ideal conditions before definitive surgery include absence of purulence, a robustly granulated wound base, and a care environment that can protect the region from soiling. Preoperative optimization should assess and address nutritional status—including prealbumin as a surrogate of protein intake—verify glycemic control in patients with diabetes and ensure the absence of acute medical instability.[16] While such optimization criteria seem most aligned with deeper lesions, the general principle stands that the wound and host must be prepared to support durable closure. (It bears noting that, in general, stage 1 and 2 injuries do not require operative measures, whereas stage 3 and 4 lesions may.[16]) Surgical strategies span a spectrum that can be tailored to patient anatomy, comorbidities, and functional goals:

- Surgical debridement. Excision of devitalized or infected tissue and bone is the first operative step when sepsis or heavy necrosis persists despite conservative care, converting the wound to a clean, vascular bed amenable to healing or reconstruction.
- Skin grafting. Although technically straightforward, split-thickness grafts demonstrate high failure rates in advanced-stage pressure injuries because they lack bulk and long-term durability over pressure-bearing prominences.
- Flap reconstruction. Pedicled muscle, myocutaneous, or fasciocutaneous flaps, as well as free flaps, provide robust, vascularized coverage and padding. Flap selection depends on anatomical site, the patient's need for ambulation or sitting tolerance, and underlying comorbidities that affect both donor and recipient sites.[18] (See Table. Flap Options for Common Anatomic Sites of Pressure Injuries for representative choices.[18])

Postoperative care is integral to long-term success: strict off-loading, pressure mapping or positioning protocols, drain management, protection from moisture and shear, and early engagement of rehabilitation to retrain safe transfers and postures help preserve flap integrity. Nutritional support and glycemic control remain essential in the convalescent period to sustain collagen synthesis and immune competence. Adjunctive modalities may be considered in selected cases. Hyperbaric oxygen therapy has supportive evidence for enhancing healing by increasing oxygen tension within hypoxic wound tissues and periwound zones, thereby facilitating fibroblast function, collagen deposition, and bacterial killing.[19] As with any adjunct, integration should be individualized, weighing logistical burden, access, and the broader multidisciplinary plan. Across all phases of care, the therapeutic framework for pressure injuries can be organized into five mutually reinforcing pillars:

1. Prevention of additional ulcers. Ongoing risk assessment (e.g., Braden scale), staff education, microclimate control, and prompt device adjustments reduce the likelihood of new lesions forming in high-risk zones.
2. Decreasing pressure on the wound. Aggressive off-loading via positioning schedules, appropriate seating/bed interfaces, and attention to transfer techniques minimizes recurrent ischemia.
3. Wound management. Thoughtful cleansing, moisture balance, debridement where indicated, bioburden control, and dressing selection create conditions conducive to granulation and re-epithelialization.
4. Surgical intervention. Reserved for complex or deep injuries, surgery aims to eradicate necrosis, eliminate dead space, and furnish resilient soft-tissue coverage when conservative measures plateau.
5. Improving nutritional status. Protein–energy adequacy and correction of micronutrient deficits bolster the host's reparative capacity and immune function, improving outcomes and durability.

Decisional pathways should be stage-aware: stage 1 and 2 injuries typically respond to conservative measures—meticulous off-loading, optimized dressings, moisture and microclimate control, and nutritional support—while stage 3 and 4 ulcers frequently require surgical consideration, particularly when undermining, persistent necrosis, or recurrent infection impedes progress.[16] That said, even advanced wounds sometimes improve substantially with disciplined nonsurgical care if off-loading is reliable and comorbidities are optimized; hence, timing of surgery should be individualized and multidisciplinary. In summary, the effective management of pressure injuries blends prevention-first thinking with methodical, evidence-informed interventions once ulceration has occurred. Prevention hinges skin hygiene, hydration/nutrition, high-quality support surfaces, and rational turning protocols tailored to risk, stage, and microclimate needs. After ulcer onset, the emphasis shifts to off-loading, infection control and drainage, judicious debridement, and dressing selection keyed to infection status and exudate level, with NPWT as a valuable bridge in complex wounds.[16][17] For patients who meet optimization criteria and whose wounds demand it, reconstructive surgery offers durable coverage, chosen from a repertoire of debridement, grafting (with caveats), and flap options matched to anatomical and functional demands.[18] Hyperbaric oxygen may serve as an adjunct to enhance oxygen delivery to ischemic tissues and catalyze healing biology in selected contexts.[19] Throughout, attention to nutrition, glycemic control, and multidisciplinary coordination—

from nursing and rehabilitation to surgery and infectious diseases—aligns daily tactics with long-term goals: preventing new ulcers, reducing pressure on existing wounds, driving progressive healing, and restoring durable function for the patient.

Staging

Multiple classification frameworks have been devised to stage pressure ulcers, reflecting the breadth of global clinical practice and the diversity of care settings in which these injuries occur. In 2020, Kottner and colleagues undertook a comprehensive review of the most frequently used systems worldwide, bringing together investigators from nine countries to compare their conceptual underpinnings and practical implications. The review's central finding was that, despite terminological and structural differences, the conceptual meaning of the major systems is broadly comparable. Consequently, clinicians are advised to adopt the classification that is most entrenched and operationally supported within their regional context, thereby maximizing consistency in documentation, communication, and quality improvement. In the United States, the most widely accepted approach is the National Pressure Injury Advisory Panel (NPIAP) system, which organizes lesions principally by ulcer depth as the staging axis and serves as the common language for interdisciplinary teams, registries, and research reporting.[4]

Within the NPIAP framework, Stage 1 denotes intact skin exhibiting nonblanchable erythema, an early warning sign of microvascular compromise that does not yet involve structural loss of the epidermis or dermis. Stage 2 represents partial-thickness skin loss affecting the epidermis and dermis, typically manifesting as a shallow open ulcer, abrasion, or intact/ruptured serum-filled blister; the hallmark is that deeper supporting tissues remain preserved. Stage 3 describes full-thickness skin loss extending into the subcutaneous tissue but not breaching the underlying fascia. At this stage, slough or eschar may be visible, exudate can be malodorous depending on bioburden and necrosis, and undermining or tunneling may be present while muscles, tendons, and bone remain uninvolved. Stage 4 indicates full-thickness tissue loss through the fascia, with substantial tissue destruction and potential exposure or involvement of muscle, bone, tendon, or joint structures, frequently accompanied by pockets, sinus tracts, and extensive undermining. Finally, lesions are deemed Unstageable when the true depth cannot be determined because slough or eschar obscures the wound bed; in such instances, staging must be deferred until sufficient debridement clarifies the extent of damage. This depth-oriented taxonomy not only structures bedside assessment but also anchors therapeutic decision-making and prognostic counseling, acknowledging that depth correlates with both complexity of care and time to closure.[4]

The NPIAP guidance also recognizes Deep Tissue Injury (DTI) as a distinct category to capture injuries that originate at the bone–muscle interface under the combined effects of prolonged pressure and shear. Clinically, DTI may present as intact or non-intact skin displaying persistent, non-blanching, deep red, maroon, or purple discoloration, sometimes accompanied by blood-filled blisters. These chromatic cues reflect a deeper pathophysiology in which muscle and fascia are compromised before overt epidermal loss occurs, creating a "tip-of-the-iceberg" scenario wherein superficial appearance underestimates underlying destruction. Importantly, DTI should not be applied to lesions best explained by vascular, traumatic, neuropathic, or dermatologic etiologies, since mislabeling can obscure root causes and misdirect interventions. Appropriate recognition of DTI encourages immediate off-loading and close monitoring to prevent rapid evolution into full-thickness defects as superficial layers eventually fail.

Table 2. NPIAP staging and first-line management priorities (with selected outcome notes).

Stage / Category	Defining features	Typical depth / structures	First-line management	Notes / outcomes
Stage 1	Intact skin, non-blanchable erythema	No tissue loss	Off-loading; microclimate control; barrier protection	Reversible with prompt measures [4]
Stage 2	Partial-thickness loss (epidermis/dermis)	Shallow ulcer/blister	Moisture-balancing dressings (e.g., silicone foam); off-loading	>70% heal by 6 months [21]
Stage 3	Full-thickness to subcutaneous tissue, not through fascia; possible slough/eschar	Subcutis; undermining/tunneling may occur	Debridement as indicated; exudate-matched dressings; NPWT for heavy exudate; off-loading	~50% heal by 6 months [21]
Stage 4	Full-thickness through fascia; possible muscle, bone, tendon, joint	Deep structures exposed	Surgical consult; debridement; NPWT; reconstructive planning; strict off-loading	~30% heal by 6 months; high recurrence [21][22]
Unstageable	Depth obscured by slough/eschar	Unknown until debrided	Do **not** debride stable dry heel eschar; otherwise debride to stage; off-loading	Stage after visualization [4][16]
Deep Tissue Injury (DTI)	Persistent deep red/maroon/purple discoloration; intact or non-intact skin	Muscle–bone interface injury from pressure + shear	Immediate off-loading; close surveillance	Exclude vascular/neuropathic/traumatic causes [4]

Prognosis:

The outlook for patients with pressure injuries is heterogeneous and hinges on anatomic location, stage at presentation, and the timeliness and adequacy of treatment. Many investigations in this domain have emphasized prevention—often reporting reductions in incidence within specific institutions—rather than healing rates after therapy is initiated, which complicates cross-study comparisons of recovery trajectories. Nevertheless, available outcome data provide clinically meaningful benchmarks. Over a six-month treatment horizon, Stage 2 injuries have been documented to heal in more than 70% of cases, Stage 3 injuries in approximately 50%, and Stage 4 injuries in about 30%.[21] A separate study that did not stratify by stage reported complete resolution in 41.7% of patients during a mean follow-up of 1.8 years, reflecting the protracted course commonly observed in complex wounds.[22] That same analysis quantified recurrence by site, noting particularly high rates for sacral ulcers (57%), with lower but notable recurrence for ischial (14%), trochanteric (7%), calcaneal (7%), and other locations (14%).[22] These site-specific patterns underscore how local biomechanics, exposure to shear during sitting or transfers, and challenges in maintaining dryness and hygiene contribute to long-term vulnerability even after apparent healing. A host of patient-level factors modulate prognosis. Advancing age, larger wound size, higher stage, poor nutritional status, and chronic comorbidities (e.g., vascular disease, diabetes, renal impairment) have all been associated with slower healing and increased complication rates.[23] Conversely, individuals with higher mean body mass index, higher mean hemoglobin, and a greater likelihood of undergoing reconstructive plastic surgery have demonstrated improved healing trajectories, possibly reflecting better substrate for tissue repair, oxygen-carrying capacity, and access to definitive coverage when indicated.[22] Although the literature is methodologically diverse and sometimes limited by small samples or retrospective designs, there is broad agreement that many patients require lifelong prevention and surveillance, even after wound closure, to mitigate recurrent breakdown and associated morbidity.[24]

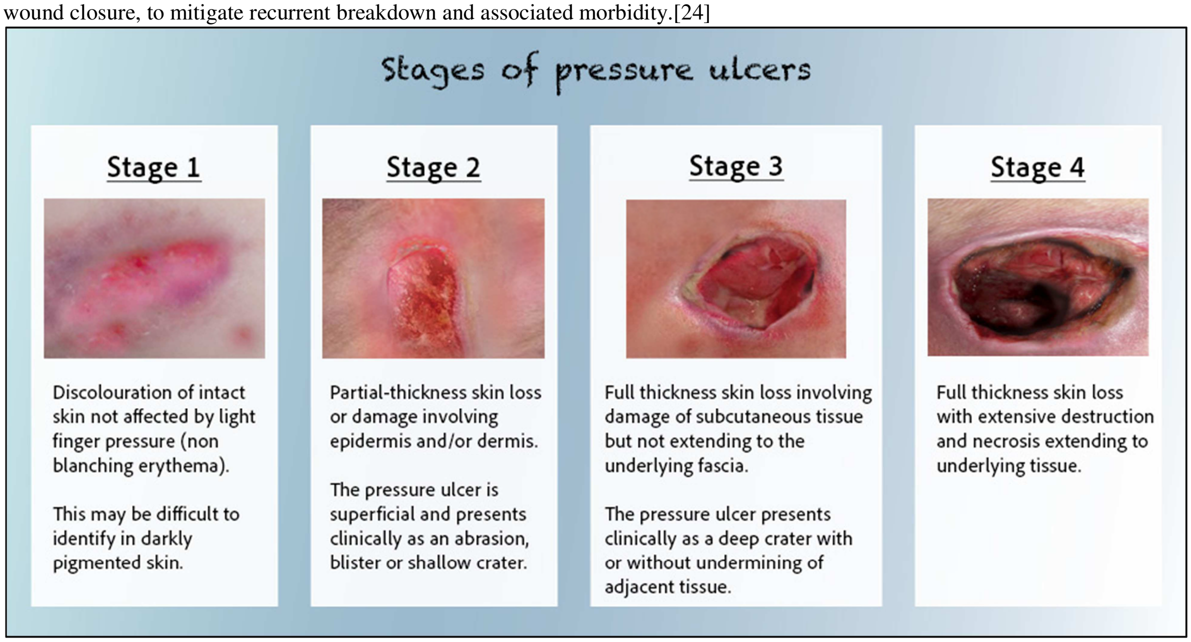

Figure 2: Stages of Pressure Ulcers.

Complications:

Decubitus ulcers predispose to a spectrum of complications, with infection being both the most common and the most consequential. Stage 3 and 4 lesions, in particular, demand rigorous management, as their depth and tissue loss increase the risk of life-threatening sequelae. Microbiological surveys consistently reveal mixed aerobic and anaerobic flora within these wounds, reflecting contamination from skin, gastrointestinal, or environmental sources and the propensity for biofilm formation that resists host defenses and topical therapies.[25] When infection spreads beyond the soft tissues, periostitis, osteomyelitis, and septic arthritis may ensue; chronic tracts can culminate in sinus formation. Systemic invasion with resultant septicemia is especially perilous in debilitated hosts whose inflammatory and hemodynamic reserves are already strained. Beyond infection, the wounds themselves exert a catabolic burden: persistent exudation and inflammatory turnover can lead to substantial fluid and protein losses, with estimates of up to 50 grams of body protein lost daily from a draining ulcer, predisposing to hypoproteinemia and malnutrition that further retard healing.[25] Chronic anemia—from ongoing bleeding and inflammatory suppression of erythropoiesis—and even secondary amyloidosis have been reported in the context of longstanding lesions.[25] Postoperative complications can arise when postoperative care is inadequate, including hematoma or seroma, wound dehiscence, abscess formation, and postoperative wound sepsis; all can jeopardize reconstructive success and necessitate reoperation. Epidemiologically, pressure injuries correlate with increased mortality risk: among older adults, presence of a pressure injury confers a 3.6-fold rise in mortality within 21 months compared with peers without such injuries. Yet the causal pathways remain debated. Thomas et al. provided evidence that pressure injuries are not a direct cause of death; rather, the shared comorbidities that predispose to both ulceration and death likely drive the association, highlighting the intertwined roles of frailty, immobility, and systemic disease.[26]

Postoperative and Rehabilitation Care:

Principles that guide preoperative risk mitigation remain critical after surgery. The postoperative plan must reinforce off-loading, meticulous skin care, incontinence control, and nutritional optimization, recognizing that even a well-executed reconstruction can fail under renewed pressure or microclimate stress.[18] In the immediate postoperative period, safe transfers from the operating table to air-fluidized or other advanced support surfaces should be performed with scrupulous attention to minimizing shear and stretch across newly inset flaps. Patients are typically maintained flat on the prescribed support surface for the first four weeks, a strategy that protects flap perfusion and mechanical integrity while early healing consolidates. Thereafter, they may transition cautiously to a semi-sitting posture under clinician supervision, with vigilant monitoring for any signs of congestion or ischemia. Sitting is generally deferred until six weeks after surgery, at which point trials begin with 10-minute sessions. Immediately following each session, the flap should be inspected for discoloration, temperature changes, or wound-edge separation that might signal compromised perfusion or undue tension. Over the subsequent two weeks, sitting duration is incrementally increased—often by 10-minute increments—toward a target of two hours, provided that serial examinations remain reassuring. Concurrently, patients are trained in pressure-relief maneuvers, such as performing a 10-second lift every 10 minutes when seated, to unload ischial and sacral regions and to promote capillary reperfusion. This graduated reintroduction of upright activity, paired with education on safe transfers, cushion use, and posture, seeks to reconcile the goals of mobility and independence with the biomechanical imperatives of flap protection.

Successful rehabilitation depends on a shared, realistic care plan endorsed by the patient, family members, and the broader social support network. The plan should delineate responsibilities for off-loading, hygiene, nutrition, and follow-up, and should anticipate obstacles such as limited caregiver availability or home environments ill-suited to specialized equipment. When robust support cannot be assured, referral to inpatient rehabilitation or a skilled nursing facility is recommended to ensure adherence to positioning protocols, wound surveillance, and nutritional regimens during the critical early phase of recovery.[18] Interdisciplinary coordination—among plastic surgery, nursing, rehabilitation medicine, nutrition, and social work—magnifies the likelihood that surgical gains will translate into durable function. In the long term, periodic reassessment of equipment (e.g., wheelchair cushions, mattresses), routine skin checks using mirrors or caregiver assistance, and reinforcement of pressure-relief behaviors are essential to prevent recurrence and to maintain the integrity of reconstructed sites. In sum, the NPIAP staging system offers a depth-based lexicon that aligns clinical appearance with management intensity and anticipated course, while the DTI category captures the unique scenario of deep tissue failure before cutaneous breakdown.[4] Prognosis is stage- and site-dependent, with healing rates declining as depth increases and recurrence particularly common over the sacrum.[21][22] Complications—from polymicrobial infection and protein-energy losses to postoperative failures and elevated mortality risk—underscore the systemic stakes of these ostensibly local lesions.[25][26] Finally, postoperative and rehabilitative strategies—anchored in off-loading, microclimate control, nutrition, and structured reintroduction of sitting—translate surgical success into sustained outcomes, provided social supports are adequate and interdisciplinary follow-up is robust.[18]

Conclusion:

Pressure injuries remain a high-impact, largely preventable source of morbidity, cost, and mortality risk across care settings. The convergent evidence summarized here emphasizes a prevention-first paradigm grounded in standardized language and reliable processes. Depth-based NPIAP staging and ICD-11 coding provide a common lexicon for assessment, communication, surveillance, and research. Early risk stratification, particularly with the Braden scale, should trigger protocolized bundles that address mobility, microclimate, moisture, nutrition, and device fit from the moment of admission, when incident lesions most often arise. Mechanistically, sustained loading with superimposed shear produces microvascular occlusion, tissue deformation, and ischemia; moisture and friction degrade barrier defenses; and systemic inflammatory states (e.g., COVID-19) can intensify local injury via cytokine amplification. Once ulceration occurs, the priorities are rigorous off-loading, control of infection (including drainage of abscesses), timely debridement of devitalized tissue, and judicious dressing selection matched to infection status and exudate volume; negative-pressure wound therapy is an effective adjunct for highly exudative or complex cavities. Antibiotics are reserved for cellulitis or systemic infection, acknowledging the relatively low, often superficial incidence of osteomyelitis beneath exposed sacral bone. Surgical reconstruction is considered for stage 3–4 wounds that fail optimized conservative care, with goals of eradicating necrosis, obliterating dead space, and furnishing durable, well-vascularized coverage; success depends on preoperative optimization and disciplined postoperative off-loading and rehabilitation. Outcomes vary by stage and site, with stage-dependent healing probabilities and high sacral recurrence, underscoring the need for lifelong secondary prevention. Multidisciplinary coordination—nursing, rehabilitation, nutrition, surgery, and infectious diseases—aligns daily tactics with durable function, while ongoing quality improvement maintains reliability under routine and crisis conditions alike.

References:

1. Chou R, Dana T, Bougatsos C, Blazina I, Starmer AJ, Reitel K, Buckley DI. Pressure ulcer risk assessment and prevention: a systematic comparative effectiveness review. Ann Intern Med. 2013 Jul 02;159(1):28-38.
2. Gaspar S, Peralta M, Marques A, Budri A, Gaspar de Matos M. Effectiveness on hospital-acquired pressure ulcers prevention: a systematic review. Int Wound J. 2019 Oct;16(5):1087-1102.
3. Mervis JS, Phillips TJ. Pressure ulcers: Pathophysiology, epidemiology, risk factors, and presentation. J Am Acad Dermatol. 2019 Oct;81(4):881-890.
4. Kottner J, Cuddigan J, Carville K, Balzer K, Berlowitz D, Law S, Litchford M, Mitchell P, Moore Z, Pittman J, Sigaudo-Roussel D, Yee CY, Haesler E. Pressure ulcer/injury classification today: An international perspective. J Tissue Viability. 2020 Aug;29(3):197-203.
5. Levine JM. Historical perspective on pressure ulcers: the decubitus ominosus of Jean-Martin Charcot. J Am Geriatr Soc. 2005 Jul;53(7):1248-51.
6. Anders J, Heinemann A, Leffmann C, Leutenegger M, Pröfener F, von Renteln-Kruse W. Decubitus ulcers: pathophysiology and primary prevention. Dtsch Arztebl Int. 2010 May;107(21):371-81; quiz 382.

7. Bansal C, Scott R, Stewart D, Cockerell CJ. Decubitus ulcers: a review of the literature. Int J Dermatol. 2005 Oct;44(10):805-10.
8. van Marum RJ, Meijer JH, Ribbe MW. The relationship between pressure ulcers and skin blood flow response after a local cold provocation. Arch Phys Med Rehabil. 2002 Jan;83(1):40-3.
9. Padula WV, Delarmente BA. The national cost of hospital-acquired pressure injuries in the United States. Int Wound J. 2019 Jun;16(3):634-640.
10. Swafford K, Culpepper R, Dunn C. Use of a Comprehensive Program to Reduce the Incidence of Hospital-Acquired Pressure Ulcers in an Intensive Care Unit. Am J Crit Care. 2016 Mar;25(2):152-5.
11. Gefen A, Ousey K. COVID-19: pressure ulcers, pain and the cytokine storm. J Wound Care. 2020 Oct 02;29(10):540-542.
12. Witkowski JA, Parish LC. Histopathology of the decubitus ulcer. J Am Acad Dermatol. 1982 Jun;6(6):1014-21.
13. Rhoads DD, Wolcott RD, Percival SL. Biofilms in wounds: management strategies. J Wound Care. 2008 Nov;17(11):502-8.
14. Wong D, Holtom P, Spellberg B. Osteomyelitis Complicating Sacral Pressure Ulcers: Whether or Not to Treat With Antibiotic Therapy. Clin Infect Dis. 2019 Jan 07;68(2):338-342.
15. Munoz N, Posthauer ME, Cereda E, Schols JMGA, Haesler E. The Role of Nutrition for Pressure Injury Prevention and Healing: The 2019 International Clinical Practice Guideline Recommendations. Adv Skin Wound Care. 2020 Mar;33(3):123-136.
16. Boyko TV, Longaker MT, Yang GP. Review of the Current Management of Pressure Ulcers. Adv Wound Care (New Rochelle). 2018 Feb 01;7(2):57-67.
17. Batra RK, Aseeja V. VAC Therapy in Large Infected Sacral Pressure Ulcer Grade IV-Can Be an Alternative to Flap Reconstruction? Indian J Surg. 2014 Apr;76(2):162-4.
18. Ricci JA, Bayer LR, Orgill DP. Evidence-Based Medicine: The Evaluation and Treatment of Pressure Injuries. Plast Reconstr Surg. 2017 Jan;139(1):275e-286e.
19. Kranke P, Bennett MH, Martyn-St James M, Schnabel A, Debus SE, Weibel S. Hyperbaric oxygen therapy for chronic wounds. Cochrane Database Syst Rev. 2015 Jun 24;2015(6):CD004123.
20. Okhovati S, Esmaeili M, Shariat E. Effect of Intensive Care Unit Nurses' Empowerment Program on Ability in Visual Differential Diagnosis of Pressure Ulcer Classification. Crit Care Nurs Q. 2019 Jan/Mar;42(1):89-95.
21. Bluestein D, Javaheri A. Pressure ulcers: prevention, evaluation, and management. Am Fam Physician. 2008 Nov 15;78(10):1186-94.
22. Walter GP, Seidel W, Giustina RD, Bins-Ely J, Maurici R, Narciso-Schiavon JL. PROGNOSTIC FACTORS IN PATIENTS WITH PRESSURE SORES IN A UNIVERSITY HOSPITAL IN SOUTHERN BRAZIL. Acta Ortop Bras. 2017 Nov-Dec;25(6):243-247.
23. Takahashi PY. Pressure ulcers and prognosis: candid conversations about healing and death. Geriatrics. 2008 Nov;63(11):6-9.
24. Qaseem A, Humphrey LL, Forciea MA, Starkey M, Denberg TD., Clinical Guidelines Committee of the American College of Physicians. Treatment of pressure ulcers: a clinical practice guideline from the American College of Physicians. Ann Intern Med. 2015 Mar 03;162(5):370-9.
25. Teasell R, Dittmer DK. Complications of immobilization and bed rest. Part 2: Other complications. Can Fam Physician. 1993 Jun;39:1440-2, 1445-6.
26. Thomas DR, Goode PS, Tarquine PH, Allman RM. Hospital-acquired pressure ulcers and risk of death. J Am Geriatr Soc. 1996 Dec;44(12):1435-40.