Review Article

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Pressure Ulcer: Review Article

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ABSTRACT

This article look into the etiopathogenesis, assessment, prevention and management of pressure ulcer. It also covers the role of pressure release devices and newer treatment methods in healing process of these wounds. Pressure ulcers occur from friction and shear and are progressive. It is usually found in bedridden and immobile people. Spinal cord injury patients have loss of sensation which adds on the problem. Pressure ulcers are preventable usually and based on severity management changes.

Keywords: Pressure Ulcer; Reconstruction; Hyperbaric Oxygen Therapy; NPWT.

INTRODUCTION

Pressure ulcer develop when there is prolonged pressure on a specific area of the skin, typically over bony prominences, such as the hips, heels, sacrum, and elbows. These sores can be particularly problematic for individuals with limited mobility or those who spend muchtime in a bed or wheel chair. According to severity, pressure sores are categorized from Stage I (mild) to Stage IV (severe). Reconstruction becomes necessary in more advanced stages where there is significant tissue damage, including muscle and bone involvement. The goals of pressure ulcer reconstruction include promoting healing, preventing infection, and restoring function and appearance. Pressure sore reconstruction refers to the surgical or medical procedures aimed at repairing and restoring tissues damaged by pressure sores, also known as pressure ulcers or bedsores. The common approaches to pressure sore reconstruction are debridement, flap cover, skin grafting, Negative Pressure Wound Therapy (NPWT) and rehabilitation.

Pressure ulcers are a type of injury that breaks down the skin and underlying tissue when a constant pressure is applied for a certain period resulting in tissue ischemia, loss of nutrition and oxygen supply to the tissues, and finally tissue necrosis. "Pressure ulcers" is a term used widely in many countries and has been accepted as a Europe wide term by the European Pressure Ulcer Advisory Panel (EPUAP). They are also known as 'bedsores', 'decubitus ulcers'. The word 'decubitus' derived from the Latin 'decumbo' or 'decumbere', meaning 'to lie down' or 'recline',since the ulcer occur commonly over areas of bony prominences

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in recumbent position, e.g., the sacrum, trochanter, heel, and occiput. The term 'pressure ulcer' describes these ulcers better with pressure as an important etiologic factor.

The site of pressure ulcer depends upon posture. Common sites of pressure ulcer in the supine position are the occiput, scapula, olecranon, sacrum, and heel;. In the lateral position are the ear, acromion process, greater trochanter, lateral condyle of femur, and lateral malleolus and in prone position are zygoma, acromion process, female breasts, pubis, patella, metatarsal over distal foot dorsum, and toes. In the sitting position sites are the shoulder blades, lower back, sacrum, ischial tuberosity, and heel.

Etiopathogenesis

Normally intracapillary pressure at the arterial end is 30–40 mmHg.[1] Microcirculatory occlusion occurs beyond this pressure and this, in turn, initiates a downward spiral toward ischaemia, tissue death and ulceration.[2,3] Compression of soft tissue occurs and shearing of tissue occur between be or chair an tissue, when the person is sitting or lying, or because something is pressing into the body, such as a shoe, a prosthesis, a surgical appliance or clothing elastic. Blood vessels within the distorted tissue are compressed, angulated or stretched out of their usual shape and blood is unable to pass through them.[4] This causes tissue

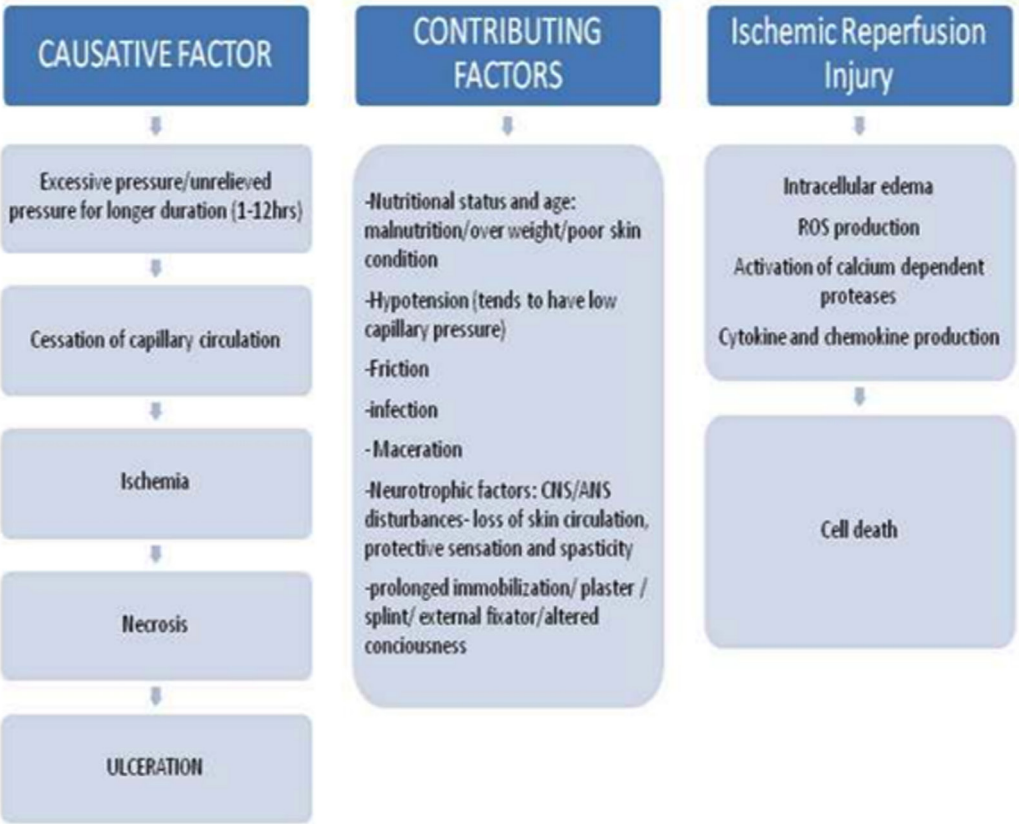

Fig. 1: Etiopathology of Pressure Sore

ischaemia. It also obstruct lymphatic flow, leading to the accumulation of metabolic waste products, proteins and enzymes in the affected tissue. (Fig 1).

1. *Pressure*

When constant pressure is applied, soft tissues change themselves to accommodate the external shape. This is known as tissue creep.[5] It decreases the external pressures and also exaggerate internal changes of soft tissues that again reduce the vascular supply of already compromised areas due to vascular kinking. If ischemia persists for 1-2 hours, necrosis takes place and pressure ulcers occurs.[6] Due to prolonged and constant pressure, the chances of skin atrophy with thinning of this protective barrier, make the skin more susceptible to minor compression. The height of the available tissue cover over the bony prominence is not the only determining factor for developing pressure sores. Although the covering of sole of foot is soft tissue, they have a vasculature is can with

stand considerable forces. On the sacrum and ischial tuberosity on the other hand, although there is a relatively thick soft tissue cover and a wide supporting surface, the blood vessels are not meant for weight bearing, so even with fairly light compression, pressure ischemia can develop rapidly. Hence, the soles of feet do not develop pressure sores even after prolonged weight bearing in ambulatory patients unless there are underlying causes making them insensate and more prone to pressure damage. The pressure points on various positions are mentioned in Fig. 3.

2. *Shear*

Shearis considered to be more significant than

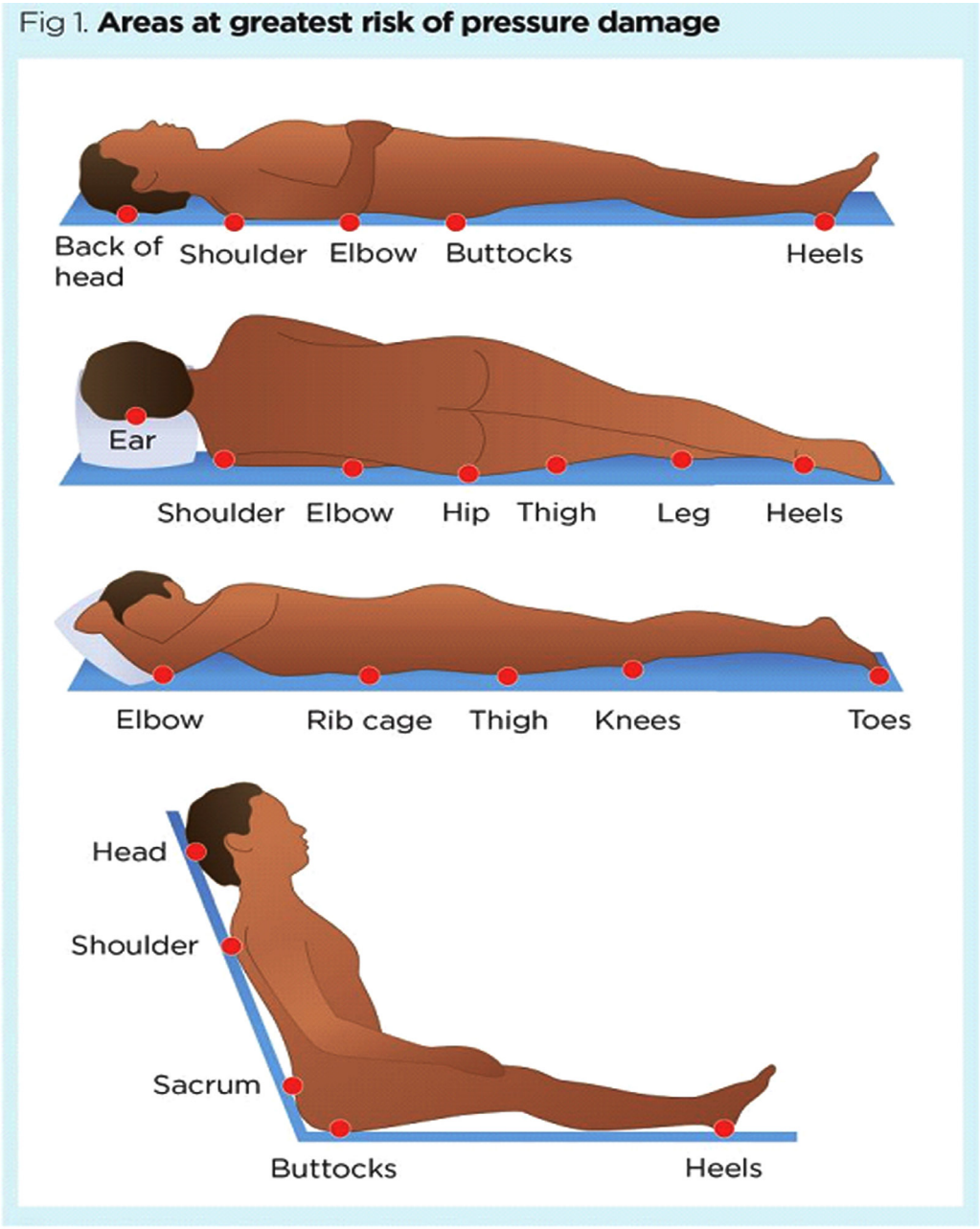

Fig. 3: Pressure Points

pressure in the causation of pressure ulcers.[7] Ischial tuberosities, heels, shoulder blades, and elbows are more prone for shear forces. These are areas on which the body is frequently supported when in a position (such as sitting or lying semi-recumbent) which allows forward slide. Superficial pressure ulcers caused by shearing tend to have an uneven appearance.

3. *Friction*

Friction, along with pressure and shear is also

found as a cause of pressure ulcers.[8] Friction can cause pressure ulcers both indirectly and directly. In the indirect sense, friction is necessary to generate the shearing forces. Shear and friction will act together and causes skin breakdown.

4. Immobility

Immobility alone does not cause pressure ulcers, but in the presence of other factors it can cause them. If the senses are intact, immobility will not lead to pressure ulcers, as the patient can communicate easily. Conversely, comatose patients, even with intact sensation, may develop pressure ulcers because they cannot communicate the pain of an elevated pressure threshold. Tissue ischemia causes pain, so these patients often ask to change their position. Patients with orthopedic casts should be encouraged to report any discomfort and pain to avoid iatrogenic pressure ulcers. Tissue deformation leads to ischemia, increases exercise resistance to reduce stress, and promotes vascular function to restore blood flow to the affected area. These defensive moves are often the result of people not realizing they are making them. However, if these emergency interventions are insufficient to reduce ischemia, the central nervous system is stimulated by discomfort and pain to ensure that pain treatment is provided without permanent damage. When the pressure drops and the blood pressure returns, local blood vessels begin to dilate and blood flow increases, this is called reactive hyperemia. As a result, bright red skin appears on the breast, often called erythema blanching because it is white, as opposed to the dark red non-blanching erythema that indicates tissue damage. Reactive hyperemia causes a rapid restoration of the balance of oxygen and carbon dioxide. It also removes waste products. Erythema disappears when the tissue returns to a resting state. Patients who cannot develop reactive hyperemia cannot recover from the stress of ischemic attacks, leading to tissue damage. Clinically, this manifests as white spots in areas of pressure areas that do not change color as quickly to red reactive occlusion as in healthy people. Instead, the white patches will last for several minutes and then gradually fade to a more normal skin color with little or no visible hyperemia.

5. Combined Pathology

When the reactive hyperaemia cycle stops to function adequately, a pressure ulcer will almost certainly develop. Predisposing factors for pressure ulcers are

- Loss of movement
- Failure of reactive hyperaemia
- Loss of sensation

6. Indirect Causes

a. Age-related physiological alterations can lower the threshold for pressure-induced injury in elderly patients.
b. Any condition that is associated with prolonged, impaired wound healing such as diabetes mellitus.
c. Any condition that is associated with a low tissue oxygen tension is a major cause of pressure ulcers. These include: Heart failure, atrial fibrillation, myocardial infarction, and chronic obstructive pulmonary disease.
d. Peripheral vascular disease.
e. Contractures and spasticity.
f. Loss of sensations, the pain signal that would normally cause an immobile individual to change position is lost.
g. Paralysis and insensibility with skin atrophy.
h. Nutritional conditions such as malnutrition, hypoproteinemia, and anaemia can cause significant delays in wound healing.
i. Moisture causes maceration, which predisposes the skin to injury.
j. Mental health conditions - people with severe mental health conditions such as schizophrenia or severe depression have an increased risk of pressure ulcers because of poor nutrition, associated conditions like diabetes or incontinence, poor personal hygiene.

Grading of Pressure Sore

Staging of Pressure ulcer may done using National Pressure Ulcer Advisory (NPUAP) Panel's Updated Pressure Ulcer Staging System pressure ulcer classification/staging system.[10,11] The staging system provides idea about the amount of anatomical tissue loss in a pressure ulcer. (Fig. 2)

1. Suspected Deep Tissue Injury

Deep tissue injury may be difficult to detect. Usually diagnosed by painful, firm, mushy, boggy, warmer or cooler tissue followed by persistent purple or maroon discolored intact skin or blood-filled blister at pressure point. The word "suspected DTI" may be added to the clinical diagnosis and staging.

2. Stage I Pressure Ulcer

It is diagnosed by intact skin with non-blanchable redness over pressure point. Dark skin people the

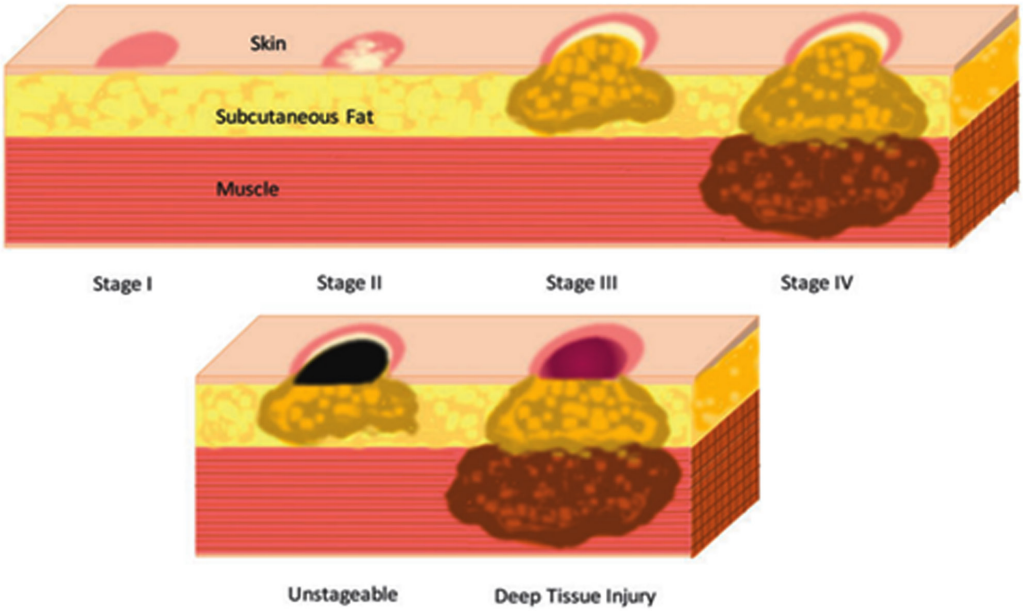

Fig. 2: Stages of Pressure Ulcer

color may be different than surrounding skin. It may be firm, warmer or cooler and painful.

3. *Stage II Pressure Ulcer*

It is characterized by a shallow ulcer with a red pink wound bed, without slough due to partial thickness loss of dermis. There may be an intact or open/ruptured serum filled blister. Surrounding skin may be shiny with tense skin due to subcutaneous fluid collection.

4. *Stage III Pressure Ulcer*

Subcutaneous tissue/fat is exposed with or without slough and/undermined edges due to full thickness loss of skin. Muscle, tendon, or bone, are not visible.

5. *Stage IV Pressure Ulcer*

Loss of skin and subcutaneous tissue with exposed bone, tendon, or muscle with or without slough and/undermined edges.

6. *Unstageable Pressure Ulcer*

Correct depth of tissue injury may not be possible as the base of the ulcer is covered by thick moist soft slough and/or dry tough eschar in the wound bed.

Risk Assessment

All patients admitted to hospital should be assessed for risk, including changes in mobility or treatment. There are many ways to measure risk. The Norton scale measures five categories, from a low of 1 to a high of 4: physical, mental, activity, mobility, and inactivity. A total score below 14 indicates a high risk of pressure ulcers.[12] The Braden scale (Table 3) is also similar, giving scores of up to 4 in the brush categories of hearing, sight, moisture, sports, play, nutrition and communication. A higher score means less risk.[13]

There are many bandages designed to cover high points such as the sacrum and heel, as well as bandages designed to wrap the injured body (especially the feet). However, it should be noted that some dressings can cause problems. For example, during the cutting process, pressure may increase on the edges of the support. There are individual beds (egg crate beds, sky sheep, etc.) that reduce the height of the large space.[14] Compared to cotton, silk is the best at preventing pain because it reduces friction and skin damage.[15]

Prevention

All patients who use dentures or require a wheelchair must be properly adjusted for proper fit and adequate padding. If there is a significant change in weight or body behavior that affects compliance, the application process must be repeated. Sweat, urine and feces can cause maceration of the skin, and if the skin covers the raised areas, the onset of the skin can be painful. Keeping the skin clean and dry is an important part of caring for at-risk patients. Even with sufficient padding, it is important to change position regularly, as even low pressure can cause pain in the long term.

The support area for pressure relief can be divided into static devices and dynamic devices. The device, which tends to distribute pressure over

***Table 3*:** Braden scale for predicting pressure sore risk

Severe Risk: Total score 9 *High Risk*: Total score 10-12 *Moderate Risk:* Total score 13-14 *Mild Risk*: Total score 15-18				Date of Assess				
Risk Factor	**Score/Description**				**1**	**2**	**3**	**4**
Sensory Perception Ability to respond meaningfully to pressure-related discomfort	1. *Completely Limited* – Unresponsive (does not moan, flinch, or grasp) to painful stimuli, due to diminished level of consciousness or sedation, OR limited ability to feel pain over most of body surface.	*2. Very Limited* – Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness, OR has a sensory impairment which limits the ability to feel pain or discomfort over ½ of body	*3. Slightly Limited* – Responds to verbal commands but cannot always communicate discomfort or need to be turned, OR has some sensory impairment which limits ability to feel pain or discomfort in 1 or 2 extremities.	4. *No Impairment* – Responds to verbal commands. Has no sensory deficit which would limit ability to feel or voice pain or discomfort.				
Moisture Degree to which skin is exposed to moisture	1. *Constantly Moist*– Skin is kept moist almost constantly by perspiration, urine, etc. Dampness is detected every time patient is moved or turned.	*2. Often Moist* – Skin is often but not always moist. Linen must be changed at least once a shift.	*3. Occasionally Moist* – Skin is occasionally moist, requiring an extra linen change approximately once a day.	*4. Rarely Moist* – Skin is usually dry; linen only requires changing at routine intervals.				
Activity Degree of physical activity	*1. Bedfast* – Confined to bed. each shift in bed or chair.	*2. Chairfast* – Ability to walk severely limited or nonexistent. Cannot bear own weight and/or must be assisted into chair or wheelchair	*3. Walks Occasionally* – Walks occasionally during day, but for very short distances, with or without assistance. Spends majority of	*4. Walks Frequently*– Walks outside the room at least twice a day and inside room at least once every 2 hours during waking hours				
Ability to change and control body position	*1. Completely Immobile* – Does not make even slight changes in body or extremity position without assistance.	*2. Very Limited* – Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently	*3. Slightly Limited* – Makes frequent though slight changes in body or extremity position independently.	4. *No Limitations* – Makes major and frequent changes in position without assistance.				
Nutrition Usual food intake pattern 1 NPO: Nothing by mouth. 2 IV: Intravenously. 3 TPN: Total parenteral nutrition	1. *Very Poor* – Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement, OR is NPO1 and/or maintained on clear liquids or IV2 for more than 5 days.	*2. Probably Inadequate* – Rarely eats a complete meal and generally eats only about ½ of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement OR receives less than optimum amount of liquid diet or tube feeding.	3. *Adequate* – Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products) each day. Occasionally refuses a meal, but will usually take a supplement if offered, OR is on a tube feeding or TPN3 regimen, which probably meets most of nutritional needs.	*4. Excellent* – Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.				

Table Cont...

Friction and Shear	1. *Problem* - Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures, or agitation leads to almost constant friction.	2. *Potential Problem* – Moves feebly or requires minimum assistance. During a move, skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	3. *No Apparent Problem* – Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.
Total Score	Total score of 12 or less represents *High Risk*		

a wide area, contains bedding materials such as air, water, gel, beads, silicone, foam, and a fur pad. The electronic device eliminates long-term pressure on the anatomical area by changing the pressure under the patient. Many areas of support include: elevated bed replacement, airless bed, and smoke inhalation. Alternating bed pressure reduces bed pressure contact time by creating alternating high and low pressure conduits in the bed. Low Air Loss (LAL) beds allow patients to float in the warmth of heated air. Circulating air balances the patient's pressure and dries the skin.

Turn every 4 hours and use a special foam mattress to reduce the risk of pressure ulcers compared to turning every 2 hours using a pad. A cushion containing gel, foam, air or water can provide pressure relief. Studies have shown that the maximum subcutaneous pressure is within 2 cm of the ischial tubercle. The thicker the pad, the lower the subcutaneous pressure, the best pad is 8 cm. Pressure relief is provided to the patient and should be performed at least every 15 minutes while the patient is seated.

Protein intake is especially important for maintaining nitrogen quality, and vitamin/ mineral supplements are recommended for unfit patients. Prealbumin is used as a short-term test of nutritional adequacy. Albumin is also beneficial, but its long half-life means a better understanding of long-term nutrition.

Diagnosis and Treatment

Staging and documentation of wound size should be performed. Other examinations include location, condition of surrounding skin, presence of tissue undermining and tunneling, and amount of exudate, odor, and tenderness. The pressure ulcer healing scale (PUSH tool) is a commonly used tool developed by the NPUAP that scores pressure ulcers based on wound size, underlying tissue type, and amount of exudate (Table 1).

Table 1: PUSH tool for pressure assessment

Points	Area, cm^2 (Length x width)	Tissue Type	Exudate Amount
0	0	Closed	None
1	<0.3	Epithelial Tissue	Light
2	0.3-0.6	Granualtion Tissue	Moderate
3	0.7-1.0	Slough	Heavy
4	1.1-2.0	Necrotic Tissue	
5	2.1-3.0		
6	3.1-4.0		
7	4.1-8.0		
8	8.1-12.0		
9	12.1-24.0		
10	>24.0		

The Bates-Jensen Wound The Bates-Jensen Wound Assessment Tool assesses wounds according to size, depth, wound margins, tissue undermining, type and amount of necrotic tissue, type and amount of exudate, skin color, presence of edema, induration, granulation, and epithelialization.[16] Other tools such as a pressure ulcer status tool and the Sessing scale are also used.[17] Assessment and monitoring should be done in a careful and consistent manner. CT or MRI is used to assess the extent of tissue involvement and to determine if osteomyelitis is present. In the extremities, adequacy of perfusion should be assessed using the ankle-brachial index and vascular studies. A pressure ulcer patient should be thoroughly evaluated for associated myoneuropathy electromyography (EMG) and nerve conduction studies (NCS), nephropathy

(renal function tests), cardiopathy (lipid profile, ECG, and echocardiogram), and chest X-ray if indicated. Furthermore, the patient should be examined to rule out underlying morbidities such as anemia, hypoproteinemia, and diabetes. Once surgical intervention is planned, routine examinations for fitness for anesthesia and surgery such as hemoglobin, total and differential count, bleeding time, clotting time, PT/APTT/INR, blood grouping, blood sugar, microscopic examination of urine, liver tests (including total proteins and serum albumin), ESR, C-reactive protein, chest x-ray should be performed. Tissue culture and sensitivity (C/S) and C/S blood for bacteria and fungi, tissue biopsy (to quantify bacterial load may be performed as indicated. Biopsy may be indicated to rule out malignant changes in chronic long-term pressure ulcers).

The main treatment involves removal of the problematic source of pressure, adequate debridement of all areas of infection, devitalized tissue, and regular wound care to promote the healing process. The basic principles of surgical treatment of pressure sores remain essentially unchanged since they were enumerated in a report by Conway and Griffith more than half a century ago:

- Excision of the ulcer, surrounding scar, underlying bursa, and soft-tissue calcifications, if any.
- Radical removal of underlying bone and any heterotopic ossification.
- Padding of bone stumps and filling dead space.
- Resurfacing with large regional pedicled flaps.
- Grafting the donor site of the flap, if necessary.

1. Wound Bed Preparation

- Cleaning the wound and careful skin care are the most important parts of the treatment. The process involves removal of surface contamination and careful excision of all dead tissue. This is debridement. In addition to conventional surgical debridement, there are other types of debridement such as mechanical debridement which involves the use of repeated wet to dry dressings to remove deposits, enzymatic debridement using enzymes (papain-urea, collagenase, fibrinolysin, deoxyribonuclease) to liquefy dead tissue and remove it with dressings The literature also mentions biological debridement, or larvae, and larval therapy (in which the larvae eat all the dead tissue and clean the wound without damaging the living tissue). Worms also help fight infection by releasing substances that kill bacteria and stimulate the healing process.[18,19]
- Surgical debridement with a blade or scissors is the most commonly used and most effective method of debridement in skilled surgical hands. Some mechanical debridement techniques include:

Table 2: Types of Wound Dressings

Type of Dressing	Advantages	Disadvantages	Ideal wound
Aliginate dressing	Absorbent, infrequent changes	Expensive	Infected wounds
Foam dressing	Absorbrnt, provide padding	Expensive	Infected wounds, fragile surroundding skin, Stage I and for prevention
Gauze dressing	Inexpensive, microdebridement	Frequent changes	Large complex wounds with exudate or biofilm
Honey dressing	Mild antibiotic	Poor efficacy	Stage II with mild infection
Hydrocollod dressing	absorbent	Expensive	Wound with minimal discharge, Stage II and Stage III
Hydrogel dressing	Hydrating	Moves easily	Dry or dehydrated wounds, unifected grannulating wounds
Silver dressing	Antibiotic	prevents epithelialization	Infection wounds, remove once infection is cleared
Transparent film dressing	Barrier from bodily fluids, infrequent changes	Not porous, can rip skin on removal	Stage I, Stage II without exudate

- Cleaning and pressure irrigation – When removing dead tissue using high-pressure water jets.
- Ultrasound – Low frequency energy waves used to remove devitalized tissue.
- Laser focused beams of light are used.
- Debridement is basically done to convert a chronic wound into an acute wound so that it can go through the normal stages of healing.

Wound Dressings

The dressing used for different stages of wound healing is specialized for each stage. These are classified as non-absorbent, absorbent, debriding, self-adhesive and many others. Dressings are usually occlusive, so ulcers heal better in a moist environment. (Table 2) If the ulcer is clean and dry, occlusive dressings are usually changed weekly, and more frequent changes are avoided because dressing changes remove healthy cells along with debris. Contaminated or oozing wounds may require more frequent dressing changes, sometimes every few hours. Heavily contaminated ulcers are treated with negative pressure wound therapy (NPWT). Special dressings and bandages are used to protect and speed up the healing process of pressure ulcers. These dressings include:

- *Hydrocolloid Dressings:* These contain a special gel that encourages the growth of new skin cells in the ulcer and keeps the nearby healthy area of skin dry.
- *Alginate Dressings:* These are made from seaweed that contains sodium and calcium known to speed up the healing process. Honey impregnated alginate dressings are known to accomplish total wound healing to pressure ulcers.
- *Nano Silver Dressings:* These use the antibacterial property of silver to clean the ulcer.
- *Creams and Ointments:* To prevent further tissue damage and help speed up the healing process, topical preparations, such as cream and ointments are frequently used.

1. *Antibiotics*

Antibiotics are usually only prescribed to treat an infected pressure ulcer and prevent the infection from spreading. If tissue infection exists, antibiotics are necessary to treat the infection, but effort must be made to debride the ulcer thoroughly and leave all viable tissues only, otherwise antibiotics alone will not clean up the ulcer. Topical antibiotics should be avoided because their use may increase antibiotic resistance and allergy. Antiseptic cream may also be applied topically to pressure ulcers to clear out any bacteria that may be present.

2. *Negative Pressure Wound Therapy (NPWT)*

This is an invaluable tool in the management of pressure sores and involves the application of sub-atmospheric pressure to a wound using a computerised unit to intermittently or continuously convey negative pressure to promote wound healing. NPWT, is effective for deep, cavitating, infected and copiously discharging pressure ulcers, particularly with exposed bone. Its benefits include

- Assists granulation.
- Applies controlled, localised negative pressure to help uniformly draw wounds closed.
- Helps remove interstitial fluid allowing tissue decompression.
- Helps remove infectious materials and quantifies exudates loss.
- Provides a closed, moist wound healing environment Promotes flap and graft survival. Both hospital and domiciliary use.
- Reduces hospital/dressings/nursing cost (if we can discharge the patient to home).

3. *Newer Research*

Many products are available to aid wound healing but should be prescribed only under strict medical advice, as they still require further research to determine their effectiveness. These include:

- Growth factors and cytokines.
- Hyperbaric oxygen (HBO).
- Skin graft substitutes (bioengineered skin).
- Connective tissue matrix.
- Expanded epidermis.
- Epidermal stem cells.
- Bone marrow (BM) or adipose tissue derived stem cell (ASC) therapy.
- *Cytokines and growth Factors*

Chronic pressure ulcers show high levels of inflammation and disruption of the collagen matrix, along with increased apoptosis and decreased levels of growth factors and their receptors. Patients treated with GM-CSF or bFGF had higher levels of the respective cytokines after treatment.

After treatment with exogenous bFGF, the bFGF gene was upregulated, suggesting auto induction of the cytokine. Both cytokines and growth factors may play a major role in the treatment of pressure ulcers in the future.

- *Hyperbaric Oxygen Therapy*

Hyperbaric oxygen therapy (HBO) is used to treat pressure ulcers. The HBO chambers are fitted with specially designed devices equipped with a controlled pressure seal and automatic safety valves. A constant pressure of 22 mm Hg (1.03 atmospheres absolute) is maintained inside the chamber using pure oxygen at a flow rate of 2-8 L/ min with direct discharge to atmosphere.

a. It increases oxygen transport to wound area stopping further tissue damage.
b. It facilitates growth of new capillaries (angiogenesis) improving the microcirculation.
c. It speeds up wound healing by reducing inflammation and swelling.
d. It relieves pain.
e. It reduces infection by eliminating bacteria directly and increasing capacity of white blood cell to fight infection.
f. It improves microcirculation and elimination of toxins in the blood.
g. It enhances the effect of some antibiotics h. It stimulates the release of stem cells from the BM.
h. It decreases blood viscosity and risk of thrombosis and stroke.
i. It improves lymphatic circulation.
j. It improves bone density and mineralisation and speeds up bone healing.
k. l. It enhances peripheral nerve regeneration for improved sensitivity.
l. It prepares tissue and bone for grafting before surgery.
m. It speeds up healing after surgery and improves chances of graft survival.

- *Skin Substitutes (Bio-Engineered Skin)*

Cultured keratinocytes are used to treat various types of wounds.[20] For partial/full skin defects, cultured dermal substitute (CDS) is the most effective therapy. Cultured epidermal substitute and cultured skin substitute are also used as biological wound dressings. This artificial dermis induces angiogenesis and fibroplasia in deep, poorly vascularized tissue defects with less vascular invasion. However, it is difficult to apply a collagen matrix to pressure ulcers because they are usually accompanied by infection with the discharge of excessive amounts of exudate or pus and are generally exposed to external forces that prevent graft fixation. Allogeneic CDS effectively treats intractable ulcers, while BM cell implantation combined with allogeneic CDS is used in the treatment of severe ischemic ulcers.

- *Bone Marrow/Adipogenic Stem Cells*

"Cell therapy" is defined as a set of strategies that use living cells for therapeutic purposes. Such therapy aims to repair, replace or restore the biological function of the damaged tissue or

	Primary closure
Sacral pressure ulcer	Reverse dermal flap
	Inferiorly based random skin rotation flap
	Transverse lumbosacral arterial and randomLimberg flap
	Thoracocolumbar-sacral arterial /random flap
	Superior gluteus myoplasty
	Turnover gluteus myopathy
	Gluteus maximus musculocutaneous flap
	Gluteus maximus musculocutaneous island flap
	Gluteus maximus fasciocutaneous flap
	Gluteal fasciocutaneous rotation-advancement flap with V-Y closure
	Bilateral gluteus advancement flap
	Gluteus plication closure
	Sensory island flaps
	Gluteal thigh arterialized flap
	Expansive gluteus maximus flap
	Parasacral perforator-based musculocutaneous flap
	Parasacral peforator-based fasciocutaneous flap
Ischial pressure ulcer	Primary closure
	Biceps femoris musculocutaneous flap
	Random posterior thigh flap ± biceps femoris myoplasty
	Gluteal thigh flap
	Inferior gluteus maximus musculo plasty
	Sliding gluteus maximus flap
	Inferior gluteus musculocutaneous flap
	Tensor fasciae latae with vastus lateralis flap
	Interior gluteus musculocutaneous island flap
	Lateral thigh fasciocutaneous flap
	Gracilis musculocutaneous flap
	Anterolateral thigh fasciocutanous island flap
	Gracilis musculocutaneous flap (with sartorius as a double muscle unit)
	Rectus abdominis musculocutaneous flap
	Hamstring musculocutaneous flap
	Inferior gluteal artery perforator flap.

Table cont....

Trochanteric pressure ulcer	Anteriorly based random thigh flap Tensor fascia lata musculocutaneous flap (island) Random bipedicle flap Vastus lateralis myoplasty Tensor fasciae latae musculocutaneous flap Vastus lateralis musculocutaneous flap Tensor fasciae latae musculocutaneous flap (bipedicle) Gluteus medius tensor fasciae latae musculocutaneous flap Tensor fasciae lata emusculocutaneous flap V-Y advancement Distally based gluteus maximus flap Tensor fasciae lata emusculocutaneous flap (innervated) Gluteal thigh flap Expansive gluteus maximus flap.

organ. Bone marrow (BM) mononuclear cells can be easily obtained in large numbers of aspirates without extensive handling or culture before transplantation, and the cells can be transplanted directly without in vitro expansion. By using the whole mononuclear fraction, no potentially beneficial cell type is missed, and MNCs from the patient's own BM promote angiogenesis, which appears to be a key factor for optimal skin wound healing. The reparative functions of MSCs are thought to include the secretion of factors such as vascular endothelial growth factor or FGF, which could help prevent apoptosis, promote angiogenesis, aid matrix reorganization, and increase the recruitment of circulating MSCs. harvest Although this method is invasive and painful, it is effective in wound healing.

Reconstruction

If there is no microbial growth (especially Streptococcus haemolyticus, Methicillin-resistant Staphylococcus Aureus, Pseudomonas), no osteomyelitis, and the granulation is healthy, the wound can be considered for definitive repair and reconstruction according to the reconstruction ladder.

The advantages of single stage treatment of pressure ulcers include fewer anesthetic episodes, shorter hospital stay, earlier rehabilitation and lower costs. However, multistage treatment should be considered in patients who have pressure ulcers on the front and back of the trunk at the same time.

Surgical coverage options for pressure ulcers include primary closure, random skin flap/Limberg flap, myoplasty plus skin graft, pedicle muscle, myocutaneous, fascial, or fasciocutaneous flap, perforator flaps, free flaps, and tissue expansion. Flap selection depends on many factors such as ulcer location, level of spinal injury, history of previous ulceration and surgery, ambulatory status and potential, daily habits, educational status, motivation level, and associated comorbidities. The flap provides padding to the bony stumps and fills the dead space. Composite flaps increase durability. The flap should not interfere with the adjacent areas of the flap to preserve all future options for the flap cover of the second option. The suture line of the flap should fall away from the area of direct pressure. By planning large flaps, tension on the suture line is minimized. Possible loss of function and need for rehabilitation should be considered in ambulatory patients. For outpatients, an option such as a skin graft may also be considered. One can choose reconstructive option according to the site involved.

Post-operative Care

Measures like pressure relief, shear, friction, moisture, skin care, incontinence, spasticity, and nutrition should be continued aggressively through the post-operative period, in addition to standard post-operative care. The patient is allowed weight bearing on the operated site after 15 days of complete wound healing. Patient can sit for 15 minutes once or twice a day, then gradually increasing the length and frequency of sitting periods until discharge. Pressure release maneuvers are done at least every 15 minutes while the patient is seated, and the surgical site inspection regularly for signs of recurrence.

CONCLUSION

It is important to note that prevention is the key in managing pressure sores. Regular repositioning, proper wound care, and the use of pressure-relieving devices can help prevent the development of pressure sores in individuals at risk. Along with this, early intervention and a multidisciplinary approach involving healthcare professionals from various specialties are essential for successful pressure sore management and reconstruction.

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