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# **REVIEW ARTICLE**

# **PRESSURE ULCERS: A REVIEW**

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ARTICLE INFO	ABSTRACT
Article History:	Pressure ulcers are a localized area of tissue necrosis that develop when soft tissue is compressed
Received 20 <sup>th</sup> September, 2018	between a bony prominence and an external surface. This condition has been recognised for at least
Received in revised form	5000 years and its incidence has been on the rise due to an increasing geriatric population. All the
24 <sup>th</sup> October, 2018	while the cost of managing pressure ulcers has also been on the rise, overburdening health care
Accepted 16th November, 2018	delivery systems worldwide, especially more so in developing nations. Currently pressure ulcers are
Published online 31 <sup>st</sup> December, 2018	seen as a preventable entity, therefore prevention should be a priority in their management. The
	management of established pressure ulcers has progressed over the past 50 years and todays practice
Key Words:	consists of serial wound dressings with debridement if required. More orthodox reconstructive
Pressure Ulcer,	surgical procedures are reserved for grade 3 and 4 ulcers. There has been a drastic reduction in the
Decubitus Ulcer,	associated mortality and morbidity associated with pressure ulcers, but literature still reports
Bedsore,	complications in long standing cases such as acute sepsis, heterotopic ossification, joint sepsis,
Pressure Sore.	malignant transformation and the development of osteomyelitis of the underlying bone. This review
	article aims to outline the mechanism of development, presentation, aetiology, diagnosis, prevention
	and management, both non-surgical as well as surgical and newer advances in treatment of pressure
	ulcers.

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# INTRODUCTION

Pressure ulcers are a type of injury that develops over an area of soft tissue when it is compressed between an underlying bone and a hard external surface leading to ultimate necrosis. This is seen in cases where in which there is prolonged weight bearing, such as in bedridden patients due to chronic illness or paralysis. The clinical sequelae of this damage are pressure ulcers with infection, and exposure of bone. In order of occurrence, the surfaces most commonly involved are those over the sacrum, calcaneus, ischium, and greater trochanter. There is a much greater appreciation of moisture-associated dermatitis as a distinct entity from pressure ulcers. Many sacral and gluteal ulcers that previously could have been considered a pressure ulcer are now more appropriately termed as moistureassociated dermatitis. The National Pressure Ulcer Advisory Panel (NPUAP) added deep tissue injury as a distinct stage of pressure ulcers (National Pressure Ulcer Advisory Panel, 2007).

**Etiopathology:** The development of pressure ulcers is broadly multifactorial, but the ultimate reason behind ulceration is tissue ischemia. An application of a constant pressure of 70 mmHg

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for more than a couple of hours produces irreversible tissue damage (Dinsdale, 1974). Histopathological changes due to application of pressure on the tissues include blood flow occlusion resulting in hypoperfusion and ultimately ischemia, if the initiating insult is not removed. Myocytes are more susceptible to hypoxic insult than cutaneous tissues (Lindan, 1961; Nola and Vistnes, 1980).

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Significant factors leading to development of pressure ulcers include-

- Skin moisture, as it leads to cutaneous maceration. Cases where increased moisture will be a contributory factor include urinary and fecal incontinence, hyperhidrosis and wound discharge.
- Loss of fat and muscle, either physiological as a result of ageing or pathological as in malnutrition.
- Prolonged immobilization, there are areas of bony prominences with thin overlying tissue, which get compressed easily against the hard surface on which the patient is lying leading to subsequent ischemia and necrosis.
- Paralysed patients are more prone to develop pressure areas due to development of large insensate areas and loss of motor function, which would in effect immobilize the patient (Ganong, 1989).

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Predisposing factors leading to the development of pressure ulcers include systemic illnesses like congestive heart failure, anemia, diabetes, peripheral vascular diseases, immunocom promised states and neurological diseases. Those at risk of development of pressure ulcers, commonly develop ulcers when immobilized for an extended duration of time. In the supine position due to illness, injury or even due to surgery on the operating table. The most common sites where pressure ulcers develop include the occipital area, the occipital area, the scapular ridge, the spinous process, the sacrococcygeal area, and thecalcaneus bone. If the patient isplaced on their lateral aspect, the trochanteric area is subjected to pressure and consequently develops cutaneous ulceration.

# **Clinical wound evaluation**

Pressure ulcer evaluation should be done as thoroughly as possible and lesions be staged as objectively as possible, this can be aided by following a recognized staging system. The staging system for pressure ulcers was first proposed by Shea in 1975 (Shea, 1975) and after agreement, finalised by the National Pressure Ulcer Advisory Panel, United States, in 1989.

The various stages of pressure ulceration are as follows-

- **STAGE I-** characterised by an acute inflammatory response. Non blanchable erythema is a feature, albeit hard to notice, especially more so if he patient is dark complexioned. Indirect signs include- in duration, increased warmth and hardness of the cutaneous tissue. An ulcer in this stage could be reversed if the initiating insult is removed and appropriate treatment instituted.
- **STAGE II-** partial thickness skin loss involving the entirety of the epidermis and part of the dermis. Appears as a blister or an abrasion.
- **STAGE III-** full thickness skin loss which may extend into the subcutaneous tissue till the deep fascial layer, which is unaffected. Appears as a deep crater with or without undermining.
- **STAGE IV-** full thickness skin loss with necrosis extending into muscle, bone, joint or surrounding soft tissue with/without undermining or sinus tract formation.

Other aspects of initial wound evaluation include-

- Past medical history- pressure ulceration is always secondary to a primary illness or trauma and a knowledge of it helps in putting up a comprehensive management plan. History should be sought to find out whether the patient is suffering from any spinal cord injury, stroke, spina bifida or other neurological illness. Conditions such as chronic lung diseases, heart disease, diabetes and immunocom promised states might affect healing and the ability of the patient to tolerate anaesthesia if surgery is indicated (Lazarus *et al.*, 1974). History of bowel and bladder control is also essential, since these are known precipitants for pressure ulceration.
- Nutritional assessment- history of food intake and appetite.
- Physiotherapeutic evaluation- for identification of factors which lead to ulceration, including evaluation of mobility, range of motion, transfer, sitting, joint

movement, contractures, type of wheelchair and cushion, and measurement of sitting pressure.

- Lab investigations- for general health evaluation and fitness for surgery
- Risk assessment- to formulate a plan for prevention of ulcers
- Wound assessment and evaluation- proper clinical examination of the ulcer with assessment of site, size, depth, staging, presence of discharge/necrosis/undermining/sinus tracts/infection, presence of granulation tissue and epithelization

# Specific diagnostic investigations:

- Radiological imaging studies-
  - Plain x-ray pelvis AP view and b/l hip x-rays in lateral position- important diagnostic information can be ascertained and also help in pre-operative planning
  - X-rays of spinal column, if pelvic obliquity is present, which causes a pelvic tilt with results in pressure on the ischium
- Sinogram study- sometimes pressure ulcers can present with a sinus opening cutaneously, these are termed as closed ulcers and are commonly seen in ischial and trochanteric areas. In such cases sinograms can be undertaken, but information gathered is not always useful
- Computerised tomography (CT)- can be used to demonstrate soft tissue structure and fluid filled spaces in their different densities. The most important use of CT scans would be to diagnose a deep seated pelvic abscess which can be treated conservatively using needle aspiration (Rubayi *et al.*, 1993).
- Magnetic resonance imaging (MRI)- can be used to diagnose osteomyelitis involving underlying bone. MRI is better than CT scan in distinguishing between soft tissue and bone, and MRI was to replace the CT scan in the diagnosis of osteomyelitis (Ruan *et al.*, 1998). Disadvantages of MRI include non-availability, high cost, inability to diagnose osteomyelitis with certainty in the absence of bone necrosis and inability to differentiate between acute and chronic osteomyelitis.
- Magnetic resonance angiogram (MRA)- special test using MRI to visualize the arterial system of the pelvis, hip, and thighs. It is a non-invasive procedure and can provide a good view of the arterial system in the surgical area. Can be used when there is hip heterotopic ossification (HO) to be excised to examine the relationship between the arterial system of that area and the HO to avoid any injury to the blood vessels during excision of the HO.
- Diagnosis of osteomyelitis and bone biopsy- needle bone biopsy can be undertaken either under local an aesthesia or without as in the case of patients with spinal injury.
- Wound swab for culture/sensitivity and microbiological examination- pressure ulcers are always colonised with polymicrobes including aerobic and anaerobic organisms Pseudomonas like aeruginosa, Proteusmirabilis, Escherichia coli, Klebsiella sp., Enterobactersp., Providencia and sp. Staphylococcusaureus; the anaerobe is Bacteroides sp.

## **Preoperative management**

Surgical management of pressure ulcers is needed in ulcer stages 3 and 4, after giving the ulcer adequate time to respond to local wound treatment, which is usually around 4 weeks. Clinically the wound should be as clean as possible with no signs of infection, discharge or necrosis present. Local wound preparation and care would consist of-

- Debridement- sharp debridement of necrotic tissue or eschar is recommended. Enzymatic debridement should be employed in patients on anticoagulants.
- Local wound care- it is imperative that the ulcer bed is kept clean and dressed to maximise chances of healing. There are a multitude of options available for this. In the presence of slight necrotic tissue or pale granulation tissue, 1% silver sulfadiazine cream can be used after cleaning the ulcer with normal saline, till healthy granulation tissue appears. This can later be changed to just saline soaked dressings. In case of deep ulcers, silver sulfadiazine soaked gauze dressings can be employed, alternatively Dakin's 0.25% solution soaked dressings can be used. The wound should be packed lightly as tightly packed dressings would prevents development of granulation tissue. Once the cavity is filled with healthy granulation tissue, dressing with normal saline is done thrice daily to prevent drying.
- Negative pressure wound therapy- the most commonly used is the VAC dressing. Most common setting of negative pressure is 125 mmHg. Application of VAC removes interstitial fluid, thus helping in healing and cleaning of the wound bed. Disadvantages of VAC include unavailability, high cost, restriction of patient mobility, discomfort or pain and inability to apply over certain anatomical areas effectively.

Another aspect of perioperative care is proper nutritional assessment of the patient. Generally, the subset of patients with pressure ulcers have poor nutritional values. Malnutrition is an important risk factor for pressure ulcer development and degree of malnutrition corelates with stages of ulceration [10]. Comprehensive nutritional assessment of the patient should include the following-

- Dietary intake- factors leading to inadequate dietary intake should be inquired including poor appetite, food intolerance, allergies, and difficulty in chewing and swallowing, which is prevalent in high tetraplegia and advanced neurological disease patients. Patients with stage IV ulcers usually have a poor appetite leading to malnutrition (Bergstrom and Braden, 1992).
- Anthropometric measurements- body weight, body mass index, triceps skin fold, and mid-arm muscle circumference should be assessed. A decrease in total body weight by less than 80 % and lower body mass indicate severe malnutrition and development of pressure ulcer (Ek *et al.*, 1991).
- Biochemical tests- indicate the nutritional status of a patient. Patients with serum albumin levels above 3.5g/dl have a lower incidence of pressure ulcers (Bergstrom and Braden, 1992). Low levels of haemoglobin reduce tissue oxygenation leading to slowed healing and anaemia is found in almost all patients with deep ulcers (Salzberg *et al.*, 1996). Patients should usually have a haemoglobin level of at

least 10 g/dL prior to surgery. Low white blood cell counts (below 1500/cu mm) are associated with pressure ulcer development (Galpin *et al.*, 1976). This association could be non-nutritional and be associated with immunocompromised states or administration of steroids. Caloric intake should be maintained at about 30–40 kcal/kg ofbody weight/day with 1.5 g–2.0 g/kg of body weight of proteins (Breslow *et al.*, 1993). Micronutrients essential for wound healing should also be provided as part of dietary plans. It isrecommended to give oral zinc sulphate in doses of 220 mg/day in the acute stage of wound healing. Optimal wound healing may occur withvitamin C intake of 10–20 mg daily, the same as when 500 mg of vitamin C is given orally (Kessels *et al.*, 1995).

As with any other patient undergoing surgery, pre-operatively fitness for surgery should be sought and in addition medical consult also undertaken for the same. A preoperative screening for heart and lung disease should be performed by chest x-ray, ECG, and echo of the heart in patients with suspected heart disease, pulmonary function test and blood gases should be performed for patients with respiratory problems and kidney function tests should be done insuspectedkidney disease patients.

# General surgical principles

- Surgery is indicated in stage III and IV pressure ulcers. In addition, surgery could be employed in pressure ulcers whichdo not heal, despite local wound care, or a wound that is extensive insize or interferes with the daily life of the patient.
- Reconstructive procedures are decided on a patient to patient basis, but in general a reconstructive ladder is followed- direct closure --- skin graft --- skin flap --- composite flap --- free flap --- tissue expansion.
- Principles of flap design-
  - Geometrical design is the way the flap ismoved toward the defect by advancement, rotation, or transposition. The flap donor site can be closed directly or, if the donor site is large enough, it may require a skin graft.
  - The anatomical content of the flap could either be cutaneous or fascio-cutaneous or muscle onlyor musculocutaneous.
  - The blood supply to the flap can be random,axial (arterial pedicle), or free flap microsurgical(the artery and vein).
- Flap selection- following points should be kept in mind while determining the type of flap to be used-
  - Patient's primary disease. The surgeon should select a flapthat will fill the ulcer defect and heal the wound in a short time. In addition to excellent skin surface and good padding over the bone, consideration of recurrence risk in certain types of patients requires leaving a reserve of sufficient skin and muscles, especially if the patient is in a young age group.
  - The flap should not impact the motor function of the patient in walking, climbing stairs, or flexion of the hip or knee.
  - For each anatomical ulcer location, there is aprimary flap to be used for a primary virgin ulcer, i.e., a first-time ulcer should be considered with attention to the primary disease of the patient.

 In recurrent ulcers, the selection of flap is more complicated and depends on the local tissue available to be used. Taking into consideration the primary disease of the patient, the choice of fasciocutaneous flap or distant muscle flap depends on which primary muscle flaps have been used previously.

#### Specific reconstructive considerations

ISCHIAL ULCERS: These account for around a quarter of all pressure ulcer cases and are common in paralysed individuals. The ulcer can manifest clinically as a stage IV ulcer over the ischial area with ischial bone exposed in the base of the ulcer. In case of long standing cases, the floor of the ulcer is covered with granulation tissue whereas in acute cases necrotic tissue can be seen. The ulcer base can represent the necrotic periosteum of the ischial bone. The ulcer can extend under the gluteus maximus muscle and the hipjoint, and infection can descend into the posterior thigh compartment to form a necrotizing fasciitis. Ischial ulcer can extend superiorly into the coccygealarea or inferiorly in the perineal area to involve the urethra in male patients. In certain cases, bowel diversion may be undertaken, like in cases where there are multiple ulcers perianally, bilateral ischial ulcers or coccygeal ulcer. This is to avoid post-operative infection and flap failure. The diverting colostomy procedure can be temporary or permanent, according to the patient's wishes. A temporary colostomy can be reversed within 6months after flap surgery to demonstrate complete healing and no breakdown in the new flap, which indicates patient compliance to prevent flap breakdown. A double-loop type colostomy is indicated when the patient is at an end stage for flap surgery and the anus is to be closed permanently during the procedure of disarticulation and total thigh flap. The choice of surgical reconstruction depends on many factors, including whether the ulcer is primary or recurrent, the size of ulcer, and whether the patient is ambulatory or sensory. For the closure of defects in stage IV ischial ulcers, the following options are available-

- Inferiorly based gluteus maximus musculocutaneous flap
- Hamstring muscles advancement musculocutaneous flap in V-Y closure
- Gracilis muscle transfer and posterior thigh fasciocutaneous advancement flap
- Gracilis muscle musculocutaneous rotation flap
- Gracilis muscle transfer and medial thigh fasciocutaneous rotation flap
- Gracilis muscle transfer and posterior thigh fasciocutaneous rotation flap
- Gracilis muscle transfer and direct closure
- Advancement of gluteus maximus flap as anisland flap
- A nonhealing stage III ulcer can be closed byskin graft or local fasciocutaneous flap

## Sacral ulcers

Sacral pressure ulcers can develop if a patient is kept in supine position without constant change of position or use of special air/water beds. These ulcers can also be seen afterthe rehab stage when an insensate patient is discharged to integrate back into normal life and begins sitting in a wheelchair, as seen in spinal cord injured patients. Some authors recommend the use of lumbar fasciocutaneous flaps to close a sacral defect (Kato *et al.*, 1999). The use of these flaps is not advised in patients with spinal cord injury or spina bifida because these subset of patients would have undergone multiple surgeries around the vicinity of lumbar area, with resultant scarring and decreased number of perforators. Thus, the other viable option would be flaps based on the gluteus maximus muscle and is durable since it provides an adequate soft tissue cushion over bony areas not covered by muscle (Stevenson *et al.*, 1987).

Solitary sacrococcygeal ulcers can be repaired by-

- Gluteus maximus musculocutaneous rotation flap
- Bilateral gluteus maximus musculocutaneousrotation flap
- Gluteus maximus advancement island flap
- Gluteus maximus splitting transposed flap

Complex and recurrent ulcers can be managed by

- Reuse of the gluteus maximus rotation flap
- Gracilis muscle tunnelled around the analcanal to the coccygeal area
- Vastus lateralis muscle flap covered withextended tensor fascia lata flap, posterior thigh flap, or skin graft

#### **Trochanteric ulcers**

This type of pressure ulcer is commonly seen in chronic insensate patients who lie on the side of their body. Anatomically, the greater trochanter is covered with anatomical bursa and skin; therefore, if ulceration occurs, it will involve the skin and the underlying bursa exposing the tendinous part of the vastus lateralismuscle origin. Healing in a stage IV ulcer may notoccur because of the nature of the tissue and the formation of granulation tissue in the bursal cavity which is colonized by bacteria; consequently, surgical closure is indicated in this condition. In any reconstructive surgery undertaken it is important to completely excise the trochanteric bursa and shave the trochanteric bone. The commonly used flap in reconstruction is the tensor fascia lata myofasciocutaneous flap described by Nahai as a musculocutaneous flap (Nahai et al., 1979) and as a V-Y advancement flap by Lewis (1981). Due to the small size of the muscle, usually the defect will be covered with only the fasciocutaneous component. The tensor fascia lata flap can be used as n island flap, V-Y advancement flap, or a rotation flap. All these modifications and their utilization depend on the size and location of the defect. An alternative to the fascia lata flap is a flap based on the gluteus maximus (Hurwitz, 1988). In cases where the defect is extensive and communicating with the hip joint, the Girdlestone procedure and flap reconstruction using the vastus lateralis or rectus femoris have to be used (Schmidt et al., 1997).

# **Complications of flap surgery**

Non-specific complications following flap surgery include the following-

- Post-operative anemia due to intraoperative blood loss, leading to delayed healing. Patient may require iron supplementation and in severe cases blood transfusion
- Post-operative hypoproteinemia may be seen due to increased catabolism and surgical insult. Patient will require a high protein diet. Usually the protein levels start normalizing by the 3<sup>rd</sup> to 4<sup>th</sup> post-operative week.

• Deep venous thrombosis (DVT)- due to extended immobilization. This complication is not usually seen in chronic spinal cord injured patients because of their history of long-term paralysis and limited ambulation. Their venous muscle pumpsystem is accustomed to pumping the venous blood while the patient is in sitting position in a wheelchair. Therefore, surgery does not increase the risk of DVT unless if there is a change in the coagulability of the patient's blood, an increase in the viscosity of the blood (e.g., from dehydration or extensive hip and pelvic surgery), or a history of DVT.

# Specific complications associated with flap reconstruction in pressure ulcer patients include

- Wound dehiscence- usually occurs in the 2<sup>nd</sup> or 3<sup>rd</sup> postoperative week at the time of suture removal. Managed by local wound care and electrical stimulation of the wound (Baker *et al.*, 1996).
- Wound edge necrosis- if skin is sutured under tension or if skin vascularity is compromised due to previous surgeries. Managed by debridement of necrotic edges and local wound care.
- Seroma formation- detected after suture removal when serous fluid discharges from the flap wound. Seroma formation under the flap is secondary to bursa development under the flap, which may result from severe uncontrolled spasticity, early removal of drainage tube, obstruction in the drainage tube by a clot, or when dead space under the flap is not obliterated completely. Management of this complication is aggressive irrigation of the space under the flap with normal saline, using a catheter inserted under the flap. The open area is then packed with a small strip of gauze soaked in normal saline or Dakin's solution and changed twice daily. This management decreases the bacterial colonization of the space under the flap, which eventually will help to heal the open flap, and the flap will adhere back to its base. If conservative management is unsuccessful in closing the bursa, surgical management is indicated and entails opening part of the flap, debridement of all the granulating tissue or excising the bursa, and flap closure under a drainage system.
- Wound infection- flaps close to the anus and perineum are more prone to develop infection than flaps in other areas of the body. Management is wound drainage and irrigation and wound packing with Dakin's solution. Intravenous antibiotics are used if clinical signs of sepsis are present, such as fever or an increased leucocyte count with increased neutrophils.
- Hematoma- can occur secondary to inadequate hemo stasis intraoperatively. Post-operative DVT prophylaxis can also contribute to hematoma formation. The end products of hematoma are toxic and can cause tissue necrosis. Management is by opening the flap, evacuation of the hematoma and thorough irrigation using saline. Thereafter the wound is packed with saline or Dakin's solution packs and changed at least twice daily. If this fails, then curettage of the cavity and flap closure with a closed drainage system is done in the operation theatre.
- Fat necrosis- can occur in patients with thick subcutaneous layers of fat. Usually manifests in the 1<sup>st</sup> post-operative week as a yellow fat droplet laden

discharge from the wound edges. Regular dressings are undertaken and if these fail, debridement of the necrotic fat done. If local wound care measures fail then flap revision surgery is needed.

• Flap necrosis- complete flap necrosis can occur in the distal part of the flap occasionally due to extended flap design or suturing under tension. There is epidermolysis with subsequent dermal necrosis. This complication is seen mainly in fasciocutaneous flaps such as the extended tensor fascia lata flap or medial thigh fasciocutaneous flap. Management at early stages is by keeping the epidermolysis part of the flap well hydrated with bacitracin ointment to keep deep dermal layers hydrated in turn. If full cutaneous necrosis ensues, debridement has to be done and local wound care / VAC dressings undertaken. Thereafter a skin graft can be applied or flap used if bone is expose.

# **Pressure ulcer prevention**

Patients at risk of developing ulcers should be identified and have their skin inspected for any changes and damage at least twice daily. The skin should be kept clean and dry. Special mattresses should be used to eliminate pressure. Currently these special mattresses are classified based on their static or dynamic nature. Static surfaces (such as foam filled mattresses, air-filled mattresses, fluid-filled mattresses) do not require electrical power, while dynamic surfaces (such as alternating air pressure mattresses or pneumatic ripple beds) require electrical power for shifting and redistributing the pressure within the surface. Other integrated electronic beds like air fluidised beds (Clinitron or Kin Air bed) (Hargest and Artz, 1969) and electronic moving air mattresses require high technology and heavy machinery to let air and ceramic sphere particles support the object on a stream mechanically; are often costly, noisy and not easily available. Due to lack of substantial evidences and researches, it is difficult to firmly conclude about relative effects of support surfaces. Those who are wheelchair bound can be provided with custom designed gel and pneumatic wheelchair cushions which are easily available, and they help to distribute the load more evenly and help in preventing ulcer formation. In addition, patients and their caretakers are advised to conduct pressure release movements or weight shifts on regular intervals to prevent pressure concentration and tissue damage. It is important that the patient understands that there is always a need to do pressure relief and give several options on how to perform pressure reliefs in a variety of settings. Pressure relief in sitting needs to be done every 15-30 min (McDonald, 2001).

## Conclusion

Pressure ulcers are a frequent complication in long term hospitalized bed-ridden patients or those with chronic illnesses limiting mobility. The incidence of pressure ulcers has been on the rise and the cost of managing pressure ulcers has increased dramatically in hospitals and in the community due to the overall increase in healthcare costs worldwide. The impact of pressure ulcers is significant in terms of both financial and nonmonetary costs and its occurrence has been recognised as a marker for quality of care. The best method of reducing the burden of pressure ulcers is by instituting stringent protocols to identify patients at risk of development of pressure ulcers along with preventive measures. These preventive measures include the use of special mattresses and patient/caregiver education on the need to keep on changing position frequently. In stage III and IV ulcers, surgery is the only therapeutic option that could prevent recurrence of pressure ulcers. When these are located in sacral, ischial or trochanteric regions, it is important to cover the defects with a thick flap to give more support and protection to the areas which are subjected to pressure. The best results could be accomplished, by utilizing myocutaneous flaps or cutaneous flaps with muscle interposition, which might guarantee a successful repair and a good long term recurrence free survival.

# REFERENCES

- National Pressure Ulcer Advisory Panel. NPUAP pressure ulcer stages/categories [Internet].2007. Available from: http://www.npuap.org/wpcontent/uploads/2012/01/NPUAP-Pressure-Ulcer-Stages-
- Categories.pdf Dinsdale SM. 1974. Decubitus ulcers: role of pressure and friction in causation. Arch Phys Med Rehabil55:147–155
- Lindan O.1961. Etiology of decubitus ulcers. Arch Phys Med Rehabil, 2:774-783
- Nola GT, Vistnes LM. 1980. Differential response of skin and muscle in the experimental production of pressure sores. *Plast Reconstr Surg*, 66:728
- Ganong WF. 1989. Review of medical physiology. Appleton & Lange, San Mateo
- Shea JD. 1975. Pressure sores: classification and management. *Clin Orthop.*, 172(112):89–100
- Lazarus GS, Cooper DM, Knighton DR et al. 1994. Definitions and guidelines for assessment of wounds and evaluation of healing. Arch Dermatol., 130:489–493
- Rubayi S, Soma C, Wang A. 1993. Diagnosis and treatment of iliopsoas abscess in spinal cord injury patients. Arch Phys Med Rehabil., 74:1186–1191
- Ruan CM, Escobedo E, Harrison S, Goldstein B. 1998. Magnetic resonance imaging of non-healing pressure ulcers and myocutaneous flaps. *Arch Phys Med Rehabil.*, 79:1089
- Bergstrom N, Braden B. 1992. A prospective study of pressure sore risk among institutionalized elderly. J Am Geriatr Soc., 40(8):747–758

- Ek A, Unosson M, Larsson J. *et al.* 1991. The development and healing of pressure sores related to the nutritional state. Clin Nutr 10:245–250, {Scientificevidence-II}
- Salzberg CA, Byrne DW, Cayten CG et al (1996) Anew pressure ulcer risk assessment scale for individualswith spinal cord injury. Am J Phys Med Rehabil75:96–104
- Galpin JE, Chow AW, Bayer AS. et al. 1976. Sepsis associated with decubitus ulcers. Am J Med., 61:346–350
- Breslow RA, Hallfrish J, Guy DG. et al. 1993. Theimportance of dietary protein in healing pressure ulcers. J Am Geriatr Soc., 41:357–362
- Terriet G, Kessels A, Knipschild P. 1995. Randomized clinical trial of ascorbic acid in the treatment of pressure ulcers. J Clin Epidemiol., 48:1453–1460
- Kato H, Hasegawa M, Takada T. et al. 1999. The lumbar artery perforator based island flap: anatomical study and case reports. *Br J Plast Surg.*, 52:541
- Stevenson TR, Polock RA, Rohrich RJ, Vander Kolk C.A. 1987. The gluteus maximus musculocutaneous island flap: refinements in design and application. *Plast Reconstr Surg*, 79:761
- Nahai F, Hill HL, Hester TR. 1979. Experiences with the tensor fascia lata flap. *Plast Reconstr Surg.*, 63:788
- Lewis VL Jr, Cunningham BL, Hugo NE. 1981. The tensor fascia lata V-Y retro position flap. Ann Plast Surg 6:34
- Hurwitz DJ (1988) Re Ramirez: the distal gluteusmaximus advancement musculocutaneous flap for coverage of trochanteric pressure sores (letter). Ann Plast Surg 20:198
- Schmidt AB, Fromberg G, Ruidisch M-H (1997)Applications of the pedicled vastus lateralis flap for patients with complicated pressure sores. Spinal Cord 35:437
- Baker L, Rubayi S, Villar F, Demuth SK (1996) Effect of electrical stimulation waveform on healing of ulcers in human beings with spinal cord injury. Wound Repair Regen 4:21–28
- International review. London: Wounds International; 2010. Pressure ulcer prevention: Pressure, shear, friction and microclimate in context. A consensus document.
- Hargest TS, Artz CP. A new concept in patient care: The airfluidized bed. AORN J. 1969;10:50–3
- McDonald H (2001) Preventing pressure ulcers. Rehab Manage 10:5–20

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