# MANAGEMENT OF PRESSURE ULCERS IN GENERAL SURGERY WARDS

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Abstract: The objective of this study is to focus in discussing the surgical treatment of pressure ulcer, and also to overview the pathophysiology, causes and risk factors of PU. We searched MEDLINE, EMBASE, and CINAHL and Cochrane databases for relevant English language studies with human subjects, published up to the end of 2016, identify relevant randomized controlled trials (RCTs), reviews and meta-analysis studies. Current search was performed using the terms; pressure ulcer, pressure sore, decubitus, bedsore, surgical treatment. Bibliographies of identified articles were further reviewed for more relevant articles to be included in this review. The pressure ulcer patient's way through the system depends on the local organization. In general, pressure ulcer patients are treated in your area. Debridement can be carried out by the local cosmetic surgeons. Comprehensive debridement needs to be performed in the operating room, however minor debridement is commonly performed at the bedside. Although much of these patients are insensate, others are unable to communicate pain sensation. Pain medication must be administered freely, and crucial signs might show pain understanding. It is crucial to cover the flaws with a thick flap to provide more support and protection to the locations which go through pressure and to lower the incidence of reoccurrences.

Keywords: Ulcers, RCTs.

#### 1. INTRODUCTION

Pressure ulcers (PUs) (also known as bed sore, pressure sore and decubitus ulcer) is a potentially uncomfortable effect of failing in medical and nursing care that frequently occurs in individuals with restricted mobility, long hospital stays; sever health problem and malnutrition (1,2,3).

In general, the most typical pressure points are: sacrum, ileac crest and in-between butts, shoulders, lateral or inner element of the knees, prominences of the head, ankle location, the heel and elbow. The pressure sores can establish either ostensibly or from within the deep tissue <sup>(3,4)</sup>.

Pressure ulcers have actually afflicted humans since antiquity. Overall, 85% of people with spinal cord disorders establish pressure ulcers throughout their life time and approximately 8% die from them <sup>(4)</sup>. Pressure ulcers increase length of stay, escalate the expense of treatment, and hinder quality of life. Numerous physical, pharmacologic, and surgical approaches have actually been tried in the treatment of pressure ulcers. Sir John Staige Davis is credited with being the very first to try surgical cure of pressure ulcers <sup>(5)</sup>. Now, a number of years later on, lots of choices are offered for surgical management of pressure ulcers, including direct closure, skin grafting, skin flaps, and musculocutaneous flaps <sup>(6)</sup>.

In patients with pressure ulcers or for those patients who are at high risk of developing pressure ulcers, a relevant history of movement, previous immobility, neurological impairment (eg, paraplegia, multiple sclerosis), and a clinical assessment defining significant pressure points encountered in daily life are critical for developing preventive and restorative interventions <sup>(7,8)</sup>. Patient positioning and approaches to lower pressure-related tissue damage are amongst the most

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important treatment parts. PUs are classified by the level of visible tissue damage, where stage I PUs exhibit non-bleachable erythematic (i.e., redness) on intact skin, phase II PUs are partial density ulcers, and stages III and IV ulcers include full-thickness damage (**Figure1**) <sup>(9)</sup>. They are thought to occur from mix of extrinsic forces such as pressure, shear, and friction and intrinsic factors such as age, malnourishment, and consciousness level that influence a person's tissue tolerance <sup>(10,11)</sup>. Previous research studies have determined the list below factors as increasing the probability of developing a pressure ulcer: immobility, admission to the ICU, poor nutrition, incontinence, hypoalbuminemia, spinal cord injury, stroke, reduced level of awareness, fractures and/or major orthopedic procedure, advanced age, injury, reduced perfusion, bad wound healing, insufficient nursing care, and chronic disease <sup>(12,13)</sup>.

The objective of this study is to focus in discussing the surgical treatment of pressure ulcer, and also to overview the patho-mechanisms, causes and risk factors of PU.

#### 2. METHODLOGY

We searched MEDLINE, EMBASE, and CINAHL and Cochrane databases for relevant English language studies with human subjects, published up to the end of 2016, identify relevant randomized controlled trials (RCTs), reviews and meta-analysis studies. Current search was performed using the terms; pressure ulcer, pressure sore, decubitus, bedsore, surgical treatment. Bibliographies of identified articles were further reviewed for more relevant articles to be included in this review.

#### 3. RESULTS

#### o Pathophysiology, etiology, and risk factors:

The NPUAP <sup>(27)</sup> defines a pressure ulcer (or "sore") as a soft tissue injury arising from unrelieved pressure over a bony prominence, resulting in ischemia, cell death, and tissue necrosis. This definition is more inclusive than the related terms bedsore or decubitus ulcer that suggests ulcerations only over bony prominences in the recumbent position (sacrum, trochanter, heel, occiput, and back), however not ulcerations from pressure areas in the seated position (e.g. ischial tuberosities) <sup>(14)</sup>. For that reason, a comprehensive list of bony prominences vulnerable to press ulcer in SCI need to include ischial tuberosities, trochanters, sacrum, heels, malleoli, back, occiput, scalp, and elbows. A number of research studies determine the most typical sites of occurrence to be the ischium (28%), the sacrum (17 - 27%), the trochanter (12 - 19%), and the heel (9 - 18%) <sup>(15,16,17)</sup>. In all these vulnerable locations, pressure ulcers occur when external pressure exceeds capillary pressure (12 - 32 mmHg), and ischemia of tissue begins to display a spectrum of injury patterns <sup>(18)</sup>. The pathological sequelae of ischemia, necrosis, and anoxia can be reversed at the ischemic stage if the factors triggering injury are recognized and gotten rid of.

Pressure ulcers are brought on by numerous pathogenic systems, including direct pressure  $^{(19,20)}$  and microcirculatory ischemia  $^{(21)}$ . Despite an increasing occurrence of pressure ulcers in the United States, the molecular mechanisms behind their advancement have actually not been totally illuminated. Tissue modifications in action to pressure may include structural changes in the dermis and cellular deformation followed by extracellular and cytoskeletal matrix modifications  $^{(22)}$ . The skin of the non-healing edge of the pressure ulcers, along with that of diabetic and venous foot ulcers, reveals increased expression of c-myc and activation of nuclear  $\beta$ -catenin that causes formation of thick, hyperproliferative skin  $^{(23)}$ 

Most of individuals impacted with pressure sores are those having health conditions (physical or mental) that motivate immobility, particularly those who are restricted to bed or chair for extended amount of times. Several other health conditions that affect blood supply and capillary perfusion, such as type-2 diabetes, can make an individual more susceptible to push ulcers. Age is likewise a factor that the bulk (approximately two-third) of pressure ulcers occur in aging people (60-80 years of age) (24). To put it more merely, any individual, with or without a medical condition, who is incapable of avoiding extended durations of an uninterrupted compression, is at a risk of pressure ulcers. Majority of the patients impacted with pressure ulcers regularly develop it over a bony prominence. Majority of the cases supposedly are affected over the area where skin covers bones such as sacral, ischial and trochanteric pressure ulcers (25) and the lower extremities these are seen in the malleolar, heel, patellar and pretibial areas represent roughly 25% of all pressure sores (26). (Figure 1) describes the numerous direct and indirect causes of pressure ulcers (26).

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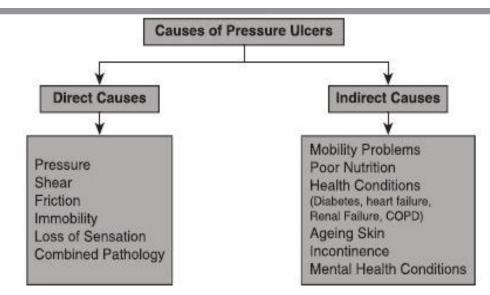
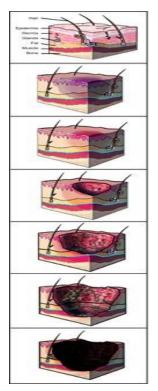


Figure1: Cause of pressure ulcer (26)

According to the NPUAP classification (**Figure 2**) <sup>(27)</sup>, the pressure ulcers that have to be surgically treated are those in Stage 3 and Stage 4 <sup>(27)</sup>. These NPUAP staging standards can be taught, however staging guidelines can be tough to apply, particularly in the SCI population. Pain is typically a presenting symptom of a stage I ulcer in the patient with undamaged feeling, however the absence of protective experience in patients with SCI positions them at higher risk for postponed discussion of pressure ulcers. The darker skin coloring of African Americans and other ethnic cultures can mask phase I ulcers, resulting in disparities in the reporting of the intensity of initial presenting pressure ulcers among racial groups in some studies <sup>(15,16,17)</sup>. Darker skinned patients are for that reason, at increased risk and ought to be carefully kept an eye on. In addition, in analyzing the above guidelines, it is essential to recognize that deep tissue injury and unstageable ulcers are descriptive phases and do not easily equate to definitive wound care suggestions or treatment. Deep tissue injury can advance to a phase I - IV and unstageable ulcers often require debridement (see Treatment below) for the sore's depth to declare itself as a definitive stage of tissue loss. Unstageable ulcers frequently represent complete thickness ulcer ranging from stage III to IV <sup>(27)</sup>.



#### Normal Skin

Normal skin showing intact epidermis, dermis, and the deeper layers of subcutaneous fat, muscle and bone.

#### Suspected Deep Tissue Injury:

Purple or maroon localized area of discolored intact skin or bloodfilled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer or cooler as compared to adjacent tissue.

#### Stage I:

Intact skin with non-blanchable redness of a localized area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its color may differ from the surrounding area.

#### Stage II:

Partial thickness loss of dermis presenting as a shallow open ulcer with a red pink wound bed, without slough. May also present as an intact or open/ruptured serum-filled blister.

#### Stage III:

Full thickness tissue loss. Subcutaneous fat may be visible but bone, tendon or muscle are not exposed. Slough may be present but does not obscure the depth of tissue loss. May include undermining and tunneling.

#### Stage IV:

Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present on some parts of the wound bed. Often include undermining and tunneling.

#### Unstageable:

Full thickness tissue loss in which the base of the ulcer is covered by slough (yellow, tan, gray, green or brown) and/or eschar (tan, brown or black) in the wound bed.

Figure 1: NPUAP guidelines for staging pressure ulcers. (27)

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#### Surgical technique for management of PU:

#### (Surgical debridement)

Operative debridement to treat pressure ulcers has been thoroughly explained, with the very first report of personnel debridement to adequately prepare the wound bed for recovery published in 1950 by Cannon et al <sup>(28)</sup>. Since then, a number of reports have detailed particular aspects of the surgical technique <sup>(29,30,31)</sup>, although couple of have provided a step-by-step description and subsequent outcomes. No matter whether a pressure ulcer heals subsequently by myocutaneous flap coverage or by secondary intent, surgical debridement to get rid of infection is an important part of injury bed preparation. Prior to operation, the surgeon checked out the wound for weakening, using a swab or gloved finger (**Figure 2**) <sup>(31)</sup>. Undermining is defined as the destruction of tissue or ulcer extending under the skin edges such that the pressure ulcer measures larger at its base than at the skin surface area <sup>(31)</sup>. For patients with ischial or sacral pressure ulcers, a rectal examination was carried out, and the location overlying the rectum was shown with a marking pen. During debridement, the perirectal fibrinous tissue might be removed, however deep penetration to the rectal muscle need to be prevented <sup>(31)</sup>.



Figure 2: Exploration of the wound for undermining can be performed with a gloved finger or a sterile cotton swab (31)

One important study <sup>(32)</sup> performed surgical treatment and showed the wound was extensively prepped in order to record the outer location of weakening. Sections of the overlying tissue were resected to expose locations of undermining. This tissue usually includes healthy-appearing skin as well as the underlying subcutaneous tissue and fascia, and it is resected in triangular segments, using electrocautery to reduce bleeding. As (**Figure 3&4**) <sup>(32)</sup> illustrate, the peak of the triangle should extend to the outermost area of the location of weakening, and the base of the triangle ought to be adjacent to the external edge of the wound. The resected area needs to be limited to the minimum amount needed to expose the pockets of undermining and to allow for debridement of underlying necrotic tissue and full exposure of the wound bed. This resection will likewise permit deep packing in dressing modifications. Although healthy skin is removed, this skin generally heals faster than tissue at the base of the wound <sup>(32)</sup>.

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Figure 3: Outline of triangular segment of undermining (32)

Figure 4: Skin covering undermining removed (32)

#### • Reconstructive surgical techniques:

#### A. Direct closure:

Direct closure is a simple, Rarely suggested method in pressure ulcer surgical treatment <sup>(33)</sup>. It is the procedure of requiring the edges of the pressure ulcer surface area together where tissue has vanished. This will lead to increased tension in the superficial tissue and a deep cavity, factors getting involved in the development of pressure ulcers and dehiscence <sup>(33,34)</sup>. In many cases where the absence of tissue is not undue, direct suture may occasionally be possible and is then a good idea. In the authors' experience very few pressure ulcers appropriate for direct closure.

#### B. Skin grafting:

Skin grafting works for elimination of a problem. A fairly basic procedure, it nevertheless needs a well vascularized bed and immobilization throughout healing. The graft generally heals in about 10 days; however, the patient should abstain from mechanical loading of the graft for about 3 weeks. A split-thickness skin graft is thin and unpliable and therefore deteriorates quickly when subjected to pressure and friction. Split-thickness skin grafting is suggested in large, shallow, and well-granulating pressure ulcers, and in cases where non-physiological high mechanical pressure is not a factor. A full-thickness skin graft has much better mechanical homes than a split-thickness skin graft but imposes more demands on the recipient bed. It is more vulnerable to mechanical pressure than a regional flap, which is why a flap is often preferred to a graft (33).

#### C. Local flaps:

Flaps can be classified according to the type of vascular supply or the kinds of tissue in the flaps (ie, skin, fascia, and muscle). Random flaps have no particular blood supply. Because of the lack of well-defined vessels providing blood throughout the flaps, they are of restricted dimensions and include a single or a few tissue layers (skin, subcutis, fascia). Flaps bring distinct vessels (axial flaps) have a much broader variety of reconstructive capabilities with regard to tissue, size, and adaptability constituents (35,36). Fasciocutaneous flaps have a better blood supply than many random cutaneous flaps. The additional padding provided by the fascia is of minimal significance from the view of pressure circulation. The subcutaneous tissue in between the skin and fascia will all be incorporated in the fasciocutaneous flaps, but subcutaneous tissue is of minimal value in pressure ulcer treatment because of its low resistance to pressure and tear and its modest blood supply. Fasciocutaneous flaps appropriate for the reconstruction of selected stage III or IV pressure ulcers without underlying osteomyelitis and without nonphysiological loading. Scrotal flaps have been utilized to cover perineal pressure sores. In theory, it must be helpful that the skin is able to slide on a wide range of fascias, however we have not discovered this kind of flap satisfying. Myocutaneous flaps are the treatment of choice in reconstructive procedures for deep pressure ulcers (33,34,37,38). The myocutaneous flap uses the very best chance for supplying the pressure ulcer cavity with sufficient bulk consisting of both excellent blood supply (muscle) and normal integumental cover (full-thickness skin). However, it needs to be noted that muscle has a low tolerance for ischemic injury. From an anatomical perspective, there is no reason for transposition of muscle into a pressure ulcer cavity, due to the fact that these pressure point areas do not, under normal situations, include muscle. Experiments suggest that muscle underneath pressure-loaded skin is helpful (Figure 5) (39).

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Figure 5: Right myocutaneous V-Y hamstring flap covering a right ischial pressure ulcer (39)

#### Advanced and unconventional procedures:

Sensate flaps (ie, flaps containing intact sensory nerves) have actually been utilized in spine-- hurt patients to provide skin cover with sensation (37) to the otherwise anesthetized skin. Recurrence rates might be minimized with using sensate flaps (33,40), but brand-new sensation can have the type of undesirable dysesthesias. This makes the patients move any loading to just outside the sensate part of the flap, to an insensitive area, and a new pressure ulcer can establish (40). An available flap can be raised from areas with undamaged experience, such as the tensor fascia lata flap to the pelvic region (40). Free flaps are muscle or myocutaneous flaps absolutely devoid of the donor site and connected to vessels at the recipient site by microsurgical strategies. Free flaps in pressure ulcer surgical treatment are only rarely explained in the literature. Free flaps include resource-demanding and time-consuming procedures and most likely should be restricted to use as a last option. Tissue expansion has actually been introduced in the treatment of pressure ulcers as skin expansion (33,40) or growth of a number of tissue layers (33). Broadening the tissue surrounding a pressure ulcer by slowly inflating expanders underneath the tissue in question increases the availability of regional tissue. However, experience in this field is limited. Enhancing the tissue covering former or threatening pressure ulcer areas has actually been attempted using carbon fiber pads. Minns et al (41) discovered the technique encouraging, with a success rate of 68%.

#### **CONCLUSION** 4.

The pressure ulcer patient's way through the system depends on the local organization. In general, pressure ulcer patients are treated in your area. Debridement can be carried out by the local cosmetic surgeons. Only in cases where interventions besides standard treatment are anticipated will the patient be examined by experts. The plastic cosmetic surgeons who will perform the reconstructive procedures ought to be included if surgical treatment is expected. Debridement is focused on removing all debilitated tissue that serves as a tank for ongoing bacterial contamination and possible infection.

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Comprehensive debridement needs to be performed in the operating room, however minor debridement is commonly performed at the bedside. Although much of these patients are insensate, others are unable to communicate pain sensation. Pain medication must be administered freely, and crucial signs might show pain understanding. It is crucial to cover the flaws with a thick flap to provide more support and protection to the locations which go through pressure and to lower the incidence of reoccurrences.

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