TELE-REHABILITATION GUIDELINE
Pressure Ulcers

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I. Definition, assessment, and diagnosis

A. Definition

1. Pressure ulcers are sores caused by ischemia due to elevated or prolonged pressure to the skin and underlying tissue, and occur most often over bony prominences.
2. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear.
3. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue.
4. There are numerous grading systems but one widely used system is the National Pressure Ulcer Advisory Panel (NPUAP) pressure ulcer stages. [1] In this system the stage is determined by the depth of the tissue damage observed and is used primarily for initial assessment of a pressure ulcer.
   a. Stage 1 Pressure Injury: Non-blanchable erythema of intact skin:
   Intact skin with a localized area of non-blanchable erythema, which may appear differently in darkly pigmented skin. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Color changes do not include purple or maroon discoloration; these may indicate deep tissue pressure injury.
   b. Stage 2 Pressure Injury: Partial-thickness skin loss with exposed dermis:
   Partial-thickness loss of skin with exposed dermis. The wound bed is viable, pink or red, moist, and may also present as an intact or ruptured serum-filled blister. Adipose (fat) is not visible and deeper tissues are not visible. Granulation tissue, slough and eschar are not present. These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel. This stage should not be used to describe moisture associated skin damage (MASD) including incontinence associated dermatitis (IAD), intertriginous dermatitis (ITD), medical adhesive related skin injury (MARSI), or traumatic wounds (skin tears, burns, abrasions).
   c. Stage 3 Pressure Injury: Full-thickness skin loss:
   Full-thickness loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.
   d. Stage 4 Pressure Injury: Full-thickness skin and tissue loss:
   Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled edges), undermining and/or tunneling often

occur. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss this is an Unstageable Pressure Injury.

e. Unstageable Pressure Injury: Obscured full-thickness skin and tissue loss: Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a Stage 3 or Stage 4 pressure injury will be revealed. Stable eschar (i.e. dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.

f. Deep Tissue Pressure Injury: Persistent non-blanchable deep red, maroon or purple discoloration:
Intact or non-intact skin with localized area of persistent non-blanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone-muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle or other underlying structures are visible, this indicates a full thickness pressure injury (Unstageable, Stage 3 or Stage 4). Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions.

B. Assessment

1. Assessment of individual with a pressure ulcer
   a. Complete history
   b. Physical examination and laboratory tests
   c. Psychological health, behavior, cognitive status, and social and financial resources
   d. Availability and use of personal care assistance
   e. Positioning, posture and related equipment
2. Assessment of the pressure ulcer
   a. Anatomical location
   b. Size (length, width, depth and wound area)
   c. Stage
   d. Exudate/odor
   e. Necrosis
   f. Undermining
   g. Sinus tracts
   h. Infection
   i. Healing (granulation and epithelialization)
   j. Wound margins/surrounding tissue

II. Management and treatment recommendations

A. Normal physiological wound healing is a complex, dynamic process which can be divided into four main phases: hemostasis, inflammation, proliferation, and tissue remodeling.[2 3 4]

1. Immediately following injury, the healing response begins with the activation of the coagulation cascade and the creation of a blood clot. Various chemotactic factors, including platelet-derived growth factor (PDGF), stimulate the migration of blood

neutrophils and monocytes, helping to initiate the second phase of healing, the inflammatory response.[5]

2. Inflammation is responsible for containing, neutralizing, diluting, or walling off the injurious agent or process. Acute inflammation lasts only minutes to days depending on the extent of injury as leukocytes, predominately neutrophils, invade the wound and clear it of debris. As the neutrophils diminish, the wound moves to the chronic inflammation phase with increased activity of macrophages and lymphocytes. This debrides the wound and promotes wound healing. Interleukins and tumor necrosis factor (TNF) concurrently promote angiogenesis and stimulate fibroblasts activity, leading to the wound transitioning to the proliferative phase.[6]
3. Granulation tissue formed during the proliferative phase of healing provides a temporary weak tissue layer in the wound. New blood vessels are produced by budding or sprouting from pre-existing vessels in a process known as neovascularization or angiogenesis. Angiogenesis is essential for granulation tissue formation and improves the overall tissue perfusion (30).
4. Reepithelialization: Migration and proliferation of keratinocytes from the wound edges leads to the formation of a thin epithelial layer over the developing granulation tissue is an important healing step in the proliferation phase of wound healing. Wounds left uncovered and open to the air develop a layer of eschar, or scab, over the granulation tissue. Although this layer of dried wound exudate and dead cells protects the wound from environmental contamination, it also prevents keratinocyte migration so that reepithelialization cannot occur. Reepithelialization may thus be accelerated by preserving an optimally moist environment.
5. Collagen repair starts in the proliferative phase and extends into the final remodeling phase. Remodeling of the ECM occurs as granulation tissue is reduced via apoptosis. [7] Collagen is constantly being synthesized and degraded in order to gain strength through the reorientation of collagen fibers, as wound tissue approaches full strength. Collagen deposition in normal wound healing reaches a maximum by 21 days after the wound is created; however, it may take up to 2 years or more for a scar to completely form. If the remodeling process is disrupted, the equilibrium between ECM deposition and degradation is lost, leading to the development of fibrosis and excessive scarring.[8] Scar tissue contains fewer cells than normal skin and never achieves the same tensile strength as the original skin it has replaced.[9]

B. Nonsurgical
   1. Cleansing
      a. Use minimal mechanical force when cleansing with gauze.
      b. Use enough irrigation pressure to enhance cleansing without causing trauma to viable tissue. Wound irrigation at a pressure of 4 to 15 psi (pounds per square inch) is recommended.[10]
      c. Use normal saline/wound cleansers.
      d. Avoid long term use of antiseptics as it delays healing due to cytotoxic properties.
      e. Use hydrotherapy for management of large pressure sores with significant exudate or necrotic tissue with a pulse lavage system. Whirlpool therapy has fallen out of favor due to cross-contamination, size of equipment, and necessity of extensive clean up after each use.

2. Debride devitalized tissue or eschar from pressure ulcers is a well-accepted practice to decrease bacterial count and promote healing.[11]
3. Dressings
   a. Ensure that ulcer bed is moist and surrounding intact skin is dry.
   b. Schedule dressing changes.
   c. Use dressing that will control exudate but not desiccate ulcer bed or macerate surrounding tissue.
   d. Loosely fill pressure ulcer cavities with dressing material to avoid dead space; avoid over-packing.
   e. Monitor all dressing placements.
   f. Perform dressing changes on schedule based on individual, ulcer and condition of dressing.
   g. Consult dressing manufacturer for general information.
4. Electrical stimulation combined with standard wound interventions for the treatment of severe (Grade III or IV) pressure ulcers can be helpful if standard care alone is not being successful. The mechanisms by which ES promotes wound healing are not fully understood, leading to a need to optimize delivery of treatment.[12]The addition of ES was recommended by the AHCPR (Agency for Health Care Policy and Research) Clinical Practice Guidelines in 1994. Three separate meta-analyses [13][14] [15]have attempted to consolidate the many varying clinical reports on the success of this technique. A recent study using anodal and cathodal ES administered 50 minutes a day, 5 times a week, showed that ES as a treatment strategy was feasible in a clinical practice to improve periwound skin blood flow and promote healing of Stage 2 to Stage 4 pressure sores in patients with neurological injuries.[16] A Canadian review of its benefit noted that it was safe, had low quality evidence of benefit and would increase cost to health care system.[17]
5. Negative pressure wound therapy (NPWT) is based on the theory that the negative pressure facilitates drainage of wound exudates and enhances wound healing through a number of mechanisms.[18] [19]
   a. NPWT is proposed to decrease the bacterial load and edema while concurrently promoting an improved local circulation and increasing granulation.
   b. NPWT devices consist of a suction pump with foam and occlusive dressing to create negative pressure on the wound being treated. Despite the lack of official guidelines on the use of NPWT, it has been widely used in clinical practice and there have been consensus reports. [20].[21]
   c. NPWT is indicated when the following clinical criteria are met: Anatomical surfaces that allow a tight seal; Adequately prepared wounds, for example, debrided, free of eschar, and necrotic materials; adequate blood supply to the wound; excessive wound drainage cannot be managed with routine dressings; patient compliance with the device.
   d. Effective NPWT will produce a response within 2 to 4 weeks. Discontinuation is recommended if there is less than 30% wound size reduction after 4 weeks.
   e. Despite its popularity, there has been a struggle to provide scientific evidence to support the use of NPWT for wound healing. A consensus review in 2011 showed evidence base is strongest for the use of NPWT in nonischaemic diabetic foot ulcers, followed by pressure sores and weakest in venous leg ulcers. [22]

6. Therapeutic ultrasound is a deep heating modality that is commonly used for pressure ulcer healing. Its deep heating property is theorized to improve the vascularity of the wound tissues, thus improving healing. However, there is only limited evidence from clinical trials[23].
7. Electromagnetic Therapy has been shown to increase the blood flow, collagen formation, and also granulocyte infiltration in both in vitro and animal models to induce healing, however, clinical trial evidence is again lacking. [3]
8. Hyperbaric oxygen therapy (HBOT) is not well studied as a treatment option for pressure sores. In a 2015 Cochrane review there was a short term but not a long term benefit in the treatment of diabetic foot ulcer, but the authors noted various flaws in design and/or reporting that decreased confidence in the results. The felt that more and better designed trials were needed to properly evaluate HBOT in people with chronic wounds. The review article did not find any studies that treated pressure sores with HBOT.[24] In another review by the British medical journal no studies were found that addressed HBOT and pressure sores.[25]

6. Reassessment
   a. Monitor on a scheduled basis, document changes, and modify treatment if no healing over a 2-4 week period.
   b. Document status of sore and any changes to pressure ulcer management at least once weekly.
   c. Review individual risk factors.
      1) Impaired mobility is commonly due to paralysis or other concurrent medical conditions, leading to an increased risk of pressure ulcer development, especially in the hospital setting,
      2) Nutritional status can be measured by various means: food intake, body weight, body mass index (BMI), serum total protein, albumin, and serum prealbumin. Serum albumin level less than 3.5 mg/dL has also been associated with pressure ulcer development [26] Serum prealbumin has a shorter half-life than serum albumin, and therefore, is a more sensitive indicator for measuring nutritional status.
      3) Anemia has been identified as a risk factor in both the general[27]and the spinal cord literature[28] [29]Hemoglobin level below 12.0 to 14.0 g/dL has been associated with the development of pressure ulcers.[30]
      4) The local environment at the skin-support interface, termed the microenvironment, has a localized effect on the risk to pressure ulcer development. This umbrella term covers several factors including temperature, moisture, and acidity.
      5) Skin temperature provides an indicator of tissue perfusion. It has been found that immobile patients do not recover preloading skin temperatures after a period of prolonged loading. [31]However, elevated body temperatures will raise metabolic activity increasing the need for cellular oxygen. Animal studies have found that deep tissue damage occurs at 10° above room temperature whereas even higher temperatures caused both cutaneous and subdermal damage.[32] In some patient populations, such as those with SCI, dysfunctional temperature regulation may produce abnormal variations in microrenvironmental temperature.

6) Moisture from sweat or incontinence will both super-hydrate the epidermis and alter the acidity. Skin maceration will both soften the stratum corneum and increase the friction coefficient of the epidermis, which promotes adhesion of the skin to the support surface and increases shear, easy sloughing, and ulceration. The net effect is rapid destruction of the epidermis and reduced antibacterial properties. [33]

d. Evaluate healing process with instrument or other measurement standards.

C. Surgical referral is recommended for patients with complex, deep, stage III with undermining tracks or stage IV pressure ulcers that do not respond to standard wound care.techniques.

1. Preoperative care
   a. Local wound infection
   b. Nutritional status
   c. Bowel regulation
   d. Severe spasms/contractures
   e. Comorbid conditions
   f. Previous ulcer surgery
   g. Smoking cessation
   h. Osteomyelitis management
   i. Urinary tract infection (UTI) treatment
   j. Heterotopic ossification assessment
   k. Anemia management
2. Postoperative care
   a. Positioning
   b. Air-fluidized bed
   c. Sitting protocol after bed rest
   d. Unloading of surgical area for 4-8 weeks
   e. Patient education to prevent recurrence of pressure ulcer

D. Complications of pressure ulcers

1. Non-surgical
   a. Tissue/bone infection
      1) Identify presence of bone tissue in ulcers not responding to treatment.
      2) Obtain a tissue/bone biopsy for culture if necessary.
   b. Immobility caused by bedrest and pressure ulcer management. Address:
      1) Nutritional deficiencies
      2) Dehydration
      3) Decreased range of movement (ROM)
      4) Deconditioning
   c. Manage hypergranulation tissue which can impede healing.
   d. Psychosocial impacts of pressure ulcers and associated immobility. The patient may need the following:
      1) Vocational rehab
      2) Peer counseling/support groups
      3) Psychotherapy/family therapy
2. Surgical
   a. Infectious disease complications of surgical intervention
   b. Wound dehiscence and/or wound separation after surgical closure

c. Risk of delayed infection and abscess development
d. Development of hematoma or seroma

III. Risk factors, prevention and nutrition
A. Risk Factors: Assess and document risk in admission and reassess on routine basis; assess demographic, physical/medical and psychosocial risk factors associated with prevention.
B. Prevention
1. Avoid prolonged immobilization.
2. Assist with pressure relief.
3. Use intraoperative pressure reduction strategies.
4. Avoid improper seating position in wheelchair or use of improper wheelchair cushion.
5. Conduct daily visual and tactile inspections of skin especially at the ischium, sacrum/coccyx, trochanters and heels.
6. Turn and reposition in the bed every 2 hours, avoiding stretching/folding of soft tissues and prevent shearing of skin with reposition; avoid side-lying on trochanter
7. Environmental support: pressure reducing support surfaces, prevent moisture accumulation on or around the skin, avoid temperature elevation of the skin, use pillows or cushions for unloading bony prominences (do not use donut-type devices as they will surround the sore and cut off blood supply to the area in the middle of the donut); use supportive surfaces for the bed or the wheelchair.
8. As well as an appropriate wheelchair, an effective cushion is essential to provide a seating system that will minimize the risk for pressure ulcer development. There are a wide and continuously increasing range of pressure relief cushions available. The main classes of pressure relief cushions foam, viscoelastic gel, or flotation with air. Some cushions will combine two or more classes of material in order to achieve improved pressure relief properties, in combination with postural stability. USA TechGuide is an online resource maintained by the United Spinal Association that provides complete and constantly updated reviews of currently available pressure relief cushions. [34]Donut-rings should not be used as pressure relief cushions because, although pressure is relieved at the center of the ring, pressure distribution around the perimeter leads to high regional pressures and blood flow occlusion, thus exacerbating the overall risk.
9. Static support mattresses are appropriate for patients without a pressure ulcer or who can be positioned so that no load is applied to the pressure ulcer at any time. Dynamic mattresses include low air loss mattresses and air-fluidized beds, and are frequently only used for patients with existing pressure ulcers, due primarily to high usage costs. However, all immobile patients are at an increased risk of pressure ulcer development and the recommended 2-hourly turning schedule for pressure relief sometimes poses a staffing challenge. If loading over an existing pressure ulcer cannot be avoided at all times then a dynamic support mattress should be used.
10. Use an individualized pressure relief technique and a pressure reducing seating system. Conduct pressure relief technique every 15 minutes when sitting in a wheelchair. This can be done using the arms for a side or forward leaning technique or if unable a manual versus power weight shift system can be used with either a tilt or recline system or an alternating pressure cushion.
11. Gluteal Neuromuscular Electrical Stimulation System can regularly stimulated muscles to increase the health of the muscle and surrounding soft tissues. Bogie et al. have

investigated the use of a 4-channel gluteal electrical stimulation system (GSTIM), specifically designed to decrease risk factors associated with pressure ulcer development for individuals with SCI.[35] Subjects with a GSTIM system showed statistically significant decreases in ischial region pressure over time, together with increased gluteal cross-sectional area and enhanced regional blood flow. Sacral nerve root stimulation or surface FES can induce sufficient gluteus maximus contraction and significantly reduce ischial pressure.[36]

12. Minimizing bedrest for pressure ulcer treatment is recommended because prolonged bedrest can lead to many complications, thus effective clinical management without bedrest for patients with pressure ulcers is desirable. Prone trolleys allow patients to gain some mobility but provide only limited function and mobility in the community environment. Standing wheelchairs or standing frames can provide full, extended pressure relief over pelvic region wounds, thus allowing remobilization of individuals with existing pressure ulcers. Standing wheelchairs also have multiple systemic health benefits including increases in lower extremity range of motion, upper extremity strength, bladder, and other systemic functions together with decreased spasm and pain.[37]
13. Dynamic wheelchairs and cushions provide an alternative approach to a full standing wheelchair. A dynamic configuration wheelchair varies individual system components in order to relieve pressure over areas of tissue breakdown. [38]Dynamic wheelchair cushions provide a means to provide automatic weight-shifting due to cyclic inflation and deflation of air cells in high-load regions and may provide benefits similar to those achieved using an advanced wheelchair system.

C. Nutrition through dietary intake
   1. Anthropometric measurements
   2. Labs
      a. Prealbumin
      b. Albumin
      c. Total protein (TP)
      d. Hemoglobin (HgB)
      e. Transferrin
      f. Total lymphocyte count
   3. Adequate nutritional intake to meet needs
      a. Calories
      b. Protein
      c. Micronutrients (zinc, Vitamin C, A and E)
      d. Appropriate fluids

This guideline was developed to improve health care access in Arkansas and to aid health care providers in making decisions about appropriate patient care. The needs of the individual patient, resources available, and limitations unique to the institution or type of practice may warrant variations.

Guideline Developers
Guideline developed by Stephanie Pulikkottil Joseph, MD, in collaboration with the TRIUMPH team led by Thomas Kiser, MD, and Rani Lindberg, MD.

Reviewed and updated by Thomas Kiser, MD April 2018.

Selected References

[1] The National Pressure Ulcer Advisory Panel. NPUAP Pressure Ulcer Stages/Categories. 2016. http://www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-injury-stages/ (Accessed 3/31/2018)

[2] Pressure Ulcers - *Chester H. Ho and Kath Bogie DELISA'S PHYSICAL MEDICINE & REHABILITATION: PRINCIPLES AND PRACTICE - 5th Ed. (2010)*

[3] Eming SA, Brachvogel B, Odorisio T, et al. Regulation of angiogenesis: Wound healing as a model. *Prog Histochem Cytochem*. 2007;42:115-170.

[4] Braddock M, Campbell CJ, Zuder D. Current therapies for wound healing: electrical stimulation, biological therapeutics, and the potential for gene therapy. *Int J Dermatol*. 1999;38:808-817.

[5] Moore K. The scientific basis of wound healing. *Adv Tissue Bank*. 2001;5:379-397.

[6] Anderson JM, Gristina AG, Hanson SR, et al. *Host Reactions to Biomaterials and Their Evaluation*. San Diego, CA: Biomaterials Science Academic Press, Inc.; 1996:165-173.

[7] Desmouliere A, Redard M, Darby I, et al. Apoptosis mediates the decrease in cellularity during the transition between granulation tissue and scar. *Am J Pathol*. 1995;146(1):56-66.

[8] Lorena D, Uchio K, Costa AM, et al. Normal scarring: importance of myofibroblasts. *Wound Repair Regen*. 2002;10(2):86-92.

[9] Clark RAF. *The Molecular and Cellular Biology of Wound Repair*. 2nd ed. New York, NY: Plenum Press; 1996.

[10] Rodeheaver GT, Pettry D, Thacker JG, et al. Wound cleansing by high pressure irrigation. *Surg Gynecol Obstet*. 1975;141(3):357-362.

[11] Rodeheaver GT. Pressure ulcer debridement and cleansing: a review of current literature. *Ostomy Wound Manage*. 1999;45(suppl 1A):80S-85S; quiz 86S-87S.

[12] Ennis WJ, Lee C, Gellada K, Corbiere TF, Koh TJ. Advanced Technologies to Improve Wound Healing: Electrical Stimulation, Vibration Therapy, and Ultrasound-What Is the Evidence?

[13] Gardner SE, Frantz RA, Schmidt FL. Effect of electrical stimulation on chronic wound healing: a meta-analysis. *Wound Repair Regen*. 1999;7(6):495-503.

[14] Cullum N, Nelson EA, Flemming K, et al. Systematic reviews of wound care management: (5) beds; (6) compression; (7) laser therapy, therapeutic ultrasound, electrotherapy and electromagnetic therapy. *Health Technol Assess*. 2001;5(9):1-221.

[15] Akai M, Kawashima N, Kimura T, et al. Electrical stimulation as an adjunct to spinal fusion: a meta-analysis of controlled clinical trials. *Bioelectromagnetics*. 2002;23(7):496-504.

16 Polak A, Kucio C, Kloth LC, Paczula M, Hordynska E, Ickowicz T, Blaszczak E, Kucio E, Oleszczyk K, Ficek K, Franek A. A Randomized, Controlled Clinical Study to Assess the Effect of Anodal and Cathodal Electrical Stimulation on Periwound Skin Blood Flow and Pressure Ulcer Size Reduction in Persons with Neurological Injuries. Ostomy Wound Manage. 2018 Feb;64(2):10-29.

[17] Lambrinos A, Falk L, Ali A, Holubowich C, Walter M. Electrical Stimulation for Pressure Injuries: A Health Technology Assessment. Ontario Health Technology Assessment Series; Vol. 17: No. 14, pp. 1–106, November 2017.

[18] Morris GS, Brueilly KE, Hanzelka H. Negative pressure wound therapy achieved by vacuum-assisted closure: evaluating the assumptions. *Ostomy Wound Manage*. 2007;53(1):52-57.

[19] Orgill DP[1], Bayer LR. Negative pressure wound therapy: past, present and future. Int Wound J. 2013 Dec;10 Suppl 1:15-9.

[20] Sibbald RG, Mahoney J; V.A.C. Therapy Canadian Consensus Group. A consensus report on the use of vacuum-assisted closure in chronic, difficult-to-heal wounds. *Ostomy Wound Manage*. 2003;49(11):52-66.

[21] Gupta S. Guidelines for managing pressure ulcers with negative pressure wound therapy. *Adv Skin Wound Care*. 2004;17 (suppl 2):1-16.

[22] S. Vig, C. Dowsett, L. Berg, C. Caravaggi, P. Rome, H. Birke-Sorensen, A. Bruhin , M. Chariker, M. Depoorter, R. Dunn, F. Duteille, F. Ferreira, J.M. Francos Martı´nez, G. Grudzien, D. Hudson, S. Ichioka, R. Ingemansson, S. Jeffery, E. Krug, C. Lee, M. Malmsjo, N. Runkel, *, International Expert Panel on Negative Pressure Wound Therapy [NPWT-EP], R. Martin, J. Smith. Evidence-based recommendations for the use of negative pressure wound therapy in chronic wounds: Steps towards an international consensus. Journal of Tissue Viability (2011) 20, S1-S18.
[23] Baba-Akbari Sari A, Flemming K, Cullum NA, et al. Therapeutic ultrasound for pressure ulcers. *Cochrane Database Syst Rev*. 2006;3: CD001275.
[24] Kranke P, Bennett MH, MartynSt James M, Schnabel A, Debus, SE, Weibel, S. Hyperbaric oxygen therapy for chronic wounds. Cochrane Database of Systematic Reviews. 6, 2015.
[25] Reddy M. Pressure ulcers: treatment. BMJ Clin Evid. 2015 Dec 11;2015. pii: 1901.
[26] Blaylock B. A study of risk factors in patients placed on specialty beds. *J Wound Ostomy Continence Nurs*. 1995;22(6):263-266.
[27] Bansal C, Scott R, Stewart D, et al. Decubitus ulcers: a review of the literature. *Int J Dermatol*. 2005;44(10):805-810.
[28] Scivoletto G, Fuoco U, Morganti B, et al. Pressure sores and blood and serum dysmetabolism in spinal cord injury patients. *Spinal Cord*. 2004;42(8):473-476.
[29] Fuoco U, Scivoletto G, Pace A, et al. Anaemia and serum protein alteration in patients with pressure ulcers. *Spinal Cord*. 1997;35(1):58-60.
[30] Rochon PA, Beaudet MP, McGlinchey-Berroth R, et al. Risk assessment for pressure ulcers: an adaptation of the National Pressure Ulcer Advisory Panel risk factors to spinal cord injured patients. *J Am Paraplegia Soc*. 1993;16(3):169-177.
[31] Baldwin KM. Transcutaneous oximetry and skin surface temperature as objective measures of pressure ulcer risk. *Adv Skin Wound Care*. 2001;14(1):26-31.
[32] Kokate JY, Leland KJ, Held AM, et al. Temperature-modulated pressure ulcers: a porcine model. *Arch Phys Med Rehabil*. 1995;76(7):666-673.
[33] Reger SI, Ranganathan VK, Sahgal V. Support surface interface pressure, microenvironment, and the prevalence of pressure ulcers: an analysis of the literature. *Ostomy Wound Manage*. 2007;53(10):50-58.
[34] http://www.usatechguide.org/cushion_reviews.php. Accessed April 8, 2018..
[35] Bogie KM, Reger SI, Levine SP. Therapeutic applications of electrical stimulation; wound healing and pressure sore prevention. *Assist Technol*. 2000;12:50-66.
[36] Liu LQ, Ferguson-Pell M. Pressure Changes Under the Ischial Tuberosities During Gluteal Neuromuscular Stimulation in Spinal Cord Injury: A Comparison of Sacral Nerve Root Stimulation With Surface Functional Electrical Stimulation. Arch Physical Medicine and Rehabilitation 2015;96:620-6.
[37] Dunn RB, Walter JS, Lucero Y, et al. Follow-up assessment of standing mobility device users. *Assist Technol*. 1998;10(2):84-93.
[38] Makhsous M, Rowles DM, Rymer WZ, et al. Periodically relieving ischial sitting load to decrease the risk of pressure ulcers. *Arch Phys Med Rehabil*. 2007;88(7):862-870.