# Initial Management of Minor Burns

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**Abstract:**

Burns are among the most common injuries in children and adults. Most burns cover less than 5-10 percent of the total body surface area and the management focuses on the local care of the burn wound. Increased understanding of the cellular and molecular mechanisms of cutaneous wound healing has led to the development of new therapeutic agents and wound dressings. While burn specialists play a pivotal role in the management of burn victims, most minor burns are initially managed by emergency and primary care and physicians. This paper will review the pathophysiology and management of the burn wound.

**MeSH Words**: burns, wound healing, synthetic dressings, antimicrobials

## Introduction

Burns are one of the most common injuries in both children and adults.[1,2] Over the last few decades advances in resuscitation and critical care have led to a significant reduction in the morbidity and mortality of burn victims.[3] Since most burns cover less than 10 percent of the total body surface area[4] the management of burns usually centers on the care of the burn wound. Increased understanding of the cellular and molecular mechanisms of cutaneous wound healing[5] has led to the development of new therapeutic agents and wound dressings. While burn specialists play a pivotal role in the management of burn victims, most minor burns are initially managed by emergency and primary care and physicians.

The primary goals of acute burn wound management are prevention of infection and the promotion of optimal wound closure by reepithelialization which results in minimal aesthetic and functional impairment. The current review is intended for emergency physicians, and other health care practitioners who care for minor burns. Fluid resuscitation and management of inhalation injury and multiorgan failure will not be discussed in this article.

| Depth | Histology | Appearance and Sensitivity | Outcome | Treatment |
|---|---|---|---|---|
| 1st degree | Epidermis only | Erythema, blanches with pressure, sensitive to touch | Heals without scarring within several days | Topical or systemic anti-inflammatory agents and/or analgesics |
| 2nd degree<br>Superficial | Epidermis and superficial dermis; most epidermal appendages intact | Erythema, blisters, moist, elastic; blanches with pressure, sensitive to touch | Heals within 1-3 weeks with minimal scarring | Topical antimicrobial agents or specialized dressings |
| Deep | Epidermis and most of dermis; many epidermal appendages destroyed | White appearing with erythematous areas, dry, waxy, less elastic, reduced blanching and sensitivity to touch | Healing last >3 weeks, often with significant scarring and contractures | Enzymatic débridement or surgical excision followed by grafting |
| 3rd degree | Epidermis and entire dermis; destruction of all epidermal appendages | White, tan or charred, thrombosed vessels, dry and leathery, does not blanch; anesthetic | Does not heal; severe scarring and contractures | Surgical excision followed by grafting |

**Table 1. Burn Depth Classification**

## Epidemiology and Etiology of Burns

More than 1 million burn injuries occur each year in the US[2] resulting in over 700,000 emergency department visits, 45,000 hospital admissions, and 4,500 deaths from burns per year.[6] Most burns are minor involving less than 10 percent of the total body surface area.[3,4] Most fatal burns are the result of residential fires resulting in nearly one death every 2 hours and one injury every 23 minutes.[7] The majority of burns in children result from scalding while those in adults are mostly secondary to direct contact with flame, hot objects, chemicals, or electricity.

## Pathophysiology of Burns

The primary injury in burns is the result of irreversible tissue necrosis at the center of the burn due to exposure to heat, chemicals, or electricity. The extent of this injury is dependent on the temperature (or concentration) and the duration of exposuret.[8,9] Surrounding the central zone of necrosis is a zone of ischemia in which there is a reduction in the dermal microcirculation.[10] This ischemic zone may progress to full necrosis over the next few days unless the ischemia is reversed. At the periphery of the burn is a third zone of hyperemia characterized by a reversible increase in blood flow. The pathogenesis of reduced dermal microcirculation in the zone of ischemia is complex and incompletely understood.[11] Activation of the clotting and complement systems by the initial insult is followed by the local recruitment of inflammatory cells with the release of a large number of inflammatory mediators[12] that increase vascular permeability and result in transudation of large amounts of fluid and protein into the interstitial space.[13] A great deal of research has been directed to developing methods aimed at attenuating the inflammatory response. Antioxidants such as glutathione and xanthine oxidase inhibitors as well as the free radical scavengers vitamin C and vitamin E have been used with some success to treat burns in both animals and humans.[14, 15] In addition, topical and systemic treatments of burns with anti-inflammatory agents such as ibuprofen and corticosteroids have also met with a variable degree of success.[16, 17] However, there is no evidence to support the routine used of any of the above agents for minor burns.

## Burn Classification

One of the major determinants of burn therapy and prognosis is the severity of the injury. Burns are classified according to their depth and total

**Table 2. Burn Unit Referral Criteria[26]**

1. Partial thickness burns greater than 10% of TBSA.
2. Burns involving the face, hands, feet, genitalia, perineum, or major joints.
3. Third-degree burns in any age group.
4. Electrical burns, including lightning injury.
5. Chemical burns.
6. Inhalation injury.
7. Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality.
8. Any patient with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity and mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be initially stabilized in a trauma center before being transferred to a burn unit. Physician judgment is necessary in such situations and should be in concert with regional medical control plan and triage protocols.
9. Burned children in hospitals without qualified personnel or equipment for the care of children.
10. Burn injury in patients who will require special social, emotional, or long-term rehabilitative intervention.

*Excerpted from*: Guideline for the Operations of Burn Units (pp. 55-62). Resources for Optimal Care of the Injured Patient: 1999, Committee on Trauma, American College of Surgeons.

surface area (Table 1). First degree burns are limited to the epidermis and are characterized by erythema and pain. They heal within several days without leaving any scars. Second degree burns involve all of the epidermis as well as parts of the underlying dermis. Second degree burns are further classified based on the depth of dermal involvement. Superficial partial thickness burns involve the upper layers of the dermis only and are characterized by the appearance of blisters and weeping. These injuries are sensitive to the touch, are painful, and blanch with pressure. Since epidermal appendages are preserved, these wounds usually heal within 2-3 weeks with minimal scarring. In contrast, deep partial thickness wounds involve deeper layers of the dermis and are often difficult to distinguish from third degree or full thickness burns that involve the entire thickness of the dermis. Deep dermal burns are covered with a layer of injured dermis (white or red in appearance) that does not blanch with pressure, and usually do not heal for at least 3 weeks. This often results in significant scarring and contractures, especially in children. Full thickness burns may appear as thick brown or tan and have a leathery texture.

The extent of injury is best described using the percentage of the total body surface area that sustained second or third degree burns. This may be estimated using standardized body charts such as the Lund Browder chart[19] that takes into account age-related changes in surface area. In patients 10 years and older the "rule of nines" can be used. With small burns the area can be estimated by comparing it to the palm of the patient's hand that represent approximately 1% of the TBSA over a wide range of ages.[21, 22] Errors in estimating the burn size are common when physicians "guess" the size, often resulting in overestimation by 100 percent or more.[23] In general, burns covering more than 10-15 percent of the total body surface area require fluid resuscitation and transfer to a burn unit.

Classification of burns into minor and major injuries depends on their location, depth and surface area (Table 1). Other considerations include the patient's age and the presence of comorbid conditions or associated injuries. Major burns should be referred to a burn unit.[24]

**Burn First Aid**

The depth of the burn can be reduced by immediate cooling with cold tap water.[25] Cooling of the burn also reduces pain and improves outcome.[26] Direct contact with ice or ice water, however, should be avoided. Copious irrigation, after removal of any pieces of metal or dry powder, is especially important with chemical burns. Blisters should be left intact and any non-adherent garments and jewelry should be removed. The burns should then be covered with a clean cloth or dressing and burned extremities should be elevated.

**Table 3. Burn Dressings**

| Type of Dressing | Advantages | Disadvantages | Examples |
|---|---|---|---|
| **Absorptive**<br>Gauze<br>Foams | Absorbent, non-adherent, expandable | Limited to small and regular shaped wounds | Allevyn (Smith & Nephew, Largo, FL)<br>Biopatch (Johnson & Johnson Medical, Arlington, TX)<br>Curafoam (The Kendall Company, Mansfield, MA)<br>Flexzan (Dow Hickham, Sugar Land, TX)<br>Lyofoam (Convatec, Princeton, NJ)<br>Mepilax (Molnlycke Health Care, Gotenberg, Sweden)<br>Vigifoam (Bard, Murray Hill, NJ) |
| **Occlusive** | | | |
| Polyurethane Films | Waterproof, transmit $O_2$, $CO_2$, and water vapor, transparent, thin | Non-absorptive, frequent dressing changes, dressing removal may disrupt neo-epidermis, requires intact surrounding skin | Bioclusive (Johnson & Johnson Medical, Arlington, TX)<br>Blisterfilm (The Kendall Company, Mansfield, MA)<br>Carrafilm (Carrington, Irving, TX)<br>Mefilm (Molnlycke Health Care, Gotenberg, Sweden)<br>Op-Site (Smith & Nephew, Largo, FL)<br>Tegaderm (3M Healthcare, St. Paul, MN)<br>Transeal (DeRoyal, Powell, TN) |
| Hydrocolloids | Absorbs wound exudates, limited moisture and gas transmission, protective cushioning | Opaque, bulky, | Comfeel (Coloplast, Holtedam, Denmark)<br>Cutinova (Smith & Nephew, Largo, FL)<br>DuoDerm (Convatec, Princeton, NJ)<br>Hydrocol (Dow Hickman, Sugar Land, TX)<br>NuDerm (Johnson & Johnson Medical, Arlington, TX)<br>Tegasorb (3M Healthcare, St. Paul, MN) |
| Alginates | Absorptive | Frequent dressing changes, less-studied than other dressings | Algiderm (Bard, Murray Hill, NJ)<br>Algosteril (Johnson & Johnson Medical, Arlington, TX)<br>Carasorb (Carrington, Irving, TX)<br>Curasorb (The Kendall Company, Mansfield, MA)<br>Kaltostat (Convatec, Princeton, NJ)<br>Melgisorb (Molnlycke Health Care, Gotenberg, Sweden)<br>SeaSorb (Coloplast, Holtedam, Denmark)<br>Sorbsan (Dow Hickman, Sugar Land, TX) |
| Hydrogels | Rehydrate dry wounds | Non-absorptive | Curagel (The Kendall Company, Mansfield, MA<br>Flexderm (Dow Hickman, Sugar Land, TX<br>FlexiGel (Smith & Nephew, Largo, FL)<br>Nu-gel (Johnson & Johnson Medical, Arlington, TX)<br>Vigilon (Bard, Murray Hill, NJ) |
| Composites | Combine durability and barrier function, less frequent dressing changes | Requires immediate débridement, often under anaesthesia | Alloderm (LifeCell, Branchburg, NJ)<br>Apligraf (Organogenesis Inc., Canton, MA)<br>Biobrane (Dow Hickman, Sugar Land, TX)<br>Integra (Ethicon Inc., Somerville, NJ)<br>TransCyte (Smith & Nephew, Largo, FL) |

**History and Physical Examination**

Careful attention to the details surrounding the injury is required in order to exclude inhalation injury and other injuries, such as head, neck or torso trauma that result from falling. Injury within a closed space and exposure to toxic fumes should heighten the suspicion for inhalation injury. With children, an inconsistent or implausible history should prompt communication with child protective services to rule out child abuse. The presence of comorbid conditions that may affect the healing process (such as diabetes mellitus and other immunocompromising conditions) should be sought. Tetanus immunization status and the presence of drug allergies should also be obtained.

The presence of facial swelling, hoarseness, stridor, singed nasal hair, carbonaceous sputum, respiratory distress or an altered mental status suggest the presence of inhalation injury and should prompt rapid referral to a burn center. The presence of circumferential burns should be noted early in the evaluation since this may result in a compartment syndrome necessitating an emergent escharotomy (incision of the eschar with cautery or surgical blade). A compartment syndrome is suggested by the presence of severe pain (especially with passive motion), numbness, weakness, decreased capillary refill or pulses. A measured compartment pressure of greater than 30 mmHg requires immediate consultation for escharotomy.

While sometimes inaccurate, careful examination by an experienced physician remains the most reliable method for determining burn depth.[27] Other methods that have been evaluated to help determine burn depth, such as doppler flow meters , thermography, fluorescence of intravenous dyes, MRI, and wound biopsy, are still under investigation.[27] Since burns are dynamic injuries, close follow-up is often required before determining ultimate depth. If burn depth is unclear, prompt referral to a burn specialist is necessary.

**Diagnosis and Treatment of Burn Wound Infections**

While relatively rare, local infection is suggested by the presence of increasing erythema, pain, systemic fever, and purulence. This may result in conversion of a superficial burn to a deep burn. Patients with suspected infection should be started on an oral antibiotic that covers *staphylococcus aureus* and *streptococcus pyogenes* and referred to a burn specialist.

**Local Burn Wound Care**

Burns should be cleaned with a mild soap and clean water or saline. Gentle removal of ruptured blisters and any residual non-viable epidermis or debris should be performed using gauze or a non abrasive sponge. The authors prefer leaving intact blisters alone since they help maintain a moist wound environment that enhances reepithelialization and angiogenesis.[28-30] Numerous studies indicate that routine removal of blisters or the necrotic epidermis delays healing of burns.[31-34] One study also demonstrated that leaving blisters intact reduces bacterial colonization.[35] Some prefer to remove large or tense blisters since they tend to burst under less than ideal conditions and may serve as media for microbial proliferation.

Routine administration of systemic antibiotics is not recommended. Patients with prior tetanus immunoprophylaxis require tetanus toxoid if more than 5 years have elapsed since their last booster. Patients in whom adequate immunoprophylaxis cannot be verified should receive passive immunization with 250 units of intramuscular tetanus immune globulin in addition to active immunization.

*First-Degree Burns*

First-degree burns do not require wound dressings or topical antimicrobial agents. Pain may be managed with oral or topical analgesics or anti-inflammatory agents. Itching can be treated with cool compresses or oatmeal based solution supplemented with oral anti-histamines, especially before bedtime, when indicated.

*Superficial Second-Degree Burns*

Superficial burns can be treated using an open or closed method. With the open method of wound care antimicrobial agents are used to minimize bacterial proliferation and fungal colonization. The routine use of antimicrobial agents has resulted in a substantial decrease in the mortality associated with burn injuries.[36] Because of its relatively low toxicity and ease of use, silver

sulfadiazine is the most widely used topical antimicrobial agent presently.[37] Silver sulfadiazine has wide antimicrobial coverage, good eschar penetration and its application is comfortable. It rarely may result in transient leukopenia and allergy due to its sulfa component. It should be avoided in newborns and pregnant women due to the risk of kernicterus. *In vitro* studies suggest that the use of silver sulfadiazine retards healing. Its clinical efficacy can be attributed to its antibacterial and occlusive effects. Mafenide acetate (Sulfamylon), while painful on application to second degree burns, has good eschar penetration and is well suited for treating burns over cartilage such as the ears and nose. Mafenide is also a good agent for focal full thickness burns and for minor burns that have been neglected prior to initial treatment. Burns of the face or neck can be treated with a topical antimicrobial agent such as bacitracin. Since their effect is limited, most topical antimicrobial agents should be applied twice daily as a thin layer after gently washing off the burn wound with a mild soap under running water. After applying a topical antimicrobial agent the wounds can be covered with multiple layers of gauze to absorb the wound transudate and prevent contamination of the burn from external sources. The dressing also splints the wound and reduces painful stimuli. Soaking the dressings in saline may facilitate their removal during dressing changes. The open method is best suited for large, heavily contaminated, or weeping burns with heavy exudation.

With the closed wound care method, the burn surface is washed and then covered with one of many synthetic or biological occlusive dressings (Table 3) creating a moist wound environment. This method is best suited for smaller burns and one's with little exudation. While many wound dressings are available there is no evidence supporting one over the others. Certain of the occlusive dressings appear to enhance re-epithelialization and may improve the cosmetic outcome.[38] When properly used, occlusive dressings do not increase the risk of wound infection.[39] The most commonly used dressings include the polyurethane films[40, 41] (such as Tegaderm [3M, St. Paul, Minn.] and OpSite [Smith & Nephew, Largo, FA]) and hydrocolloids[42] (such as DuoDerm [Convatec, Skillman, NJ]). The advantage of the hydrocolloids is their ability to absorb variable amounts of transudates and their cushioning effect. Leakage of fluid from under the occlusive dressing necessitates removal of the dressing, washing the wound and reapplication.

Acticoat (Smith & Nephew, Largo, FA) is a non-adherent nanocrystalline silver coated material which has recently been introduced as a burn wound dressing. In addition to creating a moist wound-healing environment, sustained delivery of silver ions has inherent antibacterial activity. Acticoat combines the advantages of anti-microbial agents and occlusive dressings and is less painful to remove than conventional dressings.[43]

*Deep Partial Thickness Burns (deep second-degree burns)*

The outer layer of deep second degree burns is composed of exposed collagen which has been denatured. This layer must be debrided before reepithelialization can occur. The topical use of an enzymatic debriding agent such as collagenase may result in earlier reepithelialization and healing.[44] Débridement can also be accomplished with dermabrasion performed under general anesthesia by a burn specialist. Deep partial thickness burns should be covered with a topical antimicrobial agent and referred to a burn specialist.

*Full thickness (third degree) Burns*

Full thickness burns should be treated on the initial visit with a topical anti-microbial agent and a dressing. The patient should be admitted to the hospital or a Burn Center for definitive care which involves excision of the eschar and skin grafting or primary closure if the wound is sufficiently limited.

*Alternative Therapies*

A number of alternative therapies have been evaluated for burns. However, none of these agents have been evaluated in large well-designed clinical trials. In regions where more expensive and modern therapies are not available, alternative therapies may be used. "Natural" products that have been evaluated for the treatment of burns include aloe vera,[45] papaya extract,[46] boiled potato peels,[47] banana leaves[48] and crude honey.[49]

**Instructions to Patients for Outpatient Management of Minor Burns**

With the open method of treatment, burns should be gently cleaned with a mild soap and water twice daily followed by reapplication of the antimicrobial agent and a clean dressing. With the closed method, the dressing should be observed twice daily for the appearance of any underlying fluid collections. Burns involving the extremities should be elevated to reduce swelling. All patients should report to their physician or emergency department if they develop increasing pain, redness, swelling, fever, chills, or a foul smelling drainage from the wound, as this might suggest infection.

**Pain Management**

While the pain resulting from burns, especially partial thickness injuries, is usually severe, its treatment is often neglected.[50] Furthermore, pain may persist long after the initial injury and can delay full rehabilitation.[51] The pain is often exacerbated during movement and wound care. Psychological factors influenced by the circumstances surrounding the injury or the patient's ability to tolerate pain also play a role.

*Initial Treatment of Pain*

Due to their potency and relative safety, opioids are the most commonly used analgesics.[52] Their sedating effects may also be beneficial. The choice of the route of administration is dictated by the severity of the burn. With most minor burns, oral combinations of hydrocodone or oxycodone with or without acetaminophen are sufficient. The patient may require additional medication for dressing changes. The total intake of acetaminophen should not exceed 4 grams/day, and doses should be weight adjusted in children.

The efficacy of non-steroidal anti-inflammatory drugs such as ibuprofen and naproxen is often hampered by a "ceiling" effect in which higher doses do not provide a greater analgesic effect. These drugs may be useful in very small burns, but are not as effective as the opioid medications. The use of NSAIDS in the presence of impaired renal function may have a deleterious effect upon the kidneys. The risk of gastrointestinal ulceration following a burn should caution against the prolonged or excessive use of these medications. There is also an increased interest in the use of anticonvulsants, tricyclic antidepressants and membrane stabilizers for control of pain in burns, but strong evidence for their use is lacking.[53]

**Scarring and Hyperpigmentation**

The development of hypertrophic scarring, usually 6-8 weeks after injury, is one of the most problematic aspects of burn healing and may result in significant aesthetic and functional impairment. While its exact incidence is unknown, a recent report suggests that many patients with burns develop hypertrophic scarring.[54] Hypertrophic scars are raised, firm, painful, itchy and red to deep purple in color. They are characterized by excessive deposition of collagen in the dermis both by persistent inflammation and fibroplasia.[55] Unlike keloids, hypertrophic scars are limited to the original confines of the injury. Maturation of the burn scar with reduction in its bulk and redness usually occurs over the next 6-18 months. Factors that influence the development of hypertrophic scars include genetic predisposition, age, race, anatomical location, depth and presence of infection or local tension.[55]

Several methods have been used to treat hypertrophic scars including scar massage,[56] application of local pressure[57] (pressure garments, conformers, transparent face masks, casts, or splints), use of silicone materials[58] (with or without pressure), intralesional injection of corticosteroids,[59] and surgery. Silicones are thought to act via application of local pressure, hydration and occlusion. Use of local pressure and silicone is continued at least a year. Hydration of the skin with commercially available emollients may help reduce itching. Current evidence does not support routine use of topical vitamin E preparations.[60] Patients who develop hypertrophic scars should be referred to a burn specialist.

**References**


1. Department of Health, Education, and Welfare. Reports of the epidemiology and surveillance of injuries. Atlanta: Centers for Disease Control, 1982. (DHEW publication no. (HSM) 73-10001.)

2. Brigham PA, McLoughlin E. Burn incidence and medical care use in the United States: estimate, trends, and data sources. *J Burn Care Rehabil* 1996;17:95-107.

3. Saffle JR, Davis B, Williams P. Recent outcomes in the treatment of burn injury in the United States: a report from the American Burn Association Patient Registry. *J Burn Care Rehabil* 1995;16:219-232.

4. Auchinloss JM. Survey and retrospective analysis of out-patient burns. *Br J Clin Pract* 1975;29:251-253.

5. Singer AJ, Clark RAF. Cutaneous wound healing. New Engl J Med 1999;341:738-746.

6. Demling RH. Burns. *N Engl J Med* 1985;313:1389-1398.

7. Monafo WW. Initial management of burns. New Engl J Med 1996; 335:1581-6

8. Peate WF. Outpatient management of burns. *Am Fam Physician* 1992;45:1321-130.

9. American Burn Association. Available at http://www.ameriburn.org Accessed October 22, 2003.

10. Ahrens M. The US fire problem overview report: leading causes and patterns and trends. Quincy (MA): National Fire Protection Association, 2001.

11. Moritz AR, Henriques FC. Studies of thermal injury. II. Am J Pathol 1947;23:695-720.

12. Singer AJ, Berruti L, Thode HC, McClain SA. Standardized burn model using a multi-parametric histological analysis of burn depth. *Acad Emerg Med* 2000;7:1-6.

13. Jackson D. The diagnosis of the depth of burning. *Br J Surg* 1953;40:588-596.

14. Arturson G. Pathophysiology of the burn wound and pharmacological treatment: the Rudi Hermans Lecture, *Burns* 1996;22:255-274.

15. Demling R, Lalonde C. Early postburn lipid peroxidation: Effect of ibuprofen and allopurinol. *Surgery* 1990;107:85.93.

16. Arturson G, Johnson CE. Trans-capillary transport after thermal injury. *Scan J Plast Reconstr Surg* 1979;13:29-38.

17. LaLonde C, Nayak U, Demling R, et al. Aerosolized glutathione prevents the increase in plasma hydrogen peroxide and evolving lung dysfunction seen in response to smoke inhalation injury. J Burn Care Rehabil 1997;18:S70.

18. Mann R, Foster K, Kemalyan N, et al. Intravenous vitamin C and clinical burn resuscitation. *J Burn Care Rehabil* 1997;18:S87.

19. Hughes GS, Francoc SF, Means LK, Bohan DF, Caruana C, Holland M. Synergistic effects of oral nonsteroidal drugs and topical corticosteroids in the therapy of sunburn in humans. Dermatology 1992;184:54-58.

20. Pedersen JL, Moiniche S, Kehlet H. Topical glucocorticoid has no antinociceptive or anti-inflammatory effects in thermal injury. Br J Anesth 1994;72:379-382.

21. Lund CC, Browder NC. The estimate of areas of burns. *Surg Gynecol Obstet* 1944;79:352-358.

22. Nagel TR, Schunk JE. Using the hand to estimate the surface area of a burn in children. Pediatr Emerg Care 1997;13:254-255.

23. Sheridan RL, Petras L, Basha G, et al. Planimetry study of the percent of body surface represented by the hand and palm: sizing irregular burns is more accurately done with the palm. J Burn Care Rehabil 1995;16:605-606.

24. Hammond JS, Ward CG. Transfers from emergency room to burn center: errors in burn size estimate. J Trauma 1987;27:1161-1165.

25. Guidelines for the Operations of Burn Units. Resources for Optimal Care of the Injured

Patient: 1999, Committee on Trauma, American College of Surgeons.

26. Raine TJ, Heggers JP, Robson MC, London MD, Johns L. Cooling the burn wound to maintain microcirculation. J Trauma 1981;21:394-7.

27. Guidelines 2000. Cardiopulmonary resuscitation and emergency cardiovascular care: International Consensus on Science. Circulation 2000;102 (Supplement).

28. Heimbach D, Engrav L, Grube B, Marvin J. Burn depth: a review. World J Surg 1992;16:10-15.

29. Winter GD. Formation of the scab and the rate of epithelization of superficial wounds in the skin of the young domestic pig. Nature 1962;193:293-294.

30. Hinman CD, Maibach H, Winter GD. Effect of air exposure and occlusion on experimental human skin wounds. Nature 1963;200:377-378.

31. Dyson M, Young SR, Hart J, Lynch JA, Lang S. Comparison of the effects of moist and dry conditions on the process of angiogenesis during dermal repair. J Invest Dermatol 1992;99:729-733.

32. Gimbel NS, Kapetansky DI, Weissman F, Pinkus HKB. A study of epithelization in blistered burns. Arch Surg 1957;74:800-803.

33. Wheeler ES, Miller TA. The blister and the second degree burn in guinea pigs: the effect of exposure. Plast Reconstr Surg 1976;57:83.Forage AV. The effects of removing the epidermis from burnt skin. Lancet 1962;2:690-693.

34. Forage AV. The effects of removing the epidermis from burn skin. Lancet 1962;2:690-693.

35. Singer AJ, Thode HC Jr., McClain SA. The effects of epidermal débridement of partial thickness burns on infection and reepithelialization in swine. Acad Emerg Med 2000;7:114-119.

36. Swain AH, Azadian BS, Wakeley CJ, et al. Management of blisters in minor burns. BMJ 1987;295:181-

37. Monofo WW, Freedman B. Topical therapy in burns. *Surg Clinics* 1987;67:133-145.

38. Papini RPG, Wilson APR, Steer JA, McGrouther DA. Wound management in burn centres in the United Kingdom. *Br J Surg* 1995;82:505-509.

39. Nemeth AJ, Eaglstein WH, Taylor JR, Peerson LJ, Falanga V. Faster healing and less pain in skin biopsy sites treated with an occlusive dressing. *Arch Dermatol* 1991;127:1679-1683.

40. Hutchinson JJ. Prevalence of wound infection under occlusive dressing: a collective survey of reported research. *Wounds* 1989;1:123-133.

41. Neal DE, Whalley PC, Flowers MW, Wilson DH. The effects of an adherent polyurethane film and conventional absorbent dressings in patients with small partial thickness burns. Br J Clin Pract 1981;35:254-257.

42. Eaglstein WH. Experiences with biosynthetic dressings. J Am Acad Dermatol 1985;12:434-40.

43. Afilalo M, Dankoff J, Guttman A, Lloyd J. DuoDerm hydroactive dressing versus silver sulphadiazine/Bactigras in the emergency treatment of partial skin thickness burns. Burns 1992;18:313-6.

44. Tredget EE, Shankowsky HA, Groeneveld A, Burrel R. A matched-pair, randomized study evaluating the efficacy and safety of Acticoat silver-coated dressing for the treatment of burn wounds. J Burn Care Rehabil 1998;19:531-537.

45. Soroff HS, Sasvary DH. Collagenase ointment and polymyxin B sulfate/bacitracin spry versus silver sulfadiazine cream in partial thickness burns: A pilot study. J Burn Care Rehabil 1994;18:253-260.

46. Visuthikosol V, Chowchuen B, Sukwanarat Y, et al. Effect of aloe vera gel to healing of burn wound: a clinical and histologic study. J Med Assoc Thai 1995;78:403-409.

47. Starley IF, Mohammed P, Schneider G, Bickler SW. The treatment of pediatric burns using topical papaya. Burns 1999;25:636-639.

48. Subrahmanyam M. Honey dressing versus boiled potato peel in the treatment of burns: a prospective, randomized study. Burns 1996;22:491-493.

49. Gore MA, Akolekar D. Evaluation of banana leaf dressing for partial thickness burn wounds. Burns 2003; in press.

50. Subrahmanyam M. A prospective randomized clinical and histological study of superficial burn wound healing with honey and silver sulfadiazine. Burns 1998;24:157-161.

51. Singer AJ, Thode HC Jr. National analgesia prescribing patterns in Emergency Department patients with burns. Burn Care & Rehab 2002;23:361-365.

52. Laterjet J, Choinere M. Pain in burn patients. Burns 1995;21:344-348.

53. Stoddard FJ, Sheridan RL, Saxe GN, King BS, King BH, Chedekel DS, et al. Treatment of pain in acutely burned children. J Burn Care Rehabil 2002;23:135-156.

54. Pal SK, Cortiella J, Herndon D. Adjunctive methods of pain control in burns.

55. Taun T, Nichter LS. The molecular basis of keloid and hypertrophic scar formation. Molecular Medicine Today 1998;January:19-24.

56. Carr-Collins JA. Pressure techniques for the prevention of hypertrophic scars. Clin Plast Surg 1992;19:733-743.

57. Reid WH, Evans JH, Naismith RS, Tully AE, Sherwin S. Hypertrophic scarring and pressure therapy. Burns 1987;13:S29-32.

58. Van Der Kerchove E, Stappaerts K, Boeckx W, et al. Silicones in the rehabilitation of burns: a review and overview. Burns 2001;27:205-214.

59. Azad S, Sacks L. Painless steroid injection for hypertrophic scars and keloids. Br J Plast Surg 2002;55:534.

60. Baumann LS, Spencer J. The effects of topical vitamin E on the cosmetic appearance of scars. Dermatol Surg. 1999;25:311-5.

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