FACULTY DISCUSSION & STUDY GUIDE

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Burns; Difficult Soft Tissue Infections

VII.A. Pathophysiology, Resuscitation, Pulmonary Complications

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Financed with a grant from The Hoopes Foundation.

A. Resuscitation

1. What would be the hemodynamics of a 30 y.o. burn patient with a 50% body surface area burn prior to resuscitation in terms of the following: intravascular volume, red cell mass, total body water, cardiac output? (Intravascular volume would be markedly diminished because of the mechanisms outlined in question 1, red cell mass would be only slightly diminished secondary to direct red cell damage by the burn, total body water would be essentially the same as preburn except for a minimal loss due to direct burn damage, cardiac output would be markedly diminished.)
    a. What is the mechanism(s) for the above? (The loss of intravascular volume is explained by the loss of vascular system integrity and the transudation of fluid and protein into the interstitial space; cardiac output is markedly diminished in the burn patient due to not only diminished intravascular volume but also perhaps a direct negative cardiac inotropic effect of some circulating agent as a result of the burn injury.)

2. In a patient with a large, 50% burn from the waist upward, what explains the edema formation in the lower extremities during resuscitation? (A tremendous increase in permeability particularly in the venules occurs with loss of venule and capillary integrity that allows transudation of fluid and small molecular weight proteins from the intravascular system into the interstitial space. This shift creates diminished intravascular volume and increase in interstitial fluid volume that results clinically in the appearance of edema.)
    a. What are some possible explanations for this loss of integrity? (The exact mechanism is not known but the culprits appear to be histamine release from mast cells, activation of the

proteolytic cascade including kinins, and vasoactive products as prostaglandins – all of which can create not only an increase in vascular permeability but also an increase in relative intravascular hydrostatic pressure in the micro circulation.)

3. Explain the role of the alteration of Starling's forces in the production of burn edema. (The Starling equation states that in normal circumstances fluid moves from the intravascular space across the capillary membrane and into the interstitial space but that fluid is equally removed by the lymphatic system so that edema does not occur. The fluid shift in that direction is driven by the intravascular hydrostatic pressure which is greater than the interstitial hydrostatic pressure. Yet, a positive gradient exists in the opposite direction, interstitial to intravascular space by the difference between plasma colloid osmotic pressure, higher than interstitial colloid osmotic pressure. That difference is driven by a higher concentration of protein within the intravascular rather than the interstitial space. As a result of the burn injury the capillary and venule membranes become permeable to plasma proteins including albumin which then "leak" into the interstitial space thus negating, in fact, even reversing the normal difference in colloid osmotic pressure. Of course, the same dramatic increase in permeability allows fluid as well to move easily from the vascular into the interstitial space.)
    a. What impact do these pathophysiologic changes have on the fluid resuscitation regime? (The tremendous loss of vascular volume in large burns requires a large volume of administered intravascular fluids-see below. Also, the loss of capillary integrity to the passage of intravascular proteins would also imply that colloid or protein administration in the first 24 hours would be counterproductive – as well as expensive.)

4. How was the Parkland or Baxter formulas <u>initially</u> derived? (The Parkland formula, 4ml/kg/% body surface area burn with ½ administered in the first eight hours post burn, was developed by Baxter and Shires at Parkland hospital. The formula was derived as a result of both experimental and clinical or retrospective review of successful vs. unsuccessful resuscitated patients vs. the volume in fluids given.)
    a. What was the role of colloid in the Parkland formula? Why? (Colloid was given *after* the first 24 hours post burn usually as albumin and at a volume of approximately 0.5cc of colloid/kg/% body surface area burn administered with D5w to maintain urine

output. Restoration of capillary membrane integrity is necessary such that administered colloid remains intravascular, an event that may be incomplete at 24 hours post-burn.)

b. What are the current thoughts about the role of colloid in resuscitation? (Three principal schools of thought exist on the use of colloid in burn resuscitation: One that incorporates colloid from the initiation of resuscitation, another that avoids colloid altogether and resuscitates through the whole resuscitation period with crystalloid, and a third group that utilizes colloid on a selective basis after the first 24 hours.)

c. Recommended urine output? (0.5 - 1.0 cc/kg/hr in adults.)

5. What is "fluid creep" and what are the adverse effects of fluid creep? (Fluid creep is the term applied to a relatively recently-described phenomenon that references administration of resuscitation fluids considerably greater than those predicted by a formula such as the Parkland formula. The adverse effects of fluid creep are those of abdominal compartment syndrome, the necessity for more frequent escharotomies, and possibly an increased incidence of pulmonary complications.)

    a. How can the diagnosis of abdominal compartment syndrome be established and what are some of the clinical signs? (Increase in bladder pressure greater than 20 mm of Hg, the appearance of impaired ventilation with high peak airway pressures, worsening metabolic acidosis and hemodynamic instability.)

6. What clinical scenarios may require greater than predicted fluid resuscitation volume? (Delay in initiation of therapy, inhalation injury, which may appear counter intuitive, electrical injury perhaps advancing age.)

7. What particular challenges does the pediatric patient represent for resuscitation and how are the burn resuscitation formulas modified for the pediatric age group? (Characteristically, pediatric patients require greater volumes of fluid for resuscitation than the various formulas would calculate. Also, free water, D_5W, should be added because of increased free water requirements and the necessity to avoid hypernatremia.)

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8. Discuss the use of pulmonary artery catheters for monitoring. (The results of the use of central pressure monitoring through a Swann-Ganz catheter are inconclusive and mixed. Much of the information has been derived from the use of pulmonary artery catheters in trauma patients for monitoring pulmonary wedge pressure, cardiac output, and O_2 consumption. Although these indices correlate more closely with the actual physiologic state than urine output, the wide spread use of pulmonary artery catheters have not led to an increased survival in the burn patient. An optimal application would be in the elderly, patients with preexisting cardiovascular disease and patients who are not adequately resuscitated and more information is needed to direct further resuscitation.)
    a. What would be the Swann-Ganz readings in an early (4 hour post burn) large burn inadequately resuscitated? (Pulmonary wedge pressure would be extremely low as well as cardiac index. The systemic vascular resistance would be quite high.)

9. Discuss the following:

    Swann-Ganz reading in a 25 y.o. 20 hours post burn, 50% BSA with a pulse of 96, normal BP, urine output of 50-60cc/hour: PAP 30/15, PCWP of 2, C.O. 3.5L per m^2. (The patient is adequately resuscitated.)

10. Discuss the pros and cons of the use of hypertonic saline in burn resuscitation. (The theory underlying the use of hypertonic saline is the administration of the required amount of sodium ion yet much smaller volumes of fluid. The proposed benefits were those of decreased tissue edema, less necessity for escharotomies, and fewer pulmonary complications. The potential problems include hypernatremia and hyperosmolarity with possible CNS and renal complications and is probably of historical interest only.)

11. What are the two objections to the use of Dextran as a resuscitation vehicle? (Dextran has a number of potential problems including coagulopathy, allergic reaction, and can interfere with blood typing.)

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B. Inhalation Injury

1. In general terms, describe the impact of inhalation injury in burn patients, i.e., frequency, correlation with burn surface area, effect on mortality. (Inhalation injury has a positive correlation with body surface area burn and has an additive effect on mortality. As outlined below, the frequency of occurrence hinges on the circumstances surrounding the burn injury.)
    a. Discuss the three clinical stages of inhalation injury. (The first phase within 24 hours consists of carbon monoxide poisoning, hypoxemia, and bronchospasm, all which can result in acute pulmonary insufficiency. The second phase consists of pulmonary edema, atelectasis and tracheobronchitis with respiratory failure. The third stage is bronchial pneumonia, characteristically occurring after 3-4 days post injury.)

2. Briefly discuss the toxic components of smoke and the effect on the respiratory tract. (The toxic components are secondary to a chemical injury induced by the products of incomplete combustion, aldehydes and ketones.)
    a. Describe the effects of this pathophysiologic process. (The resultant chemical or irritative tracheobronchitis can result in mechanical plugging of the distal bronchial airway. The combination of the irritative damage to the larynx and the edema induced by the resuscitation phase can produce complete upper airway obstruction.)
    b. Briefly and succinctly what is the pathophysiology of lower airway injury in inhalation injury? (The damage to the terminal bronchioles can produce premature airway closure during the respiratory cycle with focal atelectasis. These areas of atelectasis produce ventilation/perfusion mismatches. The systemic response to burn injury of increased capillary permeability occurs within the lungs as well with transudation of fluid into the alveoli, further exacerbating the deleterious effects of inhalation injury.)

3. What is the pathology of airway injury that sets the stage for the onset of infectious complications, tracheobronchitis and pneumonia? (The stage for the development of the third phase of inhalation injury, bronchial pneumonia, is set by mucosal damage of the tracheobronchial tree with bronchorrhea

and slough, the increase in pulmonary secretions and diminished ciliary response and bacterial colonization by intubation of the airway or from the burn wound.)

4. Diagnostic findings to establish inhalation injury? (A careful history may be one of the most important diagnostic findings, namely, a history of burn injury sustained in a closed space such as a house fire. Other important physical findings are singed nasal hair, intraoral soot or sooty sputum. The presence of hoarseness may indicate damage to the larynx and, of course, stridor would indicate impending upper airway obstruction.)

5. Discuss the laboratory diagnosis of the inhalation injury, including a critique of each modality. (Although not considered a laboratory study, chest xray at the early stage is normal as well as arterial blood gases. Other laboratory methods that have been utilized are Xe 133 nuclear scans and pulmonary function tests both of which are not logistically feasible and have not been incorporated routinely into the diagnosis of inhalation injury.)
    a. Expand on the role of fiberoptic bronchoscopy. (Early fiberoptic bronchoscopy can play an important diagnostic and therapeutic role. The findings of soot and inflamed mucosa within the trachea and large bronchi is indicative of a significant inhalation injury. As the inhalation injury unfolds with secondary small airway edema and airway plugging, bronchoscopy can perform pulmonary toilet and directly administer bronchodilators.)

6. What is the pathophysiology of carbon monoxide (CO) poisoning? (Exposure to carbon monoxide results in a stable intracellular compound within the red blood cell that is preferential and will replace oxygen. The carbon monoxide thus shifts the hemoglobin-oxygen curve to the left that will impair oxygen diffusion at the level of the tissues. Carbon monoxide can not be measured by arterial blood gases since laboratory measurement of blood gases is dependent on the partial pressure within the plasma not the hemoglobin or carboxyhemoglobin content. Level of carboxyhemoglobin is measured by spectrophotometry.)
    a. Outline levels and a comparable clinical picture.

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% Levels	Carbon Monoxide Intoxication
CO Hgb	Symptoms
0 – 5	Normal value
15 – 20	Headache – confusion
20 – 40	Disorientation, fatigue, nausea, visual changes
40 – 60	Hallucination, combativeness, coma, shock state
60 or above	Cardiopulmonary arrest

* CO Hgb – carboxyhemoglobin

b. Discuss the management of carbon monoxide poisoning and give the half-life of carbon monoxide with different treatment modalities. (The treatment of carbon monoxide toxicity involves the displacement of the carbon monoxide moiety from the intracellular hemoglobin by the administration of oxygen. The half-life of carboxyhemoglobin on 100% oxygen is reduced from 180 minutes to about 30 minutes. Hyperbaric oxygen can result in even a shorter half-life but the logistical difficulty is the placement of a patient into a hyperbaric oxygen atmosphere, i.e., chamber, during the resuscitation period, a period marked by hemodynamic instability.)

7. True or false: The optimal management of the airway in the burn patients dictates endotracheal intubation only with appearance in the postburn period of dyspnea and stridor? (False. Once the clinical signs of upper airway obstruction and stridor are apparent, the patient may require tracheostomy instead of endotracheal intubation, attended by increased morbidity and even mortality.)

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a. What are some clinical characteristics that may point toward early or "prophylactic" intubation? (Head and neck burns, large > 50% body burn, full-thickness circumferential burns of the trunk, high clinical suspicion of inhalation injury.)

8. In an intubated patient with circumferential third degree burns of the trunk, what are some reliable indications of the necessity for escharotomies? (The mechanics of respiration can be restricted significantly by a circumferential truncal eschar. As a result the airway pressures will rise to produce the same tidal volume, the chest wall component of dynamic compliance will decrease if calculated, P_{CO2} increases and shunting or a widened A-a DO_2 will be apparent on 100% oxygen. To summarize:
   - Rising P_{CO2} on arterial blood gases
   - A widened A-a DO_2 on 100% O_2
   - Decrease in dynamic compliance

   Increase in airway pressures for the same tidal volume)

9. Discuss the efficacy of the following: systemic steroids, prophylactic antibiotics and the use of PEEP. (Although steroids would seem indicated to diminish the inflammatory response and antibiotics to forestall infection, both have been proven to have no beneficial effect on inhalation injury and may be detrimental in addition. PEEP or positive end expiratory pressure may be of benefit to address the V-Q mismatch of ventilation and perfusion by addressing the problem with premature airway closure in the terminal bronchioles and focal atelectatic changes.)

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VII.A. Burns: Pathophysiology, Resuscitation, Pulmonary complications

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VII. B. Burns: Wound Care and Coverage

A. Topical Microbial Agents/Burn Wound Infection

1. *Briefly*, describe the natural progression of the disease process of burn wound infection. (Initially, the burn wound is relatively sterile except for some bacteria in the skin appendages and hair follicles, most commonly S. aureus. If not prevented or minimized, bacterial colonization will initially occur in a more superficial portion of the burn wound and then gradually penetrate into the deeper eschar. The next phase is proliferation of the bacteria at the interface between nonviable tissue, the eschar, and the deeper viable tissue. Once that proliferation reaches a critical mass or concentration, the microorganisms then are capable of invasion of the adjacent viable, undamaged soft tissue.)

2. Define "burn wound sepsis." (Bacterial growth reaching a level of much greater than 10^5 organisms per gram of tissue with invasion of viable subcutaneous tissues and blood vessels. The diagnosis is made by full thickness wound biopsy.)

3. Differentiate clinically and laboratory between burn wound cellulitis and burn wound infection. (Cellulitis, as the name implies, demonstrates erythema and edema of adjacent unburned skin/soft tissue and may be the precursor to burn wound infection. Burn wound infection implies an active invasion of unexcised eschar as well as adjacent viable tissue. Ordinarily, this development means that the bacterial colonization has proliferated to the point of 10^7 or 10^8 and is accompanied by or closely followed by systemic sepsis. Diagnosis can be established by histologic examination of viable tissue. Clinical diagnosis is suggested by profound changes in the appearance of the burn wound and eschar such as hemorrhagic or color changes. These changes can also occur in already-excised burn wounds.)
    a. What is the specific and cardinal histologic sign of burn wound infection? (The presence of microorganisms in unburned tissue.)

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4. Outline a staging or grading system for the histologic assessment of the burn wound colonization or invasion and why that system is consistent with burn wound pathophysiology. (A system dividing burn wound infection into that of colonization or invasion parallels what occurs with burn wound infection evolving into burn sepsis. Superficial penetrating colonization is reflective of the process of bacterial colonization within the eschar up to the point of proliferation of the bacteria in the subeschar space. Staging of the invasive process is similar, namely, the early presence of bacteria in the viable tissue immediately beneath the subeschar space and then progressive invasion of that bacteria into normal tissue with invasion of small blood vessels and lymphatics.)

5. Burn wound sepsis and pneumonia are the two most common infections in burn patients. What is the most common mechanism by which these patents contract pneumonia? (Most commonly by aerosolization of bacteria emanating from the wound surface during manipulation of the patient. This is also the reason is why topical antimicrobials decrease the incidence of pneumonia.)

6. What is the basic philosophy underlying conservative management of burns with topical agents? (The philosophy is to treat superficial partial-thickness burn wounds with an agent capable of suppressing bacterial colonization until re-epithelialization occurs, hopefully, prior to hypertrophic scarring, normally 2-3 weeks. Extension of that same philosophy to deeper burns requires the application of a topical antimicrobial agent to minimize infection while the eschar spontaneously separates and granulation tissue develops capable of supporting a split thickness skin graft. Separation of the eschar occurs as a result of subeschar proliferation and invasion of bacteria. Those bacteria then lyse the nonviable tissue that allow the eschar to separate, a prolonged process. As discussed in the next session, unless extenuating circumstances exist, the contemporary management of deeper burns is excision and coverage.)
    a. What is the evolution over time of the type of microorganism that colonizes the burn wound? (Initially, gram-positive cocci such as S. aureus are the predominant organisms, replaced in time by gram-negative organisms such as Pseudomonas. With even additional time fungi, yeast such as Candida and drug-resistant organisms become dominant.)

b. Name two contemporary organisms of concern? (MRSA and acinetobacter)

7. What is the basic issue with antimicrobial ointments, as different from creams discussed below, such as Bacitracin, Neosporin, etc.? (The agents rapidly produce secondary involvement with fungus and molds and some have a significant, 10%, incidence of hypersensitivity reactions.)

8. Discuss the possible concerns in complications in the use of silver sulfadiazine. (In large body burns in the early post-burn period leukopenia and thrombocytopenia occurs, an effect that is usually transient. The cream can not be used in patients with a sulfa allergy and should not be used on the face because of potential for eye irritation or injury. Probably the most significant historical concern has been the probable impairment of re-epithelization.)
    a. What are the two principal components? (As the name implies silver and a sulfa compound, sulfadiazine.)
    b. What is the ability of silver sulfadiazine to penetrate the eschar? (Does penetrate the eschar but to a limited degree-see below.)

9. Discuss the principal concerns and complications in the use of mafenide acetate in treatment of burns. (Mafenide acetate or Sulfamylon has an excellent eschar penetration ability. The agent also seems quite effective against Pseudomonas but appears to have little activity against fungi or yeast. Sulfamylon can be quite painful in partial thickness burns and cannot be used in patients with sulfa allergies. The principal complication, particularly in large burns, is the development of a metabolic acidosis as a result of the inhibition of carbonic anhydrase characteristic. The metabolic acidosis can also lead to potassium loss and an increase in respiratory rate.)

10. Briefly, discuss the solutions of silver nitrate, Dakin's, and acetic acid. (Silver nitrate solution as a primary agent for the treatment of large body burns is probably of historical interest only. The agent does not penetrate the eschar and can produce hyponatremia and requires close monitoring of electrolytes in large surface burns. Dakin's solution is dilute bleach and has toxicity to tissue cells. Dilute acetic acid can be effective against Pseudomonas but is relatively infrequently used.)

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11. Discuss the mechanism of action of silver as an antimicrobial agent and the practical clinical applications. (Silver is a heavy metal and as such is toxic to bacteria but has an additional antimicrobial dimension that is not clearly understood. To be active, silver must be soluble in solution since the silver ion binds to proteins in the serum and/or tissues, so silver concentrations must be maintained least at a minimal level. The metallic form is inert and must become ionized to become reactive and thus bind to proteins in cell membranes. Whether the silver is applied in solution, silver nitrate, or as a cream, silver sulfadiazine, application must be done two to three times daily.)
    a. To avoid the necessity of frequent application, discuss silver-eluting agents. (Dressings have been developed that incorporate nanocrystalline silver that elutes the active silver ion in an uncharged form when exposed to water/fluid and are changed every three days. Examples include Acticoat, Aquacell and Mepilex)
    b. Discuss potential problems with the use of silver-impregnated dressings. (The principal problem is staining of the adjacent unburned skin as well as the care keeper's clothing, dressings etc., the possibility of argyria from systemic absorption of the silver, and the potential problem of retardation of wound healing.)

12. What is the role of intravenous antibiotics in treatment of burn patients? (Prophylactic intravenous antibiotic use is discouraged. Systemic antibiotics are indicated for those patients that demonstrate sepsis. Burn patients have altered binding and metabolism of antibiotics and serum levels must be frequently monitored.)

13. What explains why adequate serum levels of some antibiotics may require more frequent dosing than normal? (The hyperdynamic and hypermetabolic state of the burn patient results in a shortened half life for most pharmaceutical agents.)

14. Give some clinical and laboratory evidence for the establishment of the diagnosis of burn wound infection. (Probably the most important clinical finding indicative of burn wound infection is an

alteration in the appearance of the burn would including color. A transformation to a more dark or violaceous eschar can be indicative of the evolution into burn wound infection. Probably the most important laboratory indication, outside of systemic signs of sepsis, is a quantitative wound biopsy with a result of $>10^5$ bacteria colonies.)

15. What is the treatment for suspected invasive burn wound infection? (Treatment of the systemic sepsis, of course, but also the application of a eschar-penetrating agent such as sulfamylon on a 2-3 times per 24 hour basis. The keystone of the treatment is surgical excision of the infected tissue with application of topical antimicrobial agents in solution.)
    a. From an infection point of view, what is the logic of early excision of eschar? (Removal of necrotic eschar appears to reverse many immunologic abnormalities and defects that are seen in the burn patient. Secondly, the longer burn wounds remain open, the longer they are risk for infection. Timely closure minimizes risk.)
16. What are the most likely non-bacteria organisms to produce burn wound infection? (Candida, fungi such as Aspergillus and herpes simplex virus.)

B. Burn Wound Excision and Closure

The readings that have been included provide both enthusiastic endorsement of burn wound excision as well as perhaps a more balanced perspective.

1. Describe the three zones of burn wound injury. (The theoretical basis for tangential excision is the three zones of injury. The deepest or central zone is the nonviable eschar, the surrounding zone is the zone of stasis or possible microvascular compromise of blood flow and potentially reversible injury, and finally, the least-injured zone of hyperemia or increased blood flow.)

2. What are the principal advantages of burn wound excision? (Early burn wound excision has the potential for reduction of mortality, shortening of hospital stay, and not subjecting the patients to prolonged daily wound debridement. Early burn wound excision of deep partial thickness burns may also avoid hypertrophic scars.)

a. Disadvantages? (Increased blood loss, potential for excision of a burn that will heal spontaneously without hypertrophic scarring. Shortening of hospital length of stay in large burns has not been definitively proven.)

3. In those studies that have demonstrated improved survival, that improvement has been limited to what group? (Patients 16-30 years of age without inhalation injury and 40-60% body surface area burn.)

4. Although discussed in more depth in the next module, metabolism and nutrition, what is the basis for the abnormal metabolic response to the burn injury? (The burned tissue, skin and subcutaneous fat, promotes an inflammatory response at the junction of the eschar and the underlying viable tissue. Bacterial proliferation can attract PMN's that in turn are responsible for the release of inflammatory mediators. The systemic response to these inflammatory mediators is increased metabolism and energy expenditure, weight loss and immunodepression.)

5. Discuss the technique and the perspective of tangential excision of partial thickness burns of less than 20-25% total body surface area. (Tangential excision is the use of large knives to remove sequential slices of burn tissue followed by the application of split thickness skin grafts. The sequential excision of burn tissue is continued until a pattern of dermal bleeding, indicative of viable tissue, is encountered. The papillary dermis will have diffuse and pinpoint pattern of bleeding rather than coarse as seen in the reticular dermis. One perspective is that the blood loss is substantial: approximately 100 cc's for every 1% body surface area excised, the wounds may heal without hypertrophic scarring without excision, and the demonstration of improved functional or cosmetic result is unproven. Immediate application of skin grafts, either auto- or allo-, are mandated to preserve their residual dermis. Some shortening of hospital stay may occur, but at the expense of general anesthesia and blood transfusions.)

6. What are two different concepts, not techniques, of burn wound excision? (Early or acute total excision of the burned wound and staged excision and grafting with the extent of excision guided by available donor sites.)
    a. In those units that have adopted early primary excision of the burn wound, in general, what is the approach in very large, greater than 60% TBSA burn? (In general, fascial rather than tangential excision and the treatment plan is completion of the excision by the fifth day postburn and coverage with very thin widely meshed skin grafts ± Integra.)

7. Describe in detail the techniques of tangential excision, fascial excision, and methods to control blood loss. (Tangential excision, as described above, consists of sequential excision of deeper layers of burned eschar until a viable layer is reached, whether deep dermis or subcutaneous fat. Fascial excision in contrast, excises burned and unburned tissue to the level of the deep fascia. The pros and cons of each is that fascial excision results in less blood loss but can create a substantial aesthetic contour defect. Tangential excision results in substantially greater blood loss but limits excision to burned tissue only. Some methods that can control blood loss include the use of tourniquets on extremity excision as well as pharmacologic products such as fibrin spray, epinephrine/thrombin soaks,ect. The use of tumescence solution, dilute epinephrine solutions in Ringer's lactate or saline, also have the capability to reduce blood loss.)

8. Describe the essentials of the operative management of a large body burn. (Methods need to be provided to prevent hypothermia which consist of a warm operative room and perhaps warming lamps, expectant blood loss with early replacement including methods to minimize blood loss such as the use of tourniquets and compressive dressings.)

C. Coverage of the Excised Burn Wound

Excision of specific anatomical areas, e.g., hands, will be addressed in the Burn Reconstruction module. A more detailed discussion of skin substitutes is also included in the biomaterials module I. G. Biomaterials.

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1. What is the essential difference between a synthetic dressing as Biobrane and allograft when applied to a wound? (A synthetic dressing may *adhere* to the wound as a result of fibrin deposition but will not become *incorporated*. At 24-48 hours the allograft skin will begin to vascularize as the capillaries migrate from the wound bed into the graft and the graft begins to "take", duplicating the sequence seen in autografts.)
    a. Advantage of the allograft? (Both materials in the early period, 24-48 hours, can reduce evaporative loss and perhaps even adhere to the wound as the result of fibrin deposition. As the allograft becomes vascularized the capability to decrease bacterial count is enhanced. Synthetic dressings require frequent dressing changes in order to decrease bacterial count.)
    b. What wound characteristics must be present to allow allograft take? (Exactly the same as autograft, namely, free of infection and a well vascularized bed.)

2. Differentiate the ultimate fate of the epidermis vs. the dermis components of an allograft placed on an open wound. (Even with vascularization, if not provided with sufficient diffusion of oxygen and nutrients the epidermis may be lost. The epidermis has the Langerhans cells which contains the histocompatibility antigens and as such will be the stimulus for rejection. Rejection consists of loss of the epidermis although the dermis may survive and persist for an extended period of time because of the diminished patient antigenicity in very large burns. Rejection followed by secondary infection, particularly in the adnexal structures within the dermis, frequently results in lysis of the entire autograft. Unfortunately, the process of rejection of the epidermis often leads to sufficient inflammation and infection that the remaining dermis is lyzed as well and the quality of the wound may regress.)

3. Discuss methods of preparation of allograft including the pros and cons of each. (The behavior of refrigerated or fresh allograft is the same as stored autograft namely after five days tends to lose viability precipitously. Cryopreserved allograft is frozen and retains some viability for a prolonged period of time. Although treated, frozen allograft loses substantial viability in comparison to fresh allograft. The benefit is obviously prolonged shelf life in comparison to fresh allografts. As such, commercially available allograft is almost always frozen.)

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4. Summarize the overall risk and clinical usefulness of each. (Fresh allograft is excellent protection and coverage for excised burned wounds and is also a good test for the suitability of wounds for autografting. The cost and limited availability are the principal limitations. The risks include the possibility of viral disease transmission, although that occurrence is extremely rare. Frozen allografts are good, although not as excellent as fresh allograft, for coverage of excised burns and are a more practical use as a test of the suitability of a wound for autografting. Frozen allograft shares the same risk of disease transmission as fresh allograft.)

5. Describe how allograft could be utilized to determine the suitability of autografting and wound closure of a chronic granulating wound. (Allografts are not viewed as "foreign" for the first few days after transplantation before the type II hypersensitivity reaction is stimulated. As such, the allograft simulates autografting and if infection supervenes, the wound is too heavily colonized.)

6. Describe the use of combined auto-and allografts in large burns. Comment on pros and cons. (The effort in very large burns is to mesh the autograft widely perhaps even at a 9:1 ratio. To do so, creates large amount of open wound because of the wide mesh. Allograft is placed over the autograft in effort to close, temporarily, the open wounds. The difficulty is the allograft will be recognized as foreign and undergo rejection with the secondary effect of lysis, infection, ect., described above with loss of both auto – and allograft.)

7. What are CEAs or cultured epithelial autografts? (CEAs are developed from a skin biopsy taken from the patient and then cultured, eventually yielding a preparation of sheets of epithelial cells about 6-12 cells thick.)
    a. Describe the performance and limitations. (The culture process requires about three weeks. The end product lacks long term durability and the overall take or engraftment rate is quite low. The expense is high. The lack of durability stems from the absence of dermis and delayed breakdown with blistering and open wounds are not infrequent.)

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8. Are porcine xenografts vascularized by the host? (No. Adherence as well as **sealing** the wound against evaporative fluid loss are an advantage of xenografts, but they do not vascularize.)

9. Although dermal analogs are discussed in the biomaterials module, briefly describe the composition and use of the following: Integra and human acellular dermis. (Integra is bovine collagen fibers immersed in shark ground substance or a glycosaminoglycan and an outer layer of a silicone polymer. Integra is intended to provide dermal wound coverage until incorporation into the host is manifest by a thin layer of vascularized tissue beneath the silicone, ordinarily at about two or three weeks. Thin autografts can then be applied. Integra is expensive and in a contaminated wound may become infected.

Human acellular dermis is a commercially available dermal substitute and is cryopreserved cadaver acellular dermis. Although used widely in breast and abdominal reconstruction, Alloderm has had a limited application in burns.)

10. Discuss the composition of as well as the benefits and limitations of Biobrane. (Biobrane can seal the wound and prevent evaporative loss and bacterial contamination. The principal limitations are the accumulation of fluid and susceptibility to infection as a synthetic material. Biobrane has no inherent bacteriostatic capability and is a synthetic bilaminar substitute composed of nylon and silicone. The Biobrane can be easily dislodged by patient movement.)

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VII. B. Burns: Wound Care and Coverage

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VII.C. Burns: Nutrition and Metabolic Support, Electrical and Chemical Burn Injury

A. Nutrition and Metabolic Support

1. The primary mediators of the large burn hypermetabolic response are? What are the effects of same? (The catabolic hormones of catecholamines particularly epinephrine, cortisol, and glucagon, all of which can lead to proteolysis, gluconeogenesis as well as an increase in production of endogenous corticosteroids, decreased protein and lipid synthesis.)
    a. Since muscle protein catabolism leads to a decrease in lean body mass, what are the negative consequences of importance to the burn patient? (Diminished capacity for wound healing, increased predisposition to sepsis, diminished immune response.)

2. What are the outcomes of the unaddressed hypermetabolic response in the major burn injury? (Increased resting energy expenditures, increased cardiac work and O_2 consumption, muscle wasting, hepatic dysfunction.)
    a. What is the magnitude of the hypermetabolism? (For burns greater than 40% BSA and dependent on the opinion of which of the readings distributed, from 50-100% greater than the basal metabolic rate.)
    b. What is the broad index of this response? (Increased nitrogen excretion in the urine reflects the protein breakdown as a result of the burn hypermetabolic response.)
    c. True or false: Wound closure returns the basal metabolic rate to normal. (False – even with healing and wound closure, basal metabolic rate, BMR, is in the range of 130% and even at one year resting energy expenditure is 110-120%)

3. Rank the following pathologic states in terms of magnitude of protein breakdown as reflected by increased nitrogen excretion: Starvation, 40% + burn injury, severe sepsis, multiple skeletal trauma. (40%+ burn injury, multiple skeletal trauma, severe sepsis, starvation.)

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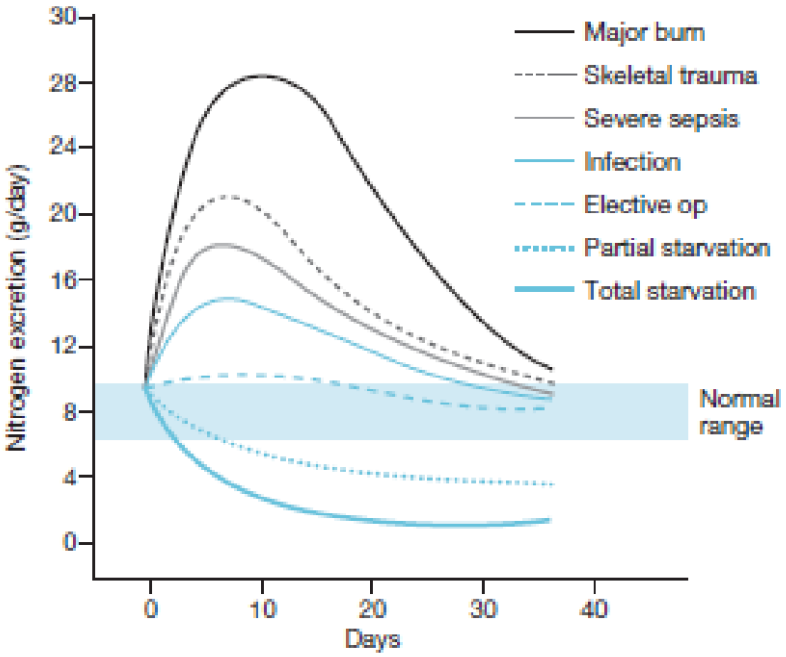

Figure 29.1 A classic illustration of nitrogen excretion following injury compared to starvation and other conditions. Burn injury evokes the most pronounced catabolism of any clinical condition, with nitrogen excretion exceeding 25 g/day (150 g of protein, almost a half-pound of lean body mass!). Notice also the dynamic 'crescendo–decrescendo' nature of this process: nitrogen excretion (and metabolic rate) rises from near-normal levels just after injury to reach a maximum at 7–14 days post burn, declining slowly thereafter throughout recovery. This illustrates the impossibility of using static formulas to estimate nutritional requirements accurately at every point throughout the course of burn treatment. *(Reproduced with permission from Long et al, 1979.[10])*

Saffle JR, Graves C, and Cochran A, Chapter 29: "Nutritional support of the burned patient", Herndon, D. N., Total Burn Care, Saunders, (2012); pgs. 333-353.

a. What is the estimated maximal weight loss in a 40%+ body burn without routine nutritional support? (25%) When? (2-3 months post burn).

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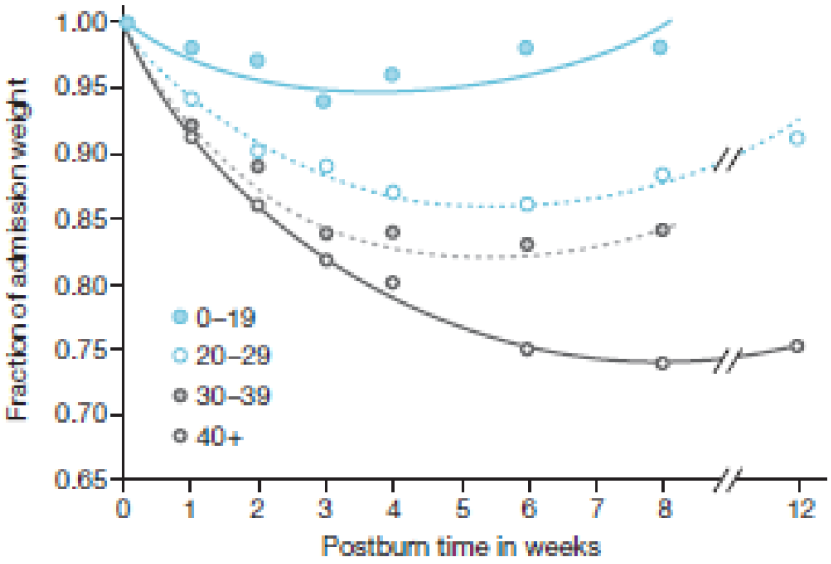

Figure 29.2 Weight loss following burn injury as documented in the era before routine nutritional support. Dramatic losses of lean body mass occur within a week or two of injury, and progress continuously in the absence of effective nutritional support. By 4–6 weeks patients with major burns have lost 15% of lean body mass or more: this can be a fatal degree of inanition. *(Reproduced with permission from Wilmore, 1974[13].)*

Saffle JR, Graves C, and Cochran A, Chapter 29: "Nutritional support of the burned patient", Herndon, D. N., Total Burn Care, Saunders, (2012); pgs. 333-353.

4. What is the primary method to modulate the burn hypermetabolic response? (The single most effective method is early excision and grafting of the burn, namely wound closure. One of the readings suggest in the first 72 hours or perhaps the first week. Other measures are discussed below.)

5. Describe the role of ambient temperature control and non-operative methods and the physiologic basis for same to assist in modulation of the hypermetabolic response. (Burn patients sustain a tremendous increase in water and heat loss through the burn wounds, both of which result in energy loss and expenditure. Increase in the ambient temperature in the patient's environment, hospital room and OR, to 28-33°C, attenuates this component of the hypermetabolic response.)

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6. Describe the efficacy of the following in attenuation of the post-burn systemic inflammatory response: anti-inflammatory agents as ibuprofen, glucocorticoids, monoclonal antibodies. (Essentially, none of those modalities have proven to be efficacious in treatment of the systemic inflammatory response.)
    a. If the above modalities are ineffective, what are the essential elements of address of same? (The obvious: adequate fluid resuscitation, use of vasoactive agents as necessary, excision of the burns, enteral support, prompt diagnosis and treatment of burn wound sepsis.)

7. What is indirect calorimetry? (Measurements of volume of expired gas and the concentration of oxygen, O_2, and carbon dioxide, CO_2.)
    a. What is the respiratory quotient? (Ratio of CO_2 produced/O_2 consumed or VCO_2/VO_2)
    b. What is the role of bedside indirect calorimetry? (Calculate resting energy expenditure)

8. Discuss address of the nutritional needs of the burn patient. (The measurement, as described above, of the resting energy requirements by beside indirect calorimetry can establish nutritional needs. These needs can be delivered most optimally by enteral feedings. Parenteral feeding by the intravenous route has significant drawbacks including bacterial intestinal translocation, loss of gut motility, and impaired immune response.)
    a. What are some methods of monitoring nutritional status and support? (Because of wide fluctuations with infections, ventilator support, and fluid balance, body weight is notoriously unreliable but easily obtained. Nitrogen balance performed 1 - 2x/per week with 24 hour urine collection will provide an estimation of nitrogen losses. Serum markers as prealbumin may provide a rough estimate if performed serially.)

9. Give a formula for calculating caloric needs. (The most widely used states 25 cal/Lg body weight + 40 cal/% BSA burned. Formulas tend to overestimate caloric needs. Another estimate is 1.4 x the resting energy expenditure)

10. The major energy source for burn patients is? (Carbohydrates)

a. Why? (Essentially, the fuel for healing wounds and the metabolic pathways is carbohydrates.)
b. The paradox is that despite increased demands and production of glucose, utilization is hampered by what? (The presence of insulin resistance resulting in hyperglycemia which also mandates for strict blood glucose control with exogenous insulin)

11. Give an estimate of the needs of carbohydrate, fat, and protein, expressed in per kg for a large body burn. (7g of carbohydrate, 1g of fat, and 2g of protein or 28 + 9+ 10 calories or a total of 47-50 calories per kg; less so in smaller burns perhaps more so in larger burns.)

12. Overfeeding can result in what? (Overfeeding, particularly of carbohydrates, can result in an increase in fat synthesis with fatty filtration of the liver. Overfeeding can further intensify the hyperglycemic characteristic of the major burn inflammatory response, rendering glucose control more difficult. Careful calculation of nutritional needs is imperative.)

B. Chemical Burns

1. What is the chemical property that differs the extent of acid vs. alkali burns? (Acids cause coagulation necrosis with precipitation of protein that will limit the depth-extent of the injury by neutralization of the agent. An exception may be hydrofluoric acid. See below. Alkalis create liquefaction necrosis by dissolution of the tissue protein and continuation of the extent and depth of injury.)
    a. What is the keystone and implications of treatment? (Copious irrigation with water. Irrigation of alkali burns may be necessary initially for a prolonged, 1-2 hours, and repeated every 4-6 hours for 24 hours. Achievement of a neutral skin pH as measured by litmus paper is the treatment goal.)

2. Hydrofluoric acid is a commonly used agent in industry. Besides the acidic component of the injury by exposure, what are the principal electrolyte disorders? (The fluoride ion combines with tissue calcium and magnesium resulting in hypocalcemia and hypomagnesaemia. Calcium and magnesium are essential elements of cellular function and lethal cardiac arrhythmias can occur. Hydrofluoric acid may also result in liquefactive necrosis secondary to the free fluoride.)
    a. Give a general classification of concentration of hydrofluoric acid exposure vs. injury. (Concentrations greater than 50% cause immediate tissue destruction and pain; 20-50% the injury may be apparent within several hours; concentrations less than 20% may take up to 24 hours to become apparent.)
    b. For significant exposures, what agent can be used to assist in neutralization of the hydrofluoric acid? (Application of a topical gel of 2.5% calcium gluconate as well as calcium gluconate injections of 5-10% concentration into the subcutaneous tissue of the wound.)

3. Phosphorous is an incendiary compound used in military armaments as well as some agricultural fertilizers. What is the method for identification of soft tissue particles and the treatment of same? (Use of a Wood's light will assist in identification of embedded particles by ultraviolet light. Injection of a solution of 0.5% copper sulfate will assist in neutralization as well as identification of particles. The copper agent has its own potential for toxicity and renal injury and lacking availability, irrigating with water may be equally as efficacious.)

C. Electrical Burns

1. What is the essential equation that estimates the degree of tissue damage by an electrical injury? (Ohm's Law = V/R, or the intensity of the current in amperage.)
    a. In fact, only one factor in the equation, voltage, is known. So, how can that number be useful? (Electrical injuries are classified as high vs. low based on the magnitude of the voltage. High voltage injuries are considered greater than 1,000 volts, low voltage less than 1,000, although as the readings explain the tissue resistance can be important as well. See below.)

   b. How does Joule's Law enter the picture in terms of tissue damage? (Adds the element of time so the duration of heat or energy transfer to the tissues is considered.)

2. Discuss the element of resistance and how that factor can vary widely. (The skin resistance of a moist palm is much less than that of a heavily calloused hand. A hierarchy of resistance exists in the different tissues.)
   a. List the hierarchy of the specific tissue resistance from low to high. (Nerve, blood vessels, muscle, skin, bone, and tendon. Bone and tendon are relatively avascular and acellular compared to the other tissues on the list.)

3. Differentiate the types of electric burns that occur from a high voltage source. (Three types of electrical burns can occur and include contact, arc, and flash. The first two are considered "true" electrical injury or burns. The contact burn occurs from either the entrance into or exit from the body of the current source. An arc burn occurs when the current from a high voltage source is transmitted and conducted by air from source to the contact point on the body. Characteristically, the generation of heat by the arcing process can be 2,000-3,000 degrees. A "flash" burn is when clothes are ignited and skin burns occur that are not essentially different than thermal burns.)
   a. Give some characteristics of arc and contact electrical burns. (The contact points will sustain full-thickness skin injury but the extent of the skin injury will not reflect the magnitude of underlying tissue damage. The readings discuss the more extensive nature of the underlying muscle injury and the implications for treatment. In addition to the arc component of the contact injury, arc burns also may occur, dependent on the voltage, on flexion creases of the extremities; wrist, antecubital fossa, and axilla as well as popliteal fossa and the groin area.The skin burns at these skip areas conceal a much larger area of muscle and soft tissue injury as a result of the high temperatures generated by the arc phenomenon.)

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4. Outline the principle, beyond ATLS, of the initial, 24 hours care of the electrical burn patient. Begin with assessment. (The history needs to center on the circumstances surrounding the injury, namely, if a fall occurred or momentary loss of consciousness occurred. If the voltage of the current source can be determined that number should be ascertained. The physical exam centers on the contact points of the head, chest, abdomen, and extremities because of the implications of each.)
    a. Give the implications of the specific body region of the contact points. (Contact points on the head have potential for CNS disturbances as well as the eventual reconstructive needs for exposed, damaged skulls. Contact points of the chest may have resulted in direct cardiac or pulmonary injury. An entrance or exit point on the abdomen may conceal underlying visceral injury. The same principles apply to contact injury of the extremities, concealed deep tissue injury beneath essentially intact skin.)

5. What are the three principal treatment needs or considerations for the first 24 hours? (EKG monitoring for cardiac arrhythmias, fluid resuscitation, the necessity for extremity decompression. Cardiac monitoring in the presence of direct chest contact injury may require monitoring beyond 24 hours. The volume of fluid resuscitation hinges on the degree of muscle injury. The presence of macroscopic or visible myoglobinuria mandates fluid administration beyond that of body surface area burned in order to achieve urine output of 1+cc/kg/hour and urine clear of pigment. A rough guideline is 9cc/% BSA/kg.

Extremities should be monitored clinically and with compartment pressures for the possible necessity of decompression by fasciotomies. Unlike thermal injury burns, the restrictive structure is the deep muscular fascia.Most high ultra voltage contact points on the hand, presumably entrance, will require early fasciotomies of the forearm.)

6. What are the essential elements in wound care of the electrical burn? (Serial debridement every 24-48 hours, temporizing wound coverage with allograft, awareness of possibility of deep muscle damage, early definitive wound coverage and/or amputation.)

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VII.C. Burns – Nutrition, Metabolic Support; Electrical and Chemical Burns

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VII.D.1: Burns: Burn Reconstruction – (Eyelids, Ears, Nose, Cheek/Face, Neck)

Editorial Note: Burn reconstruction of the nose is similar to reconstruction in skin cancer, Module III.B.3.

A. General

1. The readings establish the dilemma in treatment of acute facial burns. Outline the dilemma. (If burns are sufficiently deep, healing will be principally by contracture and the production of hypertrophic scars. If that outcome can be predicted, then early excision and skin grafting will provide a more desirable outcome. Yet, the excision of a superficial burn that would otherwise spontaneously reepithelialize will result in an inappropriately grafted face. The readings offer 3 weeks as the watershed point to permit healing if depth of burn cannot be ascertained.)

2. Give a classification system of facial burn scarring. (Essentially two classes. A normal-appearing facies without deformity but diffuse hypertrophic scars. The other is scarring, contracture, and distortion of facial features.)

3. What are the specific stigmata of facial burn scars? (The stigmata of facial burn scars consist of lower lid ectropion, a shortened nose with inadequate soft tissue coverage and skeletonization, alar flaring, and a short retruded upper lip. Eversion of the lower lip occurs because of scar contracture with inferior displacement. Overall, the facial features are flat with loss of jawline definition.)

4. What are some non-operative modalities employed to treat hypertrophic scarring? (The use of prefabricated, custom-made, silicone-lined compression masks and/or compression garments are conventional and traditional methods to address facial burn scars.)

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5. What lasers have been principally utilized for the treatment of hypertrophic burn scars? (Either ablative, the CO_2 laser, or 585 nm pulsed dye laser. Although a wide variety of lasers can be employed, the only other laser reported is the intense pulsed light.)
    a. What measures or assessment instruments can be utilized? (The two tools are modified Vancouver Scar Scale and patient observer assessment scale or POAS.)

6. Give a perspective on the use of laser therapy in the management of burns. (Although laser therapy, particularly the pulsed dye laser, has been described and advocated in the early and late management of burn scars, the readings offer a note of caution. A systematic review of past contributions revealed low quality studies and methodology such that no clear evidence of efficacy could be established.)

B. Eyelids

1. Discuss the examination of the eye in burn patients. (Vision, conjunctival burn, pupillary reaction and extraocular muscles, clarity of cornea and anterior chamber, corneal staining with fluorescein strip, measurement of intraocular pressure.)

2. Discuss the management of alkali burns of the eye. (Alkali burns tend to cause more penetration injuries than acid, and emergency treatment with irrigation is the cornerstone. A litmus test can document that neutral pH has been reached in the cul-de-sac. Intraocular pressure should be followed and the patient started on 1% atropine drops. Complications are symblepharon and ulceration.)
3. What are the methods available to protect the cornea in instances of severe lid loss? (A saran- wrap moisture chamber, scleral lens, or a Gunderson conjunctival flap and even simply antibiotic ointment alone in less severe cases. The use of ointment during the early days post-burn is sufficient. If more aggressive procedures are necessary, a Gunderson flap that consists of mobilization of conjunctiva to place over the cornea may be necessary. Early consideration should be given if persistent staining of the cornea indicates exposure. Full-thickness graft release may be

necessary with the recognition that additional release later in the post-burn period will also be necessary.)

4. Other than contracture and exposure, what other mechanism that is an outcome of burned eyelids can predispose to corneal ulceration? (Destruction of the Meibomian glands on the lash margin will result in loss of the lipid component of tears. The lipid component in addition to the aqueous and mucin components provides protection and helps to minimizes the evaporation of the tears.)

5. What were the recommendations and thoughts about temporary tarsorrhaphy for protection? (The readings were of the opinion that temporary tarsorrhaphy has less of a role than in the past because they are not effective and often dehisce and break down. They recommended early skin grafting even on granulation tissue if necessary.)

6. Briefly, describe the technique of permanent tarsorrhaphy. (An intermarginal tarsorrhaphy is the most straight forward and is accomplished by excision of a strip of conjunctiva over the superior, the upper eyelid, and the inferior, the lower eyelid, margin of the tarsus to create a raw surface, placed at the most lateral aspect of the lids. Two horizontal sutures of a 5^{o} prolene are placed with the use of small silastic catheter tubing to prevent lid skin erosion by the sutures.)
7. Describe the masquerade procedure. (Probably an extremely uncommon operation. Consists of suturing the upper and lower eyelid tarsal or lid conjunctiva to each other and application of a skin graft over the raw surface of the conjunctiva. Second stage consists of division of the skin graft. Clearly, a number of shortcomings exist in this procedure.)

8. Define symblepharon. (Adhesion of the eyelid to the underlying globe with loss and obliteration of the conjunctival-lined sulci. Address would require release and the liberal use of mucosal graft.)

9. Discuss the management of burn ectropion, including early vs. late, upper vs. lower eyelid, flaps vs. types of skin grafts. (The indications for early ectropion release are corneal exposure, irritation, and risk of ulceration. If possible, release should be deferred as long as feasible to permit maturation of

the burn scar. The release should be oriented transversely and extend to and beyond each canthus, with the incision reasonably close to the lid margin in the lower eyelid and palpebral fold in the upper lid. The readings state that flaps have no role in release of eyelid contractions and advocate full-thickness skin grafts for the lower eyelids and thick split grafts for the upper lids. What the readings did not discuss was the "third dimension" release of burn ectropion, namely, the importance of complete release of skin, orbicularis, and septum to accomplish a complete release. A oversized bolster should be applied to the graft which will essentially push the lash margin upward and serve as a tarsorrhaphy. A Frost suture can be utilized for upward traction during the release but is not necessary postoperative.)

10. Describe a technique for release-reconstruction of epicanthal folds. (A variety of procedures exist most of which center on the use of a multiple Z-plasty concept. The jumping man is the use of several advancement flaps to address the contracture as represented by the straight line of the trunk.)

11. What methods are available for eyebrow reconstruction? (Free scalp grafts, either single or multiple strips, as well as punch grafts, an island flap on the superficial temporal vessels, and scalp flap.)

12. Discuss the pros and cons of grafts vs. flap approach to eyebrow reconstruction. (The authors quote the Pensler study that determined that actually partial loss and misalignment was less common in free grafts than in the vascularized flaps. Probably gender, status of the other brow, and the degree of scarring on the recipient side all should be considered in choice of reconstruction.)

C. Ear

Partial and total ear and nose reconstruction are both addressed in another module. (Skin Tumors)

1. What is the principal concern with acute deep burns of the ear? (Secondary infection of the cartilage, the development of chondritis.)

a. What is the primary treatment to minimize the development of burn chondritis? (Application of Sulfamylon as well as avoidance of compression of the ear.)
b. What are the findings in acute burn chondritis? (Red, tender, swollen ear with obliteration of the normal anatomical landmark.)
c. Treatment of choice for burn chondritis. (Systemic antibiotics as well as bivalving and debridement of the clearly necrotic cartilage. The outcome is frequently, even usually, a significant deformity.)

2. Give one classification of ear deformity secondary to burns. (Subtotal absence of the helical rim, total absence of the helical rim, ear lobule deformity, and meatal stenosis.)
    a. What is the most common? (Loss of the superior helical rim.)

3. Outline the pros and cons of excision of burned ear tissue and skin grafting. (The obvious pro is that the burned skin can be excised and replaced with either thin full thickness or split thickness grafts with preservation of the underlying cartilaginous framework. The disadvantages or cons are at least two: one, the ear, could heal satisfactorily without deformity; second, most commonly, when the skin has sustained a full thickness burn because of the absence of any significant tissue between skin and perichondrium the perichondrium is damaged as well. Clearly, large areas of auricular cartilage without perichondrium will not sustain a skin graft.)
    a. What is an alternative for coverage? (Probably the most popular is a temporoparietal fascial flap covered by skin graft. Although, this technique has been utilized in post-traumatic as well as congenital ear loss, in severe burns the ear cartilage is significantly damaged and will often result in a deformed ear without discernible normal landmarks.)

4. Describe the use of a alloplastic or synthetic ear reconstruction. (As mentioned in the readings, silastic framework has fallen out of favor as an alternative to the use of autogenous cartilage because of high exposure and extrusion rates. A more recent development is the use of porous polyethylene or Medpor as a framework, an extension of the experience with congenital microtia,

covered with a temporoparietal fascial flap. Little experience with the use of Medpor in total burn reconstruction has been published to date but results are promising.)

5. The readings describe the use of tissue expansion with small expanders for ear reconstruction. What do you believe would be the principal limitation(s)? (Quite frequently the tissue in the peri-auricular area is scarred from the burn injury. The use of expanded scalp is quite problematic because of the hair follicles.)

6. Describe the use of prosthetic ear reconstruction with the use of osteo-integration. (The essential principle of osteo-integration is the placement of titanium implants that allow for some element of tissue/bone ingrowth and a second or third stage placement of the prosthetic ear portion of the implant. The soft tissue cover must be thin and minimally scarred, however.)

D. Face/Cheek

1. Discuss and describe the regional aesthetic units of the face in burn reconstruction.

(The seams of any flap or skin graft reconstruction should be placed along a border between the units to accomplish optimal camouflage. This concept would imply seams below the lower border of the body of the mandible, the peri-orbital region and an interface between the temple and cheek.)

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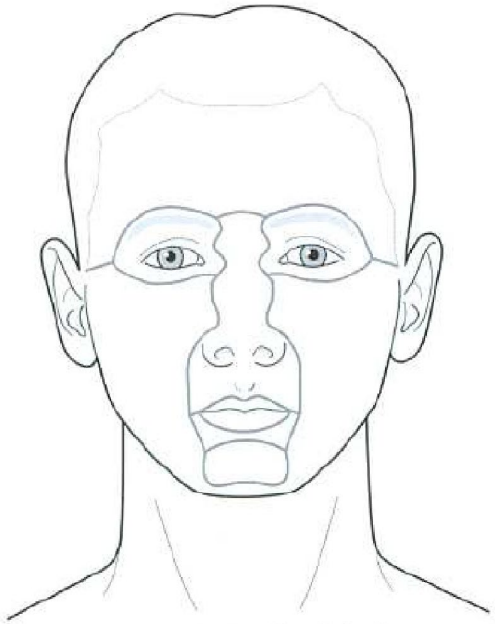

Figure 14.11 Regional aesthetic units of the face.

Dougherty, W. R. and Spence, R. J., Chapter 14 "Reconstruction of the burned face/cheek: acute and delayed." Sood, R., Burn Surgery: Reconstruction and Rehabilitation. Saunders Elsevier (2006), p. 242.

a. What is the "gold standard" in facial burn reconstruction? (The use of full thickness skin grafts.)
b. What are some time guidelines re: excision and grafting of facial burns? (As the readings outline, burns that have not healed by three weeks will heal by contracture and secondary epithelialization with resultant hypertrophic burn contractures and scars.)
c. Discuss primary excision and skin grafting versus grafting onto a granulating surface. (Split-thickness skin grafts placed on granulation tissue are prone to substantial more contracture than skin grafts placed on a freshly excised wound. The same principal of a hiatus of 2-3 weeks post-burn to allow for possible complete healing by reepithelialization. So, primary excision of the burn combined with an application of a thick skin-graft is preferable.)

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2. What is the hierarchy of correction of burn scars/contractures in the face? What are the treatment implications? (The eyelids, the lower lip/chin unit, the upper lip, cheeks, and finally the nose. The readings classify facial burn reconstruction into two categories: one, no facial distortion but diffuse burn scarring without contractures and; second, facial burn contracture and deformities.The division would appear to be somewhat simplistic even though a facial deformity as eyelid or ectropion or nasal loss may not be present, diffuse scarring may still dictate the necessity for a reconstructive plan. The point established by the readings is that perhaps limited Z-plasties, local excisions, etc. may suffice. Perhaps the division into peripheral and central has more concise reconstructive implications. See question below.)

3. Contrast the reconstructive needs of the peripheral aesthetic units of the forehead, temple, and cheeks with the central units. (In general, the featureless flat, peripheral facial reconstructive units can be addressed with full-thickness skin grafts and are preferred over flaps, particularly the forehead and temple. The cheek, as the readings describe, may require the use of expanded transposition flaps harvested from the shoulder to provide a more full and round appearance. The central units are the nose and lips. Release, in the instance of the lower lip, or resurfacing, in the instances of the upper lip and the nose, may optimally be addressed with full-thickness skin grafts harvested from the “blush” area. The “blush” area refers to skin above the clavicles and provides an optimal color match and texture. (Reconstruction of the nose is covered in the module III. B. 3. Nose Reconstruction.)

4. What is the advisability of a rotational-transposition flap from the neck to address the cheek? (Probably not advisable because of the downward traction from gravity and the secondary occurrence of lower lid ectropion unless the area is small and can be addressed with a cervicofacial flap. Similarly, expanded neck flaps rotated rather than advanced are at a less risk for the same complication.)

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5. Give a general category of methods of burn reconstruction of the face/cheek with pros and cons of each. (A broad classification would be full thickness skin grafts, either direct or harvested from expanded skin, split thickness skin grafts, flaps, flaps either expanded or not expanded, and synthetics such as Integra. One of the readings provides an algorithm for the use of each. Expanded neck and adjacent supra-clavicular shoulder skin if available provides an elegant one unit reconstruction. FTSG if harvested from expanded supra-clavicular skin is also extremely useful as a large sheet type skin replacement, but neither may be available. The use of Integra as a two-stage approach with application of a thin split thickness skin graft after vascularization of the Integra is a an option but recontracture and reoperation may be an attendant complication.)

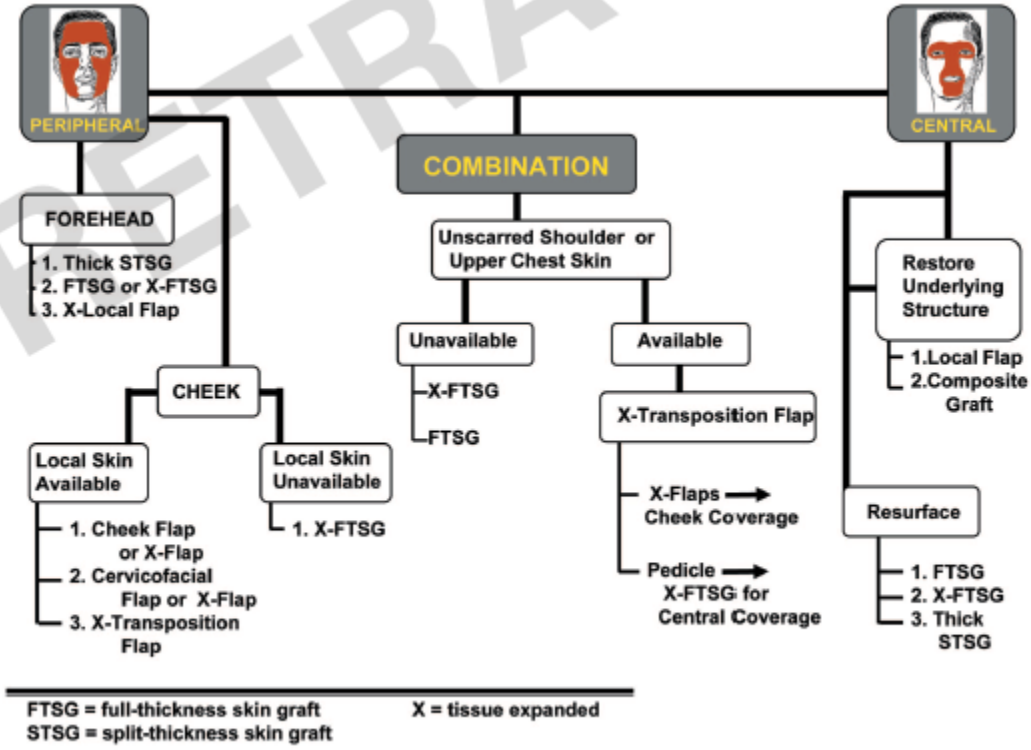

Fig. 4. Algorithm for reconstruction of large facial deformities.

Spence, R. J., "An Algorithm for Facial Reconstruction Using an Expanded Transposition Flap." *Plast. Reconstr. Surg.* (2008); 121:798.

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E. Neck

1. What measures can be used in the burn unit to minimize neck contracture occurrence? (Efforts to avoid patient positioning that flexes the cervical spine/neck.)

2. A patient has a severe anterior cervical burn contracture and you plan release of same. What are the anesthesia considerations? (The principal complication is the airway. An awake nasotracheal intubation and/or fiber optic intubation is the contemporary management. More traditional techniques are the release of the contracture under local anesthesia and then routine intubation, not often employed today.)

3. Discuss the technical points and release of neck scar contractures. (Transverse release should be placed, as in other joint scar releases, from mid axial to mid axial point which would imply the mid cervical lateral line. Less severe contractures would not require that extent of an incision. With respect to depth, platysma is almost always incised and released and the head/neck progressively extended as the contracture release is accomplished. Not infrequently, the cervical fascia beneath the platysma needs release as well. Vertical lateral incisions, if scar excision is accomplished, should be designed as a z or an s to minimize contracture.)

4. Discuss the use of Integra and release of neck burn contractures. (Placement of Integra, a dermal substitute, is done after contracture release and overlapped 2-3mm onto native skin. Either a bolster or VAC is employed for the next two weeks in combination with a splint, fabricated by occupational therapy. After two weeks the silicon layer is removed and a thin split thickness skin graft placed over the Integra. The advantage is, if a dermal template is not utilized, a thick split thickness graft, 14+/1,000 inches in thickness will be necessary. Regardless of technique, postoperative splinting for 4-6 months is necessary to prevent recontracture.)

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VII.D.1. Burn Reconstruction (Face, ears, neck, nose, eyelid, cheek)

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VII.D.2 Burns: Burn Reconstruction

(Hand/Upper Extremity, Axilla, Breast, Foot, Splints and Scar Management)

A. Splints and Scar Management

1. Discuss the correct positioning for application of splints to the elbow and the wrist. (The elbow should be splinted in full extension minus 5-10°; the wrist splinted with slight extension 15-20° with neutral radial-ulnar deviation and in neutral pronation-supination.)
    a. To splint the wrist the splint should be how far distally? The distal palmar crease, the proximal palmar crease, or the proximal carpal row? (The distal aspect of the proximal carpal row. If the splint is to mid palm or more distal, MCP joint flexion can not be obtained because of the blocking action of the splint. See "Hand VIII A" for further discussion of hand splinting.)

Table 1. POSITIONING FOR ACUTE BURNS

Region/ Joint	Position of Function	Splints	Accessory Devices
Neck	Neutral or slight extension Neutral rotation	Neck conformer splint Triple component neck splint	No pillows under head Small pillow under neck
Shoulders	Neutral rotation/elevation	Figure-of-eight soft splint	Soft vertical roll between scapulae
Axilla	Abduction: 70°–80° Horizontal adduction: 15°–20°	Airplane splint	Arm trough Bedside table Abduction wedge
Elbow	Extension: −5° to −10° Forearm: neutral to supinated	Volar based extension splint	
Wrist	Neutral to slight extension Neutral radial/ulnar deviation	Intrinsic-plus splint Wrist cock-up	
Hand	Dorsal/circumferential burn MCP joints 50°–70° of flexion IP joints in extension Palmar burns MCP and IP joints in extension Thumb in full abduction Digits abducted	Intrinsic-plus splint C-bar for thumb Hand extension splint Halo or horseshoe splint	K-wire fixation Webspace inserts

Jordan RB, Daher J, and Wasil K, "Splints and scar management for the acute and reconstructive burn care", Luce EA, ed., *Clinics in Plastic Surgery*, (January 2000); 27(1):72.

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2. What element of the splinting regimen may need to be added during the proliferative phase of healing? (Some type of progressive motion such as the use of a rubber band traction to improve MP joint function, levered against a static wrist splint. The protocol is a combination of splinting and exercise.)

3. What is a method to conservatively address a PIP joint contracture with splinting? (A "joint-jack" or wire spring PIP extension splint as pictured in one of the readings.)
    a. A similar approach to elbow contracture? (If less than 30° serial splinting may regain nearly full extension. A commercial dynamic elbow splint is also available.)

4. What is a common sequela of chin and anterior cervical burns in addition to neck contracture? (Lower lip eversion with loss of oral competence and exposure of the mandibular dentition.)
    a. The principal objective in splinting of perioral burns is? (Prevention of microstomia)

5. What is the common thread or element of the use of garments to treat hypertrophic burn scars? (Pressure)
    a. The physiologic basis? (The mechanism is not truly understood but what occurs is reorientation of the collagen bundles, diminished levels of ground substance including chondroitin and hyaluronic acid. Whether these effects are due to decreased fibroblast proliferation and thus unopposed collagenase activity or some other molecular mechanism is not known.)

6. Describe some methods for both early and late compression. (Early compression can be done with elastic bandages, Coban, ect to reduce tissue edema. Late compression can be provided by custom-measured and fitted garments that can provide even uniform pressure to burn scars. Commercially available garments are manufactured by Jobst.)

7. Describe the use of facial mask in burn scars. (Usually composed of a transparent mask manufactured after a plaster mold is made. Such masks are almost always of a single unit. A variety of inserts can be placed within the mask itself.)

B. Hand/Upper Extremity

1. Describe the position of the burn claw hand deformity. (Wrist flexed, MP joints extended, PIP joints flexed with boutonierre deformity with early DIP joint flexion and later extension. Thumb adducted with contracted first webspace shortened with recurvatum deformity of the MP joint.)

2. What are the three processes responsible for burn wound edema, and how does that edema have a detrimental effect on hand function? (The three processes are direct burn injury, "leaky" capillaries due to a circulating vasoctive kinin, and a change in oncotic pressure due to leakage of protein from an intravascular to an extravascular or interstitial site. Dorsal edema of the hand limits skin flexibility and so decreases MP joint flexion; the fluid is protein rich with matrix metalloproteinases that set the stage for inflammation and scarring.)

3. In what position should the MP joints be splinted and why? PIP joints? The thumb? (MP joints should be splinted in flexion, 80º, because the collateral ligaments are stretched in that position; the PIP joints in extension to prevent contracture and adherence of the volar plate and check-rein ligaments, and the thumb in abduction with some opposition. This posture is the position of function.)

4. What is the role of wrist splinting in management of the burned hand? (The wrist in extension will utilize the tenodesis effect that is secondary to the stretched long finger flexors that possibly will facilitate splinting of the MP joint in flexion.)

5. Failure to properly splint the thumb can lead to what? (First webspace adductor contracture; hyperextension deformity of the MP joint)

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6. What is the anatomical explanation for the susceptibility of the little finger MCP joint to a hyper extension deformity? (The unique mobility of the carpometacarpal joint, as well as the loss of the normal transverse palmar arch because of soft tissue contracture.)
    a. What can be the consequent effect on the PIP joint? (If sufficient hyperextension deformity of the MCP joint occurs, a zigzag deformity results in a PIP joint contracture or pseudo-boutonniere.)

7. Describe the indications and the technique of an upper extremity escharotomy? (Radial and ulnar incisions from just above the elbow to the level of the wrist.)
    a. What is the technique for digital escharotomy and intrinsic fasciotomy? (Mid-axial incisions for release of digits and two dorsal hand incisions for release of the intrinsics; both of these procedures are controversial whether they have a beneficial role.)

8. What is the consequence of full-thickness palmar burns? (Cupping deformity of the hand that pulls the radial and ulnar components centrally with loss of full abduction of the thumb and conversion of the palmar mild concave curve to a severe or cup deformity.)

9. What is the underlying or consequent problem related to failure to splint the PIP joint in extension in digital burns? (If the middle phalanx is in flexion, the volar plate and check-rein ligaments are slack or relaxed which can lead to contracture of same. The base of the proximal phalanx may also place pressure on the central slip particularly with burn loss of the overlying thin skin with the result of attenuation, exposure, and ultimate loss of the central slip. Loss of the central slip can lead to a boutonniere deformity; contracture of the volar plate and check rein ligaments can lead to flexion contracture of the PIP joint.)

10. What is a maneuver that will determine severity of PIP joint contractures in burned hands? (If the flexion contracture can be relieved by flexion of MP joint and extension of PIP joint, complete

extension with that maneuver is type I, incomplete is type II, and fixed PIP joint position regardless of MP joint position is type III.)

11. Discuss the readings management plan of the burned hand in the subacute period including pros and cons. (Only prompt application of compression hand dressings and elevation prior to the onset of burn wound edema of the hands allows proper splinting. This position, described in a prior question, is maintained for 4 or 5 days with the use of tissue pressure monitoring if necessary. Depending upon the patient's general condition, extubated and has diuresed the excess fluid, removal of the dressings at 5 days permits inspection of the burn wound and initiation of a program of therapy. The patient is replaced in bulky dressings for a day or two and then nighttime and daytime splints when not in therapy with burn wound dressing changes. If still intubated with little ability to cooperate, reapplication of the bulky dressing for an additional two days is advisable. Because of the risk of burn sepsis beneath a closed dressing after seven days, a switched burn wound dressing should be accomplished. A decision about burn wound excision needs to be made if the burns are clearly 3rd degree. If not, that decision is deferred until some point 15- 20 days post burn, if healing has not taken place substantially or almost completely by that juncture. This plan should be aborted at any point that the patient fails to continue to progress with evident decrease in ROM and participation in daily therapy.)

12. Discuss the potential benefits and limitations of tangential excision of hand burns. (Tangential excision defines the depth of burn, but commits to grafting, which may lead to unnecessary excision and grafting of wounds that would otherwise heal satisfactorily.)

13. What is the underlying or consequent problem related to failure to splint the PIP joint in extension in digital burns? (If the middle phalanx is in flexion, the volar plate and check-rein ligaments are slack or relaxed which can lead to contracture of same. The base of the proximal phalanx may also place pressure on the central slip particularly with burn loss of the overlying thin skin with the result of attenuation, exposure, and ultimate loss of the central slip. Loss of the central slip can lead to a boutonniere deformity because of the progressive volar displacement of the lateral bands;

contracture of the volar plate and check rein ligaments can lead to flexion contracture of the PIP joint. The risk of burn dressing sepsis after seven days, a switched burn wound dressing should be accomplished.)

14. Give a classification of burns syndactyly.

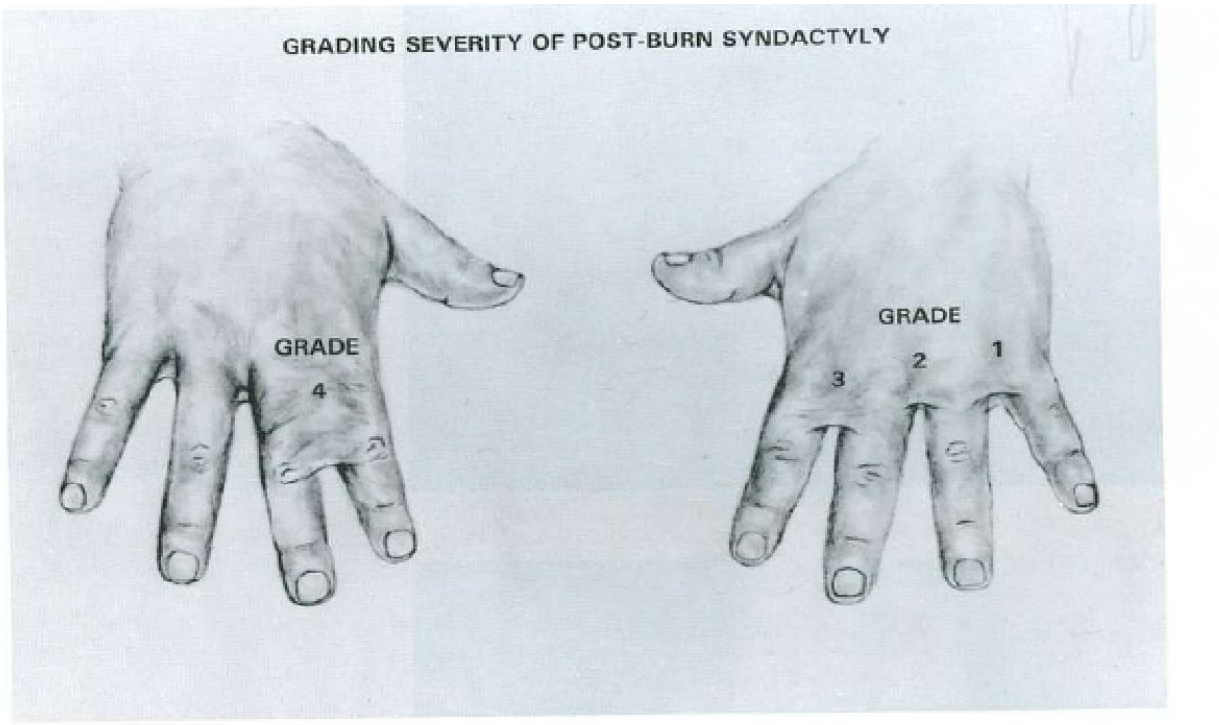

Figure 10–16. Alexander et al classified web space contractures as 1, 2, 3, 4, as shown in this illustration. (From Alexander et al.[7] Reprinted with permission.)

Achauer BM, Chapter 10"Burns of the upper extremity", Burn Reconstruction, Thieme Publisher Compant, (1991); 116.

15. A number of procedures have been described for the release of burn syndactly. What is the problem and what is the common thread throughout those procedure options? (Secondary contracture of the webspace and distal migration of the dorsal, and occasionally palmar, skin components of varying severity from mild to severe, namely obliteration of the webspace, occasionally as far distal as the PIP joint. Release of quite mild forms can be done with local flaps such as multiple z-plasties but severe forms require split-or full-thickness skin grafting. The

common component of most options is release of the contracture and use of the dorsal skin to suture to the palmar skin release to define the cleft and then placement of skin grafts in the resultant donor site defect. The use of multiple smaller flaps are at risk for ischemic necrosis and secondary healing with the final result an incomplete release of the syndactly.)

16. Discuss the prevention of and address of first webspace contracture. (As outlined earlier, proper positioning of the thumb into a posture of abduction-opposition is important to prevent secondary contracture of skin, adductor, and first dorsal interosseous muscles. Once established, release of quite minor contractures can be accomplished with four-flap Z-plasties but much more commonly will require at the minimum split-thickness skin grafts after release. Since the adductor and first dorsal interosseous are both adductor muscles of the thumb more severe contractures will require release of the adductor fascia or in more extreme cases the muscle itself. If so, closure of the wound with a flap may be necessary.)

C. Axilla

1. What is the appropriate position to splint the shoulder to minimize axillary contractures? (Abduction and external rotation)

2. What is the argument and a specific anatomical explanation for early release of axillary contractures? (Although some may advocate to allow maturation of the burn contracture since skin contracture recurrence rate is lower with release of a mature rather than an immature contracture, some specific anatomical reasons exist for early release. These include foreshortening of the principal adductors of the shoulder, the pectoralis and to lesser degree the latissimus. Also, shoulder joint capsule contracture can occur.)
    a. What are the components of the anterior and posterior axillary folds? (The principal muscular components of the anterior axillary fold are the pectoralis major and minor; the components of the posterior axillary fold are the latissimus and the teres major.)

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3. Give a classification of axillary burn contractures. (Type I anterior and/or posterior axillary fold only which usually can be released with some type of Z-plasty or V-Y plasty. Type II is involvement of both the anterior and posterior axillary folds and usually split thickness skin grafts are necessary after release. Type III is involvement of the entire axilla except for apex or cupola and flaps may be necessary. Type IV includes severe scarring of the axilla and adjacent areas with loss of shoulder joint motion.)

4. Give the pros and cons of the following coverage methods after axillary contracture release: Z-plasties, thick split-thickness skin grafts, fasciocutaneous flaps. (Z-plasties can be useful for very mild axillary contractures but involve the use of transposition of burn skin and are subject to necrosis of the tips and recontracture. Split thickness skin grafts are a reliable method, yet require long term splinting of the axilla, and occasionally re-release and re-grafting. Fasciocutaneous flaps provide full-thickness coverage and are not likely to recontract but often are not available because of burn involvement of the adjacent soft-tissue and also the donor site can not be closed primarily.)
5. What principle should be followed with release of axillary contractures? (First, the principle that joint release usually involves skin/soft-tissue release from mid axial point to mid axial point of the joint; second, adequate release of both the superficial and, rarely, deep fascia.)

6. The readings describe the use of thoracodorsal artery perforator flap. What have we learned about the TAP flap? (In only about 60-65% of patients is a true thoracodorsal perforator present, the implication is the absolute necessity for a Doppler identification of such perforator as with all perforator flaps.)

7. The vessel that supplies the scapular flap and parascapular flap arises from the triangular space. Name the vessel and define the three sides of the triangular space. (The vessels are the circumflex scapular and the triangular space is bounded by the long head of the triceps on the lateral aspect, the teres minor superiorly and the teres major inferiorly. This anatomy also discussed in the Head/Neck Tumor reconstruction module IV B2.)

a. What is the parascapular flap? (A flap based just on the descending or vertical branch of the circumflex scapular vessels. The drawings on the reference included, Duncan and Smith, on two successive pages illustrate actually the parascapular flap only. The vascular supply to the scapular flap is the transverse branch.)

8. Alternate option for the treatment of axillary contractures is use of Integra. Describe same. (The protocol is much the same as the use of Integra elsewhere, namely, the complete release of the contracture, placement of the Integra with or without a VAC, followed 2-3 weeks later by application of thin split thickness skin grafts. In all likelihood, prolonged splinting would be necessary in this scenario as well.)

D. Breast

1. Essentially, what are the three reconstructive problems in the burned breast? (One, full thickness burns in prepubescent girls can destroy all or a portion of the breast bud and have an effect on ultimate breast growth. Two, extensive involvement of the skin envelope of the breast may result in unyielding contracted soft tissues that do not permit proper breast development in adolescence. Third, the nipple areola complex may be destroyed and may require reconstruction.)

2. Discuss timing and technique of release of a constricted breast mound. (The timing is best accomplished when the opposite breast, if present, has begun development. The inframammary crease is mapped out on the contracted breast and a 180 degree release is accomplished through scar tissue and down to the deep fascia. To create a natural cleavage plane. Thick, 0.016" split thickness skin grafts are placed into the resultant defect both on the inferior pole of the breast and on the adjacent chest wall. Certainly, one alternate option to split-thickness skin grafts is the use of Integra, a 2-3 week hiatus and then followed by secondary skin grafts. The combination of scar contracture release, placement of a tissue expander, and Integra is probably not recommended as a combined procedure since inevitably the inferior portion of the tissue expander will be beneath the Integra with prolonged and incomplete vascularization of the Integra as a consequence.)

a. Superior pole restriction. (Basically the same principle, although more commonly some scar excision in addition may be accomplished. Pressure garments are worn in the extended postoperative period as well as custom-made silicone molds.)

3. What was the sequence advocated by the readings? (The readings cite very substantial experience with burn breast reconstruction and advocates release when the retardation of breast development by the burn scar is detected, proposed excision of scar contracture of the superior portion of the breast and a deep incisional release to the chest wall to create a natural cleavage plane. A thick split-thickness skin graft is placed into the defect. One year later the expander is placed and ultimately an implant. Secondary subpectoral breast implants with or without prior tissue expansion may assist in achievement of symmetry and an adequate breast mound.)

4. Why not place a tissue expander as a first aid, followed by a infra mammary release and skin grafts? (Thickened fibrotic skin will expand poorly. Incision/excision of scarred skin-soft tissue will set the stage for successful expansion. See the following questions.)

5. If tissue expansion is employed, what is the timing and the purpose? (*After* adequate release of the breast mound has been accomplished with split thickness skin grafting *and* the estimation is that implants alone will not overall provide balance then a tissue expander can be placed in the subpectoral plane and exchanged with a second stage breast implant.)

6. What options exist for reconstruction, not release, of the breast mound? (The usual options in breast reconstruction. A valuable alternative to, for example, the standard latissmus flap is the thoracodorsal perforator flap, if the lateral chest wall skin and soft tissues are not badly damaged and if a perforator can be identified.)

7. What are the options in nipple reconstruction? (Not many. The burned skin will obviate the use of standard techniques such as the skate flap, ect. that rely upon dermal circulation. A composite graft of toe pulp may be an option as well as the use of alloderm stuffed into the burned skin envelope or

a circular release and purse-string suture to mimic a nipple, followed by full thickness skin graft and perhaps tattoo to reconstruct areola.)

E. Foot

The readings established the importance of burn injuries to the feet since foot burns are one of the indications for a referral to a burn center.

1. What are the important factors to be included in the assessment of a burn of the foot? (Some are fairly obvious such as depth of burn. Others deserve emphasis such as weight bearing vs. nonweight bearing components of the foot as well as associated circumferential burn injury of the lower extremity.)

2. Discuss stance and swing phases and weight loading during gait. (60% of the duration of the gait is spent in the stance phase which is initiated once heel strike occurs and terminates with the push off of the fore foot. During the stance phase the weight load is split between the metatarsal heads and the heel. Of the metatarsals the first supports 1/3 of the load with the remaining distributed over the full remaining metatarsals although the fifth may share more compared to the other three. Once the load is shifted from heel to the forefoot, some of that increase in load is shifted to the second metatarsal head. Discussed also in lower extremity module, VI A2.)

3. What is the role of the plantar fascia? (Prevents longitudinal collapse of the arch of the foot, provides a platform to support body weight. The fascia is the principal stabilizing structure of the foot.)

4. Define the “rocker-bottom” foot deformity and its treatment. (The terminology refers to soft-tissue contracture of the dorsum of the foot as well as extensor tendon shortening and plantar subluxation of the phalanges and a complete reversal of the normal convex-concave plantar arch. Treatment requires release of the contracture often including tenotomy of the extensors and application of thick skin grafts. The toes may need to be K-wired into extension.)

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5. Describe the following: medial plantar flap, flexor digitorum brevis flap, abductor hallucis flap. (Medial plantar artery is a major pedal branch of the posterior tibial artery and a flap consisting of the instep glaborous skin can be fashioned on this artery; the muscle bellies of the short flexors of the toes can be rotated posteriorly to cover small heel defects as can the principal abductor of the big toe, the abductor hallucis.)

6. Describe the principal limitations of free tissue transfer for foot defects. (The use of free muscle transfer with overlying skin graft provide less shear and more durability than myocutaneous flaps. The problem is the use of insensate tissue for the weight bearing portion of the sole of the foot.)
    a. Would the provision of neurotized flaps be of benefit in this situation? (Theoretically, yes; practically, the results are no different than non-neurotized flaps.)

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