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Anesthesia and the Thermal Injury Patient: A Current Literature Review

John Hoscheid

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John Hoscheid, BAN, RN, SRNA

University of North Dakota

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Abstract

Problem and Purpose: A major burn is one of the most devastating physiologic and psychologic insults known (McCall & Cahill, 2005). The 2007 American Burn Association fact sheet estimates that each burn center has approximately 200 admissions each year and that the other 5,000 hospitals admit approximately 3 burn injuries on average per year. The recent approach to move some burn care to the rural setting will have a major effect on the rural anesthesia practice. The purpose of this study was to enhance the Nurse Anesthetists ability to care for the burn patient.

Conceptual Framework: This literature review study is based on a physiological framework of the burn process (Sitzer, 1991). Current literature has been based on the physiological response of the burn victim.

Analysis Methods: Extensive analysis of recent published literature on the care of the burn victim was completed. Further analyses of treatments that may be the future of burn care were completed. The literature review focused on the most recent five years of research.

Results: Fluid resuscitation continues to be a changing field of study. A large portion of treatment facilities continue to use crystalloid fluids for resuscitation. There are certain times where colloids are used. Pulmonary injury is also an ongoing threat. When encountered it is a significant key to morbidity and mortality. Many treatments used in other areas of health care are showing promise in the care of the burn victim. Treatments to decrease the effects of inflammatory cytokines are coming into favor in the care of the burn victim.

Implications: Much of the research discovered and dispersed within this study was directed to increase the knowledge on modes of care that are accepted in the treatment of the thermal injury customer encountered in many health care facilities today. With the great need for specialized care in the health care environment, the anesthesia provider may not encounter many situations where burns are involved. This may assist in the current decrease in mortality and the shortening of the length of stay in the hospital setting (Akerlund, Huss, & Sjoberg, 2007).

Anesthesia and the Thermal Injury Patient: A Current Literature Review

A major burn is one of the most devastating physiologic and psychologic insults known (McCall & Cahill, 2005). The injuries are multifaceted and consist of multiple organs and possibly in accordance with other significant traumatic injury. The American Burn Association (ABA) estimates that approximately 500,000 burn injured patients obtain care in a medical facility ("Burn Incidence and Treatment in the US," 2007). Historically this amount has been much higher. The American Burn Association fact sheet produced in the year 2001 estimated that the number of burn patients encountered in the previous year was slightly over one million.

The most recent fact sheet released by the ABA states that there are at least 4,000 deaths per year with some aspect of burn injury. Many of those deaths were in residential fires, nearly 3,500. The other 500 deaths occurred in motor vehicle, aircraft crashes, contact with electricity, chemicals or hot liquids, and other sources (Miller, et al., 2006). The amount of deaths has decreased in recent history and the current research attributes it to the increase in education of society and the treatment of burn care.

Of the 500,000 patients that encounter some form of health care for the injuries, there are very nearly 40,000 admissions to hospitals due to the injury ("Burn Incidence and Treatment in the US," 2007). There are 132 hospitals in the United States with a self designated burn center (Klein, Nathens, Heimbach, & Gibran, 2007). More than 60% of the burn injuries result in admission to one of these specialty centers. With the improvement of transport and stabilization methods this number has increased in recent years. The 2007 ABA fact sheet estimates that each burn center has approximately 200 admissions each year and that the other 5,000 hospitals admit approximately 3 burn injuries on average per year. Many of these burn patients who seek medical help have been treated in similar ways for the last 10 to 15 years.

In many cases, the large regional burn centers are based in metropolitan areas and the anesthesia care provided is a partnership of Certified Registered Nurse Anesthetists and Anesthesiologists. If this is the case, the majority of burn center admissions that need some form of anesthesia care see a variety of caregivers. The concentrations of specialized burn care to a relatively very few specialized centers dispersed through a large geographical area requires the triage, referral and transport of many smaller non burn center facilities (Klien et al., 2007).

One mostly rural state in the mid-west contains a significant amount of hospitals with varying levels of care provided. According to the Nurse Anesthetist resource in that state, 60 percent of these hospital anesthesia programs are covered by anesthetist only groups. The anesthetist is used as a resource in the trauma stabilization in many of these facilities as well as in the operating theatre. Nurse Anesthetists are now contributing much of there expertise in the realm of pain control in the pain clinic setting as well.

The recent approach to move some burn care to the rural setting will have a major effect on the rural anesthesia practice. Many burn centers have integrated more comprehensive outpatient care in their treatment regiment (Sagraves et al., 2007). This shift will call for the collaboration between centers on the basis of patient care, cost, travel, patient satisfaction, and regional burn center capacity (Sagraves et al., 2007). A recent study on an approach to the rural care of the burn patients suggest that many patients with a burn of less than 10% body surface area could be efficiently treated in the rural outpatient setting. This study by Sagraves and partners enforces the fact that the collaboration with a burn center is a must.

Problem

Not all thermal injury patients are near a facility specialized in burn care. This fact mandates the need for patient stabilization if transport is needed. Due to the paucity of specialized burn care centers in the US, long transport times are very common (Klein et al., 2007). The outcome of these patients is very dependent on the care provided before and during the transport process (Klein et al., 2007). With the majority of the facilities in North Dakota placed in the rural setting, it would be safe to assume that the anesthesia provider would encounter some need for burn care in his or her career.

The need for education on the recent practices in the care of burn patients is needed for all providers. The down trending of the number of thermal injury patients can correlate with the increased need for education on the specific care on a semi-regular basis. Although clinical outcomes are vastly better than they were 50 years ago, new modes and techniques of care are constantly being encountered. These encounters are challenging the abilities of event the specialized care giver in the burn setting (Sheridan & Tomkins, 2004).

Purpose

The purpose of this study was to enhance the Nurse Anesthetists ability to care for the burn patient. The advent of improvements in prevention as well as treatments has drawn the need for updated education for the provider. Much of the research discovered and dispersed within this study is directed and increasing the knowledge on modes of treatment that are accepted in the care of the thermal injury customer encountered in many health care facilities today. With the great need for specialized care in the health care environment, the anesthesia provider may not encounter many situations where burns are involved. This may assist in the current decrease in mortality and the shortening of the length of stay in the hospital setting (Akerlund, Huss, & Sjoberg, 2007).

This summarization of the current research being done in the field of burn care will enhance the ability of the anesthesia provider in the aspects of recent accepted care as well as what to expect in the near future.

Significance of Study

Immediate aggressive fluid administration is universally advocated; however, the choice of fluid, timing of infusion, and endpoints to be monitored remain controversial (Ahrns & Harkins, 1999). Under normal circumstances, the loss of fluid is regained though the ingestion of water in some form or another. In many burn instances, the patient is unable to care for him or her self and there is an inability to fulfill the need by him self or her self. The burn patient is in many cases unable to replace such large volumes and therefore may suffer many clinical sequelea. Current knowledge is showing that with large amounts of fluid infused, the edema process greatly compounds itself (Demling, 2005). As stated above, the choice of fluid is of significant controversy and there are many formulas available for the resuscitation of the burn injury survivor. The selection of the appropriate formula is currently based on what was historically used by the health care facility. There may be a better way in the current research.

Airway and pulmonary injury are major causes of death in burn patients (Kovac, 2006). Inhalational injury with associated burn has consistently shown increased mortality in burned victims (Barrow, Spies, Barrow, & Herndon., 2004). The new research currently being done with respect to the care of the pulmonary care of the thermal injury patient may decrease the high mortality that is associated with these injuries. The frequency of pulmonary injury occurs in approximately 20% to 30% of burns requiring admission (Edelman, Khan, Kempf, &White, 2007). Research has shown that respiratory tract infection is the most common complication that patients endure with the thermal injury (Edelman, et al., 2007). Edelman and cohorts (2007) stated that the respiratory tract complications have emerged as the dominant killer of patients with thermal injury. If the interruption of this pulmonary injury is to be successful, current knowledge will need to be disseminated to the providers on the front line.

Pain has always been a mounting issue in the thermal injury patient. Opiate therapy has been the accepted protocol in the care of thermal injury victims. Opiate therapy is not without multiple side effects (Cuignet, Mbuyamba, & Pirson, 2005). The advanced therapy associated with future burn care may be a perfect theatre for the use local anesthetics. Advancements in pain control methods for other diseases could possibly aid in the care of the thermal injury patient.

The three topics discussed above where chosen due to the ability of the anesthesia provider to intervene in each issue. Anesthesia providers are continually providing care involving the fluids, pulmonary, and pain issues of all patients. The knowledge of the current research may provide a better outcome for the clinical environment.

Research Questions/Hypothesis

The following questions were addressed in this study:

1. Is there an optimal fluid resuscitation and calculation available for the thermal injury patient?

2. Are there new treatments available for the treatment of airway and pulmonary injury in the burn patient?

3. What is currently being studied for the alleviation of burn pain?

It is the hypothesis that there are beneficial treatments that are currently being researched and may optimize the regimen of the critically burned patient.

An operational definition of crystalloid is a substance that is smaller in chemical makeup to a colloid. The solution could be transported through a semi-permeable membrane. An operational definition of a colloid is a solution with small insoluble particles that are uniformly dispersed throughout a continuous medium that does not settle readily. It does not transfer through membranes very easily ("Merck Source Illustrated Dictionary," 2005).

Fluid resuscitation is operationally defined as the volume of fluid needed and used for the rehydration of the burn patient. This volume is determined by several measurements in clinical use today. The Parkland and the Evans formula are just two measurements used to determine the amount of volume needed for the burn patient (Bert, Gyenge, Bown, Reed, & Lund, 1997).

Inhalational injury is a diagnosis given the thermal injury patient that is found to have airway edema, inflammation, mucosal necrosis, and soot deposits found on bronchoscope evaluation (Hunt, Agee, & Pruitt, 1975). Inhalational injury is an imprecise term used for the description of a wide range of airway and pulmonary problems associated with thermal injuries (Garner & Jenner, 2005). The study done by Garner and Jenner also states that inhalational injury by thermal means can also be termed smoke inhalation.

The operational definition of pain is defined as an unpleasant sensory or emotional experience associated with actual or potential tissue damage, or described in terms of such damage (Barash, Cullen, & Stoelting, 2006).

Study Framework

This quasi-experimental study is based on a physiological framework of the burn process (Sitzer, 1991). A burn has been noted to be an injury to the dermis as well as possibly other underlying structures that have been the result of contact with heat that the skin may not be able to disperse. This heat may come from many sources, including from a combustion source. Others may come from corrosive chemicals, electricity, or a form of radiation (Kovac, 2006).

Thermal injuries have been described as significantly disfiguring in many cases. When the question arises of what someone thinks a burn is, the picture is not an elegant one. Thermal injury occurs when a heat source comes in contact with any bodily organ and the amount of heat is in greater capacities than the body can dissipate. When mucous membranes come in contact with the thermal source, be it hot liquids, direct flame, or radiant energy, damage occurs at the cellular level (Hansen, 1998). Chemical burns can occur with the contact with the skin or when inhaled as is what occurs in smoke inhalation. Internal damage can occur with the consumption of chemicals through the oral orifices. An esophageal burn has been recorded in the anesthesia setting due to the inadequate cleaning of a Transesophageal Echocardiogram utensil (Venticinque, Kashyap, & O'Connell, 2003). Electrical burns can occur with the exposure of the body to high voltage current that travels through the grounded body tissues. This may occur with standard electrical sources or can be the result of a lightning strike.

It has been widely accepted that burn injury has been separated into three different classifications for the description of the burn. The first-degree also known as the superficial burn the cellular damage is found in the epidermal layer of the skin. The skin thereby continues to maintain its barrier function. The second-degree burn, also know as partial thickness burns, can consist of destruction of the epidermis and a portion of the dermis. An injury with a full destruction of the epidermis and the dermis may also be considered in a second degree injury. Either way the barrier function of the skin is lost in the second degree injury. The third degree injury or full-thickness burn is the complete destruction of the skin and part of the subcutaneous tissue is involved (Kovac, 2006). Some texts describe a fourth degree burn as well. The fourth degree burn is used to describe a burn that has infiltrated the beyond the third degree into the tissues of the muscle and bone underlying the injury site. The Hansen text (1998) also describes the possibility of the electrical injury being classified in the fourth degree designation. It is very rare that only one degree of burn is encountered. Most of the injuries contain an assortment of depths. For instance, a second degree burn may be surrounded by an area of first degree injury. This is blamed for the manifestation of pain in the burn victim. The depth of one site may have terminated the nerve impulses, but the area surrounding this zone may have sensation intact.

Burn severity has been described in one of two ways when the assessment is needed. The rule of nines has been accepted as the easiest form of assessment. In this calculation, the anterior and poster thorax is each considered an 18% surface area. The anterior and posterior arm surfaces are considered to be four and a half percent each. The legs each have a nine percent surface area in the anterior and posterior aspects. The groin is considered one percent and the head is calculated with nine percent of the body surface area. The continually accepted way of calculating the surface area of the injury is to use the palm diameter of the patient without the fingers. This is considered to be a one percent surface area (Kovac, 2006). The rule of nine charts also makes a change in the estimation when discussing children. For instance, a one-year-old has the surface area of 19% when associated with each arm. The trunk is considered 32 percent and each leg consists of 15 percent of the body surface area (Kovac, 2006). The Lund-Browder chart also describes a useful way of describing the surface area of the burn, but is not used as often as the rule of nine charts (Demling, 2005).

The first degree burn is clinically manifested as red and painful site. Blisters may occur over the twenty four hours post burn. The second degree burn is described as redness and blisters that occur immediately after the burn. The wound is painful at this depth. When the blisters open the nerve endings may be exposed and the pain is increased. The Advanced Trauma Text considers the deep second degree and the third degree burns to be less painful. This is due to the destruction of the pain receptors in the burn site. The pain may still persist due the surrounding burn injury. Certain physiological changes occur in the burn patient. Immediately post burn there is a marked decrease in cardiac output and an increase in the heart rate. This significant change is caused by very dynamic changes in the patient at the cellular level. Cellular proteins are damaged to a significant extent in the thermal injury. This injury is the major manifestation of the edema encountered in burns. The loss of the collagen cross linking at the cell level is blamed for the creation of abnormal osmotic and hydrostatic pressure gradients. Intravascular fluid movement then to the periphery then occurs. The capillary leak remains to be a topic that little is known about and continues to be controversial through current literature (Sheridan & Tomkins, 2004).

The current idea is that the burn wound consists of a three dimensional mass of damaged tissue. The center of which consists of a zone of coagulation. The surrounding area of the coagulation site is the zone of stasis. This site initially starts as a perfused area, but soon becomes plugged with red blood cells. The occurrence of this is due to the loss of the plasma to the interstitium. The margin of the injury site is the zone of hyperemia. The direct thermal effects cause the microvasculature to dilate and the endothelial lining does not maintain the plasma and intravascular proteins (Demling, 2005). Some of the cellular damage is reversible at this time, but over the first forty eight hours the inflammation then occurs. The inflammatory response in the zone of stasis is responsible for the burn edema and the shock (Hansen, 1998).

The permeability increase of the capillaries, vasodilation, increased extravascular osmotic activity in damaged tissue, and the infiltration of the tissues by leukocytes with the release of many vasoactive substances is the culprit of the edema found after the thermal injury (Lehnhardt, et al., 2005). Burn shock encountered at these zones is due to the endogenous inflammatory mediators that include interleukins, histamine, serotonin, kinins, oxygen free radicals, lipid peroxides and products of the eicosanoid acid cascade

(Sheridan, 2002). The cyclooxygenase end products such as thromboxane, prostacyclin, and prostaglandins E and F2 have also been deemed as involved in the inflammatory process of the burn injury. Products of lipooxygenase like the leukotrines B4, C4, D4, and E4 have also been associated with the increase in edema (Sener, Kabasakal, Cetinel, contuk, Gedik, & Yegen, 2005). Thromboxane can especially be damaging to the vasculature due to the vasoconstriction and its effects on the platelet aggregation. This process may increase the ischemic area found after the initial thermal injury (Sheridan, 2002).

The amount of edema that is encountered with burns increases with the total body surface area of the burn. For example, peak edema after a 10% burn forms at approximately three hours and peak edema after 40% burn forms at approximately 12 hours (Demling, 2005). It is understood that the capillary permeability will usually decrease after the first twenty four hours of the burn (Bert, et al., 1997). After the first twenty four hours the capillary beds of the direct thermal injury will maintain the poor permeability (Hansen, 1998).

After the initial resuscitation phase the patient may encounter multi-system organ failure as well as sepsis. Hyper metabolism occurs and a process of hyperthermia may occur as the inflammatory process occurs. The profound stress response may also be a trigger for the increase in temperature.

Infection is largely inevitable with the burn victim. With the loss of barrier function of the skin and with the translocation of bacteria across the gut wall (Hansen, 1998). This occurs with the decrease in gut perfusion associated with the sympathetic response in traumatic situations. Hansen (1998) also describes that invasive line placement as well as the other responses can be associated with more edema, rhabdomyolysis, disseminated intravascular coagulation, and acute respiratory distress syndrome. All of these processes increase the likeliness of the aforementioned cytokine release and an increase of inflammation.

Review of Literature

Inflammatory process and osmotic pressures

Much study has been done recently on the inflammatory process post thermal injury and the addition of fluids to decrease the injury and increase positive outcomes. Demling has done significant research on the edema process. The study of fluid flux, or flow of fluid across capillary membranes, was advanced by the physiologist Earnest Starling in the late eighteen hundreds (Demling, 2005). This fluid flux is essentially the fact that hydrostatic pressures that force fluid out of the capillary into the interstitium were counterbalanced by colloid osmotic pressure. This colloid osmotic pressure is maintained by large proteins found in human plasma (Demling, 2005). With the loss of the proteins in the seepage of serous fluid in the burn patient it is understandable that the negative pressure of the fluid in the capillaries is lost. It was found during one study on protein loss that an average of almost 17 grams of protein are lost on a wound area of ten percent TBSA in eight hours, with a peak value around twenty grams accumulating in the first eight hours of the second day after admission (Lehnhardt, et al., 2005). Lehnhardt and cohorts discuss the need to replenish the albumin, but not within the first 24 hours of the burn.

Much discussion has occurred in recent years about the addition of albumin to the arsenal of post burn treatments (Wharton & Khann, 2001). Demling's 2005 research discusses the size, in angstroms, of the leaky capillary membranes. It was found that the albumin leaked easily from the injured site, but the larger synthetic molecules in Dextran for example would theoretically stay intravascular for longer periods of time (Demling 2005).

Hettiaratchy and Papini (2004) stated that in the first eight to twelve hours there is a general shift of fluid to the interstitial spaces. The vasculature begins to close the large spaces at different time periods post burn depending on the total body surface area and the depth of the burn (Hettiaratchy & Papini, 2004). If a mixture of colloid resuscitation is given after the first eight hours, and depending on the total body surface are of the burn, the research shows a possibility of a decreased need for large volumes to be used (Hettiaratchy & Papini, 2004).

Fluid treatment modalities in the care of burn patients

Resuscitation with standard protocols is characterized with large volumes of crystalloid fluid (Lactated Ringers) using the Parkland formula (Bert, et al., 1997). Burn patients loose large amounts of fluid form the capillary leaking which in turn pulls large protein molecules from the intravascular space (Sheridan, 2002). These protein molecules have many purposes in the body. One is to keep the colloid osmotic pressure gradient to keep intravascular fluid volume adequate (Sheridan, 2002). In recent years, there has been much discussion regarding the addition of albumin or other colloids to burn resuscitation regimes (Wharton, & Khanna, 2001).

Early tragedies caused an interest in the sequence of injury in the burn patient. After the 1930 Rialto Concert Hall and the 1942 Coconut Grove fires, various resuscitation formulas based on body weight and burn size were developed (Sheridan, 2002). Most of these formulas are still in use, in one form or another, today. The Parkland Formula is the most widely used in the United States and consists of replacement of the volumes with Lactated Ringers and is calculated by multiplying the surface area burned by the patients weight in kilograms and then by 2 – 4 milliliters (Hagstrom, Wirth, Evans, & Ikeda, 2002). This volume is to be infused over the first 24 hours post burn, with half given in the first 8 hours. The Brooke formula is a treatment calculation that incorporates Lactated Ringers at 2ml/kg/% burn with the addition of colloid at 0.5 mls/kg/% burn (Hagstrom, et al., 2002).

The Baxter formula is calculated with the four milliliters of lactated ringers multiplied by the patient weight in kilograms then multiplied by the total body surface area of the burn. Similar to the parkland formula the first half is given over the first eight hours post burn and the remaining over the next sixteen hours. The daily maintenance fluid is given concurrently (Kovac, 2006). The discussion of the fluids is rather controversial even to this day, and most cases are covered with crystalloid in the initial setting (Demling, 2005).

Hypertonic saline has been discussed as a treatment of choice in several current articles. Sheridan contributes a formula for the treatment of burns that includes such hypertonic saline. This formula adds 250 mEq/L of hypertonic saline to maintain urine output of at least 30 milliliters per hour (Sheridan, 2003). The last formula found is the Demling formula. It consists of Dextran 40 in saline and is infused at a rate of 2 ml/kg/hr, then Lactated Ringer's as needed to maintain target urine output (Hagstrom, et al., 2002).

With all of these possible formulas it is easy to find that there is no one perfect formula and that the choice of which to use is quite easily controversial (Thomas, Kramer, & Herndon, 2002).

Total burn determination for fluid need

The other factor involved in this resuscitation dilemma is that treatment is based on retrieval of information with the assessment skills of the provider dealing with the burn patient. Inaccurate initial assessment of the size of the burn injuries can result in suboptimal management of burn injuries (Wong, Heath, Maitz, & Kennedy, 2004). Wong and associates also state that studies have documented that complications arise from patients with inaccurate assessment and treatment in the burn patient. In one study, it was found that 30% of the patients studied were over resuscitated, one of them critically. In that same study, 47 % were under resuscitated, five of them critically (Hagstrom, et al., 2002).

The burn process is one that causes much edema post injury. The edema process is an abnormal accumulation of liquid in cells, tissues, or cavities of the body (Demling 2005). Edema that is formed post thermal injury is an expansion of the interstitial liquid volume (Demling 2005). Many patients with cutaneous burn injuries of more than 30% of their total body surface area had hyperpermability not only at the burn site, but also at regions distant from the injury (Enkhbaatar & Traber, 2004). This edema process has lead to known compartment syndromes and recently has lead the diagnosis of multi-organ system failure secondary to a systemic inflammatory response syndrome, which is currently the greatest source of mortality in the burn populations (Brown, & Muller, 2004).

As discussed previously, the amount of fluid used for resuscitation is based significantly on the TBSA of the burn and that there have been many difficulties in standardizing the measurement of the area burned. Several studies have suggested that over and under resuscitation can have dire consequences. The monitoring of urinary output has well been established as means for volume replacement needs (Klein, Nathens, Emerson, Heimbach, &Gibran, 2007). Continuous replacement of the urinary output has been suggested as well. This closed loop suggestion had the effect of a significant reduction in the hourly urine output measurements under target (Hoskins, et al., 2006). The literature also reminds providers that the urinary output should not be considered the gold standard, but should be used as a part of a larger multifaceted tool for the volume replacement of the burn victim (Huang, Yan, & Yang, 2005).

Volume replacement has changed very little in the operative setting when the victims are several days post burn. Large blood loss during the burn reconstruction has always required large amounts of blood replacement (Losee, Fox, Hua, Cladis, & Sarletti, 2005). Total blood volumes have been replaced. Some have reported as much as 50 percent of the TBV being lost with the treatment of one extremity (Losee, et al., 2005). There has been an idea of transfusion free burn surgery, but studies are still needed on the safety of such procedures. Today, it seems that the majority of current research requires large amounts of crystalloid in the initial setting and other blood products to be given after the first 24 hours of the burn.

Inhalational Injury

There are three factors that have recently been associated with increased morbidity and mortality in the burn victim. These factors are associated with an age greater than 60 years old, a total body surface area of burn grater than 40 percent, and inhalational injury (Kovac, 2006). If all three risk factors are found in the burn victim, there is a 90 percent chance of death occurring from the thermal injury (Kovac, 2006). Upper airway assessment and the history from the events of the burn can clue care givers into the presence of the pulmonary injury.

Direct injury from the thermal energy is usually limited to the supraglotic airways (Garner & Jenner, 2005). The injury found in the deeper areas is usually as result of chemicals being released from the thermal environment. This is due to the response that conscious victims have in the heated environment. These patients show a reflex laryngospasm that prevents the heat from continuing to the rest of the lung (Garner & Jenner, 2005). This is why most burn injuries do not have associated inhalational injury.

Burn victims with inhalational injury often require increased fluid amounts than the victim with only dermal injury (Endorf & Gamelli, 2007). The inhalational injury is usually definitely diagnosed with the assessment of the airway with a bronchoscopy. This information and the events of the burn will determine the type of treatment for the patient. Either way the injury must be assessed as soon as possible because intubation of the trachea may be impossible if edema occurs. Intubation for airway protection and possible ventilatory support should be considered early in the phase of resuscitation (McCall & Cahill, 2005)

The lung is often the first organ to undergo failure in the dying burn patient. Infection of the respiratory tract is the most common complication in the burn patient (Edelman, et al., 2007). With the emergence of the discovery that respiratory complications from inhalational injury have a significant effect on the outcomes of the victim current research has been looking for treatments to intervene.

The inhalational injury affects the lung similarly to other tissues of the body. When the organ is injured, it begins the inflammatory process. With the lungs this inflammatory process can progress to pneumonia. If associated burns with the inhalational injury patient were at least 20 percent of the TBSA, there was a pneumonia rate of at least 60 percent (Edelman, et al., 2007). In the study done by Edelman et al., they found that optimal outcomes were discovered with supplemental oxygen and significant pulmonary toilet. The pulmonary toilet protocol included frequent suctioning as well as frequent bronchoscopy assessment. Nebulized albuterol, N-acetylcysteine, and in some cases heparin were given to the inhalational injury patient with pneumonia. Most of the patients with the inhalational injury were intubated and on mechanical ventilation. Volume control ventilation was the one of choice and tidal volumes of five to seven milliliters per kilogram were used and permissive Hypercapnia occurred when necessary (Edelmen, et al., 2007). Pressure control and high frequency jet ventilation were also used, but only on occasions where volume control was inadequate.

Monitoring of oxygenation is often difficult with the burn victim. The damage to the peripheral tissues frequently makes it difficult to assess the oxygen saturation in these patients. Technology has incorporated new knowledge and has developed the capability to monitor saturations within the esophageal membrane. This esophageal pulse oximetry probe can be easily placed with little to no contraindications and has been shown to be accurate in patients undergoing surgical procedures (Pal, Kyriacou, Dumaran, Fadheel, Emamdee, Langford, & Jones, 2005).

Future of Pulmonary Care

Current research has shed some light on the multisystem failure treatment modalities. The new research has focused on the cause of the inflammatory process and how to interrupt it. It has been found that plasma concentrations of vitamin E are significantly decreased in the thermally injured patient and was associated with an increase in mortality (Morita, Traber, Enkhbaatar, Westphal, Murakami, Leonard, & Cox, 2006). Incidentally, Morita and cohorts also found that there was a decrease in circulating vitamin E in the ARDS victim as well. This study found that, if sheep had been given large doses of vitamin E prior to the burn, there was an improved clinical response to the burn injury. Obviously it is impossible to know that a patient will receive a burn and therefore providers will be unable to give such large doses. This treatment may in the future be proposed to those with a high possibility of inhalational thermal injury, but it is not of significant help in the current clinical setting. Morita et al. (2006) discovered that by increasing the levels of vitamin E an associated improvement in pulmonary function was noted. This was found with the improved oxygenation and a reduction in pulmonary micro vascular leakage. Morita and cohorts also found that oral vitamin E also prevented an increase in reactive nitrogen species of the 3-nitrotyrosine variety. During the study it was found that paralytic ileus was common in the burn patient and decreased the absorption of the vitamin from the gastrointestinal tract (Morita, et al., 2006).

The oxidation damage to the lung was decreased with the nebulization of the vitamin E directly onto the lung tissues. Improvement of lung damage was also found with the nebulization of sterile saline (Enkhbaatar & Traber, 2004). Aerosolized tissue plasminogen inhibitor also showed improvement of pulmonary function when tested with sheep after burn and smoke inhalation in the lab setting (Enkhbaatar, Murakami, Cox, Westphal, Morita, Branntly, Burke, Hawkins, Schmalstieg, & Taber (2004). With this in mind, Morita and group (2006) found through study that vitamin E prevented the edema formation of the bronchi and decreased pulmonary micro vascular leakage and therefore decreased the amount of blockage noted post inhalational thermal injury.

As previously discussed, many inflammatory mediators are released as a result of the burn process. Leukotrienes, one of the major products of the lipid peroxidation mechanism, have been a topic of study recently. Historically the leukotriene blockers have been used in the treatment of asthma and other lung diseases. Sener and cohorts (2005) studied the effects of leukotriene blockers on pulmonary thermal injury and found it to be beneficial. Their findings illustrate that leukotriene receptor antagonism reverses the inflammatory reaction to thermal trauma in the lungs and other organs. This research has implicated that there may be a role for these chemicals in the treatment of inhalational injury as well as other multisystem organ failures (Sener, et al., 2005).

Sener, Sehirli, Velioglu-Ogunc, Ercan, Erkanli, Gedik, & Yegen (2006) also worked with the idea that by decreasing the significant increase in metabolic rate of the thermal injury patient the inflammatory response with be diminished. The findings of the study were found by decreasing the metabolism rates in rats with the chemical propylthiouracil. This chemical produced a hypothyroid state in which less neutrophil infiltration occurred and therefore less inflammatory cytokines were produced. This therefore diminished the amount of burn induced multisystem failure in the injury (Sener, et al., 2006). Although this study was done in the laboratory setting it does show promise for the future of care in the burn inflammatory setting.

Anesthesia and Pain Control Measures

Current literature states that there is no induction and maintenance agent that is any better than any others. Most of all of the anesthetic agents have been used for the inductions and like all other anesthetics it should be planned for each patient specifically. Propofol has been used successfully in the thermal injury theatre and in some cases may decrease the amount of stress induced gastrointestinal ulcers. Paralyzing agents are needed for the rapid sequence induction with intubation. Succinylcholine has been used in the past and historically has not been used after the first eight hours of the burn. This is due to the up regulation of acetylcholine receptor sites and the large amount of potassium release when stimulated by succinylcholine. Current research states that it should be avoided after the first four hours and not used until at least one year post thermal injury. Non depolarizing agents can be used but the dosage must be increased in those patients with greater than twenty five percent surface area burn (Kovac, 2006).

There is sufficient literature to support the use of all vapor anesthetics. Caution is advised if halothane is used and there is epinephrine in the local anesthetics injected or soaked in the gauze covering graft sites. This is due in part to the hypersensitivity to catecholamines with halothane. The other anesthetic agents can be used without issue. Much of the time sevoflurane and desflurane are used due to the cost and the ability to wake the patient sooner than that with the other agents (Kovac, 2006).

Etomidate is no longer a drug of choice in the burn setting as Etomidate can have an adrenal suppressant effect. In the long term, this can be detrimental to the resuscitation of the burn victim (Kovac, 2006).

It has often been found that even with high doses of opiates and benzodiazepines it has been difficult to control burn pain. At a certain point the analgesic effects peak and the side effects continue to be exposed in the patient. Current literature on the use of dexmedetomidine has shown some promise. Dexmedetomidine is an alpha two adrenergic agonist that was approved for use by the Food and Drug Administration in 1999. It was released for the use in short term sedation of patients who are on ventilators in the intensive care setting (Walker, MacCallum, Fischer, Kopcha, Saylors, & McCall, 2006). Dexmedetomidine has been shown to significantly decrease opiate requirements in adults when compared with propofol (Venn & Grounds, 2001). Hypotension and bradycardia are the most common side effects of dex. It seems that these side effects were more prevalent with the loading doses of dexmedetomidine (Walker, et al., 2006).

Patients that have burns severe enough for admission to a health care facility warrant the use of opiates for the pain experienced after the thermal injury (Patterson, Hoflund, Espey, & Sharar, 2004). Potent opiates are the mainstay for the control of burn pain in the current literature (Kovac, 2006). Patterson and cohorts (2004) explained that the opiate group of analgesics is most commonly used because they are potent, the benefits and risks are familiar to many providers and they can provide some dose dependent degree of sedation. This sedation may be very helpful in burn cares after the initial injury. Opiates are tolerated well with intravenous injection especially when abnormal gut motility is in question. Intramuscular injection of medications is avoided in trauma due to the inadequate rate of absorption with the sympathetic responses noted (Patterson, et al., 2004). Transmucosal administration is also a possibility in the burn patient especially with those experiencing poor IV access and with children (Patterson, et al., 2004).

Ketamine has been a popular medication with dressing changes in the thermal injury victim with advantages that include analgesia, dissociative anesthetia, sympathetic stimulation, and minimal respiratory depression (Kovac, 2006). Ketamine, with its absence of respiratory sequelea, suggest that it may also be the drug of choice for induction in patients experiencing severe facial burns (Ceber & Hayrettin, 2006). Ketamine given alone has been shown to produce severe hallucinations. The administration of a benzodiazepine has been shown to decrease the frequency of such reactions (Gilger, Spearman, Dietrich, Spearman, Wilsey, & Zayat, 2001).

In experimental studies with burned rats, the use of ketamine has been correlated with a decrease in the levels inflammatory cytotoxins. After being burned, the rats were found to have increased levels of some interleukins and tumor necrosis factor (Ceber & Hayrettin, 2006). Ketamine has also been known to suppress the production of tissue necrosis factor, Interleukin six, and reactive oxygen metabolites in human blood (Larson, Hoff, Wilhelm, Buchinger, Wanner, & Gauer, 1998). Ketamine has been studied in the form of peripheral creams or injections. Oatway, Ried, and Sawynok (2003) studied the effect of injections of ketamine on the burn site. This study showed that ketamine also has some antihyperalgesic effects at the injury site. This same study used amitriptyline and the researchers were able to prove that this chemical has analgesic properties as well as antihyperalgesic properties (Oatway, et al., 2003). Again this study was done in the laboratory environment with rat subjects.

Local anesthetics and opiods medications have been injected into the subarachnoid space for the control of surgical pain. This type of anesthetic has also been used for the alleviation of labor pain. Currently studies have focused on other medications that could be used to bathe the spinal cord for the alleviation of pain. Gabapentin, also known as Neurontin, is one such drug. It is a structural analog of y-aminobutyric acid (GABA) which is a common chemical in the body. In anesthesia anything that potentiates GABA may enhance anesthesia. Gabapentin can also alter the hyperpathic state observed after nerve injury states (Rosner, Rubin, & Kestenbaum, 1996). It has been suggested that Gabapentin may consist of a new class of drugs, the gabapentinoids (Jun & Yaksh, 1998). Although gabapentin may reduce N methyl D aspartate evoked currents, it needs such high concentrations when injected intrathecally that it may not be of use in the future (Jun & Yaksh, 1998). This procedure continues to need further research for the treatment of thermal injury patients.

Various other techniques have been used to alleviate pain in the burn patient. In an attempt to decrease narcotic need regional anesthesia has been revisited. Currently regional nerve blocks, with local anesthetic, have been used for debridement and skin grafting procedures. In many instances, continuous nerve blocks have been used to overcome the short lived action of the local anesthetic. This has been debated as well. One study found that a single shot fascia illiaca block was just as effective as a continuous nerve block, but lacked the possibility of toxicity that the continuous infusion contained (Cuignet, et al., 2005). This level of blockade was also high enough, and anesthetized more of the lower extremity, to give more surface area for graft retrieval. Cuignet and cohorts also discovered that there was a lower failure rate when attempting to place this type of block. Ample support is also found for the use of continuous peripheral nerve blocks (Dadure, Acosta, & Capdevila, 2004). Peripheral nerve blocks are a great alternative for patients with severe burns, alleviating chronic background pain as well as procedural pain. The peripheral nerve block does not necessarily mean that the local anesthetic has to be injected to be useful. Local anesthetic creams are gaining popularity in the care of the burn victim. Larger graft sites do warrant peripheral nerve anesthetic, but recently the creams are in greater use for smaller graft retrieval sites (Gupta, Bhandari, & Shrivastava, (2007). We have been using regional anesthesia for other procedures and it could be incorporated into the thermal injury regimen rather easily.

Study Limitations

This study was done with the literature review of articles produced within the last five to seven years. Although much has been studied in recent burn care, little change has occurred. The project was limited to the three topics discussed and it is obvious that there are many other areas in burn care that could be covered.

Discussion

Burn surgery is a young specialty in an environment rich with organizational and technical innovations (Sheridan, 2003). To continue to provide optimal care much must be done to educate the players on the burn care team. With the significant amount of education that is being done with the care and prevention of fires, the number of burn patients have diminished. This decrease means that many providers of anesthesia may see very few burn patients in their career. The ability to maintain a level of understanding in the burn theater means that much research must be disseminated efficiently. Many new procedures have been developed and are being developed in the research setting. These procedures may revolutionize the care of the thermally injured patient.

Recommendations for Nursing Practice

As discussed earlier, the fluid resuscitation continues to be a topic of contention (Demling, 2005). There are many different formulas available for the resuscitation of these burn victims. The Brooke, modified Brooke, Evans, and the Parkland formulas continue to be used today (Sheridan & Tompkins, 2004). The Parkland formula seems to be used most often. The calculation used, multiplies four milliliters times the percent surface area of the burn and the patients weight in kilograms. This amount is then divided into two equal portions and the first half is given within the first eight hours of the burn and the rest is given in the next 16 hours. These fluids are all crystalloid in nature, usually Lactated Ringers (Kovac, 2006). The Baxter is a modification of the Parkland. It administers daily maintenance fluids concurrently with the same amount calculated for the Parkland formula (Kovac, 2006). Kovac (2006) also describes the Brooke formula that consists of two milliliters of crystalloid multiplied by the surface area of the burn and the patient weight in kilograms. This is also given in the same fashion as the others. Half is given in the first 8 hours and the other half in the next 16 hours. The difference is that colloid is given at the rate of 0.5 milliliters multiplied by percent burn and weight in kilograms. This colloid total is given over the next 24 hours with the normal maintenance requirement. Some of these formulas have added the use of albumin or a synthetic colloid. The synthetic colloid seems to be the best choice currently due to the diameter of the molecule. This is the volume that is maintained in the vascular the longest (Demling, 2005). With the recent knowledge on the size of the seepage sites in the capillaries there has been renewed interest in the addition of large synthetic colloids to the arsenal of the burn patient providers. The key seems to be to maintain at least one milliliter per kilogram per hour urinary output. This has had the greatest effect on maintaining organ perfusion (Hoskins, et al., 2006)

Much work continues with the inaccurate estimation of burn surface areas. It is widely accepted that the palmar surface of the victims hand is equal to one percent of the body surface area. This measurement should be adhered to and this will alleviate many miscalculations. These inaccuracies cause much trouble with the volume resuscitations that ensue after the burn process (Sagraves, et al., 2007). Over hydration can cause increased edema in the pulmonary vessels as well as in the periphery. This increase in edema can potentiate the inflammatory process and worsen the multisystem failure that may occur (Sagraves, et al., 2007).

The anesthetic implications have not changed much in recent years. The induction medications continue to be similar to any other case. Propofol may be a better choice in the future. Etomidate is not recommended due to the adrenal suppression, and thiopental cost has risen due to its decrease in usage. Paralytic agents have been modified and the use of succinylcholine has diminished. It is recommended that it should not be used after the first eight hour post burn until at least one year after the burn trauma. The nondepolarizing medication recommendation has change to include an increase in the dose used if the burn surface area is greater than twenty five percent (Kovac, 2006).

There are many possibilities in regional anesthesia care of the burn patient. Continuous versus single shot local anesthetic continues to be debated. It is agreed upon that either process will decrease the need for large amounts of opiate therapy. This therefore decreases the significant amount of detrimental side effects of high dose opiate therapy. This is not without the cost of possible toxicity in local anesthesia injections (Cuignet, et al., 2005).

Monitoring for the pulmonary injury is an ongoing task. Pulmonary toilet must be maintained in the inhalational burn victim to significantly affect the outcomes. The invention and use of tools that have been developed continue to aid in the care of these burn patients. The carbon monoxide and methemoglobin oxygen saturation probe will have a significant effect on the care of the inhalational injury victim. Results are rapid and the treatment can be initiated earlier with this utensil (Barker, Curry, Redford, & Morgan, 2006).

Recommendations for Nursing Education

Nursing education needs to be centered on the assessment of the burn injury. As discussed earlier the determination of the burn size makes a significant effect on the treatment of the burn patient. Under treatment has been shown to increase morbidity and mortality. Over estimation of the burn injury has also increased the length of stay at the hospital and can cause issues with pre-existing disease processes with these patients.

The availability of other treatments is continuously changing and the nurse provider must be aware of the updated information in the treatments of thermal injury patients. Careful observation of the current research will increase the competency of the nurses that provide care to the burn victims. Educational programs must update the curriculum with research findings in the literature versus only teaching what has been printed in the educational text books.

Nursing education could also increase the education in the care of patients with regional anesthetics. This type of anesthesia has obviously increased the comfort levels of patients and has decreased the opiate use in many patients. If nursing can decrease the many side effects of the opiate therapy, the burn sequelea may not be as significant.

Nursing Research

Pulmonary injury and the inflammatory process will be an interesting topic in the future. It seems that, as the study of the inflammatory chemicals that are produced after a thermal injury continues, there is significant promise in the long term care of these victims. Pulmonary sequelea continue to be the most significant predictor of mortality in the burn victim and much more study must be done.

Ketamine has come back in favor for the care of these patients. New studies have found that it may have effects on nitric oxide and tissue necrosis factor. Ketamine and amitriptyline may be used in the periphery in the future. Laboratory studies have found that ketamine exhibits antihyperalgesia as well as amitriptyline. Amitriptyline has also shown to have some local anesthetic actions (Oatway, et al., 2003).

Intrathecal medications for the alleviation of pain have been used in the recent past. These medications have been limited to local anesthetics and opiates. The ongoing research to find more ways to control pain may have a significant impact on burn care. Jun and Yaksh (1998) have shown promising work in the laboratory setting injecting

Gabapentin and 3-Isobutyl y-Aminobutyric Acid intrathecally to rats. This could expand the pain control measures of the provider for thermal injury patients.

Research needs to continue with the multiple uses of many medications. Many of the side effects of medications that we use today can possibly have significant effect on the future care of the burn victim. Nurses are on the front line and provide the much needed care that these victims desire. Nurses have done much to advance the pulmonary care of thermal injured patients. Vitamin E, Saline, Tissue Plasminogen Inhibiter as well as many other medications may become treatments of choice in the burn victim with pulmonary sequelea as well as other critical care patients with pulmonary injury.

Anesthesia Recommendations

Burn surface area should be calculated with the rule of nines formula. A quick reference is to consider the palm of the patient as one percent surface area of the victim. After calculation of the surface area the most common formula used is the Parkland IV resuscitation formula. In this formula, four milliliters is multiplied by the surface area of the burn and the patient's weight in kilograms. This amount must be given in the first twenty four hours after the burn. The first half needs to be given within the first eight hours after the burn. This is not to be considered a hard fast rule. Stable vital signs and urinary output of at least one half to one milliliter per kilogram per hour must be achieved. Caution must be used in calculation. Over and under estimation of volumes could cause other medical issues during resuscitation. Colloid can be added in special situations. In most cases it is recommended after the first 24 hours unless measured albumin levels are below two and one half milligrams per deciliter.

Oxygen at 100 percent should be used in resuscitation, especially when high amounts of carboxyhemoglobin are suspected. Induction agents should include Propofol or Pentothal. Etomidate should not be used due to the adrenal gland suppression that may occur with use. Paralysis can be achieved with the use of succinylcholine only within the first four hours of the burn injury. After which the nondepolarizing agents must be used. If the total surface area of burn is greater than 25 five percent the amount of the nondepolarizing agent must be increased. Dexmedetomidine is considered a valid choice for medication during dressing changes and or debridement. Ketamine use can be used for anesthesia purposes. Injections have been shown to decrease tissue necrosis factors and may be beneficial in decreasing multisystem inflammatory responses. Further research is being done in regional injection of ketamine and should not be used in this fashion at this time.

Initial care of the burn patient with inhalational injury, or the question of inhalation injury must have the airway controlled as soon as possible. If the control of the airway is not done it may be impossible to control in later moments after the injury. Mucomyst and or normal saline nebulization may decrease the amount of pulmonary injury. In the future more medications may be available for the treatment of inflammation in the lungs as well as other multisystem failure. The burn care research needs to continue on the use of leukotriene receptor blockers and the use of propylthiouracil induced hypothyroidism. Anesthesia providers may be using these in the near future, but as stated more research is needed. Nebulized vitamin E may also assist in care of acute lung injury, but until more research is completed it should not be used.

Regional anesthetic nerve blocks and local anesthetic creams are showing promise in graft harvest and debridement sites. The benefit of a decreased need for opiate use for pain control has proven that it is a valid choice for an anesthetic plan.

Summary

Burn anesthesia continues to be an ever evolving specialty. With the advent of new treatments the patients have a better outlook than ever before. As the amount of burn victims that seek health care decreases, the burn experienced anesthesia provider is getting harder to find. Continued research in the care of the burn victim will aid the provider in giving the best possible care available.

References

Ahrns, K.S., & Harkins, D.R. (1999). Initial Resuscitation After Burn Injury: Therapies, Strategies, and Controversies. *American Association of Critical Care Nurse Clinical Issues, 10* (1), 46-60.

Akerlund, E., Huss, F.R.M., & Sjoberg, F., (2007). Burns in Sweeden: An analysis of 24,538 cases during the period 1987-2004. *Burns, 33*, 31-36.

Barash, P.G., Cullen, B.C., & Stoelting, R.K. (Eds.). (2006). *Clinical Anesthesia* (5^{th} ed.). Philidelphia: Lippincott Williams & Wilkins.

Barker, S.J., Curry, J., Redford, D., & Morgan, S. (2006). Measurement of Carboxyhemoglobin and Methemoglobin by Pulse Oximetry. *Anesthesiology, 105*, 892-897.

Barrow, R.E., Spies, M, Barrow, L.N., & Herndon, D.N. (2004). Influence of demographics and inhalational injury on burn mortality in children. *Burns*, *30*, 72-77.

Bert, J., Gyenge, C., Bown, B., Reed, R., & Lund, T. (1997). Fluid resuscitation following a burn injury: implications of a mathematical model of micro vascular exchange. *Burns, 23 (2), 93-105.*

Brown, TH., & Muller, MJ. (2004). Damage limitation in burn surgery. *Injury, International Journal of the Care of the Injured*, *35*, 697-707.

Burn Incidence and Treatment in the US: 2007 Fact Sheet. Retrieved April, 3, 2007. http://www.ameriburn.org/resources_factsheet.php?PHPSESSID=a5aa91ae a44226e5638277c02ca9605c

Ceber, M., & Hayrettin, B. (2006). Ketamine May be the First Choice for Anesthesia in

Burn Patients. *Journal of Burn Care & Rehabilitation, 27* (5), 760-762.

Cuignet, O., Mbuyamba, J., & Pirson, J. (2005). The Long-Term Analgesic Efficacy of a Single-Shot Fascia Iliaca compartment Block in Burn Patients Undergoing Skin-Grafting Procedures. *Journal of Burn Care & Rehabilitation, 26* (5), 409-415

Dadure, C., Acosta, C., & Capdevila, X. (2004). Perioperative Pain Management of a Complex Orthopedic Surgical Procedure with Double Continuous Nerve Blocks in a Burned Child. *Anesthesia and Analgesia, 98*, 1653-1655.

Demling, R. (2005). The Burn Edema Process: Current Concepts. *Journal of Burn Care & Rehabilitation, 26* (3), 207-227.

Edelman, D.A., Khan, N., Kempf, K., & White, M.T. (2007). Pneumonia After Inhalational Injury. *Journal of Burn Care and Research, 28* (2), 241-246.

Endorf, F.W., & Gamelli, R.L. (2007). Inhalation Injury, Pulmonary Perturbations, and Fluid Resuscitation. *Journal of Burn Care & Rehabilitation, 28* (1), 80-83.

Enkhbaatar, P., Murakami, K., Cox, R., Westphal, M., Morita, N., Brantley, K., Burke, A., Hawkins, H., Schmalstieg, F., & Traber, L. (2004). Aerosolized tissue plasminogen inhibitor improves pulmonary function in sheep with burn and smoke inhalation. *Shock, 21* (1), 70-75.

Enkhbaatar, P., & Traber, DL. (2004). Pathophysiology of acute lung injury in combined burn and smoke inhalation injury. *Clinical Science, 107*, 137-143.

Garner, J.P., & Jenner, J. (2005). Prediction of Upper Airway Closure in Inhalational Injury. *Military Medicine, 170* (8), 677-682.

Gilger, M.A., Spearman, R.S., Dietrich, C.L., Spearman, G., Wilsey M.J. Jr, & Zayat, M.N. (2001). Safety and effectiveness of Ketamine as a sedative agent for pediatric GI endoscopy. *Gastrointestinal Endoscopy, 59*, 659-653.

Gupta, A., Bhandari, P.S., & Shrivastava, P. (2007). A study of regional nerve blocks and local anesthetic creams for donor sites in burn patients. *Burns, 33*, 87-91.

Hansen, M. (Eds.). (1998). *Pathophysiology Foundations of Disease and clinical Intervention. Piladelphia*: W.B. Saunders Company.

Hagstrom, M., Wirth, GA., Evans, GD., & Ikeda, CJ. (2002). A Review of Emergency Department Fluid Resuscitation of Burn Patients Transferred to a Regional, Verified Burn Center. *Annals of Plastic Surgery, 51*, 173-176.

Hettiaratchy, S., & Papini, R. (2004). Initial management of a major burn: I-overview. *Burn Medical Journal, 328*, 1555-1557.

Hettiaratchy, S., & Papini, R. (2004). Initial management of a major burn: II-assessment and resuscitation. *Burn Medical Journal, 329*, 101-103.

Hoskins, S.L., Elgjo, G.I., Lu, J., Ying, H., Grady, J.J., Herndon, D.N., & Kramer, G.C. (2006). Closed Loop Resuscitation of Burn Shock. *Journal of Burn Care & Rehabilitation, 27* (3), 377-385.

Huang, Y., Yan, B., & Yang, Z. (2005). Clinical study of a formula for delayed rapid fluid resuscitation for patients with burn shock. *Burns, 31*, 617-622.

Hunt, J.L., Agee, R.N., & Pruitt, B.A., Jr., (1975). Fiberoptic bronchoscopy in acute inhalational injury. *Journal of Trauma, 15*, 641-649.

Jun, H.J., & Yaksh, T.L., (1998). The Effect of Intrathecal Gabapentin and 3-Isobutyl y-Aminobutyric Acid on the Hyperalgesia Observed After Thermal Injury in the Rat. *Anesthesia and Analgesia, 86*, 348-354.

Klein, M.B., Nathens, A.B., Emerson, D., Heimbach, D.M., & Gibran, N.S. (2007). An

Analysis of the Long-Distance Transport of Burn Patients to a Regional Burn Center. *Journal of Burn Care & Rehabilitation, 28* (1), 49-55.

Kovac, A.L. (2006) Anesthesia For Burn Patients. *Current Reviews for Nurse Anesthetists, 28* (8), 85-100.

Larson, B., Hoff, G., Wilhelm, W., Buchinger, H., Wanner, G.A., & Gauer, M., (1998). Effect of intravenous anesthetics on spontaneous and endotoxin-stimulated cytokine response in cultured human whole blood. *Anesthesiology, 89*, 1218-1227.

Lehnhardt, M., Jafari, H.J., Druecke, D., Steinstraessser, L., Steinau, H.U., Klatte, W., Schwake, R., & Homann, H.H. (2005). A qualitative and quantitative analysis of protein loss in human burn wounds. *Burns, 31*, 159-167.

Losee, J.E., Fox, I., Hua, L.B., Cladis, F.P., & Serletti, J.M. (2005). Transfusion Free Pediatric Burn Surgery: Techniques and Strategies. *Annals of Plastic Surgery, 54* (2), 165-171.

McCall, J.E., & Cahill, T.J. (2005). Respiratory Care for the Burn Patient. *Journal of Burn Care & Rehabilitation, 26* (3), 200-206.

Merck Source Illustrated Dictionary. Retrieved February, 20, 2006. From www.mercksource.com.

Miller, S.F., Bessey, P.Q., Schurr, M.J., Browning, S.M., Jeng, J.C., & Caruso, D.M. (2006). National Burn Repository 2005: A Ten-Year Review. *Journal of Burn Care & Research, 27* (4), 411-436.

Morita, N., Traber, M.G., Enkhbaatar, P., Westphal, M., Murakami, K., Leonard, S.W., & Cox, R.A. (2006). Aerosolized Alpha-Tocopherol Ameliorates Acute Lung

Injury Following Combined Burn and Smoke Inhalation Injury in Sheep. *Shock, 25* (3) 277-282.

Oatway, M., Reid, A., & Sawynok, J. (2003). Peripheral Antihyperalgesic and Analgesic Actions of Ketamine and Amitriptyline in a Model of Mild Thermal Injury in the Rat. *Anesthesia and Analgesia, 97*, 168-173.

Pal, S.K., Kyriacou, P.A., Kumaran, S., Fadheel, S., Emamdee, R., Langford, R.M., & Jones, D.P. (2005). Evaluation of esophageal reflectance pulse oximetry in major burn patients. *Burns, 31*, 337-341.

Patterson, D.R., Hoflund, H., Espey, K., & Sharar, S. (2004). Pain management. *Burns, 30*, A10-A15.

Rosner, H., Rubin, L., & Kestenbaum, A. (1996). Gabapentin adjunctive therapy in neuropathic pain states. *Clinical Journal of Pain, 12*, 56-58.

Sagraves, S.G., Phade, S.V., Spain, T., Bard, M.R, Goettler, C.E., & Schenarts, P.J. (2007). A Collaboative Systems Approach to Rural Burn Care. *Journal of Burn Care & Rehabilitation, 28* (1), 111-114.

Sener, G., Kabasakal, L., Cetinel, S., Contuk, G., Gedik, N., & Yegen, G.C. (2005). Leukotriene receptor blocker montelukast protects against burn-induced oxidative injury of the skin and remote organs. *Burns, 31*, 587-596.

Sener, G., Sehirli, O., Velioglu-Ogunc, A., Ercan, F., Erkanli, G., Gedik, N., & Yegen, B. (2006). Propylthiouracil (PTU)-induced hypothyroidism alleviates burn-induced multiple organ injury.*Burns, 32*, 728-736.

Sheridan, RL. (2002). Burns. *Critical Care Medicine, 30* (11), S500-S514.

Sheridan, R.L. (2003). Burn care: results of technical and organizational progress.

Journal of American Medical Association, 290, 719-722.

Sheridan, R.L., & Tompkins, R.G. (2004). What's New in Burns and Metabolism. *Journal of American College of Surgeons, 198* (2), 243-263.

Sitzer, V.M. (1991). Physiologic framework of extrinsic controls. *Journal of Cardiovascular Nursing, 4*, 1-9.

Thomas, SJ., Kramer, GC., & Herndon, DN. (2002). Burns: Military Options and Tactical Solutions. *The Journal of Trauma Injury, Infection, and Critical Care, 54* (5), S207-S218.

Venn, R.M., & Grounds, R.M. (2001). Comparison between Dexmedetomidine and propofol for sedation in the intensive care unit patient and clinician perspectives. *British Journal of Anesthesia, 87*, 684-690.

Venticinque, S.G., Kashyap, V.S., & O'Connell, R.J. (2003). Chemical Burn Injury Secondary to Intraoperative Transesophageal Echocardiography. *Anesthesia and Analgesia*, 97, 1260-1261.

Walker, J., MacCallum, M., Fischer, C., Kopcha, R., Saylors, R., & McCall, J. (2006). Sedation Using Dexmedetomodine in Pediatric Burn Patients. *Journal of Burn Care & Rehabilitation, 27* (2), 206-210.

Wharton, SM., & Khanna, A. (2001). Current attitudes to burns resuscitation in the UK. *Burns, 27*, 183-184.

Wong, K., Heath, T., Maitz, P., & Kennedy, P. (2004). Early In-Hospital Management of Burn Injuries in Australia. *ANZ Journal Surgery, 74*, 318-323.