The science of burns is essentially the study of the response to injury. Burns teach us that it is amazing what the human body can tolerate. It has been well documented that burns are the “ultimate” insult and the body’s response is the most profound of any other injury or illness. Understanding how the body deals with this response to injury is fascinating. How the body heals in response to this severe insult is also fascinating. (J Burn Care Res 2008; 29:2–11)

When I tell others that I take care of people with burns I usually get a response such as “wow, how could you do that?” or “I have great admiration for you but there are not too many people who would do that.” Some just have the look of “boy I am glad I don’t have to deal with those people.” The goal of this address, then, is to answer the question—why do you do burns? The answer is, unfortunately, not easy to express. Yes, I love taking care of this patient population but it is difficult to express to others why I am a burn doctor. After a great deal of consideration I thought that I would express my love for my field by presenting a few cases. These cases will help me express my reasons, and your reasons, for considering a career in treating people with burns. As required, I will de-identify the patient’s names so as not to violate any Health Insurance Portability and Accountability Act (HIPAA) regulations.

CASE 1: H.D.

H.D. sat on a wall
H.D. had a great fall
All of the King’s soldiers . . .
Couldn’t put H.D. back together again

Who are these King’s soldiers? We, the entire burn community, are the King’s soldiers. We try to put the pieces together. It is not easy, and the result usually is not perfect. We obviously need to strive for improvements. Is it worth it? I hope I can convince you that all of the hard work is worth it. The hope is that you will understand why I do burns and I hope that I will incite others to consider a career in treating burns.

CASE 2: V.N.

Four-year-old Asian boy spills soup on his shirt
Small second-degree burn to the chest and abdomen
All areas heal within 2 weeks except for the upper chest
Upper chest heals in 18 days
Upper chest develops hypertrophic scarring
Rest of healed burn not visible
Hypertrophic scar recurs after excision and closure (Figure 1).

CASE 3: J.B.

Three-year-old boy struck by Molotov’s cocktail (gang-related attack on mother)
Runs down street screaming—increasing burn size
Ninety-five percent total body surface area (TBSA) burn
Abdominal compartment syndrome—laparotomy during resuscitation
Gradual coverage with autograft, allograft, investigational composite skin substitute
Three months later—grafted and on floor
Central line infection—Klebsiella
Septic shock within hours
CPR several times
Recurrent abdominal compartment syndrome
Renal failure—dialysis
Acute respiratory distress syndrome (ARDS)—maximal vent settings
Three more months in intensive care unit (ICU)
Thrombosis of all great veins
Severe cardiomyopathy (considered heart transplantation)
Corneal opacification of one eye
Significant scarring
Discharged to supportive parents after 9 months

CASE 4: S.C. (SAME FIRST NAME AND AGE AS MY DAUGHTER)

Eighteen-month-old girl “dipped in hot water” Abuse confirmed
Forty percent TBSA third-degree burns to legs and lower trunk
Course complicated by respiratory distress and sepsis
Grafted and doing well
Friday before planned discharge on Monday (DG on call for the weekend)
Patient does not “look right” while eating lunch

Oxygen saturations 75% on 100% oxygen
Transferred to ICU
Intubated
Hypotensive on pressors (epinephrine)
Complete renal failure
Dies 6 hours after ICU admission
Autopsy—1 cm vegetation on mitral valve (*Staphylococcus aureus*)

In my review of these cases I will describe why I love taking care of burn patients. The reasons can be broken into three categories:

1. The science of burns
2. The team approach
3. The human side of burns

THE SCIENCE OF BURNS

The science of burns is essentially the study of the response to injury. Burns (and J.B. of case 2) teach us that it is amazing what the human body can tolerate. It has been well documented that burns are the “ultimate” insult and the body’s response is the most profound of any other injury or illness. Understanding how the body deals with this response to injury is fascinating. How the body heals in response to this severe insult is also fascinating. Some of my favorite questions are:

1. Why does an injury progress or change over time?
2. Why is the ultimate healing of the wound imperfect or even pathologic?
3. Why does a local injury (in the legs for instance) affect distant organs (such as the lungs)?
4. Why is there a threshold size of a burn that leads to a systemic response?
5. Why is there a variable response?

The other fascinating aspect of burns is that you can study the response to burn injury on multiple levels or tiers (from large to small, small to large):

- person—organ—tissue—cell—organelles—signaling networks—proteins—genes—molecules—molecular interactions—gene regulation—protein production—signaling networks—cellular communication—organ interactions—human response—epidemiology—prevention. Some investigators focus on one tier whereas some of us can delve into each of these levels. I will give the reader cursory descriptions to peek interest in the science of burns.

HEALING AND SCAR FORMATION

Let us start by examining case 2 (V.N.)—the child who develops a hypertrophic scar in a small part of the

Figure 1. In this typical scald burn most of the wound healed in 10 days whereas the hypertrophic areas required 18 days to heal. The result is a burn that is barely noticeable except for the slow-to-heal areas where hypertrophic scars developed. Although these scars can be excised and closed primarily, they tend to recur. Understanding why this delay in initial closure leads to scarring is a key goal for future research.
original wound. We observe that the initial injury leads to local edema and a local inflammatory response. We also notice that the area that healed within 2 weeks did not scar and the area that did not heal as rapidly led to an ugly and persistent hypertrophic scar. This observation is not new; Edwin Deitch reported this observation decades ago. The answer to why hypertrophic scarring occurs, however, has never been discovered. The even more basic question is why do we heal? We know that injury induces pain and this signal participates in the initiation of healing. The healing process starts immediately. Clotting brings platelets that, besides assisting with hemostasis, release all forms of growth factors to attract inflammatory cells and other cells of healing. Clotting factors also participate in initiating healing. Activation of pain neurons and clotting are the “first calls to other cells” that damage has occurred and repair is needed.

One can think of healing as re-creation of a barrier. One can liken the barrier function to be a fortress that has a giant outer wall that keeps the “bad guys” (microorganisms) from invading the organism. The outer wall is the epithelium (epidermis for skin)—our first line of defense. The epithelium keeps the microorganisms out and keeps the vital nutrients and water inside. Dermis does not “seal” the barrier but, instead, gives the “fortress wall” its “strength.” The fortress wall protects the citizens (cells) who perform specific tasks in discrete factories (organs). Rapid closure of the epithelium (outer wall) minimizes invasion and allows for the inner fortress to remain relatively unchanged. In other words, rapid epithelial closure minimizes the formation of scarring and leaves the distant organs unaffected.

The second line of defense is the inflammatory system. Our leukocytes could be considered the soldiers who valiantly fight invading organisms. Like all soldiers, leukocytes use weapons to fight the enemy. In our bodies these weapons include oxygen radicals and proteases, plus others. Also like soldiers, these weapons damage more than the enemy, but also resident cells and tissues. The leukocytes also send messages (cytokines) when they need reinforcement with fighting these battles. Although there are many soldiers, there are some cells that orchestrate the roles of others. For the wound, the macrophage is a sergeant and the neutrophil is the private who is sent in to sacrifice himself before others.

Because the outer barrier is lost, the leukocytes call in the fibroblasts (construction workers) to create a temporary but imperfect obstruction to the invading microorganisms. They try to recreate a dermis but the epithelium must repair itself. To “feed” the troops and construction workers, a new blood supply needs to be created. Thus, angiogenesis must occur with the deposition of the provisional matrix. This “lousy barrier” for us is called “granulation tissue.” It does provide some protection from infection but the microorganisms still can invade; so the soldiers must remain at the wound until a new epithelium (fortress wall) is created. In other words, inflammation persists until a new epithelium is created. After a prolonged battle against microorganisms, the soldiers decide that the lousy barrier (or scar) needs to persist despite the return of the epithelium. Thus, hypertrophic scarring is the end result of prolonged exposure to the environment.

Finally, the inflammatory cells and fibroblasts use one more strategy to try to close the hole in the wall of the fortress—they attempt to shrink the wound. The fibroblasts “grab” the collagen that they produce and “pull” the matrix to shrink the size of the hole in the fortress wall (contract the wound). The force that these armies of cells create is so strong that they actually move the fortress walls. The combined effort of these myofibroblasts leads to contractures that can pull fingers into the hand or a lip on to a chest (Figure 2). The contracting forces persist for months and if it were not for therapists our results would be much worse than they are.

One of the most important goals of research should be to find the early signals that lead to hypertrophic scarring. If we find the signals that initiate and prevent the elimination of scarring then we would greatly help mankind. We know of some factors that increase scar formation: persistent inflammation and growth factors are key players. The most important growth factor seems to be transforming growth factor-β1 (TGF-β1). It has been found to convert scarless fetal healing to one of scarring. Increased levels of TGF-β1

Figure 2. A typical burn scar contracture that is the result of the contracting of myofibroblasts. The child cannot raise her arm above 90° and her neck has severely limited mobility.
have been found in patients with hypertrophic scarring.\textsuperscript{4} Tension in a wound will increase TGF-\(\beta\) levels, which in turn, increases collagen deposition, leading to the “banding” that we commonly observe in burn contractures.\textsuperscript{5} Releasing this tension will decrease TGF-\(\beta\) levels and reduce scarring.\textsuperscript{6} Finally, the intracellular signaling of TGF-\(\beta\) occurs through proteins called Smads.\textsuperscript{7} It has been discovered that there are differential signaling pathways after TGF-\(\beta\) is bound to its receptor. If signaling occurs through the Smad2 pathway then the response is proinflammatory whereas if the Smad3 pathway is chosen, then the Smad2 pathway then the response is proinflammatory. If signaling occurs through the Smad2 pathway then the response is proinflammatory whereas if the Smad3 pathway is chosen, then the response leads to increased collagen deposition.\textsuperscript{8} There is even an inhibitory Smad (Smad 7) that interferes with TGF-\(\beta\)1 signaling. Therefore, TGF-\(\beta\) may have duel roles in the response to injury. It is well known to be a regulator of the immune response and a stimulator of scar formation. Maybe, in its attempt to control inflammation, it also tries to create the lousy barrier (scar). Future studies will focus on the role of this growth factor. Members of various organizations tied to tissue repair, the ABA and Wound Healing Society for example, should develop a task force to solve the scarring dilemma in people. The task is made more difficult because there is no good animal model of scar formation.

**THRESHOLD INJURY SIZE AND THE SYSTEMIC RESPONSE TO INJURY**

What about the systemic response to injury? The third (J.B.) and fourth (S.C.) cases illustrate how a larger wound can lead to a total body response. With a small wound, the soldiers quickly control the situation and close the wound without a major response of the remaining soldiers within the fortress. The resident citizens are totally unaffected. In other words, there is no need for a distant-organ response. Once the injury becomes larger than around 20\% TBSA, there is a systemic response that is associated with the global edema, hypermetabolism, and a catabolic response. In other words, all of the population within the fortress must participate in the response to the invasion. These massively burned patients are at increased risk of infection and if their wounds are not closed expeditiously scarring is quite common. Investigators have tried to determine what causes these systemic responses. The initial theory was that the products of invading bacteria directly injured tissues but direct injury or bacterial toxins lead to only limited damage. It turns out; instead, that the body’s response leads to a great deal of the injury. One could consider that the damage caused by inflammatory cells is the result of “friendly fire.” When the burn is large enough to attract a threshold number of leukocytes, then local mediators (cytokines, proteases) spill into the systemic circulation and injure distant organs—lungs, kidneys, and others. Leukocytes are primed with the first insult (the burn) but when exposed to a second insult (such as infection) they produce an excessive amount of cytokines that diffuse throughout the body to affect distant organs.\textsuperscript{9} The same agents that are designed to destroy microorganisms damage distant tissues.

The “spill-over” of cytokines is also detected in the “command center” (the brain), which attempts to help the body respond to the inflammatory insult. The hypothalamus and anterior pituitary respond to these “danger signals” by releasing factors that increase the release of catecholamines and corticosteroids. The resulting hypermetabolism results in resetting the baseline temperature to around 38.5°C, and leads to leukocytosis, tachycardia, and tachypnea. Thus, the typical signs of infection for other illnesses are the “norm” for burn patients. Because these mild changes in temperature or vital signs are used as indicators for systemic inflammatory response syndrome and sepsis in other diseases, members of the ABA have recently held a Consensus Conference to Define Burn Sepsis and Infection.\textsuperscript{10} Standard definitions for burns will be the result of this effort. If the response to injury is not controlled, the patient may develop injury to the various organs (lungs—ARDS, kidneys—acute tubular necrosis, heart—depressed function and cardiomyopathy, brain—altered mental status, and liver damage—increased serum bilirubin).

There are typical hemodynamic changes that are routine for patients with extensive burns—excessive capillary leakage during resuscitation and sepsis, vasodilation—decreased systemic vascular resistance and increased cardiac output, increased platelet consumption—thrombocytopenia, and signs of inadequate perfusion. If one were to drill down into the mechanisms of these changes it turns out that the lining of the vasculature—the endothelium—has a great deal to do with these changes.\textsuperscript{11} Endothelial cells detect damage and produce cytokines and receptors that attract and bind leukocytes. In other words, endothelial cells “direct traffic” to the site of injury. Their mediators lead to further damage to the endothelium and surrounding tissues. The endothelial damage leads to ongoing thrombosis that leads to disseminated intravascular coagulopathy and decreased perfusion to capillary beds. In response to the localized tissue, nitric oxide is released that leads to vasodilation of the surrounding capillary beds that lead to the decreased systemic vascular resistance.\textsuperscript{12}
The mechanisms that regulate leukocyte production of cytokines have also been elucidated. Simple organisms lack lymphocytes and thus are unable to produce antibodies but they do respond to invading microorganisms by synthesizing defensive proteins. All organisms have developed receptors—called pathogen recognition receptors—that recognize proteins or DNA/RNA of invading organisms—called pathogen-associated molecular patterns. These receptors have persisted in all vertebrates, including humans. When they recognize a foreign protein, they turn on the cellular signaling machinery to release a responding “defensive” protein (typically a cytokine). We have two immune response systems: the innate immune system, which recognizes these pathogen-associated molecular patterns, and the adaptive immune system, which is the cell-mediated and antibody system regulated by the lymphocytes. The adaptive system exists in higher organisms (vertebrates) whereas the innate system exists even in single-cell organisms. I will not review the adaptive system but will cover a few points about the innate system to illustrate the complexity of the system. The most well-described receptor system for the innate immune system is the toll-like receptor (TLR) group. There are at least 10 TLRS in humans with the most studied being TLR4, which responds to lipopolysaccharide (LPS) of gram-negative bacteria, and TLR2, which responds to proteins of gram-positive bacteria.

The complexity increases if one examines how LPS interacts with TLR4 (partially represented in Figure 3). LPS, released from gram-negative bacteria, is

**Figure 3.** A partial diagram of the initial signaling of LPS to TLR4 is demonstrated. LPS is initially bound by LPS-binding protein. The combined proteins bind to CD14, which, in turn, interacts with TLR4. Adapter proteins then influence intracellular signaling. If signaling occurs through MyD88, NF-κB is activated to produce cytokines. If a different adapter protein is present (such as TRIF) then signaling occurs through IRF3 to ultimately initiate the production of IFNβ. This sketch touches on the complexity of the cellular regulation of the response to injury.
bound outside the cell by LPS-binding protein, which in turn binds to the membrane-bound CD14. CD14, which has no signaling capabilities, carries the complex to TLR4, which also interacts with other “adapter” proteins (MyD88 and MD2). This receptor complex can have varying responses depending on which adapter proteins bind to TLR4 (MyD88, TRIF, or 3 others). Multiple other intracellular proteins are involved in intracellular signaling, with nuclear factor (NF)-κB being a key mediator of many of the responses. NF-κB signaling is inhibited from entering the nucleus by another protein IκB that when eliminated allows for NF-κB to bind promoters on DNA. This binding leads to the production of cytokines, which lead to the response to injury.

To add further complexity to the system there are all sorts of counter-inflammatory mediators that regulate the degree of inflammation. One of the most commonly described inhibitors of inflammation is cortisol (a glucocorticoid) that is released in response to stress (Figure 4). These corticosteroids must bind to a cytoplasmic receptor (glucocorticoid receptor [GR]), form a dimer, and then bind to glucocorticoid response element on DNA to counter the effects of TLR4 signaling. The degree of counter-regulation is also altered by genetic variations in the glucocorticoid receptor, failure to form dimers, and the exposure of genes through regulators of histone formation (histone acetylation and deacetylation). To make matters even more complicated, minor changes (polymorphisms) in the glucocorticoid receptor have profound effects on how the cell responds to injury. For example, GRα is responsible for normal signaling. There is also a GRβ, which binds glucocorticoids but does not bind to the glucocorticoid response element. It acts as a “decoy” to inhibit glucocorticoid signaling. These descriptions just touch on the complexity of our ability to respond to injury.

**VARIABLE RESPONSE TO INJURY**

Another fascinating area is the investigation of why similar patients respond differently to injury. It is not uncommon to have two patients, even brothers, who have similar sized burns with one surviving with minimal problems and the other dying after multiple
complications. Why does one do well and the other succumb to the injury? Research has started to find some explanations for the differential responses but we still have a long way to go. Some explanations for different responses to injury include the genetic makeup of the patient. For instance, some studies suggest that there are minor variations of certain proteins (“polymorphisms” for instance) that allow one to respond to stress in a better way than another.20 Certainly, the environment in which the patient is injured and treated may influence the outcome, and finally, the condition of the patient at the time of the injury will affect the ultimate outcome. The classic example of the patient’s condition is the state of nutritional status. Patients who are malnourished do worse than those who are of adequate nutritional status. There are other factors that influence outcome. If someone is exposed to another insult, there may be a differential response. For a mild insult with the appropriate timing, that person may be “prepared” for a second, more severe insult. A similar second insult, however, with inappropriate timing may make that patient vulnerable for an adverse response. Developing ARDS, for instance, may be associated with one of these “two-hit” insults.

It is also well known that presence of other injuries or illnesses (cancer, renal failure) will make survival more difficult. Even more interesting is the significant amount of literature that suggests that women may handle stress better than men. The level of sex hormones, even those changes regulated by the menstrual cycle, may influence how the body responds to stress.21 Another possibility put forth by Kiho Cho, PhD, working in our laboratory, is that endogenous retroviruses may play a role.22 These retroviruses invade and then insert their DNA message into our genes. These retroviral “codes” become permanent insertions in our genetic code that are passed off to our children. Most of the time, these endogenous retroviruses are silent but evidence from our laboratory suggests that they may be activated in response to injury. These messages might contribute to immunosuppression after injury. It is obvious that the regulation of the response to injury is extremely complex and I encourage others to pursue this area of investigation.

The real challenge is how do we manage this complex and profound response to injury that occurs after a major burn? Research allows us to “translate” our bench research to clinical therapies. At the current time, management is mainly supportive but we use our knowledge to improve care. We support the patient’s hemodynamics with fluids and inotropes, support patients on ventilators, and if there is renal failure we provide dialysis. One great advance has been to provide aggressive nutritional support as the metabolic milieu continues to break down protein. There has also been a great deal of interest in attempting to reverse the catabolic response with anabolic agents such as growth hormone,23 insulin,24 oxandrolone,25 plus others. Other studies suggest that there are certain patients who fail to respond to their own glucocorticoid stimuli. These “nonresponding” patients may benefit from exogenous glucocorticoids.17

Because the destructive process is a direct result of our response to injury our goal should be to interrupt the signals that initiate this destructive process. We need to turn off the “danger signals” by blocking mediator-receptor interactions. There have been major clinical trials that treated septic patients with blockers (antibodies) against LPS,26,27 various cytokines,28 and agents that inhibit thrombosis.29 These trials have been complete failures except for activated Protein C.30 This one agent has produced statistically significant improvements in survival; however, one must wonder whether clinically relevant improvements were accomplished.

What has seemed to make a difference, however, is the philosophy of early excision of the burn wound. This philosophy makes sense. If one has a splinter, the local inflammatory response persists as long as that foreign body is present. Removing the splinter leads to rapid resolution of the local response to injury. In the same light, expeditious excision and coverage of the wound should reduce the extent of the inflammatory stimulus. Expeditious excision should also assist with reducing scar formation. These philosophies have been accepted but they have not been proven with prospective, randomized trials. Unfortunately, it is not ethical to perform these trials at this time because expectant care—waiting for granulation tissue formation before grafting—is felt to be below the standard of care.

What we can do is attempt to improve our outcomes by performing expeditious excision and closure procedures in a manner that attempts to optimize the functional and cosmetic outcome of the patient (Figure 5). Sheet grafts with thicker grafts will improve the functional and cosmetic outcome,31 but we also need the “burn team” to assist with the therapy, massage, and pressure garments to optimize the outcome. If we do not have the “team” our results will never be as good as just operating on the patient and sending them home. This is why we have burn units and not a bunch of isolated surgeons.

**THE TEAM APPROACH TO BURNS**

The second aspect of burn care that I love is the team approach. Burn units were the first group of caregiv-
ers to treat a disease process as a team and we use the team approach better than any other team. We are a group where everyone contributes and every voice is heard. Everyone has a role that helps the burn patient recover from the most severe insult that is known. The “total” care of the patient clearly improves the outcomes of our patients. Not only are we colleagues, but we also become lifelong friends. Look at the American Burn Association. Is there any other surgical or medical organization that has members which include all participants of the team? Our meetings foster an interaction among all types of specialties to work together to solve an incredible medical challenge. The team approach is why Burn Units exist and we know that our team leads to a better endpoint for the patient. Several studies have proven that centers that provide high volume care to patients lead to improved outcomes for patients.32,33 Our burn units also have excellent outcomes, especially when it comes to survival. We need to go further to demonstrate that burn survivors have improved outcomes. One way to help with demonstrating improved outcomes is to have all burn centers become verified by the American Burn Association. Further studies should focus on outcome analyses to demonstrate the efficacy of burn centers.

Let us look at the team for a minute. The team really provides care from the initial rescue until the survivor’s return to society. The comprehensive care is all-inclusive and the team is with the survivor for life. We can divide the team into those who provide the rescue and initial first aid (“immediate care”), initial inpatient care, outpatient care, and an increasingly active team that provides aftercare.

### Rescue/First Aid (Immediate Care)
- Firefighters
- EMTs
- Paramedics
- Police
- Primary care MDs

### Initial Inpatient Care
- Burn MDs
- Other MDs
- Residents
- Nurses
- Respiratory therapists
- Dietitians
- Occupational therapist/Physical therapist
- Social workers
- Psychologists
- Child life
- Teachers
- Operating room staff
- Burn technicians
- Care coordinators
- Discharge planners
- Outreach coordinators
- Spiritual care
- Administrators
- Volunteers

### Outpatient Care
- Burn MDs
- Residents
- nurses
- Therapists
- Social workers
- Dietitians
- Burn technicians
- Care coordinators

We were once satisfied solely with survival but that goal is no longer good enough. We need to focus on more than survival. The new goal should be to make the life of that survivor as normal as possible. Optimizing functional and cosmetic outcome should be our new objectives. Assisting our patients to become productive members of society and returning to a normal life should be our new standards of care. Our newest members of the team are the professionals who participate in aftercare. The role of this team is to assist with the reintegration of all survivors back into society. As explained in the 2007 President’s Plenary Session one objective of my presidency will be to create an Aftercare and Reintegration Committee for the American Burn Association. Their mission will be to foster the support of survivors of burn injury as...
they return to society. The following members make up the Aftercare team:

**Aftercare**
- Survivor groups
- Peer councilors
- Burn camp teams and councilors (child and adult)
- School re-entry teams
- Prevention teams
- Phoenix Society (World Burn Congress)
- Any other team member volunteer

**THE HUMAN SIDE OF BURNS**

The last reason why I love to take care of burn patients involves the human side of burn care. We struggle with patients who may die at any second. I have taken care of patients so critically ill that team members have debated whether to stop or not. I have told parents that there is no more that we can do other than wait for the inevitable. Despite being so critical several of these patients survived and thrived. Months and years later, seeing his or her smile just warms everyone’s heart. I have seen children who undergone cardiopulmonary resuscitation only to wake up. After a few months, I have witnessed the rebirth of children in a vegetative state to a point that they can thank me for being his or her doctor. Because we routinely follow these children into adulthood, we watch them return with straight “A” grades, college degrees, spouses, and children. We watch our patients volunteer to encourage new patients that their struggles will be worth it if they just hang on for a few months. The thanks of friends and families also add to the reward.

One of the best way to observe the return of our patients into society is to attend the World Burn Congress of the Phoenix Society, an event that our team was fortunate enough to co-host in Sacramento this year. At this gathering of burn survivors, the caregivers are the minority. The survivors “break out of their shells” and really speak their minds. It is fascinating to hear what our former patients have to say during the daily “open microphone” sessions. I encourage all members of the American Burn Association to attend or even better, host a World Burn Congress. The best way to demonstrate the human side of burn care is to just show pictures of our successes.

There is also the human side of working with the team. The camaraderie of working with the members of our team lasts a lifetime. I have made lifelong friendships with the other burn doctors. I essentially can give any burn patient the name of the director in any burn unit in any part of the country. But I also am a friend with all members of the team. We all have common goals that are shared at meetings, with casual conversation and during multicenter trials. It is fascinating to see how hard all members of the team work during the times of stress and then “let it loose” during the annual meeting of the American Burn Association. It is a great release to see all of the dancers at the annual banquet. While we all sweat together on the dance floor, the respect that we show for each other, no matter what level of caregiver, is incredible to observe. These friendships will last forever.

I now return to H.D., our first case. H.D. fell off a wall, or maybe he suffered a massive burn. You and I are the “soldiers.” We have the common goal to work long, tireless hours to put H.D. back together knowing that we will never reach perfection. We will not only work hard, but we will also perform all levels of research to strive to improve our care. This research is fascinating to us as we labor to save lives and limbs. We live with the knowledge that we will never reconstruct H.D. as he once was but still we face the challenge and do our best. As we put H.D. back together we know that there are many who have no desire to get dirty, sweaty, or bloody to put the pieces together. Only a few special soldiers work to put H.D. and our patients back together. But at least someone, the burn team, gives it a try. Without us those happy faces would not exist.

Although we revel when we save a patient, such as J.B. with 95% TBSA burns, we are not capable of saving all of our patients. There seems to be no justice when an innocent child such as S.C. who is abused, suffers and survives the months of hospitalization only to die just before the planned discharge. We, the soldiers, take of these tragic cases as well.

When I die I believe that I (and you) will be sent to heaven. When we get there, I am sure that the hundreds of burn nonsurvivors who were privileged to go to heaven will meet us. I have a vision that S.C., who suffered that tragic death, will be there and she will come up to me and give me a big hug and say “thank you for trying.” Because we cannot save all of our patients but, again, we try. Our job is to help people live, and occasionally, help people die in a humane and dignified way.

I finish with a quote from Fyodor Dostoevsky’s *The Idiot*:

> “And on the gloom of my declining hour
> Perchance the farewell smile of love may shine”

These are the reasons why I became a burn surgeon. I wish to thank you for the privilege of being your president.
REFERENCES


