Published in final edited form as:

Lancet. 2012 March 17; 379(9820): 1013–1021. doi:10.1016/S0140-6736(11)61345-7.

BURN SIZE AND SURVIVAL PROBABILITY IN PEDIATRIC PATIENTS IN MODERN BURN CARE

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Abstract

Background—Patient survival following severe burn injury is largely determined by burn size. Modern developments in burn care have tremendously improved survival and outcomes. However, no large analysis on outcomes in pediatric burn patients with current treatment regimen exists. This study was designed to identify the burn size presently associated with significant increases in morbidity and mortality in pediatric burn patients.

Methods—Single center prospective observational cohort study utilizing the clinical data of severely burned pediatric patients admitted between 1998 and 2009. This study included 952 severely burned pediatric patients with burns over at least 30% of their total body surface area (TBSA). Patients were stratified by burn size in 10% increments, ranging from 30 to 100%, with a secondary assignment made according to the outcome of a receiver operating characteristic (ROC) analysis. Statistical analysis was performed using Student's t-test, χ^2 test, logistic regression and ROC analysis, as appropriate, with significance set at $p < 0.05$.

Findings—All groups were comparable in age (age in years: 30–39: 6.1 ± 5.1 , 40–49: 7.1 ± 5.2 , 50–59: 7.6 ± 5.1 , 60–69: 7.2 ± 5.1 , 70–79: 8.3 ± 5.9 , 80–89: 8.4 ± 5.6 , 90–100: 9.6 ± 5.4), and gender distribution (male: 30–39: 68%, 40–49: 64%, 50–59: 65%, 60–69: 59%, 70–79: 71%, 80–89: 62%, 90–100: 82%). Mortality (30–39: 3%, 40–49: 3%, 50–59: 7%, 60–69: 16%, 70–79: 22%, 80–89: 35%, 90–100: 55%), multi-organ failure (30–39: 6%, 40–49: 6%, 50–59: 12%, 60–69: 27%, 70–79: 29%, 80–89: 44%, 90–100: 45%), and sepsis (30–39: 2%, 40–49: 5%, 50–59: 6%, 60–69: 15%, 70–79: 13%, 80–89: 22%, 90–100: 26%), increased significantly ($p < 0.001$) among the groups and at a threshold of 62% TBSA. Comparison of patients with burns larger than 62% with those smaller showed significant differences in inflammatory (Cytokines), acute phase (CRP) and hypermetabolic responses (REE), as well as organ function ($p < 0.05$).

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AUTHORS CONTRIBUTIONS:

MGJ, DNH, RK planned the study, were responsible for the design, coordination and drafting the manuscript. AAM, FNW participated in the study design and helped to draft the manuscript. RK and FNW performed the statistical analysis. DNH, MGJ and CCF obtained funding.

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Interpretation—We established that in a modern pediatric burn care setting, a burn size of approximately 60% TBSA represents a crucial threshold for post-burn morbidity and mortality. Based on these findings, we recommend that pediatric burn patients over 60% TBSA burn should be immediately transferred to a specialized burn center. Furthermore, at the burn center patients should be treated with increased vigilance and enhanced therapies recognizing the increased risk for poor outcome associated with this burn size.

Keywords

burn size; survival; metabolic response; sepsis; multi-organ failure

INTRODUCTION

Predicting survival and outcomes has always been an important goal in the treatment of thermally injured patients. In the 1980s and early 1990s, clinical predictions and decisions were based mainly on burn size.^{1–3} In 1988, Ryan et al.⁴ assessed improvements in burn care using a large population of 1,665 patients treated between 1990 and 1996, and developed a prediction model for individual patient risk and identified burn size of 40% TBSA as a risk factor for poor outcome. This model was based on injury characteristics, initial treatment, and biochemical markers during admission and hospital stay.⁴ However, over the last ten years, additional improvements in care have further reduced morbidity and improved survival rates and outcomes following extensive burn injuries. Novel drug therapies, new grafting techniques and materials, and life support systems combined with more sensitive monitoring methods, and have all led to improved care after severe thermal injury.⁵

In this study, we analyzed the post-burn prognosis in pediatric patients. Especially in children, the probability of survival and the incidence of complications are important to know as a reference for the decision for the transfer to a specialized burn center, as well as for treatment decisions at admission and during hospital course. This study focused on children, with the advantage that in children and adolescents, the hospital course is mainly determined by the impact of the burn injury and not by preexisting co-morbidities. Additionally, in this patient population the estimated life expectation is not taken into account and decisions are based on survival probability. The aims of this study were: 1) to determine the burn size, as expressed by total body surface area (TBSA), presently associated with elevated morbidity and mortality; 2) to assess outcomes in relation to burn size; and 3) to correlate patient outcomes with biochemical markers and measures of organ dysfunction. The investigation provides a rationale for clinical decisions based on survival, expected complication rates and outcome after burn injury.

PATIENTS AND METHODS

The study was approved by the Institutional Review Board of the University Texas Medical Branch, Galveston, Texas. Informed consent was obtained from each subject, parent or child's legal guardian. All acutely burned patients arriving at our burn center were included in this analysis to be in accordance to the previously published trial and to compare the outcomes from the previous study to our study.⁴

Nine-hundred and fifty-two thermally injured children admitted to the burn unit with burns of 30% of their total body surface area (TBSA) or greater were enrolled between 1998 and 2008. Patients were resuscitated according to the Galveston formula with 5000 cc/m² TBSA burned + 2000 cc/m² TBSA lactated Ringer's solution, given in increments over the first 24 hours as necessary. Within 48 hours of admission, all patients underwent total burn wound

excision, and their wounds covered with autograft. Any remaining open wound areas were covered with homograft. Admission and treatment criteria were based on the guidelines published by the American Burn Association. All patients received similar nutritional treatment according to a standardized protocol. Intake was calculated as 1500 kcal/m² body surface + 1500 kcal/m² area burned.⁶ Patient demographics, injury characteristics and the hospital course, morbidity, and mortality were recorded. Sepsis was defined according to the modified ACCP/SCCM criteria.^{7,8} Multi-organ failure (MOF) was assessed according the DENVER2 score.⁸ We further determined the time between operations, considering this reflective of donor site healing, and therefore, an estimate of wound healing and re-epithelization. Patient data was collected prospectively, processed and analyzed with Microsoft Access®, Excel® Microsoft Corporation Inc. (Redmond, WA, USA).

Indirect calorimetry

As part of our routine clinical practice, all patients underwent resting energy expenditure (REE) measurements within one week of hospital admission and weekly thereafter during their acute hospitalization. REE was measured using a Sensor-Medics Vmax 29 metabolic cart (Yorba Linda, CA) and calculated as described by Mlcak et al⁶ (n=696).

Liver-size changes

Liver ultrasound measurements (n=405) were made with the HP Sonos 100 CF echocardiogram (Hewlett Packard Imaging Systems, Andover, MA). The liver was scanned using an Eskoline B-scanner and liver size/volume was calculated using a formula as previously described.⁹ Actual size was then compared to predicted size for healthy volunteers.

Hormones, proteins, and cytokines

Blood and urine were collected from every patient at admission, and during the acute stay till discharge and used for serum hormone (n=479), protein (n=713), and cytokine (n=461) analysis. Serum hormones and acute phase proteins were determined using HPLC, nephelometry (BNII, Plasma Protein Analyzer Dade Behring, MD), and ELISA techniques. Nephelometry represents an older technique and in our burn center this technique gave reliable and consistent data. The Bio-Plex Human Cytokine 17-Plex panel was used with the Bio-Plex Suspension Array System (Bio-Rad, Hercules, CA) to profile expression of cytokines and inflammatory mediators.

Statistical Analysis

For biochemical measurements we applied the Confidence Interval 2σ (CI= 0.9544997) to improve reliability. Student's t-test, Chi-square analysis, linear and multiple logistic regression analysis were used as appropriate. For the cut-off analysis, Receiver Operating Characteristic (ROC) analysis was used. Statistical analysis was performed using Microsoft Excel® and Systat Software Sigmaplot® version 3.5 and Sigmaplot®, Systat Software Inc. (San Jose, CA, USA). Data are expressed as means±SD or SEM. Significance was accepted at p<0.05.

RESULTS

Demographics and clinical outcome

Burn size ranged from 30–100% TBSA and most patients had burns between 40–50% TBSA. Based on burn size, patients were stratified into seven groups using 10% increments between 30 and 100% TBSA. Gender distribution, ethnicity, and time from burn to admit were similar in all groups (detailed demographics are shown in Table 1). Incidence of

inhalation injury and patients age increased significantly ($p<0.001$) with burn size whereas patients with larger burns were referred significantly ($p<0.001$) faster to the specialized pediatric burn center. Patients with inhalation injury showed a significant ($p<0.001$) higher mortality (23%) compared to the cohort without (7%). Increased burn size was associated with prolonged length of ICU stay (Table 2). Larger burns also required a greater number of operations during acute hospitalization, starting with a mean of 2.1 operations for patients with burns of 30–39% TBSA, and increasing to 8.6 operations for 90% TBSA burns ($p<0.05$). The time required between surgeries also increased accordingly ($p<0.05$). MOF, including the maximum DENVER2 and Sepsis scores, was also correlated with increasing burn size, showing a significantly higher incidence for burns over 60% TBSA ($p<0.05$). The same effect was observed in the number of minor infections (under 60%, 2.8 to 4.1, and over 60% 7.7 to 10.4 per 10% group) during the length of stay in the ICU ($p<0.05$). Mortality increased significantly ($p<0.001$) relative to burn size (Figure 1A). A tremendous increase in mortality was evident starting at 60% TBSA with the Kaplan-Meier survival curve showing a significant difference in short and long-term survival ($p<0.05$) among the groups over and under 60% TBSA. However, among patient groups with burns smaller than 60% TBSA, there were only minor increases in mortality starting at 3% up to 7%. To validate these findings, we performed ROC analysis for mortality to determine the critical value for survival (Figure 1B), with the cut-off identified as 62% TBSA. The ROC curve (Figure 1C) validates this finding with a fair value of $A=0.81$ for prediction of survival.

Using multiple logistic regression, after adjusting for inhalation injury, gender, age and burn to admit time, TBSA over 60% is a strong predictor of mortality with an the odds ratio of dying 10 times higher ($p<0.001$) than patients below the threshold and confirmed that burn size is the main factor for the estimate of survival. Inhalation injury has an odds of dying a nearly 3 times higher ($p<0.001$) than those without. Patients age ($p=0.3489$) and time from burn to admission ($p=0.6651$) are not associated with mortality differences in this patient population during hospitalization (Table 3).

Inhalation injury contributes to mortality and as shown in Supplemental Tables 1 and 2, patients with inhalation injury have a significant higher mortality in each examined burn group compared to non-inhalation injured patients (Supplemental Tables 1 and 2). A ROC cutoff analysis for burn size with inhalation injury was not performed because the majority of patients with inhalation injury had a significantly larger burn size skewing the data to a higher cutoff than a lower which would be an observational error.

Metabolic response

Glucose metabolism—Significantly ($p<0.05$) higher blood glucose and insulin levels in the group with bigger burns over the whole study period, with a slight normalization by day 60 were observed. (Figure 2A, B).

Indirect calorimetry—Analysis of REE revealed significantly higher values at discharge compared to admit in each group. These values significantly ($p<0.05$) increased with burn size (Figure 2C) reflecting the caloric needs after burn injury.

Liver changes—Liver size normalized for age, expressed as percent predicted, increased from admit to discharge with increasing burn size. While only a slight increase in liver size was observed in patients with burns up to 69% TBSA, a massive and significant increase occurred between admit and discharge for patients with larger burns (Figure 2D).

Biomarkers

Proteins and triglycerides—Production of liver constitutive proteins are shown in Figures 2 and 3. Both burn size groups showed a decrease in all measured proteins, which began to normalize following the first week post-injury. Total Protein (Figure 2E), Apolipoprotein B (Figure 2F), Apolipoprotein A1 (Figure 2H), Prealbumin (Figure 2I) and Transferrin (Figure 2J) were all significantly ($p<0.05$) decreased in the larger burn group. Regarding Retinol binding protein (Figure 2K), a change after day 10 post-burn was observed, with the expression of RBP significantly less in the smaller burn group.

Triglycerides (Figure 2L) were also significantly higher in the larger burn group at several time-points.

Cytokines

All measured cytokines (Figure 3A–J) showed major differences between the two burn size groups over the monitored time period from immediately post-burn up to 60 days after the injury ($p<0.05$).

In the larger burn group, Interleukin-8 and -10 (Figure 3H, I) remained significantly elevated over most of the study period. MIP-1b (days 2 to 28) (Figure 3C) and IL-13 (days 2 to 22) (Figure 3J) also remained significantly elevated ($p<0.05$).

Figure 3 shows the cytokine response grouped for important cellular response mechanisms, as well as macrophage activity, cell death and repair mechanisms. Levels of TNF- α and G-CSF (Figure 3A, D) were significantly and constantly elevated from day 2 to day 60. IFN- γ and GM-CSF (Figure 3B, E) were significantly higher in patients with burns above 60% TBSA between the days 2 and 22.

Established markers of the inflammatory response are shown in Figure 3G and K, where we selected IL-6 and C-reactive protein (CRP). These showed an immediate rise after burn, with significant differences throughout the whole study period. The liver synthesized protein CRP increased more slowly after day 1 post-burn then increased rapidly starting day 8.

Organ specific markers of kidney and liver function

Liver and renal function were monitored by aspartate transaminase (AST), total bilirubin (Figure 3L, M) blood urea nitrogen (BUN) and serum creatinine (CRE) (Figure 3N, O).

Blood urea nitrogen remained elevated over the first 60 days, with significantly higher values over the observational period in the larger burns group. CRE was significantly elevated up to day 28 and then remained similar in both groups. Representative of liver impairment, total bilirubin was significantly ($p<0.05$) elevated in the larger burns over the whole study period, while AST remained elevated for the first 34 days.

PANEL: RESEARCH IN CONTEXT

Systematic review

In February 2011, we conducted a literature search for outcomes and mortality after severe burn injury. Beginning with the first study from 1949, publications documented the improvements in survival over the decades and provided information on the current state of survival under the aspect of the extent of burn and co-factors with a last major analysis in 1998.¹ Clinical investigations reporting the results of interventional clinical trials and observational studies suggest significant improvements in burn care.^{2–3} Novel surgical

management of the burn wounds, monitoring, and pharmaceutical treatment options and regimens propose improved overall survival rates during the past decade.^{11,15,18,22}

Interpretation

In this analysis, we determined the current crucial cut-off burn size for survival in modern pediatric burn care. We are able to show that a burn area of 62% TBSA is a hallmark for survival. A detailed analysis of the patients stratified in deciles for burn size provides an estimate of co-morbidities during the ICU stay and shows an increasing incidence of multi-organ failure, infectious complications, length of hospital stay, and mortality with a major tilt point around 60% TBSA burn. Furthermore, a large panel of established and novel biomarkers for organ function, metabolism, and inflammation indicates that major differences between the groups exist and confirms the findings of other clinical studies. Results of this study show that pediatric burn patients with a burn size greater than 62% TBSA are at a significantly higher risk for clinical complications and dying.

DISCUSSION

Building upon previous studies,^{4,10} this study identified the critical burn size determining major complications and survival with state of the art, modern burn care. In this study we accumulated a large patient population, which we used to study the crucial cut-off for survival and to corroborate with biochemical markers. The population in this study showed a nearly equal mortality for patients with burns up to 60% TBSA, but for those with larger burns, the mortality rate increased tremendously. Important to note are the relatively low mortality rates among even the most extensively burned patients (55% for over 90% TBSA), which in our opinion is attributable to an enhanced and aggressive treatment regimen.

As cofactors contributing to mortality besides the extent of the burn, we identified inhalation injury. The presence or absence of inhalation injury affects outcomes and our data indicate that inhalation injury worsens post-burn morbidity and mortality. We did not conduct a ROC cut-off analysis for patients with burns and presence of inhalation injury because of the uneven distribution of these patients. Patients with inhalation injury suffer from larger burns when compared to patients with absence of inhalation injury, which would skew the data towards larger burns. We recommend that the treating physician needs to keep in mind that the presence of inhalation injury significantly impacts post-burn outcomes. Our interest is, that patient's age and the time from injury to admission to a specialized burn center did not significantly contribute to in-hospital mortality.

We found that mortality peaks within the first twenty days after admission and shows a prolonged elevation related to burn size. In accordance with these findings, the incidence of sepsis and MOF combined with maximum DENVER2 score and infections correlate with burn size. For burns up to 49% TBSA, the incidence of these complications remains at a level of 6–12% for MOF, and 2–6% for sepsis but increases up to 27–45% for MOF and 15–26% for sepsis in the group with burns over 60% TBSA burn size. As expected, the number of necessary surgeries during the acute stay increased with the size of burn. These results are reflected by important organ-specific clinical parameters, as well as metabolic and inflammatory response.^{11,12} Insulin resistance, a hallmark post-burn was confirmed in this study.^{13–15} We demonstrated that insulin resistance worsens with increased burn size, with higher blood glucose levels found in the extensively burned group despite higher serum insulin levels. Alterations in glucose metabolism are associated with changes in lipid metabolism. Serum triglycerides significantly increase and elevations persists significantly longer with a larger burn size. These metabolic changes were also reflected in metabolic rate determined by REE. Patients with larger burns have profoundly higher metabolic rates and demands for a prolonged period of time compared to smaller burns. Metabolic demands are

reflected in organ function. The larger the burn, the worse liver structure and function with profoundly depleted constitutive proteins and dramatic hepatomegaly is, which is most likely due to an edema formation early after burn and hepatic lipid infiltration later in hospital course.^{11,12} Our data also indicate profound impact of kidney and pulmonary function most likely due to the massive protein catabolism and wasting post-burn. The aforementioned data all indicate that severely burned patients undergo a massive hypermetabolic response affecting all organs and contributing to post-burn morbidity and mortality.

The hyper-metabolic state can be at least partially attributed to the inflammatory response. Inflammatory mediators G-CSF, IL-8 and IL-10 showed an immediate increase with a decrease over time in both populations, however with significantly higher levels in the > 60% TBSA burn group. Interleukin-8 targets a relatively wide range of cells including primary neutrophil granulocytes, mast cells, macrophages, endothelial cells and keratinocytes. Several studies revealed that IL-8 causes localized inflammation leading to oxidative stress.^{14,16} This would correlate with the tremendous difference between groups over the whole study period, reflecting the posttraumatic tissue inflammation, which is seen clinically after burn injury.^{12,17,18} The anti-inflammatory cytokine IL-10¹⁹ shows a similar pattern as IL-8. A key cytokine for inducing inflammatory processes is TNF- α ^{18,20,21}, the expression of which is characteristic and can be clearly attributed to the effects of burn size. Whereas, the group with smaller burns showed significantly lower levels and a slight decrease over time, the data from extensively burned patients showed a prolonged elevation of this cytokine. The complex signaling pathway leads to several effects throughout the whole body of injured patients. TNF- α induces the production of acute phase proteins including CRP in the liver,²² the expression of IL-6,¹⁴ stimulates phagocytosis in macrophages²³ and migration of neutrophil granulocytes²⁴ in infected or damaged tissues. TNF- α also affects hormonal regulatory processes, especially hypothalamic thermoregulation²⁵ and insulin sensitivity²⁶ all present in severely burned patients.²⁷

The importance of our biomarkers determined in this study is that they reveal trajectories differentiating survivors from non-survivors matched for burn size. These novel trajectories are of great interest as we can clearly identify molecules that are altered according to burn size and the next step would be to propensity match patients and determine differences in these trajectories. The propensity matching would further eliminate the amount of fluid resuscitation or outside factors that could cause an artificial iatrogenic modulation of these biomarkers. However, in this study, we found that biomarker profiles are profoundly different between the different burn sizes, and particularly so during later time points, indicating that it is not fluid or resuscitation dependent but rather endogenous synthesis and expression.

Besides the injury-driven inflammatory response, we can show that the incidence of clinically relevant infections increases with burned area. The loss of the dermal barrier, profoundly increased metabolism, and an impaired immune system lead to a higher incidence of burn wound and nosocomial infections increasing with burn size. The impact of these processes on important organ systems such as renal and liver function was assessed by monitoring routine laboratory parameters. Blood urea nitrogen and serum creatinine levels were significantly elevated in the larger burns group. Comparing both parameters, we found similar results as in the inflammatory markers. Differences in serum creatinine disappeared after day 28 post-burn, whereas BUN remained significantly different. As an explanation for these findings, we propose that metabolic breakdown and protein degradation related to burn size leads to a significantly higher prolonged nitrogen load in this burn group. The impact on the liver as a pivotal organ for metabolic homeostasis is reflected in levels of the enzyme AST and total bilirubin. Bilirubin is an established parameter for monitoring liver function,

and was significantly elevated in the larger burn group. The initial elevation during the first two days after injury might be explained with the breakdown of damaged red blood cells and the transfusion of red blood cells given during the burn wound excision and grafting procedures. AST is a non-liver specific marker⁹ that can also be found in red blood cells, heart, pancreas, kidneys and muscle. Therefore, it may not specifically reflect liver damage. The tremendous elevation seen during the first week in burn patients over 60% might be explained by severe tissue damage to the skin following burn injury.

We would like to mention that a limiting factor, which may be seen as advantageous by some when observing the end points used in this study, was that admitted patients at our hospital received maximal treatment regardless of the severity of the burn injury (no DNR orders written). This treatment regimen does not necessarily apply to all burn patients at all centers. Another limitation, yet a potential advantage, is that this study is a single-site study not including outcomes from other hospitals. The cutoff of around 60% TBSA, therefore, may or may not apply to other burn centers. However, the data from a single center eliminates the differences of multiple study sites due to homogenous treatment protocols. In summary, the results of this study verify the clinically expected greater incidence of mortality and major changes in morbidity with increasing burn size, with a major divergence noted at around 60% TBSA burns. We show that the incidence of co-morbidities increase with burn size and also show a major increase over the calculated cut-off value. Moreover, this depends on a series of metabolic changes and clinically important organ monitoring parameters which were also found to follow this pattern.

The relevance of this work is that we established that in a modern pediatric burn care setting, a burn size of approximately 60% TBSA represents a crucial threshold for post-burn morbidity and mortality. Based on these findings, we recommend that pediatric burn patients over 60% TBSA burn should be immediately transferred to a specialized burn center. Furthermore, at the burn center, patients should be treated with increased vigilance and enhanced therapies recognizing the increased risk for poor outcome associated with this burn size.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding: This work is support by grants from Shriners Hospitals for Children (8660, 8760, and 9145), National Institutes of Health (R01-GM56687, R01 GM087285-01, T32-GM008256, and P50-GM60338), NIDRR (H133A020102), CFI Leader's Opportunity Fund (Project #25407), and Physicians' Services Incorporated Foundation.

We would like to thank all the individuals who participated in this study. We also would like to thank all the research staff, Eileen Figueroa, Steven Schuenke and Alisihia Goldberg from the Institute for Translational Sciences for their assistance in manuscript preparation.

We would like to thank Ruxandra Pinto from TECC, Sunnybrook Health Sciences Centre, Toronto, Ontario for her statistical suggestion and corrections, and incredible help.

ROLE OF FUNDING SOURCE:

All funding sources had no involvement in the collection, analysis, and interpretation of data.

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Figure 1. Kaplan Meier survival curve (A) and Cut-Off determination (B) using sensitivity and specificity validated by ROC analysis (C).

Figure 2. Changes in the metabolic response: Insulin resistance increasing with burn size demonstrated with glucose and serum insulin levels (A, B). Increased REE (C). Impaired protein production of liver synthesized proteins (D–K) and elevated triglyceride levels (L).

Figure 3. Increased inflammatory response shown by pro-inflammatory cytokines (A–J) and CRP (K). Attenuated organ function of liver (L, M), increase of liver size according to burn size (N) and kidneys shown with organ specific markers (O).

Table 1

Patient Demographics

	All	30-39	40-49	50-59	60-69	70-79	80-89	90-100	P value
n	952	180	260	171	123	85	82	51	
Gender									
Male n (%)	628 (68)	122 (68)	167 (64)	112 (65)	73 (59)	61 (71)	51 (62)	42 (82)	
Female n (%)	324 (32)	58 (32)	93 (36)	59 (35)	50 (41)	24 (29)	31 (38)	9 (18)	
Ethnicity									
Afro American (n)	73 (8)	18 (10)	24 (9)	7 (4)	10 (8)	6 (7)	4 (5)	4 (8)	
Caucasian (n)	154 (16)	48 (27)	32 (12)	24 (14)	13 (11)	10 (12)	12 (15)	15 (29)	
Hispanic (n)	697 (73)	106 (59)	200 (77)	136 (80)	96 (78)	67 (79)	61 (74)	31 (61)	
Other Origin (n)	28 (3)	8 (4)	4 (2)	4 (2)	4 (3)	2 (2)	5 (6)	1 (2)	
Type of Burn									
Flame n (%)	637 (67)	109 (61)	167 (64)	116 (68)	85 (69)	64 (75)	68 (83)	28 (55)	
Scald n (%)	215 (23)	59 (33)	69 (27)	39 (23)	24 (20)	13 (15)	5 (6)	6 (12)	
Other n (%)	100 (10)	12 (7)	24 (9)	16 (9)	14 (11)	8 (10)	9 (11)	17 (33)	
Age admit (years)	7.3 ± 5.3	6.1 ± 5.1	7.1 ± 5.2	7.6 ± 5.1	7.2 ± 5.1	8.3 ± 5.9	8.4 ± 5.6	9.6 ± 5.4	<0.001
Inhal Injury n (%)	321 (34)	28 (16)	85 (33)	50 (29)	55 (45)	38 (45)	38 (46)	27 (53)	<0.001
TBSA burn	55 ± 18	34 ± 3	44 ± 3	54 ± 3	64 ± 3	73 ± 3	83 ± 3	95 ± 3	<0.001
TBSA third (%)	38 ± 27	17 ± 14	27 ± 17	36 ± 20	47 ± 22	56 ± 25	69 ± 26	76 ± 34	<0.001
Burn to admit (days)	3.6 ± 4.3	3.7 ± 4.8	4.4 ± 4.9	3.9 ± 4.0	2.8 ± 3.1	2.6 ± 2.6	3.0 ± 4.0	2.5 ± 4.1	<0.001
Admit to excision	1.0 ± 1.6	1.2 ± 1.9	1.0 ± 1.7	0.9 ± 1.4	0.8 ± 1.1	0.9 ± 1.2	0.9 ± 1.5	0.9 ± 2.2	0.07

Table 2

Clinical Outcome Parameters

	All	30-39	40-49	50-59	60-69	70-79	80-89	90-100	P value
n	952	180	260	171	123	85	82	51	
OR (n)	3.8 ± 3.2	2.1 ± 1.5	2.7 ± 1.8	3.3 ± 2.1	4.8 ± 2.9	5.0 ± 3.2	6.6 ± 4.4	8.6 ± 5.7	<0.001
Time btw OR (days)	4.9 ± 2.3	4.0 ± 2.2	4.4 ± 1.7	4.9 ± 3.2	5.2 ± 1.6	5.4 ± 1.5	5.6 ± 1.7	5.9 ± 3.0	<0.001
LOS ICU (days)	25.9 ± 22.7	13.5 ± 12.1	18.8 ± 11.5	23.7 ± 14.3	34.5 ± 17.6	40.3 ± 28.7	52.5 ± 28.9	72.8 ± 52.0	<0.001
LOS/TBSA	0.5 ± 0.3	0.4 ± 0.3	0.4 ± 0.3	0.4 ± 0.3	0.5 ± 0.3	0.5 ± 0.4	0.6 ± 0.3	0.8 ± 0.5	<0.001
LOS/TBSA 3 rd	1.2 ± 2.9	1.3 ± 2.1	1.3 ± 2.3	1.5 ± 5.4	0.9 ± 1.3	1.0 ± 1.4	0.8 ± 0.5	1.1 ± 0.4	0.08
Died n (%)	120 (13)	5 (3)	7 (3)	12 (7)	20 (16)	19 (22)	29 (35)	28 (55)	<0.001
Max DENVER2	3.4 ± 1.8	2.5 ± 1.6	2.8 ± 1.3	3.4 ± 1.3	4.0 ± 1.7	4.2 ± 2.0	4.6 ± 2.2	5.3 ± 2.1	<0.001
MOF n (%)	154 (17)	10 (6)	16 (6)	21 (12)	33 (27)	25 (29)	36 (44)	23 (45)	<0.001
Sepsis n (%)	89 (9)	3 (2)	14 (5)	11 (6)	19 (15)	11 (13)	18 (22)	13 (26)	<0.001
Infections (n)	5.8 ± 5.9	2.8 ± 1.8	3.8 ± 2.9	4.1 ± 3.7	7.7 ± 6.4	9.3 ± 9.6	8.6 ± 6.1	10.4 ± 8.0	<0.001

Table 3

Multiple logistic regression.

Parameter/Effect	Odds Ratio Estimates			P value
	Point Estimate	95% Wald	Confidence Limits	
Burn Area (% TBSA) ≥ 60	10.07	5.56	18.22	<.0001
Inhalation Injury (Yes)	2.97	1.81	4.85	<.0001
Gender (Female)	2.26	1.37	3.71	0.0013
Age at Admit (Years)	1.022	0.98	1.07	0.3489
Burn to Admit (Days)	0.99	0.92	1.06	0.6651