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Differences in resuscitation in morbidly obese burn patients may contribute to high mortality

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Abstract

Objective—The rising number of obese patients poses new challenges for burn care. These may include adjustments in calculations of burn size, resuscitation, ventilator wean, nutritional goals as well as challenges in mobilization. We have focused this observational study on resuscitation in the obese patient population in the first 48 hours after burn injury. Prior trauma studies suggest a prolonged time to reach end points of resuscitation in the obese compared to non-obese injured patients. We hypothesize that obese patients have worse outcomes after thermal injury and that differences in the response to resuscitation contribute to this disparity.

Methods—We retrospectively analyzed data prospectively collected in a multi-center trial to compare resuscitation and outcomes in patients stratified by NIH/WHO BMI classification (BMI: normal weight 18.5-24.9, overweight 25-29.9, Obese 30-39.9, morbidly obese ≥40). Due to distribution of body habitus in the obese, total burn size was recalculated for all patients using the method proposed by Neaman and compared to Lund-Browder estimates. We analyzed patients by BMI class for fluids administered and end points of resuscitation at 24 and 48 hours. Multivariate analysis was used to compare morbidity and mortality across BMI groups.

Results—We identified 296 adult patients with a mean TBSA of 41%. Patient and injury characteristics were similar across BMI categories. There were no significant differences in burn size calculations using Neaman vs. Lund-Browder formulas. Although resuscitation volumes exceeded the predicted formula in all BMI categories, higher BMI was associated with less fluid administered per actual body weight ($p=0.001$). Base deficit on admission was highest in the morbidly obese group at 24 and 48 hours. Furthermore, these patients did not correct their metabolic acidosis as well as lower BMI groups (p -values 0.04 and 0.03). Complications and morbidities across BMI groups were similar, although examination of organ failure scores indicated more severe organ dysfunction in the morbidly obese group. Compared to normal weight patients, being morbidly obese was an independent risk factor for death (OR = 10.1; CI 1.94-52.5, $p=0.006$).

Conclusions—Morbidly obese patients with severe burns tend to receive closer to predicted fluid resuscitation volumes for their actual weight. However, this patient group has persistent metabolic acidosis during the resuscitation phase and is at-risk of developing more severe multiple organ failure. These factors may contribute to higher mortality risk in the morbidly obese burn patient.

Keywords

burns; resuscitation; obesity

Introduction

Obesity consistently increases morbidity and mortality in both acute and chronic illnesses^{1,2}. The incidence of obesity is increasing in the general population, challenging providers to adapt to this co-morbidity. In particular, critically ill obese patients present unique challenges. There seems to be consensus that obese patients have increased days on the ventilator, ICU days and total hospital days compared to normal weight patients^{3,4}. After blunt trauma, obese patients have a persistent metabolic acidosis and require larger fluid volumes to reach desired end points of resuscitation⁵⁻⁷.

In burn care, the obese patient poses many clinical challenges such as accurate measurement of burn size, resuscitation, mechanical ventilation, wound infections, mobilization, effective DVT prophylaxis and nutritional goals. There is a paucity of literature on how best to treat the obese burn patient both initially and throughout recovery and rehabilitation. Obesity appears to be associated with higher risk of complications and deaths after major burns^{8,9}. When Purdue and colleagues retrospectively examined a cohort of 1988 patients, they found that obese patients had much higher odds of death than normal weight patients¹⁰. The resuscitation phase may have an important impact on these outcomes. Current knowledge indicates that both under and over-resuscitation increases morbidity^{11,12}. Resuscitation difficulties, including prolonged acidosis, correlate with worse patient outcomes^{13,14}. Furthermore, early organ failure and early rapid decline are the most common trajectories to death (80%) in severely burned patients¹⁵.

A better understanding of how obesity alters the early injury response after burn is needed to improve clinical outcomes. The objective of this study was to assess the impact of BMI on resuscitation during the first 48 hours following injury. We hypothesized that differences in early resuscitation in morbidly obese burn patients may contribute to worse outcomes.

Methods

Study design

This study was a retrospective analysis of data prospectively collected in a multi-center trial “Inflammation and the Host Response to Injury, the Glue Grant” to compare differences in resuscitation and outcomes in adult patients across body mass index (BMI) groups. Patients were stratified by NIH/WHO Body Mass Index (BMI) classification and compared: normal weight = 18.5-24.9, overweight = 25-29.9, obese = 30-39.9, and morbidly obese ≥ 40.

Due to differences in the body habitus of obese patients, we questioned the accuracy of estimated burn size in our database. Using three-dimensional whole body scanning, Neaman and colleagues previously developed a new method to calculate body surface area for specific body parts, stratified to each of the NIH/WHO BMI classifications¹⁶. Given that our dataset contained percent burn by body part, we first calculated the ratio of burns/body part according to Lund-Browder. We then applied this ratio to the Neaman estimates and

derived a recalculated burn size. The sum of burned areas constituted the recalculated “Neaman” TBSA burns. We then compared this “Neaman” burn size to the original Lund-Browder estimates. The intent was to assess whether differences in resuscitation in the obese patients could be explained by discrepant burn size estimates.

We examined resuscitation parameters across BMI groups including: volume of fluids given in the first 24 and 48 hours, urine output, incidence of acute kidney injury, serum lactate, pH and base deficit. The main outcomes of interest were differences in fluids administered, commonly used resuscitation end points, multiorgan failure, complications and mortality.

Selection of study subjects

Study enrollment criteria included all adult patients from 2000-2008 with TBSA $\geq 20\%$ admitted to one of the participating burn centers. Burn center admission and enrollment into the study occurred within 96 hours of injury. In patients enrolled after 48 hours, data from the time of admission was entered in the Database retroactively. Patients were excluded from study participation if they died within 48 hours of injury following the medical decision to withdraw support or decision ‘not to resuscitate’. Underweight patients, defined as a BMI <18.5 , were excluded due to predictably worse outcomes compared to normal weight patients; the physiology of which is distinct compared to the increased morbidity and mortality seen in the obese patient population¹⁷⁻¹⁹.

Study data are collected, verified and entered into a central database. Study data were abstracted and analyzed according to protocol approved by the University of Washington Institutional Review Committee.

Patient care protocols

Fluid resuscitation during the study was based on the Parkland formula estimate of 4ml/kg/TBSA administered intravenously over the first 24 hours. Adjustments to this protocol were made to maintain mean arterial pressure >60 mmHg and urine output >30 ml/hr^{20, 21}. There was no consensus of whether actual weight or ideal body weight should be implemented into resuscitation estimates with higher BMI.

Study outcomes

The outcomes of interest in this study included accuracy of burn size calculations in the obese population, differences in fluid administered during resuscitation in the first 48 hours after burn injury and whether these differences correlate to increased morbidity and mortality in the morbidly obese patient. To assess these outcomes, urine output and serum creatinine changes were analyzed to calculate the incidence of acute kidney injury as defined by the Acute Kidney Injury Network (AKIN) criteria^{22, 23}. AKIN criteria defines acute kidney injury as a 1.5 fold increase in creatinine or ≥ 0.3 mg/dL above baseline creatinine or urine output of less than 0.5 ml/kg/hr for more than 6 hours. End points of resuscitation captured included serum lactate, pH and base deficit. Unresolved base deficit (BD) was defined as worsening base deficit at 24 hours and base deficit values at 48 hours that are greater compared to 24 hours post injury. Morbidity and mortality data included incidence of acute respiratory distress syndrome (ARDS), sepsis and severity of multiorgan failure (by Denver 2 scores).

Statistical analysis

Analyses were performed using the STATA 10 program (College Station, Texas®) with actual p values reported. Multivariate analysis was used and data was adjusted for age, gender, total body surface area burned (TBSA) and inhalation injury. Differences in resuscitation were compared using Kruskal-Wallis for continuous data and chi-square for

categorical data. Whenever differences were detected among weight categories, we performed posthoc pairwise comparisons using the Dunn-Sidak test for continuous data and univariate logistic regression for categorical data in order to evaluate for individual group differences. Logistic regression and cox proportional hazard models were performed to assess for differences in length of hospital stay, ICU stay, ventilator days, incidence of sepsis, ARDS, multi-organ failure, nosocomial infections and mortality stratified to each BMI classification.

Results

Patient and injury characteristics

Retrospective analysis of the database identified 296 adult patients who met the study criteria. Stratified by BMI classification, there were 106 normal weight patients, 95 overweight, 80 obese and 15 morbidly obese. This is a severely burned cohort of patients with an average burn size of 41%, a high rate of inhalation injury (36-53%) and APACHE 2 scores around 20. Mean age was 40 years old. Men outnumbered women except in the morbidly obese class (Table 1). Overall, patient and injury characteristics across BMI categories were not statistically different except for the predominance of female sex in the morbidly obese group ($p=0.004$).

Recalculation of TBSA Burned

For each body location of the Lund-Browder estimates, injury size was recalculated as a ratio of the surface area using the Neaman method, stratified to each BMI classification. The total burn size for each method was compared. In aggregate, it showed no clinical or statistical difference between the two methods of calculating burn size (Figure 1). Therefore, we continued our analysis using the original Lund-Browder calculations. However, we note that individually, several patients' burn size estimates changed dramatically. For instance, Neaman calculation dropped an obese patient's TBSA to 6% although it was originally estimated at 20% by Lund-Browder (Figure 1).

Resuscitation data

Fluids administered were expressed as ratio of observed/expected volume of fluids, based on the Parkland formula of 4 ml/kg/% TBSA burn. Comparisons differed greatly depending on whether actual or ideal body weight is assumed in the predicted formula. Although not necessarily used clinically, ideal body weight serves as a useful benchmark to index resuscitation volumes across weight classes. Obese patients received a significantly lower volume of fluids per actual body weight compared to normal weight patients ($p=0.001$). When indexed to ideal body weight, the volume of fluid received was predictably higher in the morbidly obese patients ($p=0.01$) and confirmed by posthoc comparison (Figure 2). In fact, when resuscitation is indexed to ideal body weight, the morbidly obese group received a significantly higher volume of resuscitation compared to all other groups ($p<0.05$ for all pairwise comparisons). As BMI increased, urine output increased in milliliters per kilogram for both actual and ideal body weight. Though significant by initial Kruskal-Wallis rank test, posthoc pairwise comparisons did not confirm statistical differences in urine output between the weight categories (Figure 3). Finally, there was no difference in the incidence of acute kidney injury among the groups as determined by AKIN criteria (Table 3).

In evaluating the degree of metabolic derangement by analyzing end points of resuscitation across BMI groups, there were higher median base deficit values on admission in the morbidly obese group with declining median values in the obese, overweight and normal weighted groups. Initial lactate levels were consistently elevated across all BMI categories (Figure 4). Figure 5 summarizes the resolution of metabolic acidosis measured by base

deficit among BMI categories. At 24 hours, 33% of morbidly obese patients had increasing base deficit values compared to 6-12% in other BMI classes. At 48 hours, an even higher percentage of morbidly obese patients had unresolved base deficit (45%). Further analysis by univariate logistic regression confirmed that morbidly obese patients had significantly worse base deficits compared to normal weight patients at 24 ($p=0.02$) and 48 hours ($p=0.05$). Thus, morbidly obese patients have difficulty resolving their initial metabolic acidosis and these derangements persist longer than in their counterparts.

Incidence and severity of complications

Analysis of complications over the entire length of the patients' hospitalization in each BMI category was compared to normal weight patients. We found that the incidence of acute respiratory distress syndrome (ARDS), development of multi-organ failure (MOF) and sepsis was not significantly different among the 4 weight categories compared to the normal weight patients in our cohort. Still, morbid obesity was an independent risk factor for mortality in the burn patient (OR = 10.1; CI 1.94-52.5, $p=0.006$) (Table 2) when correcting for age, burn size, inhalation injury and sex.

We further explored potential causes for the excess mortality seen in the morbidly obese group. We found that in spite of equivalent incidence of multiorgan failure and sepsis, the cause of death in 57% of the morbidly obese group was sepsis and/or multi-organ failure. But multi-organ failure is not an all-or-none binary complication. To better understand the severity of multi-organ failure, we divided Denver scores into tertiles (0-3: none to mild, 4-7: moderate, 8-12: severe). We observed a greater severity of organ failure in the morbidly obese group: 4 out of 15 patients were categorized as severe (26.6%), whereas none had moderately severe organ failure. Potentially a shift from moderate to severe organ failure has occurred in the morbidly obese subjects compared to other weight categories, although it did not reach statistical significance ($p=0.09$, Figure 6). There were no differences in hospital length of stay, ICU stay or days on the ventilator.

Discussion

The obesity epidemic is changing our approach to patient care in the United States and burn care is no exception. Currently there is little data to define the factors that affect morbidity and mortality in the obese burn patient. In this observational study we examined resuscitation factors that may contribute to the disparity in outcomes.

In this observational multicenter study, our principal finding was that morbidly obese patients have a significantly increased risk of mortality after thermal injury. We sought to analyze factors during early resuscitation that may influence or contribute to this finding. We found that the morbidly obese burn patients have a delayed resolution of their base deficit by 48 hours. This suggests that morbidly obese patients take a longer time to reach end points of resuscitation and normalize their metabolic derangements. This finding is consistent with findings in the trauma literature in the obese population⁵. A persistent base deficit is known to correlate with a poor prognosis and may contribute to the higher mortality rate in the morbidly obese patient^{25,13, 14}. Though the incidence of multi-organ failure was similar, there was a trend toward greater severity of dysfunction in the morbidly obese. Perhaps a shift toward more severe multiorgan dysfunction occurred in the morbidly obese group. Further investigation revealed that within the morbidly obese group, 57% died of multi-organ failure with or without sepsis. It seems the morbidly obese patients have either increased severity of multi-organ failure, or decreased ability to recover from the insult.

Contrary to our expectation, we note that only morbidly obese patients had excess mortality in this cohort. This is perhaps consistent with recent findings that some excess weight does not necessarily confer higher mortality risk. A recent population study has highlighted a lower risk for all-cause mortality in overweight individuals and an equivalent risk in the mildly obese compared normal weight patients²⁶. In trauma and critical care however, there is a growing body of literature that demonstrates obesity is an independent risk factor for mortality^{3, 27, 28}. As well there is evidence that obesity may be a risk factor for more severe multi-organ failure in trauma patients²⁹. This is consistent with the observations in our cohort. It has also been noted that end points of resuscitation may be delayed in the severely injured obese patient²⁵. Given that acidosis persists at 48 hours in obese patients, future studies should track metabolic derangements beyond the initial resuscitation phase. Potentially this could lead to better understanding of their inflammatory response and whether these metabolic end points are appropriate indicators of resuscitation in this patient population. The question remains, will optimization in early resuscitation affect the observed increased multiorgan failure and mortality in the obese population?

Limitations

Despite our desire to define the best resuscitation method for overweight and obese patients, this retrospective study cannot answer this fundamental question. By titrating to urine output, the participating centers administered less fluid per actual body weight in the first 24 hours in obese patients. Despite a similar incidence of acute kidney injury, resolution of base deficit still lagged in the morbidly obese. Although it may be tempting to argue that this subset should have received more fluid, we cannot make this inference without data to confirm this hypothesis. A prospective comparative study is necessary to resolve best resuscitation practice in obese patients. A limitation of this study includes a lack of a uniform approach to resuscitation with no consensus (standard operating procedures) on fluid resuscitation in the obese patient²⁰. Nevertheless, it allowed us to perform an observational study of current practice. Furthermore, it underscores the importance of multicenter collaborative efforts, because only 15 subjects were morbidly obese (BMI>40) in this large cohort.

Conclusions

In conclusion, this study corroborates previous findings that morbidly obese burn patients have a higher mortality than their normal weighted counterparts. It provides a starting point to examine unique challenges in the morbidly obese burn patient, and their response to severe thermal injury. Further study is needed to define optimal resuscitation in the obese population, as well as to understand the most potent contributors to higher mortality.

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Disclosure

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References

1. Berrington de Gonzalez A, Hartge P, Cerhan JR, et al. Body-mass index and mortality among 1.46 million white adults. *The New England journal of medicine*. 2010; 363(23):2211–9. [PubMed: 21121834]
2. Edmonds RD, Cuschieri J, Minei JP, et al. Body adipose content is independently associated with a higher risk of organ failure and nosocomial infection in the nonobese patient postinjury. *The Journal of trauma*. 2011; 70(2):292–8. [PubMed: 21307724]
3. Akinnusi ME, Pineda LA, El Solh AA. Effect of obesity on intensive care morbidity and mortality: a meta-analysis. *Critical care medicine*. 2008; 36(1):151–8. [PubMed: 18007266]
4. Hogue CW Jr, Stearns JD, Colantuoni E, et al. The impact of obesity on outcomes after critical illness: a meta-analysis. *Intensive care medicine*. 2009; 35(7):1152–70. [PubMed: 19189078]
5. Winfield RD, Delano MJ, Dixon DJ, et al. Differences in outcome between obese and nonobese patients following severe blunt trauma are not consistent with an early inflammatory genomic response. *Critical care medicine*. 2010; 38(1):51–8. [PubMed: 19661803]
6. Winfield RD, Delano MJ, Cuenca AG, et al. Obese patients show a depressed cytokine profile following severe blunt injury. *Shock*. 2012; 37(3):253–6. [PubMed: 22266966]
7. Foldi V, Csontos C, Bogar L, Roth E, Lantos J. Effects of fluid resuscitation methods on burn trauma-induced oxidative stress. *Journal of burn care & research : official publication of the American Burn Association*. 2009; 30(6):957–66. [PubMed: 19826270]
8. Sheridan RL, Rue LW 3rd, McManus WF, Pruitt BA Jr. Burns in morbidly obese patients. *The Journal of trauma*. 1992; 33(6):818–20. [PubMed: 1474621]
9. Ghanem AM, Sen S, Philp B, Dziewulski P, Shelley OP. Body Mass Index (BMI) and mortality in patients with severe burns: is there a “tilt point” at which obesity influences outcome? *Burns : journal of the International Society for Burn Injuries*. 2011; 37(2):208–14. [PubMed: 21129850]
10. Purdue GF, Hunt JL, Lang ED. Obesity: a risk factor in the burn patient. *The Journal of burn care & rehabilitation*. 1990; 11(1):32–4. [PubMed: 2312589]
11. Pruitt BA Jr. Protection from excessive resuscitation: “pushing the pendulum back”. *The Journal of trauma*. 2000; 49(3):567–8. [PubMed: 11003341]
12. Greenhalgh DG. Burn resuscitation. *Journal of burn care & research : official publication of the American Burn Association*. 2007; 28(4):555–65. [PubMed: 17665515]
13. Tremblay LN, Feliciano DV, Rozycki GS. Assessment of initial base deficit as a predictor of outcome: mechanism of injury does make a difference. *The American surgeon*. 2002; 68(8):689–93. discussion 93–4. [PubMed: 12206603]
14. Jeng JC, Lee K, Jablonski K, Jordan MH. Serum lactate and base deficit suggest inadequate resuscitation of patients with burn injuries: application of a point-of-care laboratory instrument. *The Journal of burn care & rehabilitation*. 1997; 18(5):402–5. [PubMed: 9313119]
15. Swanson JW, Otto AM, Gibran NS, et al. Trajectories to death in patients with burn injury. *The journal of trauma and acute care surgery*. 2013; 74(1):282–8. [PubMed: 23271104]
16. Neaman KC, Andres LA, McClure AM, Burton ME, Kemmeter PR, Ford RD. A new method for estimation of involved BSAs for obese and normal-weight patients with burn injury. *Journal of burn care & research : official publication of the American Burn Association*. 2011; 32(3):421–8. [PubMed: 21562463]
17. Tremblay A, Bandi V. Impact of body mass index on outcomes following critical care. *Chest*. 2003; 123(4):1202–7. [PubMed: 12684312]
18. Gupta R, Knobel D, Gunabushanam V, et al. The effect of low body mass index on outcome in critically ill surgical patients. *Nutrition in clinical practice : official publication of the American Society for Parenteral and Enteral Nutrition*. 2011; 26(5):593–7. [PubMed: 21947642]
19. Garrouste-Orgeas M, Troche G, Azoulay E, et al. Body mass index. An additional prognostic factor in ICU patients. *Intensive care medicine*. 2004; 30(3):437–43. [PubMed: 14767583]
20. Klein MB, Silver G, Gamelli RL, et al. Inflammation and the host response to injury: an overview of the multicenter study of the genomic and proteomic response to burn injury. *Journal of burn care & research : official publication of the American Burn Association*. 2006; 27(4):448–51. [PubMed: 16819346]

21. Klein MB, Hayden D, Elson C, et al. The association between fluid administration and outcome following major burn: a multicenter study. *Annals of surgery*. 2007; 245(4):622–8. [PubMed: 17414612]
22. Englberger L, Suri RM, Li Z, et al. Clinical accuracy of RIFLE and Acute Kidney Injury Network (AKIN) criteria for acute kidney injury in patients undergoing cardiac surgery. *Critical care*. 2011; 15(1):R16. [PubMed: 21232094]
23. Pisitkun T, Tiranathanagul K, Poulin S, et al. A practical tool for determining the adequacy of renal replacement therapy in acute renal failure patients. *Contributions to nephrology*. 2004; 144:329–49. [PubMed: 15264421]
24. Englberger L, Suri RM, Li Z, et al. Clinical accuracy of RIFLE and Acute Kidney Injury Network (AKIN) criteria for acute kidney injury in patients undergoing cardiac surgery. *Critical care*. 2011; 15(1):R16. [PubMed: 21232094]
25. Winfield RD, Delano MJ, Lottenberg L, et al. Traditional resuscitative practices fail to resolve metabolic acidosis in morbidly obese patients after severe blunt trauma. *The Journal of trauma*. 2010; 68(2):317–30. [PubMed: 20154544]
26. Flegal KM, Kit BK, Orpana H, Graubard BI. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. *JAMA : the journal of the American Medical Association*. 2013; 309(1):71–82. [PubMed: 23280227]
27. Neville AL, Brown CV, Weng J, Demetriades D, Velmahos GC. Obesity is an independent risk factor of mortality in severely injured blunt trauma patients. *Archives of surgery*. 2004; 139(9): 983–7. [PubMed: 15381617]
28. Bochicchio GV, Joshi M, Bochicchio K, Nehman S, Tracy JK, Scalea TM. Impact of obesity in the critically ill trauma patient: a prospective study. *Journal of the American College of Surgeons*. 2006; 203(4):533–8. [PubMed: 17000398]
29. Ciesla DJ, Moore EE, Johnson JL, Burch JM, Cothren CC, Sauaia A. Obesity increases risk of organ failure after severe trauma. *Journal of the American College of Surgeons*. 2006; 203(4):539–45. [PubMed: 17000399]

Figure 1. Burn Size: Lund-Browder vs. Neaman Method. Results are reported as Box-whisker plots with Median, Interquartile and absolute ranges represented. There are no significant differences between the groups.

Figure 2.

Observed/expected fluid ratios in the first 24 hours of resuscitation, indexed to actual and ideal body weight. (*) Indicates $p < 0.05$ compared with normal weight category (**) Indicates $p < 0.05$ compared with all other weight groups by posthoc pairwise comparison. Results are reported as Box-whisker plots with Median, Interquartile and absolute ranges represented.

Urine output (ml/kg) first 24 hours post injury

Figure 3.

Urine output during the first 24 hours of resuscitation, indexed to actual and ideal body weight. Results are reported as Box-whisker plots with Median, Interquartile and absolute ranges represented. There are no significant differences between the groups.

Figure 4.

Initial base deficit and lactate levels. Results are reported as Box-whisker plots with Median, Interquartile and absolute ranges represented. There are no significant differences between groups.

Figure 5.

Incidence of unresolved base deficit expressed as the percentage of patients with increasing base deficit at 24 hours and base deficit values at 48 hours > 24 hours post injury. (*)

Indicates $p < 0.05$ difference with normal weight categories

Figure 6.

Incidence of organ dysfunction: normal-to-mild, moderate or severe organ failure by BMI class. No significant differences between groups.

Patient and Injury Characteristics, there were no difference between the groups except for gender. The cohort is predominately male with a significantly higher number of females in the morbidly obese group.

Table 1

	NORMAL (n=106)	OVERWEIGHT (n=95)	OBESE (n=80)	MORBIDLY OBESE (n=15)	P-value
AGE	41.1 (15.6)	42.8 (17.2)	39.1 (14.2)	39.2 (12.1)	0.84
MALE (%)	82	76	74	40	0.004
APACHE	20.8 (9.3)	19.9 (9.1)	21.5 (9.1)	21.8 (9.8)	0.78
TBSA (%)	40.7 (19.2)	41.1 (18.7)	39.6 (16.8)	42.5 (11.3)	0.48
INHALATION INJURY (%)	40	38	36	53	0.47
BAUX SCORE	81.8 (22.0)	83.9 (23.2)	78.7 (20.1)	81.7 (20.5)	0.48

Data are reported as means (std dev) or percent where indicated.

Risk of in-hospital complications, with normal weight group as reference, Odds ratio adjusted for age, sex, burn size and inhalation injury.

Table 2

	OVERWEIGHT OR (95% CI)	OBESE OR (95% CI)	MORBIDLY OBESE OR (95% CI)
ARDS	1.07 (0.6-1.7)	1.42 (0.8-2.3)	1.26 (0.5-3.3)
Multi-organ failure	0.96 (0.5-1.6)	1.36 (0.7-2.4)	1.60 (0.5-4.6)
Multi-organ failure and/or Sepsis	1.01 (0.6-1.7)	1.42 (0.7-2.5)	2.31 (0.8-6.2)
Mortality	0.55 (0.2-1.4)	0.65 (0.2-1.8)	10.1 (1.9-52.5) *

Reported odds ratios are adjusted for age, sex, burn size and inhalation injury.

* Indicates p = 0.006 compared to normal weight patients.

Table 3

Incidence of early acute kidney injury (AKIN Criteria).

	Normal Wt n=106	Over Wt n=95	Obese n=80	Morbid Obese n=15	P-value
Actual Body Weight Index	35 (33)	37 (39)	25 (31)	5 (33)	0.62
Ideal Body Weight Index	29 (27)	28 (29)	18 (23)	3 (20)	0.81

Data are reported as n (%). There are no significant differences between the groups.