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Papers presented

Systemic hypotension is a late marker of shock after trauma: a validation study of Advanced Trauma Life Support principles in a large national sample

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Abstract

Background: Systolic blood pressure is used extensively to triage trauma patients as stable or unstable, contrary to Advanced Trauma Life Support recommendations. We hypothesized that systemic hypotension is a late marker of shock.
Methods: The National Trauma Data Bank was queried (n = 115,830). Base deficit was used as a measure of circulatory shock. Systolic blood pressure was correlated with the presence and the severity of base-deficit derangement.
Results: Systolic blood pressure correlated poorly with base deficit (r = .28). There was wide variation in systolic blood pressure within each base-deficit group. The mean and median systolic blood pressure did not decrease to less than 90 mm Hg until the base deficit was worse than −20, with mortality reaching 65%.
Conclusions: We validated the Advanced Trauma Life Support principle that systemic hypotension is a late marker of shock. A normal blood pressure should not deter aggressive evaluation and resuscitation of trauma patients.

Keywords: Hemorrhagic shock; Blood pressure; Hypotension; Metabolic acidosis; Injuries; Advanced Trauma Life Support

Hemorrhage is the second leading cause of death in trauma patients, exceeded only by deaths caused by traumatic brain injuries [1,2]. If the shock state becomes severe, the survival rate is less than 50%, which highlights the need for early detection of occult hemorrhagic shock [3]. Several clinical and laboratory measures have been developed to detect the presence of shock in critically injured patients [4–7]. Clinical indicators include tachycardia, tachypnea, hypotension, cutaneous hypoperfusion, mental status alterations, and oliguria [8–11]. Laboratory indicators of shock include hypoxemia, lactate, systemic, and gastric mucosal acidosis, and base deficit [12–14].

Systolic blood pressure (SBP) is used extensively in prehospital settings and emergency departments to classify trauma patients as hemodynamically stable or unstable. This occurs despite the Advanced Trauma Life Support (ATLS of the American College of Surgeons—Committee on Trauma) long-standing principle that systemic hypotension is a late sign of shock, and therefore is a late measure of hemodynamic instability [2]. ATLS defines 4 stages of shock, based on the estimated percentage of blood volume lost. Notably, it is not until patients reach class III, which represents a 30% to 40% loss of blood volume (>1,500 mL in a 70-kg adult), that a measurable decrease in SBP is expected to occur [2].

The purpose of this study was to evaluate the ATLS principle that hypotension is not a reliable indicator of the presence of shock in a large national sample of trauma patients. We hypothesized that systemic hypotension is a late sign of shock, and, hence, SBP should be considered an unreliable means of detecting the presence of shock.

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Methods

The National Trauma Data Bank (NTDB) of the American College of Surgeons, the largest nationwide database of trauma patients, with more than 400 participating trauma centers and more than 1 million individual records, was queried (NTDB, version 4.0, 2004; N = 1,130,581). We used base deficit in the emergency department as a marker of shock because it has been validated previously as a reliable marker of shock severity in injured patients [11,14–16]. Base deficit was measured in a small proportion of patients (n = 122,159; 11%). Patients with an emergency department base-deficit value between +10 and −30 were included (n = 117,686). The first SBP measured in the emergency department was used to determine the presence or absence of systemic hypotension, which was defined as SBP less than 90 mm Hg. Patients with incomplete information on SBP were excluded. The final study population consisted of 115,830 patients.

We first validated base deficit as a marker of shock in the study population. Logistic regression analysis showed that base deficit was an independent predictor of mortality after accounting for mechanism of injury; Injury Severity Score; Glasgow Coma Scale (GCS); SBP; age; presence of head, chest, and abdominal injuries; comorbidities; and complications. These covariates were included in the model based on their clinical relevance to the dependent variable (ie, death). A best-fit model was developed using forward and backward elimination techniques, and after considering all possible 2-way interaction terms. The final model contained base-deficit groups, SBP, GCS, mechanism of injury, and 2-way interactions between age and GCS, Injury Severity Score and GCS, and SBP and GCS. The removal of additional terms, including base-deficit groups, resulted in a worse fitting model. The final model was a good predictor of mortality with a Hosmer-Lemeshow statistic of 5.75 (P .68), and 92% concordance between the observed and predicted mortality. Patients were then stratified by severity of shock by dividing the base-deficit level into 7 groups, based on the magnitude of derangement (Table 1). The validity of base deficit as a measure of shock was confirmed by the observation that mortality increased with worsening base deficit.

The relationship between severity of shock and SBP was measured using 2 approaches. First, the mean and median SBP was measured within each shock group. Second, the correlation between SBP and base deficit was measured using the Pearson correlation coefficient. The results are presented as the mean ± SD, medians with interquartile ranges, and Pearson r. A P value of less than .05 was considered significant for all statistical analyses. SPSS (SPSS Inc., Chicago, IL) and SAS (SAS, Inc. Cary, NC) were used for all statistical analyses.

Results

In the study population, 64% of patients were 15 to 44 years of age, 71% were male, 82% sustained a blunt injury, and 74% were treated at a level 1 trauma center. The mean GCS was 12 ± 5, the Injury Severity Score was 16 ± 13, and the base deficit was −2 ± 5. The overall mortality rate was 10%, with a mean length of stay of 9 ± 15 days. Systemic hypotension was noted in 7,910 patients (7%). Compared with patients with a higher SBP, hypotensive patients had a significantly higher mortality rate (37% vs. 8%, $P < .001$), a higher rate of penetrating injuries (26% vs. 15%, $P < .001$), a higher incidence of complications (26% vs. 14%, $P < .001$), more comorbidities (42% vs. 25%, $P < .001$), more chest injuries (47% vs. 33%, $P < .001$), more abdominal injuries (36% vs. 20%, $P < .001$), a lower GCS (9 ± 5 vs. 12 ± 4, $P < .001$), higher Injury Severity Score (26 ± 17 vs. 15 ± 12, $P < .001$), and worse base deficit (−8 ± 8 vs. −2 ± 5, $P < .001$). There was a negligible difference in the incidence of head injury (41% vs. 40%, $P \leq .05$).

Emergency department SBP correlated poorly with base deficit. The Pearson correlation coefficient, although statistically significant at a P value of .01 or less, was very low (r = .28), indicating that the correlation between SBP and severity of shock was very poor. Wide variations in SBP were found within each base-deficit group (Fig. 1). SBP decreased progressively with worsening base deficit, but the mean and median values did not decrease to less than 90 mm Hg until base deficit was worse than −20, with mortality approaching 65% (Table 1).

Comments

This study shows that systemic hypotension in trauma patients does not occur until the degree of shock is profound, and, hence, is an unreliable means of detecting clinically significant shock. This study validates the long-standing ATLS principle that hypotension is a late marker of shock in a large national sample of trauma patients. Although the relationship between systemic hypotension and outcome is well documented, few studies have shown the magnitude of shock and degree of anaerobic metabolism

Table 1
Mortality, mean, and median systolic blood pressure stratified by base-deficit groups

Base deficit		N	Systolic blood pressure, mm Hg		Mortality, %
Group	Value		Mean ± SD	Median (25%–75%)	
1	10.0 to 0.0	42,198	137 ± 29	137 (120–153)	6
2	−.1 to −5.0	47,124	134 ± 28	133 (118–150)	7
3	−5.1 to −10.0	18,313	123 ± 31	124 (104–142)	15
4	−10.1 to −15.0	5131	111 ± 38	111 (90–135)	33
5	−15.1 to −20.0	1907	98 ± 47	100 (76–129)	52
6	−20.1 to −25.0	811	84 ± 54	90 (53–120)	65
7	−25.1 to −30.0	346	71 ± 55	78 (0–112)	66

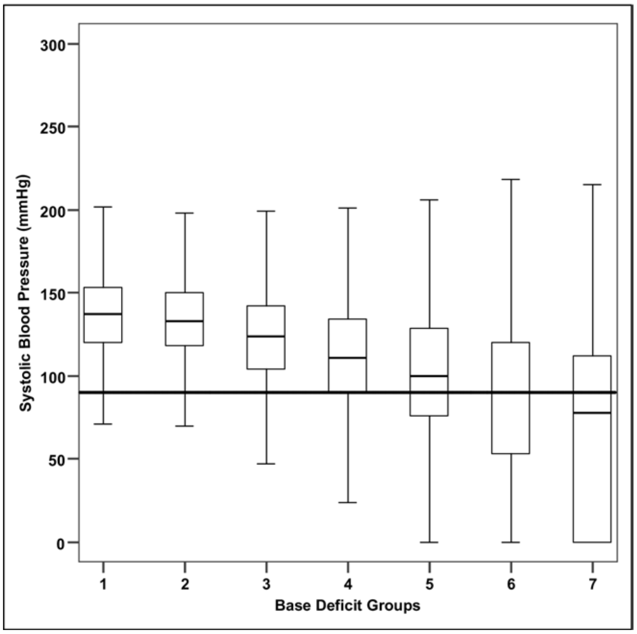

Fig. 1. This box-and-whiskers plot shows the median and interquartiles of SBP by base-deficit groups. Boxes represent the interquartile range depicted as the difference between the 75th and 25th percentiles. The line within the box represents the median. The reference line is at a systolic value of 90.

that must be present before systemic hypotension is likely to occur [1,3].

SBP is probably the most commonly used vital sign for the triage of injured patients. A SBP of less than 90 mm Hg is accepted widely as an indicator of the need for emergent interventions [17,18]. For example, the revised trauma score does not assign points for SBP status until the value decreases to less than 90 mm Hg [19]. Several practice guidelines such as prehospital Emergency Medical Service and trauma activation protocols use systemic hypotension as an indicator of hemodynamic instability. However, although our data suggest that the presence of hypotension is an ominous prognostic sign that requires a rapid response, in many patients, a significant degree of shock may be present long before the SBP decreases to less than 90 mm Hg.

As a corollary, SBP measurements higher than 90 mm Hg do not reliably indicate hemodynamic stability. These findings can be explained at a physiologic level. In the event of hypovolemia resulting from hemorrhage, physiologic compensatory mechanisms such as increased systemic vascular resistance and tachycardia are invoked to maintain perfusion of the most vital organs at the expense of causing ischemia in others [9]. These intrinsic compensatory mechanisms may maintain blood pressure within the normal range, despite significant hemorrhage resulting in severe anaerobic metabolism. This is particularly true in trauma patients because most of them are young, otherwise healthy individuals who are able to maintain a normal blood pressure until volume loss and concomitant reduction in organ perfusion has become severe [20]. However, with continuing hemorrhage, these homeostatic mechanisms begin to fail and blood pressure decreases. If the depth and duration of hypotension has been severe, the shock state may become refractory to treatment, with only transient improvements in blood pressure noted despite resuscitative efforts [21].

The most important implication of our findings is that using systemic hypotension as an indicator of adequate circulation may delay urgently needed diagnostic and therapeutic interventions until the complications become severe, or shock becomes refractory to treatment. The feasibility and reliability of alternative means of detecting early stages of shock, such as near-infrared spectroscopy, transcutaneous oxygen levels, and heart rate variability currently are being explored, but have not yet been validated clinically [22–25].

The study has a few potential limitations. The most important limitation was that base deficit was reported in only 10% of patients in the database, resulting in a large number of exclusions. This suggests that base deficit was measured selectively, probably in patients who were more severely injured, and less likely to have occult hypoperfusion. This should not significantly compromise the study because if blood pressure was not a good predictor of shock in more severely injured patients, it is unlikely to have better clinical usefulness in less severely injured ones. This study also was limited by the fact that only a single blood pressure measurement was available in the database, and not serial measurements. Early aggressive treatment of shock may improve perfusion rapidly, even though base deficit remains abnormal. However, our objective was to measure the relationship between blood pressure and shock, and this study clearly shows that initial blood pressure is a random predictor of patients who have ongoing or recent significant systemic hypoperfusion. This objective could be achieved adequately by looking at a single point in time, and does not necessarily require serial measurements. Another potential limitation of our data is that the exact time of measurement of blood pressure and base deficit are not known, although both were obtained in the emergency department. A significant delay between the time the blood pressure was measured and the time base deficit was obtained may alter the relationship between the two. Finally, we used base deficit as a measure of shock because several prior studies have shown that it has an excellent correlation with hemorrhagic shock and outcome [11]. However, base deficit may be altered by factors such as hypoventilation, poisoning, alcohol intoxication, and other causes of systemic acidosis.

In conclusion, despite these limitations, our study validates the current ATLS principle that systemic hypotension is a late marker of shock, and should not be depended on as a sole criterion for classifying patients as stable or unstable. Additional information, such as global measurements of perfusion and signs of adequate end-organ perfusion, is needed before patients can be classified as being stable. Hence, a normal blood pressure should not deter aggressive evaluation and resuscitation of trauma patients. Other, more-sensitive, measures of shock are needed that can be used at the bedside noninvasively, rapidly, and repeatedly.

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Discussion

Frederick Moore, M.D. (Houston, TX): Failure to recognize the severity of shock is a common, sometimes fatal, mistake in the initial evaluation of trauma patients with potentially life-threatening injuries. Most seasoned clinicians have learned this lesson the hard way. Therefore, it has become a basic tenet in ATLS that systemic hypotension is not an early sensitive indicator of shock. In the current study, the authors have used the very large NTDB of the American College of Surgeons to test the hypothesis that systemic hypotension is a late marker of shock. They have chosen Emergency Department (ED) base deficit (BD) to be their index of the severity of shock. They first show, as others have previously shown, that ED BD is an independent predictor of mortality. Using admission SBP in the ED, they next show that systemic hypotension (defined by the cut-point of SBP <90 mm Hg) does not correlate well with worsening BD. One study limitation that the authors acknowledge is that BD was reported in only 10% of database patients. In the discussion, they argue that this should not significantly compromise the study because if blood pressure is not a good predictor of shock in more severely injured patients, it is unlikely to have better clinical utility in less severely injured ones. However, I would like to caution the authors. They are using BD as their surrogate for shock. BDs were most likely obtained because the bedside clinician felt the patients were high risk and, therefore, BD is more likely to be predictive of mortality. If the same data were available in all NTDB patients (ie, admission SBP, ED BD, and mortality), the results of this analysis would be much different. Another potential limitation (not acknowledged) is that many trauma centers use automated blood pressure machines. At the 2003 Western Trauma Association meeting, Jim Davis and associates from Fresno reported that automated blood pressure (BP) determinations were consistently higher than manual-cuff BP, particularly in hypotensive trauma patients. Based on this report, we now mandate that a manual-cuff blood pressure be obtained at the beginning of all major trauma evaluations. I was quite surprised how resistant our ED nurses were to the implementation of this as a standard of care. I suspect the use of automated BP machines is common practice in other trauma centers and thus admission ED SBP data obtained from NTDB is not as accurate as one might assume. I have 3 questions for the authors:

1. While you report that BD is an independent predictor of mortality, you do not present the results of your logistic regression model. How did the odds ratios for BD compare to that of the other predictors? What was the area under the ROC curve for BD predicting mortality?
2. Your basic tenet is that if clinicians solely rely upon admission ED SBP to determine the severity of shock, then unnecessary delays in the evaluations in treatment will occur. In reality, the clinicians use other information that is readily available. One factor is

age. In logistic regression, you identified that age was an independent predictor of mortality. What effect does age have on the ability of SBP to predict base deficit? I suspect that the optimal cut-point for SBP prediction is age dependent.

3. Several years ago at this forum, David Partrick from Denver reported that systemic hypotension was common in children with severe head injury who had no evidence of significant hemorrhage. The following year, Walt Biffl, again from Denver, reported the same phenomenon in adult trauma patients. Forty percent of your study patients had head injuries (mean GCS = 12 ± .5). The patients you classified as being in shock had a significantly lower GCS of 9 ± .5. Were these GCS measured when the patient was in shock or were they obtained postresuscitation? Do you think that GCS is a confounding variable in this dataset and, if so, how did you control for it?

Ron Sing, M.D. (Charlotte, NC): We are concerned about the accuracy of base deficit. We have had problems with base deficit as the original base deficit isn't always very negative and then, 30 minutes later, it is −10. For your practice, what are you doing to evaluate patients for shock? What clinical or laboratory evaluations are you using to make decisions on your patient resuscitations?

Jennifer Parks, M.P.H.: In response to Dr. Moore's first question, the odds ratios for our stratified base deficit levels in the regression model were comparable to other variables in the model such as GCS, age, ISS, and first systolic blood pressure in only the less-severe base-deficit levels. Once base deficit reached −10 or worse, the odds ratios corresponding with that level of derangement were much more impressive. The area under ROC curve was not calculated. In response to Dr. Moore's second question, we did not examine the interaction between systolic blood pressure and age. Some preliminary analysis of our data did not reveal any significant differences in optimal cut-points for SBP by age. There may be other modifiers as well, such as gender, comorbidities, mechanism of injury, etc. We are looking at the data in more detail and plan to report it in a subsequent study. We utilized the first recorded GCS in the emergency department. We did not study the relationship between head injury and shock. The potential confounding due to GCS on mortality was controlled for through the use of regression modeling. Finally, in response to Dr. Sing's question, NTDB only records the first base deficit obtained in the emergency department. The results of this study suggest that relying on systemic hypotension alone is inadequate. We use a combination of traditional markers of shock to monitor response to resuscitation, including heart rate, blood pressure, base deficit, lactate levels, and invasive monitors.