Hypotension Begins at 110 mm Hg: Redefining "Hypotension" With Data

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Background: **Clinicians routinely refer to hypotension as a systolic blood pressure (SBP) ≤90 mm Hg. However, few data exist to support the rigid adherence to this arbitrary cutoff. We hypothesized that the physiologic hypoperfusion and mortality outcomes classically associated with hypotension were manifest at higher SBPs.**

Methods: **A total of 870,634 patient records from the National Trauma Data Bank with emergency department SBP and mortality data were analyzed. Patients (140,898) with severe head injuries, a Glasgow Coma Score ≤8, and base deficit (BD) <5, or missing data items were excluded from analysis. Admission BD, as a measure of metabolic hypoperfusion, was evaluated in 81,134 patients and mortality was plotted against SBP.**

Results: **Baseline mortality was <2.5%. However, at 110 mm Hg, the slope of the mortality curve increased such that mortality was 4.8% greater for every 10-mm Hg decrement in SBP. This effect was consistent to a maximum of 26% mortality at a SBP of 60 mm Hg. Hypoperfusion (change in the slope of BD curve) began to increase above baseline of 4.5 at a SBP 118 mm Hg.**

Conclusion: **Taking the BD and mortality measurements together, this analysis shows that a SBP ≤110 mm Hg is a more clinically relevant definition of hypotension and hypoperfusion than is 90 mm Hg. This analysis will also be useful for developing appropriately powered studies of hemorrhagic shock.**

Key Words: **Hypotension, Shock, Trauma, Base deficit, Systolic blood pressure, Mortality.**

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Physicians and surgeons have sought to understand shock and define its physiologic implications since the term was first coined by LeDran in 1743 from his reflection of experiences treating gunshot wounds.[1] In 1872, Gross defined the shock state as "a manifestation of the rude unhinging of the mechanism of life".[2] By the 20th century, there was already an evolving body of medical literature that characterized the hemodynamic responses to hemorrhage. In 1910, Dr. Yandell Henderson formulated the concept of the venopressor mechanism stating "blood pressure is, so to speak, the fulcrum of the circulation . . . . . shock, as surgeons use the word, is due to failure of the fulcrum".[2] In concurrence, through their investigative efforts, Wiggers, Blalock, Cannon, and Scudder all independently stressed that hypotension was the harbinger of shock.[1] Clinical studies from this era and subsequent eras referred to hypotension as systolic blood pressures (SBPs) in the range of 60 mm Hg to 90 mm Hg.[3–6]

Blood pressure and heart rate continue to serve as steadfast indicators for early diagnostic and therapeutic decision making after injury. Current standards typically use SBP as one of the measured values to determine the patient injury severity and as a triage tool. Although hypotension in trauma patients is often defined as an SBP ≤90 mm Hg, little data exist to support the dogmatic adherence to this arbitrary value. Current trauma triage relies on abnormal physiologic criteria to determine a patient's mode of transport, priority of treatment, destination for treatment, injury severity, mortality, and need for possible life-saving interventions. However, the physiologic portion of most of these existing triage tools is based on the presence of abnormal vital signs in the patient, particularly an SBP ≤90 mm Hg. Common vital signs are used because these measurements are usually readily obtainable at the site of injury and therefore may provide a "snapshot" of patient stability. However, such an assumption is problematic because the physiology of the trauma patients suffering from severe hemorrhage is often dynamic and may not reflect the true degree of hypoperfusion present because of normal physiologic compensatory mechanisms. Furthermore, previous literature has shown that significant hypoperfusion occurs in hypovolemic laboratory experiments as well as in blunt and penetrating patients with trauma despite normal, standard vital signs especially in young, healthy patients.[7,8]

Compensatory mechanisms allow significant reductions in central circulating blood volume, stroke volume, and cardiac output to occur well before changes in arterial blood

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pressure. Such physiologic compensations can thus mask the true nature and severity of many traumatic injuries, leading to under appreciation of the severity of injury, under triage, and increased mortality. In fact, a SBP of 90 mm Hg signifies impending cardiovascular collapse and underscores the necessity for improved indicators to improve triage and initiate early intervention strategies to improve postinjury mortality.[9] Although an initial SBP ≤90 mm Hg has been previously shown to provide higher sensitivity and better specificity for indication of mortality and outcome than most traditional vital signs (respiratory rate, heart rate, etc.),[10–14] more sensitive markers of acute hypoperfusion are needed if hemorrhage and circulatory shock are to be recognized in their early stages when lifesaving interventions are most likely to be successful. Thus, the current process and practice of prehospital and early hospital trauma triage and treatment may be significantly improved by providing a more liberal definition of hypotension, which may provide an earlier and better indicator of blood volume loss and impending circulatory collapse. For this reason, we hypothesized that physiologic hypoperfusion and mortality outcomes classically associated with hypotension would be manifested at SBPs >90 mm Hg.

PATIENTS AND METHODS

A retrospective review of patient records from the National Trauma Data Bank (NTDB) version 5.0 were analyzed for this study. Records consist of patient trauma registry data collected from trauma centers in the United States and Puerto Rico. Patient records used for analysis are distributed across multiple record files with common record identifiers. Data items for analysis were aggregated from the NTDB data files containing demographics, emergency department, complications, and outcome records. A total of 870,634 patient records from the NTDB with emergency department SBP and mortality data were analyzed. To exclude severe head injury and traumatic brain injury (TBI), patients with a total Glasgow Coma Score ≤8 and base deficit (BD) <5 were used as exclusion factors. A total of 140,898 patients were excluded by this mechanism for the subsequent investigation.

Analysis was performed on SBP relative to overall mortality, mortality excluding brain injury, number of complications, number of ventilator days, number of intensive care unit (ICU) days, total length of hospital stay, age, and gender. Summary data were plotted and filtered using window average filters to decimate peaks in the resulting dataset. Inflection points in the filtered dataset were determined by calculating the data slope for each point in the filtered data set. Median or average line slope estimation filters were used to generate a filtered slope set for each variable. All filters were executed using a moving window across values in the resulting dataset to generate the filtered results. A five-point moving window was used for generation of filtered datasets for all variable summaries with SBP as the independent variable. Analysis of age was performed by using a double moving average window with an overlapping range of 3 years. Point of inflection in the final filtered data set was determined by the point at which the filtered slope values maintain a positive slope and do not cross the zero line.

Receiver operator characteristic (ROC) curves were generated to analyze the power of association between the SBP and mortality. ROC values were analyzed to generate the optimal SBP that maximizes both the sensitivity and specificity of the ROC curve.

RESULTS

The overall dataset including TBI had a baseline mortality <5%. Figure 1 shows the plot of SBP compared with mortality and BD for the overall dataset. This dataset had the initial SBP inflection point at approximately 115 mm Hg. Slope of the line below SBP 115 mm Hg had an increase of approximately 6% in mortality for every 10 mm Hg, with a maximum of 40% mortality at SBP 60 mm Hg.

Excluding TBI, patients in this analysis had a baseline mortality of <2.5%. Figure 2 shows SBP compared with mortality and BD. At 110 mm Hg (lower arrow), the slope of the curve increased such that there was a 4.8% increase in mortality for every 10 mm Hg decrement in SBP. This effect was consistent to a maximum of 26% mortality at a SBP 60 mm Hg. Hypoperfusion, indicated by a change in the slope of BD curve, began to increase above baseline of 4.5 at a SBP 118 mm Hg (Fig. 2, upper arrow).

Complications during hospital stay demonstrated the same pattern as mortality. Baseline average rate for complications was approximately 0.05 complications per patient (5

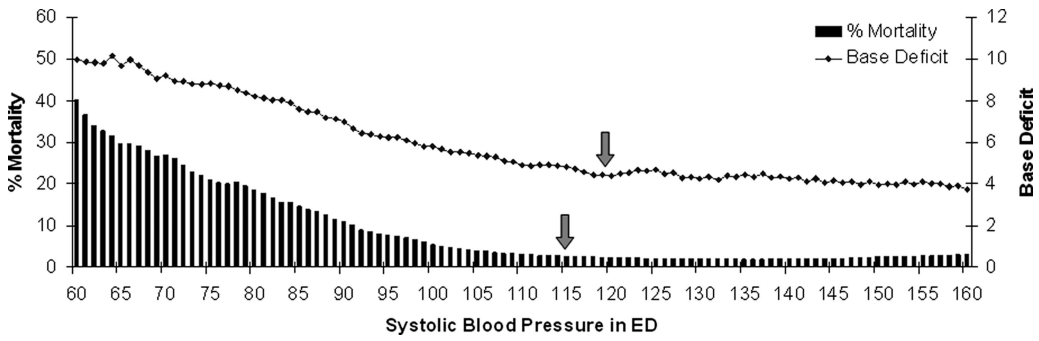

Fig. 1. *SBP analysis (including brain injuries).*

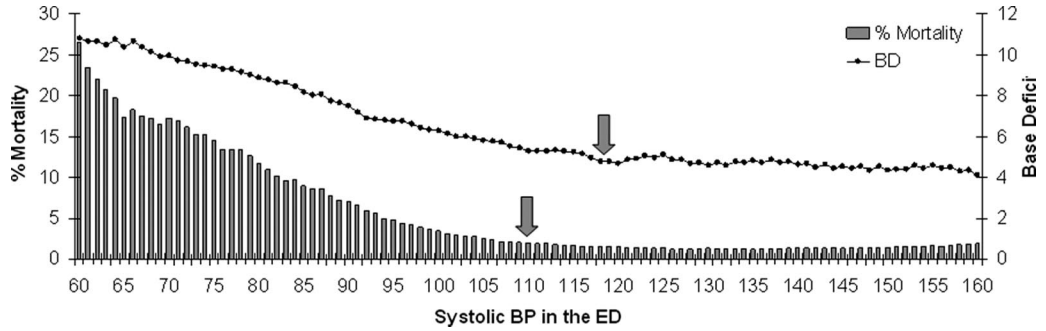

Fig. 2. *SBP analysis (excluding traumatic brain injury).*

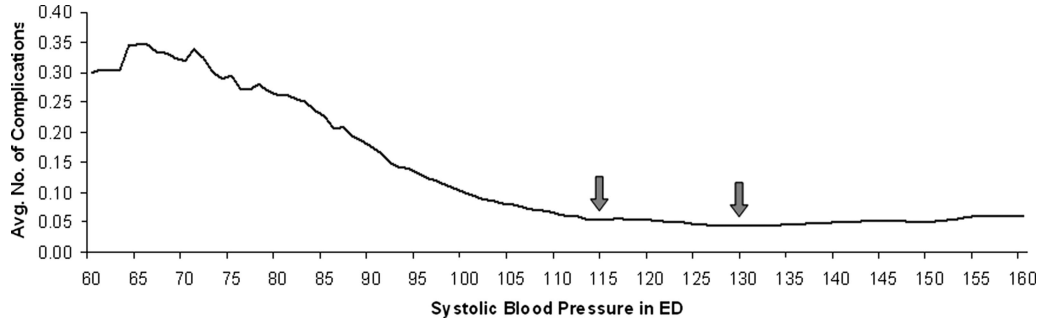

Fig. 3. *SBP analysis for complications.*

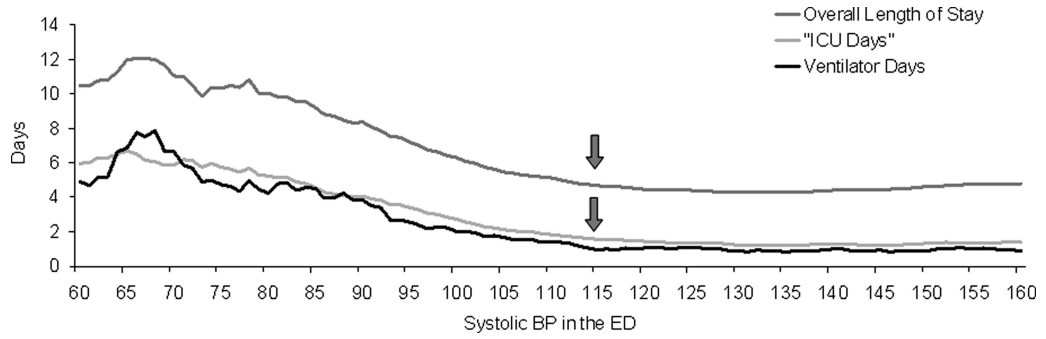

Fig. 4. *SBP analysis for overall length of stay, ICU days, and ventilator days.*

complications per 100 patients analyzed) above SBP 130 mm Hg (Fig. 3). Analysis revealed a SBP inflection point at approximately 115 mm Hg characterized by a continuous increase up to a mean of 0.35 complications per patient at a SBP of 65 mm Hg. Complications rose at a rate of 6.7% for every 10 mm Hg decrement in SBP.

Analysis for length of stay (LOS) (number of ICU days) and number of ventilator days revealed a common point of interest at a SBP of 116 mm Hg (Fig. 4). Baseline LOS was on average 4.4 days for SBP >116 mm Hg, increasing to an average of 12 days for SBP of 65 mm Hg. Rate of increase for LOS was on average 1.6 days for every drop of 10 mm Hg below SBP 116 mm Hg. Baseline for number of ICU days was on average 1.3 days for SBP >116 mm Hg, with an average increase of 6.7 days with a SBP of 65 mm Hg. The increase rate for ICU days had an average of 1.1 days for every drop of 10 mm Hg in SBP. Average number of ventilator days was approximately 1.0 for SBP >116 mm Hg. This increased to a maximum of 7.9 for SBP 68 mm Hg, with a mean rate of increase of 1.2 days per 10 mm Hg drop in SBP.

Age analysis consisted of analyzing the mortality rates to determine a cutoff at which mortality starts to increase relative to age. Figure 5A shows the mean mortality graph versus age. Analysis of these data revealed a threshold of 43 years, at which mortality continuously increased relative to age. For patients <43 years of age, the point of interest was found to be at an SBP of 108 mm Hg. However, for patients ≥43 years, this point was moved to an SBP of approximately 117 mm Hg. Average difference in mortality at SBP 108 mm Hg was found to be 3.2% higher for patients ≥43 years. Figure 5B shows the SBP plots for the two age groups with the associated points of interest.

Gender analysis showed only a slight difference between male and female groups at the baseline values above SBP of 108 mm Hg for men and 110 mm Hg for women. Figure 6

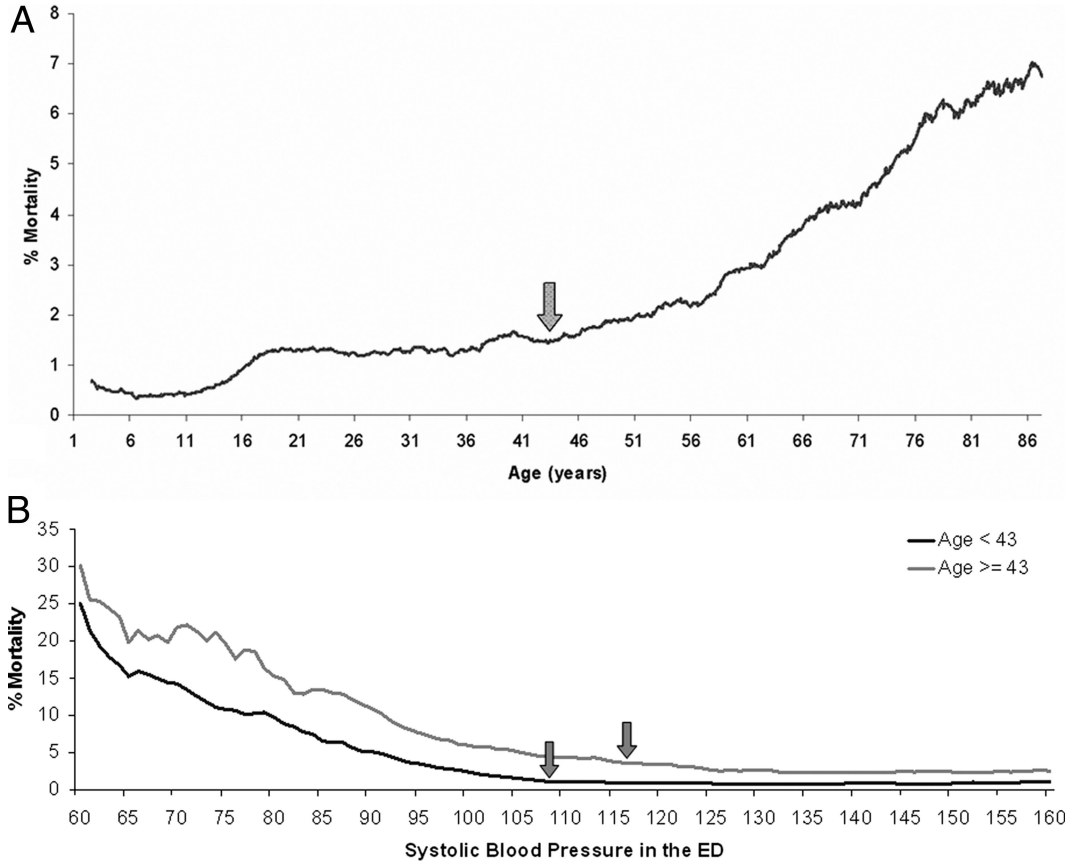

Fig. 5. (A) *Age analysis of mortality.* (B) *SBP analysis by age cutoff.*

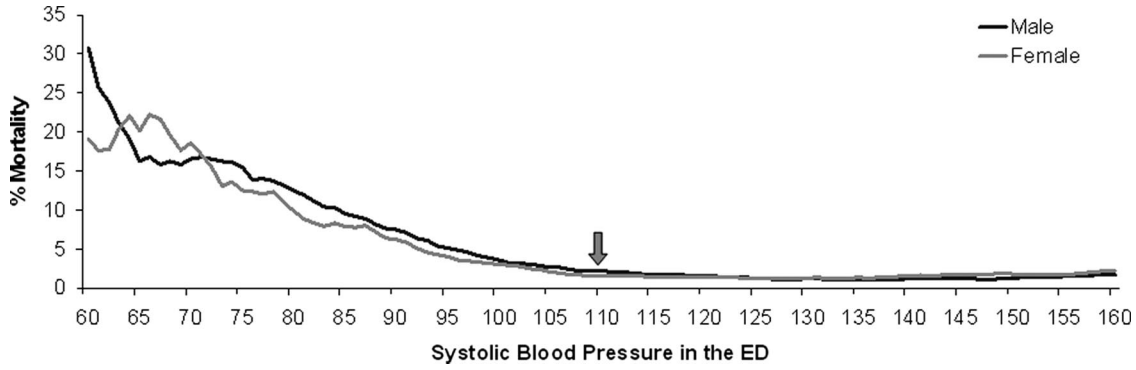

Fig. 6. *SBP analysis for gender.*

shows the mortality graph for both sets. Baseline mortality had a mean of 2.2% for the male group versus 1.7% for the female group. In men, the increase in mortality rate was 4.6% per 10 mm Hg decrease in SBP. For the female group, the inflection point for increased mortality was found to be at an SBP of 110 mm Hg with the rate of mortality increasing 4.4% per 10 mm Hg incremental decrease in SBP.

Analysis for the association of SBP with mortality (Fig. 7) resulted in an ROC area of 0.582 (95% CI, 0.577–0.588; $p < 0.001$). The maximum combination of sensitivity and specificity was found at an SBP of 123 mm Hg with a sensitivity of 0.47 and specificity of 0.697. In addition, an analysis of sensitivity, specificity, and positive predictive value was performed for the individual values of 90 mm Hg and 110 mm Hg (Table 1).

Evaluation of the dataset with respect to BD and its association with mortality inclusive and exclusive of brain injury revealed an initial inflection point at BD of 4 (Fig. 8).

DISCUSSION

Health care providers define shock by the level of blood pressure and the presence of nonspecific signs and subjective symptoms such as weak pulse character, low blood pressure, cyanosis, decreased capillary refill, restlessness, and an al-

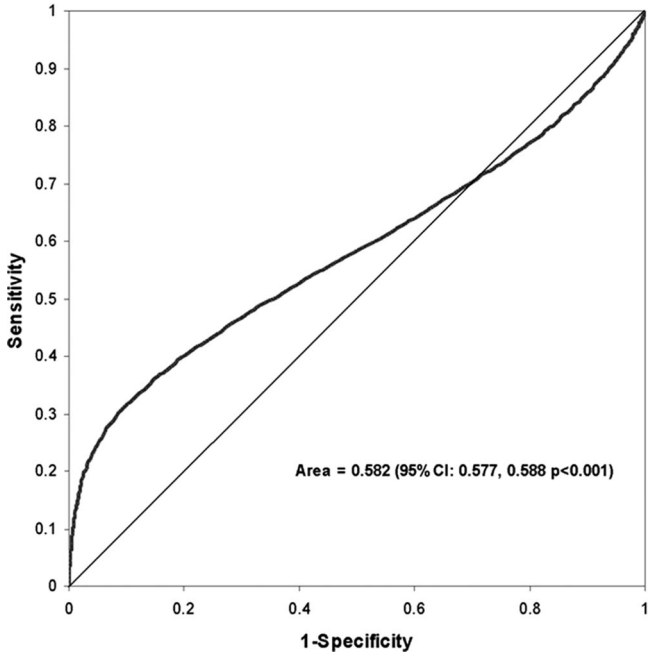

Fig. 7. *ROC curve for the association between SBP and mortality.*

Table 1 **SPB 90 mm Hg Versus 110 mm Hg**

	SBP <90 mm Hg	SBP <110 mm Hg
Sensitivity (%)	18.7	33.7
Specificity (%)	97.6	87.5
Positive predictive value (%)	13.7	5.2

tered level of consciousness. Furthermore, successful treatment with the reestablishment of these signs and symptoms to a preconceived baseline value was used to guide resuscitation and therapeutic endpoints. Unfortunately, these signs and symptoms are imprecise, subjective, and observer dependent and do not always reflect the true underlying physiologic process, compensatory reserve, and degree of hypoperfusion present. Thus, the appearance of the classical cut point of 90 mm Hg (hypotension) or other signs and symptoms of hypoperfusion and shock do not mark the beginning of circulatory failure, but rather represent a clinical manifestation of initial physiologic decompensation.[15,16]

Previous literature has shown that blood pressure measurements early in the course of shock are not well correlated with blood flow or cardiac output and that significant hypoperfusion occurs in hypovolemic laboratory experiments as well as in blunt and penetrating trauma patients despite normal, standard vital signs, especially in young, healthy patients.[7,8] Compensatory mechanisms allow significant reductions in central circulating blood volume, stroke volume, and cardiac output to occur well before changes in arterial blood pressure.[17] Such physiologic compensations can thus mask the true nature and severity of many traumatic injuries. In fact, this physiologic compensation is reflected in the lack of association between SBP and mortality as demonstrated by an area under the ROC curve of 0.58 for our cohort of patients. Similarly, the individual values of 90 mm Hg and 110 mm Hg demonstrate relatively weak sensitivity and specificity for mortality outcomes.

The association between hypoperfusion and poor outcomes in trauma patient is well described.[3,18] Our study corroborates the relationship between admission metabolic acidosis and patient survival. Because many of the markers of hypoperfusion are invasive or difficult to obtain in the prehospital or resuscitative environment of the emergency room, healthcare providers must currently make rapid decisions about priorities of triage and care, application of interventions, and transport destinations based upon isolated physiologic data points (e.g., arterial pressure, heart rate, and respiratory rate) without the benefit of observing dynamic trends inherent to trauma physiology. Based on our results, the current use of SBP ≤90 mm Hg as the marker for hypoperfusion in standard trauma triage criteria has little scientific evidence.

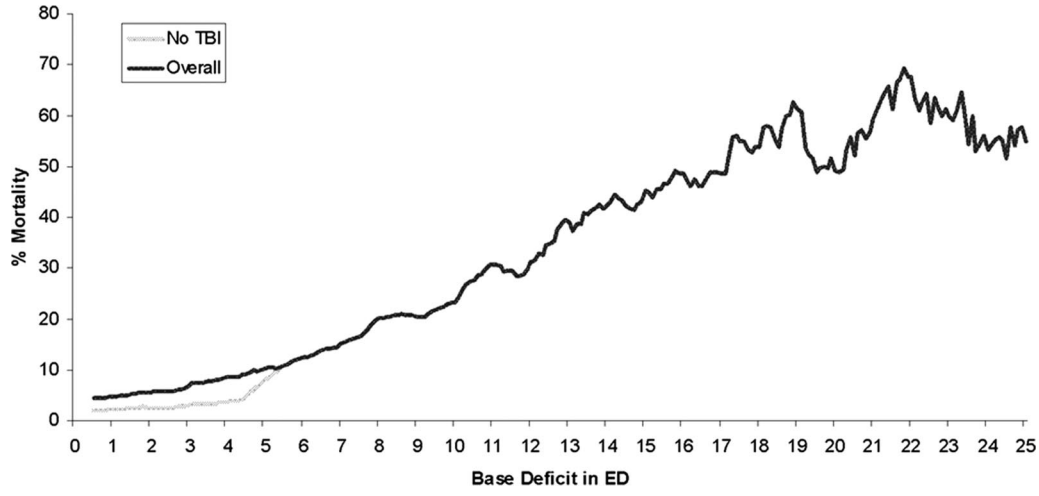

Fig. 8. *Mortality analysis for base deficit.*

Our data suggest that an SBP of 110 mm Hg after injury is associated with the onset of profound physiologic and outcome consequences. Interestingly, an SBP of 115 mm Hg appears to be associated with the inflection point of BD and corresponds to the inflection point at which complications, ICU days, and ventilator days increase. Our results suggest that using the current standard of 90 mm Hg to represent shock increases the risk for the insidious evolution of shock with attendant increases in injury-associated complications and the use of acute care resources. Our analysis suggests that this effect seems to be independent of both the age and gender of the patient.

In support of our contention, there have been a few series that have highlighted the lack of clinical sensitivity of 90 mm Hg.[19–21] In a study of 19,409 injured patients, Arbabi stratified patients by 30 mm Hg increments and demonstrated a baseline mortality of approximately 5% and a similar incremental rise in mortality. Interestingly, in the sample with an SBP 90 mm Hg to 119 mm Hg, patient mortality was 13.4%, consistent with the results of our current analysis.[19] In a hallmark study, Franklin and colleagues evaluated the impact of prehospital hypotension in a cohort of 299 injured patients with at least one documented episode of field or emergency department hypotension (SBP $<$90 mm Hg), as a valid indicator of trauma team activation. In this series, patients with field and ED hypotension had an emergent surgical indication in 79% of patients with a mortality of 44%. Patients who had only one field or ED episode of hypotension had lower rates of surgical indications and mortality.[20] Hypotension was validated as an important correlate of outcome, but the definition of hypotension was not questioned. To further validate the contention of our current study, a contemporary analysis performed by Edelman included 2,071 patients with injuries requiring exploratory laparotomy with gastric, small bowel, or diaphragm injuries. The baseline mortality of patients with SBP $\geq$110 mm Hg was less than 1%; however, mortality increased to 5% in the population with SBP between 90 mm Hg and 110 mm Hg. Concomitant with this increase in mortality were significant increases in LOS and infection.[22]

Our data provide an evidence-based approach for redefining the onset of circulatory shock based on an SBP of 110 mm Hg and subsequently providing earlier decision support for applying clinical interventions. However, our results cannot underscore the important concept that significant reductions in SBP at any time appear as late physiologic responses and consequently reflect an outcome rather than a predictor. This relationship is best supported by our observation that the threshold for tissue hypoperfusion (progressively increasing BD) appeared at an SBP $\geq$110 mm Hg (Fig. 1). It is therefore paramount that noninvasive measures of physiologic variables that track early changes in blood volume such as pulse pressure,[9,15] stroke volume,[9] and heart rate variability[15] be pursued for development of improved algorithms to support early decisions for triage and intervention.

Our study has notable limitations. The data used for our analyses were derived from the NTDB of the American College of Surgeons. By its nature, the NTDB has a number of inherent limitations, including its basis as a composite registry for numerous state, regional, and hospital databases. Deficiencies in these contributing entities are reflected in the overall database. By default, the NTDB is a convenience sample, and does not truly represent a population-based dataset because of the nature of facilities that contribute to the dataset. Other potential limitations of the database cited internally by the NTDB include data validation because of the size of the database, output issues associated with nonsystematic sampling, and selection and information bias.

We emphasize that this analysis is based upon the first SBP in the emergency department and thus the same threshold may not be applicable to the prehospital setting. Limitations of the NTDB dataset include lack of prehospital time and resuscitative fluid volumes. With this knowledge, the SBP measures obtained in the emergency department may be more valid measures of patient acuity because patients most likely had at least the initiation of resuscitative therapy. Another potential limitation of our study was blood pressure measurement itself. Many emergency departments, such as those who contribute to the NTDB, use automated blood pressure recording devices for utility and documentation. Previous investigations have demonstrated a propensity for automated devices to overestimate the true manual blood pressure by approximately 10 mm Hg.[23,24]

CONCLUSION

Our current analysis shows that an SBP $\leq$110 mm Hg is a more clinically relevant definition of hypotension and shock than is 90 mm Hg. We stress that the refined threshold SBP of 110 mm Hg is a diagnostic tool and not an endpoint of resuscitation. However, because of the overall lack of predictive value of SBP, this value cannot be used in isolation and highlights the need to develop treatment and outcome algorithms in which SBP will be an integral part. Because hemorrhage accounts for almost 50% of current combat fatalities, and up to 80% of civilian trauma fatalities, the development of new approaches for early detection of shock in trauma patients will continue to be a research priority. This analysis will be useful for developing appropriately powered studies of hemorrhagic shock in the future. In addition, our study will be important in the genesis of appropriate combinations of indices that yield increased sensitivity for shock to affect earlier postinjury intervention strategies and predict adverse outcomes.

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DISCUSSION

Dr. Glen A. Franklin (Louisville, Kentucky): Mr. Chair, Dr. Eastridge and colleagues have presented a different look at the definition hypotension.

Using the National Trauma Databank, they have examined the outcomes of over 700,000 trauma patients stratified by systolic blood pressure. And utilizing sophisticated statistical analyses, their data demonstrates an inflection point in the range of 110 millimeters of mercury for physiologic consequences of hypoperfusion.

The authors have openly disclosed the inherent error and bias associated with large administrative databases. The manuscript asks the question: why a systolic blood pressure of 90?

Who decided this was the cutoff for hypotension? And what data supports that practice? In fact, there is really little data to support this number, and likely it has been used for years more by tradition than by scientific merit.

I think there is really little to argue or discuss about their data and the manuscript itself, so I would like to focus my comments on the concept of resource utilization.

Do the authors have any data on the first systolic blood pressure taken in the field or other early parameters like heart rate or the presence of an endotracheal tube that would validate their results and would suggest earlier activation of the trauma team should be employed for management of these patients?

Although statistically significant due to the power of large data sets, have the authors compared the real clinical differences in that range between 90 and 110? And are there things we can do that we're not already doing that would impact the outcome of those patients?

Given the authors' military affiliation, I suspect they understand the concept of resource utilization quite well. The current recommendations for trauma team activation include a systolic blood pressure less than or equal to 90.

And, whether cleverly or cautiously, they have avoided this concept of a discussion of their manuscript. So I wanted to ask them, do they think we should now change our practice for trauma team activation to include a systolic blood pressure of 110 or less?

If so, do they have any data that would suggest we could intervene in those patients in some way differently than we already are that would improve their outcome? And have they made any recommendations to change existing military protocols for the triage of injured soldiers based on this very powerful dataset analysis?

Finally, with regard to their slide on age, I, too, like Dr. Eastridge, turn 43 in three weeks. And I am wondering should I have a medic alert bracelet made up that identifies to the EMS personnel that I should somehow be resuscitated differently than my younger cohorts?

This manuscript certainly has raised my level of suspicion for the patients between 90 and 110.

Dr. Brian J. Eastridge (Fort Sam Houston, Texas): With respect to Dr. Franklin's first question about early intervention in the field and early vital signs, we actually do have some data from an independent locally derived dataset.

We actually thought that this would be an important question to look at the analysis from the perspective of the pre-hospital environment because between the pre-hospital environment and admission, patients potentially have interventions, not the least of which is crystalloid resuscitation.

When we looked at a cohort of 7,300 patients from the Brooke Army Medical Center taken out of our registry over the last five years, we saw that there was a systolic blood pressure inflection point of approximately 107 to 108 millimeters of mercury.

So it's apparent that this actually is a valid indicator from the field also. Now what does this mean clinically? I think, realistically, because the systolic blood pressure is such an insensitive indicator, I think it's just a measure to have our residents sort of elevate their sphincter tone when they have patients that are in that 90 to 110 millimeter of mercury range.

With respect to changing clinical practice, I think, even with the inherent limitations of the National Trauma Databank and that type of database, this is pretty strong data so I would suggest that this is potentially a practice-changing type of analysis.

The other useful aspect is this 110 millimeters of mercury point, though we can't really do anything to specifically intervene, as it lowers that threshold for looking for issues for which we may need to intervene.

With respect to the last question of the age greater than 43, again, there are no particular resuscitative differences. I think it's a little distressing to now be in that category.

But I think the issue is, again, the higher index of suspicion. I think systolic pressure is an indicator in and of itself, or is it not a good indicator? I think it just highlights the need for better indicators. I think that SBP probably will be a useful adjunct in that analysis.

Dr. Steven R. Shackford (Burlington, Vermont): I would sort of like to turn the paper around a bit and look at it from the therapeutic side.

For years now we've been talking about hypotensive resuscitation and the importance of trying to maintain a blood pressure that would not "pop the clot", but at the same time maintain perfusion.

Years ago, we took this to the laboratory and created a model in a semi-awake pig, where you would injure the spleen, and the blood pressure would always drop before there was a great deal of blood loss. It seemed to us that hypotension, in fact, was protective and allowed the first stage of hemostasis to take place.

Could there be an implication of your data that we now ***must*** resuscitate our patients to 110? And what could be the downside of doing that?

Dr. Brian J. Eastridge: To be honest, I haven't thought about the issue from that standpoint.

From the standpoint you suggest, I could conceptually see that from a pre-hospital environment, we may not want to do that for the very reason of popping the clot.

However, when the patient is in the hospital environment where you have access to resources for definitive vascular control, then perhaps we should be potentially a bit more aggressive, if not at resuscitation, at least at looking for potential causes of shock. I should reiterate that the utility of our analysis is using SBP 110 mmHg as a diagnostic tool, not an endpoint of resuscitation.

So I think that's a great question that we should probably study in the future.

Dr. Kenneth L. Mattox (Houston, Texas): The year was 1982. Four individuals had missed a plane in San Diego. Their names were Frank Lewis, Howard Champion, Brent Eastman and myself.

On the back of a café napkin, we theorized on what is the level of blood pressure. Howard Champion reminded us of Bill Seiko's work in Baltimore, and Frank Lewis had just completed his computerized study.

Because the plane was very late, we finally settled on 90 - ninety as a pre-hospital EMS admitting level or in a laboratory level. I remind the group that your analysis was from the systolic blood pressure in the emergency department.

And even on this slide that you're showing on a field systolic blood pressure, I could maybe even make a major inflection point down around 85, although you have mathematically chosen it to be higher.

My purpose was to give you one origin of the 90, which was a best guest, and to raise the question if restricted fluid is good at 90 in the field, and now maybe 105–110 in the emergency department, if restricted fluid was good there, are you going to suggest that we not restrict fluid if we have this higher point?

In truth, I would suggest that we're describing the same cohort with measurement of the entry criteria at two different locations. Euro Laboratories, in San Antonio, Jill Sondeen has shown in the large animal a systolic of 80 is the point pre-resuscitation that popping the clot and shock is perhaps manifest.

So, my question is, are we merely describing this beast from two different angles?

Dr. Brian J. Eastridge: I think there is a very real potential for that. And, once again, I think that from a diagnosis and management standpoint, I think that the additional 20 degree window is really sort of a window that we and our residents should not be necessarily comfortable having our patients in.

I know that many in this room have probably stood in the trauma bay and looked at triple digit systolic blood pressures and said, "Okay, we're quasi-stable, move on with the resuscitation."

The one interesting thing is that we are talking about two levels of blood pressure. Clearly, the 90 is the point at which you pop your clot off. But I think the most important number is actually the base deficit number or the point at which you actually start to develop shock. Interestingly, even the 110 millimeters of mercury underestimates the advent or the initiation of shock.

Dr. George Kramer (Galveston, Texas): There are a few physiologists and surgeons that believe that perfusion and pressure may have been underestimated or risk of bleeding has been overestimated.

Regarding Dr. Mattox's comment about pigs and blood pressures, most of the blood pressures and much of the emphasis on this concept of limited resuscitation comes from anesthetized pigs and rats.

And even Dr. Mattox's patients are generally human and not anesthetized, so I think this data considers maybe those target end points should be suggested or reassessed.

My question is does this affect the military doctrine on hypotensive resuscitation or at least suggest that it should be looked at a little bit differently to see what the targets should be?

Dr. Brian J. Eastridge: Again, with the current operations in the world, we are very aggressive about getting this laboratory data out into the field.

We are starting to educate our hospital providers in theater. Right now, we're just talking about a recognition issue, recognizing that 90 to 110, we're still in a danger zone.

Dr. David P. Blake (Misawa, Japan): Obviously for me as well, this study does have some interesting implications. I do have a couple of questions for you.

First of all, did you sort out based on co-morbidities of these patients to see if there were other confounding factors that may have contributed to your outcome, even though it may not have changed the numbers remarkably?

Question number two is, did you sort out your data based on the injury, specifically thinking about things like spinal cord injury or burns or other reasons for hypotension – perhaps even a pneumothorax – as contributors to the hypotension which then, may not contribute to any of your outcome measures later on? I'm curious to know what you think about that.

Dr. Brian J. Eastridge: We did not specifically look at the confounding variable of co-morbidity.

When we looked at developing the definition of shock, there really isn't a good way from NTDB to ferret out those folks that would have potentially spinal, cardiogenic, tension pneumothoraxes, causes of shock. So we did not specifically isolate or exclude that subset. So that's another potential limitation, albeit likely small.