

Prehospital Hypotension in Blunt Trauma: Identifying the “Crump Factor”

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Background: Trauma activation for prehospital hypotension in blunt trauma is controversial. Some patients subsequently arrive at the trauma center normotensive, but they can still have life-threatening injuries. Admission base deficit (BD) ≤ -6 correlates with injury severity, transfusion requirement, and mortality. Can admission BD be used to discriminate those severely injured patients who arrive normotensive but “crump,” (i.e., become hypotensive again) in the Emergency Department? The purpose is to determine whether admission BD < -6 discriminates patients at risk for future bouts of unexpected hypotension during evaluation.

Methods: Retrospective chart review was performed on all blunt trauma admissions at a Level I trauma center from August 2002 through July 2007. Hypotension was defined as a systolic blood pressure ≤ 90 mm Hg. Patients who were hypotensive in the field but normotensive upon arrival in the emergency department (ED) were included. Age, gender, injury severity score, arterial blood gas analysis, results of focused abdominal sonogram for trauma (FAST), computed tomography, intravenous fluid administration, blood transfusions, and the presence of repeat bouts of hypotension were noted. Patients were stratified by BD ≤ -6 or ≥ -5 . Statistical analysis was performed using paired *t* test, χ^2 , and logistic regression analysis with significance attributed to $p < 0.05$.

Results: During the 5-year period, 231 blunt trauma patients had hypotension in the field with subsequent normotension on admission to the ED. Of these, 189 patients had admission BD data recorded. Patients with a BD ≤ -6 were significantly more likely to have repeat hypotension (78% vs. 30%, $p < 0.001$). Overall mortality was 13% (24 of 189), but patients with repeat hypotension had greater mortality (24% vs. 5%, $p < 0.003$).

Conclusion: Blunt trauma patients with repeat episodes of hypotension have significantly greater mortality. Patients with transient field hypotension and a BD ≤ -6 are more than twice as likely to have repeat hypotension (crump). This study reinforces the need for early arterial blood gases and trauma team involvement in the evaluation of these patients. Patients with BD ≤ -6 should have early invasive monitoring, liberal use of repeat FAST exams, and careful resuscitation before computed tomography scanning. Surgeons should have a low threshold for taking such patients to the operating room.

Key Words: Blunt trauma, Prehospital hypotension, Crump factor.

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Hypotension in the field after blunt trauma is itself a criterion for transport to a trauma center, but trauma team activation for this prehospital hypotension can be controversial.¹ Prehospital hypotension can be a predictor of mortality as well as the need for emergent therapeutic operation in trauma patients.^{2–5} Blunt trauma patients who remain hypotensive upon arrival to the emergency department (ED) are promptly and continuously evaluated. The patients who are transiently hypotensive in the field but normotensive upon arrival to the ED present a special problem in that they can have repeat hypotension or can “crump,” at an inopportune time, e.g., in the computed tomography (CT) scanner, radiology, etc. A means to discriminate those patients at risk for dangerous, repeat hypotension has not been identified.

One of the first signs of inadequate tissue perfusion and oxygen delivery is tissue acidosis.^{6,7} There are several markers that have been used for evaluating global perfusion and the degree of acidosis in trauma: serum pH, oxygen delivery, serum lactate, anion gap, and arterial base deficit (BD).^{6,8–15} Of these, BD has been useful in guiding trauma resuscitation and identifying patients with increased transfusion requirements, prolonged intensive care unit stay, and increased risk of shock-related complications.^{6,16,17} Morbidity and mortality significantly increase with a BD ≤ -6 in trauma patients.^{17–19}

We hypothesized that admission arterial BD can identify those blunt trauma patients who are transiently hypotensive in the field but normotensive on ED admission that are more prone to physiologic deterioration. The purpose of this article is to determine whether arterial BD can identify those who are at risk of “crumping.” Specifically, does a BD ≤ -6 portend a repeat bout of hypotension and its concomitant problems in this subset of blunt trauma patients?

METHODS

A retrospective chart review was performed on blunt trauma patients at Community Regional Medical Center (CRMC) in Fresno, CA, from August 2002 through July 2007. CRMC is a 550 bed, state designated, Level I trauma center in the center of California. Patients who were hypotensive in the field but normotensive upon arrival to the ED were included. Hypotension was defined as a systolic blood pressure (SBP) ≤ 90 mm Hg. Patients with continued hypotension upon arrival to the ED were excluded, as well as patients younger than 1 year. Age, gender, injury severity

score, arterial blood gas analysis, results of adjunctive studies (FAST, CT), intravenous (IV) fluids, blood transfusions, the presence of repeat bouts of hypotension, and mortality were noted. Patients were stratified by admission BD ≤ -6 or ≥ -5 . Statistical analysis was performed using paired *t* test, χ^2 , and logistic regression analysis with significance attributed to $p \leq 0.05$. Receiver operator curve (ROC) for BD was also used. This study was approved by the Institutional Review Board of Community Medical Centers and the University of California, San Francisco.

RESULTS

During the 5-year period, there were 9,973 trauma admissions, and 8,200 (82%) were from blunt mechanisms. Of these, 243 blunt trauma patients had hypotension in the field but were normotensive on admission to the ED, and 189 patients had admission BD data recorded.

Groups were stratified by serum base deficit: BD ≤ -6 and BD ≥ -5 . There was no significant difference in age, field SBP, field Glasgow Coma Scale, ED Glasgow Coma Scale, or intensive care unit admission rate between the two groups (Table 1). However, patients with a BD ≤ -6 had a significantly greater Injury Severity Score, IV fluid, and blood transfusion volumes administered in the ED. The difference in admission blood pressure between the two groups, although significantly different statistically, was not clinically notable.

Patients with a BD ≤ -6 were significantly more likely to have repeat hypotension, i.e., “crump,” during resuscitation. Of the 31 patients with BD ≤ -6 and repeat hypotension, 19 (61%) went emergently to the operating room. Seven of these patients had their repeat hypotension in the operating room (OR) while being prepared for surgery. Seventeen of the 19 patients required operative control of their hemorrhage: 10 laparotomies (with 1 concomitant thoracotomy), 1 sternotomy (for tamponade), 5 emergent orthopedic interventions, and 1 repair of a hemorrhagic scalp avulsion with a complex depressed skull fracture. The remaining two had emergent craniotomy as their primary operative procedure.

TABLE 1. Patient Characteristics

| | BD ≥ -5 | BD ≤ -6 | <i>p</i> |
|---|----------------|----------------|----------|
| N | 149 | 40 | — |
| Age (yr) | 35.6 \pm 1.5 | 33.0 \pm 2.7 | NS |
| SBP (field) mm Hg | 76 \pm 1 | 77 \pm 1 | NS |
| Glasgow Coma Scale (field) | 12 \pm 0 | 10 \pm 1 | NS |
| Glasgow Coma Scale (ED) | 12 \pm 0 | 11 \pm 1 | NS |
| Intensive care unit admission from ED (%) | 32 | 35 | NS |
| SBP (ED) mm Hg | 121 \pm 2 | 112 \pm 3 | 0.01 |
| Mean serum BD | -1.0 \pm 0.0 | -8.3 \pm 0.5 | <0.00001 |
| ISS | 20 \pm 1 | 31 \pm 3 | <0.0001 |
| IV fluid in ED (L) | 3.0 \pm 0.2 | 6.1 \pm 1.1 | <0.00001 |
| Blood transfusion in ED (units) | 1.7 \pm 0.4 | 5.0 \pm 1.0 | <0.001 |

NS, not significant.

TABLE 2. Patient Outcomes

| | BD ≥ -5 | BD ≤ -6 | <i>p</i> |
|-----------------------------|----------------|----------------|----------|
| N | 149 | 40 | — |
| Repeat hypotension (%) | 45 (30) | 31 (78) | <0.0001 |
| To OR from the ED | 40 (27)* | 20 (54)* | 0.003 |
| Ventilator days | 3.4 \pm 0.5 | 7.7 \pm 2.8 | 0.01 |
| Survivor length of stay (d) | 10.1 \pm 0.1 | 23.3 \pm 5.8 | 0.0002 |
| Deaths (%) | 14 (9) | 10 (25) | 0.01 |

* Of patients who did not die in ED or were not transferred.

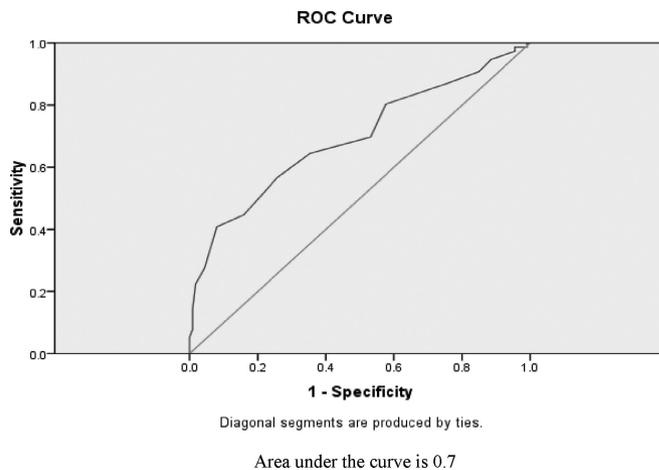


Figure 1. Receiver operator curve for BD-related to repeat hypotension.

The overall mortality was 13% (24 of 189). Patients with repeat hypotension had significantly greater mortality (24% vs. 5%, $p = 0.0003$; Table 2). Additionally, patients with BD ≤ -6 had more than a 2½-fold increase in mortality compared with those with BD ≥ -5 (25% vs. 9%, $p = 0.01$). Also, survivor length of stay, disposition to the operating room from the ED, and ventilator days were all significantly greater for the BD ≤ -6 patients (Table 2). Logistic regression analysis showed both repeat hypotension and BD ≤ -6 as significant, independent factors contributing to mortality (odds ratio, 5.5; $p < 0.01$ and odds ratio, 3.2; $p < 0.04$, respectively).

Figure 1 shows the ROC with regards to BD as a predictor for repeat hypotension. The area under the curve is 0.7. Table 3 shows the coordinates of that curve, with BD values ranging from -20.00 to +20.00. Comparison of BD ≤ -6 and BD ≥ -5 with regards to repeat hypotension shows BD ≤ -6 has a sensitivity and specificity of 41% and 92%, respectively. When BD ≥ -5 is used as a cutoff, sensitivity is 45% and specificity decreases to 84%. This trend concurs with the data in Table 3.

DISCUSSION

Prehospital hypotension (SBP ≤ 90 mm Hg) in trauma patients has been used as a criterion for trauma team activation and life-saving interventions.^{20,21} Indeed, systemic hypotension may not occur until the degree of shock is profound

TABLE 3. Coordinates of ROC Curve (Test Result Variables) for BD Related to Repeat Hypotension

| BD Values | Sensitivity | 1 – Specificity |
|--------------------|-------------|-----------------|
| Positive if \leq | | |
| -20.00 | 0.000 | 0.0000 |
| -16.50 | 0.013 | 0.0000 |
| -13.50 | 0.039 | 0.0000 |
| -12.00 | 0.053 | 0.0000 |
| -10.50 | 0.079 | 0.0009 |
| -9.50 | 0.118 | 0.0009 |
| -8.50 | 0.145 | 0.0009 |
| -7.50 | 0.224 | 0.0018 |
| -6.50 | 0.276 | 0.0044 |
| -5.50 | 0.408 | 0.0080 |
| -4.50 | 0.447 | 0.0159 |
| -3.50 | 0.566 | 0.0257 |
| -2.50 | 0.645 | 0.0354 |
| -1.50 | 0.697 | 0.0531 |
| -0.50 | 0.803 | 0.0575 |
| 0.50 | 0.868 | 0.0752 |
| 1.50 | 0.908 | 0.0850 |
| 2.50 | 0.947 | 0.0885 |
| 3.50 | 0.974 | 0.0956 |
| 4.50 | 0.987 | 0.0956 |
| 6.50 | 0.987 | 0.0991 |
| 13.50 | 1.000 | 0.0991 |
| 20.00 | 1.000 | 1.000 |

and is a late marker of posttraumatic hemorrhage.^{22,23} Parks et al.²² showed that correlation of ED SBP and severity of shock was poor. Mean SBP values did not decrease to <90 mm Hg until BD was worse than -20 mEq/L with a mortality exceeding 60%. Compensatory mechanisms can prevent a significant fall in blood pressure until up to 25% to 30% of blood volume is lost, especially in children.^{14,23} Children older than 1 year were included in our data since the average SBP is greater than 90 mm Hg starting at this age.^{4,24,25} Also, admission BD has been shown to reflect injury severity and predict mortality in children.²⁶

Blunt trauma patients who are transiently hypotensive (hypotensive in the field but normotensive upon arrival to the ED) have a mortality of 16%²¹ and pose a diagnostic dilemma. Not only can these patients have repeat hypotensive episodes at inopportune times (e.g., in the CT scanner), but repeat hypotension increases their mortality to 27%.²¹

Posttraumatic hypotension can be a significant indicator of major injury requiring intervention.^{2,3,19–22,27–30} Although there have been studies challenging the standing protocol for trauma team activation for out-of-hospital hypotension,³¹ the fact that patients who are transiently hypotensive in the field can indeed become hypotensive again in the ED gives credibility to the initial field evaluation by Emergency Medical Services personnel. Field hypotension is a valid criterion for trauma team activation and may even need to be expanded to prevent undertriage in the field.³² The American College of Surgeons Committee on Trauma guidelines states that effective trauma systems should decrease

undertriage because undertriage may result in preventable mortality or morbidity from delays in definitive care.¹ Several physiologic variables have been identified to be independent predictors of injury severity and the need for emergent intervention: altered mental status, altered respiratory status, and, of course, SBP <90 mm Hg.^{1,32}

Preoperative time spent in the ED in patients with intra-abdominal hemorrhage adds to mortality.³³ Clarke et al.³³ showed that trauma patients who were hypotensive in the ED had increased mortality as time in the ED increased. In the case of patients with transient prehospital hypotension, waiting until these patients become hypotensive again in the ED puts both the patient and the trauma team at a dangerous disadvantage. Prevention of this predicament requires costly, overaggressive monitoring, overtriage and resuscitation or an objective identifier to sort out those patients at higher risk for acute physiologic deterioration (crumpling). It is notable that of the 31 patients with BD ≤ -6 with repeat hypotension, 19 (61%) had urgent operative intervention. Seven of these 19 patients (37%) had their repeat hypotensive episode while being prepared for surgery in the OR. None of these seven patients died.

Of the 40 patients with a BD ≤ -6 and hypotension in the field, 7 had a positive FAST examination in the ED (Table 4). The remainder had either a negative FAST or no FAST examination performed at all. One of the seven sonograms was false-positive with a subsequent negative abdominal CT. Of the remaining six, four went to the OR for definitive procedures related to the positive FAST, including sternotomy for positive pericardial fluid and subsequent tamponade. Two went to the intensive care unit with isolated liver lacerations (as well as severe traumatic brain injury). All seven of these patients had repeat hypotension.

This study has the limitation of a retrospective review. A possible area of debate is that prehospital blood pressures can be inaccurate.^{21,30,34} All field blood pressures in our trauma system are performed manually, as are initial BP's in the ED. Manual blood pressure measurements have been shown to be more accurate than automated measurements in hypotensive trauma patients.³⁵

TABLE 4. Patients With BD ≤ -6 and Positive FAST in ED

| | Injury | Operation/Disposition |
|------------|--|---------------------------------------|
| Patient 1 | Complex liver laceration, small bowel rupture | Hepatorrhaphy, bowel resection |
| Patient 2 | Splenic laceration | Splenectomy |
| Patient 3* | Tamponade, right atrial rupture | Sternotomy, cardiorrhaphy |
| Patient 4† | Retrohepatic caval injury, abdominal compartment syndrome | Laparotomy, hepatectomy, caval repair |
| Patient 5 | Grade 2 liver laceration | Intensive care unit |
| Patient 6 | Grade 3 liver laceration | Intensive care unit |
| Patient 7‡ | Acetabular fracture, comminuted femur fracture, pulmonary contusions | Intensive care unit |

* FAST with pericardial fluid.

† Initial FAST negative.

‡ Negative abdominal computed tomography.

Two of the 19 repeat hypotensive patients with $BD \leq -6$ who went from the ED to the OR had craniotomy as their operative interventions. Both had emergent evacuation of subdural hematomas. Although these two patients had no hemorrhagic source of hypotension, isolated brain injury has been associated with posttraumatic hypotension.^{36,37}

Twelve of the 31 repeat hypotensive patients who had a $BD \leq -6$ did not go immediately to the OR. Eight of the 12 had traumatic brain injury in addition to multisystem trauma. Four of 12 had liver lacerations. Most had pulmonary contusions, rib fractures as well as various orthopedic injuries. There were no interventional angioembolizations performed for pelvic or solid organ injuries in this group.

An area of concern is the effect of initial resuscitation fluids on serum BD. Brill et al.³⁸ demonstrated that iatrogenic, nonanion gap, hyperchloremic acidosis from crystalloid infusion can cause a secondary BD that does not predict mortality. This study, however, examined a mixture of surgical intensive care unit patients, not blunt trauma patients in the initial stages of resuscitation as our study does. Furthermore, a prospective, observational study by Sinert et al.³⁹ concluded that BD was able to distinguish minor from major injury after 4 hours of resuscitation, irrespective of the volume of normal saline infused.

Obviously, patients with transient field hypotension and a positive FAST can have solid organ injury that responds to resuscitation. Peitzman et al. demonstrated that nonoperative management of solid organ injuries can be successful in almost 90% of patients. However, attempts at nonoperative management in hemodynamically unstable patients can contribute to significant mortality.⁴⁰ Furthermore, up to 85% of severely injured trauma patients can still have evidence of ongoing, inadequate tissue perfusion despite normalization of pulse and blood pressure.⁴¹ In addition, the use of a biochemical marker (e.g., BD) to monitor shock and resuscitation has been recommended as part of *Clinical Practice Guideline: Endpoints of Resuscitation* by Tisherman et al.⁴¹ Again, $BD \leq -6$ seems to be an important indicator of major injury as demonstrated in previous studies.^{16–18,27,41,42} Using BD as a biochemical marker of potential hemodynamic instability in patients with a positive FAST and transient field hypotension is a logical extrapolation of previous studies by Pietzman, Rutherford, Davis, and others.^{16–18,27,40–42} Although $BD \leq -6$ in this study was not very sensitive in its association with repeat bouts of hypotension, it was very specific (92%). It is noteworthy that 30% of the patients with transient field hypotension and $BD \geq -5$ still had repeat hypotension, and this concurs with the low sensitivity but high specificity of $BD \leq -6$ and its association with significant injury seen previously.⁴²

This study supports arterial $BD \leq -6$ as a useful “crump factor” in helping to discriminate which patients with transient hypotension are at risk for repeat hypotension and its associated complications. Patients with $BD \leq -6$ were more than 2½ times more likely to have repeat hypotension during resuscitation. The prehospital hypotensive patients who developed repeat hypotension in the ED increased their mortality almost 5-fold. Our data support several current

practices in the management of blunt trauma patients. First, prehospital hypotension merits trauma team activation. Blunt trauma patients with prehospital hypotension that are normotensive on arrival should have an arterial blood gas (ABG) with BD interpreted early upon admission to help identify those patients who are at risk for “crumping.” Surgeons should strongly consider taking blunt trauma patients with prehospital hypotension, a $BD \leq -6$, and a positive FAST examination directly to the OR even if they are presently normotensive in the trauma bay. In addition, such patients with a $BD \leq -6$ should have a repeat FAST if the initial study was negative. Invasive monitoring such as an arterial line and a central venous line should be placed expeditiously in blunt trauma patients with prehospital hypotension and a serum $BD \leq -6$. Since these patients are at risk for repeat hypotension and its consequences, the ability to detect hypotension as soon as it occurs and intervene with both resuscitation and intervention is paramount.

REFERENCES

1. American College of Surgeons Committee on Trauma. *Resources for Optimal Care of the Injured Patient*. Chicago, IL: American College of Surgeons; 2006.
2. Shapiro NI, Kociszewski C, Harrison T, Chang Y, Wedel SK, Thomas SH. Isolated prehospital hypotension after traumatic injuries: a predictor of mortality? *J Emerg Med*. 2003;25:175–179.
3. Lipsky AM, Gaushe-Hill M, Henneman PL, et al. Prehospital hypotension is a predictor of the need for an emergent, therapeutic operation in trauma patients with normal systolic blood pressure in the emergency department. *J Trauma*. 2006;61:1228–1233.
4. Codner P, Obaid A, Porral D, Lush S, Cinat M. Is field hypotension a reliable indicator of significant injury in trauma patients who are normotensive on arrival to the emergency department. *Am Surg*. 2005;71:768–771.
5. Kohn MA, Hammel JM, Bretz SW, Stangby A. Trauma team activation criteria as predictors of patients disposition from the emergency department. *Acad Emerg Med*. 2004;11:1–9.
6. Porter JM, Ivatury RR. In search of the optimal end points of resuscitation in trauma patients: A review. *J Trauma*. 1998;44:908–914.
7. Hoffman M. The cellular basis of traumatic bleeding. *Mil Med*. 2004;169:4–7.
8. Falcone RE, Santanello SA, Schulz MA, Monk J, Satiani B, Carey LC. Correlation of metabolic acidosis with outcome following injury and its value as a scoring tool. *World J Surg*. 1993;17:575–579.
9. Pal JD, Victorino GP, Twomey P, Liu TH, Bullard MK, Harken AH. Admission serum lactate levels do not predict mortality in the acutely injured patient. *J Trauma*. 2006;60:583–589.
10. Dunne JR, Tracy JK, Scalea TM, Napolitano LM. Lactate and base deficit in trauma: does alcohol or drug use impair their predictive accuracy? *J Trauma*. 2005;58:959–966.
11. Husain FA, Martin MJ, Mullenix PS, Steele SR, Elliott DC. Serum lactate and base deficit as predictors of mortality and morbidity. *Am J Surg*. 2003;185:485–491.
12. Martin M, Murray J, Berne T, Demetriades D, Belzberg H. Diagnosis of acid-base derangements and mortality prediction in the trauma intensive care unit: the physiochemical approach. *J Trauma*. 2005;58:238–243.
13. Bilkovski RN, Rivers EP, Horst HM. Targeted resuscitation strategies after injury. *Curr Opin Crit Care*. 2004;10:529–538.
14. Randolph LC, Takacs M, Davis KA. Resuscitation in the pediatric trauma population: admission base deficit remains an important prognostic indicator. *J Trauma*. 2002;53:838–842.
15. Kaplan LJ, Kellum JA. Initial pH, base deficit, lactate, anion gap, strong ion difference, and strong ion gap predict outcome from major vascular injury. *Crit Care Med*. 2004;32:1120–1124.
16. Davis JW, Shackford SR, Mackersie RC, Hoyt DB. Base deficit as a guide to volume resuscitation. *J Trauma*. 1988;28:1464–1467.

17. Davis JW, Parks SN, Kaups KL, Gladen HE, O'Donnell-Nicol S. Admission base deficit predicts transfusion requirements and risk of complications. *J Trauma*. 1996;41:769–774.
18. Rutherford EJ, Morris JA, Reed GW, Hall KS. Base deficit stratifies mortality and determines therapy. *J Trauma*. 1992;33:417–423.
19. Macleod JB, Lynn M, McKenney MG, Jeroukhimov I, Cohn SM. Predictors of mortality in trauma patients. *Am Surg*. 2004;9:805–810.
20. Holcomb JB, Niles SE, Miller CC, Hinds D, Duke JH, Moore FA. Prehospital physiologic data and lifesaving interventions in trauma patients. *Mil Med*. 2005;170:7–13.
21. Franklin GA, Boaz PW, Spain DA, Lukan JK, Carrillo EH, Richardson JD. Prehospital hypotension as a valid indicator of trauma team activation. *J Trauma*. 2000;48:1034–1039.
22. Parks JK, Elliott AC, Gentilello LM, Shafi S. Systemic hypotension is a late marker of shock after trauma: a validation of Advanced Trauma Life Support principles in a large national sample. *Am J Surg*. 2006;192:727–731.
23. American College of Surgeons Committee on Trauma. *ATLS Student Course Manual*. 7th ed. Chicago, IL: ACS; 2004.
24. Robertson J, Shilkofski N, eds. *The Harriet Lane Handbook*. 17 ed. Philadelphia, PA: Mosby; 2005.
25. Long JA, Klein MD. Trauma in infants and children. In: Wilson RF, Walt AJ, eds. *Management of Trauma: Pitfalls and Practice*. 2nd ed. Baltimore, MD: Williams & Wilkins; 1996:128–145.
26. Kincaid EH, Chang MC, Letton RW, Chen JG, Meredith JW. Admission base deficit in pediatric trauma: a study using the national trauma data bank. *J Trauma*. 2001;51:332–335.
27. Mackersie RC, Tiwary AD, Shackford SR, Hoyt DB. Intra-abdominal injury following blunt trauma. Identifying the high-risk patient using objective risk factors. *Arch Surg*. 1989;124:809–813.
28. Cathey KL, Brady WJ, Butler K, Blow O, Cephas GA, Young JS. Blunt splenic trauma: characteristics of patients requiring urgent laparotomy. *Am Surg*. 1998;64:450–454.
29. Edelman DA, White MT, Tyburski JG, Wilson RF. Post-traumatic hypotension: should systolic blood pressure of 90–109 mmHg be included? *Shock*. 2007;27:134–138.
30. Chan L, Bartfield JM, Reilly KM. The significance of out-of-hospital hypotension in blunt trauma patients. *Acad Emerg Med*. 1997;4:785–788.
31. Shapiro MJ, Jen J, McCormack JE. Let the surgeon sleep: trauma team activation for severe hypotension. *J Trauma*. 2008;65:1245–1250; discussion 1250–1252.
32. Lehmann RK, Arthurs ZM, Cuadrado DG, Casey LE, Beekley AC, Martin MJ. Trauma team activation: simplified criteria safely reduces overtriage. *Am J Surg*. 2007;193:630–635.
33. Clarke JR, Trooskin SZ, Doshi PJ, Greenwald L, Mode CJ. Time to laparotomy for intrabdominal bleeding from trauma does affect survival for delays up to 90 minutes. *J Trauma*. 2002;52:420–425.
34. Low RB. Accuracy of blood pressure measurements made aboard helicopters. *Ann Emerg Med*. 1988;17:604–612.
35. Davis JW, Davis IC, Bennink LD, Bilello JF, Kaups KL, Parks SN. Are automated blood pressure measurements accurate in trauma patients? *J Trauma*. 2003;55:860–863.
36. Mahoney EJ, Biffl WL, Harrington DT, Cioffi WG. Isolated brain injury as a cause of hypotension in the blunt trauma patient. *J Trauma*. 2003;55:1065–1069.
37. Patrick DA, Bensard DD, Janik JS, Karrer FM. Is hypotension a reliable indicator of blood loss from traumatic injury in children? *Am J Surg*. 2002;184:555–560.
38. Brill SA, Stewart TR, Brundage SI, Schreiber MA. Base deficit does not predict mortality when secondary to hyperchloremic acidosis. *Shock*. 2002;17:459–462.
39. Sinert R, Zehtabchi S, Bloem C, Lucchesi M. Effect of normal saline infusion on the diagnostic utility of base deficit in identifying major injury in trauma patients. *Acad Emerg Med*. 2006;13:1269–1274.
40. Peitzman AB, Heil B, Rivera L, et al. Blunt splenic injury in adults: multi-institutional study of the Eastern Association for the Surgery of Trauma. *J Trauma*. 2000;49:177–189.
41. Tisherman SA, Barie P, Bokhari F, et al. Clinical practice guideline: endpoints of resuscitation. *J Trauma*. 2004;57:898–912.
42. Davis JW, Mackersie RC, Holbrook TL, Hoyt DB. Base deficit as an indicator of significant abdominal injury. *Ann Emerg Med*. 1991;20:842–844.