Died of Wounds on the Battlefield: Causation and Implications for Improving Combat Casualty Care

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Background: Understanding the epidemiology of death after battlefield injury is vital to combat casualty care performance improvement. The current analysis was undertaken to develop a comprehensive perspective of deaths that occurred after casualties reached a medical treatment facility.

Methods: Battle injury died of wounds (DOW) deaths that occurred after casualties reached a medical treatment facility from October 2001 to June 2009 were evaluated by reviewing autopsy and other postmortem records at the Office of the Armed Forces Medical Examiners (OAFME). A panel of military trauma experts classified the injuries as nonsurvivable (NS) or potentially survivable (PS), in consultation with an OAFME forensic pathologist. Data including demographics, mechanism of injury, physiologic and laboratory variables, and cause of death were obtained from the Joint Theater Trauma Registry and the OAFME Mortality Trauma Registry.

Results: DOW casualties (n = 558) accounted for 4.56% of the nonreturn to duty battle injuries over the study period. DOW casualties were classified as NS in 271 (48.6%) cases and PS in 287 (51.4%) cases. Traumatic brain injury was the predominant injury leading to death in 225 of 271 (83%) NS cases, whereas hemorrhage from major trauma was the predominant mechanism of death in 230 of 287 (80%) PS cases. In the hemorrhage mechanism PS cases, the major body region bleeding focus accounting for mortality were torso (48%), extremity (31%), and junctional (neck, axilla, and groin) (21%). Fifty-one percent of DOW casualties presented in extremis with cardiopulmonary resuscitation upon presentation.

Conclusions: Hemorrhage is a major mechanism of death in PS combat injuries, underscoring the necessity for initiatives to mitigate bleeding, particularly in the prehospital environment.

Key Words: Military, War, Combat, Injury, Trauma, Died of wounds, Survivability.

(*J Trauma.* 2011;71: S4–S8)

At the inception of current military contingency operations, there was no organized method of combat casualty care data collection or system for the delivery of trauma care. The Joint Theater Trauma System was developed by military medical leaders to provide a systematic and integrated approach to battlefield care, resulting in minimization of morbidity and mortality and optimization of essential casualty injury care capabilities.[1–3] The Joint Theater Trauma Registry (JTTR) was developed to capture vital injury information for performance evaluation and improvement as well as combat injury epidemiology and surveillance.

Understanding the epidemiology of death after battlefield injury is vital to improving the outcomes of combat casualty care. The concept of a trimodal distribution of death after injury has been popularized in both the civilian and military settings.[4,5] Major studies of both civilian and military trauma epidemiology suggest that the majority of injury mortality occurs in the prehospital period.[6–16] From the military perspective, the lethality of injury in this environment can be impacted by measures aimed at prevention of injury, particularly personal protective equipment and changes in tactics, and through improvements in evacuation and prehospital resuscitative care. In the continuum, casualties who succumb to their injuries after admission to a hospital (died of wounds [DOW]) are reflective of the relative effectiveness of acute care of the medical treatment facility (MTF) and the trauma system. Evaluation of deaths at this level has shown to have significant performance improvement potential.[6,17,18] A substantial body of literature has analyzed hospital deaths after injury in the civilian setting.[5,6,16,17,19] Although other studies of combat mortality have been developed as a result of current contingency operations,[13,18,20,21] the current analysis is the only study to exclusively and completely investigate the DOW combat casualty.

The current analysis was undertaken to develop a comprehensive perspective of deaths that occurred after casualties reached an MTF to identify potential areas for research, development, and training.

METHODS

The JTTR was used to identify Operation Iraqi Freedom and Operation Enduring Freedom combat casualties from October 2001 to June 2009 who died from injury after admission to a military MTF.[22] As the analysis was to

Submitted for publication March 18, 2011.
Accepted for publication April 22, 2011.

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DOI: 10.1097/TA.0b013e318221147b

specifically evaluate DOW, only battle injury casualties were included in the study group. Exclusion criteria were nonbattle injury and other unnatural deaths such as drowning, electrocution, and suicide. The DOW rate was calculated by dividing the DOW count by the total number of nonreturn to duty battle injuries obtained from the JTTR for the corresponding time period. The JTTR was used to compile demographics, mechanism of injury, cause of injury, admission cardiopulmonary resuscitation (CPR) status, admission physiology, blood transfusion volume, Abbreviated Injury Scale score, and Injury Severity Score (ISS).

The identified casualty population was then used as the DOW study population for the fatality analysis at the Office of the Armed Forces Medical Examiner (OAFME). All US combat casualty deaths from theater are recovered and transported to Dover, DE, where complete identification and forensic examination are performed by the OAFME. The autopsy reports and other perimortem records, the Mortality Trauma Registry (MTR), and photographs on file with the OAFME were used by the analysis group for the conduct of the study. Cases included were DOW battle injury combat fatalities. The review panel for this study was composed of military trauma surgeons, a forensic pathologist, a military emergency medicine physician, an expert wound coder with MTR expertise, a trauma nurse, trauma epidemiologist, and surgical residents. As in the earlier mortality review, the panel used a consensus rule paradigm.[18,23] To maintain consistency and potential comparison value with past combat mortality analyses, the fatalities were classified as "nonsurvivable" (NS) or "potentially survivable" (PS)[18,20] after evaluation of the individual perimortem records listed above. As in previous analyses, when multiple wounds were identified, each was evaluated individually with respect to the potential for survivability. The consensus was to err toward the inclusion of these casualties as "potentially survivable" to facilitate the process of combat casualty care performance improvement. Specific wounds deemed to be NS were transcranial brain injury, brain stem injury, perforating cardiac injury, penetrating aortic injury, major tracheal injury below the thoracic inlet, or open pelvic injury with large soft tissue loss/traumatic hemipelvectomy. All patients were idealized to have immediate access to a US military Level III MTF with advanced surgical capabilities and more robust human, support, and materiel resources.

Institutional Review Board review for the study was provided by the Brooke Army Medical Center and the Armed Forces Institute of Pathology.

RESULTS

For the interval of the study from 2001 to June 2009, 558 DOW fatality records were reviewed. During the same period, there were 12,235 battle injury casualties recorded in the JTTR, resulting in a DOW rate of 558 of 12,235 (4.6%) over the study period. Interestingly, 232 of 456 (51%) casualties, in whom the admission status was known, presented in extremis with CPR in progress at admission. The casualties were predominantly men (97.3%), with a median age of 24 years (interquartile range of 22–29 years), and military service branch affiliation as follows: 73% Army, 15% Marine Corps/Navy, and 2% Air Force.

TABLE 1. Presentation ISS, Glasgow Coma Scale Score, and Transfusion Volume in DOW Casualties

Injury Variable	Mean (Standard Deviation)	Median (Interquartile Range)
ISS	30 (16)	27 (19−37)
Glasgow Coma Scale score	5 (4)	3 (3−3)
Units red blood cells	14 (15)	8 (4−19)

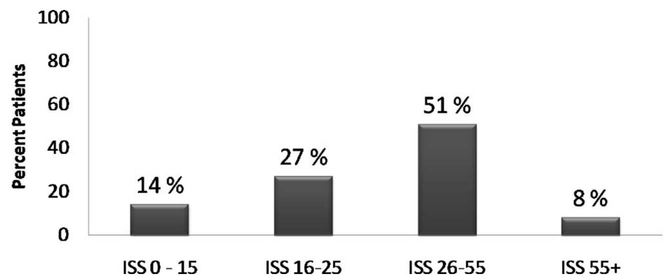

Figure 1. Distribution of injury severity in DOW.

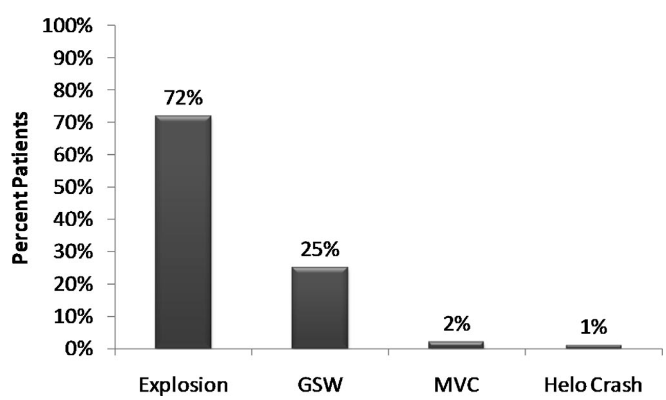

Figure 2. DOW injury agents.

Injury acuity in the DOW casualty population denoted by ISS, Glasgow Coma Scale score, and transfusion requirements is shown in Table 1. The distribution of injury severity is shown in Figure 1. Eighty-six percent of all DOW casualties had an ISS ≥16 consistent with moderate to severe injury. Fifty-nine percent had an ISS ≥25. Explosive events (72%) caused the majority of fatalities, followed by gunshot wounds (25%) (Fig. 2). DOW casualties were classified as NS in 271 (48.6%) cases and PS in 287 (51.4%) cases. After stratifying DOW fatalities as PS or NS (Fig. 3), the lethality in the NS group was predominantly characterized by traumatic brain injury (TBI; 83%). In contrast, the mortality in the PS group was predominantly acute hemorrhage (80%) caused by explosive or firearm injuries (Fig. 4). Of the fatalities caused by posttraumatic hemorrhage within the PS group, the anatomic distribution was 48% truncal, 31% peripheral extremity, and 21% junctional (Fig. 5).

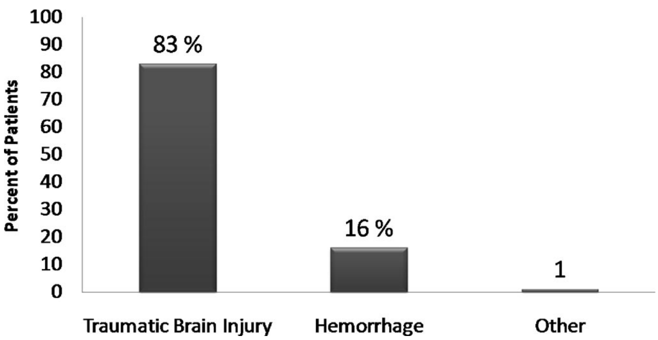

Figure 3. Mechanism of death in NS cases.

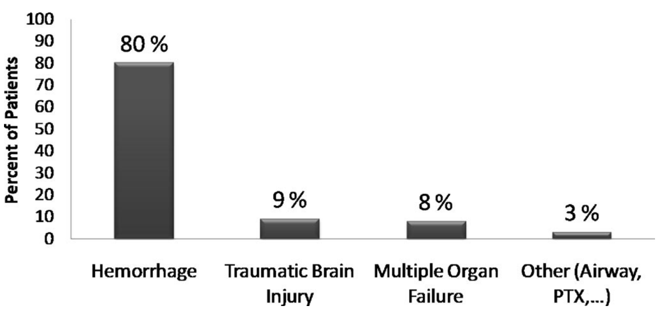

Figure 4. Mechanism of death in PS cases.

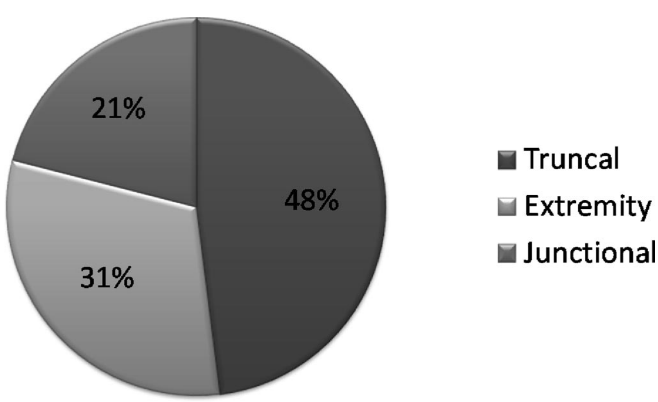

Figure 5. Main hemorrhage focus by anatomic region.

DISCUSSION

The review and analysis of injury deaths is paramount to the evaluation and improvement of trauma centers and trauma systems.[24] In one post hoc analysis of combat mortality from past conflicts, investigators surmised that improvements in education, research, and development could potentially decrease the incidence of in-hospital deaths by 8% to 25%.[25] Corroborating this evidence were two earlier studies during the current conflicts from the military medical literature. In 2004, in an analysis of a small population of special operations combat wounded who were both killed in action (KIA) and DOW, 12 of 82 (14.6%) casualties had PS injuries. Of those with PS injuries, hemorrhage was the most common mechanism of death.[20] A subsequent analysis of a casualty sample of 982 battlefield casualties composed of 75% KIA and 25% DOW by the same investigators was significant for a PS death ratio of 24%. These studies supported the proliferation of hemostatic adjuncts including tourniquets[26–29] and topical hemostatic agents[30,31] as well as the evolution and dissemination of the concepts of damage control resuscitation.[9,32–38] By evaluating the current population of those who died of their injuries after reaching a battlefield MTF, our goal was to identify opportunities for improvement across the breadth of the military trauma system.

The initial observation of significance in this analysis was that the 558 DOW casualties represented a DOW rate of 4.6% over the study period. This is striking as it compares favorably to in-hospital trauma deaths recorded in the National Trauma Data Bank and in Level I trauma centers within the United States which report rates of 3% to 5%[6,12,17,39,40] for the general injury population and up to 15.8% for specific penetrating injury populations.[41] Erring on the side of being more inclusive, we were critically introspective and included even those patients who presented in extremis with CPR (51%) ongoing at the time of presentation. These patients are inconsistently included in classic studies of in-hospital injury mortality. Had these patients been excluded, the DOW rate in this population would have been an unprecedented 2.3%. This observation is potentiated by the realization that the care is being provided in austere, often hostile conditions with numerous operational and tactical constraints that challenge the provision of injury care at all levels. Furthermore, analyzing injury variables, it was apparent that the DOW group studied in this analysis represented a group of moderately to severely injured casualties by ISS, with severe injuries comprising 59% of total injuries. The injury variable of low Glasgow Coma Scale score was consistent with the large number of casualties who presented with significant TBI or those who presented in extremis. Likewise, the number of units of blood per DOW casualty was indicative of a significant number of casualties requiring massive transfusion (>10 units red blood cells/24 h) and was associated with those who ultimately died of lethal postinjury hemorrhage. Similar results of causal mechanisms in early in-hospital death are substantiated by literature from both the civilian and military environments.[5–7,11,12,14,18,21,25,42–45]

The causality of injury from military conflict is largely not comparable with the civilian sector. In stark contrast to civilian injury patterns, 72% of casualties in the current series were caused by explosive agents. It can be difficult to sort out primary, secondary, and tertiary explosive injury effects in contemporary combat explosive injuries because of the severity of the injuries. The most current practice of the OAFME forensic pathologists is to classify the cause of death in explosions as blast injuries without further classification.

Evaluation of those casualties deemed NS was significant in that 83% were attributed to TBI. Numerous studies in the contemporary trauma literature note similar outcomes for TBI.[6,10,12,46] Mitigation strategies aimed at affecting the deleterious outcomes of TBI must stress prevention of primary brain injury with materiel and personal protective equipment technologies and minimization of secondary brain injury. Postinjury hemorrhage was also a significant mechanism of NS DOW injury. Because of the nature of the hemorrhagic source in this population including heart, aorta, and traumatic dismemberment, prevention strategies must be the main strategy for amelioration.

In contrast, as reported in previous studies of combat operations and civilian trauma centers, hemorrhage is the most substantial focus of PS battlefield injury.[10,18,20,25,33,35,44,47] Prior studies have shown that damage control resuscitation strategies decreased massive transfusion mortality from 38% to consistently below 20% in the severely injured combat casualty.[1,3,33,35,38] However, our findings support the consistent attribution of casualties who succumb to fatality after admission to a military MTF to complications of hemorrhage, underscoring the need to augment hemostatic practices with research and development. In addition, these findings champion the drive for advanced hemostatic techniques further forward onto the battlefield with agents such as freeze-dried plasma and novel topical hemostatic agents. Further stratification of the anatomic site of attributable DOW hemorrhage demonstrated that 48% was truncal, 31% peripheral extremity, and 21% junctional. The rate of peripheral extremity hemorrhage has decreased significantly over course of the conflict secondary to the dissemination of tourniquets onto the battlefield.[27–29] On the other hand, the 69% combined incidence of truncal and junctional hemorrhage as the primary mechanism of lethality stresses that more research is required to have hemorrhage control options in the tactical environment before combat casualties have exsanguinated.

This study is retrospective and thus has several limitations including the inherent limitations of large registries such as the JTTR and MTR, including misclassification bias, interobserver subjectivity/variability data interpretation, and input errors. In addition, because KIA was specifically not included in this analysis, the complete picture of the mortality profile over the spectrum of combat injury cannot be elucidated. Further analyses are in process by this group to correct this critical deficiency in future analyses. Finally, based on gross examination alone, there is potential undermeasurement of lethal TBI because of severe rotational injuries that can occur with the magnitude of forces casualties may experience during explosive events. Therefore, some NS cases classified may have been inadvertently misclassified as PS based on gross autopsy findings.

CONCLUSION

In conclusion, this comprehensive analysis of DOW combat casualties reiterates the necessity for operational strategies to enhance prevention, particularly for traumatic central nervous system injury. In addition, hemorrhage is the major mechanism of death in PS combat injuries, emphasizing the necessity for initiatives to mitigate bleeding, particularly in the prehospital environment.

REFERENCES

1. Eastridge BJ, Costanzo G, Jenkins D, et al. Impact of joint theater trauma system initiatives on battlefield injury outcomes. *Am J Surg*. 2009;198:852–857.
2. Eastridge BJ, Jenkins D, Flaherty S, Schiller H, Holcomb JB. Trauma system development in a theater of war: experiences from Operation Iraqi Freedom and Operation Enduring Freedom. *J Trauma.* 2006;61: 1366–1372; discussion 1372–1373.
3. Eastridge BJ, Wade CE, Spott MA, et al. Utilizing a trauma systems approach to benchmark and improve combat casualty care. *J Trauma.* 2010;69:S5–S9.
4. Bellamy RF, Maningas PA, Vayer JS. Epidemiology of trauma: military experience. *Ann Emerg Med*. 1986;15:1384–1388.
5. Trunkey DD. Trauma. Accidental and intentional injuries account for more years of life lost in the U.S. than cancer and heart disease. Among the prescribed remedies are improved preventive efforts, speedier surgery and further research. *Sci Am*. 1983;249:28–35.
6. Acosta JA, Yang JC, Winchell RJ, et al. Lethal injuries and time to death in a level I trauma center. *J Am Coll Surg*. 1998;186:528–533.
7. Bellamy RF. The causes of death in conventional land warfare: implications for combat casualty care research. *Mil Med*. 1984;149:55–62.
8. Bellamy RF. Contrasts in combat casualty care. *Mil Med*. 1985;150: 405–410.
9. Blackbourne LH, Czarnik J, Mabry R, et al. Decreasing killed in action and died of wounds rates in combat wounded. *J Trauma*. 2010;69:S1–S4.
10. Champion HR, Bellamy RF, Roberts CP, Leppaniemi A. A profile of combat injury. *J Trauma*. 2003;54:S13–S19.
11. Demetriades D, Kimbrell B, Salim A, et al. Trauma deaths in a mature urban trauma system: is "trimodal" distribution a valid concept? *J Am Coll Surg*. 2005;201:343–348.
12. Demetriades D, Murray J, Charalambides K, et al. Trauma fatalities: time and location of hospital deaths. *J Am Coll Surg*. 2004;198:20–26.
13. Gerhardt RT, De Lorenzo RA, Oliver J, Holcomb JB, Pfaff JA. Out-of-hospital combat casualty care in the current war in Iraq. *Ann Emerg Med*. 2009;53:169–174.
14. MacLeod JB, Cohn SM, Johnson EW, McKenney MG. Trauma deaths in the first hour: are they all unsalvageable injuries? *Am J Surg*. 2007;193:195–199.
15. Pfeifer R, Tarkin IS, Rocos B, Pape HC. Patterns of mortality and causes of death in polytrauma patients—has anything changed? *Injury*. 2009; 40:907–911.
16. Shackford SR, Mackersie RC, Holbrook TL, et al. The epidemiology of traumatic death. A population-based analysis. *Arch Surg*. 1993;128:571–575.
17. Gruen RL, Jurkovich GJ, McIntyre LK, Foy HM, Maier RV. Patterns of errors contributing to trauma mortality: lessons learned from 2,594 deaths. *Ann Surg.* 2006;244:371–380.
18. Kelly JF, Ritenour AE, McLaughlin DF, et al. Injury severity and causes of death from Operation Iraqi Freedom and Operation Enduring Freedom: 2003–2004 versus 2006. *J Trauma*. 2008;64:S21–S26; discussion S26–S27.
19. Stewart RM, Myers JG, Dent DL, et al. Seven hundred fifty-three consecutive deaths in a level I trauma center: the argument for injury prevention. *J Trauma*. 2003;54:66–70; discussion 70–71.
20. Holcomb JB, McMullin NR, Pearse L, et al. Causes of death in U.S. Special Operations Forces in the global war on terrorism: 2001–2004. *Ann Surg*. 2007;245:986–991.
21. Martin M, Oh J, Currier H. An analysis of in-hospital deaths at a modern combat support hospital. *J Trauma*. 2009;66(4 suppl):S51–S60; discussion S60–S61.
22. Holcomb JB, Stansbury LG, Champion HR, Wade C, Bellamy RF. Understanding combat casualty care statistics. *J Trauma*. 2006;60:397–401.
23. MacKenzie EJ, Steinwachs DM, Bone LR, Floccare DJ, Ramzy AI. Inter-rater reliability of preventable death judgments. The Preventable Death Study Group. *J Trauma*. 1992;33:292–302; discussion 302–303.
24. Committee on Trauma and Committee on Shock, Division of Medical Sciences. *Accidental Death and Disability: The Neglected Disease of Modern Society.* Washington, DC: National Academy of Sciences, National Research Council; 1966.
25. Blood CG, Puyana JC, Pitlyk PJ, et al. An assessment of the potential for reducing future combat deaths through medical technologies and training. *J Trauma.* 2002;53:1160–1165.
26. Beekley AC, Sebesta JA, Blackbourne LH, et al. Prehospital tourniquet use in Operation Iraqi Freedom: effect on hemorrhage control and outcomes. *J Trauma*. 2008;64:S28–S37; discussion S37.
27. Kragh JF Jr, Littrel ML, Jones JA, et al. Battle casualty survival with emergency tourniquet use to stop limb bleeding. *J Emerg Med*. 2009. ePub ahead of print.

28. Kragh JF Jr, Walters TJ, Baer DG, et al. Practical use of emergency tourniquets to stop bleeding in major limb trauma. *J Trauma*. 2008;64: S38–S49; discussion S49–S50.
29. Kragh JF Jr, Walters TJ, Baer DG, et al. Survival with emergency tourniquet use to stop bleeding in major limb trauma. *Ann Surg*. 2009;249:1–7.
30. Kheirabadi BS, Mace JE, Terrazas IB, et al. Safety evaluation of new hemostatic agents, smectite granules, and kaolin-coated gauze in a vascular injury wound model in swine. *J Trauma*. 2010;68:269-278.
31. Kheirabadi BS, Mace JE, Terrazas IB, et al. Clot-inducing minerals versus plasma protein dressing for topical treatment of external bleeding in the presence of coagulopathy. *J Trauma*. 2010;69:1062–1072; discussion 1072–1073.
32. Blackbourne LH. The next generation of combat casualty care. *J Trauma*. 2009;66:S27–S28.
33. Holcomb JB. Damage control resuscitation. *J Trauma*. 2007;62:S36–S37.
34. Holcomb JB. Optimal use of blood products in severely injured trauma patients. *Hematology Am Soc Hematol Educ Program*. 2010;2010:465–469.
35. Holcomb JB, Jenkins D, Rhee P, et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. *J Trauma*. 2007; 62:307–310.
36. White CE, Simmons JW, Holcomb JB, Aydelotte JD, Eastridge BJ, Blackbourne L. Impact of extremity amputation on combat wounded undergoing exploratory laparotomy. *J Trauma*. 2009;66:S86–S92.
37. White JM, Stannard A, Burkhardt GE, Eastridge BJ, Blackbourne LH, Rasmussen TE. The epidemiology of vascular injury in the wars in Iraq and Afghanistan. *Ann Surg*. 2011;253:1184–1189.
38. Borgman MA, Spinella PC, Perkins JG, et al. The ratio of blood products transfused affects mortality in patients receiving massive transfusions at a combat support hospital. *J Trauma*. 2007;63:805–813.
39. Claridge JA, Leukhardt WH, Golob JF, McCoy AM, Malangoni MA. Moving beyond traditional measurement of mortality after injury: evaluation of risks for late death. *J Am Coll Surg*. 2010;210:788–796.
40. Davidson GH, Hamlat CA, Rivara FP, Koepsell TD, Jurkovich GJ, Arbabi S. Long-term survival of adult trauma patients. *JAMA*. 2011;305:1001–1007.
41. Nathens AB, ed. National Trauma Data Bank Annual Report, 2010. American College of Surgeons. Available at: www.facs.org/trauma/ntdb/pdf/ntdbannualreport2010.pdf. Accessed April 8, 2011.
42. Beekley AC, Martin MJ, Spinella PC, Telian SP, Holcomb JB. Predicting resource needs for multiple and mass casualty events in combat: lessons learned from combat support hospital experience in Operation Iraqi Freedom. *J Trauma*. 2009;66:S129–S137.
43. Cancio LC, Wade CE, West SA, Holcomb JB. Prediction of mortality and of the need for massive transfusion in casualties arriving at combat support hospitals in Iraq. *J Trauma*. 2008;64:S51–S55; discussion S55–S56.
44. Eastridge BJ, Malone D, Holcomb JB. Early predictors of transfusion and mortality after injury: a review of the data-based literature. *J Trauma*. 2006;60:S20–S25.
45. Gomez R, Murray CK, Hospenthal DR, et al. Causes of mortality by autopsy findings of combat casualties and civilian patients admitted to a burn unit. *J Am Coll Surg*. 2009;208:348–354.
46. DuBose JJ, Barmparas G, Inaba K, et al. Isolated severe traumatic brain injuries sustained during combat operations: demographics, mortality outcomes, and lessons to be learned from contrasts to civilian counterparts. *J Trauma*. 2011;70:11–6; discussion 16–18.
47. Bochicchio G. Treatment of bleeding in the urban battlefield. *Surgery*. 2007;142:S78–S83.