

## Report Documentation Page

*Form Approved*  
*OMB No. 0704-0188*

Public reporting burden for the collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for Information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington VA 22202-4302. Respondents should be aware that notwithstanding any other provision of law, no person shall be subject to a penalty for failing to comply with a collection of information if it does not display a currently valid OMB control number.

1. REPORT DATE <b>01 AUG 2013</b>	2. REPORT TYPE <b>N/A</b>	3. DATES COVERED <b>-</b>		
4. TITLE AND SUBTITLE <b>Re: Traumatic cardiorespiratory arrest on the battlefield.</b>		5a. CONTRACT NUMBER		
		5b. GRANT NUMBER		
		5c. PROGRAM ELEMENT NUMBER		
6. AUTHOR(S) <b>Morrison J. J., Poon H., Rasmussen T. E., Khan M. A., Garner J. P.,</b>		5d. PROJECT NUMBER		
		5e. TASK NUMBER		
		5f. WORK UNIT NUMBER		
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) <b>United States Army Institute of Surgical Research, JBSA Fort Sam Houston, TX</b>		8. PERFORMING ORGANIZATION REPORT NUMBER		
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)		10. SPONSOR/MONITOR'S ACRONYM(S)		
		11. SPONSOR/MONITOR'S REPORT NUMBER(S)		
12. DISTRIBUTION/AVAILABILITY STATEMENT <b>Approved for public release, distribution unlimited</b>				
13. SUPPLEMENTARY NOTES				
14. ABSTRACT				
15. SUBJECT TERMS				
16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	
a. REPORT <b>unclassified</b>	b. ABSTRACT <b>unclassified</b>	c. THIS PAGE <b>unclassified</b>	<b>UU</b>	18. NUMBER OF PAGES <b>2</b>
				19a. NAME OF RESPONSIBLE PERSON

## Traumatic cardiorespiratory arrest on the battlefield

We read with interest the article by Morrison et al.<sup>1</sup> analyzing survival and the causes and times of death in patients undergoing resuscitative thoracotomy (RT) within the context of modern battlefield resuscitation. In this retrospective study of 65 patients who underwent RT, 14 (21.5%) survived. Among the 10 patients (15.4%) presenting an arrest in the field, none survived. The authors concluded that RT for patients having an arrest at the point of wounding seems to be futile, largely supporting both the US and UK military's clinical practice guidelines. Although the authors discussed that "this registry study is limited by its retrospective nature in that [they] may not have identified all eligible patients and are unable to report detailed neurological outcomes. [They] are also unable to comment on the use of cardiopulmonary resuscitation in the field because this prehospital data are not recorded within the JTTR," we would like to go further into the debate since performing cardiopulmonary resuscitation (CPR) on the battlefield is questionable. In this study, 10 patients presenting an arrest on the battlefield underwent RT in the field hospital: in real-life experience, it means that CPR was performed on the battlefield. We would like to point out that this attitude is not considered in well-established military guidelines. As with US military doctrine, current UK military protocol is not to attempt resuscitation from cardiorespiratory arrest in the presence of "effective enemy fire" on the battlefield.<sup>2</sup> Actually, the Tactical Combat Casualty Care (TCCC), a concept of prehospital casualty management specific to the combat and tactical environments, was developed in the mid-1990s in the US Special Forces community and, since evolved, earned a reputation of effectiveness across a broad spectrum of military organizations.<sup>3</sup> TCCC structures its guidelines to accomplish three primary goals as follows: first, treat the casualty; second, prevent additional casualties; and third, complete the mission. The TCCC includes the cases of casualties of blast or penetrating injury found to be without pulse, respiration, or other signs of life: in such cases, CPR on the battlefield "should not be attempted." Attempts to resuscitate

trauma patients in arrest have been discussed even in urban settings where victims are near trauma centers. In military settings, no benefit in terms of outcome has been exposed.<sup>4</sup> To illustrate, in a study by Tarmey et al.<sup>5</sup> determining the characteristics of military traumatic cardiorespiratory arrest and identifying factors associated with successful resuscitation, rates of survival from military traumatic cardiorespiratory arrest were similar to published civilian data, with 8% of patients surviving to discharge. Moreover, the authors reported that in more than 29 CPRs of 52 studied patients (56%), no casualty survived. Besides the absence of benefit in terms of survival, the cost of performing CPR on casualties on the battlefield, with what are inevitably fatal injuries, can result in additional lives lost because care could be withheld from casualties with less severe injuries. Moreover, these attempts expose rescuers to additional hazards from hostile fire. Before the combat casualty evacuation phase, as recommended by the TCCC, rescuers should consider CPR only in the cases of nontraumatic disorders such as hypothermia, near drowning, or electrocution.

To conclude, we would like to know if the authors could provide their current opinion regarding the achievement of CPR on the battlefield, with special insights on the injuries potentially sustained by first-line care givers (CPR providers on the ground, maybe exposed to hostile fire).

\*The authors declare no conflicts of interest.

**Pierre Pasquier, MD**  
**Clément Dubost, MD**  
**Stéphane Mérat, MD**  
*Intensive Care Unit*  
*Bégin Military Teaching Hospital*  
*Saint-Mandé, France*

### REFERENCES

- Morrison JJ, Poon H, Rasmussen TE, et al. Resuscitative thoracotomy following wartime injury. *J Trauma Acute Care Surg.* 2013;74: 825–829.
- Joint Service Publication JSP 999. Clinical Guidelines for Operations; 2012.
- Eastridge BJ, Mabry RL, Seguin P, et al. Death on the battlefield (2001–2011): implications for the future of combat casualty care. *J Trauma.* 2012;73(Suppl 5):S431–S437.
- Eastridge BJ, Hardin M, Cantrell J, Oetjen-Gerdes L, Zubko T, Mallak C, Wade CE, Simmons J, Mace J, Mabry R, et al. Died of wounds on the battlefield: causation and implications for improving combat casualty care. *J Trauma Acute Care Surg.* 2011;71:S4–S8.

- Tarmey NT, Parka CL, Bartelsa OJ, Koniga TC, Mahoney PF, Mellor AJ. Outcomes following military traumatic cardiorespiratory arrest: a prospective observational study. *Resuscitation.* 2011;82:1194–1197.

## Re: Traumatic cardiorespiratory arrest on the battlefield

### In Reply:

We are very grateful to Dr. Pasquier and colleagues for their thought provoking letter, discussing the role of cardiopulmonary resuscitation (CPR) on the battlefield. First, it is important to differentiate between *cardiac arrest* and *circulatory arrest* as a cause for the loss of a palpable central pulse.

Primary cardiac arrest generally relates to nontraumatic causes such as arrhythmias, electrocutions and so on and reflects a primary problem with cardiac contractility. CPR is appropriate because there is a circulating volume present with which to perfuse the systemic circulation. Circulatory arrest, as in exsanguination from hemorrhagic shock, does not initially relate to a failure of cardiac contraction but reduced cardiac preload. Untreated circulatory arrest will eventually progress to cardiac arrest owing to a failure of systemic oxygenation.

Circulatory arrest from hemorrhage is the leading cause of potentially preventable death among battlefield casualties<sup>1,2</sup> and served as the main indication for resuscitative thoracotomy in our recent study.<sup>3</sup> We are of the opinion that the management priorities in circulatory arrest are hemorrhage control and restoration of circulating volume. We do not believe that CPR is of benefit in such cases because there is no circulating volume with which to perfuse the systemic circulation.

We reported 10 cases of arrest in the field; however, this does not mean that these patients received CPR. Indeed, as all the authors have deployed to Afghanistan, serving at all medical echelons, patients do not always receive CPR in the field, as per UK prehospital care guidelines.<sup>4</sup> This largely relates to adverse tactical circumstances and other triage priorities. Furthermore, we are also unaware of any injuries sustained by a first-line care giver sustained during the performance of field CPR.

The reality is that both CPR and resuscitative thoracotomy are unsatisfactory interventions as patients have already crossed a physiologic threshold from which recovery is

unlikely. Both civilian and military trauma care providers need to focus on proactive maneuvers that can bridge the circulation to definitive hemorrhage control. Resuscitative endovascular balloon occlusion of the aorta is such an intervention, which can be deployed out with the operating room while the patient has a spontaneous circulation.<sup>5</sup> This can temporize hemorrhage control and provide afterload support until definitive hemostasis can be achieved.

\*The authors declare no conflicts of interest.

**Jonathan James Morrison, MRCS**

US Army Institute of Surgical Research  
Fort Sam Houston, Texas  
Academic Department of Military Surgery  
and Trauma

Royal Centre for Defence Medicine  
Birmingham, United Kingdom

**Henrietta Poon, MRCS**

**Todd E. Rasmussen, MD**

US Army Institute of Surgical Research  
Fort Sam Houston, Texas

**Mansoor A. Khan, FRCS**

Academic Department of Military Surgery  
and Trauma  
Royal Centre for Defence Medicine  
Birmingham, United Kingdom

**Jeffery P. Garner, FRCS**

Rotterdam NHS Foundation Trust  
Rotterdam, United Kingdom

**REFERENCES**

1. Eastridge BJ, Mabry RL, Seguin P, et al. Death on the battlefield (2001–2011): implications for the future of combat casualty care. *J Trauma Acute Care Surg.* 2012;73(Suppl 5):S431–S437.
2. Morrison JJ, Stannard A, Rasmussen TE, et al. Injury pattern and mortality of non-compressible torso haemorrhage in UK combat casualties. *J Trauma Acute Care Surg.* 2013;75:263–268.
3. Morrison JJ, Poon H, Rasmussen TE, et al. Resuscitative thoracotomy following wartime injury. *J Trauma Acute Care Surg.* 2013;74:825–829.
4. Joint Doctrine Publication 4-03.1. Clinical Guidelines for Operations; 2008.
5. Stannard A, Eliason JL, Rasmussen TE, et al. Resuscitative endovascular balloon occlusion of the aorta (REBOA) as an adjunct for hemorrhagic shock. *J Trauma.* 2011;71:1869–1872.

## The influence of mechanical ventilation in a hemorrhagic shock model

**To the Editor:**

With great interest we read the article “Still making the case against

prehospital intubation: A rat hemorrhagic shock model” by Dr. Taghavi et al. in *J Trauma Acute Care Surg.* 2012;73:332–337. Taghavi et al. have investigated the role of prehospital intubation on the survival in a hemorrhagic shock (HS) rat model. They concluded that intubation and consequently mechanical ventilation (MV) did not result in improved survival after HS. Although the studied groups are relatively low, the results suggest a detrimental effect of MV. Based on these findings, they further conclude that prehospital intubation is detrimental for patients with penetrating injury.

These findings are very interesting, and we would like to make a few comments. The authors suggest that the detrimental effect of MV can be explained by the accentuated end-organ hypoperfusion induced by decreased venous return caused by the positive-pressure ventilation. Decreased venous return caused by MV can be relevant when (very) high positive pressure is used; however the authors do not mention whether they have used high positive-end expiratory pressure levels at all. It is mentioned that the ventilator was set at a tidal volume of 8 mL/kg, which is in accordance with lung protective ventilation recommendations.<sup>1</sup>

It is well-known that MV can induce an inflammatory response, even with low tidal volumes.<sup>2</sup> We have studied the inflammatory response of MV in HS rat model.<sup>3</sup> Our data showed that HS alone has minimal effect on the development of an inflammatory response. MV (alone or in combination with HS), however, was the determining factor in inducing an inflammatory response. We therefore question whether it is more likely that the detrimental effect of MV in the presented study was caused by the inflammatory response induced by MV rather than by a decreased venous return.

When the authors extrapolated their results to the human situation, they wondered whether scoop and run would be better than intubation at the scene in penetrating injury. Most patients in profound HS in whom scoop and run is considered will need to reach the hospital as soon as possible because they will need surgery urgently. One could question whether respiratory acidosis by lack of intubation outweighs the effects induced by MV in a patient who will end up being intubated and ventilated anyway because of surgery within measurable time.

The nose coned, nonventilated, animals with hypercapnia and respiratory acidosis showed a better survival compared with ventilated rats after HS. In current protective ventilatory strategies, hypercapnia is a well-known and accepted adverse effect.<sup>1</sup> Permissive hypercapnia has even shown to contribute to the beneficial effects of protective lung

ventilatory strategies. However, the optimal ventilatory strategy and the precise contribution of hypercapnia remains unclear.<sup>4</sup> Schwartz et al.<sup>5</sup> have demonstrated in a canine model that hypercapnic acidosis applied before or after hemorrhage preserved microvascular mucosal oxygenation, suggesting that deliberate hypercapnic acidosis could serve to augment oxygenation of the splanchnic region after hemorrhage. This phenomenon might explain the fact that nonventilated rats tolerated a greater degree of shock compared with ventilated rats in the presented study. This poses the question whether the results would have been similar when both groups had had comparable CO<sub>2</sub> levels. Can the better survival in nose coned animals be attributed to the detrimental effect of MV, and/or is it caused by a possible preserving effect of hypercapnia?

Based on the previously mentioned arguments, we believe that the results can be largely explained by the inflammatory response induced by MV. It remains questionable whether the authors have really made a case against prehospital intubation after HS. In our opinion, they have demonstrated the importance of the inflammatory response after MV.

The study, however, does raise important questions about the precise role of the inflammatory response during the natural course of HS and the influence of hypercapnia on the inflammatory response in both ventilated and nonventilated rats. Therefore, we feel it would be very interesting to investigate the induced inflammatory response during the natural course of HS and the influence of hypercapnia on the development of the inflammatory response in both ventilated and nonventilated rats.

\*The authors declare no conflicts of interest.

**Karlijn J.P. van Wessem, MD, PhD**

**Luke P.H. Leenen, MD, PhD**

University Medical Center Utrecht  
Utrecht, The Netherlands

**REFERENCES**

1. Petrucci N, Iacovelli W. Lung protective ventilation strategy for the acute respiratory distress syndrome. *Cochrane Database Syst Rev.* 2007;(3):CD003844.
2. Plötz FB, Vreugdenhil HAJ, Slutsky AS, Zijlstra J, Heijen CJ, van Vught H. Mechanical ventilation alters the immune response in children without lung pathology. *Intensive Care Med.* 2002;28:486–492.
3. van Wessem KJP, Hennis MP, Heeres M, Koenderman L, Leenen LPH. Mechanical ventilation is the determining factor in inducing an inflammatory response in a hemorrhagic shock model. *J Surg Res.* 2013;180:125–132.
4. Ni Chonghaile M, Higgins B, Laffey JG. Permissive hypercapnia: role in protective lung