



Taser Safety Remains Unclear
Physiological Effects of the Taser

In reply

These two letters to the editor, and the authors' reply, were posted on the *Annals of Emergency Medicine* website on June 19, 2008. Scheduled for publication in the journal's 2008 July issue, they were written regarding Vilke et al's report of a Taser study performed on healthy human subjects, published in the journal's November 2007 issue. **Here is a link to Vilke et al's report:**

[Physiological Effects of a Conducted Electrical Weapon on Human Subjects](#)

Letter #1 CITATION: Strote J, Hutson HR. Taser safety remains unclear. *Ann Emerg Med*, July 2008; V 52, No 1, Pgs 84-85.

[http://www.annemergmed.com/article/S0196-0644\(08\)00006-1/abstract](http://www.annemergmed.com/article/S0196-0644(08)00006-1/abstract)

Letter #2 CITATION: Koscove EM. Physiological effects of the Taser. *Ann Emerg Med*, July 2008; V 52, No 1, Pg 85.

[http://www.annemergmed.com/article/S0196-0644\(08\)00005-X/abstract](http://www.annemergmed.com/article/S0196-0644(08)00005-X/abstract)

Reply CITATION: Vilke GM, Sloane CM, Neuman TS, Castillo EM, Chan TC, Kolkhorst FW. In reply. *Ann Emerg Med*, July 2008; V 52, No 1, Pgs 85-86.

[http://www.annemergmed.com/article/S0196-0644\(08\)00007-3/abstract](http://www.annemergmed.com/article/S0196-0644(08)00007-3/abstract)

CD MILLER'S REVIEW:

Vilke et al *continue* to cite the ridiculous 1997 Chan et al restraint position study as if it had a legitimate relationship to real-life situations. It does **NOT** – it *never* has. And, these authors know this!

When their study methods are challenged as being unrelated to real-life situations, Vilke/Chan et al offer the same excuse they always have; that a "high risk of death" prevents them from using "stimulant intoxicated human subjects" – prevents them from employing study parameters that come even *close* to real-life situations. Thus, the Vilke/Chan et al studies remain useful *only* as a **defense-via-confusion** ploy (something desperately sought by individuals hoping to legally and morally absolve themselves from causing someone's death with their inappropriate Taser use, and/or their employment of an asphyxial form of restraint).

Since that is the only manner in which their research information is "useful," why do *YOU* think the Vilke/Chan et al types continue wasting time and money designing and performing studies that cannot possibly yield legitimate, real-life related results?

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Taser Safety Remains Unclear

To the Editor:

We appreciate the important study by Vilke et al in the November issue of *Annals of Emergency Medicine*,¹ as it adds valuable data on conducted electrical weapon safety in an era of wide and increasing use by both law enforcement and the general public. We are concerned, however, that the “negative” results of the study may distract from the real safety issues surrounding these weapons.

In our 2006 study,² we found that deaths associated with Taser use overwhelmingly occurred in individuals who had preexisting cardiovascular disease (54%), illicit stimulant use (70%), and/or an agitated state often leading to long struggles (78%). Furthermore, many publicized deaths associated with Taser use involve multiple discharges of the weapon, often in rapid succession.

The current study consciously took these variables out of the equation, using a single 5-second discharge on healthy volunteers without any of the physiologic conditions usually encountered in Taser-related deaths.

Healthy individuals under normal circumstances can handle large physiologic and metabolic stress without noticeable clinical consequences. The real safety question is this: do these weapons cause physiologic alterations which, although possibly clinically insignificant in healthy individuals, may be harmful, or even life-threatening, when they occur in at-risk populations, such as those who are more likely to die in struggles with law enforcement?³

For example, the result that lactate rises by 1.4 mmol/L and pH drops 0.02 units may be insignificant in an unrestrained individual who can effectively compensate with respiratory and renal adjustments, but for a patient whose compensatory ventilation may be limited by restraint, whose heart may be more prone to arrhythmia due to prior insult and/or stimulant use, or whose acidosis from prolonged struggle is already near life-threatening levels, such an event could be the final insult leading to a terminal event.

Even if the relatively small physiologic changes seen in this study are tolerable, swine model studies^{4,5} have begun to suggest that multiple exposures could lead to much more impressive changes than those seen from a single discharge.

In summary, we feel that the authors' comment that there are “no clinically relevant changes” may be applicable to many individuals, but it is potentially misleading about Taser safety. Only by recreating realistic field conditions where deaths have occurred can the true danger of conducted electrical weapons be tested. In these situations, the changes noted in this study could indeed be clinically significant and potentially fatal.

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1. Vilke GM, Sloane CM, Bouton KD, et al. Physiological effects of a conducted electrical weapon on human subjects. *Ann Emerg Med.* 2007;50:569-575.
2. Strote J, Range Hutson H. Taser use in restraint-related deaths. *Prehosp Emerg Care.* 2006;10:447-450.
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4. Esquivel AO, Dawe EJ, Sala-Mercado JA, et al. The physiologic effects of a conducted electrical weapon in swine. *Ann Emerg Med.* 2007;50(5):576-583.
5. Jauchem JR, Sherry CJ, Fines DA, et al. Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of *Sus scrofa* following repeated TASER exposures. *Forensic Sci Int.* 2006;161:20-30.

Physiological Effects of the Taser

To the Editor:

Drs. Vilke et al are to be congratulated on their recent report which details simple physiological effects of the Taser on humans.¹

Was there an unpublished rationale for measuring the pH and lactate only once between baseline and 10 minutes, ie, at 1 minute? It is conceivable that a lower pH nadir and a higher lactate peak may have been discovered by measurements at 2, 4, 6, or even 8 minutes post-Tasering. While it would have entailed a small change in methodology, an additional missed opportunity in Dr. Vilke's study is measurement over time of serum catecholamines in their Tasered human volunteers.

It is interesting to note the relatively short duration of pH decline and the longer duration of lactate elevation. In Tasered humans, could one set of potentially arrhythmogenic parameters be present in an early "low pH phase," and a different set present during a longer "high lactate phase"?

Given the known onset of arrhythmias during or immediately after nonTaser electrical injuries, it is often stated that the Taser would not be termed the "immediate" cause of death in cases where a presumed arrhythmia started in a delayed fashion, eg, 5-30 minutes after Tasering.

However, Dr. Vilke's finding of a lactate elevation lasting 30 minutes after Tasering raises an important question. In a patient with agitation and sympathomimetic elevation in the presence of cocaine or methamphetamine, could further agitation or patient struggling, with additional release of epinephrine and norepinephrine, in the presence of an elevated lactate partially induced by Tasering, lead to the delayed onset of fatal

arrhythmia in that 30 minutes? Even in the absence of further struggling, is the elevated lactate level arrhythmogenic by itself? More Tasering is associated with higher levels and longer duration of elevated lactate.²

One Taser study addressed the state of cocaine intoxication, another addressed the state of an elevated epinephrine level in the absence of cocaine, both in anesthetized animals.^{3,4} Human studies in either volunteers or in the field are precluded by obvious legal and practical constraints. Restraint stress and its cardiac effects are challenging and confounding issues.⁵ A nonindustry funded study looking at Taser's effects in animals infused with both cocaine (or methamphetamine) and catecholamines, presumably more accurately mimicking the physiological state in many Tasered human events, is needed.

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1. Vilke GM, Sloan CM, Bouton KD, et al. Physiological effects of a conducted electrical weapon on humans. *Ann Emerg Med.* 2007; 50:569-575.
2. Jauchem JR, Sherry CJ, Fines DA, et al. Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of *Sus scrofa* following repeated TASER[®] exposures. *Forensic Sci Int.* 2006;161:20-30.
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In reply:

We appreciate the opportunity to respond to the letters received by *Annals of Emergency Medicine*. One letter noted that our study design consciously took the variables of agitation and stimulant drug use "out of the equation," and this is correct. At the time this study was designed, there were no other published human studies evaluating the Taser. Our Human Subjects Committees had concerns given recent media reports of sudden deaths following Taser use, and limited our study subjects to healthy individuals. A controlled study investigating the Taser effects on stimulant intoxicated human subjects could offer

additional knowledge beyond that of our current study; however, we believe such a study would not be approved by human subjects committees. There was no unpublished rationale to the timing of blood draws.

We disagree with comments regarding the pH drops seen in our study and reiterate that changes of 0.02 units are minor and of limited clinical significance. Additionally, there has been no work published to demonstrate that a restrained individual will have “compensatory ventilation. . . limited by restraint.” In fact, quite the opposite is true. Position and restraint have been shown to be essentially physiologically neutral and there is no reason to assume, nor data to support, the speculation that an individual who is restrained could not compensate for a pH change of 0.02 units.¹⁻³

Comments on the elevated lactate levels need to be taken into context. Though elevated, these are modest elevations at best, and lactate levels 5 times higher than produced by the Taser activation are seen with just exercising on a stationary bicycle.⁴ Comments that we did not use subjects “whose acidosis from prolonged struggle is already near life-threatening levels” also deserves additional comment. First, as noted above, our goal was to determine whether the Taser device posed any safety risk to humans at rest. Additionally, there has been no published work to demonstrate that prolonged struggle in restraint can produce “life-threatening levels” of acidosis. In fact, prior work has demonstrated that struggle in restraint results in significant lower oxygen consumption (and thus acid production) when compared with moderate-intensity running.³ However, we agree that preexisting acidosis should be tested and are in the midst of studying the effect on subjects following an aggressive exercise regimen.⁴

One letter references that animal studies in swine subjected to multiple Taser exposures results in more impressive physiologic changes. References to swine models must be taken into context and interpreted carefully and critically. These animals are sedated, anesthetized, and paralyzed (and thus cannot compensate) and as such do not represent well the human condition during field uses of the Taser. In the 2 specific animal studies cited by the letter, the actual deployment of Tasers was far beyond that of typical or suggested field use with one studying 18 5-second exposures in 3 minutes and the other having 20 5-second exposures in 31 minutes.^{5,6} Furthermore, in such a model compensatory ventilation (respiratory alkalosis) cannot take place.

Finally, the comment that our conclusion was “potentially misleading” was taken out of context and is misleading itself. The complete conclusion published was: “We conclude that a 5-second exposure of a TASER X-26 to healthy subjects does not result in clinically significant changes in ventilatory or blood parameters of physiologic stress. This study offers a foundation for the understanding of the effects of a single Taser activation in humans.” This is all that we were able to conclude based on our study design and results and feel it is intellectually honest and accurate.

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2. Chan TC, Vilke GM, Clausen J, et al. The effect of oleoresin capsicum “pepper” spray inhalation on respiratory function. *J Forensic Sci.* 2002;47:299-304.
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Effect of Activated Charcoal on Citalopram-Induced QT Prolongation

To the Editor:

We read with great interest the article “Activated Charcoal Decreases the Risk of QT Prolongation after Citalopram Overdose” in the November 2007 issue of *Annals of Emergency Medicine*.¹ The authors raise an excellent question regarding the utility of single-dose activated charcoal in these times of increasing nonuse of gastrointestinal decontamination and provide a hypothesis for future research.

We did, however, note some limitations that may diminish the validity of this article but focus future work in this field. Though the authors have previously reported an association