To view a sample EKG that has been transmitted via cell phone go to www.gmail.com and access the account: user name: SMALLTOWNED, password: 12LEADEKG (click "me" to open the file).

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1. Bradley BH. Strategies for reducing the coor-to-balloon time in acute myocardial infarction. *NEJM*. 2006; 355:2308-2320.
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In response to Tong TC, et al. Comparative treatment of alpha-amanitin poisoning with *N-acetylcysteine*, benzylpenicillin, cimetidine, thioctic acid, and silybin in a murine model

To the Editor:

We read with interest the study by Tong et al comparing several antidotes for amatoxin-induced hepatocellular injury.[1] We have direct experience with the use of cimetidine as an antidote for Amanita hepatotoxicity in animals and humans and were interested by the differences between the results obtained by Tong et al and prior published results.[2] In an effort to reconcile the different results, we noted that the dose of cimetidine administered by Schneider et al was actually greater than that given by Tong et al, contrary to their statement in their manuscript. Rather than 120 mg/kg/day administered in divided doses every 6 hours, the dose administered by Schneider et al was 120 mg/kg concurrently with alpha-amanitin administration in one experimental group, followed by a second dose. In a second experimental group, administration was delayed as in the study by Tong et al. It is our concern that the lower dose administered in the present study may not have been adequate to replicate the results.

We would also like to report that we have used similar dosing in humans (200 mg/hour by intravenous delivery) for the treatment of Amanita phalloides hepatotoxicity. Determination of efficacy in our human patients is compromised by the lack of suitable controls, knowledge of the dose of toxin, use of high dose penicillin in addition to cimetidine, as well as other variables that cannot be controlled in the clinical environment.

Absent sufficient controls, we attempted to evaluate efficacy of the treatment by comparing our patients in terms of severity with larger studies previously published. Fantozzi et al reported a case series of 160 patients with Amanita toxicity and classified them into mild, moderate and severe (groups A, B, and C) based on transaminase levels and prothrombin times.[3] Sabeel et al reported a case series of 41 patients with Amanita toxicity similarly classified into 3 groups on the same criteria.[4] Based on transaminase values, our patients 3 and 4 were mild (C-group), patient 1 was moderately severe (B-group) and patient 2 was severely affected (A-group).

Calculation of the disappearance rates (not presented) indicates that our patients' rates of disposal of AST are about twice the published rates in the moderately and severely poisoned groups. Fantozzi et al reported 3 deaths in their severe group and 1 death in their moderate group.[3] Thus, there is no evidence to suspect that cimetidine was detrimental to the course of our 4 patients. Comparison of our human subjects with historical controls reveals a greater survival (not statistically significant), but also a more rapid return to baseline transaminase values, implying a decrease in ongoing hepatocellular injury and, in fact, a positive effect in humans.

While we withhold comment regarding the other therapies evaluated by Tong et al, we would recommend that investigators not be dissuaded from the evaluation of cimetidine as potential therapy of hepatotoxicity induced by amatoxins.

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1. Tong TC, Hernandez M, Richardson WH, et al., Comparative treatment of alpha-amanitin poisoning with N-acetylcysteine, benzylpenicillin, cimetidine, thioctic acid, and silybin in a murine model. *Ann Emerg Med*. 2007;50:282-288.
2. Schneider SM, Borochovitz D, Krenzelok EP. Cimetidine protection against alpha-amanitin hepatotoxicity in mice: a potential model for the treatment of Amanita phalloides poisoning. *Ann Emerg Med*. 1987;16: 1136-1140.

3. Fantozzi R, et al. Clinical findings and follow-up evaluation of an outbreak of mushroom poisoning–survey of Amanita phalloides poisoning. *Klin Wochenschr*. 1986;64:38-43.
4. Sabeel AI, Kurkus J, Lindholm T. *I*ntensive hemodialysis and hemoperfusion treatment of Amanita mushroom poisoning. *Mycopathologia*. 1995;131:107-114.

In reply:

We thank Dr. Salhanick for his clarification of this difference between our study and that by Schneider et al in which 1 of 3 experimental groups poisoned by alpha-amanitin was administered cimetidine 4 hours after poisoning.[1] While both of our experimental subjects effectively received a 120 mg/kg/day dose of cimetidine, our dosing was indeed divided and given over 24 hours and then repeated for an additional day. Schneider et al chose to administer a single dose without repeat administration.

As we noted in our discussion, direct comparisons with previous studies are problematic given differences in methodology. In addition to repeat administration, for instance, our results were also evaluated by more than one standardized measure of hepatotoxicity (serum aminotransferase measurements and grading on a validated scale of histologic injury). As we acknowledged in our study, the choice of dosing could have influenced the results for all treatment groups. Our results indicate a lack of efficacy with the antidotal therapy chosen, but clearly this applies only to the dosages selected as ours was not a dosing study. Selected dosages were based on a review of the existing literature for this species.

We are grateful to Dr. Salhanick et al for sharing his insight and experiences with amatoxin poisoning and encourage others to continue their efforts to find effective antidotal therapy.

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1. Schneider SM, Borochovitz D, Krenzelok EP. Cimetidine protection against alpha-amanitin hepatotoxicity in mice: a potential model for the treatment of Amanita phalloides poisoning. *Ann Emerg Med*. 1987;16:1136-1140.

In Response to "To Sit or Not to Sit"

To the Editor:

I enjoyed the careful work the authors of "To Sit or Not to Sit" put into their article.[1] It supported both prior articles and the perception of bedside clinicians. However, it overlooked 1 key point.

The majority of emergency physicians work rotating 12 hour shifts like some draconian throwback to the 19th century. Fatigue in the final few hours can easily compromise medical quality and patient safety.

Just do the math: (12 hour shift) X (2 patients per hour) X (8 +/- minutes of sitting down per patient) versus standing for an entire shift. That's almost 2 full hours of "rest" per shift. After 35 years in emergency medicine, I know that "sitting" is better for both the patient and myself.

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1. Johnson RL, Sadosty AT, Weaver AL, et al. To sit or not to sit? *Ann Emerg Med*. 2008;51:188-193.

In reply:

We would like to thank Dr. Alexandra Murphy for her editorial and her insightful comments regarding the provider-patient interaction and would also like to thank Dr. Larry Romane for his remarks on provider fatigue.[1-2]

In "To Sit or Not to Sit?" we utilized 2 sham questions to distract providers from the true intent of the study.[3] The responses to these questions provide data that address the perceived gap in our research. The data for the sham questions demonstrate that the providers randomized to the standing position were more likely to report that their feet hurt ($p=0.002$) and likewise, that their back hurt ($p=0.010$) than those randomized to sit. While not our primary objective, our study does suggest that from a provider-centric standpoint, sitting may matter. While posture was not shown to influence patient perceptions of interaction quality, since sitting has been proven to increase patient estimates of time spent at the bedside and prolonged standing has been associated with occupational-related health problems, why not sit?[3-5]