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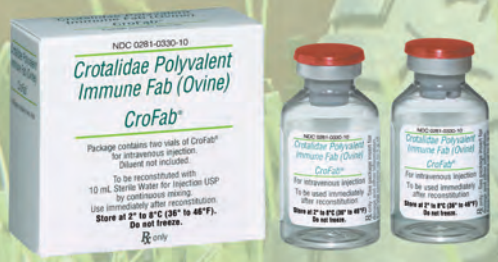

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Emergent Endotracheal Intubation and Mortality in Traumatic Brain Injury

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Objectives: To determine the relationship between emergent intubation (emergency department and field intubation cases combined) and mortality in patients with traumatic brain injury while controlling for injury severity.

Methods: Retrospective observational study of 981 (35.2% intubated, 64.8% not intubated) patients with TBI evaluating the association between intubation status and mortality. Logistic regression was used to analyze the data. Injury severity measures included Head/Neck Abbreviated Injury Scale (H-AIS), systolic blood pressure, type of head injury (blunt vs. penetrating), and a propensity score combining the effects of several other potential confounding variables. Age was also included in the model.

Results: The simple association of emergent endotracheal intubation with death had an odds ratio (OR) of 14.3 (95% CI = 9.4 – 21.9). The logistic regression model including relevant covariates and a propensity score that adjusted for injury severity and age yielded an OR of 5.9 (95% CI = 3.2 – 10.9).

Conclusions: This study indicates that emergent intubation is associated with increased risk of death after controlling for a number of injury severity indicators. We discuss the need for optimal paramedic training, and an understanding of the factors that guide patient selection and the decision to intubate in the field.
[*West*JEM. 2008;9:184-189.]

INTRODUCTION

Aggressive emergent airway management for traumatic brain injury (TBI) has become a mainstay of most prehospital emergency management system (EMS) protocols. The rationale for emergent intubation is to establish an airway and prevent secondary brain injury. Despite the widespread adoption of endotracheal intubation for head-injured patients, there is little evidence from research demonstrating its life-saving value in prehospital care. Although numerous studies show an increase in mortality associated with prehospital intubation,[1-4] some have shown an associated decrease in mortality.[5] Considerable efforts have been made to determine if the observed association between prehospital intubation and mortality is real, or an apparent association caused by confounding variables.

There are a number of possible explanations for the excess mortality among head-injured patients who receive prehospital intubation. The decision or ability to intubate in the field may be markers of injury severity that are currently not accounted for in the usual list of potential confounders.[6] Other intrinsic physiologic variables could make intubation detrimental for some patients, or perhaps intubation is being performed on individuals who do not meet the current criteria for intubation. Misplaced endotracheal tubes may be detrimental to patients, and inadequate training or experience in intubation among paramedics has also been recognized as a problem that could

contribute to mortality.[1,3,7-9] In one well-developed prehospital system, there was a 13% rate of unsuccessful intubation.[8] One study of field intubation reported as many as 25% of tubes are misplaced when protocols for verification of tube placement are not followed carefully.[10]

The use of neuromuscular blockade in the field could ease some of the difficulty in placing endotracheal tubes and reduce tube misplacements.[11-13] Several studies of neuromuscular blockade (NMB) assisted intubation in some aero-medical and ground EMS services suggest greater intubation success rates in patients where NMB is employed.[11,13-15] Bulger et al.[16] have demonstrated that NMB is associated with increased survival and improved outcomes among TBI cases. However, the use of NMB agents in the field has been criticized elsewhere, and data in support of the protocol's effectiveness in decreasing mortality risk are regarded as inconclusive.[17-19]

Hyperventilation is another factor related to increased mortality among field-intubated patients with TBI. Although hyperventilation decreases intracranial pressure by cerebral vasoconstriction, the practice also may diminish cerebral blood flow to ischemic levels.[20] Ghajar[21] notes that decreased mortality and improved outcomes are the result of practices such as increasing cerebral perfusion, which would reduce the impact of secondary injury. Chestnutt[22] also provides an extensive review of the problem of secondary brain injury**.**

Enumeration of the many possible causes of the relationship between field intubation and mortality is beyond the scope of this paper. Many studies have focused on improving field practice with the ultimate aim of reducing mortality. The present study is a retrospective observational study of the association of emergent intubation and mortality while controlling for a range of potential confounding variables associated with injury severity. We hypothesized that the excess of mortality among emergently intubated cases would be effectively eliminated when accounting for injury severity.

METHODS

Study Design, Setting and Data Collection

Data for this study were obtained from the University of Alabama Birmingham Injury Control Research Center (UAB-ICRC) as an ongoing, prospective, longitudinal study of persons with various injuries including cohort with traumatic brain injury (TBI). Medical record information was abstracted for all individuals admitted to any of eight participating hospitals in the central and northern Alabama area with TBI. From this database of potential participants, those who met the inclusion criteria were asked to participate in the longitudinal study. Criteria for inclusion in this study were: a) having sustained TBI between 1989 and 1992, b) residing and having been injured in Alabama, c) being at least 18 years of age when injured, and d) participating in regularly scheduled telephone follow-up interviews conducted by UAB-ICRC personnel.

Participants: Selection and Characteristics

All data used in this intubation outcome study were found and recorded during the initial hospital record abstraction performed during the recruitment period from 1989 to 1992. Missing data are not the result of longitudinal follow-up loss. The study base began with the 1,204 persons with TBI. Case identification was based on ICD-9 codes for skull fracture or intracranial injury (ICD-9 codes 800-803 and 851-854). Missing values on some variables (Head/Neck Abbreviated Injury Scale [n= 19], mortality status [n=31], and propensity score [n= 173]) caused the loss of 223 participants in the analysis of the relationship between intubation status and mortality. To determine if the remaining cohort of 981 participants was biased by the loss of these participants, we compared this group to the original sample of 1,204 on the outcome variable (death). All variables included in the logistic regression model are summarized in Table 2. There were no significant differences in the distributions of any of these variables between the original sample of 1,204 and the remaining cohort of 981 participants. The two groups also did not differ in the distribution of mortality outcome or gender.

Table 1 presents the demographic characteristics for this initial sample. Missing values accounted for the loss of some participants in the initial cohort, and Table 1 indicates the number of participants with valid data for each variable. Intubated cases were older, more seriously injured, and were more likely to have had a penetrating injury. The remaining variables associated with intubation status are components of the propensity score discussed below. Mortality outcome was known for 1,173 participants, and 216 (18.4%) died before hospital discharge.

Methods of Measurement

Each participant was assessed on the Head/Neck Abbreviated Injury Scale (H-AIS), with scores ranging from 1 (minor) to 6 (unsurvivable).[23-24] There were no cases of minor injury (H-AIS=1) or unsurvivable injury (H-AIS=6) in the database. Nineteen cases with H-AIS coded as missing were excluded from the primary analysis. Therefore, the effective range of H-AIS for this study was 2 (Moderate) to 5 (Critical). Mortality events were counted if death occurred before the time of regular discharge from the hospital**.**

Intubation status was collected from hospital admission records, but the abstract summary does not distinguish between field and emergency department intubations. Paramedics in Alabama at the time of the study did not then, and do not now, use neuromuscular blocking agents to permit intubation of difficult cases. Participants who were not at any time intubated during prehospital or emergency department care made up the 'not intubated' group (N= 636, 64.8%). This group also included any cases that were intubated following hospital admission because the intubation occurred outside the time frame under consideration. There were 345 (35.2%) intubated cases in the database. No data were collected on other less common forms of intubation, such as a temporary surgical airway.

Table 1. Comparison of Intubated and Not Intubated Groups on Demographic Variables, Selected Propensity Score Components, and Mortality

Variable	Intubated (n = 428) 35.6%	Not Intubated (n = 776) 64.4%	Significance Test[a]
Sex (n = 1204)			
Men	324 (37.2%)	548 (62.8%)	0.78, 0.59-1.01
Women	104 (31.3%)	228 (68.7%)	
Race (n = 1204)			
White	296 (35.7%)	532 (64.3%)	1.05, 0.81-1.4
Non-white	132 (35.1%)	244 (64.9%)	
Death (n = 1173)			
Yes	168 (77.8%)	48 (22.2%)	10.2, 7.2-14.5
No	244 (25.5%)	713 (74.5%)	
Marital Status (n = 1204)			
Single	184 (37%)	314 (63%)	
Married	152 (35.2%)	280 (64.8%)	$\chi^2 = 10.4$
Divorced	24 (27.9%)	62 (72.1%)	df = 5
Separated	3 (30%)	7 (70%)	
Widowed	18 (24.7%)	55 (75.3%)	$p = 0.06$
Unknown	47 (44.8%)	58 (55.2%)	
Age in Years (n = 1194)			
Mean Age (SD)	41 (19.6) (n = 776)	36.6 (17.5) (n = 428)	$t_{(968)} = 4.01$ $p < 0.0001$
Head Abbreviated Injury Scale (n = 1204)			
Moderate (2)	124 (27.6%)	326 (72.4%)	$\chi^2 = 137.1$ df = 4
Serious (3)	90 (25.9%)	257 (74.1%)	
Severe (4)	94 (38.5%)	161 (61.5%)	
Critical (5)	111 (77.1%)	33 (22.9%)	$p < 0.0001$
0 or Unknown	9 (47.4%)	10 (52.6%)	
Seizures (n = 1126)			
Yes	32 (62.8%)	19 (37.2%)	3.4, 1.9-6
No	358 (33.3%)	717 (66.7%)	
Cardiac Arrest (n = 1183)			
Yes	127 (70.9%)	52 (29.1%)	5.9, 4.2-8.4
No	293 (29.2%)	711 (70.8%)	
Skull Fracture (n = 1177)			
Yes	15 (20.5%)	58 (79.5%)	0.46, 0.26-0.83
No	397 (35.9%)	707 (64.1%)	
Injury Mechanism (n = 1196)			
Blunt	341 (32.6%)	706 (67.4%)	2.6, 1.8-3.7
Penetrating	83 (55.7%)	66 (44.3%)	
Intentional Injury (n = 1169)			
Yes	103 (42.9%)	137 (57.1%)	1.5, 1.1-2.0
No	314 (33.8%)	615 (66.2%)	

[a] Odds ratios and 95% confidence intervals are presented for dichotomous variables e.g., sex, race), Chi-square (χ^2) analysis was performed for variables with multiple categories (e.g., marital satus). A t-test for mean difference was performed for the variable 'age.'

Statistical Power

Power analysis calculations indicated that a logistic regression with a sample size of 981 achieves power of .91 at an alpha level of .05. This corresponds to an odds ratio (OR) of 1.7 for the association between intubation and death.

RESULTS

A simple bivariate analysis examining the relationship of intubation and mortality showed that the odds of dying before hospital discharge for those who were intubated were 14 times higher than the odds for those who were not intubated (OR = 14.3, 95% CI = 9.4 – 21.9). The next step was to determine if factors related to injury severity and age would eliminate this association. We constructed a multivariate logistic regression model to determine the impact of potential confounders on the association between intubation and mortality. The variables included in the model were H-AIS score (range =2-5), injury type (blunt vs. penetrating), systolic blood pressure (categorical), endotracheal intubation status, and a propensity score. These variables were chosen because they are frequently included in similar studies, were available in the database, and are known correlates of mortality.

We controlled for multiple potential confounding variables by the use of propensity scores.[2] Propensity scores are estimates of the conditional probability of receiving the treatment (intubation) given its relationship to potential confounding variables. The variables included in the creation of the propensity scores were selected because of their potential relationship to intubation status and mortality, as well as their relative completeness in the data set. The propensity score was developed using logistic regression with endotracheal intubation status as the outcome variable. The variables included in the propensity score model were race (white vs. nonwhite), intentionality (intentional vs. unintentional injury), alcohol use at the time of injury (present vs. absent), seizure activity (present vs. absent), skull

fracture (present vs. absent), and cardiac arrest (present vs. absent). Five variables included in the propensity score were significantly related to endotracheal intubation status in the multivariate analysis and were retained in the calculation of the propensity scores. Intentional injury (OR = 1.9, 95% CI = 1.3 - 2.7), seizure activity (OR = 3.9, 95% CI = 2.0 - 7.5), and cardiac arrest (OR = 8.4, 95% CI = 5.5 -12.8) were associated with an increased likelihood of intubation. Skull fracture (OR = 0.3, 95% CI = 0.18 - 0.68) was associated with a decreased likelihood of intubation.

Table 2 summarizes the results of the logistic regression model where the inclusion of covariates and the propensity score affects a downward adjustment on the risk of death associated with intubation as compared to the crude odds ratio. However, contrary to the hypothesis, the risk of death associated with intubation is not eliminated. The risk of death was almost six times greater for the intubated TBI patient (OR = 5.9, 95% CI =3.2 – 10.9). Significant covariates in the model include age, injury severity, systolic blood pressure, and the propensity score. Blunt trauma was associated with decreased mortality compared to penetrating injury. Greater age, lower systolic blood pressure (between 61 – 100 mm Hg), and H-AIS = 5 (critical) were all associated with greater mortality.

Table 2. Logistic Regression for Outcome Variable Death

Variable	Died (n = 173) 17.6%	Survived (n = 808) 82.4%	Adjusted odds ratio with 95% confidence interval
Intubation			
No	30 (4.7%)	606 (95.3%)	---
Yes	143 (41.5%)	202 (58.5%)	5.9, 3.2 - 10.9
Injury Mechanism			
Blunt	132 (15.2%)	739 (84.8%)	---
Penetrating	41 (37.3%)	69 (62.7%)	4.9, 2.1 - 11.5
Head Abbreviated Injury Scale			
Moderate (2)	36 (9.2%)	355 (90.8%)	---
Serious (3)	30 (10.4%)	259 (89.6%)	1.1, 0.5 - 2.3
Severe (4)	21 (11%)	170 (89%)	0.63, 0.3 - 1.6
Critical (5)	86 (78.2%)	24 (21.8%)	19.1, 8.7 - 41.9
Age in Years			
Mean Age (SD)	42.7 (20.8)	39.6 (18.5)	1.4, 1.2 - 1.6
Systolic Blood Pressure (mm Hg)			
>160	28 (21.2%)	104 (78.8%)	0.77, 0.3 - 2
141 - 160	23 (11.5%)	177 (88.5%)	0.6, 0.21 - 1.5
121 - 140	35 (12.4%)	248 (87.6%)	0.78, 0.33 - 1.8
101 - 120	31 (16.8%)	154 (83.2%)	---
81 - 100	21 (36.2%)	37 (63.8%)	3, .99 - 9.03
61 - 80	8 (42.1)	11 (57.9%)	5.9, 1.3 - 27.8
≤ 60	27 (26%)	77 (74%)	1.04, .4 - 2.7
Propensity Score	---	---	5.5, 4 - 7.5

DISCUSSION

We began with a simple odds ratio of 14.3 between intubation status and mortality. In a multivariate model adjusted for injury severity, the association drops to an almost six times increase in mortality among intubated patients. Clearly, accounting for a broad range of injury severity factors reduces, but does not eliminate, the mortality risk associated with intubation. Because many studies that control for the same injury severity factors have achieved similar results, the possibility that the results are due to inadequate statistical control of injury severity factors is unlikely. Before considering factors that may account for the association between emergent intubation and mortality, the relationship between mortality and H-AIS score deserves comment. In this study, the proportionate odds of death indicate that the critically injured group (H-AIS =5) accounted for most of the effect. There was no difference in risk of death among those with lesser degrees of injury (H-AIS = 2, 3, 4), but the odds of death were 19 times greater for the critically injured intubated cases than for those who were not intubated. The H-AIS alone obviously would not have provided adequate control for the effects of increasing injury severity in this model and did not optimally stratify the mortality risk of patients with TBI.

Batchelor et al.[24] indicated that it is not yet possible to determine which of several systems developed after the H-AIS is superior because of limitations in current methods used to assess goodness-of-fit (eg, Hosmer-Lemeshow statistic). Nevertheless, trauma-scoring models such as International Classification of Diseases Injury Severity Score (ICISS) have shown promise, especially when combined with age and other trauma measures. Although much work remains in the classification of trauma and prediction of outcomes, it is also clear that the field is advancing beyond the H-AIS and Injury Severity Score (ISS) models. Better systems are available, but it will take time for registries to change over to these systems and to develop sufficient numbers of cases for analysis.

The lack of a neuromuscular blockade protocol for ground emergency medical technicians (EMTs) or emergency physicians in Alabama during the recruitment phase of this study means that every intubated case, regardless of H-AIS, would have had a blunted gag reflex, itself an indicator of serious injury. Those who were intubated in the moderate, serious, and severe injury groups were probably more seriously injured than the H-AIS suggests. Mortality increased only slightly among the injury severity groups until a dramatic increase was observed for the critically injured group. Similar patterns of the relationship of H-AIS

and mortality have been reported elsewhere.[23,25]

Even if the H-AIS were a better predictor of mortality, it could not inform paramedic practice because it is determined long after the initial presentation observed by the EMT in the field. Further, along with Wang et al.[2] and others, our study relied on the ICD-9 code to include patients. This information is also unavailable to the paramedic in the field. Unless all patients who meet criteria for intubation based on field observations, and not just those with ICD-9 codes consistent with TBI, are found to have increased morbidity and mortality when intubated emergently, it will remain difficult for the EMS community to significantly change practices based on these results, however compelling.

Returning to other factors that may account for the increased mortality among intubated cases, it is suggested that factors prompting a decision to intubate should be included in future studies. The guidelines for intubation, as well as the observations and evaluations of the emergency responder, should be carefully studied as a means of improving criteria useful in rapidly assessing the need for intubation. A framework for understanding the cognitive processing demands on paramedics confronting potential intubation cases has been proposed.[26] Their model highlights the complex host of interacting variables that enter into the decision to intubate. Algorithms that standardize and inform practices are essential in approaching relatively uncomplicated cases, but probably would not extend to ambiguous cases that send conflicting signals to the paramedic about the advisability of intubation.[27]

Interest in cognitive processes indicates a shift in research emphasis away from post-intubation factors that affect mortality (i.e., injury severity) and toward pre-intubation factors that influence the decision to intubate at the scene. A small focus group study of paramedics indicated that paramedics view intubation as a key component of their professional identity, suggesting that it is a highly valued professional activity.[28] Thus, paramedics who place a high value on intubation may be more likely to intubate hopeless and unsalvageable cases. The emergency responder who encounters very seriously injured cases recognizes that many will die regardless of treatment. Under these circumstances, there is a very low risk of further harm while there may be a very large potential benefit if an airway is established and the person survives. There is no comparable set of circumstances at the lower end of the injury severity spectrum, so the most seriously injured, and perhaps a very large proportion of the mortally injured, would receive endotracheal intubation. Bulger et al.[16] reported that 95% of the severe TBI cases in their study were intubated, a proportion high enough to contain more than a few cases beyond rescue. The key to understanding the relationship between intubation and mortality is in the appearance of the case to the emergency responder at the time the decision to intubate is made. Despite the large number of studies that have been conducted, calls for the restriction or elimination of field intubation seem premature given that research into on-the-scene decision-making processes is beginning to emerge.[29]

LIMITATIONS

This study has the drawbacks of a retrospective study on an existing dataset and cannot be used to imply causality. Our data set only confirms emergent intubation status by the time of hospital admission. An unknown number of these cases occurred in the field, and some undoubtedly occurred in the ED. We do not have the ability to extract the site of intubation, the number of attempts, the body habitus of the patient or more detail about the causes of morbidity or mortality from the database. Because data were collected from multiple hospitals, a retrospective review of the charts from as many as 17 years ago at all of these hospitals is prohibitive. Moreover, these data were collected in Alabama where rapid sequence intubation (RSI) protocols are not in use by ground EMT services. The most recent research shows that in 2004-2005, less than 12% of Alabama EMTs licensed to perform intubation actually did so during the year. Of the almost 1400 intubations performed in the field, about 92% required more than one attempt.[30] Thus, current intubation success rates in Alabama are low, and it is difficult to imagine that field performance was any better when our data were collected.

Several investigators have reported that hyperventilation is a significant contributor to increased mortality noted in patients with TBI who are intubated. Because prehospital and the ED patients in our database were all hyperventilated by protocol this remains an important potential confounder in our data set. Patients intubated in the field are still hyperventilated at least 70% of the time, even though it is no longer part of the regular treatment protocol.[31]

CONCLUSION

Results of this study indicate that emergent intubation is associated with an increased risk of death even after controlling for potential confounders related to injury severity. Future research that combines factors that influence patient selection with injury severity measures will more adequately determine the degree of relationship between emergent intubation and mortality. The effectiveness of field and emergent intubation should ultimately determine if this practice continues to have a place in the paramedic armamentarium.

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Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources, and financial or management relationships that could be perceived as potential sources of bias.

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REFERENCES

1. Davis DP, Peay J, Sise MJ, et al. The impact of prehospital endotracheal intubation on outcome in moderate to severe traumatic brain injury. *J Trauma.* 2005; 58:933-939.
2. Wang, HE, Peitzman, AB, Cassidy, LD, Adelson PD, Yealy DM. Out-of-hospital endotracheal intubation and outcome after traumatic brain. *Ann Emerg Med.* 2004; 44:439-450.
3. Bocchichio GV, Ilahi O, Joshi M, Bocchichio K, Scalia TM. Endotracheal intubation in the field does not improve outcome in trauma patients who present without an acutely lethal traumatic brain injury. *J Trauma.* 2003; 54:307-311.
4. Arbabi S, Jurkovich GJ, Wahl WL, et al. A comparison of prehospital and hospital data in trauma patients *J Trauma.* 2004; 56:1029-1032.
5. Winchell RJ, Hoyt DB. Endotracheal intubation in the field improves survival in patients with severe head injury. *Arch Surg.* 1997; 132:592-597.
6. Floccare DJ, Cushman JT. Endotracheal intubation in the field: Pro position. *Hosp Med.* 2005; 66; 89-91.
7. Wang HE, Lave JR, Sirio CA, Yealy DM. Paramedic intubation errors: Isolated events or symptoms of a larger problem? *Health Affairs.* 2006; 25:501-509.
8. Wang HE, Kupas DF, Paris PM, Bates RR, Costantino JP, Yealy DM. Multivariate predictors of failed prehospital intubation. *Acad Emerg Med*. 2003; 10:717-724.
9. Silvestri S, Ralls GA, Krauss B, et al. The effectiveness of out-of-hospital use of continuous end-tidal carbon dioxide monitoring on the rate of unrecognized misplaced intubation within a regional emergency medical services system. *Ann Emeg Med.* 2005; 45:497-503.
10. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. *Ann Emeg Med.* 2001; 37:32-37.
11. Murray JA, Demetriades D, Berne TV et al. Prehospital intubation in patients with severe head injury. *J Trauma.* 2000; 49:1065-1070.
12. Silverstone P. Pulse oximetry at the roadside: A study of pulse oximetry in immediate care. *Br Med J.* 1989; 298:711-13.
13. Sloane C., Vilke GM, Chan TC, et al. Rapid sequence intubation in the field versus hospital in trauma patients. *J Emerg Med.* 2000; 19:443-444.
14. Cooper A, Discala C, Foltin G. Pre-hospital endotracheal intubation for severe head injury in children: A reappraisal. *Sem Pediatr Surg.* 2001; 10:3-6.
15. Bulger EM, Copass MK, Maier RV, Larsen J, Knowles J, Jurkovich GJ. An analysis of advanced prehospital airway management. *J Emerg Med. 2002* 23:183-89.
16. Bulger EM, Copass MK, Sabath D, Maier RV, Jurkovich GJ. The use of Neuromuscular Blocking Agents to Facilitate Prehospital Intubation does not Impair Outcome following Traumatic Brain Injury. *J Trauma*. 2005; 58:718-24.
17. Wang HE, Yealy DM. Out-of-hospital rapid sequence intubation: Is this the "success" we envisioned? *Ann Emerg Med.* 2002; 40:168-171.
18. Wang HE, Davis DP, O'Connor RE, Domeier RM. Drug-assisted intubation in the prehospital setting (Resource document to the NAEMSP position statement). *Prehosp Emerg Care.* 2006; 10:261-271.
19. Davis DP. Prehospital intubation of brain-injured patients. *Curr Opin Crit Care.* 2008; 14:142-148.
20. Stocchetti N, Maas AIR, Chieregato A, van der Plas AA. Hyperventilation in head injury: A review. *Chest.* 2005; 127:1812-1827.
21. Ghajar J. Traumatic brain injury. *The Lancet*. 2000; 256:923-929.
22. Chestnut RM. Care of Central Nervous System Injuries. *Surg Clin North Am*. 2007; 87:119-156.
23. Demetriades D, Kuncir E, Murray J, Velmahos GC, Rhee P, Chan L. Mortality prediction of Head Abbreviated Injury Score and Glasgow Coma Scale: Analysis of 7,764 head injuries. *J Am Coll Surg.* 2004; 199:216-222.
24. Batchelor JS, McGuinness A, Ryan J. Adult trauma scoring systems: A critical review. *Trauma.* 2001; 3:175-180.
25. Gennarelli TA, Champion HR, Copes WS, Sacco WJ. Comparison of mortality, morbidity, and severity of 59,713 head injured patients with 114,447 patients with extracranial injuries. *J Trauma.* 1994; 37:962-968.
26. Wang HE, Katz S. Cognitive control and prehospital endotracheal intubation. *Prehosp Emerg Care*. 2007; 11:234-239.
27. Wang HE, Kupas DF, Greenwood MJ, Pinchalk ME, Mullins T, Gluckman W, Sweeney TA, Hostler D. An algorithmic approach to prehospital airway management. *Prehosp Emerg Care*. 2005; 9:145-155.
28. Thomas JB, Abo BN, Wang HE. Paramedic perceptions of challenges in out-of-hospital endotracheal intubation. *Prehosp Emerg Care.* 2007; 11:219-223.
29. Zink BJ, Maio RF. Out-of-hospital endotracheal intubation in traumatic brain injury: Outcomes research provides us with an unexpected outcome. *Ann Emeg Med.* 2004; 44:451-453.
30. Stephens SW, Brown TB. Field intubation by out-of-hospital rescuers: The Alabama experience. *Acad Emerg Med* Suppl. 1 2006; 13:S85.
31. Warner KJ, Bulger EM. Does pre-hospital ventilation effect outcome after significant brain injury? *Trauma.* 2007; 9:283-289.

Delayed Complications of Emergency Airway Management: A Study of 533 Emergency Department Intubations

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Objectives: Airway management is a critical procedure performed frequently in emergency departments (EDs). Previous studies have evaluated the complications associated with this procedure but have focused only on the immediate complications. The purpose of this study is to determine the incidence and nature of delayed complications of tracheal intubation performed in the ED at an academic center where intubations are performed by emergency physicians (EPs).

Methods: All tracheal intubations performed in the ED over a one-year period were identified; 540 tracheal intubations were performed during the study period. Of these, 523 charts (96.9%) were available for review and were retrospectively examined. Using a structured datasheet, delayed complications occurring within seven days of intubation were abstracted from the medical record. Charts were scrutinized for the following complications: acute myocardial infarction (MI), stroke, airway trauma from the intubation, and new respiratory infections. An additional 30 consecutive intubations were examined for the same complications in a prospective arm over a 29-day period.

Results: The overall success rate for tracheal intubation in the entire study group was 99.3% (549/553). Three patients who could not be orally intubated underwent emergent cricothyrotomy. Thus, the airway was successfully secured in 99.8% (552/553) of the patients requiring intubation. One patient, a seven-month-old infant, had unanticipated subglottic stenosis and could not be intubated by the emergency medicine attending or the anesthesiology attending. The patient was mask ventilated and was transported to the operating room for an emergent tracheotomy. Thirty-four patients (6.2% [95% CI 4.3 – 8.5%]) developed a new respiratory infection within seven days of intubation. Only 18 patients (3.3% [95% CI 1.9 – 5.1%]) had evidence of a new respiratory infection within 48 hours, indicating possible aspiration pneumonia secondary to airway management. Three patients (0.5% [95% CI 0.1 – 1.6%]) suffered an acute MI, but none appeared to be related to the intubation. One patient was having an acute MI at the time of intubation and the other two patients had MIs more than 24 hours after the intubation. No patient suffered a stroke (0% [95% CI 0 – 0.6%]). No patients suffered any serious airway trauma such as a laryngeal or vocal cord injury.

Conclusions: Emergency tracheal intubation in the ED is associated with an extremely high success rate and a very low rate of delayed complications. Complication rates identified in this study compare favorably to reports of emergency intubations in other hospital settings. Tracheal intubation can safely be performed by trained EPs.
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INTRODUCTION

Tracheal intubation is a critical, often lifesaving, procedure performed frequently in emergency departments (EDs) across the world. Due to the often critical condition that patients requiring intubation present with, the nature and incidence of potential complications is important to consider. Several studies have reported immediate complications associated with ED intubations.[1-4] These studies, with the exception of rare case reports, have focused only on complications apparent during the ED visit. In the same teaching hospital ED, we previously reported a series of 610 consecutive intubations that occurred over a one-year period to determine the type and incidence of immediate complications.[5] We found an immediate complication rate of 8.0%, most of which were minor. Tayal et al.[6] also studied intubations in an emergency medicine (EM) training program and found an immediate complication rate of 3.4%. Immediate complications that have been consistently identified include esophageal intubation, mainstem bronchus intubation, aspiration, oxygen desaturation and hypotension. Unfortunately, emergency airway management potentially can result in very serious complications that are not immediately apparent or recognized in the ED. For example, significant cardiac, neurological and respiratory complications may occur but may not become evident until several days later in the intensive care unit (ICU).[7-10] The objective of this study is to determine the incidence and nature of delayed complications of tracheal intubation performed in the ED of a teaching hospital where intubations are performed by emergency physicians (EPs).

METHODS

Study Design

This was a one-year retrospective study of all intubated ED patients with an accompanying prospective 30 consecutive intubation cohort study. All patients intubated were eligible for enrollment. Structured data forms were cross-referenced to professional billing services to ensure a 100% capture rate. In the retrospective arm, all patients intubated in the ED during the academic year from July 01, 1997 to June 30, 1998 were included in the study and for the prospective arm all patients intubated in the one month period from January 03, 1999 to January 31, 1999 were included.

Study Setting

The University of California, Davis, Medical Center is an urban Level 1 Trauma Center with an annual ED census of approximately 60,000 patients. The ED is staffed full-time with EM residents and attending physicians. Approximately 30% of all patients arrive by ground ambulance or aeromedical transport. About 300 patients per year arrive by ambulance already intubated. These patients were not included in the study unless they required reintubation in the ED. The overall acuity of patients is high; 28% presenting to the ED are admitted to the hospital. Roughly half of all ED intubations are performed on patients with medical disorders and half on patients with trauma. Nearly all of these intubations are conducted by EM residents supervised by EM faculty. All intubations are entered into an airway registry with standardized data entry.

Airway management at the institution is the ultimate responsibility of the attending EP, who determines which resident will perform the intubation and what technique is used. The senior resident (third year post-graduate resident) usually manages all the trauma intubations and the most difficult medical airways. Routine medical intubations are typically managed by the second-year resident, and occasionally by the first-year resident. Rarely, medical students and residents from other specialties intubate patients under the direct supervision of the attending physician.

Airway equipment includes conventional straight and curved blade laryngoscopes. A cart with additional airway equipment is available for difficult airways; however, this study was performed before the introduction of videolaryngoscopes. The majority of patients in need of intubation undergo rapid sequence intubation (RSI). If they are considered a difficult airway, they undergo awake intubation without the use of a paralytic agent. Succinylcholine, vecuronium, and rocuronium are the immediately available neuromuscular blocking agents for RSI. Etomidate, midazolam and ketamine are the readily available sedative-hypnotic agents. For all intubations, patients are placed on a cardiac monitor, an automated non-invasive blood pressure monitor and a pulse oximeter. Patients are routinely preoxygenated prior to intubation attempts. Bag-valve mask ventilation is avoided before intubation unless necessitated by hypoxemia or inadequate ventilation. All patients undergoing oral intubation have cricoid pressure applied to prevent passive regurgitation and minimize gastric distension. All patients with blunt trauma are intubated with cervical spine in-line cervical stabilization maintained throughout the procedure. Tracheal intubation is confirmed by colorimetric end-tidal carbon dioxide detection in patients with a pulse. An aspiration syringe esophageal detector device is also used if the patient is in cardiac arrest. Auscultation of the chest is performed and a chest radiograph is obtained after intubation to determine the position of the tip of the endotracheal tube.

Residents receive a structured curriculum on airway management. The technique of surgical cricothyrotomy is also taught, and all EM interns attend a procedure lab and perform cricothyrotomies on cadavers under the supervision of an attending physician. The intern does a mandatory one-month rotation in anesthesia during the first year and performs approximately 50 intubations on stable patients in the operating room. During the second year, residents expand upon their airway experience by performing intubations in the ED, in the ICU, and in the prehospital setting during their EMS rotation. During their last year, each senior resident

Table 1. Definitions of Delayed Complications

Complication	Definition
Acute Myocardial Infarction	ECG or enzyme evidence*
Acute Stroke	CT scan indicative of ischemic changes or physician note indicating the diagnosis of a new stroke
Respiratory Infection	CXR indicating a new infiltrate or positive sputum culture or physician note indicating the diagnosis of a new pneumonia
Airway Trauma	Physician note indicating the presence of airway trauma

* **The Joint European Society of Cardiology/American College of Cardiology Committee.** Myocardial infarction redefined - a consensus document of the joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction. *Eur Heart J* 2000; 21:150-13 (Also published in *J Am Coll Cardiol* 2000; 36:959-69.

Table 2. Demographics of all patients intubated (N = 553)

Characteristic	No. of Patients	% of Patients
Average Age = 46 (3 months - 99 years)		
0 - 5 years	24	4.3%
6 - 16 years	30	5.4%
17 - 60 years	340	61.5%
> 60 years	159	28.8%
Race		
White	305	55.2%
Black	111	20.1%
Hispanic	62	11.2%
Pacific Islander	7	1.3%
Asian	38	6.9%
Other	24	4.3%
Unknown	6	1.1%
Sex		
Female	190	34.4%
Male	363	65.6%

performs an average of 40 difficult intubations on trauma patients and unstable medical patients.

Data Collection and Analysis

A structured data collection form was developed prior to the collection of both retrospective and prospective data. It was first pilot tested and refined prior to use in the study. Information collected included demographic data, past medical history, prior history of RSI in the previous six months, and the induction agent used (if any).

Delayed complications were pre-determined to fall into four major categories: cardiac, neurologic, respiratory and airway. An attempt was made to identify patients that suffered from an acute myocardial infarction, an acute stroke, aspiration pneumonia or upper airway trauma. Complications that occurred within seven days of intubation were considered *potentially* related to the intubation. Complications that occurred within two days of intubation were considered as *possibly* related. All medical records, including radiology reports, culture results, and progress notes were reviewed to seek evidence of complications. The time of onset of an infection was determined by when the earliest sign of infection became apparent. Definitions for each of the variables that could have subjective interpretation were prospectively developed and are summarized in Table 1. All data collection was by a single abstractor, with quality review of selected charts by the senior author.

For the retrospective portion of the study, all patients intubated in the ED from July 01, 1997 to June 30, 1998 were identified by using the airway registry database of patients at our institution and the database from the professional billing service. For the prospective portion of the study, all patients intubated in the ED from January 03-31, 1999 were identified within 24 hours of intubation. These patients were then prospectively followed as inpatients for seven days to identify any complications. All aspects of the medical record were reviewed including initial demographic data, past medical history, past steroid use history, peri-intubation vital signs, medication use and potential complications. If the patient was discharged prior to seven days, the medical record was obtained to evaluate for any further visits within the seven-day period. Data from the prospective arm of the study was used to validate the complication model created from the retrospective arm.

Summary descriptive statistics were performed. For selected complication rates, 95% confidence intervals were performed using Stata statistical software (Release 6, Stata Corporation, 1999). This study was approved by the University of California, Davis Institutional Review Board (IRB).

RESULTS

During the retrospective study period, 540 tracheal intubations were performed in the ED. Of these, 523 (96.9%), were included in the study. The medical record could not be obtained for the remaining 17. For the prospective arm of the

study, 30 intubations were identified and had data collection forms completed. The total number of intubations with data forms in both the retrospective and prospective groups is 553 (entire study group). Table 2 summarizes the demographics of the study patients.

The overall success rate for ED endotracheal intubation was 99.8%. Only one patient (0.02%) could not be successfully intubated. This patient was a seven-month-old infant with subglottic stenosis. He was mask ventilated and went to the operating room for an emergent tracheostomy. Three other patients required surgical management of the airway by cricothyrotomy in the ED. All were successful. Eighty patients (14.5%) were intubated without the use of sedative-hypnotic agents, 453 patients (81.9%) received etomidate, 10 patients (1.8%) were induced with midazolam, one (0.2%) with lorazepam, one (0.2%) with fentanyl, five (0.9%) with ketamine, and in three (0.5%) the induction agent is unknown.

Table 3 summarizes the long-term complications for the entire study group. Thirty-four patients (6.2%) developed a new respiratory infection within seven days of intubation and determined to be *potentially* related to the intubation. This does not include patients who were admitted with a diagnosis of pneumonia. In the prospective group, three patients (10%) developed a new respiratory infection. Eighteen patients (3.3%) for the entire study group and two (6.7%) for the prospective group developed respiratory infections that became apparent within forty-eight hours *possibly* related to the ED intubation. For the entire study group, no patient suffered a cerebrovascular accident, but three patients (0.5%) suffered an acute myocardial infarction within seven days of intubation. None of these patients was in the prospective group. Only one patient ruled in for a myocardial infarction within 48 hours of intubation without cardiac symptoms closely antecedent to the intubation. No patient had evidence of major airway trauma (defined as a traumatic injury to the pharynx, larynx, vocal cords or trachea).

DISCUSSION

Several large prospective studies have reported their rates of successful intubation and acute complications with ED intubation.[5,11] These studies have concluded that RSI in the hands of trained EPs is a safe and effective procedure with a low rate of immediate complications. EP rates of complications compare favorably to those done by anesthesiologists or critical care medicine attendings.[12,13] This study confirms that endotracheal intubation by EPs is a highly successful procedure. Only one patient (0.2%) experienced failed airway management in the ED.

Prior studies, with the exception of scattered case reports, have not examined the rate of long-term or non-immediate complications from ED intubations by EPs.[3,7-10,14,15] We found that long-term complications, as defined as occurring within seven days, are also uncommon. This study found a low incidence of new respiratory infections within the first seven days (6.2%). The rate of new respiratory infections occurring within 48 hours and more likely to be intubation related (aspiration) was only 3.3%. Our results are also similar to another study of emergent intubations outside the operating room.[2] In that study of 297 patients intubated in an ICU, 12 (4%) were felt to have probably suffered an aspiration during airway management.

Three of our study patients suffered myocardial infarctions within seven days of intubation, but none were felt to be associated with the intubation. One patient was likely having an infarct at the time of intubation. He expired within 24 hours of intubation. One patient was intubated secondary to severe blunt trauma. His infarction occurred five days after intubation and is likely unrelated. The last patient had a myocardial infarction between 24 and 48 hours of intubation. His electrocardiogram did not become ischemic until more than 24 hours after intubation. Therefore, this is also unlikely to be secondary to intubation. No cases of major airway trauma were found in either the retrospective or prospective arms of the study.

LIMITATIONS

One study limitation is that the majority of data is obtained from a retrospective review of registry data and is dependent upon the quality of registry data entry and also medical chart documentation. It is possible that not all long-term complications were recorded in the progress notes or discharge summary, or that some were missed by the data abstractor. For these reasons, a prospective arm of the study was included to try for a validation of the rate of complications. The rate and pattern of complications were very similar in both arms of the study. This supports the validity of the larger retrospective data set. Another limitation is that these data are 10 years old. However, emergency

Table 3. Delayed Complications of Intubation

Complication	Prospective (N = 30)	All Patients (N = 553)	95% CI's
New Respiratory Infection			
(≤ 7 days)	3 (10.0%)	34 (6.2%)	4.3 - 8.5%
(≤ 2 days)	2 (6.7%)	18 (3.3%)	1.9 - 5.1%
Acute MI			
(≤ 7 days)	0	3 (0.5%)	0.01 - 1.6%
(≤ 2 days)	0	1 (0.2%)	
Acute Stroke	0	0	0 - 0.6%
Airway Trauma	0	0	0 - 0.6%

airway management practices, with the exception of the recent use of videolaryngoscopes, have changed little over the last decade. Most patients still undergo direct laryngoscopy in the ED and receive the commonly used RSI drugs succinylcholine and etomidate.

CONCLUSION

In summary, we found that tracheal intubation was performed in our ED with an extremely high success rate and is associated with a very low rate of long-term complications. These rates compare favorably to reports of emergency intubations in other settings. This further supports the safety of rapid sequence intubation, and emergent tracheal intubation by EPs.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources, and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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REFERENCES

1. Talucci RC, Shaikh KA, Schwab CW. Rapid sequence induction with oral endotracheal intubation in the multiply injured patient. *Am Surgeon*. 1988; 54:185-187.
2. Schwartz DE, Matthay MA, Cohen NH. Death and other complications of emergency airway management in critically ill adults. A prospective investigation of 297 tracheal intubations. *Anesthesiology*. 1995; 82:367-376.
3. Ooi GC, Irwin MG, Lam LK, Cheng SW. An unusual complication of emergency tracheal intubation. *Anaesthesia*. 1997; 52:154-158.
4. Slobodkin D, Topliff S, Raife JH. Retrograde intubation of the pharynx: an unusual complication of emergency cricothyrotomy. *Ann Emerg Med*. 1992; 21:220-222
5. Sakles JC, Laurin EG, Rantapaa AA, Panacek EA. Airway management in the emergency department: a one-year study of 610 tracheal intubations. *Ann Emerg Med*. 1998; 31:325-332.
6. Taryle DA, Chandler JE, Good JT, Jr., Potts DE, Sahn SA. Emergency room intubations--complications and survival. *Chest*. 1979; 75:541-543.
7. Fan CM, Ko PC, Tsai KC, Chiang WC, Chang YC, Chen WJ, Yuan A. Tracheal rupture complicating emergent endotracheal intubation. *Am J Emerg Med*. 2004; 22:289-293
8. Besmer I, Schupfer G, Stulz P, John M. [Tracheal rupture: delayed diagnosis with endobronchial intubation]. *Anaesthesist*. 2001; 50:167-170.
9. Miller JS, Itani KM, Oza MD, Wall MJ. Gastric rupture with tension pneumoperitoneum: a complication of difficult endotracheal intubation. *Ann Emerg Med*. 1997; 30:343-346.
10. Gray B, Huggins NJ, Hirsch N. An unusual complication of tracheal intubation. *Anaesthesia*. 1990; 45:558-560.
11. Tayal VS, Riggs RW, Marx JA, Tomaszewski CA, Schneider RE. Rapid-sequence intubation at an emergency medicine residency: success rate and adverse events during a two-year period. *Acad Emerg Med*. 1999; 6:31-37.
12. Ma OJ, Bentley B, II, Debehnke DJ. Airway management practices in emergency medicine residencies. *Am J Emerg Med*. 1995; 13:501-504.
13. Kovacs G, Law JA, Ross J, Tallon J, MacQuarrie K, Petrie D, Campbell S, Soder C. Acute airway management in the emergency department by non-anesthesiologists. *Can J Anaesth*. 2004; 51:174-180.
14. Harris R, Joseph A. Acute tracheal rupture related to endotracheal intubation: case report. *J Emerg Med*. 2000; 18:35-39.
15. Moschini V, Losappio S, Dabrowska D, Iorno V. Tracheal rupture after tracheal intubation: effectiveness of conservative treatment. *Minerva Anestesiol*. 2006; 72:1007-1012.

The Effect of Single-Bolus Etomidate on Septic Patient Mortality: A Retrospective Review

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Background: Because of its many desirable properties, etomidate is widely used as an induction agent for endotracheal intubation. However, some have recently called into question the safety of etomidate for even single-bolus use due to its known effects on adrenal suppression.

Objectives: We sought to compare the in-hospital mortality between septic patients given etomidate and those given alternative induction agents for intubation.

Methods: We performed a retrospective chart review of intubated septic patients treated in our hospital. We collected data from patients over the age of 18 with sepsis who were intubated in the pre-hospital setting, in our emergency department, or on the wards of our hospital, and calculated the in-hospital mortality of each group.

Results: We identified 181 patients with sepsis who were intubated over the study period; 135 received etomidate and 46 received alternative agents or no induction agent. Baseline characteristics, vital signs, and laboratory values were similar between the two groups. Of the 46 patients receiving alternative agents or no agent, 18 died, yielding an unadjusted mortality of 39.1% (95% CI 25.5% to 54.6%), while of the 135 patients receiving etomidate, 63 died, for an unadjusted mortality of 46.7% (95% CI 38.1% to 55.4%), *P*=0.38.

Conclusion: We found a non-statistically significant 7.6% absolute increase in mortality in patients given etomidate in our small-sized study population.
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INTRODUCTION

Use of the drug etomidate for continuous sedation in mechanically ventilated patients was found to have detrimental effects on patient mortality shortly after its introduction, presumably because of its measurable adrenal suppression. The package insert now cautions against prolonged infusion, citing the "hazards of prolonged suppression of endogenous cortisol and aldosterone production" (Bedford Laboratories, Bedford, OH).[1-5] Several authors have recently called into question the safety of etomidate for even single-bolus use, citing its demonstrated capacity to cause relative adrenocortical insufficiency after a single bolus.[6-14] Additionally, a recent retrospective analysis suggests an association of etomidate with worse outcomes in septic patients.[15] Nevertheless, no rigorous, prospective, randomized study has demonstrated a clinically significant adverse outcome from either continuous or single-dose use of etomidate.

In the emergency department (ED), etomidate is widely used as an induction agent for endotracheal intubation

Table 1. Demographics and medical history*

	Etomidate N = 135		No Etomidate N = 46		
	ED N = 120 (%)	Floor N = 15 (%)	ED N = 16 (%)	Floor N = 25 (%)	Field N = 5 (%)
Median Age, years (IQR)	77 (62-83)	69 (61-82)	80 (76-83)	72 (56-76)	81 (63-93)
Gender, % Male	49	60	50	40	80
Diabetes	43 (36)	9 (60)	5 (31)	11 (44)	1 (20)
Coronary Artery Disease	39 (33)	7 (47)	8 (50)	5 (20)	2 (40)
Hypertension	68 (57)	11 (73)	10 (63)	15 (60)	4 (80)
Congestive Heart Failure	37 (31)	6 (40)	6 (38)	8 (32)	1 (20)
COPD	25 (21)	2 (13)	3 (19)	7 (28)	1 (20)
Nursing Home Residence	64 (53)	10 (67)	6 (38)	15 (60)	2 (40)
Immunosuppression	5 (4)	2 (13)	2 (13)	4 (16)	0 (0)
Hemodialysis	9 (8)	1 (7)	1 (6)	4 (16)	0 (0)
Cirrhosis	4 (3)	0 (0)	0 (0)	3 (12)	0 (0)

ED, Emergency Department; *COPD*, Chronic obstructive pulmonary disease
Immunosuppression includes patients undergoing chemotherapy, radiation, or chronic steroid use, and those diagnosed with leukemia, lymphoma, or AIDS.
* Number and percentages are given unless otherwise indicated.

because it allows for a rapid, smooth, hemodynamically stable intubation.[16,17] The implication that etomidate may increase mortality, even with a single dose, could have widespread effects given the large number of patients who receive this medication.

We sought to determine the mortality difference between patients with sepsis given etomidate and patients with sepsis given alternative induction agents by retrospectively reviewing the outcome of intubated septic patients treated in our hospital.

METHODS

Study Design, Setting, and Selection of Participants

This was a retrospective cohort study utilizing a chart review of intubated septic patients. The study was performed at a tertiary-care suburban community hospital with an annual ED census of almost 85,000 patients. We collected data from patients over the age of 18 with sepsis who were intubated in the prehospital setting, in our ED, or on the wards of our hospital over a four-year period from December 2002 to September 2006. Intubation may have occurred at any point during the patient's hospital stay or in the field prior to arrival in the ED. Therefore, intubation was performed by paramedics, emergency physicians, house staff physicians, or anesthesiologists. The study was approved by the hospital's Institutional Review Board with a waiver of informed consent.

Methods of Measurement

We created a standardized abstraction form using Microsoft Excel for recording the patient's name, age, gender, medical record number, induction agent, location and time of intubation, admit, discharge date and time, whether or not steroids were given, and whether the patient lived or died. We trained the additional abstractors in its use through individual education about location of the data in the chart. However, we were unable to blind abstractors to the study objective or to patient assignment.

We identified patients by searching the ED electronic medical record database and then confirming the diagnosis of sepsis in the inpatient electronic medical record database. We searched the terms "sepsis" and "septic" in the diagnosis field of the ED records to identify patients in the ED diagnosed as septic, and reviewed these charts to determine which patients had been intubated (either in the field or in the ED). We then searched the ED records for all patients intubated in the ED, in order to find patients who had sepsis by standard criteria but were given alternative diagnoses in the ED record (such as pneumonia, respiratory failure, hypotension, or urinary tract infection). Finally, all charts were checked against electronic hospital discharge records to ensure that sepsis was a discharge diagnosis. Patients found to have had more than one admission for sepsis were considered as independent admissions. Abstractors were given printed lists of identified patient medical record numbers from which to obtain data from our ED and inpatient records. Abstraction forms were returned to a study coordinator and entered into a master Excel spreadsheet.

We recorded into the abstraction forms times of admission, intubation, discharge, status at discharge, all intubation medications used, any use of supplemental steroids

Table 2. Vital signs and laboratory values*

	Etomidate				No Etomidate			
	Mean	**95% CI**	**Median**	**IQR**	**Mean**	**95% CI**	**Median**	**IQR**
Mean Arterial Pressure (mm Hg)	75	70 - 80	74	33	74	66 - 82	75	27
Temperature (°C)	37.2	36.8 - 37.5	37.2	1.8	36.8	36.4 - 37.2	36.8	1.1
Heart Rate (beats/min)	109	105 - 114	110	30	103	92 - 114	100	48
Serum:								
White Blood Cells (k/μL)	17.7	15.4 - 20.0	14.3	11.7	15.1	12.8 - 17.4	14.8	9.2
Hematocrit (%)	36	34 - 38	37	12	34	30 - 37	37	14
Sodium (mmol/L)	140	139 - 142	139	8	140	138 - 142	139	11
Potassium (mmol/L)	4.8	4.1 - 5.4	4.3	1.2	4.6	4.3 - 4.9	4.4	1.4
Bicarbonate (mmol/L)	20	19 - 22	20	10	25	19 - 31	22	14
Creatinine (mg/dL)	3.1	2.2 - 3.9	1.9	2.2	2.1	1.6 - 2.6	1.7	1.9
pH	7.3	7.28 - 7.33	7.34	0.22	7.26	7.21 - 7.31	7.29	0.32
Lactate (mmol/L)	3.7	3.2 - 4.2	3.1	3	3.9	2.7 - 5.1	2.3	4

IQR, Interquartile range
* Data obtained from times closest to time of intubation.

either in the ED or after admission, as well as laboratory values, vital signs, and co-morbidities. Our primary outcome was the mortality difference between patients intubated with the use of etomidate versus patients intubated using any other, or no, induction agent. We selected 10% of our charts for review and testing of interrater reliability on the variables of etomidate use, steroid use, discharge condition (dead or alive) and time of intubation.

Primary Data Analysis

Baseline demographic and clinical characteristics are described using means with 95% confidence intervals and medians with interquartile ranges (IQRs). We calculated the mortality of each of the two groups with 95% confidence intervals using the Wilson method with continuity correction (VassarStats, http://faculty.vassar.edu/lowry/VassarStats.html). We compared the unadjusted mortality between the two cohorts with a χ^2 test and performed survival analysis with Kaplan-Meier curves, using the Mantel-Cox Log Rank test to compare the two groups. We considered values of $P < 0.05$ to be statistically significant for all analyses. Analyses were performed using SPSS version 15.0 (SPSS Inc., Chicago, IL).

RESULTS

We identified 181 patients with sepsis who were intubated over the study period. Baseline characteristics were similar between the group receiving etomidate and the group receiving alternative or no induction agent, although a slightly greater percentage of patients in the non-etomidate group were immunosuppressed, on hemodialysis, or had cirrhosis (Table 1). A greater proportion of patients who did not receive etomidate were intubated after admission to the hospital when compared to the proportion of patients who did receive etomidate. Vital signs and laboratory values, shown in Table 2, were not markedly different between the two groups. Treatments rendered to patients, summarized in Table 3, were likewise not significantly different between groups, as was the percent of patients successfully extubated.

The majority of our patients, 136 (75%) were intubated in the ED, 40 (22%) were intubated after admission to the hospital, and five (3%) were intubated in the pre-hospital setting. Of the 181 patients, 135 received etomidate and 46 received alternative agents or no induction agent. Steroids were given to 108 patients (80%) in the etomidate group and 35 patients (76%) in the non-etomidate group. Of the

Table 3. Treatments administered for cohorts

	Etomidate		No Etomidate	
	(%)	**95% CI**	**(%)**	**95% CI**
Percent Receiving:				
Antibiotics	97	93 - 99	96	85 - 99
Packed RBC transfusion	36	28 - 44	28	17 - 43
Vasopressors	59	51 - 67	44	30 - 58
Steroids	80	72 - 86	76	62 - 86
Successfully extubated (%)	67	59 - 75	70	55 - 81

46 patients receiving alternative agents or no agent, 18 died, giving a mortality of 39.1% (95% CI 25.5% to 54.6%), while of the 135 patients receiving etomidate, 63 died, giving a mortality of 46.7% (95% CI 38.1% to 55.4%), *P*=0.38. Kaplan-Meier survival curves, shown in Figure 1, demonstrate a divergence that appears to favor the group not receiving etomidate, but the difference was not statistically significant (*P*=0.29). Length of ICU stay for patients receiving etomidate, 4.4 days (95% CI 3.6 to 5.1 days), was similar to patients not receiving etomidate, 5.4 days (95% CI 3.9 to 6.9 days).

All abstractors had full agreement on the use of etomidate, steroids, and discharge condition in our subset of charts reviewed for interrater reliability. Agreement for exact time of intubation was only 67%; however, the median disagreement was less than 18 minutes.

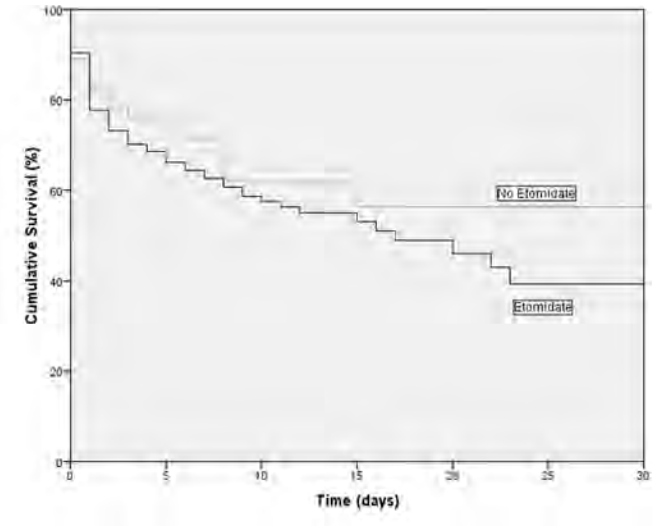

Figure 1. Kaplan-Meier estimates of survival

DISCUSSION

The primary motivation for the cessation of use of etomidate for continuous sedation in the ICU stems from a retrospective study of 428 severely injured trauma patients.[1] In this analysis, the in-hospital mortality rate was found to have increased from 28% in those given opiates with or without benzodiazepines for sedation to over 70% after the adoption of etomidate for continuous sedation, despite similar severities of illness in the two groups of patients. After the discontinuation of the use of etomidate for continuous sedation, mortality returned to 25%. Consequently, etomidate use for continuous sedation ceased while use as an agent for intubation or procedural sedation has continued to increase. Favorable hemodynamic effects combined with a lack of immediately evident adverse effects makes etomidate particularly appealing for use in critically ill patients in the ED.

Although no studies have conclusively shown clinically significant adverse outcomes from single-dose use, a recent retrospective review of 477 patients by the Corticus Study Group found an increased odds ratio of death of 1.53 for patients sedated with etomidate.[15] Additionally, a number of studies have shown adverse effects on surrogate endpoints, primarily in adrenocortical output. Reduced plasma cortisol and aldosterone levels following induction doses of etomidate have been reported to persist for up to 24 hours and appear unresponsive to adrenocorticotropic hormone stimulation.[7] Patients given etomidate have shown lower measured serum cortisol compared to those given midazolam after cosyntropin stimulation tests less than 12 hours after administration and a decreased rise in cortisol from a high-dose corticotropin test 24 hours after intubation.[7,18] A retrospective analysis of 152 patients with septic shock showed decreased responses to cosyntropin stimulation in patients given etomidate.[9] These effects are due to blockage of 11 beta-hydroxylation within the adrenal cortex.[4]

With the widespread use of etomidate in the United States for induction of anesthesia prior to intubation, the potential for worsened outcomes in septic patients when using etomidate has profound implications. In our retrospective chart review, we did not find a statistically significant difference in hospital mortality between patients given etomidate and patients given alternative agents or no induction agent for intubation. The baseline demographics, vital signs, and laboratory values in the two groups suggest that the patients were similar in severity of illness and that neither group would be expected to have significantly different outcomes. Likewise, an equivalent percentage of patients in each group had been treated with steroids.

Although we did not find a statistically significant difference in outcomes between our two groups, the difference of 7.6% in absolute mortality between groups does raise a question of clinical importance. This difference perhaps supports the suggestions of others that a well-powered, prospective, randomized trial be performed to firmly establish the safety of etomidate use in patients at risk of relative adrenal insufficiency, such as those with sepsis. Given the difference in mortality seen between our patient groups, approximately 600 patients per group would be required to obtain adequate power to obtain statistical significance, using a two-sided α of 0.05 and ß of 0.20.

LIMITATIONS

For this study, we relied on a chart review from a single tertiary care medical center and obtained data retrospectively, a method with well-known limitations. Although we performed an extensive search for patients, the possibility remains that we may not have found all septic patients intubated in this time period. We used an objective endpoint, discharge or death, limiting the need for observer interpretation, but many of our variables (times of intubation,

types of medication used, comorbidities, vital signs) were recorded manually and are therefore subject to potential errors of transcription and omission. Although we reviewed all sections of the chart to determine which medications were used for intubation, including physician entries, nursing entries, and pharmacist entries, it remains possible that patients we classified as not having received a medication such as etomidate or steroids may in fact have received these medications, and vice-versa. We received no external funding for this study, and thus were unable to hire abstractors that could be blinded to the study purpose or to patient treatment. Although our endpoints were objective, subtle biases may have affected our results in unquantifiable or unexpected ways. Despite the inclusion of patients intubated in the pre-hospital setting (who were unlikely to have received etomidate) we found that a disproportionate number of patients received etomidate, weakening the power of our study to detect statistically significant differences in mortality. In addition, many of the patients who did not receive etomidate were intubated either in the field before ED arrival or on the hospital floors after treatment in the ED, further contributing to possible confounding.

Although we had excellent agreement between abstractors in the use of etomidate, the use of steroids, and the final discharge disposition (possibly because these items were generally documented in identical locations in each electronic medical record), we had less than perfect agreement in exact times of intubation, since in many cases documentation of procedures such as intubation were provided by more than one person, including nurses, attending physicians, and resident physicians. We limited our dependence on this measure by using the day of intubation as our start point and day of discharge or death as our endpoint. We examined many commonly used variables to evaluate differences in severity of illness between patient groups, but many charts did not have a documented Glasgow Coma Scale (GCS) score, limiting our ability to calculate scores such as a Simplified Acute Physiology Score or an Acute Physiology and Chronic Health Evaluation score. We did not attempt to estimate GCS scores from the charts we reviewed. The fact remains that the clinical appearance of the patient is often not reflected in severity of illness scores, and that unmeasured differences may exist between our cohorts that might explain the difference in mortality that we observed. Moreover, the differences in location of intubation between groups may also explain these mortality differences.

CONCLUSION

We found a non-statistically significant 7.6% absolute difference in mortality between patients given etomidate and patients given alternative or no induction agent for intubation in our study population, although the study was limited by a small sample size. Given the magnitude of this difference, further investigation into the safety of etomidate when used in patients at risk of relative adrenal insufficiency may be warranted; however, the available evidence to date does not support abandoning the use of etomidate in patients with sepsis.

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Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources, and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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REFERENCES

1. Watt I, Ledingham IM. Mortality amongst multiple trauma patients admitted to an intensive therapy unit. *Anaesthesia.* 1984; 39:973-981.
2. Fellows IW, Bastow MD, Byrne AJ, Allison SP. Adrenocortical suppression in multiple injured patients: a complication of etomidate treatment. *Br Med J (Clin Res Ed)*. 1983; 287:1835-1837.
3. Ledingham IM, Watt I. Influence of sedation on mortality in critically ill multiple trauma patients. *Lancet.* 1983; 1:1270.
4. Wagner RL, White PF, Kan PB, Rosenthal MH, Feldman D. Inhibition of adrenal steroidogenesis by the anesthetic etomidate. *N Engl J Med.* 1984; 310:1415-1421.
5. Anonymous. Etomidate Package Insert. *Bedford Laboratories*. Available at: http://www.bedfordlabs.com/products/inserts/etomidate_pi.pdf. Accessed September 12, 2007.
6. Bloomfield R, Noble DW. Etomidate and fatal outcome--even a single bolus dose may be detrimental for some patients. *Br J Anaesth.* 2006; 97:116-117.
7. Malerba G, Romano-Girard F, Cravoisy A, Dousset B, Nace L, Levy B, Bollaert PE. Risk factors of relative adrenocortical deficiency in intensive care patients needing mechanical ventilation. *Intensive Care Med.* 2005; 31:388-392.
8. Jackson WL Jr. Should we use etomidate as an induction agent for endotracheal intubation in patients with septic shock?: a critical appraisal. *Chest.* 2005; 127:1031-1038.
9. Mohammad Z, Afessa B, Finkielman JD. The incidence of relative adrenal insufficiency in patients with septic shock after the administration of etomidate. *Crit Care.* 2006; 10:R105.
10. Morris C, McAllister C. Etomidate for emergency anaesthesia; mad, bad and dangerous to know? *Anaesthesia.* 2005; 60:737-740.
11. Schulz-Stubner S. Sedation in traumatic brain injury: avoid etomidate. *Crit Care Med.* 2005; 33:2723. Author reply: 2723.

12. Bloomfield R, Noble DW. Etomidate, pharmacological adrenalectomy and the critically ill: a matter of vital importance. *Crit Care.* 2006; 10:161.
13. Zed P, Mabasa V, Slavik R, Abu-Laban R. Etomidate for rapid sequence intubation in the emergency department: is adrenal suppression a concern? *Can J Emerg Med.* 2006; 8:347-350.
14. McConachie I. Etomidate controversies in emergency medicine. *Ann Emerg Med.* 2007; 50:200-201.
15. Lipiner-Friedman D, Sprung CL, Laterre PF, et al. Adrenal function in sepsis: the retrospective corticus cohort study. *Crit Care Med.* 2007; 35:1012-1018.
16. Sakles JC, Laurin EG, Rantapaa AA, Panacek EA. Airway management in the emergency department: a one-year study of 610 tracheal intubations. *Ann Emerg Med.* 1998; 31:325-332.
17. Zed PJ, Abu-Laban RB, Harrison DW. Intubating conditions and hemodynamic effects of etomidate for rapid sequence intubation in the emergency department: an observational cohort study. *Acad Emerg Med.* 2006; 13:378-383.
18. Schenarts CL, Burton JH, Riker RR. Adrenocortical dysfunction following etomidate induction in emergency department patients. *Acad Emerg Med.* 2001; 8:1-7.

Non-Invasive Method for the Rapid Assessment of Central Venous Pressure: Description and Validation by a Single Examiner

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Objectives: This study describes a means of assessing the external jugular venous pressure (JVP) as an indicator of normal or elevated central venous pressure (CVP).

Methods: Intensive care unit patients having CVP monitoring were examined. With patients in bed, the external jugular vein (EJV) was occluded at the base of the neck and observed to distend. The occlusion was then removed and the vein observed for collapse. Complete collapse was hypothesized to indicate a non-elevated CVP (≤8cm of water). In those patients whose EJV collapsed incompletely, the vein was then occluded with the finger near the angle of the jaw. With the occlusion maintained, the vein was milked downwards with the other hand to cause its emptying and was then observed for filling from below. Filling from below was hypothesized to indicate an elevated CVP (>8cm of water).

Results: In 12 of the 40 patients examined, the EJV could not be assessed (EJV not seen at all: 5, and difficult to visualize: 7). For the remaining 28 patients, 11 had a CVP > 8 cm, while 17 had a CVP of ≤ 8. EJV assessment was 100% accurate (95% Confidence Interval 88-100) in predicting whether or not a patient's CVP was greater or less than 8 cm of water.

Conclusion: EJV assessment, when visible, is accurate to clinically assess a patient's CVP in the hands of the author. Further studies are needed to see if they are reproducible by other observer. [*West*JEM. 2008;9:201-205.]

INTRODUCTION

The rapid and accurate assessment of volume status is an important adjunct in the management of many emergency department (ED) patients. Although ultrasound has been shown to be helpful in this regard, it is not available to all emergency physicians (EP) and takes a degree of skill and time to perform properly.[1, 2] The only other technological alternatives available to the EP to make such a determination are invasive.

In 1930 Lewis first described the assessment of central venous pressure, (CVP) by observing the level of distension of the internal jugular veins (IJV).[3] Right atrial pressure is translated from the vertical height of the venous column above the sternal angle, (given that the sternal angle itself is, on average, 5 cm above the right atrium).[4]

Over the subsequent decades, physicians have employed this technique of clinical examination as a rapid, non-invasive means to assess the volume status of patients. Unfortunately, this method has been found to have a low sensitivity and poor inter-observer reliability and as such is ill-suited to ED patients.[5-7]

A simpler method would be to use the much more visible external jugular vein, (EJV) for measurement. However, such measurements had, until recently, been shown to be even more unreliable than those of the internal jugular.[8] This is due to the variability of the anatomy of the vein and in some cases

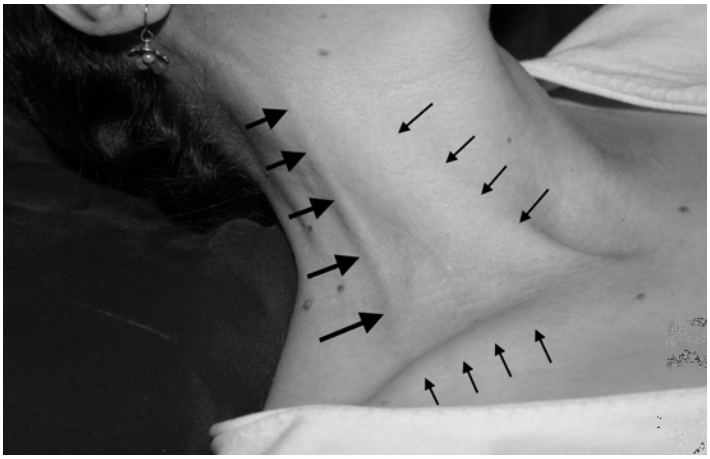

Figure 1. The surface anatomy of the external jugular vein, (indicated by the large arrows). The clavicle and sternocleidomastoid muscle are indicated by the small arrows.

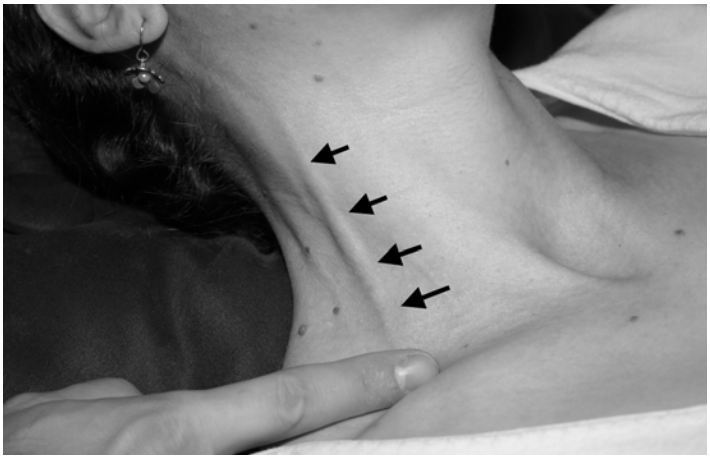

Figure 2. The external jugular vein is occluded at the clavicle and distends as it fills from above (indicated by the arrows).

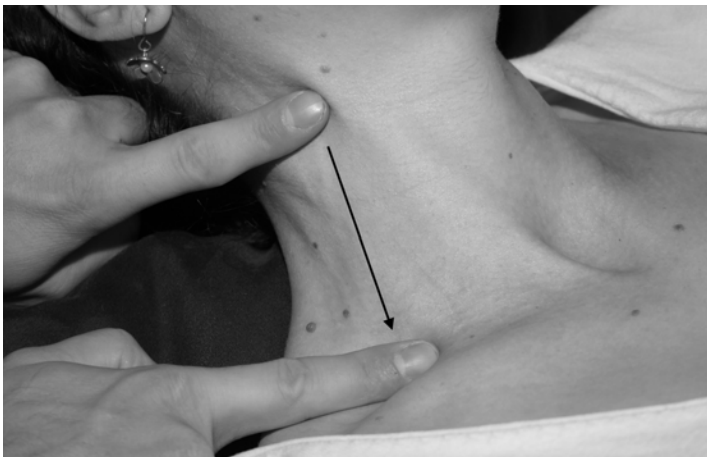

Figure 3. The second step of the technique: milking the vein empty with a lower finger in the direction of the arrow while maintaining the upper occlusion with the other hand.

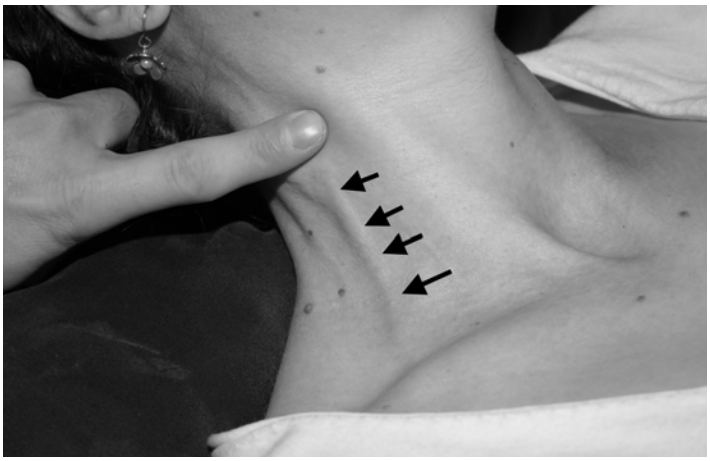

Figure 4. With the lower occlusion removed and the upper occlusion maintained, the external jugular vein fills from below, (as indicated by the arrows). The subject in the photo employed a Valsalva to stimulate a high CVP.

to partial occlusion of the vein by sclerotic valves that may prevent emptying.

In a study that compared EJV pulsation height to CVP readings from a central line,[9] Vinayak and colleagues[9] helped to dispel the notion that the EJV is unreliable. They reported that EJV pulsations were useful to determine if an intensive care unit (ICU) patient's CVP was low (≤ 5 cm of water), normal (6-9 cm) or high (≥ 10 cm) and that the EJV performed well for both low and high values. However, in a busy ED, this technique may perform less well than in an ICU, as it calls for a careful assessment of the veins to determine the height of venous pulsations. Alternatively, the EJV may be useful to rapidly establish whether a patient's CVP is elevated or not, rather than an actual value in cm. While this would preclude a diagnosis of hypovolemia, it could simplify the determination of hypervolemia vs. euvolemia or hypovolemia. We describe a method that allows for the anatomical variations of the EJV. Although described in some texts, this method is rarely used and has never been validated prospectively.[10]

This report describes the technique and details the findings of a prospective trial to validate CVP measurement using the EJV.

METHODS

We conducted a prospective, observational study in a tertiary care hospital ICU in patients with invasive CVP monitoring. ICU, rather than ED, patients were enrolled because of the higher prevalence of CVP catheters and the decreased likelihood of ongoing resuscitation on study subjects. A convenience sample was selected over a three-

month period from two hospitals. The local IRB did not require written consent.

Patients were examined by the author (JS, who was not involved in patient management and was blinded to the CVP measurement at the time of the exam) in the following manner: The primary nurse turned the monitor away prior to the investigator approaching the patient. Although CVP lines were not re-zeroed prior to the measurement being recorded, the wave-form was verified.

EJV assessment was done on whichever side of the neck that it could be most easily identified regardless of the presence of a central line. The EJV was identified as it courses medial to lateral in a rostral direction across the sternocleidomastoid muscle (Figure 1). With the patient's bed at approximately 45º, the vein was occluded (above the clavicle) and observed to distend (Figure 2). At end expiration the occlusion was removed and the vein observed for collapse. Complete collapse was hypothesized to indicate a non-elevated CVP (≤8cm of water).

Incomplete emptying was recorded as indeterminate. This finding may be due to three reasons: 1) sclerotic valves prevent emptying in a patient with a normal CVP; 2) occlusion of the vein by a fascial flap at its distal end; or 3) truly elevated CVP. To differentiate between vein obstruction consistent with the first two possibilities and a truly elevated CVP, a second step was performed in patients whose EJV collapsed incompletely or had distension prior to examination.

The vein was occluded with the finger at the angle of the jaw. The vein was milked downwards with the other hand in order to cause its emptying (Figure 3). With the upper occlusion maintained, the lower occlusion was removed and the vein observed for filling from below (Figure 4). Filling from below was hypothesized to indicate an elevated CVP (>8cm of water). Lack of filling would indicate that the patient had a non-elevated CVP and that incomplete emptying observed in the first step of the procedure was due to venous obstruction.

Patient data included age and sex. Prior to measurement, we noted whether or not the EJV was easily visualized, visualized with difficulty, or not visualized at all. This was a purposely subjective evaluation and not defined in advance. Outcome measures were the result of the examination technique, method of technique used (single or two-step reading), and the CVP reading from the central line.

Data were reported in two-by-two tables to demonstrate sensitivity, specificity and overall diagnostic accuracy.

RESULTS

We examined 40 patients. Twenty five (62.5%) were male. The average age was 40, (range 30-89). Ten patients (25%) had subclavian central lines, while 30 (75%) had IJV central catheters. In five of the 40 patients, the EJV could not be visualized at all. In the remaining 35 patients, 19 had a CVP ≤ 8 while 16 had a CVP > 8.

Table 1. Results of External JVP* Assessment (n = 35)

		JVP* Measurement Result	
		≤ 8 cm	> 8 cm
Actual CVP**	≤ 8 cm	18	1
	> 8 cm	5	11

*JVP: jugular venous pressure
**CVP: central venous pressure

Table 2. Results of External JVP* Assessment-Patients excluding seven patients with difficult to visualize veins (n = 28)

		JVP* Measurement Result	
		≤ 8 cm	> 8 cm
Actual CVP**	≤ 8 cm	17	0
	> 8 cm	0	11

*JVP: jugular venous pressure
**CVP: central venous pressure

Table 3. Results of Single and Two-step Techniques in EJV* Assessment

	CVP*** < 8	CVP*** > 8
EJVA ≤ 8 (single-step technique)**	12	3
EJVA ≤ 8 (two-step technique)**	6	2
EJVA > 8 (two-step technique)**	1	11

*EJV: external jugular vein
**EJVA: external jugular vein assessment
***CVP: central venous pressure

In the 35 patients whose EJV could be assessed, the sensitivity and specificity for elevated CVP was 91.7% (95% confidence interval (CI) 62-100) and 78.3% (95% CI 56-93) respectively. The overall accuracy of the test in determining whether or not CVP was < or ≥ 8 cm of water was 83 % (95% CI 66-93) (Table 1). Eliminating those seven patients in whom the EJV was not easily visible resulted in a diagnostic accuracy of 100% (95% CI 88-100)(Table 2).

The reason for the inability to visualize the vein at all was obesity in three and anasarca in two. In an additional seven patients the EJV could be visualized only with difficulty,

resulting in one false positive and five false negatives.

Table 3 shows the findings in all 35 patients by whether they had a one-step technique or two.

DISCUSSION

The rapid and accurate assessment of a patient's volume status is important for treating a wide range of ED patients. The determination of hypovolemia is critical in early goal-directed therapy of sepsis and in diagnosing shock ascribed to volume loss.[11] It is also helpful to be able to easily identify those patients who have adequate or elevated intravascular volume, as these patients are less likely to respond to further volume challenges in the case of shock, or may require diuresis for congestive heart failure.[12]

It has become common practice for EPs and intensivists to use CVP as a surrogate marker for intravascular volume. However, others have questioned the veracity of this and have advocated that other measures such as continuous right ventricular end-diastolic volume index, pulmonary capillary wedge pressure, or transesophageal echocardiographically-derived left ventricular end-diastolic area be used instead.[13]

While this important question has yet to be resolved, none of the alternatives to CVP are currently widely available to the EP. As such, the determination of CVP remains important.

While ultrasound provides a reliable and reproducible means of making this determination, it requires special equipment and skills. While physical examination of the IJV has been used for over 70 years, it is a skill performed inconsistently by cardiologists and non-cardiologists alike.[14, 15] A recent report on the validity of EJV assessment made this a plausible alternative for this purpose, although it too may have limited value in the ED. The assessment of the EJV as described in this paper may make the determination of elevated volume status more practical.

In the hands of the author, EJV assessment via a single- or two-step technique when indicated, proved accurate in all patients in whom the EJV could easily be visualized. This limitation is important as false results were obtained in six of the seven patients in whom the vein was not easily seen. The subjective ability to visualize the EJV was not defined a-priori. We felt this was important as it would make this technique more applicable. We acknowledge that different observers may have varying abilities to visualize the EJV and this could result in inappropriate use of the technique. For our subjects, the EJV was easily identifiable in nearly 70% of the patients.

The accuracy of the test was consistent in both single-step and two-step techniques and was not affected by whether or not the patient's true CVP was elevated. The test was performed on whichever side of the neck the EJV was visualized most easily, regardless of the presence of a central line. This was on the right in most patients but the left side proved no less accurate.

The time to conduct the test was very brief for all patients. For the single-step technique 10-15 seconds were required, while 20-25 seconds were required for the two-step test. Furthermore, in the setting of the ED the test can be performed without interrupting other patient evaluation and management. No patient reported any discomfort during the performance of the test.

In the current clinical environment, emphasizing all manner of technology to diagnose ED patients, there has been a gradual erosion in EPs' physical exam skills. This study, although limited, offers a return to traditional physical examination that provides useful information quickly.

LIMITATIONS

These results reflect the experience of only one examiner and therefore lack external validity. However, in our experience, this test is easily taught. A study to determine inter-observer reliability as well as validating accuracy in the hands of others is planned.

Many of the patients were post-operative or on mechanical ventilation and these are not the same patients as in the ED.

Unlike the technique described by Vinayak, the one described here does not distinguish between euvolemia and hypovolemia. Instead, it discriminates between those who have hypervolemia and those without. Other means must be used to stratify patients in the latter category.

A final limitation of the EJV assessment is an extension of the limitations of assessment of the IJV. Neither of these methods will be useful to assess CVP in patients with tricuspid regurgitation or stenosis, atrial septal defect or superior vena cava syndrome,-all of which will result in falsely elevated readings.

CONCLUSION

EJV assessment in the hands of the author is highly accurate to grossly determine a patient's CVP when the EJV is clearly visible. Both the single- and two-step techniques can be employed with confidence. The test can be performed during other important aspects of therapy. When the EJV is not clearly seen, other methods should be used to determine patient volume status.

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Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources, and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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REFERENCES

1. Lipton B. Estimation of central venous pressure by ultrasound of the internal jugular vein. *Am J Emerg Med*. 2000; 18:432-4.
2. Lipton BM. Determination of elevated jugular venous pressure by real-time ultrasound. *Ann Emerg Med*. 1999; 34:115.
3. Lewis T. Early Signs of Cardiac Failure of the Congestive Type. T*he British Medical Journal*. 1930; 1:849-52.
4. O'Rourke R, Braunwald E. *Harrison's Principles of Internal Medicine*. 13th ed. New York: McGraw-Hill; 1994.
5. Butman S, Ewy G, Standen J, et al. Bedside Cardiovascular Examination in Patients With Severe Chronic Heart Failure: Importance of Rest or Inducible Jugular Venous Distension. *JACC*. 1993; 22:968-974.
6. Chakko S, Woska D, Martinez H, et al. Clinical, radiographic, and hemodynamic correlations in chronic congestive heart failure: conflicting results may lead to inappropriate care. *Am J Med*. 1991; 90:353-359.
7. Stevenson LW, Perloff JK. The limited reliability of physical signs for estimating hemodynamics in chronic heart failure. *JAMA*. 1989; 261:884-8.
8. Davison R, Cannon R. Estimation of Central Venous Pressure by Examination of Jugular Veins. *Amer Heart Jour*. 1974; 87:279-82.
9. Vinayak AG, Levitt J, Gehlbach B, Pohlman AS, Hall JB, Kress JP. Usefulness of the external jugular vein examination in detecting abnormal central venous pressure in critically ill patients. *Archives of Internal Medicine*. 2006; 166:2132-2137.
10. Sapira J. *The Art & Science of Bedside Diagnosis*. Baltimore: Williams & Wilkins; 1990.
11. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M; Early Goal-Directed Therapy Collaborative Group. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001; 345:1368-1377.
12. Magder S, Bafaqeeh F. The clinical role of central venous pressure measurements. *Journal of Intensive Care Medicine*. 2007; 22:44-51.
13. Wiesenack C, Fiegl C, Keyser A, Laule S, Prasser C, Keyl C. Continuously assessed right ventricular end-diastolic volume as a marker of cardiac preload and fluid responsiveness in mechanically ventilated cardiac surgical patients. *Critical Care* (London, England). 2005; 9:R226-R233.
14. Ajuluchukwu JN, Inem VA. The examination of the jugular venous pressure (JVP): variation of technique amongst Nigerian resident doctors. *Niger Postgrad Med J*. 2000; 7:124-128.
15. Cook DJ. Clinical assessment of central venous pressure in the critically ill. *Am J Med Sci*. 1990; 299:175-178.

Use of the Trendelenburg Position in the Porcine Model Improves Carotid Flow During Cardiopulmonary Resuscitation

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Background: Cardiopulmonary resuscitation (CPR) is now widely used as a treatment for ventricular fibrillation, though numerous studies have shown the outcome of standard CPR to be dismal. Alternative methods of CPR, including interposed abdominal compression, constant aortic occlusion, and the use of intrathoracic pressure regulator, have been shown to increase cardiac output and affect the mortality rate of CPR.

Objectives: Here we suggest the Trendelenburg position as yet another method of increasing cardiac output and therefore improving the effectiveness of chest compressions. We hypothesized that the use of the Trendelenburg position during CPR would increase cardiac output as measured by carotid blood flow.

Methods: We anaesthetized six pigs and measured their pre-arrest carotid flow rate for two minutes. We then induced ventricular fibrillation in those pigs and performed open-chest CPR on them. Post-arrest carotid blood flow was measured for two minutes each at 0 (supine position), 10, 20, and 30 degrees of head-down tilt in each pig. The mean carotid flow for each degree of tilt was compared to mean carotid flow at 0 degrees of tilt using a paired student t-test.

Results: We found an increase of up to 1.4-fold in carotid blood flow during CPR in the Trendelenburg position, though only 20 and 30 degrees of Trendelenburg showed a statistically significant increase from the 0 degrees of tilt in pigs.

Conclusion: The Trendelenburg position can lead to increased blood flow through the carotid arteries during CPR in this pig model. Future studies should investigate whether this increased blood flow through the carotid arteries leads to improved brain perfusion and better neurologic outcomes. [*West*JEM. 2008;9:206-211.]

INTRODUCTION

Approximately 225,000 people in the United States and 375,000 people in Europe experience cardiac arrest every year, the majority of which occur in an out-of-hospital setting.[1,2] Cardiac arrest occurs in the form of ventricular fibrillation (VF), which is treatable by shock administration, or as pulseless electrical activity (PEA) or asystole, which are not treatable through shock administration. The degree of cardiac

and neurological damage following VF is correlated with the amount of blood flow through coronary and carotid arteries respectively. In situations when immediate defibrillation is not an option, cardiopulmonary resuscitation (CPR) should be initiated in order to temporarily maintain enough flow to the heart and the brain.[3-8]

There are many factors that can contribute to a depressed cardiac output during CPR. For example, it has been suggested that the average rescuer performing CPR can achieve adequate depth of compression in majority of cases on women but only in small percentage of men who comprise the majority of cardiac arrest victims.[9] The inadequate compression depth would decrease the CPR-generated cardiac output. In addition, incomplete chest decompression during CPR performed by a fatigued rescuer can increase the intrathoracic pressure, impair venous return to the heart, and decrease cardiac output as well as coronary and cerebral perfusion pressures.[10] Therefore, there is a need to further improve CPR-generated cardiac output and cerebral perfusion in order to compensate for such lack of compression depth and incomplete decompression. Compared to standard CPR there is an increase in cardiac output and blood flow to the heart and brain with methods such as the use of an intrathoracic pressure regulator,[11,12] interposed abdominal compression,[13-15] and constant aortic occlusion.[16,17]

We would like to propose the Trendelenburg position as yet another adjunct to CPR. The Trendelenburg position was once advocated to increase cardiac filling in low-flow states such as hypotension and shock. However, multiple studies were unable to demonstrate this hypothesized benefit.[18-23] This lack of clinical benefit may be due to the vasoconstriction and increased arteriolar vascular tone seen during the compensation stage of hypovolemic shock, which would counteract the effects of placing the patient in the Trendelenburg position. However, during cardiac arrest, the victim has no tone and one would expect the model to change. In this study we used a porcine model to measure carotid blood flow as an indirect measure of cardiac output during experimental CPR and compared this flow for varying degrees of Trendelenburg positioning.

METHODS

This study was conducted at Biosurg, Inc. in Winters, California with adherence to existing protocols. All work was performed in accordance with the "Guide for the Care and Use of Laboratory Animals" prepared by the National Research Council of the National Institutes of Health.

Animal Preparation

Six adult pigs weighing 80 to 100 kilograms were studied. All the animals received halothane and isofluorane anesthesia. They were intubated with ventilation established before being placed supine on the operating table. We obtained right carotid access using a cutdown procedure. A 4 mm non-cannulating flow probe was attached to the right common carotid artery. To allow placement of other monitoring devices we performed a median sternotomy to provide complete access to the heart, while keeping the pericardium intact over the ventricles. ECG leads were attached for continuous monitoring. A 5 French pressure measuring catheter was advanced into the femoral artery via cutdown and positioned in the aortic arch. A capnograph was set to operate from a transducer in the endotracheal tube. Flow and pressure and capnograph devices were zeroed and calibrated. The ventilation depth and rate were adjusted to achieve between 30 and 35 mmHg end-tidal carbon dioxide measurement (pETCO2). The carotid flow rate, the ECG, and the pETCO2 were obtained continuously from this point until the end of the study.

Experimental Protocol

Before inducing VF, the pre-arrest carotid flow rate for each animal was measured at 0 degrees of tilt (supine position) in order to compare with the post-arrest carotid flow rates. Each pig was then placed into VF by direct application of current from a 9-volt battery. A minimally invasive device was advanced against the pericardium. To standardize the CPR performed on each animal, the minimally invasive device was pumped at 90 to 110 beats per minute at 10 to 12 pounds of force. The minimally invasive device is made up of an 8 cm diameter expandable membrane at the end of a shaft. Initially, the membrane is collapsed within a 45 French access sheath to allow easier introduction into the subject's thorax. There is a 67 French flange in position on the sheath handle to prevent over-penetration of the device into the thorax.

We obtained baseline post-arrest carotid flow rates for two minutes with the animal at supine position. The table was then subsequently tilted to 10, 20, and 30 degrees of incline with the head placed down, and two minutes of data were collected for each degree of incline. We collected hemodynamic data at least every 30 seconds at each angle of tilt. Each animal served as its own control as it was placed in increased Trendelenburg tilts in a sequential manner, and its mean carotid flow rate was compared to both the pre-arrest and post-arrest baseline values. At the end of the experimental period, the animals were euthanized by termination of circulatory support.

Statistical Methods

We analyzed the significance of hemodynamic changes with degree of head-down tilt using a PC-based statistical analysis package. A paired student t-test was used as each animal served as its own control. Statistical significance was accepted if $p < 0.05$. The coefficient of determination r^2 was determined using the same software.

RESULTS

The mean carotid flow in the pre-arrest phase of the study

Table 1. Mean carotid flow rates (mL/min) at varying degrees of Trendelenburg in pigs were compared and analyzed. Only three and four sets of data were available for 10 and 30 degrees of Trendelenburg, respectively. The "Difference Mean (SED)" column represents the subtraction of the first value from the second value. A positive difference mean value represents an increase in carotid flow. A paired student t-test was performed with the significance level set to α =0.05.

Comparison First vs. Second	N pairs	First Value Mean (SEM)	Second Value Mean (SEM)	Difference Mean (SED)	95% CI of Difference	p-Value for Difference
Pre-arrest vs. Post-arrest (0^o)	6	442 (41)	149 (18)	-293 (41)	(-188, -398)	0.0008
Post-arrest (0^o) vs. Post-arrest (10^o)	3	165 (34)	177 (37)	12 (11)	(-35, 47)	0.3975
Post-arrest (0^o) vs. Post-arrest (20^o)	6	148 (18)	194 (25)	46 (9)	(69, 23)	0.004
Post-arrest (0^o) vs. Post-arrest (30^o)	4	128 (9)	178 (5)	50 (13)	(9, 91)	0.0343

SEM = standard error mean
SED = standard error of the difference

was 442 mL/min. After induction of ventricular fibrillation with CPR in progress this decreased to 149 mL/min (difference of 293 mL/min (95% CI: 188, 398)).

For 10 degrees of incline, only three complete sets of data were available due to technical issues with the flow meters of the other animals. These three animals had a post-arrest mean carotid flow of 165 mL/min at 0 degrees of tilt that increased to 177 mL/min at 10 degrees of tilt (an increase of 12mL/min from baseline (95% CI: -12,35)).

At 20 degrees of incline, six complete sets of data were available. The post-arrest mean carotid flow for all six pigs at this level of incline was 194 mL/min (an increase of 46 mL/min from baseline (95% CI of the difference: 23, 69)).

For 30 degrees of incline, four complete sets of data were available. These four animals had a post-arrest mean carotid flow of 128 mL/min at 0 degrees of tilt and 178 mL/min at 30 degrees of tilt (an increase of 50 mL/min from baseline (95% CI: 9, 91)) (Table 1) (Figure 1a).

Compared to baseline CPR at supine position, compressions at head-down angles of 10, 20 and 30 degrees resulted in 1.1, 1.3, and 1.4 times greater carotid flow, respectively. An alternate method of viewing the data is as a percentage of the mean carotid flow in the pre-arrest phase. The mean carotid flow at 0 degrees was 34% of pre-arrest flow. At 10 degrees of Trendelenburg the mean flow increased to 37% of the pre-arrest value, at 20 degrees this value was 44%, and at 30 degrees of tilt the mean carotid flow was 49.5% of pre-arrest value. The change in percentage of pre-arrest flow rates were tightly associated with the degrees of Trendelenburg tilt with a coefficient of determination r^2= 0.98 (Figure 1b).

DISCUSSION

Initially developed as a surgical position, the Trendelenburg position was later adopted by Walter Cannon as a treatment for shock during World War I. Although it has been widely taught and used by healthcare professionals,[24] its effectiveness as a treatment for shock has not yet been clinically shown despite numerous studies. The Trendelenburg position does not have any beneficial hemodynamic effects in hypotensive patients,[18,19] leads to no changes in tissue oxygenation in hypovolemic postoperative patients,[20] and appears to cause small and ineffective autotransfusion changes in normovolemic patients.[21] Reich and colleagues[22] report an increase in mean arterial pressure and cardiac output in hypotensive patients with coronary artery disease following placement in the Trendelenburg position, although this position led to impaired respiration in those patients to a greater extent. Furthermore, steep head-down positions have been reported to have potentially harmful effects in patients with cardiac, respiratory, and neural problems. Such effects include increased intracranial pressure and respiratory compromise.[23] There has also been a report of brachial plexus injury in a post-operative patient who underwent a long period of head-down tilt at an angle of 30-40 degrees.[25]

With the use of Trendelenburg positioning during CPR we were able to see an increase in carotid blood flow. Our results show that the use of this position improved carotid blood flow during CPR in this porcine model. We found the 20 and 30 degrees of tilt to have a statistically significant higher carotid flow. The 10 degree of head-down tilt also showed an increased carotid blood flow, although this increase was not statistically significant. Chest compressions while placing the animals in the Trendelenburg position produced up to 1.4-fold increase in the carotid flow compared to the standard CPR. It is suggested that other adjuncts to conventional CPR also result in improved circulation during cardiac arrest. Gedeborg and colleagues[16] reported an increase of 62% in carotid blood flow following balloon occlusion of descending aorta, and Yannopoulos and colleagues[12] reported an average increase

of 70% in carotid artery flow with the use of an intrathoracic pressure regulator during CPR. Similarly, Babbs[13] suggested that by combining interposed abdominal compression and chest compression the total cardiac output may show a 1.9-fold increase compared to conventional CPR alone. These adjuncts, however, have their own limitations; the use of constant aortic occlusion requires a period of conventional CPR while the balloon catheter is inserted,[16] and interposed abdominal compression requires the availability of additional rescuer personnel as well as further training.[13] Nonetheless, recent studies have suggested these adjuncts to be effective and their combination with the use of the Trendelenburg position may prove to be beneficial.

One of the major goals of cardiopulmonary resuscitation is to preserve neurological function, which in turn is dependent on the perfusion of cerebral tissue. The brain is very vulnerable to hypoxic/ischemic injury. Following cardiac arrest and CPR, approximately 80% of patients are comatose with about 40% entering persistent vegetative state.[26] Novel approaches, such as mild therapeutic hypothermia (lowering body temperature to 33ºC for 12 to 24 hours following return of spontaneous circulation), have been suggested to help with ischemic brain injury, and recent studies have shown an increase in favorable neurological outcomes following therapeutic hypothermia after resuscitation from cardiac arrest.[1,2]

Many authors have also suggested adjuncts to standard CPR to improve outcomes, including the use of intraortic balloon pumps, abdominal binding systems and counterpulsation techniques, either through abdominal compression or timed respirations. All of these adjuncts attempt to improve blood flow during standard closed-chest CPR by increasing the venous return to the thorax and heart (i.e. prime the pump).[13-17, 27-28] Given that cardiac output is a function of both the stroke volume and the rate, and that the slow ventricular filling during CPR limits the rate of effective compressions, the purpose of a good adjunct should be to increase the stroke volume in the face of a constant compression rate.

An increase in carotid blood flow through the use of Trendelenburg position is an important finding with clinical implications. An increase in carotid blood flow may improve cerebral oxygen delivery during CPR and, together with the use of mild therapeutic hypothermia, may further improve neurological outcomes following resuscitation. However, it must be noted that the "Guidelines for Cardiopulmonary Resuscitation" recommends placing the post-cardiac arrest survivors in a 30 degrees reverse Trendelenburg position with head at midline.[29] This position is thought to maximize cerebral arterial perfusion without compromising cerebral venous drainage after resuscitation, though its effectiveness has not yet been studied.[30] Therefore, placing the patient in the Trendelenburg position may theoretically lead to an

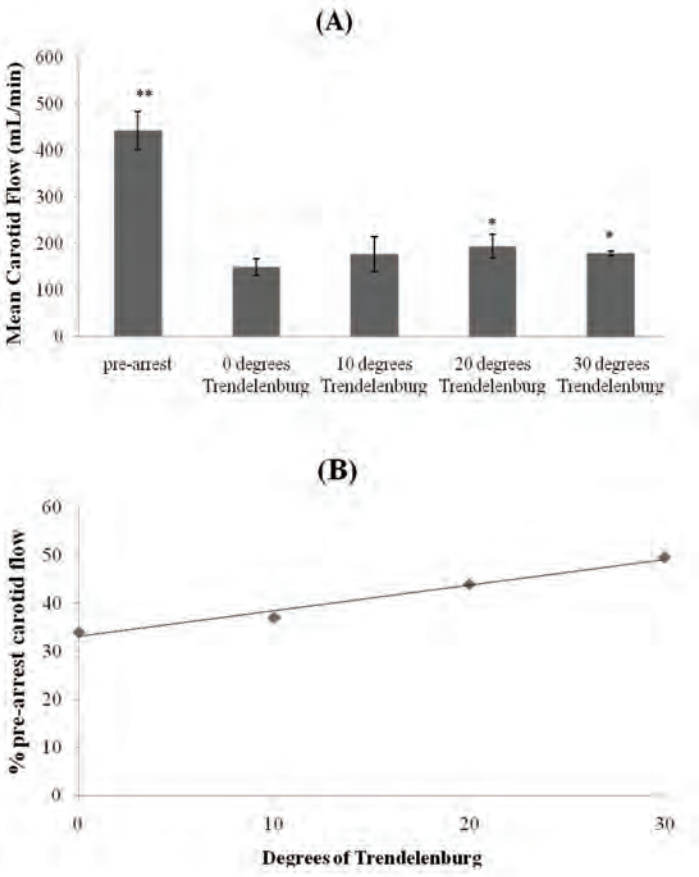

Figure 1. (A) Mean ± Standard Error Mean carotid flow rates are shown for pre-arrest and each degree of Trendelenburg tilt. * represents $p < 0.05$ and ** represents $p < 0.001$ with respect to 0 degrees of Trendelenburg as baseline. Sample size is three and four for 10 and 30 degrees Trendelenburg, respectively, and six for the other trials. (B) Changes in percent pre-arrest carotid flow show a strong linear association with the degrees of Trendelenburg tilt ($r^2=0.98$).

increase in the intracranial pressure during cardiopulmonary resuscitation, which can lead to a decrease in cerebral perfusion—the opposite of what is intended by this maneuver. This requires additional studying and is further discussed in the limitations section of our study.

Although numerous studies of the Trendelenburg position in healthy volunteers and hospitalized patients have suggested little or no beneficial hemodynamic effects, it may well be beneficial during CPR. The ability of an individual, healthy or hypotensive, to autoregulate peripheral and central vascular tone (specifically, the arteriolar vascular tone) minimizes the effect that a head-down position might have on cerebral blood flow.[31] On the other hand, a person experiencing cardiac arrest with concomitant decrease in vascular tone has lost such autoregulatory reflexes. The head-down position during cardiac arrest may lead to increased arteriolar blood flow with the aid of gravity, as the vascular tone is decreased due to

the loss of autoregulatory reflexes. Our findings support this theory as we saw an increase in CPR-generated carotid blood flow with increasing Trendelenburg.

At the same time it must be noted that the venous system is not significantly affected by the autoregulatory reflexes, considering the lower vascular tone of a venule compared to an arteriole. Yet the application of Trendelenburg could simply aid the return of the pooled venous blood from the periphery to the heart and in this way serve as the means to increase the cardiac output through increasing the preload. However, one can argue that more blood flow to the arrested heart could lead to a higher right atrial pressure and a subsequent decrease in coronary perfusion pressure (defined as the difference between diastolic aortic pressure and right atrial pressure)—another unwanted effect. Future experiments should investigate these possibilities in more detail.

LIMITATIONS

It is important to note the following limitations of our study. Firstly, the sample size was small. Secondly, venous return to the heart was not measured, and although it is believed that the Trendelenburg position would lead to increased venous flow to the heart, there is no direct evidence for that. Thirdly, our study employed an open-chest CPR technique, which may compromise any benefit of the thoracic pump model of blood flow during closed chest CPR. Regardless, open-chest CPR has been repeatedly shown to be superior to closed-chest CPR based upon standard measures of arterial pressure generated, cardiac output, coronary perfusion pressure and cerebral blood flow achieved.[32-33] Fourthly, the applicability of the pig as a model for CPR has been shown, but the appropriateness of the pig to simulate the relative effects of Trendelenburg in arrest with the comparatively small leg volume of the pig cannot be predicted. Fifthly, because return of spontaneous circulation and the neurologic outcome of CPR with Trendelenburg were not measured in this particular model there is no information as to the overall effect of this increased cerebral flow on survivors of cardiopulmonary arrest. Sixthly, though carotid blood flow was used as an indirect measure of cardiac output and cerebral perfusion, direct measurements of intracranial pressure, cerebral perfusion pressure and actual cerebral blood flow were not performed. Theoretically, if the head-down position augments forward flow, cerebral venous return through internal jugular veins may be proportionately diminished due to gravity. This may lead to decreased delivery of waste products from the CNS, as well as increased intracranial pressure and decreased cerebral perfusion pressure, negating the effects of the increased carotid flow. Furthermore, during placement in the Trendelenburg position the abdominal organs can be pushed into the thoracic cavity increasing the intrathoracic pressure. Recent studies suggest that an increase in the intrathoracic pressure would lead to an increase in the intracranial pressure, decreasing cerebral perfusion.[10-12] However, a modified Trendelenburg position with cervical flexion and elevation of the head might minimize these potential negative effects without diminishing the forward flow through the carotids. It is also possible that the increased carotid flow simply reflects the backflow of blood from the abdominal aorta as an effect of gravitational forces, and this may lead to decreased perfusion of other organs, including the kidneys and intestines. Finally, the pigs were moved from 0 to 10 to 20 to 30 degrees in a step-wise fashion, possibly exaggerating the effects of each position.

CONCLUSION

Future studies should be undertaken to: 1) investigate the efficacy of Trendelenburg position on a closed-chest CPR technique; 2) determine the effects of both standard Trendelenburg and modified Trendelenburg with cervical flexion on venous return to the heart, intracranial pressure, coronary and cerebral perfusion pressures, as well as internal jugular vein and abdominal aorta blood flow to control for the negative effects of gravity; 3) investigate return to spontaneous circulation and neurologic outcome of concomitant CPR and Trendelenburg; 4) determine flow at an initial setting of 20 to 30 degrees in the immediate post-arrest phase.

Despite the limitations of the study, in all of the animals studied carotid flow increased as the animal was placed into the Trendelenburg position. Our findings indicate that 20 and 30 degrees of head-down tilt can improve carotid flow in pigs in a statistically significant manner. Such findings merit further investigations of the use of Trendelenburg position during CPR. Whether these findings can be extrapolated to human subjects undergoing CPR remains to be seen.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources, and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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REFERENCES

1. Curfman GD. Hypothermia to protect the brain. Perspective. *N Engl J Med*. 2002; 346:546.
2. The Hypothermia after Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med*. 2002; 346:549-556.

3. Steen S, Liao Q, Pierre L, Paskevicius A, Sjöberg T. The critical importance of minimal delay between chest compressions and subsequent defibrillation: haemodynamic explanation. *Resuscitation*. 2003; 58: 249-258.
4. Kern KB, Ewy GA, Voorhees WD, Babbs Cf, Tacker WA. Myocardial perfusion pressure: a predictor of 24-hour survival during prolonged cardiac arrest in dogs. *Resuscitation*. 1988; 16:241-250.
5. Michael JR, Guerci AD, Koehler RC, Shi AY, Tsitlik J, Chandra N, Niedermeyer E, Rogers MC, Traystman RJ, Weisfeldt ML. Mechanisms by which epinephrine augments cerebral and myocardial perfusion during cardiopulmonary resuscitation in dogs. *Circulation*. 1984; 69:822-835.
6. Paradis NA, Martin GB, Rivers EP, Goetting Mg, Appleton TJ, Feingold M, Nowak RM. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. *JAMA*. 1990; 263:1106-1113.
7. von Plata I, Weil MH, von Plata M, Bisera J, Bruno S, Gazmuri RJ, Rackow EC. Cardiopulmonary resuscitation in the rat. *J Appl Physiol*. 1988; 65:2641-2647.
8. Andreka P, Frenneaux MP. Haemodynamics of cardiac arrest and resuscitation. *Current opinion in critical care*. 2006; 12:198-203.
9. Rottenberg EM. The need for a leftward shift in the flow-depth relationship during cardiopulmonary resuscitation. *Resuscitation*. 2007; 72:350-352.
10. Yannopoulos D, McKnite S, Aufderheide TP, Sigurdsson G, Pirrallo RG, Benditt D, Lurie KG. Effects of incomplete chest wall decompression during cardiopulmonary resuscitation on coronary and cerebral perfusion pressure in a porcine model of cardiac arrest. *Resuscitation*. 2005; 64:363-372.
11. Yannopoulos D, Nadkarni VM, McKnite S, Rao A, Kruger K, Metzger A, Benditt D, Lurie KG. Intrathoracic pressure regulator during continuous-chest-compression advanced cardiac resuscitation improves vital organ perfusion pressures in a porcine model of cardiac arrest. *Circulation*. 2005; 112:803-811.
12. Yannopoulos D, Aufderheide TP, McKnite S, Kotsifas K, Charris R, Nadkarni V, Lurie KG. Hemodynamic and respiratory effect of negative tracheal pressure during CPR in pigs. *Resuscitation*. 2006; 69:487-494.
13. Babbs CF. Efficacy of interposed abdominal compression-cardiopulmonary resuscitation (CPR), active compression and decompression-CPR, and Lifestick CPR: Basic physiology in a spreadsheet model. *Crit Care Med*. 2000; 28:N199-N202.
14. Babbs CF. Interposed abdominal compression CPR: a comprehensive evidence based review. *Resuscitation*. 2003; 59:71-82.
15. Xavier L, Kern KB, Berg RA, Hilwig RW, Ewy GA. Comparison of standard CPR versus diffuse and stacked hand position interposed abdominal compression-CPR in a swine model. *Resuscitation*. 2003; 59:337-344.
16. Gedeborg R, Rubertsson S, Wiklund L. Improved haemodynamics and restoration of spontaneous circulation with constant aortic occlusion during experimental cardiopulmonary resuscitation. *Resuscitation*. 1999; 40:171-180.
17. Gedeborg R, Silander HC, Rubertsson S, Wiklund L. Cerebral ischaemia in experimental cardiopulmonary resuscitation—comparison of epinephrine and aortic occlusion. *Resuscitation*. 2001; 50:319-329.
18. Taylor J, Weil MH. Failure of the Trendelenburg position to improve circulation during clinical shock. *Surg Gynecol Obstet*. 1967; 124:1005-1010.
19. Sibbald WJ, Paterson NA, Holliday RL, Baskerville J. The Trendelenburg position: hemodynamic effects in hypotensive and normotensive patients. *Crit Care Med*. 1979; 7:218-224.
20. Sing RF, O'Hara D, Sawyer MA, Marino PL. Trendelenburg position and oxygen transport in hypovolemic adults. *Ann Emerg Med*. 1994; 23:564-567.
21. Bivins HG, Knopp R, dos Santos PA, Blood volume distribution in the Trendelenburg position. *Ann Emerg Med*. 1985; 14:641-643.
22. Reich DL, Konstadt SN, Hubbard M, Thys DM. Do Trendelenburg and passive leg raising improve cardiac performance? *Anesth Analg*. 1988; 67:S184.
23. Johnson S, Henderson SO. Myth: the Trendelenburg position improves circulation in cases of shock. *Canadian Journal of Emergency Medicine*. 2004; 6:48-49.
24. Ostrow CL. Use of the Trendelenburg position by critical care nurses: Trendelenburg survey. *Am J Crit Care*. 1997; 6:172-176.
25. Craig J. Shoulder supports, brachial plexus injury and head-down tilt. *Anaesthesia*. 2004; 59:196.
26. Madl C, Holzer M. Brain function after resuscitation from cardiac arrest. *Current Opinion in Critical Care*. 2004; 10:213-217.
27. Koehler RC, Chandra N, Guerci AD, Tsitlik J, Traystman RJ, Rogers MC, Weisfeldt ML. Augmentation of cerebral perfusion by simultaneous chest compression and lung inflation with abdominal binding after cardiac arrest in dogs. *Circulation*. 1983; 67:266-275.
28. Wenzel V, Lindner KH, Prengel AW, Strohmenger HU. Effect of phased chest and abdominal compression-decompression cardiopulmonary resuscitation on myocardial and cerebral blood flow in pigs. *Crit Care Med*. 2000; 28:1107-1112.
29. Advances in cardiovascular life support—section 8: post-resuscitation care. *Circulation*. 2000; 102(suppl I):I166-I177.
30. Wright WL, Geocadin RG. Postresuscitative intensive care: neuroprotective strategies after cardiac arrest. *Seminars in neurology*. 2006; 26:396-402.
31. Hu Z, Zhao G, Xiao Z, Chen X, Zhong C, Yang J. Different responses of cerebral vessels to -30 degrees head-down tilt in humans. *Aviat Space Environ Med*. 1999; 70: 674-680.
32. Alzaga-Fernandez AG, Varon J. Open-chest cardiopulmonary resuscitation: past, present and future. *Resuscitation*. 2005; 64:149-156.
33. Benson DM, O'Neil B, Kadish E, Erpelding J, Alousi S, Mason R, Piper D, Rafols J. Open-chest CPR improves survival and neurologic outcome following cardiac arrest. *Resuscitation*. 2005; 64:209-217.

Necrotizing Vasculitis as a Complication of Propylthiouracil

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Background: Acute dermatologic conditions are a concern for acute care practitioners. Comprising 1.4% of presenting complaints to emergency departments, most skin complaints are relatively benign; however, some conditions can be quite severe. Prompt diagnosis is essential to avoid unnecessary morbidity and mortality.

Objectives: To review drug-induced vasculitis.

Case Report: We present the case of a 43-year-old female with a chief complaint of bruising to her ear, arm, and leg. She was found to have necrotizing vasculitis induced by propylthiouracil.

Conclusion: In this case, we look at the highlights of this presentation and review key aspects of cutaneous vasculitis for the practicing emergency provider.
[*West*JEM. 2008;9:212-215.]

INTRODUCTION

Acute dermatologic conditions are a concern for all healthcare professionals. With the increased national utilization of acute care services, emergency physicians must have a basic knowledge of such disorders. In 2004, "skin rash" complaints accounted for 1.4% of all emergency department (ED) visits.[1] The majority of these conditions are benign and non-emergent, with diagnoses ranging from contact dermatitis to psoriasis. However, a small number of patients present with acute dermatologic conditions requiring rapid diagnosis and treatment.

Due to the multiple etiologies associated with dermatologic emergencies, diagnosis may often be difficult. However, one of the most common causes of dermatologic presentations to acute care facilities is a drug reaction, and it has been estimated that 10–20% of cutaneous reactions to drugs are vasculitic reactions.[2,3] The prognosis for drug-induced vasculitis is usually improved with prompt removal of the offending medication.[4] We present a case in which a 43-year-old woman with a history of Graves' disease presented to the ED after two weeks of progressive palpable purpura caused by a propylthiouracil-induced vasculitis.

CASE REPORT

A 43-year-old woman presented to the ED with a chief complaint of new-onset bruising. She had been well until two weeks prior to admission, when she developed symptoms consistent with an upper respiratory tract infection (URI). At that time the patient developed a bruise on her left calf without any history of trauma. As this started to resolve she noted some bruising on the right ear four days later. She complained that she was developing bruises along her right tricep associated with increasing redness, swelling and pain over these lesions. The patient denied any history of domestic violence, liver disease, prior bruising episodes, family history of bleeding disorders, heavy menses, or anticoagulant use. Systemically the patient denied any fevers, chills, chest pain, shortness of breath, abdominal pain, hematuria or joint complaints. Her past medical history was significant for Graves' disease, which was being followed by her endocrinologist. She also reported degenerative joint disease of her left knee. Her medications included 50-mg

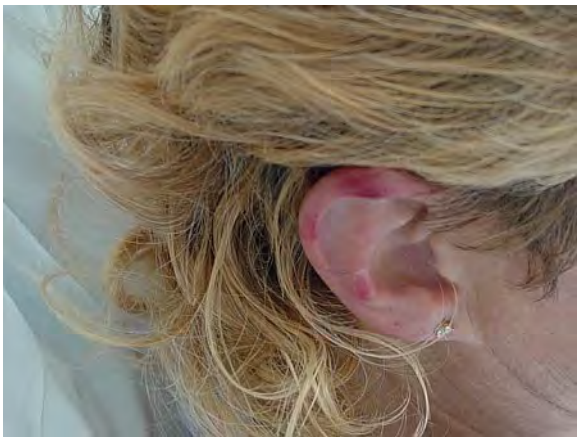

Figure 1. Skin lesion at right pinna.

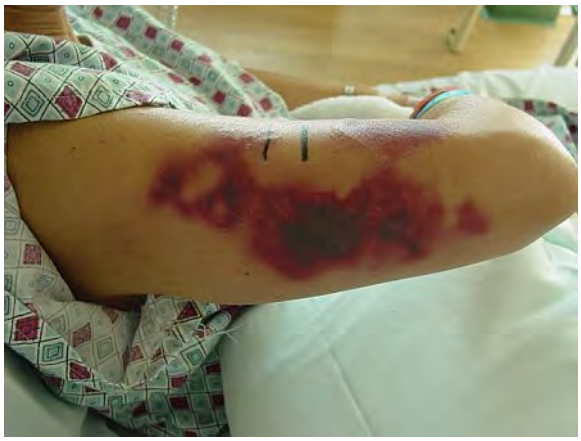

Figure 2. Skin lesion at right tricep.

propylthiouracil (PTU) each day, which she has been taking for the past nine years. She also reported the use of 400mg Motrin as needed for pain relief from her arthritis. The patient denied taking any other medications including over-the-counter medications for her recent URI symptoms. The patient stated that her mother had recently been diagnosed with diabetes and thyroid disease but denied any other family history. The patient reported a 20 pack/year tobacco history but denied any illicit drug use.

On arrival to the ED she was afebrile at 36.4°C, with a blood pressure of 116/78 mmHg, heart rate at 74 beats per minute, respirations at 14 breaths/minute with oxygen saturation at 97% on room air. On examination, she was a well-appearing woman in no acute distress. Her skin examination showed several findings. She had trace ecchymosis along the superior aspect of the helix of the right ear (Figure 1). This area was otherwise non-tender to palpation with no other abnormalities noted at the ear. She had a second ecchymotic area at her right tricep approximately 4 x 10 cm in diameter (Figure 2). The arm was tender andindurated, with some erythema at the borders of the lesion. There were no areas of fluctuance, and the neurovascular function of the arm and hand were intact. She also had one small left calf ecchymosis which was superficial and non-tender, approximately 1.5cm in diameter (Figure 3). Regarding the remainder of the examination, the patient's eyes were normal, pupils were equally round and reactive to light, and no conjunctival injection was noted. The fundoscopic examination was unremarkable bilaterally. Lungs were clear to auscultation without increase in respiratory effort. The cardiac exam showed regular rate and rhythm without murmur, rub, or gallop. The abdominal examination was non-tender with no evidence of rebound or guarding. The rectal examination was normal with guiac-negative brown stool. The neurologic examination was unremarkable.

Laboratory analyses of CBC, coagulation studies, electrolytes, urinalysis, liver function tests, and negative urine pregnancy test were unremarkable. Blood cultures were sent and eventually returned negative. Erythrocyte sedimentation rate was elevated at 54. The patient was admitted to the hospital for further, assessment and subsequent lab studies were as follows with normal ranges in parentheses. Thyroid function tests showed a TSH of 1.40 uIU/mL, a free T4 of 0.74 ng/dL (0.85-1.69), and a free T3 of 4.23 pg/mL (2.0-4.1). Antiphospholipid panel was negative. Anti-nuclear antibody screen was negative. Perinuclear-staining anti-neutrophil cytoplasmic antibodies (P-ANCA) was positive. Antithrombin III was 104% (80-120), D-dimer was 1303ng/mL (<250), and fibrinogen was 345 mg/dL (240-490).

The clinical presentation of a palpable, purpuric rash in conjunction with elevated erythrocyte sedimentation rate and ANCA titers indicated a vasculitis. In general, the differential diagnosis for vasculitis includes infectious etiologies (22.1%), autoimmune disorders (4.4%), connective tissue disease (11.4%), malignancy (4.3%), environmental exposures including medications (22.7%) or idiopathic (39% frequency).[5] In our patient, the lack of associated clinical symptoms and absence of other possible etiologic

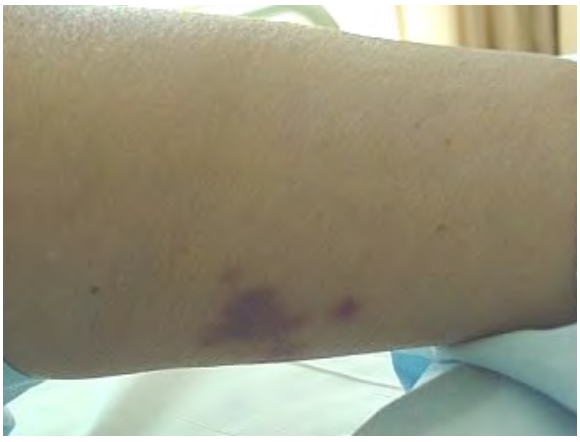

Figure 3. Skin lesion at left lower calf.

sources lead us to believe the ANCA-positive vasculitis was secondary to her PTU. Previous data suggests that vasculitic complications associated with PTU administration have a varied time of onset and have been reported anywhere from three days to seven years from the time of initiation of therapy.[6] Furthermore, despite conservative management with 60 mg prednisone per day, the patient's symptoms did not resolve until several days after admission when the PTU therapy was discontinued. The decision to continue her PTU while awaiting the diagnostic workup, rather than immediately discontinue the medication, was made due to her lack of other end-organ failure often associated with vasculitis. With the vasculitis resolving, the patient was treated with a total course of oral prednisone, 60 mg each day for 10 days, along with metoprolol, 100mg each day for her Graves' disease, and she was discharged from the hospital. The possibility of re-challenging the patient on PTU was subsequently considered after her symptoms had resolved, but given the data (albeit limited) which suggests a high recurrence of vasculitis, it was felt that other treatment options should be pursued.[6]

The patient returned for subtotal thyroidectomy to address her Graves' disease. The surgery was uneventful, and she was discharged with supplementation for hypothyroidism, hypocalcemia, and hypomagnesemia.

DISCUSSION

Vasculitis is a rare condition among the general population with one study showing an estimated lifetime incidence of biopsy-proven vasculitis at 39.6 per million; however, the number varies based on geography, tertiary vs. outpatient setting.[7] The true incidence may be higher due to the under-diagnosis of mild or subclinical presentations. The etiology of vasculitic disease can be attributed to a primary idiopathic process or to a secondary autoimmune syndrome, infectious agent, trauma, or medication. In attempting to determine an etiology, a proper history is essential. In general, drug-induced vasculitis is more likely to have cutaneous manifestation compared to idiopathic vasculitis.[8] And it is more likely to manifest with pulmonary and renal complications than musculoskeletal complaints.[9]

Drug-induced vasculitis has been reported from penicillins, sulfonamides, allopurinol, antithyroid drugs, and multiple other compounds.[10] Of the thyroid drugs propylthiouracil is the most often reported as causing drug-induced vasculitis. Ithas also been associated with other rheumatic manifestations, including serum sickness and PTU-induced lupus.[9] There is some debate whether PTU is the primary cause of rheumatologic disease or if the increased incidence is due to the underlying thyroid disease of the patients, which has been shown to predispose patients to other autoimmune conditions. PTU has been associated with various other inflammatory conditions, such as agranulocytosis, aplastic anemia, hepatitis, interstitial pneumonitis, and others.

The mechanism of PTU-induced vasculitis is not clear. Studies have shown that PTU can accumulate in neutrophils. This may allow PTU to bind to myeloperoxidase (MPO), which when oxidized can inactivate this enzyme. This inactivation with the continued presence of PTU can induce the formation of auto-antibodies, thereby stimulating other neutrophils to degranulate, causing vascular damage.[4,6,10,11,12] Clinically the development of the MPO-ANCA antibodies are more prevalent in the serum of patients treated with PTU, and studies have demonstrated that MPO-ANCA positivity was increased after being treated by PTU as compared to other hyperthyroid medications.[9,11,13] It should be noted, however, that not all patients with these antibodies developed clinical symptoms of PTU-induced rheumatic disease, which contraindicates the use of these auto-antibodies as a screening tool.[14]

There are currently 44 cases of PTU-induced vasculitis in the literature since 2002, with an apparent female and Japanese-ethnic predominance.[6] This gender predominance is likely attributed to the higher incidence of thyroid disease in women. It is unknown why Japanese ethnicity, which accounts for half of all reported cases, predisposes patients.[11] There may be a genetic predisposition to the formation of auto-antibodies, or it may be caused by variations in treatment regimens. The age of onset varies but appears to have a higher incidence in younger patients; however, the reported age range is 13-80 years old.[6,14] Of note, the development of PTU-induced vasculitis is hypothesized to be independent of the dosage of PTU prescribed or the duration of PTU treatment, based on the limited data available.[6]

The clinical presentation can be varied and complex due to systemic complications and multi-organ involvement of small-vessel disease. Signs of PTU-induced vasculitis include anemia (seen in the majority of patients), skin lesions (ulcers, purpura, subcutaneous nodules, and erythema nodosum-like and erythema multiforme-like lesions), arthralgia, fever, malaise, lymphadenopathy, hematuria/proteinuria, alveolar hemorrhage, and pleural effusion.[4,6,14] Multiple organ systems can be affected, including renal failure, cholestatic jaundice, cardiac dysfunction, pleural effusions, pneumonitis/pleuritis, and hearing loss. The most common laboratory abnormalities include neutropenia, anemia, thrombocytopenia, and elevated transaminases[10] (although our patient demonstrated none of these). Histologically the majority of patients developed crescentic glomerulonephritis or other forms of renal changes.[4,14]

Clinical diagnosis is often difficult because of the overlapping presentations of the various vasculitic diseases. Although biopsy is diagnostic, in most cases the diagnosis of drug-induced vasculitis is made by exclusion or with resolution of symptoms after withdrawal of medications. Once PTU-induced vasculitis is determined, the simple withdrawal of PTU usually causes resolution of the

symptoms after 1-4 weeks.[3] Some patients have been reported to have continued laboratory abnormalities (elevated serum creatinine, proteinuria, positive ANCA titers) throughout their clinical follow-up without evidence of clinical symptoms.[3] Patients with more severe presentations are typically treated with steroids. When considering treatment for Gravesí disease after resolution of their symptoms, re-administration of PTU is not recommended because it often causes a repeat reaction;[6,10,11] however, there does not appear to be cross-reactivity between agents.[2,10] Therefore, consideration of treatment should be a multi-disciplinary approach to weigh the risk and benefits of further medical treatment versus surgical intervention.

CONCLUSION

Drug-induced vasculitis should be considered in any patient who takes medications and develops cutaneous eruptions. Emergency practitioners should be able to differentiate benign skin conditions from those that are potentially progressive, causing morbidity and possibly mortality.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources, and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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REFERENCES

1. McCaig L, Nawar E. National hospital ambulatory medical care survey: 2004 emergency department survey. CDC Advance Data. *Vital and Health Statistics*. 2006; Number 372.
2. Dubost JJ, Souteyrand P, Sauvezie B. Drug-induced vasculitides. *Baillieres Clin Rheumatol*. 1991; 5:119-138.
3. Ekenstam E, Callen JP. Cutaneous leukocytoclastic vasculitis. Clinical and laboratory features of 82 patients seen in private practice. *Arch Dermatol*. 1984; 120:484-489.
4. Otsuka S, Kinebuchi A, Tabata H, Yamakage A, Yamazaki S. Myeloperoxidase-antineutrophil cytoplasmic antibody-associated vasculitis following propylthiouracil therapy. *Br J Dermatol*. 2000; 142:828-830.
5. Carlson JA, Ng BT, Chen KR. Cutaneous vasculitis update: diagnostic criteria, classification, epidemiology, etiology, pathogenisis, evaluation and prognosis. *Am J Dermatopathol*. 2005; 27: 504-528.
6. ten Holder SM, Joy MS, Falk RJ. Cutaneous and systemic manifestations of drug-induced vasculitis. *The Annals of Pharmacotherapy*. 2002; 36:130-147.
7. Watts RA, Jolliffe VA, Grattan CE, Elliot J, Lockwood M, Scott DG. Cutaneous vasculitis in a defined population—clinical and epidemiological associations. *J Rheumatol*. 1998; 25:920–924.
8. Wiik A. Clinical and laboratory characteristics of drug-induced vasculitic syndromes. *Arthritis Research & Therapy*. 2005; 7**:**191-192.
9. Aloush V, Litinsky I, Caspi D, Elkayam O. Propylthiouracil-induced autoimmune syndromes: two distinct clinical presentations with different course and management. *Semin Arthritis Rheum*. 2006; 36:4-9.
10. Hardy OT, Smolinski KN, Yan AC, Grimberg A. PTU-associated vasculitis in a girl with turner syndrome and graves' disease. *Pediatric Emergency Care*. 2006; 22:52-54.
11. Sato H, Hattori M, Fujieda M, Shigetaka S, Inomata H, Hoshi M et al. High prevalence of antineutrophil cytoplasmic antibody positivity in childhood onset graves' disease treated with propylthiouracil. *The Journal of Clinical Endocrinology & Metabolism*. 2000; 85:4270-4273.
12. Sheen YS, Chu CY, Yu HS. Antineutrophil cytoplasmic antibody–positive cutaneous leukocytoclastic vasculitis associated with propylthiouracil therapy. *Arch Dermatol*. 2006; 142:879-880.
13. Harper L, Chin L, Daykin J, Allahabadia A, Heward J, Gough SC et al. Propylthiouracil and carbimazole associatedantineutrophil cytoplasmic antibodies (ANCA) in patients with Graves' disease. *Clinical Endocrinology*. 2004; 60:671–675.
14. Nakamori Y, Tominaga T, Inoue Y, Shinohara K. Propylthiouracil (PTU)-induced vasculitis associated with antineutrophil antibody against myeloperoxidase (MPO-ANCA). *Internal Medicine*. 2003; 42:529-533.

Monomorphic Ventricular Tachycardia

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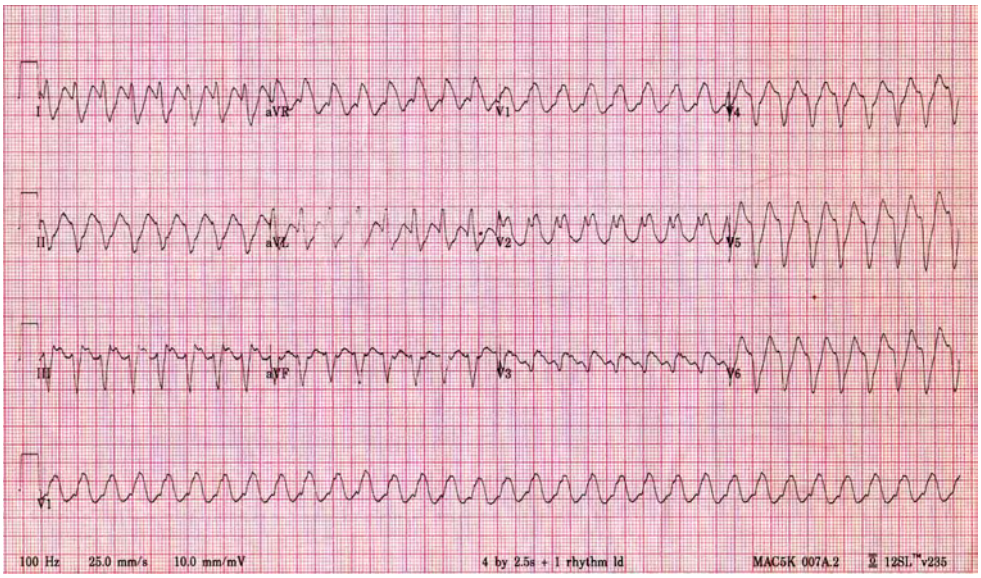

An 83-year-old male with known coronary artery disease presented to our emergency department (ED) with a complaint of palpitations and associated minor chest discomfort while on his way to play a round of golf. Vital signs were significant for a blood pressure at 122/76 mmHg, heart rate at 180 beats per minute, respiration rate at 20 breaths per minute, temperature at 37ºC, and SpO2 at 98% on room air. A 12-lead ECG showed a wide-complex tachycardia with no clearly discernible P waves and was thought to represent monomorphic ventricular tachycardia. Initial treatment with procainamide was attempted; however, within minutes of receiving this medication, his blood pressure dropped to 70/40 mmHg. Procedural sedation using etomidate and fentanyl, followed by electrical synchronized cardioversion using a biphasic defibrillator at 200J, successfully converted his rhythm to a sinus rhythm.

The differential diagnosis for a wide-complex (i.e. QRS 0.12sec) regular tachycardia with no clearly discernible P waves prior to each QRS complex includes ventricular tachycardia, supraventricular tachycardia with preexisting or rate-related bundle branch block (e.g. SVT with LBBB), and supraventricular tachycardia with antidromic conduction through an accessory pathway (e.g. antidromic WPW). Several proposed criteria may aid in the differentiation of these entities.[1,2] Contemporary teaching, however, mandates that a wide-complex tachycardia should be presumed ventricular tachycardia if the diagnosis is ambiguous. [3]

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REFERENCES

1. Brugada P, Brugada J, Mont L, Smeets J, and Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation*. 1991; 83:1649-1659.
2. Vereckei A, Duray G, Szenasi G, Altemose GT, and Miller JM. Application of a new algorithm in the differential diagnosis of wide-QRS complex tachycardia. *Eur Heart J*. 2007; 28:589-600.
3. Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/ American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines. *Circulation*. 2006; 114:e385– e484.

Spontaneous Pneumomediastinum

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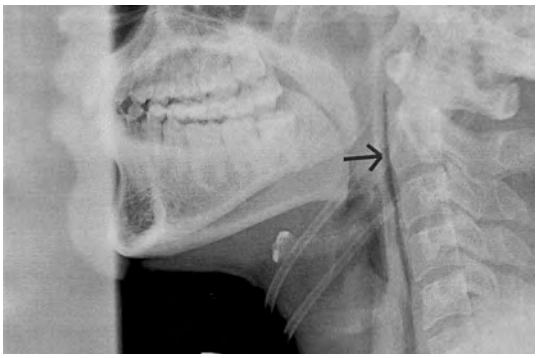

Figure 1. Lateral soft tissue neck illustrating retropharyngeal free air.

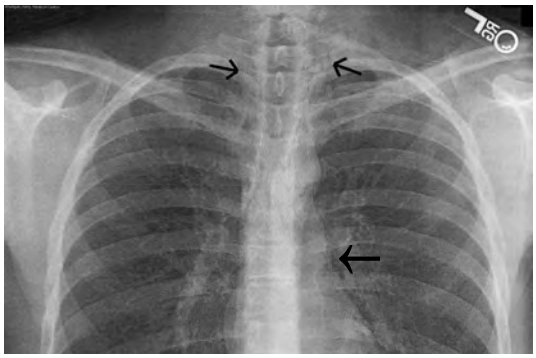

Figure 2. PA CXR showing pneumopericardium and free air in cervical soft tissues

A 23-year-old male presented to the emergency department (ED) with sore throat, chest pain and shortness of breath that started two days prior to his arrival and gradually increased in severity. Other symptoms included pleuritic chest pain, back pain, dysphagia, odynophagia and general air hunger. The patient denied any history of trauma or similar symptoms.

Spontaneous pneumomediastinum (SPM) is an uncommon condition presenting in approximately one in 1,000 to one in 40,000 ED referrals.[1,2,3] Young patients with SPM typically present with a history of asthma or recent inhalation of cocaine, methamphetamine, ecstasy, marijuana or hydrocarbons.[4,5,6,7] Other causes include barotrauma in asthmatics and COPD patients, rapid ascent in scuba divers, valsalva maneuvers, vomiting, infections, blast injuries and iatrogenic injuries from endoscopy or surgery.[8] The most common presentation is nonspecific pleuritic chest pain with dyspnea. Potential life-threatening etiologies include esophageal rupture and tension pneumothorax, but these are historically evident at presentation.[9-12] Because a subset of patients with this finding have significant pathology, extensive workups are often necessary. Treatment is generally limited to observation, with the SPM typically reabsorbing over a period of one to two weeks without intervention and only rare recurrence.[13]

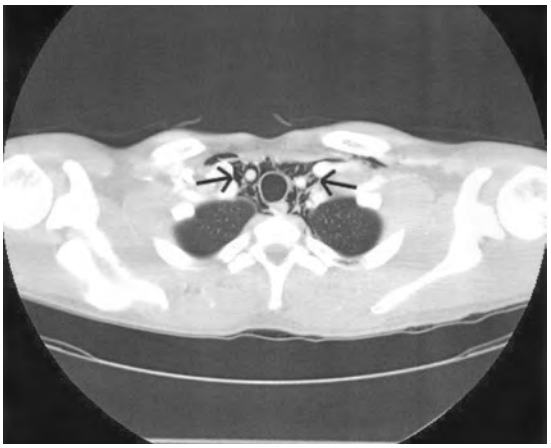

Figure 3. CT upper thorax with pneumomediastinum

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REFERENCES

1. Newcomb AE. *Chest.* 2005; 128:3298-3302.
2. McMahon DJ. Spontaneous pneumomediastinum. *Am J Surg*. 1976; 131:550-551.
3. Bodey GP. Medical Mediastinal Emphysema. *Ann Intern Med.* 1961; 54:46–56.
4. Harris R, Joseph A. Spontaneous pneumomediastinum: ecstasy; a hard pill to swallow. *Aust N Z J Med.* 2000; 30:401–403.
5. Mir B, Galvete V, Plaza V, et al. Spontaneous pneumomediastinum after cocaine inhalation. *Respiration.* 1986; 50:230–232.
6. Millar N, Spiakeman P, Hepper N. Pneumomediastinum resulting from performing valsalva maneuvers during marijuana smoking. *Chest.* 1972; 62:233–234.
7. Badaoui R, El Kettani C, Fikri M, et al. Spontaneous cervical and mediastinal air emphysema after ecstasy abuse. *Anesth Analg.* 2002; 95:1123.
8. Bartelmaos T, Blanc R, Claviere GD, Benhamou D. Delayed pneumomediastinum and pneumothorax complicating laparoscopic extraperitoneal inguinal hernia repair. *J Clin Anesth*. 2005; 17:209-212.
9. Hamman L. Spontaneous mediastinal emphysema. *Bull Johns Hopkins Hosp.* 1939; 64:1–21.
10. McMahon DJ. Spontaneous pneumomediastinum. *Am J Surg*. 1976; 131:550–551.
11. Henderson JAM, Péloquin AJM. Boerhaave revisited: spontaneous esophageal perforation as a diagnostic masquerader. *Am J Med*. 1989; 86:559–567.
12. Munsell WP. Pneumomediastinum: a report of 28 cases and review of the literature. *JAMA*. 1967; 202:129–133.
13. Lee CC, Chen TJ, Wu YH, Tsai KC, Yuan A. Spontaneous retropharyngeal emphysema and pneumomediastinum presented with signs of acute upper airway obstruction. *Am J Emerg Med*. 2005; 23: 402-404.

Ultrasound-Assisted Peripheral Venous Access in Young Children: A Randomized Controlled Trial and Pilot Feasibility Study

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Objectives: Intravenous (IV) access in children treated in the emergency department (ED) is frequently required and often difficult to obtain. While it has been shown that ultrasound can be useful in adults for both central and peripheral venous access, research regarding children has been limited. We sought to determine if the use of a static ultrasound technique could, a) allow clinicians to visualize peripheral veins and b) improve success rates of peripheral venous cannulation in young children in the ED.

Methods: We performed a randomized clinical trial of children < 7 years in an academic pediatric ED who required IV access and who had failed the first IV attempt. We randomized patients to either continued standard IV attempts or ultrasound-assisted attempts. Clinicians involved in the study received one hour of training in ultrasound localization of peripheral veins. In the ultrasound group, vein localization was performed by an ED physician who marked the skin overlying the target vessel. Intravenous cannulation attempts were then immediately performed by a pediatric ED nurse who relied on the skin mark for vessel location. We allowed for technique cross-over after two failed IV attempts. We recorded success rate and location of access attempts. We compared group success rates using differences in 95% confidence intervals (CI).

Results: We enrolled 44 children over a one-year period. The median age of enrollees was 9.5 months. We visualized peripheral veins in all patients in the ultrasound group (n=23) and in those who crossed over to ultrasound after failed standard technique attempts (n= 8). Venipuncture was successful on the first attempt in the ultrasound group in 13/23 (57%, CI, 35% to 77%), versus 12/21 (57%, CI, 34% to 78%) in the standard group, difference between groups 0.6% (95% CI -30% to 29%). First attempt cannulation success in the ultrasound group was 8/23 (35%, CI, 16% to 57%), versus 6/21 (29%, CI, 11% to 52%) in the standard group, difference between groups 6% (95% CI -21% to 34%).

Conclusion: Ultrasound allows physicians to visualize peripheral veins of young children in the ED. We were unable to demonstrate, however, a clinically important benefit to a static ultrasound aided vein cannulation technique performed by clinicians with limited ultrasound training over standard technique after one failed IV attempt in an academic pediatric ED.
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INTRODUCTION

Phlebotomy and placement of an intravenous (IV) catheter are among the more common and yet more painful procedures performed in the pediatric emergency department (ED). Despite the routine nature of these procedures, phlebotomy and IV access continue to be uncomfortable and at times difficult to perform, particularly in young children. In the most critically ill or injured children, venous access is of great importance and various techniques are routinely used to establish access.[1-3]

Previous studies in both children and adults suggest that ultrasound offers an advantage over blind techniques when placing an IV catheter into central veins of the neck and groin.[4-7] In these situations, ultrasound allows the target vessel to be directly visualized in contrast to standard methods in which location is estimated based on superficial anatomic relationships. Additionally, initial research in adults has suggested that ultrasound can also improve success rates in peripheral venous access.[8-10] To date only one study in children has assessed the association between ultrasonographic venous visualization and venous cannulation success;[11] however, there have been no randomized clinical trials on this topic in children .

We performed a randomized clinical trial to determine if a static ultrasound technique could accomplish two things: (a) allow clinicians to visualize peripheral veins in young children; and (b) improve success rates of phlebotomy and peripheral venous cannulation in children treated in the ED after a failed IV attempt. We hypothesized that ultrasound visualization would improve success rates of phlebotomy and IV cannulation.

METHODS

Study design

We performed a randomized trial of children younger than seven years old who had already undergone one failed IV attempt in the ED. We randomized patients to either continued standard IV attempts or ultrasound-assisted attempts using a "static" ultrasound technique. This trial has been registered with ClinicalTrials.gov (#NCT00557154).

Setting

The study was conducted at an academic, urban, Level 1 trauma center with an ED census of approximately 60,000, of whom 13,000 are children seen in the pediatric ED. The core nursing staff in the pediatric ED participating in this study has specialized training and experience in pediatrics.

Selection of Participants

All patients younger than seven years old who required IV access and who had failed an initial IV attempt, and were judged to be hemodynamically stable by the enrolling physician, were eligible for the study. We obtained written informed consent from the parent or guardian for each eligible patient. The study was approved by the Institutional Review Board.

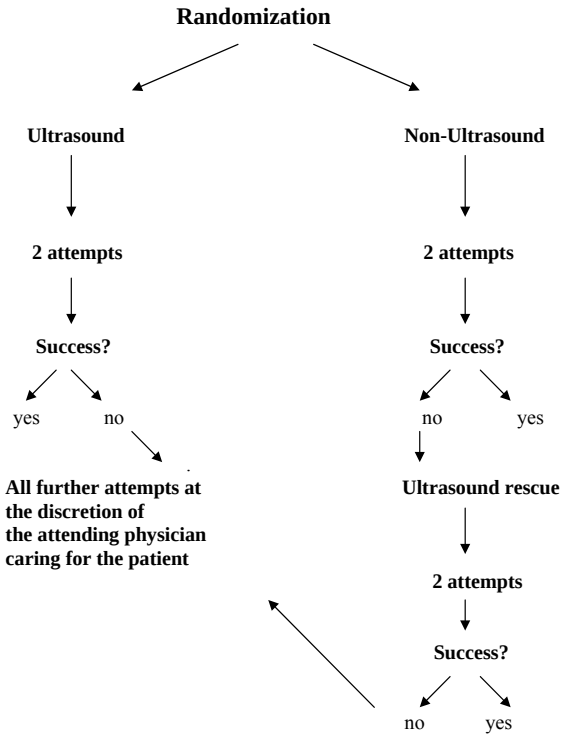

Figure 1. Randomization scheme

We used a computerized randomization scheme to determine placement into either the ultrasound-assisted or standard therapy arms. The random group assignment was placed into sealed, opaque enrollment packets that were used in sequence according to order of patient presentation. We permitted technique cross-over after two failed attempts in the assigned arm of the trial (Figure 1).

We based our sample-size calculations on an anticipated clinically important absolute difference of 25% in IV cannulation success rates between groups (i.e. improvement from 50% to 75%). We estimated that to have a power of 80% with an alpha of .05, a sample size of 132 would be required to detect this difference. Based on the patient volume at the participating ED, we anticipated that we would need one year of patient enrollment. We enrolled patients during times of participating staff availability from August 2003 through July 2004.

Interventions

All participating physicians and nurses underwent an initial training program. As staff familiarity with ultrasound was not uniform, we assumed no previous knowledge with the

technology or technique. All six of the participating physicians were board-certified emergency physicians, and three of them were board certified in pediatric emergency medicine. All participating physicians attended an approximately one-hour orientation presentation on the use of the ultrasound equipment and concepts related to peripheral vein visualization and identification, and accurate skin marking. Each participating physician demonstrated proficiency by visualizing the veins (hand, forearm and antecubital fossa) of at least one adult and one child during training. The participating nurses were part of the regular pediatric emergency department nursing staff and were instructed on the details of the study, including patient recruitment, and given an overview of the ultrasound technique. The objective of the physician and nurse training was to provide sufficient instruction to be realistic in a "real world" setting but not to create ultrasound expertise. The participating nurses were not involved in ultrasound-assisted vein localization. Vein localization in the ultrasound-assisted study arm was performed by a participating physician, who then marked the overlying skin with a dimple impression from the barrel of a pen. Venous anatomy was identified on the basis of selecting the most apparent vessel on the most accessible extremity. The participating nurses then immediately used the skin impression as a landmark for subsequent IV access attempts. Patient movement was limited to prevent relative movement of the target vein with respect to overlying skin mark. The anatomical site chosen for all attempts at venous access was at the discretion of the treatment team and was not limited by study protocol.

Ultrasound Technique

We used a 10 MHz linear transducer (Sonosite iLOOK 25, Bothell, Washington) for imaging in this trial. We employed a static "no touch" technique which involved placing a large acoustic gel ridge between the transducer and the skin (Figure 2). This large gel ridge provides a small "stand off," thus allowing better superficial vein visualization by better accommodating the inherent focal zone of the transducer. Additionally, it avoids direct contact on the skin, which inevitably collapses the underlying superficial veins (Figure 3). We did not perform the "dynamic" technique (real time venous visualization and cannulation while the ultrasound probe was in place) for this study because of the limited physical space in the extremities of small children. Our pretrial experience was that functional working space on the small child's extremity was limited, and could not typically accommodate the simultaneous use of the transducer during venipuncture.

Outcome Measures

The main outcomes of interest in this trial were the following: 1) venipuncture success rates, and 2) venous cannulation success rates. We documented the total number of skin punctures and needle passes for each attempt. In the ultrasound study arm, successful vessel sonographic visualization was required prior to subsequent puncture attempts.

We also documented IV site location of successive attempts. Among patients undergoing multiple venipuncture attempts, we monitored the pattern of anatomical progression (e.g. distal to proximal), as the protocol did not dictate this progression.

The participating nurses also reported their subjective

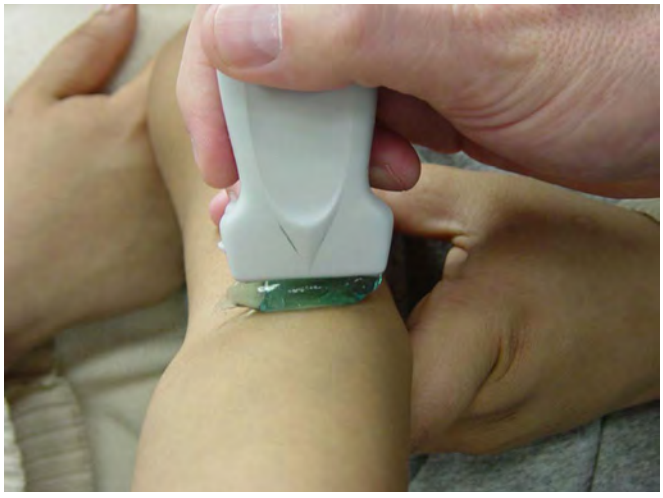

Figure 2. The static "no touch" technique. Note the thick ridge of acoustic gel between the transducer and the patient's skin. The thick layer of gel improves vessel visualization and ensures that the target vein will not be inadvertently compressed.

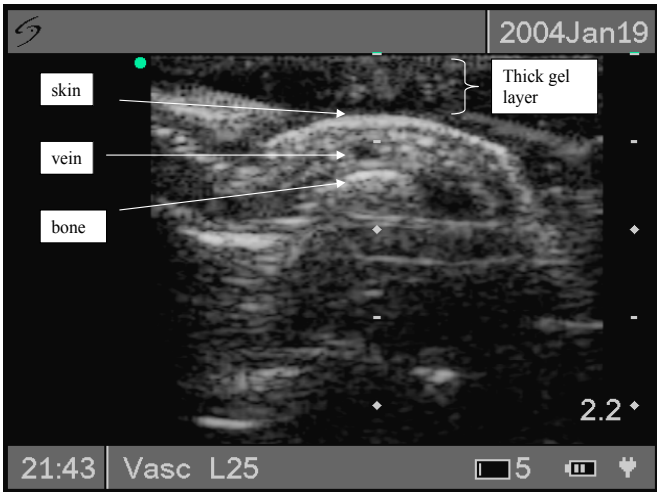

Figure 3. Ultrasound image of target vessel

ratings of anticipated difficulty of venipuncture before the procedure and rated the difficulty after the completion of the venipuncture. We used nursing-perceived difficulty to reflect the combined assessment of difficulty of vein visualization and palpation. We tracked this information for all venous cannulation attempts. In particular, we were interested in successful cannulations that were expected to be "very difficult" or "difficult" on a 5-point Likert scale (anchored by "Very Easy" and "Very Difficult").

Primary Data Analysis

We considered venipuncture to be successful if a sufficient amount of blood was obtained for laboratory analysis. We considered venous cannulation to be successful if the nurse was able to successfully cannulate the vessel as indicated by the ability to infuse fluid ("flush") into the inserted catheter.

We report counts and percentages with 95% confidence intervals (CI) and compared success rates between groups using the 95% CI of the difference in success rates. Non-parametric analyses were performed using the Wilcoxon Rank Sum test. We analyzed the data using Stata software (StataCorp. 2003. Stata Statistical Software; Release 8. College Station, TX).

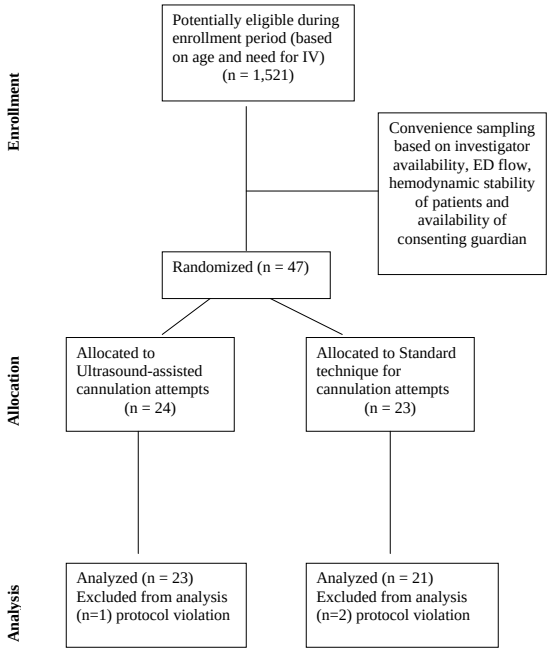

Figure 4. Diagram of patient flow through the trial

RESULTS

We randomized 47 children over a one-year period (Figure 4). Three of these were excluded from analysis due to protocol violations (N = 44). The overall median age of enrollees was 9.5 months (range one to 60 months). The various patient characteristics between study arms are shown in Table 1.

We ultrasonographically visualized peripheral veins in all patients randomized to the ultrasound group (n=23), as well as in those who crossed over to ultrasound after failed standard technique (n=8). The first "study" venipuncture attempt (i.e. all patients in this study had failed a first routine IV attempt) in the ultrasound group was successful in 13/23 (57%, 95% CI, 35% to 77%), compared to 12/21 (57%, 95% CI, 34% to 78%) in the standard group, with a difference between groups of 0.6% (95% CI -30% to 29%). First study cannulation success rate in the ultrasound group was 8/23 (35%, 95% CI, 16% to 57%), compared to 6/21 (29%, 95% CI, 11% to 52%) in the standard group, with a difference between groups of 6% (95% CI -21% to 34%).

Among the eight patients in the standard group who were crossed over to ultrasound, the "next attempt" venipuncture was successful in six, or 75% (95% CI, 35% to 97%). All sonographically-visualized veins were apparent at or distal to the antecubital fossa in the upper extremities. Likewise, in the lower extremities they were apparent at or distal to the ankle.

We tracked the pattern of progressive attempts at venipuncture and IV catheter insertion during the study. Approximately equal numbers of patients in both groups had venipuncture attempts that progressed from distal sites to proximal sites relative to initial venipuncture attempts (ultrasound = 6, standard = 5). Additionally, equal numbers of patients had anatomically neutral progression (i.e. attempt in hand with subsequent attempt in contralateral hand).

With respect to anticipated ease of puncture, we found that four of 18 (22%) (95% CI, 6% to 48%) cannulations in the ultrasound group versus 0 of 17 (95% CI, 0% to 20%) in the standard group (difference between groups 22% (95% CI, 3% to 41%)) were expected by nurse participants to be "difficult" or "very difficult" but were actually found to be "easy" or "very easy".

Table 1. Patient demographics. Age represented in months (m) with interquartile ranges (IQR). Weights are listed in kilograms (k). Hypoperfusion based on signs of poor capillary refill and volume status.

	Ultrasound (n = 23)	Standard (n = 21)	
Age	14 m (IQR, 7-24)	7 m (IQR, 5-13)	P = 0.05
Weight (k)	10 k (IQR, 8.2-12.2)	8.5 k (IQR, 5.8-11)	P = 0.2
Primary trauma -related diagnosis	2	2	
Hypoperfusion	3	5	

DISCUSSION

In this study in an academic pediatric ED, we were able to use ultrasound to visualize the peripheral veins of young children who require IV access in the ED setting after failing one IV attempt. We could not demonstrate, however, a significant difference in cannulation success between the ultrasound-assisted and the standard technique group. In our study we attempted to enroll a patient population that would potentially represent difficult venous cannulation (as defined by young patient age and the requirement of a failed previous attempt), but we were unable to achieve a sufficiently large sample size. Despite these limitations, we documented a number of cannulation attempts in which the use of ultrasound allowed IV cannulation to be easy when it was anticipated to be difficult.

Obtaining intravenous access in ill or injured children is a fundamental skill for providers who work in an emergency environment. Several techniques have been developed to assist with difficult IV access in children, and multiple techniques have been studied. Transillumination techniques have been studied and have met with variable success.[12,13] In one study, only 40% of the enrolled patients had visible palm veins with transillumination.[12] Additionally, nitroglycerin has been used as a venodilation agent and has been shown to increase the diameter of target veins.[14,15] However, adverse systemic side effects with topical nitroglycerin have been demonstrated, and its use has largely fallen into disfavor. Warming of the extremity is also often used to dilate peripheral vessels, but to our knowledge no controlled studies in the pediatric literature exists. One study demonstrated a moderate advantage of hand warming in adults.[16]

While multiple prior studies have shown that ultrasound is a useful adjunct for central venous access in both children and adults, there has only been one previous report that documents ultrasound use for identifying peripheral vessels for IV cannulation in young children. In their observational study using two extensively trained investigators to perform all the ultrasound visualizations, Schnadower and colleagues demonstrated that greater venous length was associated with higher rate of successful IV cannulation.[11] They too found that ultrasound is capable of detecting peripheral veins in this age group of children. In contrast to the Schnadower study, however, ours was a randomized trial effectiveness study, using a variety of pediatric emergency medicine clinicians with just one dedicated hour of training in ultrasound-assisted peripheral venous access in young children.

LIMITATIONS

Our study has several limitations, the most important of which was small study size. Mainly for this reason, we consider this randomized controlled trial a pilot and feasibility study, with need for further study. There were a few apparent reasons for our limited enrollment. Our pool of potential enrollees was decreased somewhat by staff members who were "early adopters" of the use of ultrasound routinely for IV placement. In fact, currently in the ED of the study institution, there is a nursing protocol for use of ultrasound for placement of difficult IVs. Unfortunately, we had smaller power than anticipated to detect the *a priori* defined minimal clinically important difference of 25% (i.e. improvement from 50-75%) in cannulation rates between groups. We were, however, able to visualize peripheral veins with the ultrasound in all patients randomized to this group, and use of ultrasound allowed IV cannulation to be "easy" when it was anticipated to be "difficult" in a substantial number of attempts compared to control patients. These two findings by themselves have important clinical implications.

We used the "static" ultrasound technique for identifying and cannulating vessels, in which the vessel is identified and the skin overlying marked with a pen tip, with the nurse immediately attempting cannulation to minimize misalignment of the skin mark with the localized vessel. Other studies that have shown ultrasound to be useful for venous cannulation in adults have used a "dynamic technique," in which the IV attempt occurs with real time visualization of the vessel. The small size of children's hands and feet precluded the use of a dynamic ultrasound technique. Instead we employed a static technique that relied on a single skin mark. However, an additional skin mark (i.e. two marks) might have provided better information regarding vein position and improved our cannulation success rates. Investigators should take this point into consideration when designing future trials.

Furthermore, randomization after just one failed IV attempt may have biased this study towards the null, as it is not uncommon for nurses to fail on the first IV attempt and succeed in the subsequent attempt without any visualization adjuncts. Randomization after two failed IV attempts rather than one, however, may have resulted in the ultrasound group appearing relatively better than the control group, as the success rate in the control group after two failed IV's would have possibly been relatively less than in the ultrasound group. In our efforts to make this a "real world" effectiveness study, we provided only one hour of formal training in ultrasound visualization of peripheral vessels to the clinicians involved in this study. Perhaps more intensive or prolonged training would have increased the success rate in the ultrasound group of this study. However, the consequence of "overtraining" may have resulted in a study that would be less generalizable. While our findings suggest that the currently available ultrasound technology allows clinicians to visualize even the small superficial veins in children, successful cannulation requires multiple additional steps. These require operator comfort and experience with ultrasound, venipuncture skills and other issues that depend on the experience of the clinician performing the procedure.

In this trial, we did not attempt to track or identify reasons

why an access attempt failed. Although the participating physicians received training in ultrasound use and the nurses had specialized pediatric skills, we did not measure or control for issues related to experience. Additionally, we could not control for potential reporting bias related to anticipated venipuncture difficulty. We acknowledge that success or failure of venipuncture might have affected subsequent reporting. Finally, the lack of observable differences between groups in this study may have also been due to the difficulty of trying to distinguish success rates compared to a standard technique performed by skilled specialized pediatric emergency nurses. Differences between groups (favoring the ultrasound group) may have been greater if nurses with less pediatric experience had participated in this study.

CONCLUSION

In summary, in this pilot and feasibility randomized controlled trial, we report our initial experience with one technique for ultrasound-assisted peripheral venous access in children after a failed IV attempt. While we were able to adequately visualize peripheral veins in all children in the study population, we were not able to show a difference in our primary outcome measures of cannulation or venipuncture success rates. However, we were able to demonstrate adequate vein visualization in all patients.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources, and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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REFERENCES

1. Brunette DD, Fischer R. Intravascular access in pediatric cardiac arrest. *Am J Emerg Med*. 1988; 6:577-579.
2. Rossetti V, et al. Difficulty and delay in intravenous access in pediatric arrests. *Ann Emerg Med*. 1984; 13:406.
3. Lozon MM. Pediatric Vascular Access and Blood Sampling Techniques. In: Roberts J, Hedges J, eds. *Clinical Procedures in Emergency Medicine*. 4th ed. Philadelphia, PA: Saunders; 2004:357-383.
4. Hudson PA, Rose JS. Real-time ultrasound guided internal jugular vein catheterization in the emergency department. *Am J Emerg Med*. 1997; 15:79-82.
5. Denys BG, Uretsky BF, Reddy PS. Ultrasound-assisted cannulation of the internal jugular vein. A prospective comparison to the external landmark-guided technique. *Circulation*. 1993; 87:1557-1562.
6. Rothschild J. Ultrasound Guidance of Central Vein Catheterization. *AHRQ.* 2001; Available at: http://www.ahcpr.gov/clinic/ptsafety/chap21.htm. Accessed October 2003.
7. Abboud PA, Kendall JK. Ultrasound guidance for vascular access. *Emerg Med Clin North Am.* 2004; 22: 749-773.
8. Costantino TG, et al. Ultrasonography-guided peripheral intravenous access versus traditional approaches in patients with difficult intravenous access. *Ann Emerg Med.* 2005; 46:456-461.
9. Brannam L, Blaivas M, Lyon M, Flake M. Emergency nurses' utilization of ultrasound guidance for placement of peripheral intravenous lines in difficult-access patients. *Acad Emerg Med.* 2004; 11:1361-1363.
10. Keyes LE, Frazee BW, Snoey ER, Simon BC, Christy D. Ultrasound-guided brachial and basilic vein cannulation in emergency department patients with difficult intravenous access. *Ann Emerg Med.* 1999; 34:711-714.
11. Schnadower D, Lin S, Perera P, Smerling A, Dayan P. A pilot study of ultrasound analysis before pediatric peripheral vein cannulation attempt. *Acad Emerg Med.* 2007; 14:483-485.
12. Goren A, Laufer J, Yativ N, Kuint J, Ben Ackon M, Rubinshtein M, Paret G, Augarten A. Transillumination of the palm for venipuncture in infants. *Pediatr Emerg Care*. 2001; 17:130-131.
13. Atalay H, et al. The use of transillumination for peripheral venous access in paediatric anaesthesia. *Eur J Anaesthesiology.* 2005; 22:317-318.
14. Vaksmann G, et al. Nitroglycerine ointment as aid to venous cannulation in children. *J Pediatr.* 1987; 111:89-91.
15. Maynard EC, Oh W. Topical nitroglycerin ointment as an aid to insertion of peripheral venous catheters in neonates. *J Pediatr.* 1989; 114:474-476.
16. Lenhardt R, et al. Local warming and insertion of peripheral venous cannulas: single blinded prospective randomised controlled trial and single blinded randomised crossover trial. *BMJ.* 2002; 325:409-410.

Acute Ischemic Stroke in a Pediatric Patient

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Acute ischemic stroke in a pediatric patient is a complex disease with a variety of etiologies that differ from adults. Though rare, they are a real phenomenon with potentially devastating consequences. Some treating institutions are using anti-thrombotic drug therapy with unclear benefits. Available literature, which is limited to case reports and retrospective reviews of databases, clouds this topic with both positive and negative outcomes. Emergency department management should focus on stabilization and resuscitation with immediate involvement of a pediatric neurologist and intensivist. The decision to use anti-thrombotic drug therapy, including anti-platelet drugs and thrombolytics, should be in consult with the specialists involved until randomized controlled trials determine their safety and efficacy in the pediatric population.
[*West*JEM. 2008;9:225-227.]

INTRODUCTION

Acute ischemic stroke (AIS) in a pediatric patient is a rare medical emergency with an incidence of only 2-3 per 100,000.[1] Cognitive and behavioral sequelae frequently arise with social implications and effects on daily living.[2] The etiologies of stroke in a child are more varied than in adults and are not always due to acute clot formation or hemorrhage. Literature on this topic, including the use of anti-thrombotic therapy for children presenting with AIS, is sparse. Studies in the adult population show anti-thrombotic drugs, including aspirin, are effective when administered using recommended guidelines.[3] The role of thrombolytics is controversial with conflicting evidence.[4,5,6] This case report describes an 11-year-old boy who presented to the emergency department (ED) of a small rural community with the diagnosis of an acute ischemic stroke. After transfer to a children's hospital and an exhaustive workup, the etiology of his stroke remained unclear. At four-month outpatient follow-up, the patient had only minimal left arm weakness. Background information on AIS in the pediatric population is presented, including ED management and the role of anti-thrombotic drug therapy.

CASE

An 11-year-old Hispanic male presented to the ED 30 minutes after the sudden onset of headache, left-sided weakness and dysarthria. His vital signs were within normal limits. Left arm and leg strength were 2/5 with a noticeable facial droop. Cardiac exam showed a regular rate and rhythm with no murmurs. Chest was clear and abdomen was soft. No petechiae were noted on his skin. Fingerstick blood glucose was normal. A CT scan of the head showed edema to the right frontal lobe without evidence of hemorrhage. Transfer for higher level of care was initiated with the presumptive diagnosis of acute ischemic stroke. A long transport time placed him at the nearest children's hospital eight hours after symptom onset. An emergent MRI showed findings consistent with a right frontal lobe and basal ganglia ischemic infarction (Figure 1). The patient was given aspirin, empiric intravenous antibiotics and antiviral medications in the ED. A thorough work-up ensued in the pediatric intensive care unit. An MRA of the brain, carotid Doppler and echocardiogram were normal. The lumbar puncture, ECG, CBC, chemistry panel, liver enzymes, cardiac enzymes and urine drug screen were normal, as were the SLE panel, Protein C and S, Factor V Leiden, anti-phospholipid antibodies, anti-thrombin III and coagulation profile, metabolic screening tests, blood and CSF cultures. The patient remained stable for the first two days and regained some motor strength. His hospital stay was complicated by a worsening headache. The CT demonstrated increased intracranial pressure that resolved following treatment with mannitol and steroids. After four weeks of extensive testing and rehabilitation, the etiology for his ischemic stroke remained elusive. The patient expended significant time and effort with a physical therapist. Upon discharge, he had regained the ability to walk and was able to

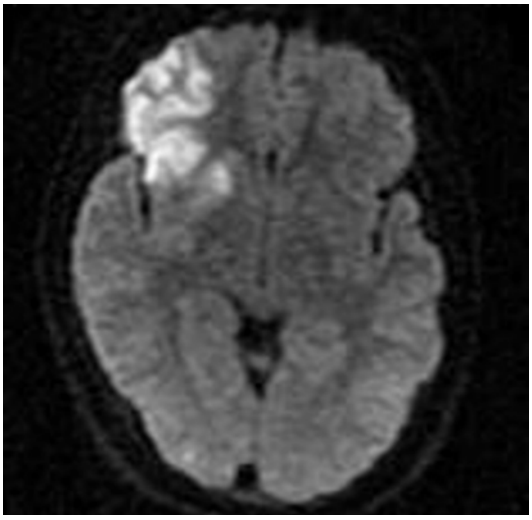

Figure. Diffusion Weighted MRI consistent with a right frontal lobe and basal ganglia ischemic infarction

move his left arm above his head. At his four-month follow-up, he had mild residual left arm weakness with no deficits in his gait or speech.

DISCUSSION

AIS in a pediatric patient is defined as a stroke occurring between the ages of one month and 18 years. This relatively rare condition varies geographically with occurrence in the United States at 2-3 per 100,000 patients.[1] By contrast, the overall incidence rate for total stroke (first-ever and recurrent of all ages) was 269 per 100,000 population.[7] Eighty percent of adult strokes are due to ischemia. While ischemic strokes in adults are usually thrombotic or embolic in nature, they may also be caused by hypoperfusion states. Cryptogenic strokes comprise 30 to 40% of all adult ischemic strokes and approximately 50% in children.[2,8]

Acute ischemic strokes in children most commonly occur between the ages of 1-5 years and least commonly in the extremes (< 1 year and > 15 years).[9] Focal neurological signs occur in three-quarters of patients, with hemiplegia the most common.[9] Despite the fact that infection accounts for 26% of cases, fever is present only 11% of the time.[9] The risk factors for stroke in children are congenital heart disease, infection, prothrombotic disorders, trauma, acquired and congenital vascular disease, sickle cell disease, metabolic disorders and mitochondrial disease.[9] Vascular disease alone accounts for one-third of cases, while metabolic disorders and prothrombotic disorders comprise 18% and 13% of cases respectively.[9] Trauma makes up an additional 11% of cases.[9] At least one risk factor is present in 90% of patients with almost 25% of patients having more than one.[9]

There appears to be demographic variation with a predilection for both gender and ethnicity. A seasonal variation may exist, as the disease most commonly occurs during the summer and least commonly during the winter.[9] African-Americans have a significantly higher risk of suffering from both ischemic stroke and hemorrhagic strokes.[10] Males carry a significantly higher risk of suffering all stroke subtypes compared to females.[10]

ED management of a child suffering from an ischemic stroke should focus on stabilization and transfer to an appropriate facility for specialty care. Stabilization begins with securing the airway, providing supplemental oxygen, establishing IV access and monitoring vitals signs and mental status. An emergent CT of the head in the ED should not be delayed. An ECG, CXR and lab work should be sent, including a complete blood count, blood and CSF cultures, chemistry, liver enzymes, cardiac markers, coagulation factors, urine analysis and urine drug screen. Empiric intravenous antibiotics and antivirals, maintenance fluid with an isotonic, non-dextrose containing fluid must be initiated in the ED.

Anti-platelet drugs are widely used in adults after literature has shown they reduce the rate of strokes.[2] Although randomized controlled trials with children have not been conducted, anti-platelet drugs are used in some centers to reduce the recurrence rate of stroke.[2] Adverse effects, such as severe bleeding or the precipitation of Reye's syndrome, are rare.[2] The use of anti-thrombotic drugs remains controversial in the pediatric literature and is not the standard of care.[3] However, anti-thrombotic drugs are being given to pediatric patients at some institutions despite a paucity of supporting literature. The benefits are unclear, and it seems their use is based on adult studies, case reports and expert opinion.[11] One study showed 1.6% of pediatric AIS patients admitted between 2000 and 2003 received thrombolytic therapy.[1] The children receiving thrombolytics had significantly higher medical costs, were less likely to be discharged home and had higher overall mortality rates.[1] Shortcomings of these findings were noted, including the small sample size and unknown severity of any of the patients at the time of presentation. Conversely, several case reports have been published showing potential benefits using thrombolytic therapy in children, including success stories across a wide age range and administration of thrombolytics well outside of the standard three-hour window used in adults.[11,12]

There are numerous etiologies for acute ischemic stroke in the pediatric population. Arterial dissection is one important cause. While most dissections occur in the internal carotid artery, children may dissect intracranially. MRI and contrast MRA show the anatomy without the risks of traditional angiography or radiation. Duplex ultrasonagraphy is useful, but CT angiogram still remains the gold standard for further investigation for suspected arterial dissection.[13] Secondly, cerebral venous sinus thrombosis may cause strokes in children. This commonly develops from extension

of infections, including acute otitis media, mastoiditis, pharyngitis, sinusitis, or meningitis. If infection is suspected, appropriate intravenous antimicrobials to include a third-generation cephalosporin, vancomycin and acyclovir should be initiated. Additionally, infectious vasculitis may occur from chronic infections such as tuberculous meningitis or rickettsial infection. Vasculitis may also be seen with common viral illnesses such as varicella and coxsackie. Lastly, cerebral vasculitis should be considered in children with either ischemic or hemorrhagic stroke, as well as strokes associated with fever or rash.[13] While erythrocyte sedimentation rate may be used as a screening tool, its interpretation should be used with caution since it does not exclude all vascular etiologies for ischemic stroke. There is no literature to support the empiric use of steroids; however, they may be given to treat cerebral edema or a vasculitic cause of stroke.

The rate of recurrence for childhood stroke may be as high as 30% and is dependent upon the etiology of the stroke.[2] Children with a hypercoagulable disorder or a vascular diagnosis have a higher likelihood of recurrent stroke. Sickle cell disease patients have a 40% chance of repeat stroke, while children with arterial dissection have a recurrence rate of 12%.[13]

Children who suffer AIS generally recover better than adults, but the effects may still be long lasting and detrimental.[14] While children who suffer a stroke have good educational and mobility outcomes, they have poorer outcomes when it comes to communication, socialization and activities of daily living.[14] Social and economic consequences, including decreased ability to work and lifelong disability, may arise. This is even more profound in children having a recurrence. The financial impact of these consequences has yet to be analyzed.

CONCLUSION

AIS in the pediatric patient is a rare but potentially devastating disease. The lack of research in this area is apparent. There is only anecdotal evidence for the use of anti-thrombotic drugs in the management of children with ischemic strokes, unlike the evidence for treating adult ischemic strokes. Due to the high rate of recurrence anti-platelet drugs should be considered and initiated in the ED.

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Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources, and financial or management relationships that could be perceived as potential sources of bias. The authors disclosed none.

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REFERENCES

1. Janjua N et al. Thrombolysis for ischemic stroke in children: data from the nationwide inpatient sample. *Stroke*. 2007; 38:1850-1854.
2. Nowak-Gottl et al. Antithrombotic drug treatment of pediatric patients with ischemic stroke. *Pediatric Drugs*. 2003; 5:167-175.
3. The International Stroke Trial (IST). A randomized trial of aspirin, subcutaneous heparin, both, or neither among 19435 patients with acute ischaemic stroke. International Stroke Trial Collaborative Group. *Lancet*. 1997; 349: 1569-1581.
4. National Institute of Neurological Disorders and Stroke rtPA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med*. 1995; 333:1581-1587.
5. Hacke W et al. Intravenous thrombolysis with recombinant tissue plasminogen activator for acute hemispheric stroke: the european cooperative acute stroke study. *JAMA*. 1995; 274:1017-1025.
6. Multicentre Acute Stroke Trial—Italy (MAST-I) Group. Randomised controlled trial of streptokinase, aspirin, and combination of both in treatment of acute ischaemic stroke. *Lancet*. 1995; 346:1509-1514.
7. Williams GR: Incidence and characteristics of total stroke in the united states. *BMC Neurol*. 2001; 1:2.
8. Lee BI et al. Yonsei Stroke Registry. Analysis of 1,000 patients with acute cerebral infarctions. *Cerebrovasc Dis*. 2001; 12:145-151.
9. Lee Y et al. Risk factors and outcomes of childhood ischemic stroke in taiwan. *Brain and Development*. 2007; 30:14-19.
10. Fullerton HJ, Wu YW, Zhao S, Johnston SC. Risk of stroke in children: ethnic and gender disparities. *Neurology*. 2003; 61:189-194.
11. Carpenter J, Tsuchida T, Lynch JK. Treatment of arterial ischemic stroke in children. *Expert Review of Neurotherapeutics*. 2007; 7:383-394.
12. Shuayto MI, Lopez JI, Greiner F. Administration of intravenous tissue plasminogen activator in a pediatric patient with acute ischemic stroke. *Journal of Child Neurology*. 2006; 21:604-606.
13. Roach SE et al. Management of Stroke in Infants and Children: A Scientific Statement From a Special Writing Group of the American Heart Association Stroke Council and the Council on Cardiovascular Disease in the Young. *Stroke*. 2008; 39:2644-2691.
14. Hurvitz E et al: Long-term functional outcome of pediatric stroke survivors. *Topics in Stroke Rehabilitation*. 2004; 11: 51-59.

Ovarian Teratoma with Torsion Masquerading as Intussusception in 4-Year-Old Child

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Background: Ovarian torsion (OT) occurs primarily in women of child-bearing age, but is rare in the pediatric population. The clinical presentation often consists of nonspecific abdominal complaints making the diagnosis difficult. Radiologic and sonographic evidence can be misleading. Although the delay in diagnosis from symptom onset is common, rapid diagnosis of ovarian torsion is imperative to prevent morbidity.

Case Report: We present the case of a four-year-old female who presented to the emergency department (ED) with a five-day history of intermittent abdominal pain and emesis. Initial diagnosis was suspicious for intussusception; however, on operative exploration, she was found to have a right adnexal torsion secondary to an ovarian teratoma. A right salpingo-oophorectomy was performed.

Conclusion: Early diagnosis of ovarian torsion may increase ovarian salvage and reduce morbidity. Faced with abdominal pain of uncertain etiology in a female child, emergency physicians should include ovarian torsion secondary to an ovarian mass in the differential diagnosis.
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INTRODUCTION

Ovarian torsion (OT) in the pediatric population is rare. Although it can be idiopathic, it is frequently due to a cystic or solid ovarian mass. The clinical presentation of OT often consists of vague abdominal complaints, making the diagnosis difficult.[1,2] Initial clinical and sonographic evidence is often misleading.[2-5] Cases of OT mimicking various abdominal pathologies have been described in the literature, but OT masquerading as intussusception is not well described.[6-8] Although the symptoms of OT are not diagnostically specific, the presentation and examination is almost always strongly suggestive of an acute surgical abdomen. Prompt surgical involvement should not be delayed by diagnostic evaluation under these circumstances. Although the delay in diagnosis from symptom onset is common, rapid diagnosis of ovarian torsion is imperative to prevent morbidity and optimize ovarian preservation.[1,9,10] Emergency physicians should consider OT caused by an ovarian mass when evaluating female patients of any age who present with lower abdominal pain.

CASE REPORT

A four-year-old female presented to the emergency department (ED) with a five-day history of severe, intermittent abdominal pain. She initially had several bouts of non-bloody, non bilious emesis that resolved after one day. Two days later, she had intermittent, crampy abdominal pain and tactile fevers. She was seen by her primary medical doctor who treated her for presumed constipation. Two days later, she continued to have episodic severe abdominal pain, recurrence of vomiting and a decrease in appetite and urine output. Upon presentation to the ED, the patient was witnessed to have several bouts of severe abdominal pain.

The patient's medical history was significant for chronic otitis media requiring myringotomy tubes. She had no recent travel and no pets at home. Her brother at home had nausea and

vomiting. The patient was born full-term by an uncomplicated repeat C-section. Medications included milk of magnesia, polyethylene glycol, and acetaminophen. She had no known drug allergies, and her immunizations were up to date.

On examination her vital signs were within normal limits. She appeared non-toxic and playful. On abdominal examination there was mild distention, diffuse tenderness, and mild guarding in the left lower quadrant. Rectal examination was negative for occult blood. While in the ED, the patient continued to have recurrent episodes of colicky abdominal pain.

Abnormal laboratory results were limited to an elevated white blood cell (WBC) count of 17.2 103/μL, elevated neutrophils of 12.4 103/μL, a urinalysis with WBC 22/HPF, and a low serum chloride level of 99 mEq/L. An acute abdominal series was unremarkable. A limited pelvic ultrasound (US) demonstrated a mass, measuring 2.9x 2.7x 2.4 cm, posterior to the bladder and left of midline, without peristalsis or internal vascularity (Figure 1). The radiologist reported the US as highly suspicious for intussusception because it demonstrated the classic target sign. A normal appendix was identified. The sonographic examination was limited due to sudden intense patient pain and consequently the ovaries were not visualized.

The pediatric surgical team was consulted, and the patient underwent diagnositic laparoscopy for treatment of a presumed intussusception with a lead point. Laparoscopy, however, showed no bowel pathology but instead revealed a complete 720° ovarian torsion with necrosis of the entire right fallopian tube and presence of an ovarian mass (Figure 2). On inspection, the liver, diaphragm, peritoneal surfaces, omentum, and pelvis were without evidence of tumor involvement. A laparoscopic right salpingo-oophorectomy was performed. The mass was placed in an endobag and removed piecemeal through a 12 mm trocar. Hair and sebaceous material were noted in the mass, supporting the gross diagnosis of an ovarian teratoma. Pathology confirmed the diagnosis of a benign, mature teratoma. Tumor markers including beta-HCG and alpha-fetoprotein were normal. The patient recovered without complications, and was discharged the following day. She will be followed with an annual examination and US of the contralateral ovary.

DISCUSSION

Although they can be found at any age, the incidence of benign cystic teratomas is highest during the reproductive years, accounting for 10% to 20% of all benign ovarian tumors.[11,12] In a 34-year study of patients presenting with ovarian teratomas (with or without torsion), 44% complained of lower abdominal pain, 25% had a mass or swelling, 21% were found incidentally, and 40% had symptoms of an acute abdomen.[13] In children, teratomas are usually benign with surgical removal being curative.[9,14] Two-thirds of children with ovarian teratomas have a palpable abdominal mass.[3]

Ovarian torsion has been defined as total or partial rotation of the adnexa around its vascular axis.[2] This torsion interferes first with the venous and lymphatic circulation and progresses rapidly to arterial occlusion. The ovary quickly becomes necrotic, and may cause eventual infection

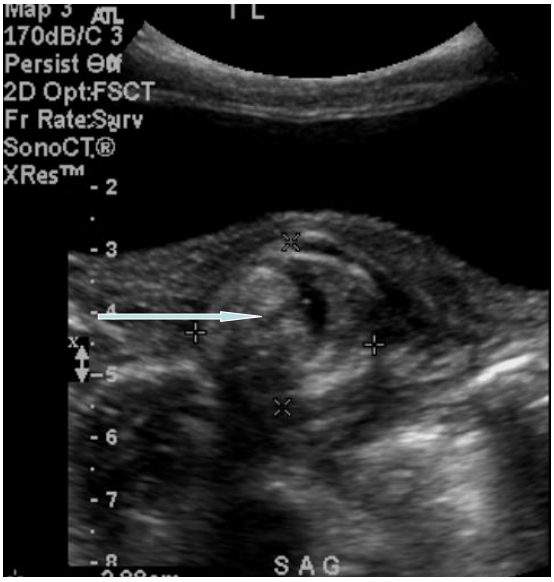

Figure 1. Limited pelvic ultrasound demonstrating a rounded mass in the left lower pelvis (arrow and crosses)

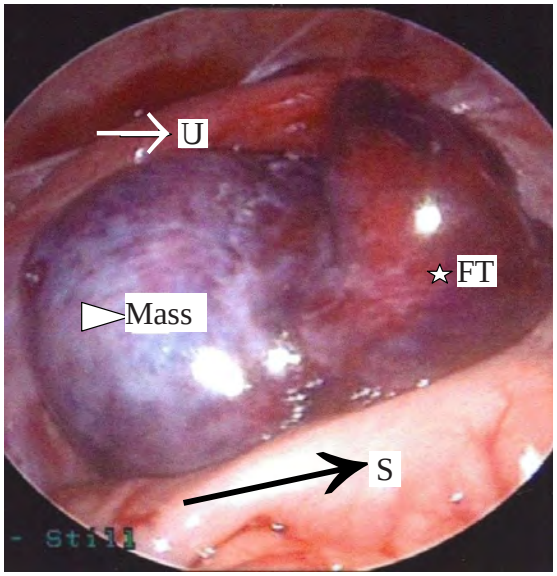

Figure 2. The torsed, ischemic left adnexa is seen between the sigmoid colon (black arrow/S) posteriorly and the uterus (white arrow/U) anteriorly. The fallopian tube (star/FT) is severely congested. The mass (triangle) has caused significant ovarian enlargement.

Table 1. Differential diagnosis for a female pediatric patient presenting with an acute surgical abdomen

Appendicitis	Ovarian torsion
Volvulus	Ovarian cyst
Peptic ulcer disease	Ectopic pregnancy
Intussusception	Tubo-ovarian abscess
Small bowel obstruction	Ruptured corpus luteal cyst
Necrotizing enterocolitis	Pelvic inflammatory disease
Incarcerated hernia	Trauma
Foreign body ingestion	Diabetic ketoacidosis

and peritonitis.[9] Delayed diagnosis past eight hours makes adnexal preservation unlikely.[10,11] Surgical salvage of ovaries in the presence of torsion varies from 0% to 31% among institutions.[2,9,15]

The clinical features of OT are variable. Houry and Abbott[15] described the cardinal features of OT as pain, nausea, vomiting, and rarely fever. Two large reviews of OT reported sudden pain (87% to 100%), nausea/vomiting (59% to 85%), and palpable abdominal mass (63%) as the most common presentations.[2,4] Other symptoms may include constipation, fever, or urinary symptoms.[1] Pain characteristics of OT are also variable, described as sharp or stabbing (70%), sudden (59%), or radiating to flank, back, or groin (51%). The most common complaint is lower quadrant pain (90%).[15] While laboratory and diagnostic tools may aid in diagnosis of OT, they should never delay a surgical consult when OT is clinically suspected. Leukocytosis is present in nearly 50% of patients, while the urinalysis is typically normal.[1] Torsion can be intermittent; therefore, diagnostic tests may be negative at the time of examination.[1]

With an incidence of 3%, ovarian torsion is the fifth most common gynecologic emergency in women.[15] The risk of OT complicating a case of ovarian teratoma is approximately 5% to 15% in adults and 3% to 16% in children.[13,16,17] Adnexal torsion accounts for up to 2.7% of all pediatric cases presenting with acute abdominal pain. Most reviews report three to five cases of torsion per year at large institutions.[10] In one study, 17% of girls who underwent surgery for presumed appendicitis were found to have OT.[9] OT should therefore be included in the differential diagnosis of the pediatric female patient with abdominal pain.

Intussusception presents most frequently between the ages three months to five years, with 60% occurring in the first year of life.[18,19] The classic triad consists of intermittent colicky abdominal pain, vomiting, and bloody mucous stools. This triad occurs only 20-40% of the time, with at least two of these findings occurring in 60% of patients.[20] The colicky abdominal pain usually lasts for one to five minutes and then abates for five to 20 minutes with the child often looking better or lethargic between episodes. Frank blood in the stool is a late and unreliable sign for intussusception. No laboratory test reliably excludes or provides a diagnosis of intussusception.[1]

As seen in our case, the presenting symptoms and physical examination in OT and intussusception can overlap. Abdominal pain and vomiting can be seen with both. The pelvic examination may reveal adnexal tenderness with a mass. In addition, each may present with rebound tenderness.[1,16] With younger children, who are unable to have a pelvic examination, ultrasound may be diagnostic.[1]

The many nonspecific signs and symptoms of OT and intussusception give a vast differential diagnosis for the female patient presenting with an acute surgical abdomen (Table 1). The lack of sensitivity and specificity of clinical parameters for OT vs. intussusception makes the diagnosis difficult. In White's study,[2] initial clinical suspicion in surgically proven cases of OT existed in only 19.2% of cases.

The primary diagnostic modality employed for suspected OT is US. The most common finding in female children is an enlarged heterogenous-appearing ovary—also described as an echogenic pelvic mass with no visualization of the ipsilateral ovary.[9,21] Doppler technology is not helpful in the diagnosis of OT because results are often equivocal.[2,21] Emergency physicians have diagnosed OT using bedside US that identified the enlarged ovarian mass and midline shift.[4,5,22]

Abdominal x-rays are neither sensitive nor specific for intussusception.[23,24] Classically, radiographs show a paucity of air in the right upper and lower quadrants with or without a soft tissue mass.[25] Many institutions use US to diagnose intussusception, and a Doppler flow study to identify areas of ischemia.[26] Classic US findings include the "target lesion" or "doughnut sign," which is the presence of several concentric rings. The target lesion averages 2.9 cm in diameter in the pediatric population.[27] The inner ring represents the proximal portion of the bowel with the outer ring representing the distal portion. Sonography is now considered a reliable means to diagnose intussusception.[25] Once diagnosed by US, a barium or air enema can be done with the intent of reducing the intussusception.

Our patient's radiographs were unremarkable. The US was read as likely intussusception, based on presence of a target lesion and consistent clinical history. The mass seen on US demonstrated no peristalsis and no internal vascularity. These are non-specific signs seen in both OT and intussusception.[4,5,27] In retrospect, the radiologist concluded that this mass was the torsed ovary. A second ultrasound radiologist who reviewed the images in a blinded fashion concluded that the images were suspicious for either an ovarian mass or intussusception. He stated that on several views the mass had the appearance of an intussusceptum within an intussuscipiens. Given the clinical history and the

limited pelvic ultrasound he agreed that it was a difficult diagnosis to make.

CONCLUSION

Pediatric ovarian torsion is rarely diagnosed in the ED by physical examination or imaging studies. Both sources can be difficult to interpret or misleading. Although laboratory and diagnostic tools may aid in diagnosis of OT, they should never delay a surgical consult when OT is clinically suspected. This case is presented to increase awareness of the key features of ovarian torsion in association with an ovarian teratoma. This difficult diagnosis accentuates the need for emergency physicians to consider OT in the differential diagnosis and workup of females presenting with abdominal pain.

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REFERENCES

1. McCollough M, Sharieff GQ. Abdominal surgical emergencies in infants and young children. *Emerg Med Clin N Am*. 2003; 21:909-935.
2. White M, Stella J. Ovarian torsion: 10-year perspective. *Emergency Medicine Australasia*. 2005; 17:231-237.
3. Sisler CL, Siegel MJ. Ovarian teratomas: a comparison of the sonographic appearance in prepubertal and postpubertal girls. *AJR Am J Roentgenol*. 1990; 154:139-141.
4. Shadinger LL, Andreotti RF, Kurian RL. Preoperative sonographic and clinical characteristics as predictors of ovarian torsion. *J Ultrasound Med*. 2008; 27:7-13.
5. Villalba ML, Huynh B, So M, Mackenzie JD, Ledbetter S, Rybicki F. An ovary with a twist: a case of interesting sonographic findings of ovarian torsion. *J Emerg Med*. 2005; 29:443-446.
6. Baker JL, Gull S, Jesudason EC, Abernethy LJ, Losty PD. Appendicitis masquerading as malignancy. *Arch Dis Child*. 2004; 89:481-482.
7. Hoey BA, Stawicki SP, Hoff WS, Veeramasuneni RK, Kovich H, Grossman MD. Ovarian torsion associated with appendicitis in a 5-year old girl: a case report and review of the literature. *J Pediatr Surg*. 2005; 40:e17-e20.
8. Kienstra AJ, Ward MA. Third place winner Three-year-old female with intermittent ovarian torsion. *J Emerg Med*. 2002; 23:375-377.
9. Kokoska ER, Keller MS, Weber TR. Acute ovarian torsion 252 in children. *Am J Surg*. 2000; 180:462-465.
10. Breech LL, Hillard PJ. Adnexal torsion in pediatric and adolescent girls. *Curr Opin Obstet Gynecol*. 2005; 7:483-489.
11. Takeda A, Manabe S, Mitsui T, Nakamura H. Laparoscopic management of mature cystic teratoma of bilateral ovaries with adnexal torsion occurring in a 9-year-old premenarchal girl. *J Pediatr Adolesc Gynecol*. 2006; 19:403-406.
12. Matz MH, Benign cystic teratomas of the ovary: a review. *Obstet Gynecol* Surv. 1961; 16:591-594.
13. Ayhan A, Bukulmez O, Genc C, Karamursel BS, Ayhan A. Mature cystic teratomas of the ovary: case series from one institution over 34 years. *Eur J Obstet Gynecol Reprod Biol*. 2000; 88:153-157.
14. Brown MF, Hebra A, McGeehin K, Ross AJ 3rd. Ovarian masses in children: a review of 91 cases of malignant and benign masses. *J Pediatr Surg*. 1993; 28:930-933.
15. Houry D, Abbott JT. Ovarian torsion: a fifteen-year review. *Ann Emerg Med.* 2001; 38:156-159.
16. Wittich AC. Adnexal torsion presenting as an acute abdomen in a patient with bilateral cystic teratoma of the ovary. *J Am Osteopath Assoc.* 2002; 102:231-233.
17. Benjapibal M, Boriboonhirunsarn D, Suphanit I, 273 Sangkarat S. Benign cystic teratoma of the ovary: a review of 608 patients. *J Med Assoc Thai*. 2000; 83:1016-1020.
18. Parashar UD, Holman RC, Cummings KD, et al. Trends in intussusception-associated hospitalizations and deaths among US infants. *Pediatrics.* 2000; 106:1413-1421.
19. Ugwu BT, Legbo JN, Dakum NK, et al. Childhood intussusception: a 9-year review. *Ann Trop Paediatr*. 2000; 20:131-135.
20. Kuppermann N, O'Dea T, Pinckney L, et al. Predictor of intussusception in young children. *Arch Pediatr Adolesc Med*. 2000; 154:250-255.
21. Servaes S, Zurakowski D, Laufer MR, et al. Sonographic findings of ovarian torsion in children. *Pediatr Radiol.* 2007; 37:446-451.
22. Johnson S, Fox JC, Koenig KL. Diagnosis of ovarian torsion in a hemodynamically unstable pediatric patient by bedside ultrasound in the ED. *Am J Emerg Med*. 2006; 24:496-497.
23. Smith DS, Bonadio WA. Losek JD, et al. The role of abdominal x-rays in the diagnosis and management of intussusception. *Pediatr Emerg Care*. 1992; 8:325-327.
24. Yang ST, Tsai CH, Chen JA. Differential diagnosis between intussusception and gastroenteritis by plain film. *Zhonghua Min Guo Xiao Er Ke Yi Xue Hue Za Zhi*. 1995; 36:170-175.
25. Byrne AT, Goeghegan T, Govender P, Lyburn ID, Colhoun E, Torreggiani WC. The imaging of intussusception. *Clinical Radiology*. 2005; 60:39-46.
26. Bhisitkul DM, ListernickR, Shkolinik A, et al. Clinical 294 application of ultrasonography in the diagnosis of intussusceptions. *Pediatr J.* 1992; 121:182-186.
27. Ko SF, Lee TY, Ng SH, et al. Small bowel intussusception in symptomatic pediatric patients: experience with 19 surgically proven cases. *World J Surg.* 2002; 26:438-443.

Fever, Sacral Pain, and Pregnancy: An Incarcerated Uterus

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Uterine incarceration is an uncommon but serious presentation in the emergency department that requires early recognition to improve maternal and fetal outcomes.

Case: A 29-year-old female, at 12 weeks gestation, presented to the emergency department (ED) with complaints of fever, sacral pain and urgency. Based on history and physical examination, she was found to have a retroverted, incarcerated uterus. After a failed attempt at reduction in the ED, her uterus was successfully reduced under general anesthesia.

Discussion: Pain and urinary difficulties, such as retention and hesitancy, are frequent in pregnancy, yet incarcerated uterus is an uncommon emergency department diagnosis that often presents with these symptoms. Clues to the diagnosis include a retroverted uterus, urinary retention, and pain in a patient presenting in the third to fourth months of gestation. Treatment is by manual reduction of the uterus. Complications range from spontaneous abortion to uterine rupture.
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INTRODUCTION

Pregnant patients present commonly to the emergency department (ED) with complaints of pain and urinary difficulties including retention and hesitancy. A patient with a complicated previous gynecologic history may be at risk for more than an ectopic pregnancy, spontaneous miscarriage, or urinary tract infection as illustrated in the case below.

CASE REPORT

A 29-year-old woman, gravida 6, para 1-0-4-1, at 12 weeks gestation, presented to the ED with a chief complaint of fever (reported maximum 103°F) and sacral pain. She denied chest pain, shortness of breath, and any vaginal discharge or bleeding. Her pain was located mid-sacrum at a 5/10 intensity with radiation to the suprapubic area. Additionally, she denied dysuria but complained of increasing difficulty with emptying her bladder. Her past medical history was significant for endometriosis with four laparoscopic procedures, an adenexal cyst, four previous miscarriages, a prior cesarean section, and Perry-Romberg syndrome (localized scleroderma of childhood). An ultrasound completed a week prior for pelvic pain revealed a viable fetus in an otherwise normal uterus with no retroversion noted.

The vital signs were as follows: blood pressure 121/53 mm Hg, pulse 94, respirations 18, temperature 100.4°F, and pulse oximetry of 100% on room air. The abdominal exam was remarkable for tenderness to palpation on the midline at and below the umbilicus. The patient's complaints of pain in the sacrum and lower lumbar spine could not be further elicited through palpation. There was no costovertebral angle tenderness. The patient reported in previous speculum examinations that her cervix could not be visualized due to severe anterior location, and this was found on our examination also. Bimanual examination revealed diffuse tenderness and a palpable mass in the posterior cul de sac consistent with a retroverted uterus, but otherwise no gross abnormalities. The remainder of her physical examination was unremarkable. Laboratory studies including a basic metabolic panel, complete blood count, and urinalysis were unremarkable. The patient was given a one-liter intravenous (IV) fluid bolus, 1mg hydromorphone IV x 2 for pain control, and ondansetron 4mg IV for nausea. An obstetric/gynecologic

consultation was requested for persistent pain.

The consultant agreed with the prior examination and lab review and made the diagnosis of incarcerated uterus due to the presenting symptoms in combination with the markedly retroverted uterus without repeat ultrasonography. Attempts to manually reduce the incarceration by intravaginal pressure in the ED were unsuccessful due to patient discomfort. She was taken to the operating room for reduction of the incarcerated uterus under spinal anesthesia followed by placement of a vaginal pessary. Her recovery was complicated with a low-grade fever and some pain in the absence of abnormalities in the chest x-ray, urinalysis, or blood count. Her postoperative pain warranted the use of parenteral narcotics and was significantly improved with the removal of the pessary. She was discharged with a viable pregnancy.

DISCUSSION

Retroversion in the first trimester is reported to occur in up to 15% of pregnancies.[1] Incarceration of the uterus is a rare complication, occurring in one of 3000 cases.[2,3,4,5,6] During a normal pregnancy, between the twelfth and fourteenth weeks of pregnancy, the gravid uterus transforms from a pelvic to an abdominal organ and a retroverted uterus will spontaneously correct as the fundus rises out of the pelvis and falls forward to its normal anatomical position.[3] However, a retroverted or retroflexed uterus can become entrapped between the subpromontory sacrum and the pubis. Factors and preexisting conditions that may predispose a patient to an incarcerated uterus include: multiparity, adhesions from endometriosis or prior pelvic inflammatory disease, anatomical abnormalities, pelvic tumors, and uterine fibroids.[2,3,6,7,] Presenting symptoms include: pelvic discomfort and lower abdominal or back pain, dysuria, urinary frequency, urinary retention, overflow incontinence, urinary stasis leading to cystitis, vaginal bleeding, rectal pressure, tenesmus, and progressive constipation.[6,7,8] If untreated, patients may go on to develop anterior uterine wall thinning or sacculation, bladder rupture, preterm labor, premature rupture of fetal membranes, spontaneous abortion, or uterine rupture during labor.[2,5,7]

Although patient symptoms are often reported as non-specific and mimic many of the normal events of pregnancy, the most common complaints are of urinary difficulties, such as retention and hesitancy, due to urethral compression.[2,5] Patients who present with these symptoms during the third and fourth months of gestation should receive a pelvic examination to assess for this serious cause of common symptoms. The typical physical examination of a pregnant woman with an incarcerated uterus often reveals a distended bladder and a fundal height that is less than expected.[3] The cervix is often not visualized with a speculum exam as it is displaced anteriorly, behind the pubic symphysis.[3,6,9] Fetal heart tones can be difficult to auscultate.[3] On bimanual examination, the fundus of the retroverted uterus is palpated as a large mass in the cul de sac.[3,6] An ultrasound exam can show a uterus displaced posteriorly with a distended bladder anterior to the uterus. In the setting of a retroverted uterus, a fundal placenta can often be mistaken as a placenta previa; as such, magnetic resonance imaging may be useful.[5]

Debates exist concerning whether to emergently reduce the uterus with older reports of fetal demise and more recent ones demonstrating good term pregnancy outcomes.[10,11] Treatment includes first draining and decompressing the bladder with an indwelling catheter. Next, after close consultation with the obstetrician, reduction of the uterus can be attempted by applying steady pressure with two fingers in the posterior vaginal fornix directing the uterus cephalad while the patient is in the dorsal lithotomy position, knee-chest position, or under anesthesia.[2,7] None of these were successful in the patient in this case, so reduction under spinal anesthesia was undertaken. Following a successful reduction, the patient is encouraged to help maintain the corrected uterine position through sleeping in a prone position and with exercises such as the knee-chest and all-fours positions.[6]

Although, this patient did not have ultrasound findings consistent with retroversion at the time of her ultrasound, she did develop symptoms consistent with uterine incarceration in the days preceding her ED visit. Her other risk factors, such as endometriosis or adhesions secondary to her previous surgeries, may have contributed in this case.

In conclusion, incarcerated uterus is a rare but important diagnosis for emergency physicians to recognize and assist in treatment. It is important to consider this diagnosis in patients entering their second trimester with complaints of urinary symptoms, vaginal bleeding and pelvic pain to ensure the safety of the mother and fetus.

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, nor the U.S. Government.

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REFERENCES

1. Fadel HE, Misenhimer HR. Incarceration of the retroverted gravid uterus with sacculation. *Obstet Gynecol*. 1974; 43:46-49.

2. Myers DL, Scotti RJ. Acute urinary retention and the incarcerated, retroverted, gravid uterus. *J Reprod Med*. 1995; 40:487-490.
3. Feusner AH, Mueller PD. Incarceration of a gravid fibroid uterus. *Ann Emerg Med*. 1997; 30:821-824.
4. O'Connell MP, Ivory CM, Hunter RW. Incarcerated retroverted uterus – a non recurring complication of pregnancy. *J Obstet Gynaecol*. 1999; 19:84-85.
5. Inaba F, Kawatu T, Masaoka K, Fukasawa I, Watanabe H, Inaba N. Incarceration of the retroverted gravid uterus: the key to successful treatment. *Arch Gynecol Obstet*. 2005; 273:55-57.
6. O'Grady JP, Pope CS, Darouian Z. Malposition of the uterus. EMedicine; 5 June 2006. Available at: www.emedicine.com/med/topic3473.htm. Accessed Sept 23, 2007.
7. Love JN, Howell JM. Urinary retention resulting from incarceration of a retroverted, gravid uterus. *J Emerg Med*. 2000; 19:351-354.
8. Crichton EC. The significance of the retroverted uterus in obstetrics and gynaecology. *S Afr Med J*. 1959; 33:129-131.
9. Nowlin WC. Incarceration of the pregnant retroverted uterus. *Med Times*. 1965; 93:743-745.
10. Gibbons JM Jr., Paely WB. The incarcerated gravid uterus. *Obstet Gynecol*. 1969; 331:842-845.
11. Hamoda H, Chamberlain PF, Moore NR, MacKenzie IZ. Conservative treatment of an incarcerated gravid uterus. *BJOG*. 2002; 109:1074-1075.

Clitoral Priapism with No Known Risk Factors

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Clitoral priapism is a rare condition that is associated with an extended duration of clitoral erection due to local engorgement of clitoral tissue resulting in pain. Although the pathophysiology is not completely understood, it has been associated with specific classes of medications, diseases that alter clitoral blood flow or others associated with small to large vessel disease. We present a case report of a 26-year-old patient who developed clitoral priapism without a clear medication or disease related etiology. The patient was treated with opiates, imipramine, non-steroidal anti-inflammatory medication, and local ice packs. She recovered uneventfully.
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INTRODUCTION

Clitoral priapism is a rare cause of clitoral pain. It is thought to be a result of alterations in local hemodynamics leading to incomplete venous or lymphatic drainage of the clitoral tissue, resulting in edema and tissue swelling with concomitant pain.[1] Due to the increasing number of medications being prescribed that can cause priapism as well as the resultant deficiency in blood flow to the clitoral region, it is paramount for the emergency physician (EP) to diagnose and treat appropriately. The prolonged erection is not associated with sexual stimulation, can last from minutes to days, and is frequently uncomfortable.[1,2] Embarrassment can preclude patients from describing the exact nature of their symptoms, emphasizing the importance of a complete physical exam that includes an evaluation of the clitoris.

Clitoral priapism has been associated with medications producing alpha blockade, inhibition of serotonin reuptake, and non-SSRI antidepressants.[1,3] It has also been associated with transitional cell carcinoma obstructing venous and lymphatic clitoral outflow.[4,5] Since cases are rare, there is inadequate evidence to determine the incidence of this condition or the likelihood of a particular cause. However, it has been shown that discontinuing the offending agent can result in a therapeutic resolution within 24-72 hours.[1,6] Other treatment modalities include imipramine, non-steroidal anti-inflammatory medications (NSAIDs), ice packs, opiates, and, rarely, intracavernous injection of adrenaline.[2,6-8] Presented here is a case of clitoral priapism of unknown etiology and no known risk factors.

CASE REPORT

A 26-year-old gravida 4 para 2 TAB 2 African-American patient presented to the emergency department (ED) with a two-day history of pain following wiping of her urethra. The patient described her pain as constant, aching and sharp, and feared she cut herself near her urethra while wiping. She has not been sexually activity for several months and denied insertion of foreign bodies. She described swelling in the region of her clitoris and attempted to alleviate the symptoms with several hot baths with minimal relief. She denied a history of trauma, vaginal pain and discharge, prior similar symptoms, and changes in hair pattern or voice. She also denied fever, dysuria, suprapubic pain, and gastrointestinal symptoms. She denied recent medication use or recreational drug use. The patient's obstetrical and uro-gynecologic history was significant for two first trimester therapeutic abortions for unintended pregnancies without complication, several urinary tract infections treated effectively, and chlamydia vaginitis treated as an outpatient. She states that all annual pap smears and pelvic examinations prior to her presentation were otherwise normal. The patient also denied a family history of sickle cell disease and cancer.

Her vital signs included a temperature of 37 degrees Celsius, blood pressure of 129/70 mm Hg, pulse of 87 beats

per minute, and respiratory rate of 16 breaths per minute. She was alert, oriented, and in moderate discomfort lying supine on the gurney. She pointed to her clitoral region when asked to point to the area of maximal pain. Her pelvic exam revealed a firm, swollen and acutely tender clitoris and clitoral hood with minimal vulvar tenderness and swelling (Figure 1). Her external genitalia including labia were normal and without lesions, lacerations, or abrasions. She had no cervical motion tenderness, os was closed, and no masses or discharge were present. Uterus was midline and non-tender. Her head and neck, cardiopulmonary, abdominal, lymphatic, and extremity exams were unremarkable.

Laboratory tests included an unremarkable urinalysis and a negative urine pregnancy test. The patient was given vicodin for pain control with minimal relief. A gynecology consultation recommended NSAIDS, imipramine, and ice packs. The patient was referred to her primary gynecologist and instructed to follow up within two days with precautions to return if loss of sensation, increased swelling, or persistent or worsening pain after 48 hours. Her follow-up was uneventful.

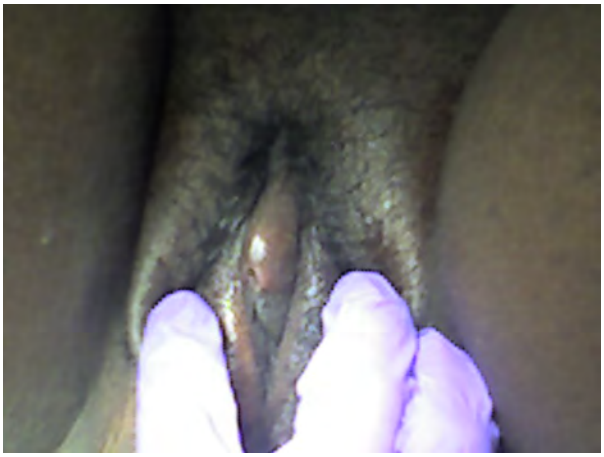

Figure 1. Clitoral priapism of patient

DISCUSSION

Priapism is defined as a persistent and painful erection of penile or clitoral tissue with a duration of more than six hours and is not associated with sexual arousal.[2] Clitoral priapism is an infrequent cause of clitoral or vulvar pain and can result in unnecessary and extensive laboratory and radiographic testing if not diagnosed appropriately through the history and clinical examination.[2] Although only infrequently associated with etiologies that can cause long-term morbidity, the episodes themselves can be incapacitating and can require multiple hospital or clinic visits for pain control, according to sporadic case reports in the literature.[2] Often a vague history is obtained as a function of embarrassment and frequently the episode has abated by the time the clinician evaluates the patient, making it difficult to diagnose. The details of specific clitoral swelling followed by pain with concomitant erection of the clitoral tissue can be suggestive of the diagnosis. Often, patients will describe an episode of perceived vulvar or labial swelling or pain that leads to clitoral swelling, erection, and extensive pain.[2] Others will describe symptoms of dysuria, anorgasmia, intestinal complaints, and even cramping pain in the pelvic or lower abdominal region within the episode of clitoral priapism.[3] A search for antecedent or proximate causes should ensue, and would likely include a complete recent medication and illicit drug history, a complete physical and gynecological examination, relevant urine and pregnancy studies, and adjunct testing depending upon the physical exam and history findings.

The mechanism of clitoral priapism is theorized to be similar to the pathophysiology of male priapism, which involves altered circulation of the corpora cavernosa and increased clitoral intracavernous pressure resulting in erection of the glans.[9] Decreasing venous outflow results in increased blood volume and pressure in the corpora. Although in men an increase in arterial inflow is an additional mechanism for producing priapism, this does not appear to be a relevant etiology in women.[9] Corporeal venous outflow obstruction is likely a result of the following causes: 1) alpha sympathomimetic blockade resulting in extended relaxation of corporal smooth muscle, 2) physical obstruction or sinus compression, and 3) venous drainage occlusion.[2,7]

Several groups of medications have been associated with clitoral priapism. although the exact mechanism for producing clitoral engorgement and pain is unknown.[1] Due to the increasing use of these agents, the condition may become more prevalent, which is why EPs should evaluate for this condition. Alpha adrenergic blocking agents resulting in smooth muscle relaxation and venous stasis in the corpus cavernosa may cause priapism.[9] Priapism is also a known adverse effect of psychotropic medications[1,10] and is a rare reported adverse effect of antidepressant treatment.[11] Trazodone, buproprion,[10] citalopram,[6] fluoxetine, paroxetine, bromocriptine, olazapine,[12] and nefazodone[10] have all been implicated as agents contributing to or responsible for episodes of clitoral priapism. Potent 5H2 receptor antagonists[3] have also been reported to cause priapism, although the mechanism is unclear since the majority of 5H2 antagonists have limited alpha blocking effects. Bromocriptine may act through an increase in oxytocin or spinal dopaminergic pathways, although clear evidence is lacking.[1]

Additionally, authors have provided evidence to support the idea that, rarely, clitoral priapism is the result of a physical obstruction of venous or lymphatic drainage and have included transitional cell carcinoma as a cause.[5] Sickle cell disease[13-15] and spinal cord injury[16] are known causes of priapism, with sickle cell far more common as a cause in males. Although this patient was of African-American decent,

she had no family history of sickle cell disease and it is not thought to be the cause in her situation.

If an anatomic source exists, often excision or other therapies need to be considered with the appropriate specialist. The complete workup must exclude other more common causes of clitoral or vulvar pain including urinary tract or pelvic infections, local trauma or abrasions, vulvovaginitis, contact dermatitis, and other causes of pelvic or abdominal pain.

No specific treatment regimen has been proven to be effective in managing clitoral priapism. However, cause-specific treatment frequently reverses the condition and has responded well, in case reports, to the usage of NSAIDS, cooling pads, and opiates, although their effectiveness is inconclusive.[2,7,17,18] When the etiology in unknown, the course of treatment becomes more confused and uncertain. While several case reports have discussed the use of imipramine as the initial drug of choice, its effect is poorly understood.[2,6,7] More likely, the patient's condition resolved because the offending agent was removed. Intracavernous administration of alpha agonists similar to the treatment of penile priapism has also been reported with success.[8] Therefore, the accepted management involves removal of the offending agent, replacement with an agent from another therapeutic class, and coordinated treatment with imipramine for symptomatic episodes.[7]

Some authors have advocated for the use of alpha adrenergic agonists in the acute episodes, including phenylpropanolamine or phenylephrine. These medications have similar alpha agonist activity when compared to imipramine but do not have the anti-cholinergic effects. One case highlights scorpion venom as a possible treatment modality.[19] Urologic and/or gynecologic consultation may be required if symptoms cannot be abated with the above remedies.

CONCLUSION

Clitoral priapism is a rare cause of vulvar pain and can be excruciating to the patient. Diagnosis may be difficult given embarrassment, vague history and inconclusive physical exam. Several causes has been linked to clitoral priapism including, alpha blocking agents, antidepressants, psychotropics, and 5H2 blockers. Other causes, such as cancer, have also been implicated. Treatment involves alpha agonists for acute attacks, imipramine, and symptomatic relief with NSAIDS and ice packs. Gynecology follow up is necessary for further evaluation.

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REFERENCES

1. Thompson JW Jr., Ware MR, Blashfield RK. Psychotropic medication and priapism: a comprehensive review. *J Clin Psychiatry*. 1990; 51:430-433.
2. Medina CA. Clitoral priapism: a rare condition presenting as a cause of vulvar pain. *Obstet Gynecol*. 2002; 100:1089-1091.
3. Klein DF. Repeated observations of yawning, clitoral engorgement, and orgasm associated with fluoxetine administration. *J Clin Psychopharmacol*. 1989; 9:384.
4. Slavin RE, et al. Locally aggressive granular cell tumor causing priapism of the crus of the clitoris. A light and ultrastructural study, with observations concerning the pathogenesis of fibrosis of the corpus cavernosum in priapism. *Am J Surg Pathol*. 1986;10:497-507.
5. DiGiorgi S, Schnatz PF, Mandavilli S, Greene JF, Curry SL. Transitional cell carcinoma presenting as clitoral priapism. *Gynecol Oncol*. 2004; 93:540-542.
6. Berk M, Acton M. Citalopram-associated clitoral priapism: a case series. *Int Clin Psychopharmacol*. 1997; 12:121-122.
7. Pohl J, Pott B, Kleinhans G. Priapism: a three-phase concept of management according to etiology and prognosis. *Br J Urol*. 1986; 58:113-118.
8. Xiao H, et al. One case report of clitoral priapism and literature review. *Zhonghua Nan Ke Xu*e. 2004; 10:524-525.
9. Keoghane SR, Sullivan ME, Miller MA. The etiology, pathogenesis and management of priapism. *BJU Int*. 2002; 90:149-154.
10. Levenson JL. Priapism associated with bupropion treatment. *Am J Psychiatry*. 1995; 152:813.
11. Hayes PE, Kristoff CA. Adverse reactions to five new antidepressants. *Clin Pharm*. 1986; 5:471-480.
12. Bucur M, Mahmood T. Olanzapine-induced clitoral priapism. *J Clin Psychopharmacol*. 2004; 24:572-573.
13. Adeyoju AB, Olujohungbe AB, Morris J, et al. Priapism in sickle-cell disease; incidence, risk factors and complications - an international multicentre study. *BJU Int*. 2002; 90:898-902.
14. Badmus TA, Adediran IA, Adesunkanmi AR, Katung IA. Priapism in southwestern Nigeria. *East Afr Med J*. 2003; 80:518-524.
15. Molitierno JA, Jr., Carson CC, 3rd. Urologic manifestations of hematologic disease sickle cell, leukemia, and thromboembolic disease. *Urol Clin North Am*. 2003; 30:49-61.
16. Gordon SA, Stage KH, Tansey KE, Lotan Y. Conservative management of priapism in acute spinal cord injury. *Urology*. 2005; 65:1195-1197.
17. Yamamoto K. Priapism successfully treated with indomethacin: report of a case. *Hinyokika Kiyo*. 1970;16:283-286.
18. Brodie-Meijer CC, Diemont WL, Buijs PJ. Nefazodone-induced clitoral priapism. *Int Clin Psychopharmacol*. 1999; 14:257-258.
19. Teixeira CE, Faro R, Moreno RA, et al. Nonadrenergic, noncholinergic relaxation of human isolated corpus cavernosum induced by scorpion venom. *Urology*. 2001; 57:816-820.

Malpractice Cases in Wound Care and a Legal Concept: Special Defense

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There is no doubt that in today's practice of emergency medicine it is imperative to be familiar with how the law relates to administrative and clinical practice. It is my pleasure to announce, as section editor, the new Legal Medicine section of the Western Journal of Emergency Medicine. It is anticipated that the articles will cover a variety of areas and cases in the law. Some articles may focus on a particular disease or entity, with representative malpractice cases, and clinical caveats. Other articles may focus on legal concepts that enter the arena of emergency medicine. I have provided brief examples of each of these in this initial manuscript. Other articles could also cover original research related to law such as the standard of care in a given clinical situation or legal concepts such as consent, do-not-resuscitate, and AMA among others. I am hopeful that it will be of great interest to the readers. We welcome submissions and contributions for consideration.
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Medical Topic – Wound Foreign Bodies

Over 12 million visits a year throughout the United States for traumatic wounds make them one of the most common reasons for an emergency department (ED) visit.[1] One emergency medicine (EM) text cites wound care as accounting for 5-20% of all ED malpractice claims and 3-11% of all dollars paid out.[2]

Ashly v Gustafson[3] – A woman broke a glass and put the pieces in a bag of trash. She did not notice that a piece was protruding and cut her ankle when it came into contact with the glass. In the ED her wound was sutured, but no-ray was taken. Nine months later she presented for persistent pain and a 2.5 cm piece of glass was removed. Again, no radiographs were done. Seven months after this visit she returned to the ED, and an x-ray revealed three more pieces of glass. She litigated with the claim that an x-ray should have been done on the first visit. At trial she was awarded $119,930. One ED physician was given 42% fault, another 25%.

Nelson v Richter[4] – A teenager cut his foot when a glass fell onto it from a counter. Although the wound was cleaned and examined, it was too small for exploration. Ten days later he returned with persistent pain. In surgery a piece of glass was removed from between two toes. The patient sued, claiming an x-ray should have been done. The defense claimed that the return visit was simply for suture removal and that surgery would have been required irregardless of time of diagnosis. A jury reached a verdict for the defense.

A retained foreign body should be assumed to be present in traumatic wounds until proven otherwise. One study showed that glass, a notorious foreign body, was present in 7% of the lacerations it caused,[5] and research indicates that plain x-ray is greater than 98% sensitive when the foreign body is radiopaque material, such as glass.[6] In a cadaver study, nonleaded glass was visualized with 90% sensitivity and a false-positive rate of 10%.[7] A volume of less than 15 mm^3 (an object less than 1/10 of an inch on either side) was associated with a higher miss rate.[7] Because failure to radiograph glass wounds is a common source of litigation, there should be a low threshold by the clinician to do so. A thorough examination of a laceration caused by glass should be carried out with maximum exposure and good lighting in a bloodless field and through a full range of motion of the affected area. Despite a negative exam, and/or a negative x-ray, upon discharge the patient should be warned of the possibility of a retained foreign body. He should also be instructed about specific signs and symptoms for which he

should return to the ED. The exam and warnings should be appropriately documented on both the chart and discharge instructions.

A 'Special Defense' to Be Aware of: Legal Concepts

We should all be aware of the four components of malpractice. (1) the physician had a duty, (2) breached the duty, (3) resulting in harm to the patient, and (4) the harm was caused by the breach of duty. Typically, if a lawyer proves all four elements are present, the physician is liable for damages. Sometimes "special defenses" may be raised to absolve the physician, even though it appears the elements are all present. For example, if a physician stopped by the roadway to help an injured victim and malpractice occurred, the physician would likely not be held liable by reason of the "Good Samaritan" special defense. Let's look at another special defense recently used in court. It has sound legal basis and was very imaginative.

Ross v Vanderbilt[8] – Kimberly Ross went to the ED with a lacerated finger. The emergency physician (EP), who was in training, determined that sutures were needed and injected lidocaine into the wound. Immediately after the injection, Ross said she didn't feel well. Her arm jerked and her eyes rolled back. The EP walked a few feet from the bedside to summon help. Ross continued jerking and fell to the floor, hitting her head.

After her fall Ross suffered loss of memory and dexterity and had personality changes. Diagnosed with a vasovagal reaction and traumatic brain injury, she brought suit for malpractice. Ross claimed that the physician should have not left her side, allowing her to fall.

On its face, this case seems to fit the criteria for malpractice. There was a duty (the doctor had taken the patient), very possible breach of duty (abandonment by the physician), injury (head trauma), and direct causation (by leaving the bedside the physician was not able to prevent the fall). The EP, however, raised a creative "special defense." In citing the "sudden emergency" (in the ED) defense, he was exonerated.

A valid defensive doctrine accepted in law, the "sudden emergency" defense acknowledges that a person confronted with a sudden or unexpected situation demanding immediate action may not use the same degree of judgment as he would in normal circumstances. Another example would be a car accident where someone is suddenly struck. In attempting to hit the brake pedal, the driver may hit the gas pedal instead and accelerate, striking another car. That driver could claim that the sudden emergency, caused him to do something he would not normally have done, and he would likely be absolved.[9]

In this initial contribution to the new medical-legal section of *West*JEM, we have presented the form of typical future articles that will often focus on a high risk area of EM (such as foreign bodies in wounds) and/or a legal topic (such as the sudden emergency doctrine). The reader will thus gain insight into particular areas of high risk and how to avoid liability and also stay abreast of legal concepts that will impact his or her practice.

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REFERENCES

1. Singer AJ, Hollandr JE, Quinn JV. Evaluation and management of traumatic laceration. *NEW ENGL J MED*. 1997; 337:1142-1148.
2. Henry GL. Specific High-Risk Medical-Legal Issues. In: Henry GL, Sullivan DJ, eds. *Emergency Medicine Risk Management*. Dallas: American College of Emergency Physicians; 1997:475-494.
3. Ashly v Gustafson, et al. Jackson County (Missouri) Circuit Court, Case No. CV97-19936.
4. Nelson v Richter. Oakland County (Mississippi) Circuit Court, Case No. 03-048262-NH.
5. Karcz A., Korn R, et al. Malpractice claims against emergency physicians in Massachusetts 1975-1993. *Am J Emerg Med*. 1996;14:341-345.
6. Russell RC, et al. Detection of foreign bodies in the hand. *J Hand Surg*. 1991; 16:2.
7. Flom LL, Ellis GL. Radiologic evaluation of foreign bodies. *Emerg Med Clin North Am*. 1992; 10:163.
8. Ross v Vanderbilt University Medical Center. No.M1999-02644-COA-R3-CV, February 18, 2000.
9. Keeton WP, Dobbs DB, Keeton RE, Prosser WL, Owen D. *Prosser and Keeton's Hornbook on Torts, 5th ed*. St. Paul, MN: West; 1984.

Open Access: The Alternative to Subscription-Based Medical Publishing

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Since the inception in 1655 of *The Philosophical Transactions of the Royal Society*,[1] one of the world's first scientific journals,[2] the subscription-based journal has been the traditional model for disseminating scientific and medical knowledge.[3,4] Granting access only through subscription can place significant barriers to information sharing, thus putting research, teaching and patient care at risk. While the internet age has made information universally available, medical and scientific knowledge has proven to be an exception. Subscription rates to scholarly journals continue to increase annually by 8-10%, far exceeding inflation rates measured by the Consumer Price Index.[5,6] Publishing in science, technology and medicine is a seven-billion dollar global industry[7] with publishers averaging 20-30% profit margins.[8] In 2003, the University of Iowa paid nearly 1.1 million dollars to Elsevier, one of the largest publishing companies in the world, and Cornell nearly 1.7 million.[9] The University of California libraries collectively paid eight million to Elsevier and 64 million for all their scholarly material.[8,10] With a growing number of libraries, including Cornell, Harvard, North Carolina and MIT, forced to cancel subscriptions,[11,12,13] it is clear that we are in a crisis situation. One potential solution is the open access model. According to the 2003 Bethesda Statement on Open Access Publishing,[14] an open access journal must fulfill two criteria: 1) the author and any copyright holder must grant free access to the publication and a license to copy, use and distribute the work publicly, and 2) the work must also be deposited immediately upon publication to at least one online repository.

While the concept of open access has been around for years, only recently has it gained traction within the scientific community. In 1998 the Association of Research Libraries (ARL) launched the Scholarly Publishing and Academic Resources Coalition (SPARC) with the specific intent of stimulating change to correct imbalances in the traditional scholarly publishing model.[4,15] SPARC has advocated tirelessly for new methods of scholarly communication such as open access, launching initiatives and partnering with scientific societies with the intent of decreasing the costs of publication/subscription and improving scientific communication.[3,15] The Budapest Open Access Initiative (BOAI) in 2001 resulted in the Budapest Statement, the first such declaration advocating and defining the principle of open access. Illustrating the widespread growth of open access, over 5,000 signatories as of now have agreed with the principles stated in the BOAI.[16] In 2003 the Howard Hughes Medical Institute, the largest private, non-profit funding organization for U.S. biomedical research,[17] drafted the Bethesda Statement, which has become the predominant definition of an open access publication.

Similarly, the World Health Organization has taken steps to expand access to biomedical literature in Third World countries by initiating a unique program, the Health InterNetwork Access to Research Initiative (HINARI). In 2002 HINARI was launched with the express purpose of providing free to low-cost online access to biomedical journals to non-profit organizations in developing countries.[18,19] Institutions in countries with a gross national income (GNI) per capita of less than $1,250 (BAND 1) are given free access to the journals provided in the HINARI database.[18,20] Institutions in countries with a GNI per capita between $1,250 and $3,000 (BAND 2) must pay a yearly fee of $1,000 to access these journals. Currently, over 2,500 institutions in 109 countries have access to more than 3,750 journals in an effort to benefit health workers and researchers and improve the quality of health in the world's poorest nations.[21]

However, HINARI has not been entirely successful. One problem is that access is dependent on an infrastructure that is often lacking or broken due to undependable power supplies and faulty internet connections,[22] which can be so

slow that articles take days to download.[23] Also, current copyright laws prohibit reproduction, dissemination, translation, etc., which provide an additional burden to the local institutions in countries where internet access is already unreliable.[24] In Peru, researchers are finding it hard to justify the $1,000 subscription fee when they are unable to access many of the promised journals sponsored by the major publishing companies in HINARI.[20] Lastly, the GNI per capita requirements themselves severely limit access to these journals in countries such as China, Brazil, and India that do not meet the requirements yet have a pressing need for up-to-date medical/scientific information.[24] HINARI's mixed performance lends further credence to the idea that open access can provide an effective solution to global access inequities and should be the future of publishing. An open access journal would bypass many of these limitations, as anyone with an internet connection anywhere would be able to access the information at anytime.

On a more positive note, large and influential funding organizations such as the National Institutes of Health (NIH), the Wellcome Trust, European Research Council, and the Canadian Institutes of Health Research have begun requiring open access as a stipulation for funding.[25] In 2006 the Wellcome Trust, the largest biomedical research charity in the world,[17] mandated that all their funded research be archived in PubMed Central and UK PubMed Central (open access online repositories of biomedical and life sciences journal literature) within six months.[25] A year later, the European Research Council, the major funding body of the European Union, followed suit, requiring that all research funded by them be put in an online repository such as PubMed Central.[25] Since December 2007, NIH-funded research must be open access through PubMed Central no later than 12 months after the official date of publication.[26] These major funding organizations realize that intellectual property produced in part by their funds should not be transferred over to a publishing company for profit, but should be free for the public's benefit.

Most universities recognize the importance of faculty keeping copyright to their written work, ideally in order to share that work with the rest of the world. For instance, the University of California policy grants copyright ownership to their faculty for their original scholarly work.[27] The copyright owner has exclusive rights to reproduce, prepare derivative works, distribute, perform and display publicly. The purpose of this policy is to fulfill the university's mission of "contributing to the body of knowledge for public good." The University of California also "encourages the creation of original works of authorship and the free expression and exchange of ideas." With authors ceding copyright ownership to publishing companies only to buy it back with subscription, one has to consider whether this promotes the public good. Information in the subscription-based model is at the consumer's cost. By contrast, open access encourages the free exchange of ideas and supports the idea that scholarly work is a public resource that should be available without impediment for the public's benefit.

Open access detractors argue that it devalues research because it does not produce enough capital to create a formidable publication. Granted, while financing remains a challenge for the open access model, a publication's value does not come from the extrinsic monetary value of a subscription but from the value of the research being published. Financing open access can be achieved in various ways, including having authors pay for publication, advertising, or charging for print copies. If an open access journal can find the means to finance its operations, it remains a viable alternative to the traditional publishing model, and with the growing support for open access it may just be the future of scientific publishing.

A 2006 study showed that the majority of emergency medicine physicians favored the open access model,[28] and it is easy to understand why. For too long, the traditional publishing model has constrained the spread of scientific knowledge. Changes in technology and society have provided us with the means to alter this dynamic, especially given the fact that global access to the internet has increased approximately 300% over the past eight years.[29] Despite its limitations, open access provides greater access to scientific knowledge. This is especially important for not only emergency medicine practitioners, but all of science, technology and medical publishing. As such, it has a strong potential to improve the quality of healthcare throughout the world.

To the best of our knowledge, the *Western Journal of Emergency Medicine* (*West*JEM) is the only peer-reviewed emergency medicine open access journal in the world that allows authors to keep their copyright and does not charge authors a publication fee. With limited access to emergency medicine research and only four major emergency medicine journals accessible by subscription, *West*JEM is paving the way for a new publication model, one in which anyone can access its cutting-edge research information.

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REFERENCES

1. Oldenburg, H. Philosophical Transactions: Giving some accompt of the present undertakings, studies, and labours of the ingenious in many considerable parts of the world. *The Philosophical Transactions of the Royal Society*. 1665; 1.
2. Guédon, J-C. *In Oldenburg's Long Shadow: Librarians, Research Scientists, Publishers, and the Control of Scientific Publishing*. Annapolis Junction, MD: ARL Publications; 2001.
3. Anton B. Issues and impacts of the changing nature of scientific communication. *Optom Vis Sci*. 2003; 80:403–410.
4. Liesegang TJ, Schachat AP, Albert DM. Perspectives: The Open Access Initiative in Scientific and Biomedical Publishing: Fourth in the Series on Editorship. Am J Ophthalmol 2005; 139:156-167.
5. EBSCO Information Services. Five year journal price increase history (2001–2005). Birmingham, AL: EBSCO Information Services; 2004: Available at: http://www.ebsco.com/home/printsubs/priceoverview.pdf. Accessed July 16, 2008.
6. Kean G. 19th Annual Study of Journal Prices for Scientific and Medical Society Journals. *JP: Newsl J Pub.* 2007. Available at: http://www.allenpress.com/static/newsletters/pdf/JP-2007-01.pdf. Accessed August 10, 2008.
7. Gooden P, Owen M, Simon S. Scientific publishing: knowledge is power. Morgan Stanley. 2002; Available at: http://www.econ.ucsb.edu/~tedb/Journals/morganstanley.pdf. Accessed July 16, 2008.
8. University of California Office of Scholarly Communication. *The facts: the economics of publishing*. 2004; Available at: http://osc.universityofcalifornia.edu/facts/econ_of_publishing.html. Accessed July 16, 2008.
9. Carnevale D. Libraries With Tight Budgets Renew Complaints About Elsevier's Online Pricing. Chronicle of Higher Education, Section: Information Technology. 2003; 50: A33. Available at: http://chronicle.com/prm/weekly/v50/i17/17a03302.htm. Accessed July 11, 2008.
10. Office of Systemwide Planning for Libraries and Scholarly Communication. *UC Libraries' negotiations with Elsevier*. The Regents of the University of California. 2003; Available at: http://libraries.universityofcalifornia.edu/news/elsevier_update.html.
11. Hane PJ. Cornell and Other University Libraries to Cancel Elsevier Titles. Information Today, Inc. Medford, NJ. Available at: http://newsbreaks.infotoday.com/nbreader.asp?ArticleID=16580. Accessed July 01, 2008.
12. Van Orsdel L, Born K. Periodicals price survey: 2004: closing in on open access. Libr J 2004; 129:45–50.
13. Guterman L. The Promise and Peril of "Open Access". *The Chronicle of higher education*. 2004; 50:A10.
14. Scientific Information Working Group. Bethesda Statement on Open Access Publishing. World Summit on the Information Society, October 30, 2003; Geneva, Switzerland. Available at: http://www.wsis-si.org/mdpi-bethesda.pdf. Accessed July 1, 2008.
15. Scholarly Publishing and Academic Resources Coalition (SPARC). SPARC. Available at: http://www.arl.org/sparc/about/index.shtml. Accessed June 23, 2008.
16. Chan L, et al. Budapest Open Access Initiative (BAOI). Available at: http://www.soros.org/openaccess/index.shtml. Accessed June 23, 2008
17. Cohen J. Science Funding: Philanthropy's Rising Tide Lifts Science. *Science*. 1999; 286: 214-223. Available at: http://www.sciencemag.org/cgi/content/full/286/5438/214. Accessed July 09, 2008.
18. World Health Organization. About HINARI. HINARI: Access to Research. Available at: http://www.who.int/hinari/about/en. Accessed July 09, 2008.
19. Aronson B. WHO's Health InterNetwork Access to Research Initiative (HINARI). *Health Info Libr J*. 2002; 19:164–165.
20. Villafuerte-Gálvez J, Curioso WH, Gayoso O. Biomedical Journals and Global Poverty: Is HINARI a step backwards? *PLoS Med*. 2007; 4: e220. doi:10.1371/ journal.pmed.0040220
21. HINARI. Press Release: 2500th Member Joins Developing Country Institutes with Low-Cost Access to World's Medical Literature. Geneva, Switzerland. Available at http://www.who.int/hinari/2500th%20member.pdf. Accessed June 23, 2008.
22. Smith H, Bukirwa H, Mukasa O, Snell P, Adeh-Nsoh S, Mbuyita S, Honorati M, Orji B, Garner P. Access to electronic health knowledge in five countries in Africa: a descriptive study. *Bio Med Central Health Services Research*. 2007; 7:72. doi:10.1186/1472-6963-7-72.
23. Brown H. For richer for poorer. *BMJ*. 2007; 335; 280-283.
24. The PLoS Medicine Editors. How can biomedical journals help to tackle global poverty? *PLoS Med.* 2006; 3 (8): e380. DOI: 10.1371/journal.pmed.0030380
25. Mayor S. Opening the lid on open access. *BMJ*. 2008; 336;688-689.
26. National Institutes of Health (NIH). NIH Public Access Policy Details. Unites States Department of Health and Human Services. Available at: http://publicaccess.nih.gov/policy.htm. Accessed June 23, 2008.
27. University of California Office of the President. *University of California policy on copyright ownership*. 1992. Available at: http://www.ucop.edu/ucophome/coordrev/policy/8-19-92att.html. Accessed July 11, 2008.
28. Rodriguez RM, Wong J, Hardy J, et al. An evaluation of emergency medicine investigators' views on open access literature. *Emerg Med J*. 2006; 23:895-898.
29. Miniwatts Marketing Group. Internet World Stats: Usage and Population Statistics. Available at: http://www.internetworldstats.com/stats1.htm. Accessed July 14, 2008.

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IN EMERGENCY MEDICAL SERVICES AND DISASTER MEDICINE

University of California, Irvine

University of California, Irvine, Department of Emergency Medicine is seeking an HS Clinical Instructor for July, 2009. UC Irvine Medical Center is a Level I Trauma center with 2200 runs/year, 40,000 ED census. The fellowship in Emergency Medical Services and Disaster Medicine, beginning July 1, 2009, combines the traditional emphasis on EMS research with the disciplines of emergency management/disaster medicine and public health. A key focus of the fellowship is health policy and health services systems research including mass casualty management and triage. Completion of American Council of Graduate Medical Education (ACGME) accredited Emergency Medicine Residency required prior to start. The two-year combined program, with an integrated Masters of Public Health, will be jointly administered by Director, EMS and Disaster Medicine. Salary commensurate with level of clinical work. Send CV, statement of interest and three letters of recommendation to:

Carl Schultz, MD.
Department of Emergency Medicine, Route 128,
UC Irvine Medical Center,
101 City Drive, Orange, CA 92868.

The University of California, Irvine is an equal opportunity employer committed to excellence through diversity.

RESEARCH FELLOWSHIP

PUBLIC HEALTH / INJURY PREVENTION

University of California, Irvine

University of California, Irvine, Department of Emergency Medicine is seeking an HS Clinical Instructor- Research Fellow for July, 2009. University of California, Irvine Medical Center is a Level I Trauma center with 2200 runs/year, 40,000 ED census. This two-year research fellowship will include formal public health education leading to an MPH degree coupled with training in injury prevention with a traffic safety focus. Completion of an ACGME accredited Emergency Medicine Residency is required prior to start. Salary is commensurate with the level of clinical work. Send CV, statement of interest, and three letters of recommendation to:

Federico Vaca, MD, MPH, FACEP
Department of Emergency Medicine
UC Irvine Medical Center, Route 128-01
101 The City Dr, Orange, CA 92868

or

Email to: fevaca@uci.edu
Phone: 714-456-6986
Website: http://www.ucihs.uci.edu/ctipr/.

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FACULTY POSITIONS IN EMERGENCY MEDICINE

American University of Beirut, Faculty of Medicine and Medical Center, Beirut, Lebanon

The Faculty of Medicine and Medical Center of the American University of Beirut, Beirut, Lebanon, is establishing a high quality Academic Department of Emergency Medicine. We are actively seeking experienced Emergency Medicine physicians for this development. Candidates must be board-certified or -eligible in Emergency Medicine by the American Board of Emergency Medicine or the American Board of Osteopathic Emergency Medicine and must have at least three years successful experience in Emergency Medicine. Excellent opportunities exist for faculty development, research and teaching. The compensation is competitive, and the position offers excellent benefits.

To apply please send a cover letter, CV and names of three references to:

Amin Antoine N. Kazzi, MD, FAAEM
Chief of Service & Medical Director, Emergency Department
AUB Faculty of Medicine and Medical Center
American University of Beirut
P.O.Box 11-0236 / Medical Dean's Office
Riad El-Solh / Beirut 1107 2020, Lebanon

For additional information, e-mail: ak63@aub.edu.lb

The American University of Beirut is an affirmative action, equal opportunity employer.

Faculty
Department of
Emergency Medicine

The Department of Emergency Medicine of the Henry Ford Health System is seeking board certified/ prepared emergency physicians to join the faculty of our Emergency Medicine Residency Program. The successful candidate will join our staff of 30 full-time faculty involved in teaching and research. The Emergency Medicine residency program is in its 32nd year and has a total of 53 residents in three tracks including EM, EM/IM, and EM/IM/Critical Care. We have an active research division with NIH funding that focuses on sepsis, the emerging role of biomarkers, neurologic emergencies including stroke, hemorrhage, seizure, and brain injuries. This ED is the birthplace of early goal directed therapy.

The Henry Ford Hospital Emergency Department is an urban, level 1 trauma center with over 90,000 patient visits per year. Our facility has 80 rooms including a 16 bed emergency critical care unit. The Henry Ford Health System is known for its comprehensive, system-wide, electronic medical record (EMR). The Emergency Department is paperless and is fully integrated into the system's EMR.

Henry Ford Health System is an integrated health system that includes 25 medical centers and six hospitals. It is the largest health system in Southeast Michigan (population approaching 5 million). The Henry Ford Hospital is a 750 bed facility serving as the flagship of the system.

Qualified candidates must have a strong interest in furthering the academic mission of the department through research and education. We offer a highly competitive salary and excellent benefits. Please forward your CV for consideration to Scott Johnson, Physician Recruiter. Email: sjohns10@hfhs.org or fax to (313)874-4677. AA/EEO

Emergency
Physician

Henry Ford Health System's Department of Emergency Medicine is seeking board certified/prepared Emergency Physicians to join the staff at Henry Ford Cottage Hospital in Grosse Pointe Farms, Michigan. Successful candidates will become members of one of the nation's largest and most experienced group practices, with more than 1,000 physicians and researchers in more than 40 specialties serving patients throughout southeast Michigan.

The Henry Ford Health System Emergency Medicine service line provided treatment for over 215,000 patients in our 5 locations last year and is rapidly expanding. Henry Ford Cottage Hospital has served thousands of patients from the Grosse Pointe and other eastside communities for more than 85 years.

The ED at Cottage Hospital has over 16,000 visits per year. This site offers CT and ultrasound as well as staff radiologist interpretation. Our ED physicians work 12 hour shifts, have PA coverage on afternoons, and also have full specialty backup. Rotation to the ED at Henry Ford Hospital (Level 1 Trauma Unit) is available.

Qualified candidates must have demonstrated abilities in the areas of clinical care, service excellence, quality improvement, academics and research. We offer a competitive salary and benefits including fully paid malpractice insurance, affordable family health benefits, and an excellent retirement program.

Please forward your CV for consideration to Scott Johnson, Physician Recruiter. Email: sjohns10@hfhs.org or fax to (313)874-4677. AA/EEO

Emergency
Physician

Henry Ford Health System (HFHS) is seeking board certified/ prepared Emergency Physicians to join the staff at the Henry Ford Fairlane Medical Center in Dearborn, Michigan. Successful candidates will become members of one of the nation's largest and most experienced group practices, with more than 1,000 physicians and researchers in more than 40 specialties serving patients throughout southeast Michigan.

The HFHS Emergency Medicine Service Line provided treatment for over 215,000 patients in our 5 locations last year. The Henry Ford Fairlane Emergency Department itself had over 55,000 ED visits. This site offers CT and ultrasound as well as staff radiologist interpretation 24/7. The 74 hours of daily physician coverage is supplemented by 16 hours of mid-level coverage. A distinct pediatric ED is open 24 hours and had over 17,000 visits last year. The Clinical Decision Unit has 6 beds and will soon be expanded to 12 beds. Full subspecialty back up is available 24/7, either on-site or at Henry Ford Hospital.

Qualified candidates must have demonstrated abilities in the areas of clinical care, service excellence, quality improvement, academics and research. We offer a competitive salary and benefits including fully paid malpractice insurance, affordable family health benefits, and an excellent retirement program.

Forward your CV for consideration to Scott Johnson, Physician Recruiter. Email: sjohns10@hfhs.org or fax to (313)874-4677. AA/EEO

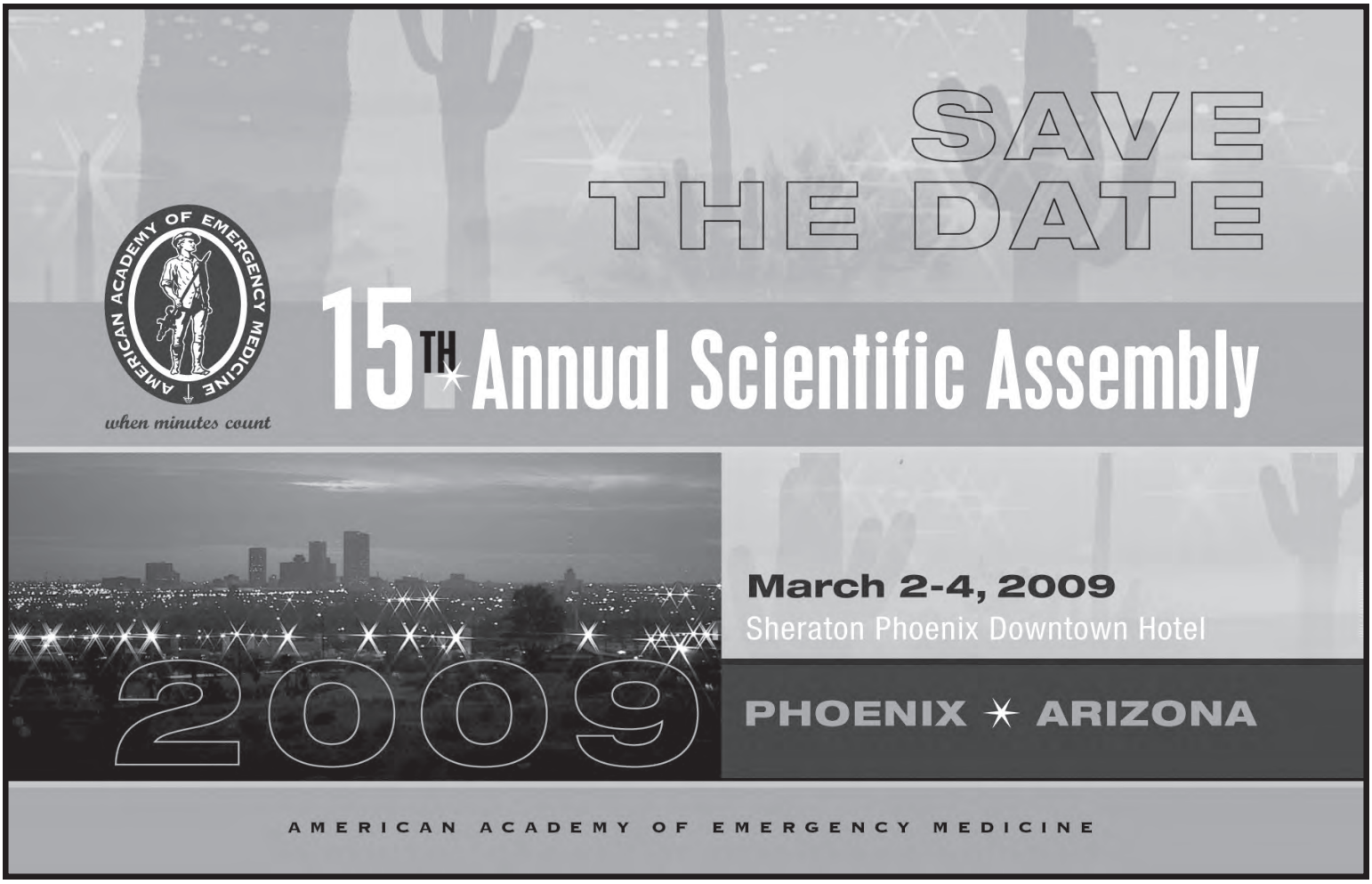
Emergency Physician

Henry Ford Health System's Department of Emergency Medicine is seeking board certified/prepared Emergency Physicians to join the staff at our new Henry Ford West Bloomfield Hospital in West Bloomfield, MI. Successful candidates will become members of one of the nation's largest and most experienced group practices, with more than 1,000 physicians and researchers in more than 40 specialties serving patients throughout southeastern Michigan.

The Henry Ford West Bloomfield Emergency Department expects over 26,000 visits in 2009 and to increase to over 48,000 by 2013. The site offers 24 hr CT and ultrasound with real time radiology interpretation and full specialty back up. The 30 hours of daily physician coverage is supplemented by 12 hours of mid-level coverage. The ED is "paperless" utilizing an electronic medical record including physician order entry (PICIS). The new 34 bed ED, opening in March of 2009, will feature large private patient rooms with baths, computer workstations in every room, a two bed trauma bay and separate Fast Track area.

Qualified candidates must have demonstrated abilities in the areas of clinical care and service excellence. Staff members are also expected to contribute to department activities in the areas of quality improvement, academics, and research.

We offer a competitive salary and benefits including fully paid malpractice insurance, affordable family health benefits, and an excellent retirement program. Please forward your CV for consideration to Scott Johnson, Senior Physician Recruiter. Email: sjohns10@hfhs.org or fax to (313)874-4677. AA/EEO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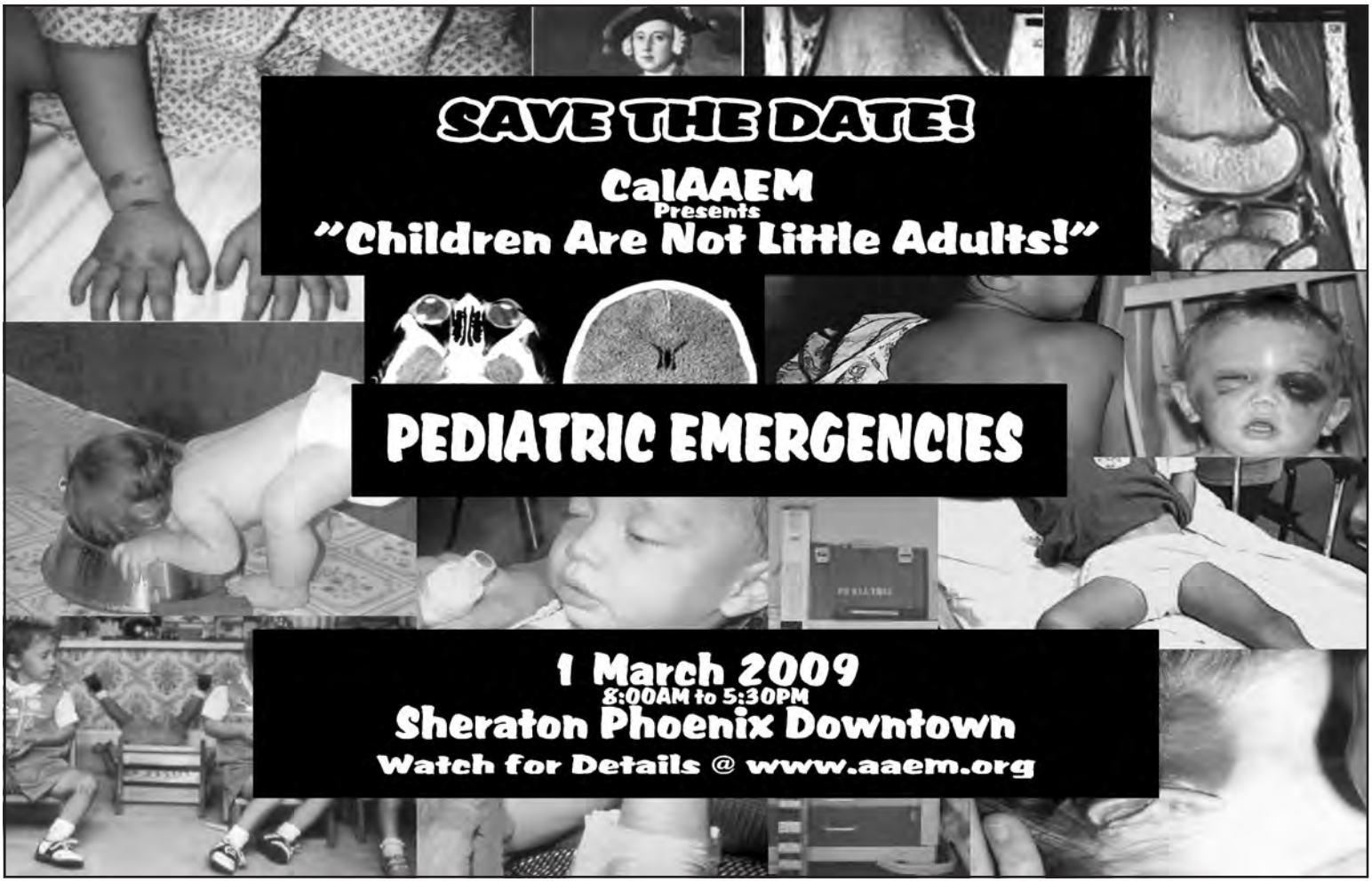
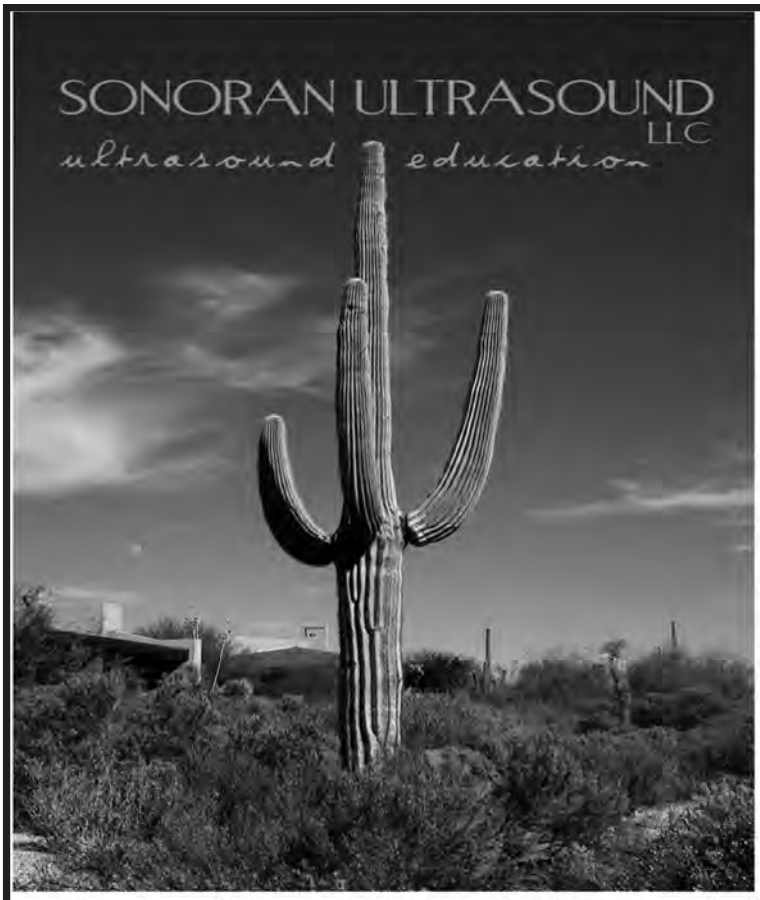
*Alaskan Airlines new service from LAX to Mammoth for $79 one-way

http://mammothmountainemconf.com





FACULTY EXPERTISE

Sonoran Ultrasound, LLC is comprised of nationally recognized emergency physician experts in ultrasound. All instructors are fellowship trained and have Registered Diagnostic Medical Sonographer (RDMS) Credential.

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DID YOU KNOW?

The South Coast Air Quality Management District (AQMD) is the air pollution control agency for Orange County and urban areas of Los Angeles, San Bernardino and Riverside counties.

AIR POLLUTION IS DIRECTLY LINKED TO:

- An estimated 5,400 premature deaths
- 140,000 asthma & lower respiratory symptoms
- 2,400 hospitalizations
- 980,000 lost work days

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For more information, contact AQMD's Clean Air Congress at:

www.CleanAirCongress.org
1-800-4-CLN-AIR or
cleanaircongress@aqmd.gov

AQMD

South Coast Air Quality Management District