



West JEM

Volume IX, Number 3, August 2008

Open Access at www.westjem.org

ISSN 1936-900X

Western Journal of Emergency Medicine

A Peer-Reviewed Professional Journal



PRSTD STD
US POSTAGE
PAID
PERMIT #646
ORANGE, CA

PEDIATRICS

- 130 **Health Literacy among Parents of Pediatric Patients Seen in the Emergency Department** (Original Research)
TP Tran, LM Robinson, JR Keebler, RA Walker, MC Wadman
- 135 **Prevalence of *Bordetella pertussis* and *Bordetella parapertussis* in Samples Submitted for RSV Screening** (Original Research)
P Walsh, C Overmeyer, L Kimmel, M Feola, J Pusavat, TA Nguyen, S Kuan, K Emery, M Rosengreen, E Mordechai, ME Adelson

EDUCATION

- 141 **Self-Reported Emergency Medicine Residency Applicant Attitudes Towards a Procedural Cadaver Laboratory Curriculum** (Original Research)
L Hoffman, MC Wadman, T Erickson, R Muelleman

TOXICOLOGY

- 146 **Tinnitus as a Measure of Salicylate Toxicity in the Overdose Setting** (Original Research)
SR Samlan, MT Jordan, SB Chan, MS Wahl, RL Rubin
- 150 **Comparison of Methanol Exposure Routes Reported to Texas Poison Control Centers** (Original Research)
M Givens, K Kalbfleisch, S Bryson
- 154 **Fluoxetine Overdose-Induced Seizure** (Case Report)
JR Suchard
- 157 **Survival after an Intentional Ingestion of Crushed Abrus Seeds** (Case Report)
L Reedman, RD Shih, O Hung
- 160 **Fatal Metformin Overdose Presenting with Progressive Hyperglycemia** (Case Report)
JR Suchard, TA Grotsky
- 165 **Images in Emergency Medicine: Chemical Pneumonitis from Hydrocarbon Aspiration** (Images in Emergency Medicine)
K Pham, J Sverchek, RA McPheeters

EMS REVIEW

- 166 **Issues and Solutions in Introducing Western Systems to the Prehospital Care System in Japan** (Review)
T Suzuki, M Nishida, Y Suzuki, K Kobayashi, SV Mahadevan

Contents continued inside



Danger can
strike
at the most
unexpected times.

The threat continues long after
the summer has ended.

Now is the right time to check your hospital's
inventory level of CroFab®
Crotalidae Polyvalent Immune Fab (Ovine).

CroFab® Crotalidae Polyvalent Immune Fab (Ovine)



Please contact Fougera® Customer Service at (800) 645-9833 for additional
CroFab® information, or to be connected to your sales representative.

Manufactured by: Protherics Inc., Brentwood, TN 37027
Distributed by: E. FOUGERA & CO. and Savage Laboratories®
both divisions of Nycomed US Inc., Melville, NY 11747

© Copyright Nycomed US Inc. 2008. All rights reserved. Rev. 2/08

Full prescribing information is available at www.fougera.com

Western Journal of Emergency Medicine

Mark Langdorf, MD, MHPE, Editor-in-Chief
University of California, Irvine School of Medicine

Sean Henderson, MD, Senior Associate Editor
Keck School of Medicine, University of Southern California

Shahram Lotfipour, MD, MPH, Managing Associate Editor
University of California, Irvine School of Medicine
Email: shl@uci.edu

Section Editors

Behavioral Emergencies

Eric Isaacs, MD
University of California, San Francisco

Critical Care

H. Bryant Nguyen, MD
Loma Linda University
Jeffrey Sankoff, MD
University of Colorado

Disaster Medicine

Christopher Kang, MD
Madigan Army Medical Center
Colleen Buono, MD
University of California, San Diego

Education

Michael Epter, DO
University of Nevada
H. Gene Hern, MD, MS
Alameda County Medical Center

Emergency Department Administration

Jeffrey Druck, MD
University of Colorado

Emergency Cardiac Care

Matthew Strehlow, MD
Stanford University

Emergency Medical Services

Christopher Kahn, MD, MPH
University of California, Irvine
David E Slattery, MD
University of Nevada

Geriatrics

Teresita M. Hogan, MD
Resurrection Medical Center, Chicago

Health Policy

Michael Menchine, MD, MPH
University of California, Irvine
Stephen Wolf, MD
Denver Health Medical Center

Infectious Disease

Robert Derlet, MD
University of California, Davis

Information Technology

James Killeen, MD
University of California, San Diego
Scott Rudkin, MD, MBA
University of California, Irvine

Injury Prevention

Bharath Chakravarthy, MD, MPH
Loma Linda University
Shahram Lotfipour, MD, MPH
University of California, Irvine

International Medicine

Tareq Bey, MD
University of California, Irvine
Chris Mills, MD
George Washington University

Legal Medicine

Greg P. Moore, MD, JD
Madigan Army Medical Center

Methodology and Biostatistics

Craig Anderson, MPH, PhD
University of California, Irvine
Christian McClung, MD
University of Southern California

Neurosciences

Kurt Denninghoff, MD
University of Arizona

Pediatric Emergency Medicine

Judith Klein, MD
University of California, San Francisco
Paul Walsh, MB, BCH, BAO, MSc
Kern Medical Center

Public Health

Harrison Alter, MD
Alameda County Medical Center

Resident/Student/Fellow Forum

Kapil R. Dhingra, MD, MBA
University of California, Davis
Laleh Gharahbaghian, MD
Stanford University

Toxicology

Jeffrey Suchard, MD
University of California, Irvine
Brandon Wills, DO, MS
Madigan Army Medical Center

Ultrasound

Seric Cusick, MD, RDMS
University of California, Davis
J. Christian Fox, MD, RDMS
University of California, Irvine

Visual Diagnosis

Eric Snoey, MD
Alameda County Medical Center
Rick McPheeters, DO
Kern Medical Center

Editorial Board

Steven Gabaeff, MD
California Chapter of AAEM

Laleh Gharahbaghian, MD
Stanford University

Sean Henderson, MD
University of Southern California

Mark Langdorf, MD, MHPE
University of California, Irvine

Shahram Lotfipour, MD, MPH
University of California, Irvine

Susan Promes, MD
University of California, San Francisco

Robert M. Rodriguez, MD
University of California, San Francisco

Scott Rudkin, MD, MBA
University of California, Irvine

Stuart Swadron, MD
University of Southern California

Editorial Staff

Suleman S. Ahmed, BS, BA
Associate Editorial and Publishing Manager
WestJEM@gmail.com
714-456-6389

June Casey, BA
Editorial Manager and Copy Editor

Christina Cheng, BS
Design Editor

Warren Wiechmann, MD, MBA
Webmaster

Sonia Johnson, MD
Graphic Design Consultant

Publisher

Department of Emergency
Medicine, University of California,
Irvine School of Medicine

Official publication of the California Chapter of the American Academy of Emergency Medicine

Articles now available on Google Scholar, eScholarship, Melvyl (The Catalog of the University of California Libraries), Directory of Open Access Journals, and MDLinx Emergency Med. Will be available in Pub Med Central and Medscape.

WestJEM, 101 The City Drive, Rt. 128-01, Orange, CA 92868-3201: Office (714) 456-6389

Acknowledgment of *WestJEM* Top Reviewers and Section Editors

Volume IX, Number 3 : August 2008
www.westjem.org

As the summer of 2008 draws to a close, *WestJEM* would like to honor the top reviewers and section editors of the past year. Without their generous contributions of time and expertise to the peer-review process, the *Journal* would not be able to advance its mission of emphasizing practical, efficient, and world-class emergency care.

To begin with, the *WestJEM* editors would like to pay special tribute to top reviewer Dr. Paul Walsh from Kern Medical Center Emergency Medicine Residency, for leading all reviewers with eight reviews in the past year. Drs. Jeffrey Druck (University of Colorado/Denver General Hospital), Melissa Givens (Darnall Army Medical Center), Laleh Gharahbaghian (Stanford University), and Craig Anderson (University of California, Irvine) all had several reviews during the same time period. Dr. Givens' reviews were done in an average three days.

Finally, special thanks to Section Editors, Dr. Rick McPheeters (Kern Medical Center, Visual Diagnosis) and Dr. Brandon Wills (Madigan Army Medical Center, Toxicology) for their enthusiasm and participation.



Western Journal of Emergency Medicine

Table of Contents,

continued

Volume IX, Number 3 : August 2008
www.westjem.org

INFECTIOUS DISEASE

- 171 **A Case of Complicated Urinary Tract Infection: *Klebsiella pneumoniae* Emphysematous Cystitis Presenting as Abdominal Pain in the Emergency Department (Case Report)**
KR Dhingra
- 174 **Herpes Zoster Ophthalmicus (Case Report)**
T Catron, HG Hern
- 177 **Complications of MRSA Treatment: Linezolid-induced Myelosuppression Presenting with Pancytopenia (Case Report)**
J Gorchynski, JK Rose

IMAGES IN EMERGENCY MEDICINE

- 179 **Images in Emergency Medicine: Splenic Infarction Due to Sickle Cell Trait after Climbing Mt. Fuji (Images in Emergency Medicine)**
A Morishima, JM Schofer, P Pelletier, JM McKee
- 180 **Images in Emergency Medicine: Pelvic Digit (Images in Emergency Medicine)**
J Carnell, C Fee

ABSTRACTS

- 181 **Society for Academic Emergency Medicine Western Regional Research Forum (Abstracts)**

Health Literacy among Parents of Pediatric Patients

T. Paul Tran, MD
Laura M. Robinson, MPA
John R. Keebler, MD
Richard A. Walker, MD
Michael C. Wadman, MD

University of Nebraska Medical Center

Supervising Section Editor: Paul Walsh, MD, MSc

Submission history: Submitted October 16, 2007; Revision Received March 31, 2008; Accepted April 5, 2008.

Reprints available through open access at www.westjem.org

Background: Health literacy is an important predictor of healthcare outcomes, but research on this topic has largely been absent from the emergency medicine literature.

Objective: We measured the prevalence of health literacy in parents or guardians of pediatric patients seen in the emergency department (ED).

Methods: This was an observational study conducted in a Midwestern urban, university-based, tertiary, Level 1 trauma center ED with 33,000 visits/year. Using convenience sampling during a three-month period, English-speaking parents or guardians of pediatric patients (< 19 yrs.) were asked to complete the short version of the Test of Functional Health Literacy for Adults (s-TOFHLA). Parents/guardians were excluded if they had uncorrected visual impairment, required an interpreter, had altered mental status, or if the patients they accompanied were the subjects of a medical or trauma activation.

Results: Of the 188 parents or guardians approached, six did not consent or withdrew, one was excluded, leaving 181 (96.3%) in the study. Of these, 19 (10.5%) had either "marginal" or "inadequate" health literacy, while 162 (89.5%, 95% CI: 84.1%, 93.6%) had "adequate" health literacy.

Conclusion: A large majority (89.5%) of English-speaking parents or guardians of pediatric patients evaluated in the ED have adequate health literacy. This data may prompt ED professionals to adjust their communication styles in the evaluation of children. Future multi-center studies are needed to confirm the findings in this pilot study.

[WestJEM. 2008;9:130-134.]

INTRODUCTION

Health literacy is defined as the ability of patients to perform the basic reading and computational tasks required to function effectively in the healthcare environment.¹ Directly or indirectly through written words, health literacy skills are required in various aspects of doctor-patient dialogue, discussion of diagnostic and therapeutic plans, use of medical tools such as nebulizers or peak flow meters, health information, follow-up instructions, or administration of home medications. Poor health literacy is associated with several adverse health outcomes including increased incidence of chronic illness, poor intermediate disease markers, sub-

optimal use of preventative resources, and increased rates of hospitalization and use of emergency services.^{1,2} The potential impact of inadequate health literacy on health outcomes is significant. Almost half of all U.S. adults, approximately 90 million people, were found to have difficulty understanding and acting on health information in a 1992 literacy survey.³ This and the recent reports by the American Medical Association (AMA) and Institute of Medicine (IOM), highlighting the importance of health literacy and its impact on health care outcomes, indicate the increasing focus on this new field of study.^{1,4}

Although significant, the issue of health literacy among

Table 1. Study Summary

	Total (n = 181)	Adequate Health Literacy		Inadequate Health Literacy
		Subtotal (%)	95% CI	Subtotal (%)
All subjects		162 (89.5%)	84.1%-93.6%	19 (10.5%)
Ethnicity				
Caucasian	78 (43.1%)	73 (93.6%)	85.7%-97.7%	5 (6.4%)
African American	80 (44.2%)	69 (86.3%)	76.7%-92.9%	11 (13.8%)
Hispanic	17 (9.4%)	14 (82.4%)	56.5%-96.2%	3 (17.7%)
Other	6 (3.3%)	6 (100.0%)	54.1%-100%*	0 (0.0%)
Insurance				
Medicaid	125 (69.1%)	108 (86.4%)	79.1%-91.9%	17 (13.6%)
Private	39 (21.5%)	38 (97.4%)	86.5%-99.9%	1 (2.6%)
Self-Pay	17 (9.4%)	16 (94.1%)	71.3%-99.9%	1 (5.9%)
Disposition				
Discharge	167 (92.3%)	148 (88.6%)	82.8%-93.0%	19 (11.4%)
Admit	12 (6.6%)	12 (100.0%)	73.5%-100%*	0 (0.0%)
Transfer	2 (1.1%)	2 (100.0%)	15.8%-100%*	0 (0.0%)
Age of Child				
0 to 5	94 (51.9%)	84 (89.4%)	81.3%-94.8%	10 (10.6%)
6 to 11	43 (23.8%)	36 (83.7%)	69.3%-93.2%	7 (16.3%)
12 to 19	44 (24.3%)	42 (95.4%)	84.5%-99.4%	1 (2.3%)
Gender				
Male	101 (55.8%)	89 (88.1%)	80.2%-93.7%	12 (11.9%)
Female	80 (44.2%)	73 (91.3%)	82.8%-96.4%	7 (8.8%)

* 97.5% one sided CI for these cases

patients seen in emergency departments (EDs) has received scant attention in emergency medicine literature. The often-quoted study by Williams et al.⁵ was performed more than a decade ago, using adult patients recruited from two adult EDs. In this 1995 study, 65% of adults had “adequate” health literacy. We undertook this pilot study to assess health literacy levels in parents or guardians of pediatric patients who presented to our ED for care. Among the patients seen in our nation’s EDs, pediatric patients (< 19 years) constitute one of the most vulnerable populations. Furthermore, pediatric visits are common, accounting for approximately 28% of the 100.4 million annual emergency visits in 2000.⁶ Anecdotal evidence suggests that ED personnel often presume that recurrent use of the ED by pediatric patients may be linked to socioeconomics, ethnicity, and literacy levels by parents or guardians of these children.^{7,8}

This is an observational study conducted at a Midwestern urban university, tertiary referral, Level 1 trauma center ED from May to August 2004. The study was approved by the local Institutional Review Board. The ED serves a greater metropolitan area of approximately three-quarters of a million

people and has a census of 33,000 visits/year, with 24.6% pediatric patients (< 19 years of age). A single trained research assistant (RA) approached consecutive English-speaking parents or guardians of pediatric patients in a given eight-hour shift for participation in the study. Potential study subjects were identified using an online log of ED patients and were approached at the end of each patient’s visit to the ED. The timing of the subject solicitation was selected to minimize parent/guardian distraction and to preserve parent/guardian and patient privacy. A balanced rotating shift schedule (morning, evening, night) covering seven days per week was used to achieve a more representative sampling of the ED population. Parents/guardians were excluded if they had uncorrected impaired visual acuity, cognitive impairment (e.g., alcohol, drugs, trauma), required a language interpreter, or the pediatric patients they accompanied were pregnant or were subjects of a medical code or trauma team activation.

After verbal consent had been obtained and documented on the chart, the RA administered the English version of the abbreviated Test of Functional Health Literacy for Adults (s-TOFHLA) using standard instructions provided by the

test maker. One of the authors (TPT) was licensed to use s-TOFHLA instrument (license #112/03) for research. Details on abbreviated s-TOFHLA are available elsewhere.⁹ Briefly, the abbreviated s-TOFHLA is a 36-item timed-reading comprehension test that measures health literacy skills of a patient in a realistic health care environment. Parents/guardians were asked to read two passages with every fifth or seventh word in the passages omitted, and to fill in the blanks from a list of four word choices. The first passage is a set of instructions for a patient in preparation for an upper gastrointestinal series. The second passage is taken from an applicant's "Rights and Responsibilities" portion of a theoretical Medicaid application. The patient's health literacy score reflects the number of correct answers and ranges from 0-36. Parents/guardians are considered to have "inadequate" health literacy if his/her abbreviated s-TOFHLA score is 0-16, "marginal" health literacy if score is 17-22, and "adequate" health literacy if score is 23-36.⁹

Along with the s-TOFHLA scores, demographic data, social, and medical information for the patients were abstracted from electronic medical records. Data were entered into an Excel® spreadsheet (Microsoft, Seattle, WA) and categorical variables were analyzed using Fisher's exact (SigmaStat™, Point Richmond, CA). Data are reported with 95% *exact* confidence interval (95% CI).

RESULTS

During the study period, 2,209 pediatric patients presented to the ED, out of 8,649 total patients (25.5%). Of these, 188 consecutive parents/guardians were approached, six did not consent or withdrew, and one had incomplete data, leaving 181 (96.28%) in the study. Of the 181 subjects, 162 (89.5%, 95% CI: 84.1%, 93.6%) scored in the "adequate" range (23-36), and 19 (10.5%) scored in the "marginal" or "inadequate" range [six participants (3.3%) scored in 0-12 range, 13 (7.2%) in 13-22 range]. The mean age for the pediatric patients was five years (interquartile range 2-11 years). Table 1 summarizes the study results. Table 2 illustrates the clinical characteristics of our ED during the study period. There were no differences in health literacy levels by ethnicity, insurance status, age, gender, or medical disposition.

DISCUSSION

In this pilot study, almost nine out of 10 parents or guardians of pediatric patients had "adequate" health literacy, as measured by s-TOFHLA. Our findings, if validated in future multi-center studies, may have a significant impact on the way healthcare professionals interact and communicate with parents or guardians of children in the ED. In the subsequent discussion, we will briefly review health literacy and its impact on healthcare outcomes.

From humble beginnings, the last two decades have witnessed the maturity of what is now a legitimate field of

study known officially as "health literacy." Up to 1,000 papers link health literacy to general health, healthcare, and health outcomes.¹⁰ Limited health literacy skills have been associated with disparity in access to care, adverse health outcomes, increased hospitalization, higher healthcare costs, increased use of emergency services, ineffective communication, inability to understand informed consent, and inability to understand verbal and written medical advice.^{2,7,11,12} In the ED and clinic settings, low health literacy adversely impacts patient-physician dynamics, communication, patient satisfaction, and resource utilization.^{7,11-13} One approach healthcare professionals have taken to counter the effects of low health literacy is to tailor health-related communications to patients' literacy and comprehension levels.¹⁴

Health literacy is commonly defined as "the degree to which individuals have the capacity to obtain, process, and understand basic information and services needed to make appropriate decisions regarding their health."¹¹ In the first U. S. government study of its kind, the 1992 National Adult Literacy Survey found that upward of 44 million Americans out of 191 million (23%) were functionally illiterate and 53.5 million (28%) had marginal reading skills.³ Although the term health literacy is closely related to *literacy* – English-language reading skills – they are not the same. Health literacy is a potpourri of reading, comprehension, and computational skills that patients must possess to navigate effectively in a healthcare environment. Not only do patients need to know how to read, they need to know how to interpret their own health issues in the context of their healthcare environment to be health literate. Subsequent to the 1992 National Adult Literacy Survey, the U.S. Department of Education conducted the first national survey on health literacy. In this 2005 study, 35% of Americans were found to have "basic" or "below basic" health literacy.¹⁵

The two instruments most commonly used to approximate health literacy skills in the clinical setting include the Rapid Estimate of Adult Literacy in Medicine (REALM)¹⁶ and the Test of Functional Health Literacy in Adults (TOFHLA).¹⁷ While the REALM can be administered quickly (< 3 minutes), it does not measure comprehension. The TOFHLA, is more comprehensive but more time-consuming (> 20 minutes) to administer. In this study, we elected to use the shortened version of the TOFHLA (s-TOFHLA) because it is a good approximation of the full TOFHLA yet more practical (< 8 minutes) in the ED setting.⁹

Research has gained momentum in the last decade with the selection of health literacy as one of the *20 Healthy People 2010* objectives by the U.S. Department of Health and Human Services¹⁸ and *20 Priority Areas for National Action* by the IOM;¹⁹ however, similar advances have not been observed in emergency medicine literature.² To our knowledge, ours is one of the first two studies in the last decade that examined health literacy among parents or guardians who accompanied

Table 2. Clinical Characteristics of the emergency department (ED).

	N	%
Total ED census during study period	8649	
Total pediatric patient census during study period	2209	25.5%
Total pediatric patients admitted	163	7.4%
Total pediatric patients admitted to ICU	57	2.6%
Insurance		
Medicaid	1512	68.4%
Private	463	21.0%
Others	234	10.6%
Top five discharge diagnoses		
Acute URI/Pharyngitis	855	38.7%
Otitis Media	386	17.5%
Laceration/Suture/Wound care	374	16.9%
Fever < 14 days	352	15.9%
Vomiting not otherwise specified	129	5.8%

pediatric patients to the ED. Compared to Williams's well known study,⁵ results from our study and those by Sanders et al.¹³ suggest that parents or guardians of children seen in the ED tend to have higher health literacy skills – 77.2% in Sanders' and 89.5% in our study vs 65% in Williams'. One possible explanation for the differences is that health literacy declines with age. Since parents of pediatric patients tend to be younger, both studies that involve parents and guardians of children (Sanders and ours) report higher health literacy. Future studies are required to confirm our findings as well as the conclusions by Sanders.

Data in this study suggest two potentially important implications. First, if parents of pediatric patients seen in the ED are in fact more health literate, ED professionals may need to adjust the patient-doctor communications accordingly. Second, fresh health literacy data are needed in the current debates regarding access to emergency care. For example, some arguments for limited access to emergency care are fueled in part by the assumption that poor health literacy may cause over-utilization of emergency services.⁸ New data may thus impact policy-making decisions regarding ED access in the future.

When the "adequate" group was compared to the "marginal/inadequate" group for any predictors of poor health literacy, no evidence emerged that ethnicity, insurance status, medical disposition, age, or gender predicted or predisposed any particular group of patients to inadequate health literacy (Table 1). In our study, pediatric patients accounted for approximately a quarter of our ED visits, with a payer mix that included 68% Medicaid patients (Table 2).

LIMITATIONS

This was a pilot study using convenience sampling of English-speaking subjects at a single site in the Midwest. Results should not be generalized to regional or national ED populations. Although a rolling enrollment design was employed, the study sample was only a small fraction of eligible patients seen in the ED during the study period. The possibility of a non-representative population could not be excluded. We did not include the Spanish version of s-TOFHLA because of the required training in cultural competency and interpreter quality. This exclusion of Spanish-speaking subjects may also have skewed the results. During the study period, however, the Spanish-speaking pediatric patients (requiring a Spanish interpreter) made up a small fraction (5.78%) of our ED's pediatric patient census.

CONCLUSION

A large majority (89.5%) of English-speaking parents or guardians of pediatric patients evaluated in the ED have adequate health literacy. This high prevalence of health literacy may prompt ED professionals to adjust their communication styles in the evaluation of children seen in the ED. While the data from this study may prompt ED professionals to adjust their communication styles in the evaluation of children, future multi-center studies are needed to confirm these findings.

Acknowledgment

The authors would like to thank Jane Meza, Ph.D., Department of Preventative and Societal Medicine, University of Nebraska Medical Center, for her statistical assistance.

Address for Correspondence: Paul Tran, MD, Department of Emergency Medicine, University of Nebraska Medical Center, 981150 Nebraska Medical Center, Omaha, NE 68198-1150. Email: trtran@unmc.edu.

REFERENCES

1. Nielsen-Bohlman L. Institute of Medicine & Committee on Health Literacy *Health literacy: A Prescription To End Confusion*. Washington, DC: National Academies Press; 2004.
2. Berkman ND, et al. Literacy and health outcomes. *Evid Rep Technol Assess (Summ)*. 2004; 87:1-8.
3. Kirsch IS. Educational Testing Service & National Center for Education Statistics *Adult literacy in America: A first look at the results of the National Adult Literacy Survey*. Washington, DC: Office of Educational Research and Improvement, U.S. Dept. of Education; 1993.
4. Ad Hoc Committee on Health Literacy for the Council on Scientific Affairs, American Medical Association. Health Literacy: Report of the Council on Scientific Affairs. *JAMA*. 1999; 281:552-557.

5. Williams MV et al. Inadequate functional health literacy among patients at two public hospitals. *JAMA*. 1995; 274:1677-1682.
6. McCaig LF. National Hospital Ambulatory Medical Care Survey: 1998 emergency department summary. *Adv. Data*. 2000; 1-23.
7. Halfon N, Newacheck PW, Wood DL, St. Peter RF. Routine emergency department use for sick care by children in the United States. *Pediatrics*. 1996; 98:28-34.
8. Vastag B. Low Health Literacy Called a Major Problem. *JAMA*. 2004; 291:2181-2182.
9. Baker DW, Williams MV, Parker RM, Gazmararian JA, Nurss J. Development of a brief test to measure functional health literacy. *Patient Educ Couns*. 1999; 38:33-42.
10. Rudd RE. Health literacy skills of U.S. adults. *Am J Health Behav*. 2007; 31:S8-S18.
11. Baker DW, et al. Health literacy and use of outpatient physician services by Medicare managed care enrollees. *J Gen Intern Med*. 2004; 19:215-220.
12. Davis TC, Wolf MS. Health literacy: implications for family medicine. *Fam Med*. 2004; 36:595-598.
13. Sanders LM, Thompson VT, Wilkinson JD. Caregiver health literacy and the use of child health services. *Pediatrics*. 2007; 119:e86-e92.
14. Williams MV, Davis T, Parker RM, Weiss BD. The role of health literacy in patient-physician communication. *Fam Med*. 2002; 34:383-389.
15. Kutner MA, National Center for Education Statistics. *The health literacy of America's adults results from the 2003 National Assessment of Adult Literacy*. Washington, DC: U.S. Dept of Education, National Center for Education Statistics; 2006.
16. Davis TC, et al. Rapid assessment of literacy levels of adult primary care patients. *Fam Med*. 1991; 23:433-435.
17. Parker RM, Baker DW, Williams MV, Nurss JR. The test of functional health literacy in adults: a new instrument for measuring patients' literacy skills. *J Gen Intern Med*. 1995; 10:537-541.
18. U.S. Department of Health and Human Services: Office of Disease Prevention and Health Promotion. Healthy People 2010. *Nasnewsletter*. 2000; 15:3.
19. Institute of Medicine, Committee on Identifying Priority Areas for Quality Improvement, Adams K, Corrigan J. *Priority areas for national action transforming health care quality*. Washington, DC: National Acad. Press; 2003.

Prevalence of *Bordetella pertussis* and *Bordetella parapertussis* in Samples Submitted for RSV Screening

Paul Walsh, MB, BCh, BAO, MSc*

Christina Overmeyer, BS†

Lauren Kimmel, BS†

Melanie Feola, BS†

James Pusavat, BS‡

Tuan Anh Nguyen*

Sam Kuan, MB, BCh, BAO*

Kirt Emery, MPH#

Martin Rosengreen, MD*

Eli Mordechai, PhD†

Martin E. Adelson, PhD†

* Kern Medical Center, Department of Emergency Medicine and David Geffen School of Medicine, University of California Los Angeles

† Research and Development Department, Medical Diagnostic Laboratories, Hamilton NJ

‡ Kern Medical Center, Department of Pathology

Kern County Department of Public Health

Supervising Section Editor: Robert W. Derlet, MD

Submission history: Submitted November 12, 2007; Revision Received April 18, 2008; Accepted May 15, 2008.

Reprints available through open access at www.westjem.org

Background: The clinical presentation of *Bordetella pertussis* can overlap with that of respiratory syncytial virus (RSV); however, management differs.

Hypothesis: First, the prevalence of *B. pertussis* is less than 2% among patients screened for RSV, and second the prevalence of *B. parapertussis* is also less than 2% among these patients.

Methods: Nasal washings submitted to a clinical laboratory for RSV screening were tested for *B. pertussis* and *B. parapertussis*, using species-specific real-time polymerase chain reaction (PCR) assays. These were optimized to target conserved regions within a complement gene and the *CarB* gene, respectively. A *Bordetella* spp. genus-specific real-time PCR assay was designed to detect the *Bhur* gene of *B. pertussis*, *B. parapertussis*, and *B. bronchiseptica*. RSV A and B subtypes were tested by reverse transcription-PCR.

Results: Four hundred and eighty-nine clinical samples were tested. There was insufficient material to complete testing for one *B. pertussis*, 10 RSV subtype A, and four RSV subtype B assays. *Bordetella pertussis* was detected in 3/488 (0.6%) (95% CI 0.1% to 1.8%), while *B. parapertussis* was detected in 5/489 (1.0%) (95% CI 0.3% to 2.4%). Dual infection of *B. pertussis* with RSV and of *B. parapertussis* with RSV occurred in two and in three cases respectively. RSV was detected by PCR in 127 (26.5%).

Conclusion: The prevalence of *B. pertussis* in nasal washings submitted for RSV screening was less than 2%. The prevalence of parapertussis may be higher than 2%. RSV with *B. pertussis* and RSV with *B. parapertussis* coinfection do occur.

[WestJEM. 2008;9:135-140.]

INTRODUCTION

The clinical presentation of *Bordetella pertussis* can

overlap with and be clinically indistinguishable from that of respiratory syncytial virus (RSV).^{1,2} The potential for

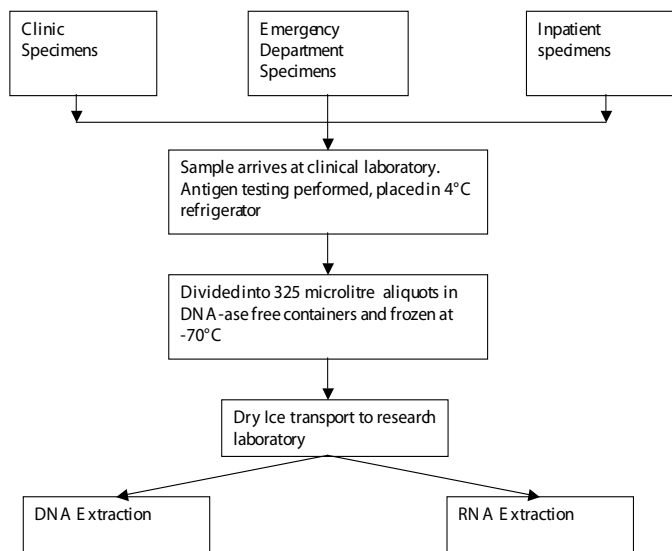


Figure 1. Specimen flow through the study.

morbidity and mortality is higher with *B. pertussis*.^{2,3} Apnea occurs in 15.9% of previously healthy infants less than six months of age who contract pertussis.⁴ Apnea rates for RSV are lower, even in hospitalized infants.⁵ This risk is largely confined to infants less than two months of age and to those with co-morbidities.⁶⁻⁹ Admission criteria reflect these differences with mandatory admission recommended for those with pertussis up to six months of age.¹⁰ Mandatory admission for infants with RSV is controversial, with conflicting published evidence and opinions, but many opt to admit all less than two months of age.^{11,12,13}

RSV epidemiological trends have remained steady¹⁴ whereas pertussis infections have climbed both locally (Kern County Department of Public Health data) and nationally¹⁵ with genomic subtypes not contained in vaccinations being increasingly represented.¹⁶ Previous work among children admitted to intensive care for bronchiolitis in the UK, and among infants less than six months of age hospitalized with bronchiolitis in Finland, suggests that despite the apparently viral nature of their illness (typically RSV), pertussis is under diagnosed.^{1,17} However this experience is not universal with some reporting the opposite.¹⁸ These studies raise the question whether infants and toddlers screened for RSV should also be screened for *B. pertussis*.

While baseline population prevalence data can help inform such a decision, these estimates from public health departments are of limited value. They reflect cases with more typical presentations in which the diagnosis was clinically suspected; even then they are prone to under reporting.^{19,20} Determining the overall baseline-expected prevalence of *B. pertussis* in patients with presentations more typically associated with RSV requires large numbers. It also requires a sample representative of the full spectrum of RSV disease that presents for treatment (primarily bronchiolitis). We tested de-identified specimens previously submitted for RSV screening to determine the prevalence of *B. pertussis* in this population.

We considered that a prevalence of less than 2% would represent an acceptable miss rate for most emergency physicians (EPs) for pertussis and tested the hypothesis that the prevalence of *B. pertussis* is less than 2% in nasal washings screened for RSV. Our secondary hypothesis was that the prevalence of *B. parapertussis* is less than 2% in nasal washings screened for RSV.

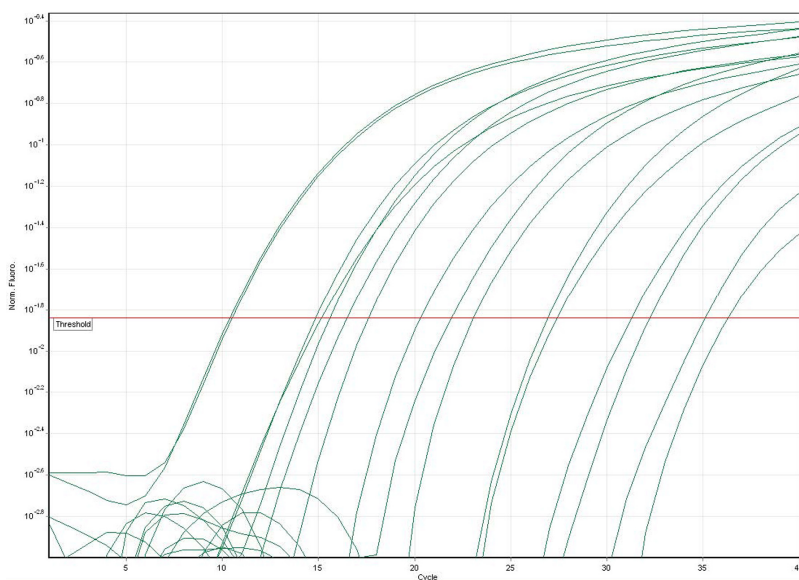


Figure 2. *B. pertussis* range of detection and standard curve ranging from 1×10^{10} copies to 1×10^2 copies.

METHODS

Our institutional review board exempted the study from review.

Setting: A single clinical laboratory serving an emergency department (ED), pediatric clinic, family practice clinic and a county hospital and a diagnostic testing laboratory specializing in the use of molecular amplification assays for the detection of viruses, bacteria, and fungi in biological specimens.

Nasal washings samples collected between October 2005 and May 2006 were first tested for the RSV antigen by the clinical laboratory. They were collected in test tubes without additives using normal saline. The remaining part of each sample was refrigerated to 4°C prior to being divided into 325 µl aliquots into DNA-ase-free transport containers and frozen to -70°C. The flow of specimens is shown in Figure 1.

Upon receipt at the diagnostic testing laboratory the specimens were thawed, and DNA was extracted using an automated Corbett Robotics X-tractor Gene system. Extracted DNA from these specimens were subsequently tested by *Bordetella pertussis* and *Bordetella parapertussis* species-specific real-time polymerase chain reaction (PCR) assays designed and optimized to target conserved regions within a complement gene and the *CarB* gene, respectively. A *Bordetella spp.* genus-specific real-time PCR assay was also designed to detect the *Bhur* gene of *B. pertussis*, *B. parapertussis*, and *B. bronchiseptica*. The characteristics and detection limits of this PCR assay (detection limit for *B. pertussis* 100 copies per reaction, detection limits for *B. parapertussis* 10 copies per reaction) are shown in Figures 2 and 3. Specificity was assessed by testing the assay against known samples of the viral, bacterial, and fungal pathogens listed in Table 1. No amplification for any of these agents was

Table 1. Pathogens for which the real time probe and primer specificity was assessed.

<i>Gardnerella vaginalis</i>	<i>Candida parapsiosis</i>	<i>Bartonella bacilliformis</i>
<i>Neisseria gonorrhoea</i>	<i>Candida tropicalis</i>	<i>Bartonella quintana</i>
<i>Neisseria meningitidis</i>	<i>Candida lusitanae</i>	Human rhinovirus 6
<i>Trichomonas vaginalis</i>	<i>Candida albicans</i>	Human rhinovirus 11
<i>Chlamydia trachomatis</i>	<i>Candida dublinensis</i>	Adenovirus type 10
<i>Chlamydia pneumoniae</i>	<i>Candida utilis</i>	Adenovirus
<i>Ureaplasma urealyticum</i>	<i>Candida krusei</i>	Coxsackie virus
Herpes simplex virus I	Influenza A	<i>Cryptococcus neoformans</i>
Herpes simplex virus II	Influenza B	<i>Babesia microti</i>
Human papilloma virus	<i>Aspergillus fumigans</i>	<i>Bacteroides fragilis</i>
Epstein Barr virus	Human herpes virus 8	<i>Mobiluncus curtisii</i>
Cytomegalovirus	Human herpes virus 6	<i>Mobiluncus mulieris</i>
<i>Mycoplasma fermentens</i>	Canine herpes virus	<i>Legionella pneumophila</i>
<i>Mycoplasma pneumoniae</i>	HTLV 1	<i>Brucellosis ovis</i>
<i>Mycoplasma genitalium</i>	Parainfluenza 2	<i>Borrelia burgdorferi</i>
<i>Mycoplasma hominis</i>	Parainfluenza 3	<i>Streptococcus pneumoniae</i>
		<i>Moraxella catarrhalis</i>

detectable for the *B. pertussis*, *B. parapertussis* or *Bordetella spp.* assays. These assays are available for clinical use from a commercial CLIA approved laboratory.

For the RSV assays, RNA was extracted from the washings using either a modified protocol with the Corbett Robotics X-tractor Gene System or with the QIAamp Viral RNA purification kit (Qiagen, Valencia, CA). Extracted RNA was subsequently tested for RSV-A and RSV-B using two separate one-step reverse transcription PCR assays targeting

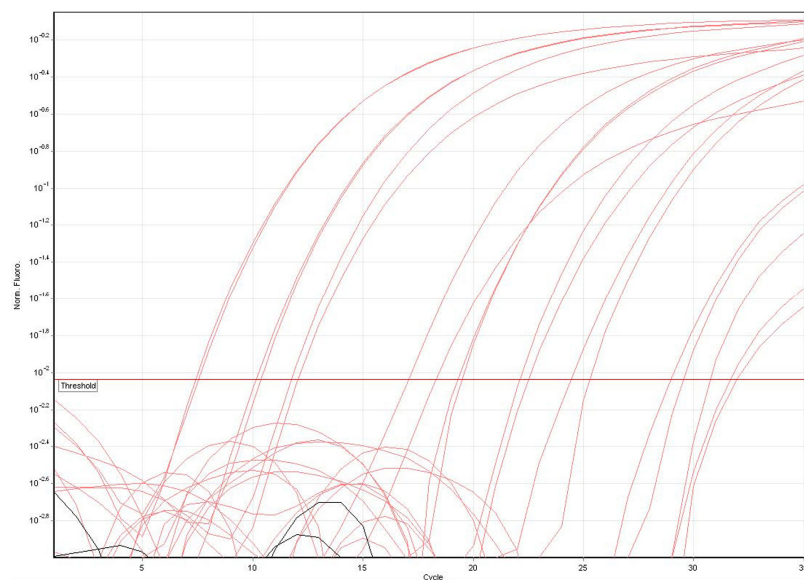


Figure 3. *B. parapertussis* range of detection and standard curve ranging from 1×10^{10} copies to 1×10^2 copies.

Table 2. Results by subtype

	RSV	<i>B. pertussis</i> +	<i>B. parapertussis</i> +
RSV A +	75 (16%)	1 (0.2%)	2 (0.4%)
RSV B +	52 (11%)	1 (0.2%)	1 (0.2%)
RSV A -	404 (84%)	1 (0.2%)	2 (0.4%)
RSV B -	434 (89%)		

genes, which encodes a phosphoprotein for each strain. The detection threshold for RSV-A and RSV-B was 100 copies per reaction.

Because we were not allowed to use patient identifiers, we used total population characteristics for all RSV samples submitted to the clinical laboratory during the study period to estimate population characteristics.

Statistical Analysis: Prevalence and exact confidence intervals were calculated for *B. pertussis*, *B. parapertussis* and RSV and co-infection. We tested the hypotheses that the proportion of cases screening positive for *B. pertussis* and *B. parapertussis* would exceed 2% with exact methods using STATA 9.2 (Stata Corp LP, College Station, TX).

RESULTS

The median age of the population sampled was 4.8 months (IQR 8.2); 356/648 (54.9%) were male. Six hundred and forty-eight specimens were received at the clinical lab. Most of these, 571 (88.4%) came from the ED, 49 (7.6%) from the pediatric and family practice clinics, and 26 (4.0%) from inpatients. Four hundred and eighty-nine (75.5%) of these submitted clinical samples were captured for additional testing. There was insufficient material to complete testing for one *B. pertussis*, 10 RSV subtype A, and three RSV subtype B assays. *Bordetella pertussis* was detected in 3/488 (0.61%) (95% CI 0.13% to 1.79%) while *B. parapertussis* was detected in 5/489 (1.02%) (95% CI 0.33% to 2.37%). Dual infection with *B. pertussis* and RSV was found in two cases. Dual infection with *B. parapertussis* and RSV occurred in three cases. The results are detailed in Table 2. One case of *B. bronchiseptica* ('kennel cough') was also found. RSV A was found in 75/479 (16%) samples and RSV B in 52/486 (11%). Local prevalence trends for the preceding 10 years are shown in Figure 4 to place the results in context.

DISCUSSION

Despite the rising local and national trend of reported cases of *B. pertussis* at the time of the study, we found very low rates of co-infection of *B. pertussis* and RSV. Our study does provide some evidence for clinicians who discount concerns about missing pertussis during RSV season, even during the current apparent resurgence of pertussis.

Our results are consistent with that of a retrospective

study of 166 children less than six-years-old hospitalized with lower respiratory tract symptoms in which a 0.6% (95% CI: 0.0% to 3.3%) prevalence was noted.¹ However, Siberry et al.¹⁸ was performed at a low point in local disease prevalence (five cases in all age groups in Baltimore) and drawn from an older population than ours; the studies are therefore not directly comparable. Our results contrast with those of Korppi and Hiltunin,¹⁷ which found an 8% prevalence in infants hospitalized with bronchiolitis who were less than six months old.

Our methodology allowed us to obtain large numbers of clinical specimens but not to obtain individual level clinical information on the patients from whom they were obtained, much less the clinicians' reasons for ordering the test. This is a feature of studies using so called "discarded clinical specimens." Such studies' results are most useful inferentially when they find a high or a low prevalence of an infectious agent. They are useful in triangulation, i.e. supporting a particular hypothesis by three different research methodologies. Triangulation is warranted when investigating conditions with high morbidity (such as causes of apnea) or when existing research provides conflicting answers.

This study suggests that when the community prevalence of pertussis is low, (our reported prevalence was 7.9/100,000 during the study period), and a 2% miss rate is acceptable to clinicians, routine screening for pertussis in patients suspected of having RSV is unwarranted. This is in contrast to studies that have looked at selected subgroups and found *B. pertussis* to be common. The definitiveness of this statement will depend on the results being replicated over a number of seasons.

LIMITATIONS

Local test-ordering patterns may help explain our low RSV rate. The sampling would have included patients

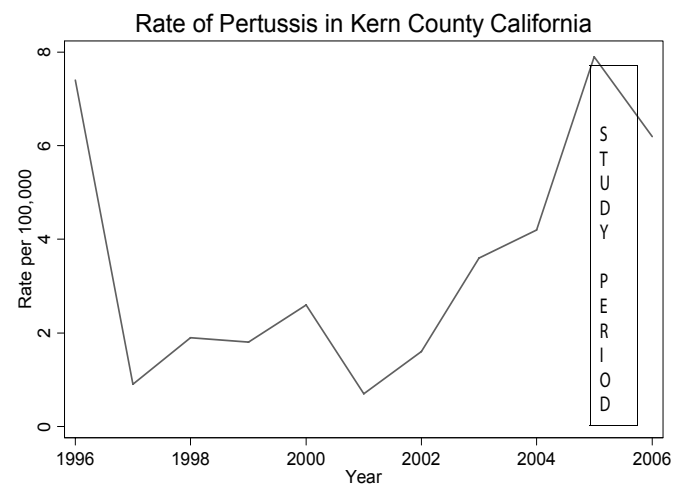


Figure 4. The context in which our sampling was performed. The box represents the sampling period.

screened for RSV early and late in the bronchiolitis season when other viruses are generally responsible. In-patient screening samples required for cohorting would also have been included in this analysis. RSV testing may have been obtained in cases of fever without source, or as part of apparent-life threatening event (ALTE) evaluations. False-negative PCR results for RSV are also a likely potential reason. This may in part be due to dilution of virus inherent in the collection of nasal aspirates. Initial specimen collection used unpreserved saline in clinical test tubes. RNA is less stable than DNA. During the uncontrolled phase (specimen going from patient to clinical laboratory) and despite refrigeration prior to aliquoting into DNA-ase-free containers, some RNA degradation could have occurred. Nasal aspirates have been shown to be markedly inferior to universal transport medium for room temperature (UTM-RT) based collection methods for PCR-based testing for respiratory viruses in general and RSV in particular.^{21,22} While this may artificially lower our detection of RSV it is less likely to have such an effect on *Bordetella spp.* Detection of bacterial DNA is much less susceptible to the collection method used (unpublished data). All of these factors may account for the difference between RSV detection in this and another contemporaneous study in our institution where a clinical diagnosis of bronchiolitis was the entry criterion and in which RSV prevalence was 55%.²³

The use of nasal washings rather than nasopharyngeal swabs (NPS) could potentially have decreased the yield of pertussis. While only NPS are considered acceptable for culture, the sensitivity of PCR mitigates against the potential loss of sensitivity by our methods.

Mistaken attribution of respiratory illness to *B. pertussis* has been described due to poor PCR assay specificity but high sensitivity.²⁴ We feel this is unlikely to be the case in our study based on the specificity testing results for our assay and the actual study results obtained.

We have not addressed here the broader epidemiology of those clinical presentations that typically prompt RSV testing. The clinical role for testing for these other agents is currently evolving. We could not control the age of patients whose samples were included; however, based on the age profile of the patients whose samples were submitted, our results should be considered applicable only to infants and toddlers.

We included only samples from a single clinical laboratory serving a single county system of clinics and hospital sites. Although this system serves a mixed urban, suburban and rural population, our patients tend to be drawn from lower socio-economic groups. This would tend to increase the prevalence of infectious diseases in general and may overestimate the prevalence that would be found in a more affluent setting. On the other hand vaccine refusal, which increases vulnerability to pertussis, tends to occur in higher socio-economic groups. Local DPT vaccination and

pertussis patterns mirror those of the rest of the state and country as a whole.

Our results must be viewed in the context of the overall prevalence of pertussis within the community. A higher prevalence could have changed our results, although the degree of change necessary to change the outcome is speculative. Nonetheless, although low by historical standards, prevalence was at a 10-year high during our study period. This is based on Department of Public Health reported case, data that inevitably carry their own limitations. Although the number of cases reported is low, it is likely an underestimate. It is estimated that between one-third and one in 20 of actual pertussis cases are reported.^{19,20} Nonetheless, the increasing trend of reported cases matches that for state and national reporting.

Our geographic locale is large. It is also a relatively sparsely populated, poor, and a medically underserved county with very low per person medical care expenditure,²⁵ again suggesting that the actual number of pertussis cases is probably higher than that reported.

Future reports will address these limitations by enrolling only ED patients and addressing clinical variables and outcomes over a number of seasons.

CONCLUSION

In a combined outpatient clinic, ED, and inpatient population, the prevalence of *B. pertussis* in nasal washings submitted for RSV antigen screening was less than 2%. The prevalence of parapertussis may have been higher than 2%. RSV with *B. pertussis* and RSV with *B. parapertussis* coinfection did occur.

Address for Correspondence: Paul Walsh, MB, BCh, BAO, MSc, Department of Emergency Medicine, Kern Medical Center Bakersfield, CA and David Geffen School of Medicine, University of California Los Angeles. Email yousentwhohome@gmail.com

REFERENCES

1. Crowcroft NS, Booy R, Harrison T et al. Severe and unrecognised: pertussis in UK infants. *Arch Dis Child.* 2003; 88:802-806.
2. Sotomayor J, Weiner LB, McMillan JA. Inaccurate diagnosis in infants with pertussis. An eight-year experience. *Am J Dis Child.* 1985; 139:724-727.
3. Smith C, Vyas H. Early infantile pertussis; increasingly prevalent and potentially fatal. *Eur J Pediatr.* 2000; 159:898-900.
4. Heininger U, Klich K, Stehr K, Cherry JD. Clinical Findings in *Bordetella pertussis* Infections: Results of a Prospective Multicenter Surveillance Study. *Pediatrics.* 1997; 100:e10.
5. Church NR, Anas NG, Hall CB, Brooks JG. Respiratory syncytial virus-related apnea in infants. Demographics and outcome. *Am J Dis Child.* 1984; 138:247-250.

6. Wang EE, Law BJ, Stephens D. Pediatric Investigators Collaborative Network on Infections in Canada (PICNIC) prospective study of risk factors and outcomes in patients hospitalized with respiratory syncytial viral lower respiratory tract infection. *J Pediatr*. 1995; 126:212-219.
7. Foo AL, Chay OM, Hiew J, Tan CK, Lim KW, Cheng HK. Severe bronchiolitis in children. *J Singapore Paediatr Soc*. 1991; 33:165-168.
8. Van Steensel-Moll HA, Van d, V, Bos AP, Rothbarth PH, Neijens HJ. Respiratory syncytial virus infections in children admitted to the intensive care unit. *Pediatrics*. 1989; 44:583-588.
9. Neilson KA, Yunis EJ. Demonstration of respiratory syncytial virus in an autopsy series. *Pediatr Pathol*. 1990; 10:491-502.
10. Fleischer G. Infectious Disease Emergencies. In: Fleischer G, Ludwig S, eds. *Textbook of pediatric emergency medicine*. 4 ed. Philadelphia: Lippincott Williams; 1999:725-93.
11. Ruddy R, Gittleman M. Pediatric Emergencies. *Pediatric Emergencies*. Atlanta: American Health Consultants; 2001:135-50.
12. Willwerth BM, Harper MB, Greenes DS. Identifying hospitalized infants who have bronchiolitis and are at high risk for apnea. *Ann Emerg Med*. 2006; 48:441-447.
13. Arms JL, Ortega HW, Finkelstein M, Reid SR. Apnea complicating RSV infection in infancy. *Ann Emerg Med*. 2006; 48:95.
14. Respiratory and Enteric Virus National Trends. Centers for Disease Control. 2005. Available at: <http://www.cdc.gov/ncidod/dvrd/revb/nrevss/trends.htm>. Accessed October 20, 2007.
15. Cherry JD. The Epidemiology of Pertussis: A Comparison of the Epidemiology of the Disease Pertussis With the Epidemiology of Bordetella pertussis Infection. *Pediatrics*. 2005; 115:1422-1427.
16. Hardwick TH, Cassidy P, Wayent R, Bisgard K, Sanden G. Changes in Predominance and Diversity of Genomic Subtypes of *Bordetella pertussis* Isolated in the United States, 1935–1999. *Emerging Infectious Diseases*. 2002; 8:44-49.
17. Korppi M, Hiltunen J. Pertussis is common in nonvaccinated infants hospitalized for respiratory syncytial virus infection. *Pediatr Infect Dis J*. 2007; 26:316-318.
18. Sibery GK, Paquette NR, Ross TL, Perl TM, Valsamakis A. Low Prevalence of Pertussis Among Children Admitted With Respiratory Symptoms During Respiratory Syncytial Virus Season. *Infect Control Hosp Epidemiol*. 2006; 27:95-97.
19. Sutter RW, Cochi SL. Pertussis hospitalizations and mortality in the United States 1985-1988. Evaluation of the completeness of national reporting. *JAMA*. 1992; 267:386-391.
20. Ward JL, Cherry JD, Chang SJ. Efficacy of an acellular pertussis vaccine among adolescents and adults. *N Engl J Med*. 2005; 353:1555-1563.
21. Walsh P, Overmeyer C, Pham K, Michaelson S, Pusavat J, Tran T, Mordechai E, Adelson M. Comparison of RSV-A RNA Recovery by PCR Using Different Methods of Specimen Preservation. *Annals of emergency medicine*. 2007; 50[3]: S38.
22. Walsh P, Lim-Overmeyer C, Gofman L et al. Pediatric Respiratory Infectious Disease Analysis: UTM-RT versus Flocked Swab Nasal Collections. *Western Journal of Emergency Medicine*. 2008; 9:72.
23. Walsh P, Caldwell J, McQuillan KK, Friese S, Robbins D, Rothenberg SJ. Comparison of nebulized epinephrine to albuterol in bronchiolitis. *Acad Emerg Med*. 2008; 15:305-313.
24. Centers for Disease Control and Prevention. Outbreaks of Respiratory Illness Mistakenly Attributed to Pertussis --- New Hampshire, Massachusetts, and Tennessee, 2004-2006. *MMWR*. 2007; 56:837-842.
25. Starr G. Comparison of funding to county hospitals. Blue Ribbon Task Force Recommendations. (Kern Medical Center). February 26, 1997. Kern County Board of Supervisors, Blue Ribbon Task Force.

Self-Reported Emergency Medicine Residency Applicant Attitudes Towards a Procedural Cadaver Laboratory Curriculum

Lance Hoffman, MD
Michael C. Wadman, MD
Tammi Erickson
Robert Muelleman, MD

University of Nebraska Medical Center

Supervising Section Editor: Michael Epter, DO

Submission history: Submitted March 5, 2008; Revision Received April 10, 2008; Accepted May 4, 2008.

Reprints available through open access at www.westjem.org

Objective: Residency applicants consider a variety of factors when ranking emergency medicine (EM) programs for their NRMP match list. A human cadaver emergency procedure lab curriculum is uncommon. We hypothesized that the presence this curriculum would positively impact the ranking of an EM residency program.

Methods: The EM residency at Nebraska Medical Center is an urban, university-based program with a PGY I-III format. Residency applicants during the interview for a position in the PGY I class of 2006 were surveyed by three weekly electronic mailings. The survey was distributed in March 2006 after the final NRMP match results were released. The survey explored learner preferences and methodological commonality of models of emergency procedural training, as well as the impact of a procedural cadaver lab curriculum on residency ranking. ANOVA of ranks was used to compare responses to ranking questions.

Results: Of the 73 potential subjects, 54 (74%) completed the survey. Respondents ranked methods of procedural instruction from 1 (most preferred or most common technique) to 4 (least preferred or least common technique). Response averages and 95% confidence intervals for the preferred means of learning a new procedure are as follows: textbook (3.69; 3.51-3.87), mannequin (2.83; 2.64-3.02), human cadaver (1.93; 1.72-2.14), and living patient (1.56; 1.33-1.79). Response averages for the commonality of means used to teach a new procedure are as follows: human cadaver (3.63; 3.46-3.80), mannequin (2.70; 2.50-2.90), living patient (2.09; 1.85-2.33), and textbook (1.57; 1.32-1.82). When asked if the University of Nebraska Medical Center residency ranked higher in the individual's match list because of its procedural cadaver lab, 14.8% strongly disagreed, 14.8% disagreed, 40.7% were neutral, 14.8% agreed, and 14.8% strongly agreed.

Conclusion: We conclude that, although cadaveric procedural training is viewed by senior medical student learners as a desirable means of learning a procedure, its use is uncommon during medical school, and its presence as part of a residency curriculum does not influence ranking of the residency program.

[WestJEM. 2008;9:141-145.]

INTRODUCTION

Simulation has been defined as a person, device, or set of conditions that present evaluation problems authentically in an environment where the student responds to them as they would under natural circumstances. Simulation training

has been advocated as a way to provide the necessary skills-practice opportunities to become a competent physician, while affording the safest environment to patients being managed by physicians in training.¹⁻⁷ Simulation training may use a variety of diagrams, improvised models made of everyday

items, manufactured mannequins of varying fidelity, human and non-human cadaver models, patient actors, or emerging computer-based virtual reality systems.¹⁻¹² The majority of medical procedural skills are acquired during medical school and residency training.¹³ Many of the procedural-training simulations involve medium to high fidelity mannequins made of plastics and rubbers that model human anatomy and tissue haptics. Despite advances, these materials do not imitate the feel of human tissue to a high degree. Consequently, this emergency medicine residency instituted a lightly-embalmed cadaver laboratory through collaboration with the Department of Anatomy as part of its didactic training for the resident physicians. The lab experience allows them to realistically practice high risk procedures in a low stakes environment and is used to test whether they are competent to perform a given procedure safely on an actual patient.

While a procedural cadaver laboratory experience has been documented in the literature for medical students being taught emergency procedures in an elective course, and for internal medicine and surgery residents, it is not widely described as a learning resource among emergency medicine residents.¹⁴⁻¹⁸ Although animal laboratories have been used for teaching procedural skills to emergency medicine residents,¹⁹⁻²⁰ we were able to find only a single study describing the use of a cadaver model in teaching emergency procedures to them.²¹ Because of this, we hypothesized that the presence of such a laboratory experience at this program will make it more likely that the applicants who interviewed for a residency position will rank this program higher than they otherwise might have if such a learning experience were not offered as part of the curriculum. Our survey's objectives were twofold: (1) to determine whether the presence of a cadaveric procedural training curriculum influenced the self-reported desirability of an emergency medicine residency when ranking a program via the National Residency Match Program (NRMP); and (2) to gain information about the most preferred or most common procedural training technique and learning preferences for various forms of procedural simulation, including a description of specific procedures being taught in medical school using various instructional techniques.

METHODS

Study Design and Population

The emergency medicine residency program at Nebraska Medical Center is an urban, university-based program with a PGY I-III format, caring for approximately 45,000 patients each year. Six resident physicians comprise each class. This study was reviewed by the Institutional Review Board and approved under the status of exempt from written consent.

Prospective subjects were identified by reviewing the list of NRMP interviewees at the emergency medicine residency program. The investigators all hold administrative roles within

the residency and have professional access to the names of the potential subjects. Each of the investigators personally interviewed each of the potential subjects between November 2005 and February 2006 as applicants for the incoming class. A description of the cadaveric procedure laboratory as part of the repeating, mandatory educational curriculum was specifically discussed with each of the applicants during their residency interview visit. It includes using lightly embalmed cadavers four times a year to instruct residents in the successful completion of many emergency procedures, including tracheal intubation and rescue airway techniques, cricothyrotomy, central venous access, intra-osseous vascular access, tube thoracostomy, and thoracotomy. The study subjects did not have an opportunity to attend a cadaveric procedure laboratory during their visit as the laboratory was not held during an interview day.

Survey Content and Administration

The survey explored learner preferences and methodological commonality of models of emergency procedural training, as well as the impact of a procedural cadaver laboratory curriculum on residency ranking. Other features of residency programs that might affect residency ranking were not included in the survey. In addition, subject demographic information was not collected in order to maintain respondent confidentiality. Prospective subjects were contacted for recruitment into the study by a series of up to three electronic mailings, each spaced approximately a week apart. A commercially available web-based survey company, SurveyMonkey (www.SurveyMonkey.com), was utilized. The names, as well as all other identifying information, of the respondents were not accessible to the investigators. The surveys were mailed after the NRMP match was completed (March 16, 2006) so that the potential subjects were free of coercion. A cover letter explaining the objectives was electronically mailed to the potential subjects at the same time as the survey. In this way, each potential respondent could decide, free of coercion and based on the content of the cover letter, whether to complete the survey, not respond in any way, or remove his or her name from subsequent mailings.

Data Analysis

Means and 95% confidence intervals were calculated for each response involving ranking of foils. Analysis of variance of ranks was used to compare responses to ranking questions.

RESULTS

Of the 73 potential subjects, 54 (74%) completed the survey, and none of the questions were skipped by the respondents.

Respondents ranked methods of procedural instruction 1-4, with 1 representing the most preferred or most common procedural training technique and 4 representing the least

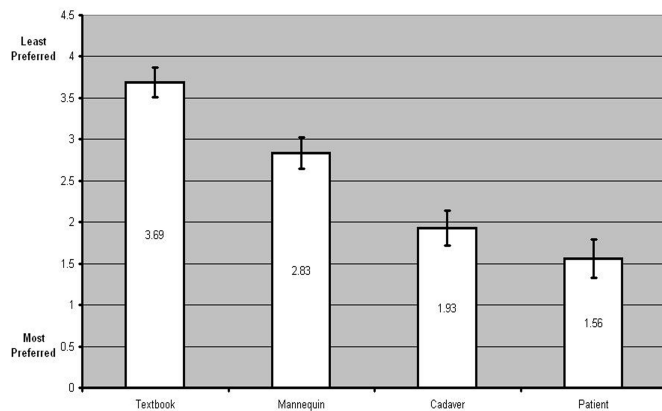


Figure 1. Learner preferences for procedural teaching modality. Response means and 95% confidence intervals are indicated.

preferred or least common technique. Response averages and 95% confidence intervals for the preferred means of learning a new procedure were as follows: textbook (3.69; least preferred; 3.51-3.87), mannequin (2.83; 2.64-3.02), human cadaver (1.93; 1.72-2.14), and living patient (1.56; most preferred; 1.33-1.79) (Figure 1). Statistical significance was reached comparing textbook v. mannequin and mannequin v. human cadaver, but not comparing human cadaver v. living patient. Response averages for the commonality of means used to teach a new procedure were as follows: human cadaver (3.63; least common; 3.46-3.80), mannequin (2.70; 2.50-2.90), living patient (2.09; 1.85-2.33), and textbook (1.57; most common; 1.32-1.82) (Figure 2). Statistical significance was reached comparing commonality of cadaver v. mannequin and mannequin v. living patient, but not comparing living patient to textbook.

The subjects were asked to indicate all of the means that were to be utilized at the program to which they matched in order to teach emergency procedural skills. The respondents indicated that 52/54 (96%) used the written descriptions and diagrams of textbooks, 51/54 (94%) used a high stakes biological model of a living patient, 47/54 (87%) used a high fidelity, non-biological model like a mannequin simulator, and 34/54 (63%) used a low stakes biological model of a cadaver.

The respondents indicated the procedures they had performed during medical school using a non-biological material model, such as a mannequin. The results were as follows: direct laryngoscopy with tracheal intubation (48/54=89%), any non-surgical rescue airway technique (33/54=61%), central venous access (19/54=35%), cricothyrotomy (14/54=26%), lumbar puncture (10/54=19%), intra-osseous vascular access (9/54=17%), and tube thoracostomy (4/54=7%). None of the respondents had performed a pericardiocentesis or thoracotomy using a non-biological material model. Two of the respondents (2/54=4%) stated that they had not performed any of these procedures using a non-biological material model during medical school.

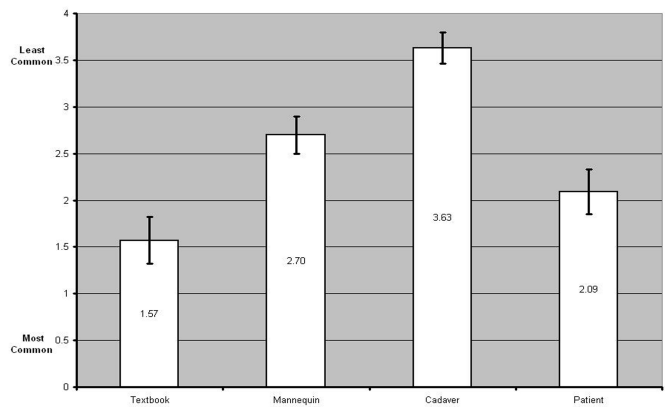


Figure 2. Prevalence of procedural teaching modality. Response means and 95% confidence intervals are indicated.

The respondents also indicated those procedures that they had performed during medical school using a biological material model, such as a cadaver model. Cricothyrotomy was the most commonly performed procedure using a cadaver model with 12/54 (22%) of the respondents answering in the affirmative. The other procedures performed by medical students using a cadaver model were as follows: tube thoracostomy (11/54=20%), direct laryngoscopy with tracheal intubation (8/54=15%), central venous access (7/54=13%), intra-osseous vascular access and pericardiocentesis (6/54=11%), any non-surgical rescue airway technique and thoracotomy (5/54=9%), and lumbar puncture (3/54=6%). Thirty-two of the 54 respondents (59%) stated that they had not performed any of these procedures using a biological material model.

Finally, when asked about the commonality of means of acquiring procedural skills, respondents indicated the procedures they had performed on a living patient while in medical school. These results were as follows: lumbar puncture (46/54=85%), direct laryngoscopy with tracheal intubation (43/54=80%), central venous access (36/54=67%), any non-surgical rescue airway technique (19/54=35%), tube thoracostomy (11/54=20%), intra-osseous vascular access (4/54=7%), thoracotomy (2/54=4%), and pericardiocentesis (1/54=2%). None of the respondents had performed a cricothyrotomy on a living patient. One of the respondents (1/54=2%) stated that he had not performed any of these procedures on a living patient as a medical student.

When asked if this emergency medicine residency ranked higher in the individual's match list because of its procedural cadaver lab, 8/54 (14.8%) strongly disagreed, 8/54 (14.8%) disagreed, 22/54 (40.7%) were neutral, 8/54 (14.8%) agreed, and 8/54 (14.8%) strongly agreed.

DISCUSSION

Previous investigations have shown that adult learners acquire and retain information best if they are actively

involved in the learning process. It is clear that medical schools across the United States have adopted teaching techniques that capitalize on these findings, many of which now use problem-based learning and small group discussion formats, rather than traditional lecture to teach information during the first two years of medical school. In more recent years, patient simulation has been gaining momentum as an effective means of learning new information and evaluating a learner's proficiency, especially for tasks that require behaviorally- or algorithmically-based information. For example, Objective Structured Clinical Examinations (OSCEs) using simulated patients are utilized by many United States medical schools as clinical evaluation tools. The American Association of Medical Colleges (AAMC) now requires that an OSCE in the form of the Clinical Skills test be taken by medical students applying for residency positions.

Simulation affords the opportunity to practice an invasive procedure with known complication rates in a safe setting where a living patient is not at risk of incurring a procedural complication because of an inexperienced operator. One challenge faced by simulation is improving tissue haptics to better approach that of living patients while making such experiences affordable for widespread use. Using lightly embalmed cadavers to gain proficiency in a procedure addresses this simulation limitation. The tissue haptics of a lightly embalmed cadaver are more realistic than those of a mannequin made of plastics and rubber. The cost of the lightly embalmed cadavers can be divided among the individuals using the cadavers for instruction. For example, the Department of Orthopedic Surgery may share the cost of cadavers with the Department of Emergency Medicine so that the limbs may be used for teaching orthopedic surgical procedures and the torso for teaching the placement of a chest tube or open thoracotomy. This is the strategy that is employed at this institution in teaching emergency medicine residents invasive procedures.

Indeed, our data support that a lightly embalmed human cadaver procedural laboratory is viewed by senior medical students as being a preferred means of learning a new procedure on par with learning the procedure on a living patient and over that of using a high fidelity mannequin or textbook instruction. Further, use of the lightly embalmed cadaver affords the added benefits of not placing a patient at risk because of an inexperienced operator while simultaneously approaching the tissue haptics of a living patient. Martin et al¹⁵ demonstrated significantly fewer pneumothoraces as a complication of central venous cannulation after implementing a procedural didactic session coupled with skill performance in a fresh cadaver model compared to those prior to this procedural skill session being completed. This survey's respondents indicate that a textbook containing written descriptions and illustrations is the most common means of teaching a medical procedure during medical school and residency while also being the least preferred means of learning

a medical procedure. These same respondents also indicate that a human cadaver laboratory is the least commonly utilized means of teaching a medical procedure in medical school and residency.

We postulated that the presence of a lightly embalmed procedure cadaver laboratory as part of the mandatory teaching curriculum of an emergency medicine residency would improve the desirability of matching at such a residency. However, the data from this study do not support this hypothesis given that an identical number of respondents stated that they "strongly disagreed" (8/54), "disagreed" (8/54), "agreed" (8/54), or "strongly agreed" (8/54), with 22/54 respondents answering "neutral," that the presence of such a procedural cadaver laboratory influenced them to rank the residency program higher than they would have if such a laboratory had not been part of the curriculum. While many factors influence the decision of where to rank a given residency program for the NRMP match, among these respondents the presence of such a laboratory curriculum does not appear to be an influential factor.

LIMITATIONS

The main limitation of this study is that it represents a survey of a limited sample of individuals who interviewed for a residency position at a single, young, Midwestern emergency medicine residency. At the time the interviews were conducted, this residency was young enough to have not yet graduated a class of physicians eligible for American Board of Emergency Medicine certification. In fact, the addition of six residents from this applicant pool of study subjects completed the first full complement of resident physicians in this PGY I-III program. This residency is located in a geographically removed area from other emergency medicine residency programs in a relatively smaller city (population of approximately 750,000 individuals including the suburban areas) compared to many other programs. These relatively individualized factors may have attracted applicants who placed a higher priority on residency characteristics not related to the specific educational curriculum. Consequently, different results might be obtained if this study were to be repeated at a more well-established program located in an area of higher population density, or located in a different geographical area of the country. A future direction of study might be comparing specific residency characteristics in order to determine those that are most influential to residency candidates when organizing their NRMP rank lists.

CONCLUSION

We conclude that, although cadaveric procedural training is viewed by senior medical student learners as a desirable means of learning a procedure, its use is uncommon during medical school, and its presence as part of an emergency medicine residency curriculum does not influence the applicants' NRMP match ranking of the residency program.

Address for Correspondence: Lance Hoffman, MD,
University of Nebraska Medical Center, Department of
Emergency Medicine, 981150 Nebraska Medical Center,
Omaha, NE 68198-1150, Email: LHoffman@unmc.edu.

REFERENCES

1. Steadman RH et al. Simulation-based training is superior to problem-based learning for the acquisition of critical assessment and management skills. *Crit Care Med.* 2006; 34: 252-253.
2. Wayne DB et al. Simulation-based training of internal medicine residents in advanced cardiac life support protocols: a randomized trial. *Teach Learn Med.* 2005; 17: 210-216.
3. Wanggren K et al. Teaching medical students gynaecological examination using professional patients-evaluation of students' skills and feelings. *Med Teach.* 2005; 27: 130-135.
4. Mueller MP et al. Teaching antiarrhythmic therapy and ECG in simulator-based interdisciplinary undergraduate medical education. *Br J Anaesth.* 2005; 95: 300-304.
5. DiGuilio E et al. Training with a computer-based simulator achieves basic manual skills required for upper endoscopy: a randomized controlled trial. *Gastrointest Endosc.* 2004; 60: 196-200.
6. Uribe JI et al. Learning curves, acquisition, and retention of skills trained with endoscopic sinus surgery simulator. *Am J Rhinol.* 2004; 18: 87-92.
7. Patil V et al. Training course in local anaesthesia of the airway and fiberoptic intubation using course delegates as subjects. *Br J Anaesth.* 2002; 89: 258-259.
8. Gallagher AG et al. Discriminative validity of the Minimally Invasive Surgical Trainer in Virtual Reality (MIST-VR) using criteria levels based on expert performance. *Surg Endosc.* 2004; 18: 660-665.
9. Gerson LB and Van Dam J. A prospective randomized trial comparing a virtual reality simulator to bedside teaching for training in sigmoidoscopy. *Endoscopy.* 2003; 35: 569-575.
10. Reznick MA, Rawn CL, and Krummel TM. Evaluation of the educational effectiveness of a virtual reality intravenous insertion simulator. *Acad Emerg Med.* 2002; 9: 1319-1325.
11. Manyak MJ et al. Virtual reality surgical simulation for lower urinary tract endoscopy and procedures. *J Endourol.* 2002; 16: 185-190.
12. Wilhelm DM et al. Assessment of basic endoscopic performance using a virtual reality simulator. *J Am Coll Surg.* 2002; 195: 675-681.
13. Crutcher RA et al. Where Canadian family physicians learn procedural skills. *Fam Med.* 2005; 37: 491-495.
14. Van der Vlugt TM and Harter PM. Teaching procedural skills to medical students: one institution's experience with an emergency procedures course. *Ann Emerg Med.* 2002; 40: 41-49.
15. Martin M et al. Training fourth-year medical students in critical invasive skills improves subsequent patient safety. *Am Surg.* 2003; 69: 437-440.
16. Tabas JA et al. A comprehensive, unembalmed cadaver-based course in advanced emergency procedures for medical students. *Acad Emerg Med.* 2005; 12: 782-785.
17. Oxentenko AS et al. A multidimensional workshop using human cadavers to teach bedside procedures. *Teach Learn Med.* 2003; 15: 127-130.
18. Martin M et al. Competency-based instruction in critical invasive skills improves both resident performance and patient safety. *Surgery.* 1998; 124: 313-317.
19. Homan CS et al. Evaluation of an emergency-procedure teaching laboratory for the development of proficiency in tube thoracostomy. *Acad Emerg Med.* 1994; 1: 382-387.
20. Custalow CB et al. Emergency department resuscitative procedures: animal laboratory training improves procedural competency and speed. *Acad Emerg Med.* 2002; 9: 575-586.
21. Proano L et al. Evaluation of a teaching laboratory using a cadaver model for tube thoracostomy. *J Emerg Med.* 2002; 23: 89-95.

Tinnitus as a Measure of Salicylate Toxicity in the Overdose Setting

Scott R. Samlan, MD*
Matthew T. Jordan, MD*
Shu B. Chan, MD, MS*
Michael S. Wahl, MD†
Rachel L. Rubin, MD*

* Resurrection Medical Center, Emergency Medicine Residency Program
† Evanston Northwestern Healthcare, Illinois Poison Center

Supervising Section Editor: Brandon K. Wills, DO, MS

Submission history: Submitted November 20, 2007; Revision Received March 10, 2008; Accepted March 14, 2008.

Reprints available through open access at www.westjem.org

Introduction: The development of tinnitus and/or hearing loss (THL) in patients receiving chronic salicylate therapy has been demonstrated. However, to date, little scientific data validates this relationship in the large single overdose setting.

Objective: To correlate salicylate levels in patients with the subjective complaint of THL, following an acute salicylate overdose.

Methods: A retrospective chart review of cases of acute salicylate toxicity and THL reported to the Illinois Poison Control Center (IPC) from 2001-2002 was performed. Data abstracted included age, gender, ingestion time, salicylate levels, and arterial blood gases.

Results: Ninety-nine cases of THL were reviewed and analyzed with mean age of 23.7 years (SD: 10.9), 30.3% male, and 82.2% intentional overdoses. The average dose ingested was 20.0 grams (SD:20.2) and the mean time from ingestion to medical care was 12.4 hours (SD: 11.1). The mean initial ASA level was 48.3 mg/dl (SD: 16.4) with 86.9% having initial level \geq 30mg/dl and 40.4% \geq 50 mg/dl. 85.9% of cases presented to the hospital with their ASA level at or past peak. The mean pH was 7.45, pO₂ = 108, pCO₂ = 28.0, and HCO₃ = 19.9.

Conclusion: In this limited study, 85.9% of patients presenting with tinnitus and/or hearing loss following a single salicylate ingestion had initial salicylate levels at or past their peak and 86.9% were in the toxic range.

[WestJEM. 2008;9:146-149.]

INTRODUCTION

Salicylates, aspirin in particular, have been used for their analgesic and anti-inflammatory properties for many years.¹ Unfortunately, toxicity from salicylates remains a common occurrence in the emergency department (ED).²⁻⁴ Moreover, it is well known that the development of tinnitus is associated with salicylate use, and its onset has been heralded as an early marker of toxicity.⁵⁻⁷ This subjective complaint is classically described as a constant ringing, or roaring sound, perceived by the patient. Other

presentations may include a subjective hearing loss or muffled hearing.⁸

Numerous reports have demonstrated the development of tinnitus and/or hearing loss (THL) in patients receiving chronic salicylate therapy.⁹⁻¹⁴ Anecdotally, its development has also been documented in the overdose setting.¹⁵ However, to date, little scientific data validates this relationship between THL and salicylate levels in patients with a large single salicylate overdose, as seen the ED. The hypothesis of this study was that the subjective complaint of

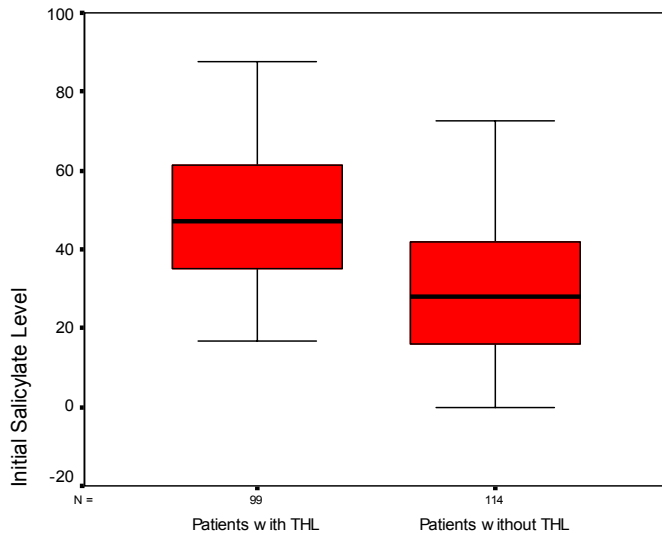


Figure 1. Boxplot of patients with and without tinnitus and/or hearing loss (THL) vs. initial aspirin levels.

tinnitus and/or hearing loss, in the setting of a large single salicylate overdose, correlates with an elevated salicylate level.

METHODS

A retrospective review of all cases of possible salicylate overdose reported to the Illinois Poison Center (IPC) from January 1, 2001 to December 31, 2002 was performed. Before the study's inception, approval was obtained from the institutional review board of Resurrection Medical Center. Potential cases were identified by a search of the computerized database (dotlab) of the poison center. The search criteria included "aspirin" or "salicylate" entered in the search field based on the American Association of Poison Control Centers (AAPCC) coding of salicylate products. The database search had no restrictions for age, sex, or exclusions based on co-ingestants. The information recorded in the database was provided by the healthcare workers involved in the care of the patient through telephone consultation with the poison center. This information was then entered into the database by the employee of the poison center.

The study setting included all emergency departments, intensive care units, and urgent care centers in the state of Illinois who contacted the poison center regarding salicylate toxicity management. Treatment and management decisions were made by the healthcare provider and poison center specialist involved in each individual case.

The raw data abstracted from call records were reviewed individually for age, gender, timing from ingestion to presentation for treatment, salicylate levels, bicarbonate levels, pH level, pCO₂ level, pO₂ level, and presence or absence of THL. All names were de-identified and deleted from the

database before data abstraction. These data elements were entered onto an Excel spreadsheet for analysis. An additional author to ensure entry accuracy reviewed approximately 10% of the collected data.

Since this was a retrospective chart review, the amount of detail provided in each chart varied and was not consistent for all charts. The time of ingestion to presentation for medical treatment was determined based on history taken by the healthcare provider and rounded off to the nearest half-hour. The presence of THL was also determined by the healthcare provider who queried this information during the initial contact with the patient. This measurement was performed utilizing a simple yes or no format. THL was defined by the poison center staff and medical provider as the subjective complaint of either ringing/roaring in the ears, "muffled" hearing, or the perception of a hearing loss by the patient.

All poison center charts containing the search criteria words were reviewed for inclusion into the study. Reasons for exclusion from the study were as follows: patients that did not present to a health care facility after making poison center contact, patients who did not ingest a salicylate as demonstrated by two consecutive serum levels below 1 mg/dl, or did not have documented salicylate levels, patients who failed to have the presence or absence of tinnitus queried by the health care worker, patients who were unable to confirm the presence or absence of THL either due to age, language barrier, or alteration in consciousness.

Data Analysis

Following data collection, descriptive statistics were performed using Statistical Package for Social Sciences for Windows Version 11.5 (Chicago, IL, 2004).

RESULTS

The database search yielded follow-up records on 592 cases of salicylate toxicity. In 335 cases the presence or absence of THL was not queried during the encounter or its documentation was not provided. In 44 cases, the patient was unable to confirm the presence of THL due to either age, language barrier, or an alteration in their level of consciousness. Only 99 of the remaining 213 cases had documented THL and were included in the study. Table 1 gives the demographic profile of all 213 documented cases, while Figure 1 is a box-plot of the initial acetylsalicylic acid (ASA) level between patients with documented THL and patients with documented no THL.

There were minimal differences between THL and no THL patients in terms of demographics or cause of ASA toxicity. Patients with documented tinnitus or hearing loss had higher initial and peak ASA levels. For patients with THL, the mean initial ASA level was 48.3 mg/dl (SD: 16.4) compared to 30.2 mg/dl (SD:18.4) for patients without THL. Also,

Table 1. Profile of patients with documented Tinnitus and/or hearing loss (THL) and no documented THL.

Profile	THL	No THL
Frequency	99	114
Gender (% male)	30.3%	27.2%
Age (Range)	23.7 (13-59)	24.9 (12-84)
Intentional overdoses	82.8%	91.2%
Initial acetylsalicylic acid (SD)	48.3 (16.4)	30.2 (18.4)
Hours from Ingestion (SD)	12.4 (11.1)	7.2 (8.8)
Reported Amount in Gm (SD)	20.0 (20.2)	17.9 (18.8)
	n= 42	n = 40
Peak acetylsalicylic acid (SD)	49.0 (15.5)	34.0 (17.3)

86.9% of THL patients had initial level ≥ 30 mg/dl and 40.4% had levels ≥ 50 mg/dl. On repeat testing, only 14.1% of cases had a further increase in ASA levels (mean increase = 8.5; SD: 4.6). Thus, 85.9% of cases presented to the hospital with their ASA level at or past peak. The average patient presented with compensated metabolic acidosis with mean pH = 7.45, mean pO₂ = 108, mean pCO₂ = 28.0, and mean HCO₃ = 19.9.

DISCUSSION

The development of tinnitus and/or hearing loss (THL) has been heralded as an early marker of salicylate toxicity.⁵⁻⁷ However, this principle has only been demonstrated in patients receiving escalating doses of salicylates as a therapeutic measure.^{12,13} In the overdose setting, THL has been cited as a symptom, but no current studies document its true relationship to the ingestion. In our limited study, we provide support for this concept in the large overdose setting. The data presented from our sample demonstrates that those patients who develop THL have a 86% chance of having a salicylate level in the toxic range, or ≥ 30 mg/dl.

The importance of this finding lies in the morbidity and mortality associated with salicylate toxicity. Intoxication can result in many deleterious effects on the human body, including death.^{16,17} As a result, the early recognition and treatment of salicylate toxicity remains paramount. In particular, prior studies have demonstrated that patients with critical serum salicylate levels have better outcomes when treatment is initiated earlier in their hospitalization.^{18,19}

In our study we utilized THL as a subjective marker of toxicity. Our results demonstrate that the patient complaint of THL, following a single salicylate ingestion, is suggestive of a toxic salicylate level. The mean initial salicylate level in the THL patients was 48.3 mg/dl. It was also noted that 85% of the patients presented to the hospital with their salicylate level at their peak. This finding coincides with a prior study that documented that the two most common findings in patients

with significant salicylate overdoses were the historical account of ingestion and the subjective complaint of tinnitus.²⁰

The treatment of salicylate toxicity includes bowel decontamination, fluid resuscitation, urinary alkalization, and extracorporeal elimination.^{8,21,22} Bowel decontamination in the form of activated charcoal is recommended for the majority of cases in salicylate overdose.^{2,21} However, beyond bowel decontamination, the consensus on other treatment modalities remains varied.^{2,3,23} In particular, recent publications have recommended the initiation of urinary alkalization when the serum salicylate level is found above 35 mg/dl.^{8,21}

As stated earlier, the measured serum salicylate level may take a significant time to obtain.²⁴ Consequently, flowcharts have been developed utilizing the patient's symptoms or other rapidly available clinical data to help guide treatment decisions.^{7,22} Most of these diagrams list THL as a marker of early toxicity typically presenting at a level of 25 mg/dl.^{5,8} The data from our study shows that 86.9% of patients had a level ≥ 30 mg/dl.

The principle that THL typically occurs around 25 mg/dl is based on previous studies over the past five decades.⁹⁻¹⁴ Mongan, et al¹³ demonstrated that THL was observed at an average salicylate concentration of 29.5 mg/dl in patients receiving chronic salicylate therapy. A study by Myers¹⁰ reported sensorineural hearing loss as the plasma salicylate level approached 30 mg/dl. As stated previously, both these studies utilized escalating doses of salicylates as a therapeutic, and both are distinctly different than the typical ED overdose setting. In our study, the mean salicylate level for those with THL was 48.3 mg/dl, much higher than this previously stated level of 25 mg/dl.

LIMITATIONS

Our study possesses several limitations. First, the self reporting of THL by the patient creates some potential problems. No accurate method was available to determine the validity of the patient's account. It is possible that some patients may have falsely denied or affirmed the presence of THL. Next, since this was a retrospective chart review, the amount of detail provided in each chart varied. Moreover, no ED or inpatient charts were obtained in this study to confirm poison center records. Lab values and patient histories were strictly based on the treating healthcare provider's phone testimony. In addition, the type of salicylate, and its form (i.e. enteric coated), were both sparsely documented making any analysis of these factors difficult. There was a limited population in our sample that had taken co-ingestants as well as prescribed medicine in conjunction with their salicylate ingestions. In reviewing the data, it seems however, that there were no known co-ingestants that would have contributed to tinnitus, but we acknowledge that this is a limitation in this study. The provided treatment modalities were also not always

identical. Their technique and timing unfortunately could have affected a patient's symptoms and lab values. The data also shows that the patients with tinnitus presented later in their course (12.4 versus 7.2 hours). But on the same note, 85.9% of the cases had the initial level above or at their peak salicylate level and the difference between initial and peak levels were very small in each group. Therefore we do not believe that the time presentation difference to be a large confounding variable. Finally, our study is limited in the fact that from a large initial study sample, only 213 had documentation of tinnitus or hearing loss. The lack of documentation of 335 patients prevents statistical testing between the groups. It may be that there were a number of patients without THL but with elevated salicylate levels. Thus, it is impossible to determine at what cutoff salicylate level we would expect THL but the evidence in this study does show that if THL occurs, we can expect an elevated salicylate level.

CONCLUSION

In the emergency department setting, it is essential to consider the diagnosis of salicylate ingestion when an overdose is suspected and a patient complains of THL. This limited study demonstrates that 85.6% of patients with single large salicylate ingestion combined with the complaint of tinnitus, and/or hearing loss, had initial salicylate level at or past their peak and 86.9% were in the toxic range.

Address for Correspondence: Shu Chan, MD, MS, Resurrection Medical Center, Emergency Medicine Residency Program, 7435 West Talcott Avenue, Chicago, Illinois 60631, E-mail: schan@reshealthcare.org.

REFERENCES

- Boettcher FA, Salvi RJ. Salicylate ototoxicity: review and synthesis. *Am J Otolaryngol.* 1991; 12:33-47.
- Yip L, Dart RC, Gabow PA. Concepts and controversies in salicylate toxicity. *Emerg Med Clin North Am.* 1994; 12:351-364.
- Proudfoot AT. Toxicity of salicylates. *Am J Med.* 1983; 75:99-103.
- Patel DK, Hesse A, Ogunbona A, Notarianni LJ, Bennett PN. Metabolism of aspirin after therapeutic and toxic doses. *Hum Exp Toxicol.* 1990; 9:131-136.
- Dargan PI, Wallace CI, Jones AL. An evidence based flowchart to guide the management of acute salicylate (aspirin) overdose. *Emerg Med J.* 2002; 19:206-209.
- Temple AR. Pathophysiology of aspirin overdosage toxicity, with implications for management. *Pediatrics.* 1978; 62:873-876.
- Krause DS, Wolf BA, Shaw LM. Acute aspirin overdose: mechanisms of toxicity. *Ther Drug Monit.* 1992; 14:441-451.
- Donovan JW, Akhtar J. Salicylates. In: Ford M, Delany K, Ling L, Erickson T, editors. *Clinical Toxicology.* Philadelphia: W.B. Sanders; 2001: 275-280.
- Jardini L, Findlay R, Burgi E, Hinderer K, Agarwal A. Auditory changes associated with moderate blood salicylate levels. *Rheumatol Rehabil.* 1978; 17:233-236.
- Myers EN, Bernstein JM, Fostiropoulos G. Salicylate ototoxicity: a clinical study. *N Engl J Med.* 1965; 273:587-90.
- Halla JT, Atchison SL, Hardin JG. Symptomatic salicylate ototoxicity: a useful indicator of serum salicylate concentration? *Ann Rheum Dis.* 1991; 50:682-684.
- Halla JT, Hardin JG. Salicylate ototoxicity in patients with rheumatoid arthritis: a controlled study. *Ann Rheum Dis.* 1988; 47:134-137.
- Mongan E, Kelly P, Nies K, Porter WW, Paulus HE. Tinnitus as an Indication of Therapeutic Serum Salicylate Levels. *JAMA.* 1973; 226:142-145.
- McCabe PA, Dey FL. The effect of aspirin upon auditory sensitivity. *Ann Otol Rhinol Laryngol.* 1965; 74:312-325.
- Beveridge GW, Forshall W, Munro JF, Owen JA, Weston IA. Acute Salicylate Poisoning in Adults. *Lancet.* 1964; 41:1406-1409.
- Strom J, Thisted B, Krantz T, Bredgaard SM. Self-poisoning treated in an ICU: drug pattern, acute mortality and short-term survival. *Acta Anaesthesiol Scand.* 1986; 30:148-153.
- Chapman BJ, Proudfoot AT. Adult salicylate poisoning: deaths and outcome in patients with high plasma salicylate concentrations. *Q J Med.* 1989; 72:699-707.
- Thisted B, Krantz T, Stroom J, Sorensen MB. Acute salicylate self-poisoning in 177 consecutive patients treated in ICU. *Acta Anaesthesiol Scand.* 1987; 31:312-316.
- Anderson RJ, Potts DE, Gabow PA, Rumack BH, Schrier RW. Unrecognized adult salicylate intoxication. *Ann Intern Med.* 1976; 85:745-748.
- Jammehdiabadi M, Tierney M. Impact of toxicology screens in the diagnosis of a suspected overdose: salicylates, tricyclic antidepressants, and benzodiazepines. *Vet Hum Toxicol.* 1991; 33:40-43.
- Flomenbaum NE. Salicylates. In: Goldfrank LR, Flomenbaum NE, Lewin NA, Howland MA, Hoffman RJ, Nelson LS, editors. *Goldfrank's Toxicologic Emergencies.* New York: McGraw-Hill; 2002: 507-518.
- Notarianni L. A reassessment of the treatment of salicylate poisoning. *Drug Saf.* 1992; 7:292-303.
- Prescott LF, Balali-Mood M, Critchley JA, Johnstone AF, Proudfoot AT. Diuresis or urinary alkalinisation for salicylate poisoning? *Br Med J (Clin Res Ed).* 1982; 285:1383-1386.
- Song W, Dou C. One-step immunoassay for acetaminophen and salicylate in serum, plasma, and whole blood. *J Anal Toxicol.* 2003; 27:366-371.

Comparison of Methanol Exposure Routes Reported to Texas Poison Control Centers

Melissa Givens, MD, MPH
Kristine Kalbfleisch, MD, MS
Scott Bryson, MD

Carl R. Darnall Army Medical Center

Supervising Section Editor: Jeffrey R. Suchard, MD

Submission history: Submitted February 20, 2008; Revision Received April 4, 2008; Accepted April 7, 2008.

Reprints available through open access at www.westjem.org

Objective: Methanol poisoning by ingestion is well represented in current emergency medicine literature. Much less described, however, is poisoning via intentional inhalation of methanol-containing products such as carburetor cleaner. This study intends to explore the exposure routes and treatment patterns of methanol cases reported to Texas Poison Centers.

Methods: All cases of methanol exposures from January 2003 to May 2005 were collected from the Texas Poison Center Network database "Toxicall." Inclusion criteria were 1) methanol as primary exposure, and 2) documented route of exposure. Exclusion criteria were unknown, dermal, and eye exposures. Data was extracted from documented calls to Texas Poison Centers and analyzed using descriptive statistics.

Results: A total of 203 cases were collected from 6 regional Poison Centers. Eighty seven cases had inhalation as the route of exposure, while 81 were methanol ingestions. Carburetor cleaner was responsible for nearly all the inhalational cases (79/87) while ingestions involved mostly windshield washer fluid (39/81) and carburetor cleaner (20/81). Seventy-eight percent of the inhalational exposures were intentional while most of the ingestions were accidental (49/75) and suicidal (18/75). An anion gap was documented in 31 of the inhalational cases and in 10 of the ingestions. Dialysis, use of fomepizole, and vision loss were documented for both types of exposure. Fifty-six percent of the inhalational group was admitted compared to 46% of the ingestion group.

Conclusion: We propose that the results obtained from our review show inhalational exposure involving methanol (e.g., "huffing") represents a significant source of toxicity in the studied population. This is in contrast to previous literature that proposed inhalational toxicity was rare and aggressive treatment usually not necessary in cases of inhalation of methanol-containing carburetor cleaners. [WestJEM. 2008;9:150-153.]

INTRODUCTION

When speaking of methanol exposures, most emergency physicians have experience with ingestions of methanol-containing products. However, inhalational abuse of carburetor cleaner and other volatile products containing methanol represents a non-trivial source of methanol exposure not commonly described in emergency medicine textbooks. Methanol is a colorless, volatile alcohol commonly used commercially in industrial solvents and products such as windshield washer fluid, glass cleaners, antifreeze, carburetor

cleaners, canned solid fuels (e.g., Sterno®), and small engine fuels. It is a common source of both intentional and accidental poisoning because of its widespread availability. Methanol is oxidized to formaldehyde by alcohol dehydrogenase (ADH) and then to formic acid by aldehyde dehydrogenase. Formic acid is primarily responsible for most of the serious sequelae observed in methanol toxicity, including metabolic acidosis and visual disturbances.^{1,2} Methanol poisoning by ingestion commonly presents with gastrointestinal complaints due to mucosal irritation including nausea, vomiting, and abdominal

pain. All exposure routes lead to central nervous system (CNS) depression, confusion and ataxia. The syndrome can progress to development of an uncompensated anion gap metabolic acidosis, brain lesions, and visual impairment, which can range from blurred vision to visual field deficits to total blindness.

Importance of the Topic

Epidemic poisonings from methanol exposure are sporadic. In 2005, of the 807 cases of methanol poisoning reported to American poison centers, 80% were unintentional, 4% had major complications and six resulted in fatalities.³ Treatment delay is associated with increased morbidity, making early recognition of clinical and laboratory clues crucial. Intentional inhalation of volatiles, including methanol-containing products, is increasing in prevalence, especially among adolescents.⁴⁻⁷ Long-term inhalant abuse is associated with violence, major depression, polysubstance abuse, and suicidality.^{8,9} This has far-reaching implications to society in general and to the emergency department (ED) as we treat significant toxicity in this growing demographic.

Goals of the Investigation

Poisoning by ingestion of methanol-containing products is well represented in the current body of medical literature; however, there is a paucity of literature dealing with their inhalation. Information is limited to a handful of small studies and case reports with mixed conclusions. Some report that methanol toxicity by inhalation or ingestion is equally dangerous, while others assert that inhalation does not result in serious toxicity.¹⁰⁻¹⁴ The goals of this investigation are to contribute evidence that toxicity from inhalation of methanol-containing products is both more common and more dangerous than previously thought and to explore the exposure routes and treatment patterns of methanol cases reported to Texas Poison Centers.

METHODS

A retrospective chart review was performed using the Texas Poison Control Center Network database, "Toxcall"®. All cases of methanol exposures from January 2003 to May 2005 were collected. Inclusion criteria were 1) methanol exposures, and 2) documented route of exposure. Exclusion criteria were unknown, dermal, and eye exposures. Data were extracted using a standardized data collection instrument by two independent investigators and reviewed for discrepancies. If a discrepancy occurred, the original chart was reviewed by both investigators to correct the discrepancy. Data were extracted from documented calls to Texas Poison Centers and analyzed using descriptive statistics. Database was managed and analyzed using Microsoft Office Excel® 2007.

Important endpoints included the type of product/exposure source (e.g., carburetor cleaner vs. windshield wiper

fluid), categorization of route of exposure (e.g., inhalation vs. ingestion), age of the exposed, and whether the exposure was intentional abuse (e.g., abused for intoxication purposes), accidental, or for a suicidal intent. The endpoints of toxicity included methanol level (if measured), the presence of an anion-gap acidosis (anion gap >12mEq/L), visual disturbances (to include any change in vision documented in the chart), the need for ethanol, fomepizole treatment and/or hemodialysis, and final ED disposition (e.g. need for admission). If endpoints were not documented in the chart then data were reported as "unknown."

RESULTS

A total of 203 cases were collected from six regional poison centers. Eight unknown exposures, five dermal, and 22 eye exposures were excluded. Eighty-seven of the remaining cases were inhalational and 81 were ingestions. Carburetor cleaner was responsible for nearly all the inhalational cases (79/87). The remaining inhalational cases were unknown products (6) or windshield washer fluid (2). Ingestions involved mostly windshield washer fluid (39/81) and carburetor cleaner (20/81), with 11 remaining cases from miscellaneous sources such as Sterno® and brake fluid and 11 cases from unknown products.

The mean age of the ingestion group was 19 (range <1 to 70 years, with 12 patients under the age of six), while the mean age of the inhalation group was 28 (range 2-62 years with one patient under the age of six). Seventy-one percent (62/87) of the inhalational group was male, and 88% (71/81) of the ingestion group was male. Eighty percent of known cases (66/83) of the inhalational exposures were intentional abuse cases, while 8 were suicidal, 9 accidental, and 4 unknown. Most of the known cases of ingestions were accidental (49/75) and suicidal (18/75) with the remaining attributable to intentional abuse (8) and unknown (6).

The mean pH for the inhalation group was 7.24 (95% CI 7.19 - 7.29) and for the ingestion group was 7.33 (95% CI 7.27-7.37). Arterial or venous source of pH was not explicitly queried. Six patients in the inhalational group suffered visual loss and two in the ingestion group. Other endpoints are described in Table 1.

DISCUSSION

Although awareness of acute toxicity from methanol ingestion has been highlighted for years in Emergency Medicine literature, this is one of the few studies looking at inhalational exposures. In 2002 Barceloux et al.¹ reported that "almost all cases of acute methanol toxicity result from ingestion, though rarely cases of poisoning have followed inhalation or dermal absorption." The results obtained from our retrospective chart review demonstrate an increased trend of inhalational exposure involving methanol (i.e., "huffing"), and the critical clinical manifestations of methanol toxicity

Table 1. Comparison of endpoints in methanol exposures

	Inhalation			Ingestion		
	Yes	No	Unknown	Yes	No	Unknown
Anion gap	31	24	32	10	28	43
Methanol level <20mg/dl	7	7	63	3	6	72
4-methylpyrazole or ethanol	27	32	28	21	36	24
Dialysis	9	48	20	5	45	31
Admission	44	35	8	31	36	14

and evidence of poisonings from an inhalation-exposure route.

A case report published in *Annals of Emergency Medicine* in 1990 described the “unusual presentation of solvent abuse” in a 17-year-old male who appeared intoxicated after inhaling a rag soaked with carburetor cleaner containing methanol, toluene and methylene chloride.¹⁴ Additionally, in 1993 Frenia and Schauben¹⁰ reported in *Annals of Emergency Medicine* of seven cases of intentional inhalation abuse of carburetor cleaner, containing methanol, toluene, methylene chloride and propane. These cases experienced CNS depression, visual disturbance (one case), death (one case), and acidosis requiring treatment with leucovorin and/or folate, ethanol infusions, reversal of acidosis and even hemodialysis (three cases). The results of our retrospective chart review build upon these reports to provide an even larger number of patient study subjects inhaling methanol (87) with 80% of those cases defined as “intentional abuse” exposures. These cases were compared to ingestion exposure subjects (81) defined as accidental (60%) or suicidal (22%). Our study too showed that the most popular methanol-containing product involved in inhalational cases was carburetor cleaner (97%).

Carburetor cleaner contains other compounds that may contribute to toxicity. Common ingredients that often comprise more than 50% of the listed ingredients in carburetor cleaners include toluene, xylene, acetone, and naphtha; however, this list is not all-inclusive. Mixtures of various hydrocarbons make up smaller percentages of ingredients. Methanol content varies based on manufacturer. The hydrocarbon component of carburetor cleaners can be expected to contribute to toxicity. Inhalation of hydrocarbons may cause pulmonary toxicity, CNS symptoms (sedation, agitation, seizures, ataxia) and cardiac dysrhythmias. Toluene deserves specific mention as it can cause renal tubular acidosis with chronic exposure. Toluene may also contribute a small amount to an anion gap metabolic acidosis since it is metabolized to hippuric acid.

A recent retrospective poison center chart review summarized 22 cases and reported that “significant toxicity following inhalation of methanol containing carburetor cleaners was rare with symptoms improving without aggressive care (e.g., dialysis, ADH blockade).”¹² A more

recent prospective observational study on seven patients after inhalant abuse of methanol-containing hydrocarbon products preliminarily concluded that “inhalant abusers of methanol products may have significantly elevated methanol and formic acid levels, but are at low risk for methanol induced complications of visual dysfunction and refractory acidosis.”¹³

Our data appear to refute these conclusions. Many of our inhalation exposure patients developed an anion gap metabolic acidosis, needed dialysis or treatment with ethanol or fomepizole, developed visual loss and required admission to the hospital. The trends observed in our study should remind us to maintain our awareness and aggressiveness of treatment of the acutely intoxicated patient to prevent the potential significant toxic sequelae that can result from the inhalation of methanol-containing products.

LIMITATIONS

Several limitations exist in our study. It is performed on only the patient population reported through the poison control system and thus is vulnerable to the inherent limitations of poison center data. Data are collected by specialists in poison information (SPI) by phone calls from the facility where the patient is receiving care. Data may be collected from nurses or physicians and are dependent on the information offered by those involved in the care of the patients and the thoroughness of the query by the SPIs. Due to the limitations of data collection in these patients, we did not do any further statistical testing other than descriptive statistics. Poison center cases are limited to those voluntarily reported by the individual or health care facilities. There could be cases that are never diagnosed or reported, which could contribute to the underestimation of this exposure. In addition, the ingestion route of exposure may be overestimated from the large number of accidental cases reported and are a result of small amounts ingested, and therefore nontoxic. One major limitation of the study that may overestimate the toxicity of the inhalational exposures is the fact that the source products contain several co-ingestants/chemicals (e.g., methylene chloride, toluene, etc.) which may be contributing to the endpoints of toxicity.

CONCLUSION

Our results indicate that inhalational exposure involving methanol represents a significant source of toxicity in the studied population. These results contrast with previous literature that proposed inhalational methanol toxicity was rare and aggressive treatment usually not necessary in cases of inhalation of methanol containing carburetor cleaners.

Address for Correspondence: Melissa Givens, MD MPH, Department of Emergency Medicine, 36000 Darnal Loop, C.R. Darnall Army Medical Center, Fort Hood TX 76548. Email: Melissa.givens@us.army.mil.

REFERENCES

1. Barceloux DG, Bond GR, Krenzelok EP, Cooper H, Vale JA, American Academy of Clinical Toxicology practice guidelines on the treatment of methanol poisoning, *J Toxicol Clin Toxicol*. 2002;40(4):415-46.
2. Hantson PE, Acute methanol intoxication: physiopathology, prognosis and treatment, *Bull Mem Acad R Med Belg*. 2005;160(5-6):294-300.
3. Lai M, Klein-Schwartz, Rodgers G, et al. 2005 Annual report of The American Academy of Poison Control Centers national poisoning and exposure database. *Clin Toxicol* 2006;44(6-7):803-892.
4. Kavet R, Nauss KM, The toxicity of inhaled methanol vapors, *Crit Rev Toxicol*. 1990;21(1):21-50.
5. Wu LT, Ringwalt CL. Inhalant use and disorders among adults in the United States. *Drug Alcohol Depend*. 2006 Oct 15;85(1):1-11. *Epub* 2006 Apr 3.
6. Chepesiuk R., Resurgence of teen inhalant use. *Environ Health Perspect*. 2005 Dec;113(12):A808.
7. Neumark YD. The epidemiology of adolescent inhalant drug involvement. *Arch Pediatr Adolesc Med*. August 1, 1998, 152(8):781-6.
8. Fleschler MA, Tortolero SR, Baumler ER, Vernon SW, Weller NF, Lifetime inhalant use among alternative high school students in Texas: prevalence and characteristics of users. *Am J Drug Alcohol Abuse*. 2002;28(3):477-95.
9. Sakai JT, Hall SK, Mikulich-Gilbertson SK, Crowley TJ. Inhalant use, abuse, and dependence among adolescent patients: commonly comorbid problems, *J Am Acad Child Adolesc Psychiatry*. 2004 Sep;43(9):1080-8.
10. Frenia ML, Schauben JL, Methanol inhalation toxicity, *Ann Emerg Med*. 1993 Dec;22(12):1919-23.
11. Winnik L, Radomska M, Wos M, Pach D, The CNS damage due to methanol poisoning in the case of 23-year-old male patient addicted to organic solvents. *Przegl Lek*. 2005;62(6):511-3.
12. LoVecchio F, Sawyers B, Thole D, Beuler MC, Winchell J, Curry SC, Outcomes following abuse of methanol-containing carburetor cleaners. *Hum Exp Toxicol*, 2004 Oct;23(10):473-5.
13. Bebartá VS, Heard K, Dart RC, Inhalational abuse of methanol products: elevated methanol and formate levels without vision loss, *Am J Emerg Med*. 2006 Oct;24(6):725-8.
14. McCormick, MJ, Mogabgab E, Adams SL. Methanol poisoning as a result of inhalational solvent abuse. *Annals of Emergency Medicine*. 1990, June 19(6):639-42.

Fluoxetine Overdose-Induced Seizure

Jeffrey R. Suchard, MD

Department of Emergency Medicine, University of California, Irvine School of Medicine

Supervising Section Editor: Brandon K. Wills, DO, MS

Submission history: Submitted November 19, 2007; Revision Received January 20, 2008; Accepted January 24, 2008.

Reprints available through open access at www.westjem.org

A 37-year-old woman experienced a witnessed generalized seizure in the Emergency Department three hours after ingesting approximately 1400 mg of fluoxetine in a suicide attempt. Although the majority of fluoxetine ingestions are benign, seizures may occur after large intentional overdoses. [WestJEM. 2008;9:154-156.]

INTRODUCTION

Fluoxetine (Prozac®, Eli Lilly and Company, Indianapolis IN) is a selective serotonin reuptake inhibitor (SSRI) commonly used to treat depression and for other psychiatric indications. The majority of fluoxetine overdoses result in a benign clinical course. The largest published case series of fluoxetine overdoses found that the most common effects were tachycardia, drowsiness, tremor, nausea, and vomiting, and concluded that such overdoses typically are “minimally toxic”.¹ Despite this suggestion that only mild to moderate symptomatology is to be expected, seizures,²⁻⁸ cardiac conduction abnormalities,⁸ and even fatalities⁹ have been associated with fluoxetine ingestions, although most of these cases involve co-ingested drugs or other confounding factors. We report the case of a witnessed, generalized seizure occurring three hours after a fluoxetine overdose in an otherwise healthy young woman.

CASE REPORT

A 37-year-old woman with a history of bulimia nervosa and depression ingested approximately seventy 20 mg fluoxetine capsules and 4-5 cans of beer in a self-professed suicide attempt. Shortly thereafter, she telephoned a friend who activated the EMS system. The patient’s prescribed medications were fluoxetine 20 mg daily and buspirone 15 mg twice daily. The patient stated that she had taken her buspirone only as directed, with the last dose on the morning of the fluoxetine overdose, about six hours earlier. She specifically denied ingesting any additional buspirone or any other medications. She admitted to “purging” herself daily for the last week. Other than some orthopedic surgical procedures, she denied any other significant past medical history, including seizures.

The paramedics arrived approximately 90 minutes post-

ingestion and found the patient to be awake, alert, sitting up, and emotionally upset. The initial blood pressure was 142/ palp and cardiac monitoring showed a sinus tachycardia at 120/minute. In the emergency department (ED) her vital signs were: temperature 37.2°C, pulse 91/min, blood pressure 132/72 mmHg, respirations 20/min, O₂ saturation 99% on room air. The emergency physician noted the patient to be alert and oriented, but with slurred speech and slow verbal response time. The patient had a non-focal neurologic exam without tremor, rigidity, or hyperreflexia, and the remainder of the physical examination was without noted abnormalities. The patient was given 50 g of activated charcoal, and blood and urine samples were obtained for baseline values and for quantitative serum acetaminophen and salicylate measurements. Serum chemistries showed sodium 138 mmol/L, potassium 4.2 mmol/L, chloride 105 mmol/L, bicarbonate 23 mmol/L, BUN 11 mg/dL, creatinine 0.6 mg/dL, glucose 81 mg/dL, salicylate 3.9 mg/dL, acetaminophen <1µg/mL, and ethanol 48 mg/dL. A qualitative urine pregnancy test was negative. An electrocardiogram revealed a normal sinus rhythm of 97 beats per minute with normal intervals (QRS 88 msec, QTc 461 msec).

Approximately three hours after the ingestion, the patient cried out and then experienced a generalized tonic-clonic seizure lasting 30 seconds witnessed by the ED personnel. The seizure resolved spontaneously, and the patient had a post-ictal period lasting five minutes. The patient received an intravenous loading dose of phenobarbital (620 mg [10mg/kg]) and was then transported without incident to a regional toxicology referral center.

On arrival to the intensive care unit, the patient was somnolent but easily arousable. Vital signs were: temperature 36.9°C, pulse 82/min, blood pressure 112/78 mmHg, respirations 24/min. A repeat physical examination was

unremarkable including the neurologic exam. Additional laboratory data obtained upon admission included serum creatinine kinase (140 IU/L), calcium (8.8 mg/dL), and a comprehensive urine drug screen (which combines the enzyme-multiplied immunoassay technique, thin layer chromatography, and gas chromatography/mass spectroscopy to detect over 1500 drugs and metabolites) that showed the presence of only phenobarbital, fluoxetine, ethanol, and caffeine. The laboratory verified that buspirone can be detected by this analysis. Quantitative serum levels of fluoxetine, norfluoxetine, and buspirone were ordered on admission. The fluoxetine level six hours after the ingestion was 922 ng/mL (therapeutic = 50-480 ng/mL) and the norfluoxetine level was 379 ng/mL (therapeutic = 50-450 ng/mL). The quantitative buspirone level could not be determined due to laboratory handling error. The patient was observed overnight without any further seizure activity or other unusual events. The psychiatry consultation and liaison service evaluated the patient the following morning and arranged for outpatient therapy. The patient was then discharged home in stable condition.

DISCUSSION

Data from human and animal trials show fluoxetine to be generally safe and with few drug interactions.¹⁰⁻¹² Fluoxetine overdose typically results in a benign clinical course, with the most common symptoms being tachycardia, drowsiness, tremor, nausea, and vomiting, and has therefore been identified as “minimally toxic in doses up to 1,500 mg and with combined plasma levels [fluoxetine plus norfluoxetine] up to 1390 ng/mL.”¹¹ With regard to potential neurotoxic effects, considerable evidence exists that fluoxetine has an anticonvulsant effect at therapeutic doses in humans and animal models.¹³ Antidepressants may display both anticonvulsant and pro-convulsant properties, with the most important determining factor being the dose.¹⁴ In a study of five different SSRIs taken in overdose, fluoxetine had the lowest incidence of inducing seizures (1%, vs. 2% for sertraline, paroxetine, and citalopram, and 4% for fluvoxamine).¹⁵ Not surprisingly then, there are few reports of seizures associated with fluoxetine in the medical literature. Many of these reports are confounded by co-ingestants and/or underlying brain disease.^{2-5,8} Only a few cases of seizure after isolated fluoxetine overdose in normal subjects have been reported,^{6,7} and there is also a case occurring after escalation of therapeutic dosing up to 60 mg/day.¹⁶

Evidence in the patient presented here for an acute fluoxetine overdose is supported not only by history, but also from the relatively high ratio of the parent substance compared to norfluoxetine, its *N*-desmethylated metabolite. The seizure does not appear to be related to serotonin syndrome, because the patient did not exhibit autonomic instability, muscular rigidity, or abnormal mental status (excluding the seizure

itself and a brief post-ictal period) as typically occur in that disorder.¹⁷ The patient also consumed some ethanol during her suicide attempt, but it is very unlikely that ethanol contributed to her seizure. Firstly, she was only an occasional ethanol consumer without a history of dependence or prior episodes of withdrawal; in such a case, the presence of ethanol would, if anything, act as an anticonvulsant. Secondly, she did not exhibit signs of autonomic instability (e.g., diaphoresis, hypertension) or tremor consistent with ethanol withdrawal. She was initially tachycardic, but this is also commonly found in cases of significant SSRI overdose.

Although acute fluoxetine overdose is believed to cause the seizure in this patient, buspirone might potentiate fluoxetine's neurotoxicity. A seizure has been reported in a patient receiving therapeutic doses of fluoxetine and buspirone for obsessive-compulsive disorder.¹⁸ In contrast, a case series of 11 patients on fluoxetine and buspirone does not describe any seizures or other neurotoxicity.¹⁹ Caffeine overdoses have also been reported to cause seizures,²⁰ and caffeine was detected in our patient's urine. When questioned about this finding, she reported that she consumed caffeine-containing beverages (e.g. tea, soft drinks), but denied excessive use. Although a serum caffeine level is not available for this patient, she was not exhibiting symptoms of caffeine intoxication such as tremor, agitation, hyperglycemia or hypokalemia.²⁰ She did have a mild tachycardia initially, but this spontaneously resolved prior to the seizure. In order for caffeine to be responsible for this seizure, one would expect the patient to exhibit prominent symptoms of caffeine toxicity. Thus, although the patient's ingestion was not a completely isolated fluoxetine overdose, since other xenobiotics were detected, there is no evidence that any other drug contributed significantly to her seizure.

CONCLUSION

We report the clinical course of a patient who had a witnessed seizure following an acute fluoxetine overdose. While the medical literature strongly suggests that most fluoxetine overdoses are benign, emergency physicians need to remain cognizant that intentional, high-dose fluoxetine ingestions may induce seizures.

Acknowledgements

David Connor, MD (Phoenix, AZ) for referring the case and assistance with early drafts.

Address for Correspondence: Jeffrey R. Suchard, MD, Department of Emergency Medicine, University of California, Irvine Medical Center, 101 The City Drive, Route 128, Orange, CA 92868. Email: jsuchard@uci.edu

REFERENCES

1. Borys DJ, Setzer SC, Ling LJ, et al. Acute fluoxetine overdose: a report of 234 cases. *Am J Emerg Medicine*. 1992; 10:115-120.
2. Weber JJ: Seizure activity associated with fluoxetine therapy. *Clin Pharmacol*. 1989; 8:296-298.
3. Hargrave R, Martinez D, Bernstein AJ. Fluoxetine-induced seizures. *Psychosomatics*. 1992; 33:236-237.
4. Prasher VP. Seizures associated with fluoxetine therapy. *Seizure*. 1993; 2:315-317.
5. Ware MR, Stewart RB. Seizures associated with fluoxetine therapy. *DICP Ann Pharmacother*. 1989; 23:428.
6. Braitberg G, Curry SC. Seizure after isolated fluoxetine overdose. *Ann Emerg Med*. 1995; 26:234-237.
7. Gross R, Dannon PN, Lepkifker E, et al. Generalized seizures caused by fluoxetine overdose. *Am J Emerg Medicine*. 1998; 16:328-329.
8. Graudins A, Vossler C, Wang R. Fluoxetine-induced cardiotoxicity with response to bicarbonate therapy. *Am J Emerg Medicine*. 1997; 15:501-503.
9. Lai MW, Klein-Schwartz W, Rodgers GC, et al. 2005 annual report of the american association of poison control centers' national poisoning and exposure database. *Clin Toxicol*. 2006; 44:803-932.
10. Stark P, Fuller RW, Wong DT. The pharmacologic profile of fluoxetine. *J Clin Psychiatry*. 1985; 46:7-13.
11. Lemberger L, Bergstrom RF, Wolen RL, et al. Fluoxetine: clinical pharmacology and physiologic disposition. *J Clin Psychiat*. 1985; 46:14-19.
12. Cooper GL. The safety of fluoxetine - an update. *Br J Psych*. 1988; 153(Suppl 3):77-86.
13. Jobe PC, Browning RA. The serotonergic and noradrenergic effects of antidepressant drugs are anticonvulsant, not proconvulsant. *Epilepsy Behav*. 2005; 7:602-619.
14. Pisani R, Spina E, Oteri G. Antidepressant drugs and seizure susceptibility: from in vitro data to clinical practice. *Epilepsia*. 1999; 40(Suppl 10):S48-S56.
15. Isbister GK, Bowe SJ, Dawo A, Whyte IM. Relative toxicity of selective serotonin reuptake inhibitors (SSRIs) in overdose. *Clin Toxicol*. 2004; 42:277-285.
16. Oke A, Adhiyaman V, Aziz K, Ross A. Dose-dependent seizure activity associated with fluoxetine therapy. *Q J Med*. 2001; 94:113-114.
17. Mills KC. Serotonin syndrome: a clinical update. *Crit Care Clinics*. 1997; 13:763-783.
18. Grady TA, Pigott TA, L'Heureux F, et al. Seizure associated with fluoxetine and adjuvant buspirone therapy. *J Clin Psychopharmacol*. 1992; 12:70-71.
19. Markovitz PJ, Stagno SJ, Calabrese JR. Buspirone augmentation of fluoxetine in obsessive-compulsive disorder. *Am J Psychiatry*. 1990; 147:798-800.
20. Dietrich AM, Mortensen ME. Presentation and management of an acute caffeine overdose. *Ped Emerg Care*. 1990; 6:296-298.

Survival after an Intentional Ingestion of Crushed *Abrus* Seeds

Lisa Reedman, MD*
Richard D. Shih, MD*†
Oliver Hung, MD*

* Morristown Memorial Hospital
† New Jersey Medical School

Supervising Section Editor: Brandon K. Wills, DO, MS

Submission history: Submitted December 23, 2007; Revision Received March 6, 2008; Accepted March 6, 2008.

Reprints available through open access at www.westjem.org

Abrus precatorius seeds contain one of the most potent toxins known to man. However, because of the seed's outer hard coat the vast majority of ingestions cause only mild symptoms and typically results in complete recovery. If the seeds are crushed and then ingested, more serious toxicity, including death, can occur.

We present a case of a man who survived an intentional ingestion of crushed *Abrus* seeds after he was treated with aggressive gastric decontamination and supportive care.

[WestJEM. 2008;9:157-159.]

INTRODUCTION

Abrus precatorius is a vine native to India and other tropical and subtropical areas of the world. Since introduction to Florida and the Caribbean, it is now commonly found throughout these areas and in the southern United States.¹ It is known by a variety of names, including jequirty bean, rosary pea, prayer bead, crab's eye, and love bean.² The vine has pods with oval seeds and a hard glossy shell. The seeds vary in color, from red, black, orange or white with black and white centers (Figure 1). While all parts of the plant are toxic, the highest concentrations are found in the seeds.¹ Due to their appearance, the seeds are often used for jewelry, beadwork and ornaments.³

Abrus contains a potent toxin, abrin, along with smaller concentrations of glycyrrhizin, aric acid, and N-methyltryptophal. Abrin is a toxalbumin, composed of two subunits, an A and B chain covalently linked through a disulfide bridge. The toxin's structure is similar to insulin, ricin, botulinum, cholera and diphtheria toxins.¹ The B chain facilitates binding to cell surface receptors, allowing the entry of the A chain into cells.⁴ The A chain acts on the 60s ribosome to inhibit EF-1 and EF-2, preventing protein synthesis and leading to cell death.^{1,5}

Oral ingestion of whole seeds often does not produce serious illness since the shell protects the toxin from digestion.⁷ Poisonings are more likely in the fall because new crops with immature seeds have a softer shell.⁶ Conversely, older seeds have brittle shells and also a greater potential for toxin exposure to the gastrointestinal tract. Chewing, grinding, or drilling the seed disrupts the hard shell, exposing more abrin to the GI tract. Most



Figure 1. *Abrus precatorius*.

effects of this toxin are limited to local GI symptoms, as digestive enzymes destroy the toxin and limit systemic absorption.

The majority of cases involving abrin are ingestions with whole seeds. These cases often cause few or mild symptoms because the shell remains intact and there is limited toxin exposure. Only one previous case documents the intentional ingestion of *Abrus* seeds that have been ground or crushed. Our case also involves a suicide attempt involving ingestion of ground *Abrus* seeds. However, unlike the previous case that led to death,

in this case with aggressive GI decontamination and supportive care, the patient survived with no sequelae.

CASE REPORT

A 27-year-old man presented to the emergency department (ED) with multiple episodes of vomiting and liquid black stools. The patient revealed that he had intentionally ingested the powder of 10 ground *Abrus precatorius* seeds in a suicide attempt approximately 30 minutes prior to ED arrival. The patient had ground up the seeds and mixed the powder with water to make a liquid slurry. After he ingested this liquid slurry, he decided that he did not want to go through with the suicide and self-administered 50 grams of activated charcoal orally just prior to presenting to the ED. Upon arrival, he was awake, alert, and oriented, and initial vital signs revealed a blood pressure of 140/100 mmHg, pulse of 130 beats/minute, respiratory rate of 12 breaths/minute, and a temperature of 99.4°F. Cardiac monitoring showed sinus tachycardia without ectopy or arrhythmias. On physical examination his head, neck, heart, lung, and neurological exams were normal. Abdominal exam revealed normal bowel sounds with diffuse mild tenderness to palpation. Rectal examination revealed black colored stools that were hemocult negative.

Intravenous fluid boluses and anti-emetics were administered. Despite the history of self-administered charcoal, activated charcoal (50 grams) were administered orally in the ED. Laboratory tests revealed: sodium of 143 mEq/L, potassium of 4.0 mEq/L, chloride of 100mEq/L, carbon dioxide of 26mEq/L, urea nitrogen of 16 mEq/L, creatinine of 1.0mg/dL and glucose of 98 mg/dL. Liver function tests and CBC were normal. Salicylate, acetaminophen, and ethanol levels were within normal limits, and urine drug screen was negative.

Upon re-evaluation, the patient continued to have diarrhea but had stopped vomiting. His tachycardia and abdominal tenderness also resolved during his ED evaluation. The patient was medically cleared after ~ 8 hours in the ED for psychiatric evaluation. One month follow-up phone call revealed no further sequelae from his ingestion.

DISCUSSION

Abrus precatorius is one of the most potent plant toxins known to man.⁶ The toxin, a protein, is poorly absorbed in the digestive tract and rarely, if ever, causes systemic toxicity via the oral ingestion. However, if injected parentally, it can enter the systemic circulation, where it has potent effects on protein synthesis, and even in small amounts can lead to fatalities.

The majority of poisonings involve children who ingest seeds, either from broken pieces of jewelry or off the native plant.¹⁰ Despite this, ingesting whole seeds produces few or mild symptoms because the shell insulates the toxin from absorption, and digestive enzymes in the gastrointestinal tract destroy the toxin. However, if the shell has been broken through chewing, drilling or grinding, increased amounts of the toxin are exposed

to the digestive system. Although poorly absorbed, the toxin can produce local GI symptoms with large exposures, such as in our case above.

Typical GI symptoms include nausea, vomiting, and diarrhea, resulting in more serious poisonings with severe dehydration and death.¹ Clotting time and platelet function appear to be unaffected by the toxin, although non-toxic agglutins are present in the seeds.⁴ There is no known toxicity level in humans, although there is a steep dose-lethality curve in animals.² In addition, animals given lethal doses of abrin did not show unusual or unique symptoms when compared to animals given non-lethal doses.⁹ Symptoms can sometimes be delayed for 1-3 days after ingestion, and the clinical course can last up to 10 days.^{2,6}

Due to the delay between ingestion and symptoms, immediate and aggressive treatment may be necessary. Gastric emptying techniques, including induced emesis, activated charcoal, gastric lavage and whole bowel irrigation, may be useful treatment modalities.⁶ There is no specific antidote for abrin poisoning, and treatment is mainly supportive with intravenous fluids and correction of electrolyte abnormalities.² In addition, stool and vomitus should not be discarded until the diagnosis is confirmed, as seed remnants may be present within these specimens.⁶

While poisonings with abrin are potentially life threatening, the majority of cases result in only mild symptoms. These patients require supportive care during the acute phase, and most recover without permanent sequelae. One case reported a four-year-old child who ingested four old and dry seeds. He was treated with induced emesis, activated charcoal, and cathartics. He fully recovered over three days and was discharged home without any sequelae. This case is typical of most abrin exposures, in that it involves ingestion of a few seeds resulting in mild GI symptoms and full recovery.¹⁰

Although severe sequelae can occur from oral ingestion of *Abrus* seeds, it is felt that the seed coating is protective and limits toxicity. Few cases involve the crushing or pulverizing of *Abrus* seeds, as in the case presented here. There is only one previously published similar case. This involves a 25-year-old male who put 20 *Abrus* seeds and graphite into a blender, and then drank most of this mixture in a suicide attempt. While he only drank a portion of this mixture, he developed severe nausea, vomiting, and diarrhea. He was brought to the ED four days after the ingestion, later developed cardiac arrhythmias, and died. The mechanism of death in this case is not entirely clear. The crushing of the seeds releases toxin from within the protective outer hard coat of the seeds. This mechanism likely increases the severity of toxicity and decreases the time to onset of symptoms. The classical delay in onset of toxicity associated with the ingestion of intact whole seeds likely does not occur when the seeds have been crushed.

Our case also involves the crushing of the *Abrus* seeds with oral ingestion in a suicide attempt. Exposure to abrin in this manner most likely increases the toxin exposure in the

gastrointestinal tract, as our patient presented with severe vomiting and diarrhea.

However, in our case the patient self-administered activated charcoal prior to ED arrival and received aggressive treatment for dehydration. Outcome in this case was good, in contrast to the previous similar case.

SUMMARY

The majority of cases of abrin poisoning involve ingestion of a small number of intact *Abrus* seeds resulting in mild symptoms and full recovery. However, abrin poisoning can be associated with severe GI toxicity, especially when the seeds are pulverized prior to ingestion as in the case presented here. It is believed that early treatment with intravenous fluids and supportive care will lead to good outcome.

Address for Correspondence: Richard D. Shih, MD, Morristown Memorial Hospital, 100 Madison Ave, Box #8, Department of Emergency Medicine, Morristown, NJ 07962. Email: Shih100@yahoo.com.

REFERENCES

1. Shih RD, Goldfrank LR. Plants. In: Goldfrank LR, Flomenbaum NE, Lewin NA, Weisman RS, Howland MA, Hoffman RS, eds. *Goldfrank's Toxicologic Emergencies*. 6th edition. East Norwalk, Connecticut: Appleton and Lange; 1998.
2. Arena JM. *Poisoning – toxicology, symptoms and treatments*. Springfield, IL: Charles C Thomas; 1986: 496.
3. Fernando C. Poisoning due to *Abrus precatorius* (jequirty bean). *Anaesthesia*. 2001; 56:1178-1180.
4. Olsnes S, Refsnes K, Pihl A. Mechanism of action of toxic lectins abrin and ricin. *Nature*. 1974; 249: 627-631.
5. Benson S, Olsnes S, Pihl A, Skorve J, Abraham AK. On the mechanism of protein-synthesis inhibition by abrin and ricin. *Eur J Biochem*. 1975; 59:573-580.
6. Davies JH. *Abrus precatorius* (rosary pea). The most common lethal plant poison. *J Fla Med Assoc*. 1978; 65:188-191.
7. Wambebe C, Amosun SL. Some neuromuscular effects of the crude extracts of the leaves of *Abrus precatorius*. *J Ethnopharmacol*. 1984; 11:49-58.
8. Routh BC, Lahiri SC. Some actions of the seeds of *Abrus precatorius*. *Bulletin of the Calcutta School of Tropical Medicine*. 1971; 19:46-47.
9. Fodstad O, Johannessen JV, Schjerven L, Pihl A. Toxicity of abrin and ricin in mice and dogs. *J Toxicol Environ Health*. 1979; 5:1073-1084.
10. Kinamore PA, Jaeger RW, de Castro FJ. *Abrus* and ricinus ingestion: management of three cases. *Clin Toxicol*. 1980; 17:401-405.
11. Bukhari AQ, Ahmad S, Qureshi S. Oral toxicology of *Abrus precatorius* linn. *J Pak Med Assoc*. 1976; 26:4-6.
12. Niyogi SK. The toxicology of *Abrus precatorius* linnaeus. *J Forensic Sci*. 1970; 15:529-536.
13. Olsnes S. Toxic and nontoxic lectins from *Abrus precatorius*. *Methods Enzymol*. 1978; 50:323-330.

Fatal Metformin Overdose Presenting with Progressive Hyperglycemia

Jeffrey R. Suchard, MD
Thomas A. Grotzky, MD

Department of Emergency Medicine, University of California, Irvine School of Medicine

Supervising Section Editor: Brandon K. Wills, DO, MS

Submission history: Submitted February 19, 2008; Revision Received April 1, 2008; Accepted May 4, 2008.

Reprints available through open access at www.westjem.org

A 29-year-old man with no history of diabetes ingested over 60 grams of metformin in a suicide attempt. He presented to the emergency department with acute renal insufficiency, severe lactic acidosis, and rapidly-progressive hyperglycemia. The patient's peak serum glucose level of 707 mg/dL is the highest yet reported in a case of metformin toxicity. Treatment included sodium bicarbonate infusion and hemodialysis, but the patient suffered several cardiac arrests with pulseless electrical activity and ultimately expired 25 hours after the ingestion.
[WestJEM. 2008;9:160-164.]

INTRODUCTION

Metformin is a biguanide anti-hyperglycemic drug which is the most commonly prescribed oral agent to treat diabetes mellitus.¹ Metformin-associated lactic acidosis (MALA) occurs infrequently with therapeutic use, occurring in 0.03 cases per 1000 patient-years.² This rate is substantially lower than the incidence of phenformin-associated lactic acidosis, which caused that biguanide drug to be withdrawn from the U.S. market in the late 1970s.²⁻⁴ In overdose situations, however, metformin is frequently associated with lactic acidosis.^{3,5-15}

Because the biguanide drugs do not enhance insulin release, as occurs with the sulfonylurea and meglitinide classes of diabetes medications, disorders of glucose homeostasis are rare with metformin. Although hypoglycemia associated with metformin has been reported, it is uncommon and often ascribed to concurrent use of other diabetes drugs.^{3,14,16} Hyperglycemia associated with metformin toxicity is even more rare, and has previously been ascribed to the patients' underlying diabetes or to acute pancreatitis.^{3,14,17,18} We report the case of an intentional overdose of metformin in a patient without diabetes which resulted in progressive hyperglycemia early in the clinical course and fatal lactic acidosis. This patient's peak serum glucose level of 707 mg/dL is the highest reported in a case of metformin toxicity.

CASE REPORT

A 29-year-old man ingested metformin in a suicide

attempt. The patient consumed the entire remaining contents of his father's prescription metformin bottle that originally contained 100 tablets of 850 mg each. The father stated that the bottle had contained at least three-quarters of its original contents, putting the ingested dose between 64 and 85 grams. The patient also consumed ethanol, but denied any other co-ingestants. The parents discovered the overdose around 6:30 a.m., about 5½ hours post-ingestion, when the patient began complaining of vomiting, diarrhea, thirst, abdominal pain and bilateral leg pain. Paramedics were called, who found the patient to be agitated with a fingerstick glucose level of 180 mg/dL.

The patient had a history of psychosis and depression, including prior suicide attempts by drug ingestion. He was not taking any prescribed medications, having discontinued olanzapine and sertraline several months earlier. The patient had no personal history of diabetes, despite the family history of type II diabetes in his father, who was taking no other anti-diabetic medications than metformin. The patient admitted to daily ethanol and tobacco use, but denied any current or past use of illicit drugs. He had no surgical history or known allergies.

Vital signs on arrival to the Emergency Department (ED) were temperature of 35.2°C (rectal), pulse of 113 beats/min, blood pressure of 129/59 mmHg, respirations at 28 breaths/min with 100% saturation via pulse oximetry on room air. The patient was awake and oriented x4, but agitated and slightly confused (GCS=14). Pupils were equal and reactive

Table 1. Initial Laboratory Evaluation (½ hour after ED arrival)

Sodium	136 mEq/L
Potassium	3.1 mEq/L
Chloride	105 mEq/L
Bicarbonate	9 mEq/L
BUN	2 mg/dL
Creatinine	2.1 mg/dL
Glucose	707 mg/dL
Calcium	9.4 mg/dL
Total Protein	7.6 g/dL
AST	60 IU/L
ALT	53 IU/L
Bilirubin	0.8 mg/dL
Acetaminophen	<5 µg/mL
Salicylate	<4 mg/dL
Lactate	>11.1 mmol/L
Ethanol	214 mg/dL
Osmolality	392 mOsm/kg
β-hydroxybutyrate	2.4 mg/dL

at 4mm and the oral mucous membranes were dry. Other than tachycardia, the heart and lung exams were unremarkable. The abdomen was mildly tender to palpation diffusely, but soft and without guarding or rebound. The patient had been incontinent of feces prior to arrival, but the stool was guaiac-negative and the patient had normal rectal tone. Electrocardiographic monitoring demonstrated a narrow-complex sinus tachycardia. A repeat fingerstick glucose upon arrival to the ED was 364 mg/dL. The patient became increasingly combative, attempting to bite and spit at the ED staff. He was placed in 4-point soft restraints, had a respiratory protection mask placed over his mouth, and was medicated with intravenous lorazepam 2 mg, dolasetron 12.5 mg, morphine sulfate 4 mg, and hydration with 0.9% saline. Within 45 minutes, the patient was no longer combative, and the physical restraints were removed.

Laboratory evaluation from blood samples obtained about one-half hour after ED arrival are shown in Table 1. Notably, the serum glucose level was 707 mg/dL associated with an elevated anion-gap metabolic acidosis. Arterial blood gas analysis showed: pH 7.10, pCO₂ 18.8 mmHg, and pO₂ 133.1 mmHg. The serum lactate level was above our clinical laboratory's reporting range (>11.1 mmol/L). Even accounting for ethanol's contribution to total osmolality, the calculated osmolal gap was highly elevated at >20 mOsm/kg. Given the presence of hyperglycemia with severe metabolic acidosis, the possibility of diabetic ketoacidosis was briefly considered; however, the serum β-hydroxybutyrate level was within normal limits (2.4 mg/

dL [0.5-3.0]) and ketonuria was absent. Urinalysis showed glycosuria and moderate hemoglobin, but no calcium oxalate crystalluria, blood cells, or signs of infection. A urine drug screen by enzyme-multiplied immunoassay technique was negative for opiates, cocaine, phencyclidine, amphetamines, propoxyphene, benzodiazepines, barbiturates, and methylenedioxymethamphetamine.

The patient was admitted to the medical intensive care unit service. Due to lack of ICU bed availability, the patient physically remained in the ED for several more hours. The nephrology service was initially contacted one hour after the patient's arrival for consideration of emergent hemodialysis.

Repeat laboratory analysis on serum samples obtained four hours after ED arrival showed decreasing bicarbonate (6 mEq/L) and continued severe hyperglycemia (672 mg/dL), despite continued IV hydration with 0.9% saline boluses. The nephrology fellow was at the bedside discussing the benefits and risks of dialysis, when the patient was witnessed to become unresponsive, vomit, aspirate, and go into pulseless electrical activity (PEA) with a heart rate of 29/min. The patient was resuscitated with chest compressions, endotracheal intubation, 2 mg IV epinephrine, 1 mg IV atropine, and 4 ampules (176 mEq) of 8.4% sodium bicarbonate, resulting in a heart rate of 110/min and blood pressure 121/88 mmHg. An intra-resuscitative ABG showed: pH 6.95, pCO₂ 55.7 mmHg, pO₂ 88.1 mmHg, and bicarbonate 12.4 mEq/L. The patient was started on a bicarbonate drip (150 mEq NaHCO₃ in 1L 5% dextrose at 200 cc/hr) and transferred to the ICU.

The patient developed non-sustained episodes of ventricular tachycardia, and then his blood pressure fell to 72/44 mmHg. A dopamine drip was started, but by the time the blood pressure stabilized the patient was found to be coagulopathic (INR 3.08; fibrinogen 113 mg/dL [156-400]; d-dimer >8.0 mcg/mL [<0.5]) and insertion of a hemodialysis catheter was delayed until the patient was treated with four units of fresh frozen plasma. The concurrent platelet count was 390 k/mm³ and the hematocrit was 47.7%. An echocardiogram showed mildly decreased left ventricular function, although serial cardiac enzymes showed progressive elevation of the CK-MB fraction and troponin I (up to 1.321 ng/mL [<0.03]). Hemodialysis was begun twelve hours after ED arrival. Three hours later, the patient again developed PEA and was resuscitated. Several more episodes of PEA recurred, and the patient expired about 19 hours after ED arrival. The last set of laboratory results, drawn five hours prior to death, showed a serum lactate level still >11.1 mEq/L, bicarbonate 12 mEq/L, and glucose 327 mg/dL. The hyperglycemia persisted despite treatment with an insulin drip (3 units regular insulin/hr) and the hemodialysis. No measurements of serum lipase or amylase were made during the patient's hospitalization.

DISCUSSION

Overdoses with metformin are relatively uncommon,

but may have serious consequences. In a five-year review of toxic exposures reported to U.S. poison control centers, only 4072 out of nearly 11 million exposures involved metformin, corresponding to less than one in 2500.³ There were a total of 9 deaths (0.2% of all metformin-related exposures), 32 cases with life-threatening signs or symptoms and/or residual disability (0.8%), and 187 cases with moderate clinical effects (4.6%). Cases of serious toxicity from metformin overdose are rare enough that single case reports and small case series continue to be published, which often describe extracorporeal methods of managing the consequent severe lactic acidosis.^{5-13,15}

Lactic acidosis may occur either with therapeutic metformin dosing or in overdose; 0.03 cases of lactic acidosis per 1000 patient-years occur with therapeutic dosing, and most of these cases occur among patients who have contraindications to metformin, such as renal insufficiency.² In overdose situations, lactic acidosis is seen much more commonly, although the exact incidence is unclear. Lactic acidosis was seen in 1.6% of metformin exposures reported to poison control centers, but only 10% of these exposures were due to intentional overdoses.³ In a review of poison control center inquiries from Germany, the incidence of metformin-associated lactic acidosis was 12.8% (14 of 109 calls); however, the incidence of attempted suicide was 56.9% in this group, suggesting that higher metformin doses were involved.¹¹ Case reports and series of severe toxicity from metformin are typically associated with severe lactic acidosis,^{5-13,15,17,18} and many of these are due to intentional overdose.^{5-10,12,13,15,18} Such publications, however, form an obviously biased group, and the actual incidence of lactic acidosis with intentional metformin overdose remains unknown.

The prognosis in cases of undifferentiated lactic acidosis is poor, with an expected case-fatality rate of 30-50%.^{2,19} In cases of metformin-associated lactic acidosis, the serum lactate level does not correlate with prognosis, even with lactate levels as high as 35.5 mmol/L.¹⁹ In cases where metformin levels have been measured, there is a poorer prognosis with lower metformin levels.¹⁹ This unusual finding suggests that patients developing lactic acidosis at lower metformin levels probably have concurrent underlying disease, placing them at higher risk both for accumulating lactate and for death.

The most striking feature of the case reported here, however, is the profound and progressive hyperglycemia, which has not been previously reported. Metformin generally does not cause significant alterations in serum glucose levels, even in overdose situations. Unlike the sulfonylurea and meglitinide classes of diabetes drugs, which stimulate insulin release from the pancreas and therefore lower the glucose level, the biguanide drugs have more complex effects on glucose homeostasis that tend to reduce hyperglycemia

without inducing hypoglycemia. These effects include: inhibition of hepatic gluconeogenesis (primarily through inhibiting hepatic lactate uptake), improving peripheral insulin sensitivity, inhibition of fatty acid oxidation, and possibly reducing intestinal glucose absorption.^{2,19} Metformin also increases production of lactate by the intestinal mucosa and suppresses pyruvate carboxylase, which impairs lactate clearance. Thus, lactic acidosis may occur in patients who have overdosed on metformin, or those with renal insufficiency, whose lactate clearance is already impaired.

Although metformin does not directly lower glucose levels, metformin-associated hypoglycemia has been reported, both with therapeutic dosing and with overdoses.^{3,14,16} Hypoglycemia was reported in 2.8% of metformin exposures reported to a poison control center.³ In many of these cases the patients were receiving concurrent treatment with sulfonylurea drugs, which likely contributed in causing hypoglycemia. Some instances of drug interactions with metformin have been associated with hypoglycemia (e.g. with enalapril, which increases insulin sensitivity)²⁰, although hypoglycemia with isolated metformin exposure is very rare.

Hyperglycemia associated with metformin overdose has occasionally been reported, but is even less common than hypoglycemia. In their review of 4072 metformin exposures, Spiller and Quadrani found only 18 cases associated with hyperglycemia (versus 112 with hypoglycemia), which they attributed as most likely due to the patients' underlying diabetes.³ Nearly 40% of cases with hyperglycemia (seven of 18) occurred in patients who died, and seven of the nine overall fatalities had hyperglycemia, suggesting that it may be associated with particularly severe metformin toxicity.

Hyperglycemia has been related to acute pancreatitis in a few cases of metformin toxicity from both intentional overdose^{11,18} and therapeutic dosing.¹⁷ In the previously published cases of pancreatitis with metformin toxicity, the degree of hyperglycemia was considerably lower than in our patient: 162 mg/dL¹⁷, 345 mg/dL¹¹, and 450 mg/dL (this final patient presented with hypoglycemia [23 mg/dL] and only became hyperglycemic after treatment with parenteral glucose).¹⁸ Similarly, our patient's peak serum glucose exceeded the highest level (698 mg/dL) reported among diabetic patients taking metformin therapeutically who went on to develop lactic acidosis.²¹ Since our patient did not have a history of diabetes, his extreme hyperglycemia is all the more unexpected. Also, we could not identify any previously published cases where the hyperglycemia progressively worsened, in the absence of administration of glucose, as occurred in our patient early in his clinical course.

The potential mechanism for the severe hyperglycemia in our patient is not clear. Nothing among metformin's known mechanisms would logically explain the progressive and severe hyperglycemia, especially since these mechanisms should tend to limit the glucose level. However, if one

considers what might occur if the patient could no longer secrete enough insulin, as may occur with pancreatitis, then a potential explanation arises. With a lack of circulating insulin, metformin's ability to enhance peripheral insulin sensitivity would count for nothing. Glucose would accumulate in the serum, since it would not be able to enter the tissues. It is also possible that a counter-regulatory hormone surge (epinephrine \pm glucagon) from the acute physiologic stress of the overdose contributed to the hyperglycemia. This mechanism is conjectural, since pancreatitis was not confirmed in our patient with serum amylase and lipase levels, nor with any radiographic study showing pancreatic inflammation. Similarly, no circulating insulin levels were measured, which would have to be low for this mechanism to work. Nevertheless, pancreatitis remains a promising potential mechanism, as our patient's clinical presentation with complaints of vomiting and abdominal pain is consistent with previously reported cases of metformin-associated pancreatitis.^{11,17,18}

Alternate toxicologic causes for this patient's hyperglycemia seem unlikely. Only a few toxins are routinely associated with hyperglycemia, including calcium channel-blockers and the ingestion of agents that specifically poison pancreatic beta-islet cells, such as alloxan, nitrophenolurea (an obsolete rodenticide), and streptozocin (a chemotherapy agent for pancreatic islet cell tumors). Calcium channel-blockers can impair insulin release, often resulting in modest hyperglycemia. Our patient had no known access to these drugs and did not present with hypotension or bradycardia. Similarly, our patient had no known access to beta-islet cell poisons, and the resultant hyperglycemia from these agents occurs in association diabetic ketoacidosis within a few days after significant exposure.

Our patient also ingested ethanol, which may have contributed to his severe toxicity. Ethanol itself may have increased the serum lactate level or induced pancreatitis. Also, the interaction of ethanol with biguanide drugs has been shown to increase lactate levels both in animal and human studies.⁴

Another potential complicating factor was the elevated osmolal gap in our patient, unaccounted for by the serum ethanol level. The presence of a highly elevated osmolal gap (>20 mOsm/kg) is most frequently caused by exposure to toxic alcohols,²² and our patient had a progressively worsening metabolic acidosis, which would be consistent with toxic alcohol poisoning. However, our patient denied ingesting anything but ethanol and metformin, and his parents confirmed that they saw no evidence at home of exposure to products containing isopropanol, methanol, or ethylene glycol. An unexplained osmolal gap may be seen in several severe disease states, including shock or sepsis,²² and was also seen in four out of nine cases of fatal metformin toxicity,³ providing an alternate explanation for its presence. We believe our

patient's severe acidosis and osmolal gap is highly unlikely to have occurred from toxic alcohol exposure for several reasons. Firstly, there was no historical evidence supporting the ingestion of any alcohol other than ethanol. Secondly, the patient did not exhibit additional clinical signs of end-organ damage, such as visual complaints from methanol toxicity, nor calcium oxalate crystalluria from ethylene glycol. The patient did have an elevated initial serum creatinine of 2.1 mg/dL, but this did not worsen as would be expected from ethylene glycol poisoning. Also, he had no ketonuria as would occur with isopropanol ingestion. Ingestion of propylene glycol might result in presentation with lactic acidosis and renal insufficiency, but such exposures are rare and severe acidosis should not occur in the presence of a supra-"therapeutic" ethanol level. Even if our patient had had severe toxic alcohol poisoning, the treatment would have included alcohol dehydrogenase inhibition, which was already effected by the presence of ethanol, and hemodialysis, which was already planned to treat his severe metformin toxicity.

CONCLUSION

Metformin toxicity is associated with development of lactic acidosis. Because metformin does not induce insulin release by pancreatic beta-islet cells, exposures infrequently induce disorders of glucose homeostasis. When abnormal glucose levels occur with metformin exposures, hypoglycemia is much more commonly seen than hyperglycemia; hypoglycemia is usually ascribed to concurrent anti-diabetic medications such as the sulfonylureas. Hyperglycemia appears to be a marker of severe toxicity in cases of metformin poisoning, and may be associated with the patient's underlying diabetes or the development of pancreatitis.

We presented the case of a non-diabetic 29-year-old man who took a large overdose of metformin and ethanol in a suicide attempt. He presented with severe lactic acidosis and a progressively increasing serum glucose level. The hyperglycemia might have been due to pancreatitis, although confirmatory studies were not obtained before the patient died. The patient's peak serum glucose level of 707 mg/dL is the highest yet reported in a case of metformin toxicity.

Address for Correspondence: Jeffrey R. Suchard, MD, Department of Emergency Medicine, University of California, Irvine, 101 The City Drive, Rt. 128-01, Orange, CA 92868. Email: jsuchard@uci.edu.

REFERENCES

1. Anonymous. Top 300 Prescriptions for 2005: By Number of US prescriptions dispensed. Available at: <http://www.rxlist.com/script/main/art.asp?articlekey=79509>. Accessed February 12, 2008.

2. Bailey CJ, Turner RC. Metformin. *N Engl J Med*. 1996; 334:574-579.
3. Spiller HA, Quadrani DA. Toxic effects from metformin exposure. *Ann Pharmacother*. 2004; 38:776-780.
4. Chan NN, Brain HPS, Feher MD. Metformin-associated lactic acidosis: a rare or very rare clinical entity? *Diabet Med*. 1999; 16:273-281.
5. Heaney D, Majid A, Junor B. Bicarbonate haemodialysis as a treatment of metformin overdose. *Nephrol Dial Transplant*. 1997; 12:1046-1047.
6. Teale KFH, Devine A, Stewart H, Harper NJH. The management of metformin overdose. *Anaesthesia*. 1998; 53:691-701.
7. Barrueto F, Meggs WJ, Barchman MJ. Clearance of metformin by hemofiltration in overdose. *Clin Toxicol*. 2002; 40:177-180.
8. Chang CT, Chen YC, Fang JT, Huang CC. High anion gap metabolic acidosis in suicide: Don't forget metformin intoxication – two patients' experiences. *Ren Fail*. 2002; 24:671-675.
9. Gjedde S, Christiansen A, Pedersen SB, Rungby J. Survival following a metformin overdose of 63 g: a case report. *Pharm Toxicol*. 2003; 93:98-99.
10. Nisse P, Mathieu-Nolf M, Deveaux M, Forceville X, Combes A. A fatal case of metformin poisoning. *J Toxicol Clin Toxicol*. 2003; 41:1035-1036.
11. von Mach MA, Sauer O, Weilemann LS. Experiences of a poison center with metformin-associated lactic acidosis. *Exp Clin Endocrinol Diabetes*. 2004; 112:187-190.
12. Harvey B, Hickman C, Hinson G, Ralph T, Mayer A. Severe lactic acidosis complicating metformin overdose successfully treated with high-volume venovenous hemofiltration and aggressive alkalinization. *Pediatr Crit Care Med*. 2005; 6:598-601.
13. Lacher M, Hermanns-Clausen M, Haefner K, Brandis M, Pohl M. Severe metformin intoxication with lactic acidosis in an adolescent. *Eur J Pediatr*. 2005; 164:362-365.
14. Spiller HA, Sawyer TS. Toxicology of oral antidiabetic medications. *Am J Health-Syst Pharm*. 2006; 63:29-38.
15. Galea M, Jelacin N, Bramham K, White I. Severe lactic acidosis and rhabdomyolysis following metformin and ramipril overdose. *Br J Anaesth*. 2007; 98:213-215.
16. Spiller HA, Weber JA, Winter ML, et al. Multicenter case series of pediatric metformin ingestion. *Ann Pharmacother*. 2000; 34:1385-1388.
17. Ben MH, Thabet H, Zaghdoudi I, Amamou M. Metformin associated acute pancreatitis. *Vet Human Toxicol*. 2002; 44:47-48.
18. Mallick S. Metformin induced acute pancreatitis precipitated by renal failure. *Postgrad Med J*. 2004; 80:239-240.
19. Lalau JD, Race JM. Lactic acidosis in metformin therapy. *Drugs*. 1999; 58(Suppl 1):55-60.
20. Talwalkar PG, Deotale P. Severe hypoglycemia in a patient with type 2 diabetes mellitus on metformin monotherapy. *J Assoc Phys India*. 2007; 55:458-459.
21. Lalau JD, Race JM. Lactic acidosis in metformin-treated patients: prognostic value of arterial lactate levels and plasma metformin concentrations. *Drug Safety*. 1999; 20:377-384.
22. Suchard J. Osmolal gap. In: Dart RD, et al., eds. *Medical Toxicology*. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2004:106-109.

Images in Emergency Medicine: Chemical Pneumonitis from Hydrocarbon Aspiration

Keimanh Pham, MD
James Sverchek, MD
Rick A. McPheeters, DO

Kern Medical Center, Department of Emergency Medicine

Supervising Section Editor: Eric R. Snoey, MD

Submission history: Submitted December 28, 2007; Revision Received February 28, 2008; Accepted March 13, 2008.

Reprints available through open access at www.westjem.org

[WestJEM. 2008;9:165.]

A three-year-old female presented to the emergency department (ED) with a chief complaint of choking for 20 minutes after ingestion of an unknown clear liquid; this was followed by one episode of emesis. On arrival she had no respiratory distress, and her symptoms had resolved. Her physical exam and vital signs were unremarkable with a room air oxygen saturation of 99% and a respiratory rate of 28 breaths per minute. The patient's family brought in the water bottle containing the clear substance, and it was identified as naphtha. An initial chest radiograph showed no infiltrates or consolidations. The patient was given nebulized saline and did not require further therapeutic intervention.

A repeat chest radiograph (Figure 1) was done four hours after the first, and it showed left lower lobe and right middle lobe infiltrates, demonstrating significant pneumonitis. Throughout her stay in the ED, she continued to have no respiratory distress, auscultation of her chest remained clear, and her vital signs normal. Nevertheless, the patient was admitted to pediatrics for observation. Her hospital stay was uneventful and she was discharged home in 24 hours after a final chest radiograph showed no interval changes.

Exposure to hydrocarbons comes in many forms, but it causes the most damage with aspiration.¹ This damage depends on the viscosity (the resistance to flow, measured in Saybolt seconds universal [SSU]); volatility (the propensity to vaporize); and the chemical side chains of the hydrocarbon. Lower viscosity, especially less than 60 SSU, and higher volatility are associated with a greater chance of aspiration with resultant pulmonary injury.²

While hydrocarbons have been reported to be toxic to various organ systems, the most frequent adverse effect is aspiration, which can cause a chemical pneumonitis from direct injury to the lung parenchyma.³ Other effects of aspiration are pulmonary edema, bronchospasm, and resultant hypoxia. On pathology, there is a necrotizing pneumonia along with direct destruction of capillaries, alveolar septae, and the pulmonary epithelium. The surfactant layer, which is composed of lipids, is made soluble by hydrocarbons, causing further damage. Subsequently, there can be atelectasis, interstitial inflammation, and hyaline membrane formation.

Patients with hydrocarbon exposure should be placed on a cardiac monitor with continuous pulse oximetry. A chest radiograph should be taken in patients with significant exposure or if they are symptomatic (tachycardia, tachypnea, hypoxia). If

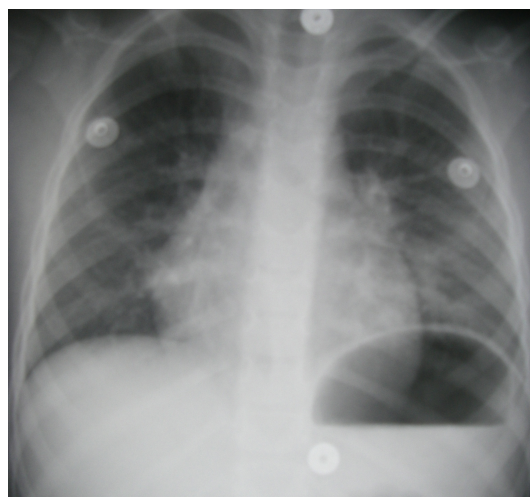


Figure 1.

aspiration is severe, patients may need intubation and positive pressure ventilation.

With minimal exposure, patients can be discharged after a six-hour period of observation. Pulse oximetry and a chest radiograph may be helpful in reassessing the patient before discharge. Symptomatic patients with more severe exposure should be admitted and observed for a minimum of 24 hours.

Address for Correspondence: Keimanh Pham, MD, Department of Emergency Medicine, Kern Medical Center, 1830 Flower Street, Bakersfield, CA 93308. Email: kiemanh@gmail.com.

REFERENCES

1. Lee DC. Hydrocarbons. Marx JA *et al.* (eds): Rosen's Emergency Medicine: Concepts and Clinical Practice, ed 6, 2006, pp2428-32.
2. Dyer S. Hydrocarbons. Wolfson AB, *et al.* (eds): Harwood-Nuss' Clinical Practice of Emergency Medicine, ed 4, 2005, pp1590-93.
3. Wax PM, Beuhler MB. Hydrocarbons and Volatile Substances. Tintinalli JE, *et al.* (eds): Emergency Medicine: A Comprehensive Study Guide, ed 6, 2004, p1124-30.

Issues and Solutions in Introducing Western Systems to the Pre-hospital Care System in Japan

Tetsuji Suzuki, EMT-P, MA*
Masamichi Nishida, MD, PhD†
Yuriko Suzuki, RN‡
Kunio Kobayashi, MD, PhD*
S. V. Mahadevan, MD#

* Graduate School of Health Information Science Teikyo Heisei University, Department of Pre-hospital Emergency Medical Care
† Teikyo University, School of Medicine, Emergency Medicine
‡ Kyoto Kujo Hospital, Department of Nursing
Stanford University School of Medicine, Division of Emergency Medicine

Supervising Section Editor: Chris Mills, MD

Submission history: Submitted November 8, 2007; Revision Received March 12, 2008; Accepted March 15, 2008.

Reprints available through open access at www.westjem.org

Objective: This report aims to illustrate the history and current status of Japanese emergency medical services (EMS), including development of the specialty and characteristics adapted from the U.S. and European models. In addition, recommendations are made for improvement of the current systems.

Methods: Government reports and academic papers were reviewed, along with the collective experiences of the authors. Literature searches were performed in PubMed (English) and Ichushi (Japanese), using keywords such as emergency medicine and pre-hospital care. More recent and peer-reviewed articles were given priority in the selection process.

Results: The pre-hospital care system in Japan has developed as a mixture of U.S. and European systems. Other countries undergoing economic and industrial development similar to Japan may benefit from emulating the Japanese EMS model.

Discussion: Currently, the Japanese system is in transition, searching for the most suitable and efficient way of providing quality pre-hospital care.

Conclusion: Japan has the potential to enhance its current pre-hospital care system, but this will require greater collaboration between physicians and paramedics, increased paramedic scope of medical practice, and greater Japanese societal recognition and support of paramedics.

[WestJEM. 2008;9:166-170.]

INTRODUCTION

Emergency medical services (EMS) in Japan have developed primarily from emergency transportation services, historically emphasizing transporting patients to the most appropriate medical facility as soon as possible. Although this is an important aspect of EMS, it no longer accurately reflects the current state of pre-hospital care in Japan.

In 1991 Japan officially established national paramedic systems that enabled emergency personnel to work similarly to paramedics in the United States.¹ Before this time ambulance personnel were not allowed to use defibrillators without authorization by a physician. Only as recently as April 2006 were Japanese paramedics given permission to administer adrenaline to patients in cardiac arrest.²

Japan is a world leader in communication technology, including standard and cellular telephone services and dispatch systems. This technological advantage, combined with well-organized emergency medical services, has enabled Japanese ambulances to arrive within six minutes of emergency “119” calls (national average in 2006).³ However, the Japanese EMS system as a whole has yet to achieve maximum success because of various legal restrictions on paramedic scope of practice. This paper introduces the current Japanese system of pre-hospital care and compares it to the systems used in the U.S. and Europe. Adopting effective aspects of Western EMS systems could lead to improved medical care and patient outcomes in Japan.

Table 1. Component facilities and responsibilities of each in emergency medical service in Japan⁵

Emergency Medical Service	Component Facilities	Responsibilities
Primary	Holiday and night-time urgent care centers, on-call private practitioners	Triage for emergency patients, treatments and prescriptions for minor cases
Secondary	Emergency notification hospitals, national or private hospitals (both single-specialty and general hospitals), occupational health hospitals, university hospitals, medical associations hospitals	In-patient care Emergency surgery
Tertiary	High-level emergency medical center, emergency medical center, university hospital emergency room, critical care unit	Diagnosis and treatment of severe or life-threatening cases (e.g. severe injury, burn of large area, acute toxicity, cerebrovascular disease, ischemic heart disease, circulatory or respiratory failure, metabolic coma, sepsis, attaching severed finger or limb)

METHODS

Government and academic publications on the Japanese system of emergency medical services were reviewed. Japanese articles were selected from Ichushi (Japana Centra Revuo Medicina), using keywords such as emergency medicine/care, pre-hospital care, emergency transport systems, and emergency medical response. Government publications, journal articles, and book chapters that were most recent or peer reviewed were prioritized. English articles were selected by searching PubMed, using the same key terms, for study of the U.S., European, and international emergency care and pre-hospital care systems. Again, the most recent or peer reviewed articles were given highest priority.

Measures of improvements are proposed based on the collective experiences and research results of the authors and other relevant researchers.

RESULTS

Japanese Emergency Service System and its History

The existing system for emergency medical services in Japan depends on two distinct entities: the fire department, which is responsible for transporting patients, and the emergency medical system, which is responsible for actual medical care.

Emergency transport service in Japan is mostly performed by the fire department in each local government district/locale. Although not originally a responsibility of the fire department, emergency transport service has been performed by local fire stations since 1986, when it became required by national law.⁴ As a natural extension, pre-hospital care is considered a major responsibility of local government.

Japanese emergency medical service is based on two national systems: the emergency notification system, which began in 1964, and a supplementary system, which began in 1977. A partial amendment of the Fire Protection Law of 1986 states: "Patients with life threatening conditions and/or with disease that

has possibility of severe worsening, and without any method of appropriately transporting [themselves] immediately to places such as medical facilities are the subjects of emergency medical services." This amendment established and legally acknowledged emergency transport as the responsibility of fire departments in the modern system of emergency medical service in Japan. Of note, this service was intended for transportation, and not for pre-hospital medical care.

In December 1997, based on the report from the Emergency Medical Service System Basic Problem Commission, the national emergency medical system (Figure 1) was established.⁵ This system has three layers of service: the primary, secondary, and tertiary emergency medical systems (Table 1). The primary emergency medical system provides what is commonly called primary (or basic) medical care. These medical facilities are often the first point of contact for patients in the system and are responsible for handling relatively mild cases. At the end of 2003, 509 holiday and night-time urgent care centers were operating, and 686 areas with on-call private practitioners were registered.

The secondary emergency medical system is the focus of Japan's emergency medical services; it provides care to patients who need hospitalization or emergency surgery. Emergency notification hospitals, national or private hospitals (both single-specialty and general hospitals), occupational health hospitals, university hospitals, and medical association hospitals comprise this system. Hospitals in various regions take turns providing such emergency medical services. However, in areas that have adequate medical resources (i.e., Tokyo), certain hospitals are permanently assigned to provide secondary emergency medical services as part of the holiday and night-time medical services of local governments. In areas where hospitals take turns providing secondary emergency medical services, pre-hospital transport times may vary widely based on the hospital's location, and hospitals with fewer resources may be

overwhelmed when they are "on duty."

The tertiary emergency medical system provides 24-hour care for patients with serious and potentially fatal conditions such as cerebrovascular events, cardiac events, and severe injuries. The highest level of medical care is provided in this system, which is supported by specialists and supervisory physicians of the Japanese Association for Acute Medicine.

In response to the 2000 Ministry of Welfare report on pre-hospital care and rescue commission, emergency medical care centers were positioned as regional centers for pre-hospital care at the local level. They were expected to establish medical control systems and provide local paramedics with instruction, guidance, and post-event evaluations. By November 2004, there were 173 emergency medical care centers throughout Japan, and new small-scale centers called "mini ERs" are planned for each secondary emergency medical system district.⁶

An important issue that has arisen is the wide variability in quality of care between populous and financially-stable major cities (such as Tokyo) and rural regions. While major cities can meet most of the criteria for establishing emergency departments (EDs) in hospitals, many of the regional hospitals have not yet met official standards. Many hospitals have been forced to quickly establish EDs in response to government calls for increased emergency facilities.⁷ According to a survey conducted in Fukuoka prefecture, only four of 28 hospitals were certified by the Japanese Association of Acute Medicine, and only nine had certified acute care physicians.⁸ Fukuoka has a population of 1.32 million (2005) and is considered one of Japan's large cities; however, the staffing in its hospitals was found to be inadequate. The researchers who conducted this study concluded that many Japanese EDs were seriously understaffed, even in large cities.

Another fundamental issue is the absolute shortage of both medical personnel and available hospital beds. Ambulances may travel long distances (mean transport time in 2006 was 57 minutes⁹) to reach an appropriate or designated hospital but may not find a readily available hospital bed. In 2007 two similar incidents in the Nara prefecture captured national attention because ambulances transporting pregnant women in critical condition were unable to find hospital EDs with available beds and qualified physicians.^{10,11} These incidents prompted improvement in communication between local hospitals and emergency dispatchers, and led to increases in hospital staff with obstetric-specific training.¹²

The Role and Issues of Paramedics in Japan

In 1991 Japan officially established the role of the paramedic and enacted related regulations, designating its workers as medical professionals and establishing that emergency medical services encompasses both medical care at the scene and transport to medical facilities.¹ Paramedics are nationally licensed, not as part of the certification within fire departments, but as a new medical profession in its own right.

The U.S. has highly trained paramedics that provide high-level

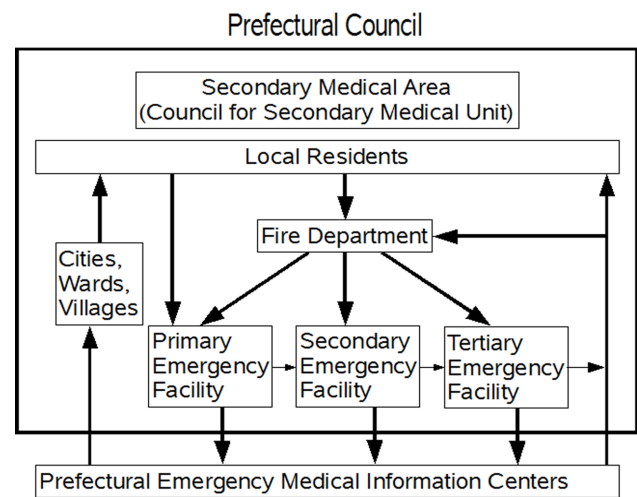


Figure 1.

emergency medical care. One difference between paramedics in the U.S. and Japan is the amount of training required. In the U.S. the classification, Emergency Medical Technician (EMT)-Paramedic, indicates that the individual has taken approximately 1200 hours of required coursework and training.¹³ The EMT-Paramedic National Standard Curriculum recommends 500-600 hours of classroom/practical laboratory, 250-300 hours of clinical training, and 250-300 hours of field internship.¹⁴ In Japan, most paramedics have a firefighter background. The paramedic job requirements include a minimum of five years of employment as ambulance personnel, approximately 509 hours of lecture, and 406 hours of hospital training or simulation in a designated training facility. This represents a total of approximately 915 hours of training; however, on-the-job training with currently certified paramedics is not required.¹⁵ In addition, there is no system for relicensing or renewal in Japan, and continuing education is up to each individual.

Another difference between the U.S. and Japanese paramedic profession is that most Japanese paramedics belong to the fire department as public civil servants and only a minority work as paramedics for private entities. U.S. paramedics may be either civil servants or private citizens. Patients in the U.S. are responsible for the costs associated with pre-hospital care, which forms much of the business of emergency medical services. Calling for an ambulance for minor conditions or when transportation to the hospital is unavailable occurs routinely in Japan but infrequently in the U.S.

Doctor Car Service

France utilizes a different EMS system in which traditionally hospital-based medical providers such as physicians may provide care outside of the hospital.¹⁶ The French system, called "SAMU," evolved because French physicians felt they were most qualified to perform resuscitation procedures in the pre-hospital setting. Pre-hospital medical professionals, including physicians, initiate medical care at the site of the incident. The system is nationally

organized by the Ministry of Health of the French government. Although commonly referred to as a "Doctor Car," the arriving medical team may not include a physician, especially for cases deemed not life-threatening. This system is independent of the fire safety service and emphasizes the independence of EMS. France, Germany, and Italy have the most established system of sending doctors in ambulances.¹⁷

In April 1991 the Japanese Lower House Social and Labor Commission Conference Minutes emphasized the introduction of "European styles of paramedic systems that routinely send physicians to the site of incident," and encouraged the evaluation of a SAMU-like system.¹⁶ In Japan, doctor cars can operate in three different ways. The first is the "docking method," which is used in the Aizu and Osaka Emergency and Life-Support Centers.¹⁸ When an emergency call is received, an ambulance is dispatched from the fire department and a doctor car is dispatched from a hospital; the two meet on the way to arrive together at the event site. The second is the "rendezvous method," in which the two arrive separately at the event site. The last method is the "Funabashi System," which was adopted by the city of Funabashi and permanently stations a doctor car in the fire house through the support of the local medical association. As an extension to this doctor car service, the Congresses passed the helicopter service law in June 2007, as a result of the success of introducing helicopters in nine base hospitals throughout Japan. The law was passed partly in response to the current shortage of physicians, especially in rural areas.¹⁶

Supporting Others in the Process of Development

Although the Japanese system is still evolving, not having yet achieved maximal efficiency or quality of care, Japan has been a leader in assisting other nations with the process of developing their emergency medical systems. One of the most notable efforts has been in Vietnam, where the Japan International Cooperation Agency has provided cars for use as ambulances as well as other emergency equipment since 1994.¹⁹ Because of increases in the number of victims from road traffic accidents in major Vietnamese cities, the lack of an adequate EMS system has become a serious issue. Japan has experienced similar issues in its process of economic and industrial development. Japanese emergency care professionals are assisting the Vietnamese in strengthening their "115" system and are encouraging the development of Vietnamese emergency care professionals.

DISCUSSION

For pre-hospital medical care in Japan to advance, there needs to be increased cooperation and communication between paramedics and physicians, support for an increased scope of practice for paramedics and increased societal support for the emergency medical healthcare workers.

Efforts made by Nara prefecture in assigning trained dispatchers to facilitate communication between hospitals and ambulances may enable a change in the system in increasing efficiency. Also, appointing a permanent facility to provide

emergency medical system services would increase the experience levels of healthcare workers. From the perspective of specific facilities and staffing levels, national or public hospitals are the best choices as permanent facilities. The challenge that needs to be overcome is administrative, political and cultural resistance to such a change.⁵

One of the methods considered as collaboration between physicians and paramedics is to delegate more procedures to trained paramedics. The more advanced skills and role of U.S. paramedics have evolved from the idea that physicians and other medical professionals consider it better to delegate routine jobs to other health professionals who have received the necessary training and who specialize in such services.²⁰ However, delegating what have traditionally been doctors' jobs is culturally at odds with many Japanese professionals, who feel that the idea of delegation intrudes into another professional's work. Such acts are considered as lacking respect for other professions. Despite such a background, modern Japanese society has embraced other Western technologies and cultures. This issue could be solved with time as other new ideas gradually became accepted in Japan.

Despite the new laws and regulations, the current status of the Japanese paramedic remains far from that of paramedics in the U.S. and Europe, who enjoy respect and social recognition and who can proactively perform medical procedures at event sites. U.S. and European systems are based on the concept that emergency medicine begins at the site of the accident or event. It is evident that trained paramedics who can perform more procedures on site contribute to better survival of patients compared with patient survival when emergency service personnel have only minimal or basic training. In a report by the Ministry of Internal Affairs and Communications, the survival rate after one month of cases of cardiopulmonary arrest reported by citizens was double when patients were treated by trained paramedics versus by general emergency service personnel (Figure 2).²¹

Bringing about change in the Japanese system to give paramedics more authority and training is largely a political issue and will require collaboration among the Japanese Medical Association, the Ministry of Health, Welfare, and Labor, and the Japanese Society for Emergency Medicine (JSEM). These groups have made initial efforts, with the JSEM in its 2003 annual symposium proposing to remove the restriction on paramedics that prohibits them from performing procedures only after an ambulance has arrived for patient transport. Hopes are high that with increased societal recognition and patient awareness, paramedics can have a larger role in emergency patient care and can be a more active medical profession.

To solve political and regulatory issues, members of the healthcare professions must gain public support. Healthcare professionals in emergency medicine and pre-hospital care have to inform the governments or community about what is needed – including increases in the number of staff and other resources, such as beds and more facility spaces. Traditionally, Japanese healthcare professionals have not been vocal in asking for their needs to be

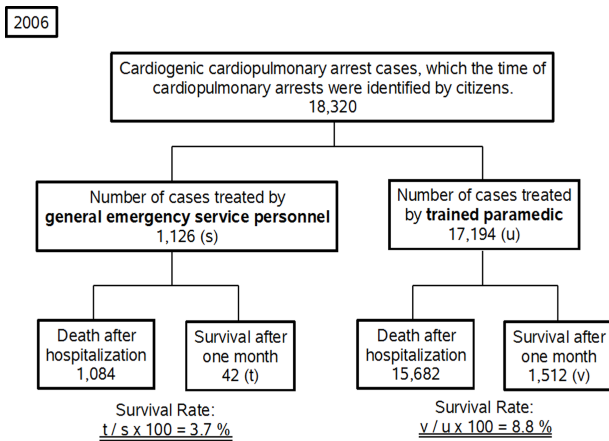


Figure 2.

filled, since they are considered helping-professionals and therefore not the ones to ask for help. But to garner societal support both emergency medical doctors and paramedics should raise the issues that they face, despite the cultural hesitations to do so.

Therefore, we would like to recommend: 1) More collaboration and cooperation between physicians and paramedics, because better results can be expected from delegating specific jobs to appropriately trained professionals. 2) Advocacy and increased recognition for the role of Japanese paramedics to provide greater support in establishing and expanding their role and training to achieve the level of paramedics in the U.S. 3) Increased societal support for emergency medical personnel and their work, needed to address the inadequate number of physicians, hospital beds, and communication professionals.

CONCLUSION

Japan has the technology to support an advanced emergency dispatch system, and doctors and paramedics are capable of performing necessary procedures. Currently, there are two methods operating in Japan: one similar to the U.S. system, in which trained paramedics arrive at site, and another similar to the European model, in which medical doctors arrive at site. However, Japanese paramedics have not yet achieved the level of ability and recognition of their U.S. and European counterparts. Other related issues of emergency medicine in Japan include shortages of emergency medical care professionals and hospital beds. The solution must address both political and cultural aspects; however, information and advocacy from medical professionals may be able to improve the situation. Future improvements lie in greater collaboration between physicians and paramedics, along with greater societal support and recognition of the paramedic profession.

Address for Correspondence: Tetsuji Suzuki, EMT-P, MA, Dept of Pre-hospital Emergency Medical Care, Graduate School of Health Information Science, Teikyo Heisei University, 2-51-4 Higashi-Ikebukuro, Toshima-ku, Tokyo 170-8445 Japan. Email: cpr119@coral.ocn.ne.jp.

REFERENCES

1. Tanigawa K, et al. Emergency medical service systems in Japan: Past, present, and future. *Resuscitation*. 2006; 69:365-370.
2. Fire Disaster Management Agency. 2007 White Paper. Available at: <http://www.fdma.go.jp/html/hakusho/h18/h18/html/id100000.html>. Accessed February 26, 2008.
3. Department of Fire Safety, Ministry of Internal Affairs and Communications. *2006 Outline of Emergency Services*; 2007.
4. Harita T, et al. A discussion for emergency transportation system, Byouin Kanri (Hospital Management). *J Japan Hosp Manag Assoc*. 2001; 39:25.
5. Kondo S. Emergency Medical System. In: Yamamoto Y, ed. *The Multiple Approaches for Emergency Medicine – Theory and Application*. Japan: Medical Journal Co. Ltd.; 2003.
6. Abe Y, et al. Shinryou to chiryou/ kyukyuiryou (Care and Treatment/ Emergency Medicine). 2003; 91(extra number issue).
7. Ezaki T et al. Current status of Japanese emergency medicine based on a cross-sectional survey of one prefecture. *Emergency Medicine Australasia*. 2007; 19:523-527.
8. Ezaki T, et al. Emergency medicine in Japan: A look at a current university hospital and the problems faced. *Emergency Medicine Australasia*. 2007; 19:296-299.
9. Miwa M, et al. The effect of the development of an emergency transfer system on the travel time to tertiary care centres in Japan. *Intl J Health Geograph*. 2006; 5:25.
10. Chunichi shinbun; May 24, 2007.
11. Mainichi Shinbun; August 27, 2007.
12. Outline of Nara Prefecture Board Meeting of Survey on Issues in August 2007 Emergency Transportation of Pregnant Women Incident. Prefecture of Nara; October 5, 2007.
13. Suzuki T. *The Mission and Reality of Private Paramedic*. Japan: Chigensha; 2004.
14. US Department of Transportation, National Highway Traffic Safety. *EMT Paramedic National Standard Curriculum*.
15. Shigeta M, Maeda K. Kyukyutaiin. Kyukyu kyumeishi kyoiku (ambulance crew and paramedic education). *Rinsho to Kyoiku (Clinical Activities and Education)*. 2000; 77.
16. Morimura H. (Dispatching emergency crew to the site of incident – French System of supporting out of hospital emergency medical care, SAMU). *Japan Traffic and Welfare Association, Asuka* 21; 2002:44.
17. Mashiko K. Trauma system in Japan: History, present status and future perspectives. *J Nippon Med Sch*. 2005; 72:194-202.
18. Hirma H. (Investigation on docking method doctor car system in Aizu General Hospital). *Kyukyugaku (Emergency Medicine)*. Japan: Herusu Publishing; 1997
19. Department of Fire Safety, Ministry of Internal Affairs and Communication. *Effects on survival results from emergency medical procedures under various circumstances*. Japan; 2007.
20. Fleischmann T et al. Emergency medicine in modern Europe *Emergency Medicine Australasia* 2007;19:300-302.
21. Kuroiwa Y. *Kyukyu iryou ni mess (Dissecting Emergency Medicine)* Information Center Publishing Division, 1990.

A Case of Complicated Urinary Tract Infection: *Klebsiella pneumoniae* Emphysematous Cystitis Presenting as Abdominal Pain in the Emergency Department

Kapil R. Dhingra, MD, MBA

University of California, Davis Medical Center, Department of Emergency Medicine

Supervising Section Editor: Sean O. Henderson, MD

Submission history: Submitted October 31, 2007; Revision Received January 11, 2008; Accepted January 21, 2008.

Reprints available through open access at www.westjem.org

This case report describes an atypical presentation of an atypical disease entity: Emphysematous Cystitis, a rapidly progressive, ascending urinary tract infection, in an emergency department (ED) patient whose chief complaint was abdominal pain and who had a urinalysis not consistent with the diagnosis of cystitis.

[WestJEM. 2008;9:171-173.]

INTRODUCTION

Emphysematous Cystitis (EC) is an uncommon, rapidly progressive bacterial infection whereby fermentation of albumin or glucose by the urinary pathogen results in gas formation in the bladder wall and lumen. Although it is an uncommon clinical entity, when present it is seen commonly in diabetic patients. Early recognition of EC in the emergency department (ED) can prevent morbidity and mortality resulting from progression of the infection to Emphysematous Pyelonephritis and Urosepsis.

CASE REPORT

A 59-year-old female presented to the ED with a chief complaint of abdominal pain. Her abdominal pain began three hours before presentation and had awoken her from sleep. This abdominal pain was described as “achy,” 5/10, intermittent, and located in the left lower quadrant without radiation. She denied nausea or vomiting and had no diarrhea or blood in her stools. Review of systems revealed that the patient had symptoms of dysuria for one day and left-sided flank pain along with her abdominal pain. She denied fevers or chills. She stated that, despite taking her prescribed insulin injections, her blood sugars have been persistently elevated above 400 with her personal glucometer. Her past medical history was significant for insulin-dependent diabetes mellitus (DM), hypertension, and prior cerebrovascular accident. She denied ever having any surgeries. She used regular insulin, Lantus (insulin glargine), and Ibuprofen. Her family history was

noncontributory, and she denied alcohol, tobacco, or illicit drug use.

Physical exam revealed a temperature of 36.5° C, respiratory rate of 18 breaths per minute, heart rate of 93 beats per minute, blood pressure of 166/81 mm Hg, oxygen saturation of 99% on room air, and capillary blood glucose of 447 mg/dL. She was awake, alert, and interacting appropriately. Her pupils were equally round and reactive to light. The cardiac exam was normal and lungs were clear with breath sounds bilaterally. The abdomen was soft with minimal tenderness to palpation in the left periumbilical and left lower quadrant. There was no guarding or rebound. Extremities were warm and well-perfused. The neurological exam revealed no deficit.

The metabolic panel revealed sodium of 132 mEq/L (normal 135-145), BUN of 45 mg/dL (normal 8-22), Creatinine of 2.0 mg/dL (normal 0.5-1.3), glucose of 473 mg/dL, and an anion gap of 8.0. A CBC revealed a white blood cell count of 12.0 K/MM3 (normal 4.5-11.0) and a manual differential with 10.10 K/MM3 Neutrophils (normal 0.80-7.70). The hepatic panel was remarkable for an alkaline phosphatase of 189 U/L (normal 35-115). The urine was visibly clear and non-turbid. Urinalysis was leukocyte esterase and nitrate negative, but revealed large occult blood, glucosuria, protein of 300 mg/DL, and 3-6 WBC/HPF (normal 0-3).

While laboratory studies were pending, the patient received one liter IV normal saline, four mg of morphine sulfate IV for pain, and 10 units of regular insulin. Despite fluid administration and analgesia, the patient continued to

have worsening 6/10 abdominal pain. Given the patient’s presentation of undifferentiated abdominal pain without clear etiology and her immunocompromised state, a non-contrast computed tomography (CT) scan of the abdomen was ordered. The CT scan revealed significant air in the bladder wall and lumen consistent with EC, severe left hydronephrotic and hydronephrosis without evidence of stones, and significant left perinephric stranding without gas in the renal parenchyma (Figure 1).

A gram of Ceftriaxone and 500 mg of Gentamycin were started empirically along with a repeat fluid bolus and placement of a Foley catheter. While awaiting consultation and admission to the Urology service, the patient complained of increased abdominal pain and was increasingly tender in the left lower quadrant and suprapubic region. Her heart rate was persistently tachycardic (in the 130s), and she developed a fever to 39.4°C. In light of developing urosepsis and the potential for rapid progression to Emphysematous pyelonephritis, Interventional Radiology (IR) was consulted and placed a left nephrostomy tube to relieve the functional obstruction of the urinary tract just eight hours after the patient presented to the ED. After nephrostomy tube placement by IR, the patient was admitted to the ICU for close monitoring. The urine culture grew out *Klebsiella pneumoniae* and the patient was continued on IV antibiotics. Three days after admission, the nephrostomy tube was removed as the patient was able to void without use of a foley catheter and was able to control her pain. She was subsequently discharged and given a prescription for Levofloxacin 500 mg every day for fourteen days.

DISCUSSION

EC, or “Cystitis Emphysematosa”, is a potentially fatal necrotizing infection that can begin in the bladder and rapidly ascend to the renal parenchyma.^{1,2} The etiology of EC is multifactorial and involves: gas-forming bacteria, decreased tissue perfusion, and elevated serum glucose in the context of an immunocompromised host such as a diabetic patient.^{1,4} The mixed fermentation process of substrates such as glucose and albumin within the urinary tract by bacteria results in the formation of H₂ and CO₂ gas both within the urinary tract and within the luminal mucosa.² In addition, bacterial endotoxin release may contribute to the inflammatory process by inducing paralysis of the urinary tract and thus urinary stasis. In this case, left ureteral stasis resulted in urine reflux into the renal parenchyma with hydronephrotic and hydronephrosis. Ultimately, this patient required placement of nephrostomy tubes to relieve the functional urinary obstruction.

Both the prevalence and the incidence of the spectrum of Emphysematous urinary tract infections are unknown, but patient demographics are significant for a predilection for middle-aged, diabetic women as was the patient that presented in this report.³ In general, patients with DM have an increased

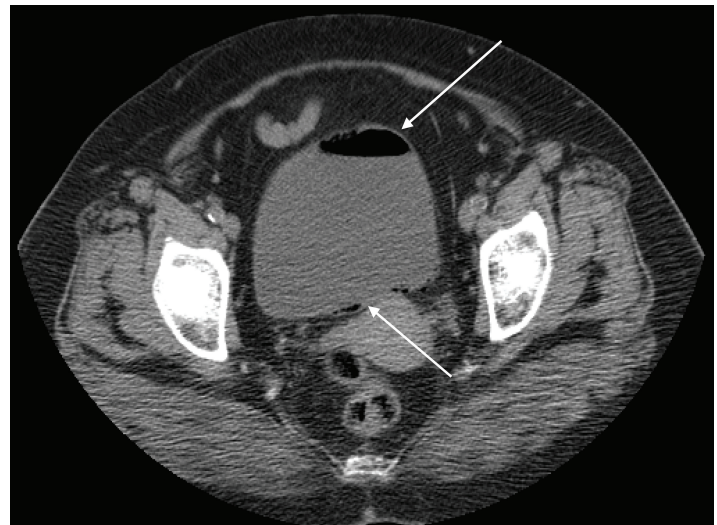


Figure 1. Abdominal CT with arrows demonstrating emphysematous changes in the bladder wall

risk of complicated urinary tract infection due to more atypical isolated urinary pathogens which thrive in the presence of an immunocompromised host.^{5,6} It is likely that the frequency of diagnosis is increasing as the prevalence of DM and use of CT scanning for undifferentiated abdominal pain is also increasing.

Table 1. Demographics and common pathogens³

Variable	Value n/N(%)
Mean age (years)	61.9
Men	49/135 (36%)
Women	86/135 (64%)
Diabetic	90/135 (66.7%)
Type 1 DM	25/52 (48%)
Type 2 DM	27/52 (52%)
Women with DM	61/86 (71%)
Men with DM	25/86 (29%)
Overall death rate	9/135 (7%)
Pathogens (n=119) (%)	
<i>Escherichia coli</i>	69 (58%)
<i>Klebsiella pneumoniae</i>	25 (21%)
<i>Enterobacter aerogenes</i>	8 (7%)
<i>Clostridium perfringens</i>	7 (6%)
<i>Candida albicans</i>	5 (4%)
<i>Pseudomonas aeruginosa</i>	3 (3%)
Other	13 (11%)

DM = diabetes mellitus

The spectrum of presentation of EC is broad; however, it is commonly associated with fever, chills, nausea, emesis, dysuria, and pneumaturia.^{7,8} Any patient with air in the bladder, not necessarily complicated by an inflammatory process or infection, will require inquiry regarding past surgical history. Both prior urologic procedure, such as cystoscopy, and/or enterovesicular fistula secondary to Diverticulitis or Crohn's disease are known to cause air in the bladder and bladder wall.

Although the diagnosis of urinary tract infection is made primarily based on patient history and urinalysis results, the diagnosis of EC is made via clinical suspicion and plain films and/or CT scan, which carries high sensitivity for the diagnosis.⁹ As in this case, the urinalysis was relatively negative and had mild pyuria without bacteriuria lessening concern for urinary tract infection. However, clinical suspicion for a complicated UTI was raised in this diabetic patient with undifferentiated abdominal pain and dysuria who later developed fever and chills. The CT scan will demonstrate presence of air within the bladder lumen and within the bladder wall. In addition, CT can confirm presence alternate sources of intraluminal gas such as an enterovesicular fistula.

Management of EC is correlated with disease severity. As in this case, empiric broad-spectrum intravenous antibiotics to cover gram-negative and anaerobic urinary pathogens was necessary to prevent disease. In rapidly progressive cases of EC, pathogens release endotoxins that create functional obstruction of the urinary tract via ureteral stasis and retrograde flow of urine into the renal parenchyma causing hydronephrosis and hydronephrosis. As a result, urinary drainage via cystectomy or nephrostomy tube placement may be necessary. Alternatively, there has been a case report of hyperbaric oxygen therapy as an adjunctive measure to the mainstays of antibiotics, urinary drainage, and fluid resuscitation in a case of EC.¹⁰

As reported, the mortality rate of EC is 7%, but rapidly progressive forms that result in Emphysematous Pyelonephritis can lead to nephrectomy and carry mortality rates between 19-75%.^{11,12}

CONCLUSION

The rapid progression of EC from a lower urinary tract infection to a complicated upper urinary tract infection warrants recognition of this disease entity by the emergency physician. Prior to the development of EC, an immunocompromised patient with an uncomplicated urinary tract infection may be treated with outpatient oral antibiotics. However, symptoms of abdominal pain or persistent dysuria in immunocompromised patients with a recently-treated, uncomplicated UTI or with a new presentation of symptoms

should be worked up as undifferentiated abdominal pain of an unknown origin. The presentation of EC can be insidious and may present as undifferentiated abdominal pain as in this report without initial evidence of urinary tract infection as seen in the screening urinalysis. In susceptible patient populations, such as in diabetic and immunocompromised patients, inclusion of EC in the emergency physician's differential diagnosis will reduce morbidity and mortality of this disease entity.

Address for Correspondence: Kapil R. Dhingra, MD, MBA, UC Davis Medical Center, Department of Emergency Medicine, 4150 V Street, PSSB Suite 2100, Sacramento, CA 95817, E-mail: kapil.dhingra@ucdmc.ucdavis.edu

REFERENCES

1. Bailey H: Cystitis emphysematosa: 19 cases with intraluminal and interstitial collections of gas. *Am J Roentgen*. 1961;86:850-862.
2. Hawtrey CE, Williams JJ, Schmidt JD: Cystitis emphysematosa. *Urology* 1974; 3:612-614.
3. Thomas AA, Lane BR, Thomas AZ, Remer EM, Campbell SC, Shoskes DA. EC: a review of 135 cases. *BJU International*. 2007 Jul;100(1):17-20.
4. Wessells H, McAninch JW. *Urological Emergencies: A Practical Guide*. 2005. 128-133.
5. J.E. Patterson and V.T. Andriole. Bacterial urinary tract infections in diabetes. *Infect Dis Clin North Am* 1997; 11:735-750.
6. Foxman B. Epidemiology of urinary tract infections: incidence, morbidity, and economic costs. *Am J Med*. Volume 113, Issue 1, Supplement 1, July 2002;5-13.
7. Mokabberi R, Ravakhah, K. Emphysematous Urinary Tract Infections: Diagnosis, Treatment and Survival. *Am J Med Sci* 2007;333(2):111-116.
8. Taussig AF. Pneumaturia with report of a case. *Boston Med Surg J* 1997;156: 769-74.
9. Bohlman ME, Sweren BS, Khazan R. Emphysematous pyelitis and Emphysematous pyelonephritis characterized by computerized tomography. *South Med J* 1991;84:1438-43.
10. McCabe JB, Mc-Ginn Merrit W, Olsson D, Wright V, Camporesi EM. EC: rapid resolution of symptoms with hyperbaric treatment: a case report. *Undersea Hyperb Med* 2004;31:281-4.
11. Huang JJ, Chen KW, Ruaan MK. Mixed acid fermentation of glucose as a mechanism of Emphysematous urinary tract infection. *J Urol* 1991 Jul;146(1): 148-51.
12. Michaeli J, Mogle P, Perlberg S, et al. Emphysematous pyelonephritis. *J Urol* 1984 Feb;131(2): 203-8.

Herpes Zoster Ophthalmicus

Thomas Catron, MD
H. Gene Hern, MD, MS

Alameda County Medical Center, Highland General Hospital, Department of
Emergency Medicine

Supervising Section Editor: Eric R. Snoey, MD

Submission history: Submitted November 15, 2007; Revision Received None; Accepted February 22, 2008.

Reprints available through open access at www.westjem.org

[WestJEM. 2008;9:174-176.]

INTRODUCTION

Herpes zoster is a common diagnosis in the emergency department (ED). Caused by the reactivation of the varicella-zoster virus (VZV), zoster usually presents as a painful dermatomal rash. In addition to skin or mucosal involvement, VZV reactivation commonly affects the ophthalmic division of the trigeminal nerve and subsequently the eye. This manifestation is termed herpes zoster ophthalmicus (HZO). HZO is considered an ophthalmologic emergency, as sequelae often include severe chronic pain and vision loss. In order to ensure proper follow up and to minimize morbidity, the accurate and timely diagnosis of HZO in the ED is extremely important. While typically an easy diagnosis to make based on history and skin findings, occasionally HZO presents as an isolated ophthalmologic process that is difficult to distinguish from other more benign causes of a red eye.

CASE REPORT

A 64-year-old Chinese man presented to the ED with five days of increasingly blurry vision in his right eye. The blurring was associated with mild photophobia and a right-sided headache which he described as a burning pain over his forehead. The patient did not wear corrective lenses and denied trauma, chemical exposure, discharge, foreign body sensation, sick contacts, or history of similar problems. Review of systems was positive for approximately one week of malaise, myalgia, and subjective fevers. The patient denied sore throat, rhinorrhea, hearing changes, rashes, or any other concerns. The patient did not know if he had ever had chicken pox or cold sores, and he denied any past medical problems, surgeries, allergies, or medications.

Physical exam revealed a well appearing male in no distress with vitals as follows: heart rate at 72 beats/minute, blood pressure at 145/84 mm Hg, respirations at 14 breaths/minute, oral temperature at 36.8°C and room air oxygen saturation at 97%. HENT exam showed no obvious rashes or lesions; however, the patient had mild hyperesthesia over his right forehead. He had normal ear canals, tympanic



Figure 1. Dendritic fluorescein uptake from Herpes zoster ophthalmicus.

membranes, nares, and oropharynx. The patient had no cervical or auricular lymphadenopathy.

Ophthalmologic exam was significant for slight right-sided conjunctival irritation with no exudates or obvious corneal scarring. The patient had normal lids and consensual pupillary reflexes with mild discomfort on ipsilateral and contralateral pupillary testing. Extra-ocular motions were intact with no diplopia. Visual acuity was 20/20 in the left eye, 20/80 in the right, and 20/20 in both. Visual fields were normal and symmetric. Fundoscopic exam revealed normal appearing eye grounds with no evidence of hemorrhage, vascular occlusion, papilledema, or retinal detachment. Intraocular pressures were normal bilaterally. Anterior chamber slit lamp exam showed no corneal ulceration or cell and flare. Fluorescein staining yielded a 7mm branched dendritic corneal lesion (Figure 1). The remainder of his exam was unremarkable.

The patient was started on oral acyclovir and a topical cycloplegic. Ophthalmology was consulted, and the patient was discharged to ophthalmology clinic, where the ED findings were confirmed. In clinic the patient was started on

topical steroids in addition to antivirals and cycloplegics, and was discharged home with early follow up by ophthalmology.

DISCUSSION

Varicella-zoster virus manifests itself as two distinct syndromes in humans. During primary infection, the virus causes chicken pox. After the initial infection, VZV remains latent in the dorsal root ganglia of sensory neurons, possibly reappearing as herpes zoster later in the patient's life.

In the United States, VZV primary infection rate reaches nearly 100% by 60 years of age; however, this rate will likely decrease with the widespread usage of the VZV vaccine. The lifetime risk of varicella zoster is between 10-20% in patients who have had chicken pox. Risks for reactivation include any decline in the T-cell mediated immune response including that caused by normal aging, HIV/AIDS, and immunosuppressive medications.

Herpes zoster ophthalmicus is a relatively common presentation of zoster. By definition HZO is reactivation of VZV in the ophthalmic division of the trigeminal nerve (V_1), and accounts for 10-25% of all herpes zoster cases.¹ While HZO does not necessarily affect the structures of the eye, many of the acute and long-term complications associated with the disease are the result of direct viral toxicity to the eye or the ensuing inflammatory response within the eye. It is thought that approximately 50% of those diagnosed with HZO will develop complications. Many of these poor outcomes can be prevented or ameliorated with early recognition, treatment, and referral.

Classically, HZO begins with flu-like symptoms including fever, myalgia, and malaise for approximately one week. Typically, patients then develop a painful unilateral dermatomal rash in the distribution of one or more branches of V_1 : supraorbital, lacrimal, and nasociliary. The skin manifestations usually begin as an erythematous macular rash, progressing over several days into papules, vesicles, and then pustules. These eventually rupture and scab, and in immunocompetent individuals will resolve over the course of two to three weeks. In about 60% of cases patients will complain of a painful dermatomal prodrome prior to the development of any rash. Ocular involvement is not invariable in HZO; however, in patients with nasociliary nerve involvement (Hutchinson's sign) some case series indicate 100% go on to develop eye pathology.² Approximately one third of those without nasociliary involvement will eventually develop eye manifestations.³ Conversely, in a small subset of patients (such as our patient), ocular symptoms will predominate.⁴

Physical exam should include a thorough ophthalmologic exam including external inspection, visual acuity, visual fields, extra ocular movements, pupillary response, funduscopy, intraocular pressure, anterior chamber slit lamp exam, and corneal exam with and without staining. As in our patient, the

differential diagnosis includes a broad range of pathology: herpes simplex keratitis, other viral or bacterial conjunctivitis, uveitis, glaucoma, trauma, chemical exposure, vascular occlusion, migraine, cluster headache, trigeminal neuritis, optic neuritis, vasculitis, and others. If the classic rash is present, a brief exam will limit this differential; however, as in our patient, this would not be adequate to diagnose a patient presenting with purely ocular involvement. Classic ocular involvement is typified by dendritic or punctate keratitis (Figure 1). This pattern of infection occurs in approximately 65% of patients with HZO;⁴ however, other eye findings are more frequent and range from simple conjunctivitis to retinal necrosis and detachment. Any structure in the eye may be involved.⁴

Diagnostic testing is rarely indicated, as diagnosis can almost always be made by a combination of history and physical. It is possible to use a Tzanck smear or Wright stain to determine whether lesions contain herpes-type virus (though these will not differentiate between VZV and other herpes viruses). Viral culture, direct immunofluorescence assay, or PCR may also be used to confirm the diagnosis.

In the ED treatment consists of local wound care, pain control, initiation of antiviral medication, and antibiotics if needed. Acyclovir and other similar antivirals have been shown to significantly decrease adverse outcomes related to HZO if started within 72 hours of initial symptoms.^{5,6,7,8} Studies report reduced pain during the outbreak, reduced likelihood of postherpetic neuralgia, increased rate of skin healing, decreased duration of viral shedding, and decreased incidence of corneal involvement. It is not proven that the patient accrues these benefits if the medications are started after the 72-hour window, but given the extremely low side-effect profile of the drugs and the serious sequelae of complications, most physicians recommend administering antivirals during the first seven to 10 days of symptoms.

Steroids (topical and systemic) may also play a role in the treatment of HZO. In some studies systemic steroids have been shown to speed skin healing and to decrease initial pain; however, there have been no definitive studies showing reduced long-term incidence of post-herpetic neuralgia or ocular complications.^{9,10} Likewise, topical steroids may be helpful in the initial management of pain due to uveitis or scleritis; however, they have a number of serious side effects and potential complications and should never be used without concomitant anti-viral treatment.⁴ Always obtain ophthalmologic consultation prior to starting steroid treatment.

Oral opiate and nonsteroidal anti-inflammatory medications are frequently indicated for pain and may be augmented by the use of cycloplegics in patients who display features of iritis.

In otherwise healthy individuals with minimal eye involvement, most sources suggest outpatient treatment with seven to 10 days of oral acyclovir at a dose of 800mg

five times daily. Other antiviral agents (valacyclovir and famciclovir) offer equivalent benefits and have reduced frequency of dosing, which may improve patient compliance. In high risk cases, admission for IV acyclovir is indicated. Admission is recommended for those with known immunodeficiency, patients on immunosuppressive medications, involvement of multiple dermatomes (which may indicate immunosuppression), retinal involvement, corneal ulceration, or serious bacterial superinfection. All patients with the possible diagnosis of HZO require ophthalmologic consultation prior to discharge from the ED in order to ensure full evaluation for more serious complications. For patients eventually discharged home, early ophthalmologic follow up is mandatory.

CONCLUSION

HZO is a potentially serious reactivation of VZV in the distribution of the ophthalmic division of the trigeminal nerve. Usually this entity presents with classic findings that make diagnosis simple; however, as was the case with our patient, HZO may easily be confused with more common and benign eye pathology. Full ophthalmologic exam is warranted in the patient with decreased vision or a red eye. Once the diagnosis of HZO is entertained, appropriate antiviral and adjunctive therapy should be initiated and ophthalmology consultation should be requested for evaluation and treatment of common complications.

Address for Correspondence: H. Gene Hern, Jr. MD, MS, Department of Emergency Medicine, Alameda County Medical Center, Highland General Hospital, 1411 E 31st St, Oakland, CA 94602-1018. Email: gene_hern@yahoo.com.

REFERENCE

1. Ragozzino MW, Melton LJ 3d, Kurland LT, Chu CP, Perry HO. Population-based study of herpes zoster and its sequelae. *Medicine*. 1982; 61:310-316.
2. Zaal MJ, Völker-Dieben HJ, D'Amario J. Prognostic value of Hutchinson's sign in acute herpes zoster ophthalmicus. *Graefes Arch Clin Exp Ophthalmol*. 2003; 241:187-191.
3. Harding SP, Lipton JR, Wells JC. Natural history of herpes zoster ophthalmicus: predictors of postherpetic neuralgia and ocular involvement. *Br J Ophthalmol*. 1987; 71:353-358.
4. Shaikh S, Ta CN. Evaluation and management of herpes zoster ophthalmicus. *Am Fam Physician*. 2002; 66:1723-1730.
5. Severson EA, Baratz KH, Hodge DO, Burke JP. Herpes zoster ophthalmicus in olmsted county, Minnesota: have systemic antivirals made a difference? *Arch Ophthalmol*. 2003; 121:386-390.
6. Morton P, Thomson AN. Oral acyclovir in the treatment of herpes zoster in general practice. *N Z Med J*. 1989; 102:93-95.
7. Huff JC, Bean B, Balfour HH Jr, Laskin OL, Connor JD, Corey L, et al. Therapy of herpes zoster with oral acyclovir. *Am J Med*. 1988; 85:84-89.
8. McGill J, Chapman C, Mahakasingam M. Acyclovir therapy in herpes zoster infection. A practical guide. *Trans Ophthalmol Soc U K*. 1983; 103:111-114.
9. Wood MJ, Johnson RW, McKendrick MW, et al. A randomized trial of acyclovir for 7 days or 21 days with and without prednisolone for treatment of acute herpes zoster. *N Engl J Med*. 1994; 330:896-900.
10. Whitley RJ, Weiss H, Gnann JW, et al. Acyclovir with and without prednisone for the treatment of herpes zoster. A randomized, placebo-controlled trial. The National Institute of Allergy and Infectious Diseases Collaborative Antiviral Study Group. *Ann Intern Med*. 1996; 125:376-383.

Complications of MRSA Treatment: Linezolid-induced Myelosuppression Presenting with Pancytopenia

Julie Gorchynski, MD, MSc*
Jeri K Rose, BS†

* CHRISTUS Spohn Memorial Hospital, Texas A&M University, Department of
Emergency Medicine

† University of North Texas Health Science Center, Texas College of Osteopathic
Medicine

Supervising Section Editor: Sean O. Henderson, MD

Submission history: Submitted March 2, 2008; Revision Received March 8, 2008; Accepted March 13, 2008.

Reprints available through open access at www.westjem.org

Methicillin-resistant *Staphylococcus aureus* (MRSA) infections have grown to epidemic proportions in the United States. With the development of increasing drug resistance of MRSA to traditional antimicrobials, there has been a search for a more effective antibiotic treatment. Linezolid is one of the most effective oral medications used for outpatient treatment of MRSA infections. We present a case of pancytopenia after outpatient treatment with linezolid. Myelosuppression is a rare but serious side effect of linezolid of which emergency physicians need to be aware in order to provide early intervention.

[WestJEM. 2008;9:177-178.]

INTRODUCTION

Since their emergence onto the clinical scene in the 1960s, methicillin-resistant strains of *Staphylococcus aureus* (MRSA) have continued to plague clinicians. MRSA is no longer solely a nosocomial infection and has now become the most common cause of skin and soft tissue infections presenting in emergency departments (EDs).^{1,2} With the expanding resistance to multiple antibiotics, physicians are faced with the challenge of finding effective outpatient therapy. Linezolid is effective against multiple strains of MRSA and is available in an oral formulation for outpatient therapy.¹ We present a case of linezolid-induced myelosuppression. Myelosuppression is a rare yet significant side effect reported during post-marketing use of linezolid. ED physicians need to be aware of this serious side effect, which may place the patient at risk for further health-related complications as a result of cytopenias.

CASE REPORT

A 73-year-old female presents to the ED complaining of generalized weakness. The patient reported weight loss due to nausea, vomiting, and decreased appetite for two months. The patient had no known drug allergies and denied tobacco, alcohol, or illicit drug use. Review of systems included dyspnea on exertion and fatigability. Past medical history included a lumbar fracture due to a motor vehicle accident, requiring lumbar fusion. The patient later developed recurrent

osteomyelitis of the lumbar vertebrae. Repeated incision and drainage of the abscesses at her previous surgical site showed persistent MRSA-positive wound cultures. Subsequently, inpatient treatment with intravenous vancomycin for MRSA osteomyelitis of the lumbar spine was instituted. The patient had been discharged on oral linezolid for continued outpatient treatment for MRSA at a dose of 600 mg twice a day for six weeks, with Zofran as needed for nausea and vomiting.

On this visit, the patient was in no acute distress and nontoxic appearing. Her mucous membranes were pale, with no gingival bleeding; the fundoscopic eye exam was normal. The lungs were bilaterally clear to auscultation; the heart had a regular rate and rhythm with no murmurs, gallops or rubs. Abdominal exam was soft, non-tender, non-distended with normoactive bowel sounds. A rectal exam revealed normal tone, brown stool was hemoccult negative. Extremities exhibited full range of motion and intact neurovascularization; the skin was without rashes and petechiae; pulses were full throughout. Exam of the back demonstrated a healed incision in the lumbar region, correlating with the previous surgeries; no erythema, tenderness, warmth, rashes, lesions, open wounds, or fluctuance were noted. Cranial nerves II-XII were intact; motor and sensory exams, reflexes, and gait were normal with no focal neurologic signs.

ED laboratory tests included a complete metabolic panel, complete blood count, urinary analysis, lipase level, cardiac

enzymes, partial thromboplastin time, prothrombin time and INR, and reticulocyte count. All labs were within normal limits, except the CBC, which reported a white blood cell count of 2,100 with a normal differential, hemoglobin of 4.2, platelet count of 64,000, with normal red blood cell indices and normal peripheral smear. EKG and chest x-ray were normal. Inpatient TIBC, iron and transferrin levels, and high reticulocyte values were consistent with iron deficiency and active bone marrow response.

The patient was admitted for treatment of linezolid-induced myelosuppression. She received IV fluids and was transfused with packed red blood cells due to symptomatic anemia. Treatment with linezolid was discontinued, and the patient was restarted on IV-administered vancomycin. Her hospitalization was uneventful, and the patient's cell counts returned to baseline, without the need for a bone marrow biopsy. A peripherally inserted central catheter line was placed and the patient was discharged home to continue outpatient IV therapy with vancomycin for her persistent MRSA osteomyelitis.

DISCUSSION

MRSA infections present most commonly as skin or soft tissue infections, in the form of cellulitis, furuncles or abscess, as well as more serious infections, such as pneumonia and sepsis. MRSA infections have historically occurred more frequently in hospitalized patients with compromised immune systems. More recently, community-acquired MRSA infections are of epidemic proportions in previously low-risk groups in the United States.^{3,4} The genetic element that confers methicillin resistance continues to evolve, creating multi-drug resistant strains. Without a definitive marker for identifying MRSA strains, outpatient treatment continues to challenge the medical community.^{1,5}

Historically, MRSA has been treated successfully with outpatient oral sulfonamides, clindamycin, rifampin, doxycycline, or a combination of these agents.⁵ With the development of increasing drug resistance of MRSA to these traditional antimicrobials, there has been a search for more effective antibiotics. One recent study demonstrated that vancomycin, linezolid, and quinupristin-dalfopristin were the most effective antibiotics against multiple strains of MRSA.¹ The parenteral administration of vancomycin and quinupristin-dalfopristin has limited their use in the outpatient setting; however, the availability of an oral formulation of linezolid has led to its increasing utilization.

Linezolid is an oxazolidinone antibiotic indicated for the treatment of Gram-positive bacterial infections, including bacterial pneumonia, skin and soft tissue infections, and vancomycin-resistant enterococcal infections. The most common adverse effects include diarrhea, nausea, and headache; less common side effects include hypertension, lactic acidosis, and elevated liver enzymes. Among the most

severe adverse effects, seen with prolonged courses of therapy, include irreversible peripheral neuropathy, optic neuropathy, and reversible myelosuppression.⁸

Oral linezolid is now being prescribed more frequently for outpatient treatment of MRSA due to the increase in multi-drug resistant microbes. In the literature, five cases of linezolid-induced myelosuppression have been reported.^{6,7} Of these cases, four reported anemia and thrombocytopenia and only one case reported pancytopenia. In this case, pancytopenia developed after only six weeks of therapy, whereas the other case reported pancytopenia developing after five months of treatment with linezolid. In addition to our patient, three of the five reported cases had their myelosuppression reversed after discontinuation of linezolid.⁷ Per the manufacturer's recommendation, patients on linezolid therapy for longer than two weeks should be monitored regularly in order to identify myelosuppression.

Emergency physicians need to be aware of this rare and serious side effect of linezolid-induced myelosuppression.

Address for Correspondence: Julie Gorchynski, MD, MSc, Department of Emergency Medicine, Texas A&M University System, CHRISTUS Spohn Memorial Hospital, 2606 Hospital Blvd. 3W, Corpus Christi, TX 78405. Email: jgorchyn@msn.com.

REFERENCES

1. Draghi DC, Sheehan DF, Hogan P, Sahm DF. Current antimicrobial resistance profiles among methicillin-resistant *Staphylococcus aureus* encountered in the outpatient setting. *Diagn Microbiol Infect Dis.* 2006; 55:129-133.
2. Ahmad K. MRSA becomes growing problem in US emergency departments. *Lancet Infect Dis.* 2006; 6:621.
3. National Library of Medicine: MedlinePlus. (2007 May 02). MRSA infection. Available at: <http://www.nlm.nih.gov/medlineplus/ency/article/007261.htm>. Accessed May 8, 2007.
4. Centers for Disease Control and Prevention. *Community-Associated MRSA Information for Clinicians.* 2005; Available at: http://www.cdc.gov/ncidod/dhqp/ar_mrsa_ca_clinicians.html. Accessed May 8, 2007.
5. Elston DM. Methicillin-Sensitive and Methicillin-Resistant *Staphylococcus aureus*: Management Principles and Selection of Antibiotic Therapy. *Dermatol Clin.* 2007; 25:157-164.
6. Spellberg B, Yoo T, Bayer AS. Reversal of linezolid-associated cytopenias, but not peripheral neuropathy, by administration of vitamin B6. *J Antimicrob Chemother.* 2004; 54:832-835.
7. Green SL, Maddox JC, Huttenbach ED. Linezolid and Reversible Myelosuppression. *JAMA.* 2001; 285:1291.
8. Pfizer, Inc. Zyvox prescribing information. 2007; Available at: www.zyvox.com/prescribingInfo.asp. Accessed May 23, 2007.

Images in Emergency Medicine: Splenic Infarction Due to Sickle Cell Trait after Climbing Mt. Fuji

Aki Morishima, MD
Joel M. Schofer, MD
Pierre Pelletier, MD
James M. McKee, MD

Naval Hospital Okinawa

Supervising Section Editor: Matthew Strehlow, MD

Submission history: Submitted April 7, 2008; Revision Received May 12, 2008; Accepted May 30, 2008.

Reprints available through open access at www.westjem.org

[WestJEM. 2008;9:179.]

A 41-year-old African American female with a history of alcoholism in remission developed acute vomiting, chills, and severe left upper quadrant abdominal pain while climbing Mt. Fuji (Shizuoka Prefecture, Japan). She presented to a Japanese hospital and was diagnosed with cholelithiasis. The studies obtained are unknown and were unavailable.

She presented two days later to a military emergency department in Okinawa, Japan (a two hour flight from Mt. Fuji) with continued severe left upper quadrant pain, increased with inspiration. Her only medication was disulfiram. The remainder of her history was unremarkable. Her vitals signs were normal, except for a blood pressure of 158/93 mm Hg. She appeared to be in moderate distress. Her physical examination was significant for tenderness in the left upper quadrant with guarding but no rebound. She had left costovertebral angle tenderness. Her urinalysis showed small blood on the urine dipstick with negative microscopy. A non-contrast CT scan of the abdomen and pelvis, obtained to look for urolithiasis, showed cholelithiasis.

A contrast enhanced CT of the abdomen and pelvis was consistent with splenic infarction (Figure 1). Hemoglobin electrophoresis obtained upon admission was consistent with sickle cell trait. Splenic infarction in patients with sickle cell trait was first reported during the Korean War in African American pilots flying in unpressurized aircraft.¹

Patients with sickle cell trait inherit a normal hemoglobin A gene from one parent and an abnormal hemoglobin S gene from the other parent. In the United States, it occurs in 7-10% of African Americans and is rare in people of other ethnicities.² Although patients with sickle cell trait are usually asymptomatic, they can develop splenic infarctions in hypoxic environments.³

Signs and symptoms typically include epigastric and left upper quadrant pain developing over 48 hours and may include guarding, rebound, splenomegaly, fever, anorexia, and vomiting. Laboratory data often show increases in lactate dehydrogenase, leukocytosis, thrombocytopenia, and anemia.^{1,2,3} A CT scan with intravenous contrast will demonstrate areas of multifocal splenic infarcts. Splenic rupture is a rare complication. Treatment

with supportive care including oxygen, analgesia, and hydration is usually adequate. Patients who are unstable may require splenectomy.² In this case, supportive care was provided and the patient recovered without sequelae. Physicians should consider splenic infarction in patients who develop suspicious symptoms after exposure to a high altitude environment.³

Address for Correspondence: Joel M. Schofer, MD, PSC
482 Box 2827 FPO, AP 96362. Email: jschofer@gmail.com.

REFERENCES

1. Chamberland DL. Splenic infarction in an African-American male with sickle cell trait. *Am J Hematol*. 2006; 82:86-87.
2. Tieman CJ. Splenic crisis at high altitude in 2 white men with sickle cell trait. *Ann Emerg Med*. 1999; 33:230-233.
3. Sheikh A. Splenic syndrome in patients at high altitude with unrecognized sickle cell trait: splenectomy is often unnecessary. *Can J Surg*. 2005; 48:377-381.

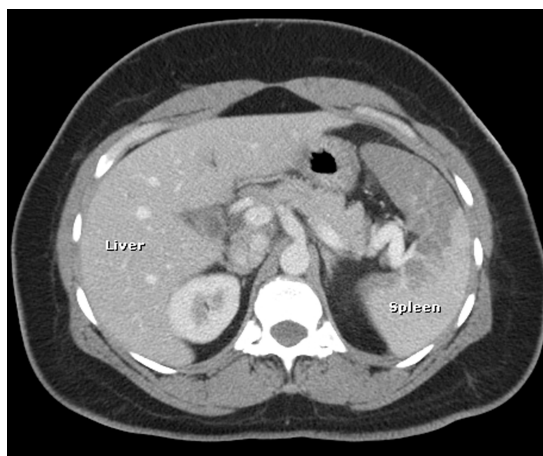


Figure 1. Contrast abdominal CT at the level of the splenic hilum shows lack of contrast enhancement in a large area of the anterior spleen.

Images in Emergency Medicine: Pelvic Digit

Jennifer Carnell, MD*
Christopher Fee, MD†

* Alameda County Medical Center, Highland General Hospital, Department of
Emergency Medicine

† University of California, San Francisco Medical Center, Division of Emergency
Medicine, Department of Medicine

Supervising Section Editor: Rick A. McPheeters, DO

Submission history: Submitted February 23, 2008; Revision Received April 1, 2008; Accepted April 3, 2008.

Reprints available through open access at www.westjem.org

[*WestJEM*. 2008;9:180.]

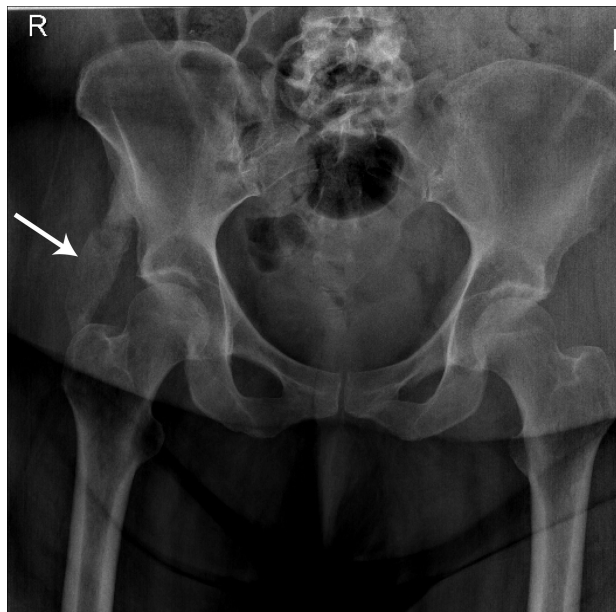
A 48-year-old female with systemic lupus erythematosus and renal failure was admitted to the hospital with pyelonephritis. While in the emergency department (ED), during transfer to a bedside commode, she slipped to the floor and subsequently reported right lateral hip pain. Examination revealed mild tenderness to palpation in that area. Radiographs were obtained and demonstrated a pelvic digit (indicated by arrow). What may be mistakenly interpreted radiographically as a fracture of the pelvic digit is actually a pseudoarticulation, a common finding near the base of the pelvic digit. Also referred to as a pelvic rib or an iliac rib, a pelvic digit is a rare, congenital anomaly that is usually asymptomatic and, therefore, found incidentally by plain radiograph. Pelvic digits are most often associated with the ilium but may also pseudoarticulate with other pelvic bones or the abdominal wall.¹ Their well-corticated appearance without defect facilitates differentiation from post-traumatic myositis ossificans, heterotopic bone formation, ligamentous calcifications and fracture.² As in the

case of our patient, radiographs are often obtained in the ED in the setting of trauma. Consequently, it is important to consider the benign entity of pelvic digit as a possibility and avoid further unnecessary work-up.

Address for Correspondence: Jennifer Carnell, MD,
Department of Emergency Medicine, Alameda County
Medical Center, Highland Hospital, 1411 East 31st Street,
Oakland, CA 94602. Email: jencarnell@gmail.com.

REFERENCES

1. Casey MC, Phancao J, Pressacco J. Answer to case of the month # 106 pelvic digit. *Can Assoc Radiol J*. 2006; 57: 51-3.
2. Nguyen VD, Matthes JD, Wunderlich CC. The pelvic digit: CT correlation and review of the literature. *Computerized Medical Imaging and Graphics*. 1990; 14:127-31.



11th Society for Academic Emergency Medicine Western Regional Research Forum

The 11th Annual Western Regional Research Forum of the Society for Academic Emergency Medicine was hosted by the University of California, Irvine Department of Emergency Medicine on March 28-29, 2008 in Costa Mesa, California. With author permission, seven selected abstracts are printed below and the remainder are published online at www.westjem.org.
[WestJEM. 2008;9:181-183.]

Reprints available through open access at www.westjem.org

1 Use of a Live Porcine Model to Detect Ocular Pathology by Bedside Sonography

Vincent L. Ball, MD; Bradley N. Younggren, MD; Stephen A. Crandall, MD
Madigan Army Medical Center

Objectives: To evaluate emergency medicine (EM) residents' ability to detect ocular pathology using portable ultrasound (US) in a live porcine model.

Methods: This was a single-blinded outcome study to determine both the validity of a live porcine model to evaluate ocular pathology and to assess the ability of EM residents to accurately diagnose ocular pathology using handheld US. Subjects were EM residents who had undergone a two-day US course as part of their EM orientation month and had reviewed a one-hour self-instruction computer tutorial on ocular US prior to the study. Vitreous hemorrhages, retro-orbital hemorrhages and intraocular foreign bodies were simulated by placement of porcine blood and metallic objects under ultrasound guidance out of view of the subject population. Residents then performed self-directed US of two eyes each and were asked to comment on any pathology observed. Time required for each scan was noted.

Results: 72 scans were performed to examine for vitreous hemorrhage, retro-orbital hemorrhage, and intraocular foreign bodies. EM residents were able to detect a significant abnormality greater than 93% (95CI 87-99%) of the time. Vitreous hemorrhage was the most detectable injury with over 95% (95CI 86-100%) accuracy. A significant abnormality was detected in the models with intraocular foreign bodies 97% (95CI 90-100%) of the time with a clear diagnosis of foreign body noted in 73% (95CI 57-89%) of the cases. Retro-orbital hemorrhage was the most difficult to detect with 62% (95CI 41-83%) accuracy. The average time taken for scanning two eyes was 458 sec. Central retinal artery flow was detected in 100% of the 26 cases in which this was documented. Accuracy of diagnoses was similar across levels of EM training.

Conclusion: EM residents can accurately diagnose significant ocular pathology using handheld US in a live porcine model. This is the first study to demonstrate the feasibility and effectiveness of such a model.

2 Violations of Match Rules: Asking for a Commitment during the Interview

H. Gene Hern, MD; Harrison J. Alter, MD; Charlotte P. Wills, MD; Eric R. Snoey, MD; Barry C. Simon, MD
Alameda County Medical Center – Highland General

Background: Applicants to residency face a number of difficult questions during the interview process. One type of question, which is expressly forbidden to ask, is that of a program asking for a verbal or written commitment to rank the program highly. The regulations governing the NRMP match expressly forbid any residency programs asking or requiring such a commitment.

Methods: We conducted a cross sectional survey of US applicants applying in five specialties (Emergency Medicine, Internal Medicine, OB/GYN, Orthopaedics, and General Surgery) during the 2006-2007 interview season through the Electronic Residency Application Service (ERAS) of the AAMC. Applicants were asked to recall if they experienced questions that asked them to provide any sort commitment (verbal or otherwise) stating they would rank a program highly. Applicants were provided with a unique ID from ERAS anonymizing them to the study authors.

Results: Of the 13,372 applicants surveyed, 6,981 returned a valid completed survey (52.2%). Overall 18.3% of applicants stated they were asked for a commitment. 15.3% of applicants to Emergency Medicine, 14.9% of those in Internal Medicine, 24.1% in Ob/Gyn, 29.3% in Ortho, and 22.4% in General Surgery were asked to commit to a program during the interview. In comparison to Emergency Medicine, ORs for other specialties asking for a commitment were: .96 (.78-1.19) for IM, 1.75 (1.36-2.25) for

Ob/Gyn, 2.29 (1.74-3.02) for Ortho and 1.59 (1.23-2.05) for General Surgery. 64.7% of applicants felt uncomfortable or very uncomfortable sharing this information, and 33.0% stated they would be less likely or much less likely to rank the program highly because of the question.

Conclusion: Applicants to residencies are being asked questions expressly forbidden by the NRMP match. Among the five specialties surveyed, Orthopaedics and Ob/Gyn have the highest incidence of this type of violation. Asking for a commitment makes applicants less likely to rank a program highly.

3 Maximizing Medical and Health Outcomes after a Catastrophic Disaster: Defining a New “Crisis Standard of Care”

Emile F. Chang, MSC; Howard Backer, MD; Tareq A. Bey, MD; Kristi L. Koenig, MD
University of California, Irvine; California Department of Public Health

Background: When healthcare demand exceeds resources during catastrophic disasters such as pandemic flu, hurricanes, or earthquakes, physicians shift focus from individual patients to populations of patients.

Objectives: To define the key principles and identify areas for future research for providing optimal care after a catastrophic disaster.

Methods: Investigators performed an exhaustive literature review to identify all articles about standard of care during a catastrophic disaster. In addition, the State of California provided access to prepublication copies of academic and stakeholder consensus documents.

Results: Analysis of 22 articles identified the following key principles for “Crisis Standard of Care” 1) prioritize population health rather than individual outcomes; 2) respect ethical principles of beneficence, stewardship, equity, and trust; 3) modify regulatory requirements to provide liability protection for healthcare providers making resource allocation decisions; 4) designate a crisis triage officer and include provisions for palliative care in triage models for scarce resource allocation (e.g. ventilators).

Conclusions: Despite the science of disaster medicine being in its infancy, several key principles exist for maximizing health outcomes during a catastrophic disaster. Evidence-based research is urgently needed to assess when to shift to the new “Crisis Standard of Care” and to determine optimal methods of educating healthcare providers to understand principles for optimizing care in a resource-poor environment. In addition, codifying the science of triage, developing and testing prognostic tools, and studying the effectiveness of crisis health risk communication strategies for healthcare workers and the public are key areas for future research.

4 Impact of Winter Resort Injuries on ACS Level I Trauma Center

Karen Danenhauer, MD; Craig Moffat, MS; Nathan Gilmore,

MD; Christy L. McCowan, MD

University of Utah Health Care; University of Pittsburgh

Objectives: To evaluate the impact of winter resort injuries on hospital resource utilization at a regional ACS level 1 Trauma Center.

Methods: Patients \geq 12 years presenting to the emergency department (ED) with an acute injury sustained at a winter resort were included in the study. Trained research assistants approached eligible patients in the ED. Missed patients were identified from the ED log and contacted by phone. ED and hospital data was obtained from trauma registry and hospital records. Patients were excluded if their injury occurred outside a winter resort, if they were inter-facility transfers, or if they did not present the day of their injury.

Results: Four hundred seventeen patients presented to the ED from local ski resorts during the 2006-07 season. Three hundred two of 417 (72.4%) patients were male. Average patient age was 32.8 +/- 16.1 years. 203 of 417 (48.7%) patients lived locally. Two hundred fifty-five of 417 (61.2%) patients arrived via ground emergency medical services (EMS), and 39 (9.4%) arrived via helicopter EMS. Two hundred fifty-five of 371 (60.6%) patients were not wearing helmets. One hundred sixty of 415 (38.6%) patients were admitted to the hospital, 47 of 160 (29.3%) were admitted for observation, and 23 (14.4%) required ICU admission. Average hospital LOS was 3.3 +/- 3 days with an average ISS of 7.6 +/- 4.6. Average ICU LOS was 58 +/- 77 hours. 14 of 23 (60.9%) ICU admissions had head injuries. Two hundred eleven of 415 (50.8%) patients required specialty intervention and/or consultation, including 73 (34.6%) trauma activations, and 105 (49.8%) orthopedic consultations. Seven patients were emergently intubated (3 by EMS), two required chest tubes, and two were taken emergently to the operating room.

Conclusions: Patients presenting with injuries from winter resorts have a significant impact on both EMS and hospital resources. Winter resort injuries have a high rate of hospital admission, specialty care, and/or trauma service evaluation. Injury prevention initiatives, including increasing helmet usage, in areas with high winter resort densities may help to increase awareness of the common and potentially serious injuries.

5 Outcome Study of Same-Level Falls in the Elderly with Momentary Loss of Consciousness or Acute Neurological Deficit

Eric S. Hegedus, MD; Samuel J. Stratton, MD
University of California, Irvine

Background: Same-level falls in the elderly may be associated with intracranial and cervical spine injury because of aging changes, co-morbid conditions, and medications such as anticoagulants.

Objectives: To determine the rate of cerebral hemorrhage, intracranial hemorrhage and cervical spinal cord injury after same-level falls in persons 65 and older who had loss of consciousness or acute focal neurological deficit associated with the fall.

Methods: Retrospective, observational outcome study of persons 65 and older who suffered a same-level fall and for whom Emergency Medical Services (EMS) were summoned. Study location was Orange County, California (population three million) and study period was from January 2006 through September 2007. Persons included in the study were reported by EMS to have a same-level fall with associated momentary loss of consciousness or focal neurological deficit. Outcomes of interest were acute cerebral hemorrhage, intracranial hemorrhage, and cervical spinal cord injury. Statistical measures included demographic information and rates for the outcomes of interest with calculated 95% confidence intervals.

Results: During the study period, 131 cases met study inclusion criteria. Mean age was 79.3 +/- 8.0 years and 48% were male. 14% died during acute hospitalization. For the study population of 131, cervical spine injury occurred in 6 cases (5%; 95% CI = 2%, 10%), cerebral hemorrhage in 10 cases (8%; 95% CI = 4%, 13%), and intracranial hemorrhage in 43 cases (33%; 95% CI = 25%, 41%). A total of 59 of 131 same-level falls with loss of consciousness or focal neurological deficit had associated cervical spine injury, cerebral hemorrhage, or intracranial hemorrhage for an overall rate of 45% (95% CI = 37%, 54%).

Conclusions: For persons aged 65 and over, same-level falls with momentary loss of consciousness or acute neurological deficit have a strong association with cerebral hemorrhage, intracranial hemorrhage and cervical spinal cord injury

6 Success Rates of Direct Laryngoscopy and Glidescope in an Academic Emergency Department

Laura Dolkas, MD; Arthur R. Smolensky, MD; John C. Sakles, MD
University of Arizona

Background: In the last 10 years there has been an explosion of alternative airway devices and rescue airway devices, such as video laryngoscopes, optical laryngoscopes and intubating laryngeal airways. The success rates of these devices compared to direct laryngoscopy are unknown.

Objectives: To compare the success rates of a standard intubating device, direct laryngoscopy, with an advanced form of video laryngoscopy known as the glidescope, as both a first-attempt airway device and as a rescue airway device.

Methods: We retrospectively studied data from an academic ED between July 1, 2007 and December 1, 2007. Faculty and residents were asked to fill out a standardized form for each intubation performed in the ED with routine critical information such as number of intubation attempts, initial device used, rescue device(s) used, level of operator, and whether the intubation was performed for a traumatic or medical resuscitation. One hundred seventy-nine intubations were studied.

Results: The level of operator performing the intubations was as follows: Attending physicians (6.7%), PGY-I (10.6%), PGY-II (25.1%), PGY-III (54.2%), Anesthesia (1.7%), and MS-4 (1.7%).

The success rate of direct laryngoscopy as a first-attempt airway device was 70% (64 of 92), while the glidescope first-attempt success rate was 79% (58 of 73). When used as a rescue device after other methods failed, direct laryngoscopy was successful 77% of the time (10 of 13), and the glidescope was successful 88% of the time (15 of 17).

Conclusions: Early data shows a trend towards superior success with the glidescope as a first-attempt airway device and as a rescue airway device in the ED setting. Future analysis of a greater number of intubations will reveal if these early trends are statistically significant enough to suggest a superior airway device in the emergency setting.

7 Near-Hanging Injuries in an Urban Emergency Department

Stuart P. Swadron, MD; Kori Sauser, MS; Aleya Hyderi; Sean O. Henderson, MD
USC/Keck School of Medicine

Objectives: Hanging is the second most common method of suicide in the United States. Few studies have examined the epidemiology of near-hanging cases presenting to an emergency department (ED).

Methods: A retrospective chart review was performed on patients presenting to an urban Level I trauma center between 1997 and 2007 with a diagnosis of near-hanging. Charts were abstracted for the following: gender, age, location of discovery, ligature, ligature marks, type of suspension, duration of hanging, cardiopulmonary resuscitation (CPR) at scene, cardiopulmonary arrest (CPA), Glasgow Coma Score (GCS) on arrival, endotracheal intubation, imaging and toxicology results, social history, injuries sustained, additional diagnoses and patient outcome. Descriptive statistics were used to summarize the data.

Results: Of 77 patients identified, 67 (87%) were male and 10 (13%) were female. The mean age was 33.9 years (range 10 to 86). Seventy-four (96%) cases were the result of a suicide attempt. The most common location of discovery was a jail cell (49%). The mean GCS on arrival was 10.4 (range 3 to 15). Endotracheal intubation was performed in the ED in 28 (36%). Hanging durations were not consistently available. Of 33 patients for whom a full chart review was performed, 29 patients (88%) survived to discharge. Three (10%) of these patients suffered CPA and one (3%) received CPR at the scene. Of six patients (18%) with a GCS of 3, two (33%) survived to discharge. Only three surviving patients (10%) suffered hypoxic brain injury. A single patient had a C-spine fracture and another developed pulmonary edema. Hanging durations of up to 10 minutes were recorded in the survivors.

Conclusions: We describe one of the largest series of near-hanging injuries presenting to an ED. Men far outnumbered women in cases of attempted suicide. The majority of patients survived, and several patients with CPA, CPR on scene, GCS scores of 3, and prolonged hanging times survived to discharge.

11th Society for Academic Emergency Medicine Western Regional Research Forum

Published Online at www.westjem.org

- 1 **Use of a Live Porcine Model to Detect Ocular Pathology by Bedside Sonography**
Vincent L. Ball, MD; Bradley N. Younggren, MD; Stephen A. Crandall, MD
Madigan Army Medical Center
- 2 **Violations of Match Rules: Asking for a Commitment during the Interview**
H. Gene Hern, MD; Harrison J. Alter, MD; Charlotte P. Wills, MD; Eric R. Snoey, MD; Barry C. Simon, MD
Alameda County Medical Center; Highland Hospital
- 3 **Maximizing Medical and Health Outcomes after a Catastrophic Disaster: Defining a New “Crisis Standard of Care”**
Emile F. Chang, MSC; Howard Backer, MD; Tareq A. Bey, MD; Kristi L. Koenig, MD
University of California, Irvine; California Department of Public Health
- 4 **Impact of Winter Resort Injuries on ACS Level I Trauma Center**
Karen Danenhauer, MD; Craig Moffat, MS; Nathan Gilmore, MD; Christy L. McCowan, MD
University of Utah Health Care; University of Pittsburgh
- 5 **Outcome Study of Same-Level Falls in the Elderly with Momentary Loss of Consciousness or Acute Neurological Deficit**
Eric S. Hegedus, MD; Samuel J. Stratton, MD
University of California, Irvine School of Medicine
- 6 **Success Rates of Direct Laryngoscopy and Glidescope in an Academic Emergency Department**
Laura Dolkas, MD; Arthur R. Smolensky, MD; John C. Sakles, MD
University of Arizona
- 7 **Near-Hanging Injuries in an Urban Emergency Department**
Stuart P. Swadron, MD; Kori Sauser, MS; Aleya Hyderi; Sean O. Henderson, MD
USC/Keck School of Medicine
- 8 **Patient Perceptions of Resident Interpersonal and Communication Skills in the Emergency Department; An Analysis by Post-Graduate Year of Emergency Medicine Residents and Off-Service Residents**
Rebecca L. Palmer, MD; Sarah S. Andrus, MD; Rongwei Fu, PhD; Emilie Weed, BS; Annick Yagapen; Daniel Knoepfelmacher; Megan Lockwood; Patrick H. Brunett, MD
Oregon Health & Science University
- 9 **A Survey of the Beliefs Regarding International Emergency Medicine among Fourth-Year Medical Students Planning on Matching in Emergency Medicine**
Elissa M. Schechter, MD; Nicholas Forget, MD; Allison Richard, MD; William K. Mallon, MD
Los Angeles County and USC Medical Center
- 10 **Administrative Support and Clinical Shift Load Varies among EM Residency Leadership**
H. Gene Hern, MD; Charlotte Page Wills, MD; Eric Katz, MD
Alameda County Medical Center; Highland Hospital; Maricopa Medical Center
- 11 **Attending and Resident Satisfaction with Feedback in the Emergency Department**
Lalena M. Yarris, MD, MCR; H. Gene Hern, Jr, MD, MS; Judy Linden, MD; David M. Nestler, MD; Cedric Lefebvre, MD; Rongwei Fu, PhD; Esther K. Choo, MD; Patrick H. Brunett, MD;
Oregon Health & Science University; Alameda County Medical Center; Boston Medical Center; Mayo Clinic; Wake Forest University
- 12 **Use of Standardized Evaluation Methods for Assessing ACGME Core Competencies**
H. Gene Hern, MD; Charlotte Page Wills, MD; Susan Promes, MD; Douglas Char, MD
Alameda County Medical Center; Highland Hospital; University of California, San Francisco; Washington University, St. Louis
- 13 **Do You Plan to Have Children? The Incidence of Potentially Illegal Questions during Resident Interviews**
H. Gene Hern, MD; Harrison J. Alter, MD; Charlotte Page Wills, MD; Eric R. Snoey, MD; Barry C. Simon, MD
Alameda County Medical Center; Highland Hospital
- 14 **Emergency Medicine Interest Group: Faculty and Preclinical Medical Students Differ in Their Preferences for Educational Objectives**
Carol K. Lee, MS4; Wendy C. Coates, MD
David Geffen School of Medicine at UCLA; Harbor-UCLA Medical Center
- 15 **Effectiveness of a Central Line Bundle Protocol including Ultrasonic Guidance in Reducing Operative Complications Associated with Central Venous Catheter Placement in a Community Emergency Department**
Alissa Arnold, BS; Sam Torbati, MD
Cedars-Sinai Medical Center

- 16 Terrain Park Injuries: Demographics and Injury Patterns**
Craig Moffat; Nathan Gilmore, MD; Karen Danenhauer, MD; Jade Bringhurst; Christy McCowan, MD
University of Utah Health Care; University of Pittsburgh
- 17 Access to Care - A Pilot Study in Los Angeles**
Rachel E. Rosenheck; Kathryn R. Challoner, MD
Harvard School of Public Health; USC/Keck School of Medicine
- 18 Time Analysis of Consultant Services in Adult ED Admissions**
Katherine A. Sutherland, MD; Joseph Stephan Stapczynski, MD
Maricopa Medical Center
- 19 Low Rate of MRSA Colonization among Residents is Stable Over Time**
Jennifer Carnell, MD; H. Gene Hern, Jr, MD; Amandeep Singh, MD; Paul Cheung, MPH; Bradley Frazee, MD
Alameda County Medical Center; Highland Hospital
- 20 Opt-Out HIV Screening in an Urban Emergency Department: Implementation of the New Centers for Disease Control and Prevention Guidelines**
Douglas AE White, MD; Alicia N. Scribner, MPH
Alameda County Medical Center; Highland Hospital
- 21 Influenza Vaccination in the Emergency Department**
Heather Woodin, MS2; Katherine M. Hiller, MD; Billie Winegard, MPH; Sid Patanwala, PharmD; Melanie DeBoer, PhD
University of Arizona
- 22 Ultrasound-Guided Needle Drainage Versus Incision and Drainage (I&D) of Skin Abscesses**
Cameron Scott, MD; J. Scott Lowry, MD; Albert B. Fiorello, MD; Billie Winegard, MPH
University of Arizona
- 23 Social Factors that Predict Penetrating Trauma: Has Anything Changed in the Past Twenty Years?**
Lisa Moreno-Walton, MD; Valerie Katz, MD; Amish Shah, MD; Jayne Lieb, MD
Lincoln Medical and Mental Health Center; Mt. Sinai Medical Center
- 24 Addition of Echocardiography to Electrocardiographic Stress Testing Clarifies Cardiac Pathology but Does not Improve Diagnostic Accuracy in Emergency Department Chest Pain Unit Patients**
Ali Ghobadi, MD; Eric Kaihwan Wei, BS; Rocky Sayegh, BS; Son Doan; Roula Saleem; Mark I. Langdorf, MD, MHPE
University of California, Irvine School of Medicine
- 25 Rapid Sequence Intubation - from the Patients' Perspective**
Deborah Kimball, MS; Sean O. Henderson, MD
USC/Keck School of Medicine
- 26 Use of Alternative Airway Devices in an Academic Emergency Department**
John C. Sakles, MD; Ross B. Rodgers, MD; David J. Olvera
University of Arizona
- 27 Diagnosis and Treatment of Acute Dental Pain in the Emergency Department**
Aaron Lessen, MD; Billie Winegard, MPH; Michael McLaughlin, MD; Daniel Beskind, MD
University of Arizona
- 28 Resident Shift-Hour Productivity: A New Method for Measuring Resident Productivity that Accounts for Variations in Hourly Patient Volumes**
James C. McClay, MD; Ronald H. Carson, BA; Michael Wadman, MD
University of Nebraska Medical Center; The Nebraska Medical Center
- 29 Incidental Ultrasound Findings of ED Patients with Biliary Symptoms**
Wesley Zeger, DO; JR Realing, MD; Paul Tran, MD; Samantha Morris
University of Nebraska Medical Center
- 30 Cocaine Use and Risk of Death in Sickle Cell Patients Who Frequently Use the Emergency Department**
Martha L. Neighbor, MD; Jaime Raul Antuna; Michael A. Kohn, MD
University of California, San Francisco
- 31 Study in the Emergency Department of Atypical Tranquilizer Efficacy (SEDATE)**
Gregory D. Folkert, MD; Sara Nikravan, MD; Michael E. Mullins, MD
Washington University
- 32 Emergency Medicine Physician Practice and Perception of Opioid-Seeking Patients**
Lisa Chan, MD; Billie Winegard, MPH; Kevin Reilly, MD; Brooke Rosonke, MD; Emily Osborn, MD
University of Arizona
- 33 Prevalence of a Positive Emergency Department Septic Screen Differs between Respiratory Syncytial Virus and Non-Respiratory Syncytial Virus Bronchiolitis**
Christopher B. Chee, MS3; Paul Walsh, MD; Kian Azimian, MD; Stephen J. Rothenberg, PhD
Kern Medical Center; David Geffen School of Medicine at UCLA; Center of Research in Population Health, National Institute of Public Health, Cuernavaca, Mexico

AS A PHYSICIAN.....

.....ARE MY ASSETS PROTECTED?

Call the Law Offices of Todd M. Litman
Specializing in Estate and Business Planning

Tustin
714-545-4640
www.ToddLitman.com

FELLOWSHIP 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 a Clinical Instructor- Research Fellow for July 2009. UC Irvine Medical Center is a Level I Trauma center with 2200 trauma 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
101 The City Dr, Route 128-01
Orange, CA 92868

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

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

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:

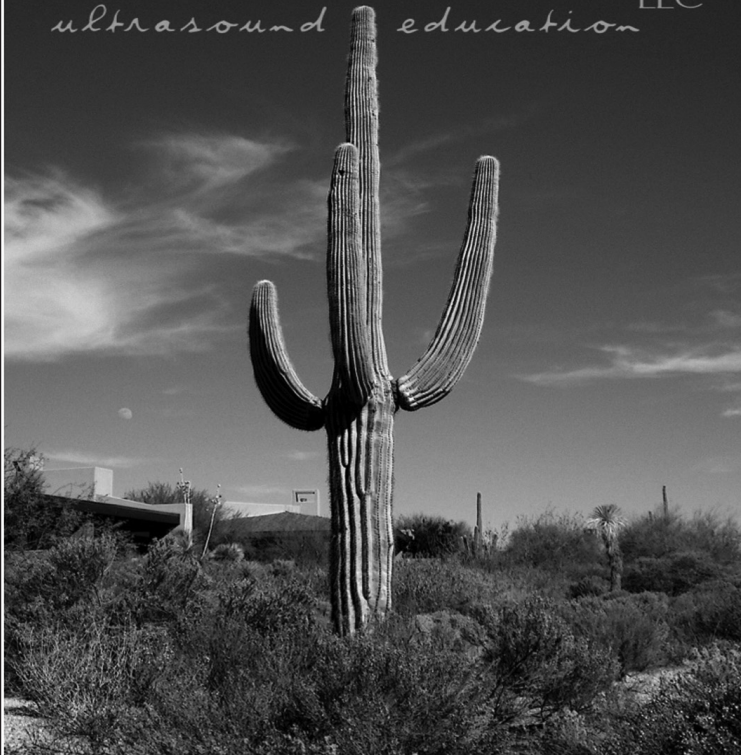
Amin Antoine N. Kazzi, MD, FAAEM
Chief of Service & Medical Director, Emergency Department
AUB Faculty of Medicine and Medical Center
American University of Beirut
P.O.Box 11-0236 / Medical Dean's Office
Riad El-Solh / Beirut 1107 2020, Lebanon

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

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

SONORAN ULTRASOUND LLC

ultrasound education



SonoranUltrasound.com • ultrasoundeducation@gmail.com • 949 - 226 - 8548

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.

Category I AMA CME. 1-Day courses qualify for 6.5 units while 2-Day courses qualify for 13 units.

IMPLEMENTATION

Every possible obstacle encountered by the non-radiologist for utilizing ultrasound is addressed and overcome upon completion of our program. Each course contains clear pathways for credentialing, quality assurance, documentation, and billing requirements.

PATHOLOGY

Video-clips of positive scans in all categories are integrated throughout course



I Went Flying This Weekend.

The Air Force Reserve is in need of flight surgeons and medical personnel who can handle critical care aeromedical transport. The pay is competitive and the benefits are excellent, but those are not the motivators that get people to voluntarily spend their days off working.

The Air Force Reserve performs about 65 percent of aeromedical evacuations assigned to the Air Force, and we need your help to keep up this pace. You won't have to move; you can continue your civilian career and, as a physician or nurse, you will join as an officer. Please take this opportunity to contribute to your country and to those who are doing the same.



AIR FORCE RESERVE

800-257-1212 • AFReserve.com/healthcare

Save the Date!

20th Annual Southern California Emergency Medicine Symposium

Wednesday, November 5th, 2008

Doubletree Hotel—Anaheim/Orange County
100 The City Drive S., Orange, CA 92868

- State-of-the-Art Lectures
- Lifelong Learning Self-Assessment Review
- Exhibitors
- Luncheon and Closing Reception
- This activity has been approved for
AMA-PRA Category 1 Credit

Jointly Sponsored By:



University of California, Irvine School of
Medicine, Department of Emergency Medicine

714-456-5239



SOCIETY OF
ORANGE COUNTY EMERGENCY PHYSICIANS

<http://www.socep.us>

SAVE THE DATE!

CaIAAEM
Presents

"Children Are Not Little Adults!"

PEDIATRIC EMERGENCIES

1 March 2009

8:00AM to 5:30PM

Sheraton Phoenix Downtown

Watch for Details @ www.aaem.org



“What I learned at Merage, they didn’t teach me in medical school.”

As a physician, you’re an expert in your field. But can the same be said for navigating health care and running a successful emergency room?

Our graduates will tell you that the Health Care Executive MBA program at the prestigious UC Irvine Paul Merage School of Business was invaluable in transforming them into savvy business people.

Over just one long-weekend a month you’ll:

- Network with top professionals in your field
- Gain a top-ranked business education
- Become an expert on running your practice

Our dynamic 24-month program will give you the business education you need to be the decision maker. Contact us today for a personal consultation or register online to attend an upcoming information session.

949.824.0561
hcemba@uci.edu
merage.uci.edu/HealthCareExecutiveMBA

Romilla Batra, MD, HCEMBA Class of 2009
Associate Program Director, Internal Residency Program
Medical Director, Internal Medicine Practice
Associate Professor of Medicine
University of California, Irvine

UCIRVINE | THE PAUL MERAGE
SCHOOL OF BUSINESS
HEALTH CARE EXECUTIVE MBA PROGRAM



909-949-0600



Visit the Exclusive Websites for Our Listings, or Visit Us at www.theAhmedTeam.com!

Our Team Represents...

33 years of combined service in the Real Estate Market
Graduate of Realtors Institute
Certified Residential Specialist
Certified Relocation Specialist
26 Step Success Program to strategically market your home
A Business Model that has an 88% referral rate attesting to its effectiveness.

We maintain a disciplined organized and detailed orientated business that is committed to personal care, superior results and bettering the community. We will never become complacent and will remain loyal to ideals of justice honesty hard work and success through giving of ourselves to others.

We are a generation of real estate and we are never too busy for any of your referrals.

Your largest IPA is downcoding your claims. Your malpractice insurance costs are through the roof. Your cash collections are down, and operating costs are up. It's your move. What are you going to do?

Call Marina!

Marina Medical

Billing Service, Inc.

*emergency medicine billing specialists
since 1981*

18000 Studebaker Road, Fourth Floor
Cerritos, CA 90703
562.809.3500
www.MarinaBilling.com

- Integrity
- Reputation
- Excellence
- Commitment
- Execution
- Experience

CARE



Ambulance Service, Inc.

PREMIUM BLS AND CCT
TRANSPORT SERVICES

Serving Orange and Los Angeles Counties

1517 W Braden Ct., Orange CA 92868

714-288-3888

www.careambulance.net

Is **SCHEDULING** your **PROVIDERS** an ongoing **HEADACHE?**
We have two solutions!

EPSKED

the venerable auto-scheduler,
scheduling emergency physicians
since 1989

WebSked

our new web-based
scheduling program,
integrates with EPSKED

Visit us at www.bytebloc.com to learn more about our programs,
or to download a free trial of EPSKED.

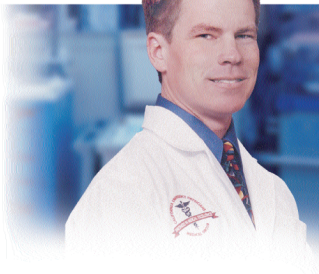
ByteBloc Software, LLC
2152 Rock Glen
Escondido, California 92026
USA
760.510.9900
service@bytebloc.com

ByteBloc
SOFTWARE



CEP — the premier democratic emergency physician partnership that has withstood the test of time — now with opportunities in four states.

CALIFORNIA • OREGON • GEORGIA • ARIZONA



When I joined CEP, I was looking for a stimulating practice environment with a group that values its partners as a long term investment. What I found was that CEP not only endeavors to treat partners with all of the fairness of a democratic group, but that opportunities to contribute to our specialty exist in a way that only a group of this size could offer. I am proud to be associated with two groups dedicated to integrity and innovation in emergency medicine—AAEM and CEP.

—Bret Ginther, MD, FAAEM
CEP Partner

For more information contact
John Gravette, **800-842-2619**
or visit our website at **www.cep.com**

CEP  **America**
CALIFORNIA EMERGENCY PHYSICIANS

The Lifestyle You Want



MedAmerica
PHYSICIAN PRACTICE PARTNERS

Do you wish you were able to spend more time enjoying life?

MedAmerica delivers just that!

For over 30 years, we have offered systems and solutions for the unique management and financial needs of an emergency medicine practice. Our proven expertise in the business of emergency medicine has enabled physician groups to operate very successfully in an increasingly complex healthcare world.

MEDAMERICA: LIFESTYLE SOLUTIONS

www.medamerica.com
hays@medamerica.com
800-476-1504

© 2006 MedAmerica, Inc.