WestJEM

Volume XV, Number 2, March 2014 Open Access at www.westjem.com ISSN 1936-900X

Western Journal of Emergency Medicine:

Integrating Emergency Care with Population Health

DIAGNOSTIC ACUMEN

Contents continued on page ii

CALIFORNIA ACEP
AMERICAN COLLEGE OF EMERGENCY PHYSICIANS

ACOEP
The American College of Osteopathic Emergency Physicians

SCHOOL OF MEDICINE
UNIVERSITY of CALIFORNIA · IRVINE
Discover · Teach · Heal

CAL/AAEM
ACADEMY OF EMERGENCY MEDICINE
California Chapter

A Peer-Reviewed, International Professional Journal

LIKE US ON FACEBOOK

FACEBOOK.COM/CALIFORNIAACEP

FOLLOW US ON TWITTER

@ CALIFORNIAACEP

CALIFORNIA ACEP

AMERICAN COLLEGE OF EMERGENCY PHYSICIANS

CALIFORNIAACEP.ORG

Western Journal *of* Emergency Medicine:

Integrating Emergency Care with Population Health

Official Journal of the California Chapter of the American College of Emergency Physicians, the America College of Osteopathic Emergency Physicians, and the California Chapter of the American Academy of Emergency Medicine

Publisher
Department of Emergency Medicine, University of California, Irvine

School of Medicine
University of California • Irvine
Discover • Teach • Heal

Available in Pub Med, Pub Med Central, CINAHL, SCOPUS, Google Scholar, eScholarship, Melvyl, Directory of Open Access Journals, Medscape and MDLinx Emergency Med.

*West*JEM, 101 The City Drive, Rt. 128-01, Orange, CA 92868-3201: Office (714) 456-6389; Email: Editor@westjem.org

Western Journal *of* Emergency Medicine:

Integrating Emergency Care with Population Health

JOURNAL FOCUS

Emergency medicine is a specialty which closely reflects societal challenges and consequences of public policy decisions. The emergency department specifically deals with social injustice, health and economic disparities, violence, substance abuse, and disaster preparedness and response. This journal focuses on how emergency care affects the health of the community and population, and conversely, how these societal challenges affect the composition of the patient population who seek care in the emergency department. The development of better systems to provide emergency care, including technology solutions, is critical to enhancing population health.

Table of Contents

continued

Policies for peer review, author instructions, conflicts of interest and human and animal subjects protections can be found online at www.westjem.com.

Western Journal *of* Emergency Medicine:
Integrating Emergency Care with Population Health

Table of Contents
continued

Western Journal *of* Emergency Medicine:

Integrating Emergency Care with Population Health

This open access publication would not be possible without the generous and continual support of our society, chapter, and department sponsors.

Professional Society Sponsors

American College of Osteopathic Emergency Physicians

California Chapter of the American Academy of Emergency Medicine

California Chapter of the American College of Emergency Physicians

Professional Chapter Sponsors

Florida Chapter of the American Academy of Emergency Medicine

Nevada Chapter of the American Academy of Emergency Medicine

Missouri Chapter of the American Academy of Emergency Medicine

Tennessee Chapter of the American Academy of Emergency Medicine

Uniformed Services Chapter of the American Academy of Emergency Medicine

Virginia State Chapter of the American Academy of Emergency Medicine

Academic Department Sponsors

Advocate Christ Medical Center
Oak Lawn, IL

American University of Beirut
Beirut, Lebanon

Baylor College of Medicine
Houston, TX

Baystate Medical Center/ Tufts University
Springfield, MA

Boston Medical Center
Boston, MA

Carl R. Darnall Medical Center
Fort Hood, TX

University of Colorado & Denver Health
Denver, CO

Eastern Virginia Medical School
Norfolk, VA

Emory University
Atlanta, GA

Florida Hospital Medical Center
Orlando, FL

George Washington University
Washington, DC

Good Samaritan Hospital Medical Center
West Islip, NY

Henry Ford Medical Center
Detroit, MI

INTEGRIS Health
Oklahoma City, OK

Kaweah Delta Healthcare District
Visalia, CA

Kennedy University Hospitals*
Turnersville, NJ

Kern Medical Center
Bakersfield, CA

Lehigh Valley Hospital and Health Network
Allentown, PA

Madigan Army Medical Center
Tacoma, WA

Maimonides Medical Center*
Brooklyn, NY

Maricopa Medical Center
Phoenix, AZ

Massachusetts General Hospital
Boston, MA

Mount Sinai Medical Center
Miami Beach, FL

New York Medical College/NY Metropolitan Center
New York, NY

North Shore University Hospital
Manhasset, NY

Regions Hospital/ Health Partners Institute for Education and Research
St. Paul, MN

Resurrection Medical Center*
Chicago, IL

Robert Wood Johnson Hospital*
New Brunswick, NJ

Southern Illinois University
Carbondale, IL

San Diego Medical Center
San Diego, CA

Stanford University
Palo Alto, CA

Temple University
Philadelphia, PA

University of Arizona
Tucson, AZ

University of California Davis*
Davis, CA

University of California Irvine*
Orange, CA

University of California Los Angeles
Los Angeles, CA

University of California San Diego
La Jolla, CA

University of California San Francisco
San Francisco, CA

University of California San Francisco Fresno
Fresno, CA

University of Florida
Jacksonville, FL

University of Illinois at Chicago
Chicago, IL

University of Maryland
College Park, MD

University of Nevada
Las Vegas, NV

University of Southern California
Los Angeles, CA

University of Texas, Houston
Houston, TX

University of Utah
Salt Lake City, UT

Upstate Medical Center
Syracuse, NY

**denotes department & residency sponsor*

International Society Partners

Sociedad Argentina de Emergencias

Sociedad Chileno Medicina Urgencia

Thai Association for Emergency Medicine

Emergency Medicine Association of Turkey

Become a *West*JEM departmental sponsor, waive article processing fees, receive print/electronic copies, and free CME advertisement space at www.calaaem.org/westjem or contact:

Marcia Blackman
Cal/AAEM: WestJEM
Phone: (800) 884-2236
MBlackman@aaem.org

or

Shahram Lotfipour, MD, MPH
Managing Associate Editor
Phone: (714) 456-2326
Shahram.Lotfipour@uci.edu

Splenic Laceration and Pulmonary Contusion Injury From Bean Bag Weapon

Amar Patel, MD*
Shannon Toohey, MD†
Megan Boysen-Osborn, MD†

* Pennsylvania State University, College of Medicine, Hershey, Pennsylvania
† University of California Irvine Medical Center, Department of Emergency Medicine, Orange, California

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted October 3, 2013; Accepted October 21, 2013
Electronically published February 19, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.10.19900
[West J Emerg Med. 2014;15(2):118–119.]

CASE REPORT

A 58-year-old male with schizophrenia presented to the emergency department after being shot by law enforcement with two bean bag rounds. He was shot once in the upper abdomen. He reportedly turned and was shot again in the left flank; both shots were at an estimated distance of twenty feet. On arrival, the patient's blood pressure was 84/52 and heart rate was 121 beats per minute. The patient was agitated and given 5mg intramuscular (IM) haloperidol, 50 mg IM diphenhydramine, and 1 mg intravenous lorazepam. The patient's chest and back exam revealed a 2.5 cm abrasion and contusion to the left flank (Figure 1). His lung examination was normal. His abdomen was thin, soft, and non-tender. He had another 2.5 cm circular abrasion to the upper abdomen/epigastrum (Figure 2). Initial hemoglobin was 13.4 g/dL. We performed Focused Assessment with Sonography for Trauma examination, which demonstrated free fluid in Morison's pouch (Figure 3) and the suprapubic window. The splenorenal space did not show any fluid.

Computed tomography of the abdomen and pelvis with intravenous contrast revealed the following: a moderately displaced fracture of the left 11th rib, buckle fracture of the left 12th rib, trace left anterior inferior pneumothorax, American Association for the Surgery of Trauma grade 4 splenic laceration (Figure 4) with moderate perisplenic hematoma, mild perihepatic hematoma, and anterior abdominal wall/rectus sheath hematoma.

DISCUSSION

The use of bean bags as a less lethal technology to

Figure 1. Left flank abrasion/hematoma from bean bag round.

Figure 2. Abdominal wall abrasion/hematoma from bean bag round.

Figure 3. Focused Assessment with Sonography examination demonstrating free fluid in Morison's pouch.

Figure 4. Computed tomography showing grade IV splenic laceration.

apprehend suspects has steadily increased since their introduction in 1994.[1] Even though fatalities are uncommon, morbidity and mortality from bean bag weapons was predicted by United States Army studies in 1974, which showed that the impact was significant enough to cause internal organ injuries.[2]

Previous case series have demonstrated a variety of injuries caused by these less lethal weapons, including: epidural hematoma[3], penetrating abdominal and thoracic wounds[4,5], hemothorax/pneumothorax, splenic laceration, testicular fracture, globe rupture, orbital fracture, compartment syndrome, and cardiac contusion, among others.[1] There have been at least three fatalities reported in the literature and news.[1,6,7] The majority of patients injured by bean bag rounds are those with psychiatric disorders.[1] Patients often present with an underwhelming physical examination.[1] The emergency physician should have a high index of suspicion for serious injuries in patients with bean bag injuries, even in the absence of significant physical examination findings.

Address for Correspondence: Megan Boysen-Osborn, MD. Department of Emergency Medicine, University of California Medical Center, 101 The City Drive, Rte. 128-01, Orange, CA 92868. Email: mbo@uci.edu.

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

REFERENCES

1. Brito D, Challoner KR, Sehgal A, et al. The Injury Pattern of a New Law Enforcement Weapon: The Police Bean Bag. Ann Emerg Med. 2001. 38:383-390
2. Shank EB, Thein BK, Campbell D, et al. "A comparison of various less lethal projectiles," LWL TR-74-79 US Army Land Warfare Laboratory, Aberdeen Proving Ground, MD, June 1974.
3. Drazin D, Shirzadi A, Hanna G, et al. Epidural hematoma and surgical evacuation from a bean bag weapon. Am Surg 2012. 78(1):E33-35.
4. Charles A, Asensio J, Forno W, et al. Penetrating bean bag injury: Intrathoracic complication of a nonlethal weapon. J Trauma 2002. 53:997-1000.
5. Olivas T, Jones B, Canulla M. Abdominal wall penetration by a police "bean bag." Am Surg 2001. 67:407-409.
6. Autopsy: Bean bag rounds fired by police killed Park Forest man, 95. Chicago Tribune, 7/28/2013. Available at: http://articles.chicagotribune.com/2013-07-28/news/chi-autopsy-bean-bag-rounds-fired-by-police-killed-park-forest-man-95-20130728_1_park-forest-man-metal-cane-bean-bag-rounds, Last accessed September 18, 2013.
7. Columbus officers cleared in bean bag death. Athens Banner-Herald, 7/27/2005. Available at: http://onlineathens.com/stories/072705/new_20050727040.shtml Last accessed September 18, 2013.

High-pressure Injection Injury with Molten Aluminum

Joseph McCarthy, DO
Christopher Trigger, MD

Lakeland Regional Medical Center, Department of Emergency Medicine, St Joseph, Michigan

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted September 12, 2013; Revision received October 5, 2013; Accepted October 28, 2013
Electronically published February 19, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.10.19443
[West J Emerg Med. 2014;15(2):120–121.]

INTRODUCTION

A previously healthy 65-year-old male presented to the emergency department complaining of a burn to his right forearm. Just prior to arrival the patient was working at a tool and die company casting products with molten aluminum where he inadvertently caused his injury. Physical examination revealed a burn to the right forearm of varying degrees of severity. A non-painful charred central area was observed with surrounding erythema and swelling (Figure 1). Patient had full motor strength of his wrist and elbow. He also had normal neuro-vascular status distal to the injury. A radiograph was performed to assess the depth of the injury (Figure 2).

DISCUSSION

High-pressure injection injury with molten aluminum.

High-pressure injection injuries typically look benign with a small site of entry, often initially painless and numb, but can quickly become painful with severe swelling that can lead to compartment syndrome and gangrene.[1] Commonly injected materials include paint, grease, and hydraulic fluid with the risk of amputation being greater than 40%.[2] Due to the serious nature of all high-pressure injections, urgent surgical consultation with likely debridement is required.

Similarly, molten metal thermal burns are often small in gross appearance, at least when compared to other thermal burns. On average, 2.3% of mean body surface is involved in the burn, but typically full thickness.[3] The severity of molten metal thermal burns can easily be overlooked and the delay in proper treatment often leads to extended hospital stays and

Figure 1. Right forearm with 2nd, 3rd, and 4th degree burns.

Figure 2. Right forearm radiograph showing radiopaque material in the subcutaneous tissues.

increased length of time to return to work when compared to earlier referral to a surgeon.[3]

Whether a high-pressure injection injury, molten metal thermal burn, or combination of both, urgent consultation to a surgeon is warranted. The patient will likely require irrigation and debridement in the operating room with possible amputation depending on the severity. The patient in this case underwent debridement and split-thickness skin grafting by a plastic surgeon that specializes in burn reconstruction. Patient had no loss of function of his affected arm and had a full recovery.

Address for Correspondence: Joseph McCarthy, DO, Lakeland Regional Medical Center, Department of Emergency Medicine, 1234 Napier Avenue, St. Joseph, MI 49085. Email: jmccarthy@lakelandregional.org.

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

REFERENCES

1. Mirzayan R, Holtom PD, Schnall SB, et al. Culture results and amputation rates in high-pressure paint gun injuries of the hand. *Orthopedics*. 2001;24(6):587–589.
2. Hogan CJ, Ruland RT. High-pressure injection injuries to the upper extremity: A review of the literature. *J Orthop Trauma*. 2006;20(7):503-511.
3. Margulies DR, Navarro RA, Kahn AM. Molten metal burns: Early treatment improves outcome. *Am Surg.* 1998;64(10):947-949.

Diagnosis of Fournier's Gangrene on Bedside Ultrasound

Christopher Coyne, MD*
Thomas Mailhot, MD*
Phillips Perera, MD†

* Los Angeles County + USC Medical Center, Department of Emergency Medicine, Los Angeles, California
† Stanford University, Division of Emergency Medicine, Stanford, California

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted September 6, 2013; Revision received September 16, 2013; Accepted October 28, 2013
Electronically published February 28, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.10.19476
[West J Emerg Med. 2014;15(2):122.]

A previously healthy 48 year-old male presented to the hospital with a 4-week history of "pimples" on his scrotum. This condition had progressively worsened, resulting in increased pain, swelling and redness to the genital region and buttocks. On physical examination, the patient was persistently tachycardic. The scrotum, penis, perineum and left buttock were erythematous, swollen and markedly tender to palpation. Furthermore, the patient's suprapubic region contained an area of necrotic tissue.

As part of the initial assessment, the patient received a bedside ultrasound (US) that demonstrated marked thickening of the scrotal fascia with edema, as well as discrete areas of subcutaneous gas (Video). Based on these ultrasound findings, in conjunction with the clinical evaluation, the patient was diagnosed with Fournier's Gangrene and intravenous antibiotics were started. He was then emergently transferred to the operating room without further advanced imaging, where he received aggressive surgical therapy with a good outcome.

Fournier's Gangrene is defined as "an infective necrotizing fasciitis of the perineal, genital or perianal regions".[1] The bacterial etiology is typically a synergistic polymicrobial infection, defined as a form of type 1 necrotizing fasciitis.[2-3] This case demonstrates that Fournier's Gangrene remains a clinical diagnosis and that while many patients receive confirmatory advanced imaging with computed tomography and magnetic resonance imaging, an expedited bedside US can allow for the diagnostic certainty to proceed rapidly with appropriate therapy.[4-5]

Video. Ultrasound of scrotum demonstrating thickening of scrotal fascia.

Address for Correspondence: Christopher Coyne, MD, Los Angeles County + USC Medical Center, Department of Emergency Medicine, 2051 Marengo Street, Inpatient Tower-Room C1A100, Los Angeles, CA 90033. Email: coyne.usc@gmail.com.

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

REFERENCES

1. Smith GL, Bunker CB, Dinneen MD. Fournier's gangrene. *Br J Urol*. 1998;81:347-355
2. Rotstein OD, Pruett TL, Simmons RL. Mechanisms of microbial synergy in polymicrobial surgical infections. *RevInfect Dis*. 1985;7(2):151-170.
3. Ustin J, Malangoni M. Necrotizing soft tissue infections. *Crit Care Med*. 2011;39:2156-2162.
4. Laucks SS 2nd. Fournier's gangrene. *Surg Clin of North Am*. 1994;74:1339-1352.
5. Kube E, Stawicki S, Bahner D. Ultrasound in the diagnosis of Fournier's gangrene. *Int J Crit Ill Inj Sci*. 2012;2:104-106

Cardiac Sarcoma: Unusual Cause of Intracardiac Contrast Filling Defect

Nathan J. Cleveland, MD, MS
Melissa Beckmann, MD

Department of Emergency Medicine, University of Nevada School of Medicine, Las Vegas, Nevada

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted November 25, 2013; Accepted December 10, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.20576
[West J Emerg Med. 2014;15(2):123–124.]

A 28-year-old female was transferred to the emergency department from her physician's office for further evaluation of tachycardia. She was being seen for a recent illness which included nausea, vomiting, diarrhea and fevers. The patient endorsed fatigue, dyspnea on exertion, and extremity edema. She had no chest pain or cough. Exam revealed a pale, fatigued, mildly ill-appearing female with bilateral lower extremity edema and diminished breath sounds on the right. Chest radiograph revealed a large right pleural effusion (Figure 1). Computed tomographic angiography of the chest was performed (Figure 2).

DIAGNOSIS:

Cardiac Sarcoma. This is a rare form of primary invasive malignant tumor of the heart. Most cardiac tumors are secondary.[1] Of primary tumors, benign myxomas are the most common.[2] About 25% of primary cardiac tumors are malignant with 95% of those being sarcomas.[3] Sarcomas often invade into adjacent structures including the myocardium, valves, vena cava and pericardium.[3]

The differential diagnosis for intracardiac contrast-filling defects includes thrombus, tumor and vegetations from endocarditis.[4] Echocardiography, angiocardiography and magnetic resonance imaging may also be helpful in establishing a diagnosis.[1,3] Large intracardiac tumors frequently result in inflow/outflow obstruction which can precipitate heart failure.[2]

Treatment of this condition is primarily operative.[5] Heart transplantation (including autotransplantation) may even be necessary.[5] Cardiac sarcomas often recur after resection. Post-surgical chemotherapy and radiation are of limited benefit.[6]

Our patient underwent surgical resection of the tumor, replacement of the tricuspid valve and reconstruction of the vena cava. She survived for several weeks after surgery but eventually succumbed to her illness.

Address for Correspondence: Nathan J. Cleveland, MD, MS, Department of Emergency Medicine, University of Nevada School of Medicine, Las Vegas, Nevada. Email: nathanjcleveland@gmail.com.

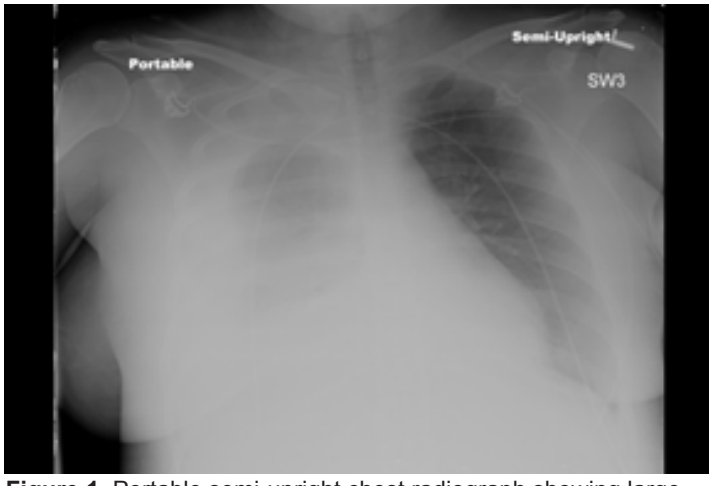


Figure 1. Portable semi-upright chest radiograph showing large right pleural effusion.

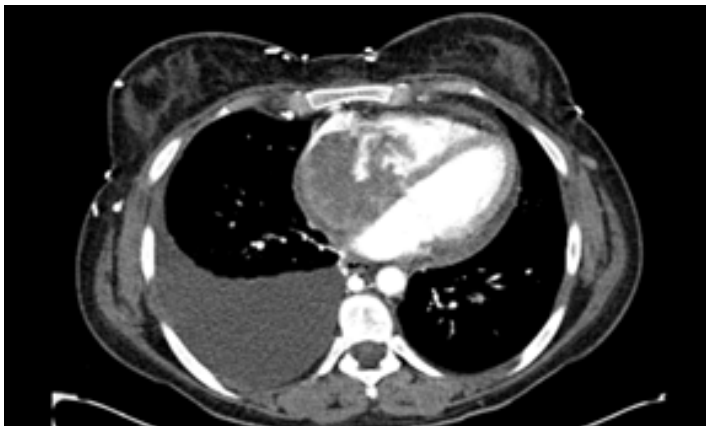


Figure 2. Axial computed tomography angiogram of the chest showing intracardiac contrast-filling defect with an associated right-sided pleural effusion.

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

REFERENCES:

1. Shapiro LM. Cardiac tumors: Diagnosis and management. Heart. 2001;85:218-222.
2. Butany J, Nair V, Naseemuddin A, et al. Cardiac tumours: diagnosis and management. Lancet Onc. 2005;6:219-228.
3. Sheppard MN, Mohiaddin R. Tumors of the heart. Future Cardiol. 2010;6:181-193.
4. Price S. Endocarditis: the complementary roles of CT and echocardiography. Br J Cardiol. 2012;19:7-9.
5. Blackmon SH, Reardon MJ. Surgical Treatment of Primary Cardiac Sarcomas. Tex Heart Inst J. 2009;36:451-452.
6. Putnam JB Jr, Sweeney MS, Colon R, et al. Primary cardiac sarcomas. Ann Thorac Surg 1991;51:906-910.

Lemierre's Syndrome

Jayten Shook, DO
Christopher Trigger, MD
Lakeland Regional Medical Center, Department of Emergency Medicine, St Joseph, Michigan

Supervising Section Editor: Sean Henderson, MD
Submission history: Submitted November 19, 2013; Accepted December 9, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.20418
[West J Emerg Med. 2014;15(2):125–126.]

A 25-year-old woman presented to the emergency department (ED) with 4 days of progressive, left-sided neck pain and swelling. Additional symptoms included sore throat, dysphagia and left otalgia. On presentation she was tachycardic, hypotensive and had an exam notable for granular pharyngitis as well as a large area of nonfluctuant induration and swelling posterior to her left mandibular angle (Figure 1). Diffuse anterior and posterior cervical lymphadenopathy was palpable on the left. She had a white blood cell count of 13.4 with a left shift and no bandemia. A computed tomography (CT) of the neck with intravenous contrast was also performed (Figure 2).

Lemierre's syndrome, also called postanginal septicemia, is a rare but potentially fatal disease characterized by septic thrombophlebitis of the internal jugular vein**.** Affected patients are typically young, otherwise healthy individuals with a recent history of tonsillitis (37%) or pharyngitis (30%) followed by severe sepsis.[1-2] Patients often present with complaints of sore throat, neck pain or neck mass as well as bone and joint pain related to septic emboli. The primary infection progresses to abscess formation within 1-3 weeks, facilitating invasion of the parapharyngeal space and internal jugular vein, leading to septic thrombophlebitis. The disease is often complicated by septic emboli traveling to the lungs and large joints. Isolated organisms include anaerobic pathogens, with *Fusobacterium necrophorum* being the most common.[2]

Early recognition and treatment are crucial as the mortality rate in untreated individuals approaches 17%. Antibiotic treatment should include intravenous anaerobic coverage with metronidazole or clindamycin which can be transitioned to oral with minimum treatment duration of 3 weeks. Anticoagulation in Lemierre's syndrome remains controversial but should be considered if thrombosis extension is noted clinically.[2] The imaging modality of choice is a contrast enhanced CT of the neck. Radiologic findings include intraluminal venous filling defects and peripheral rim enhancement of the involved segment which can measure 10-20 cm in length and most frequently includes complete occlusion.[3]

Figure 1. Large mass on left lateral neck.

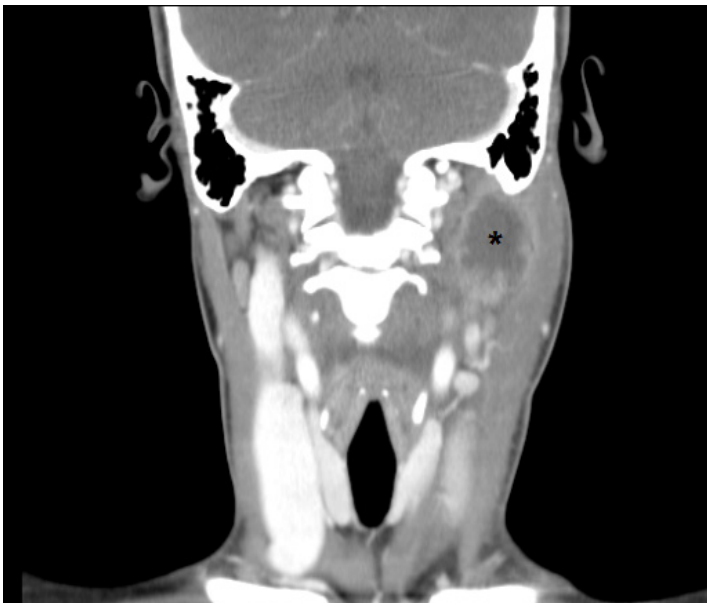


Figure 2. Coronal computed tomography of the neck with contrast demonstrating a 3.3 cm loculated abscess (*) in the left posterior triangle with adjacent cervical adenitis and left internal jugular vein occlusion.

Address for Correspondence: Jayten Shook, DO, Lakeland Regional Medical Center, Department of Emergency medicine, 1234 Napier Ave, St Joseph, MI 49085. Email: jshook@lakelandregional.org.

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

REFERENCES

1. Karkos PD, Asrani S, Karkos CD, et al. Lemierre's syndrome: A systematic review. *Laryngoscope*. 2009;119:1552–1559.
2. Johannesen K, Bødtger U, Heltberg O. Lemierre's syndrome: the forgotten disease. *J Thromb Thrombolysis.* 2013 May 18.
3. Kim BY, Yoon DY, Kim HC, et al. Thrombophlebitis of the internal jugular vein (Lemierre syndrome): clinical and CT findings. *Acta Radiol*. 2013;54(6):622-627.

Phenytoin Toxicity from Cocaine Adulteration

Carlos J. Roldan, MD

Department of Emergency Medicine, University of Texas Health Science Center at Houston, Houston, Texas

Supervising Section Editor: Rick McPheeters, DO
Submission history: Submitted October 14, 2013; Revision received November 13, 2013; Accepted December 11, 2013
Electronically published February 28, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.20010

The use of phenytoin (PHT) as a cocaine adulterant was reported decades ago;that practice is still current. Ironically PHT has also been used for the treatment of cocaine dependence. A drug smuggler developed PHT toxicity after swallowing several rocks of crack. We investigated the current trends of PHT as a cocaine adulterant and its toxicological implications. We also reviewed the clinical use of PTH in relation to cocaine. The use of PHT as cocaine cut is a current practice. This may affect the clinical manifestations and the management of the cocaine-related visits to the emergency department. West J Emerg Med. 2014;15(2):127–130.]

INTRODUCTION

Cocaine is a powerful stimulant of the nervous system and is highly addictive. Non-medical cocaine is commonly diluted (or "cut") with adulterants or diluents that mimic the drug's systemic pharmacologic properties, simulate its local anesthetic effect, or resemble the physical appearance of cocaine. Cocaine dealers incorporate these additives to enhance the total volume of their stock and therefore to increase profits. Consequently, the purity of the street cocaine is variable. Various substances, including phenytoin (PHT), have been reported as cocaine adulterants. In the early 1990s, Katz and colleagues reported several patients with PHT toxicity after smoking crack cocaine that was cut with PHT capsules .[1] The management of cocaine-related emergencies is well described in the literature. When cocaine is adulterated, clinicians must be vigilant of the unsuspected exposures from the additives; the clinical implications and its complications can be significant. Many past and present cocaine adulterants, such as Levamizole, are otherwise legitimate medications that are used to enhance or alter cocaine's pharmacological traits. This alteration of cocaine's pharmacology makes these drugs an interesting class of adulterants.[2] Regarding PHT, some have suggested the possibility that it augments cocaine's effects on the nervous system; others speculate that PHT's similarities in appearance to cocaine just make it an attractive blender.

CASE REPORT

In Houston, Texas, a 26-year-old male drug smuggler who swallowed several rocks of crack cocaine to conceal evidence while being apprehended by police was taken to our emergency department (ED) for medical clearance. Initially, he was asymptomatic, and his vital signs and physical exam were normal. After 2 hours of observation, he manifested clinical signs of cocaine toxicity including anxiety, pallor, diaphoresis, agitation, tachycardia, and hypertension. His vital signs changed; his blood pressure was 190/110 mmHg, heart rate 120 bpm, respiratory rate 22 breaths per minute, and temperature 99.6 F. His physical exam was otherwise unchanged. He was given intravenous benzodiazepine boluses and was admitted to a medical intensive care unit. Subsequently he developed confusion, truncal ataxia, diplopia, slurred speech, and multidirectional nystagmus; these changes were not explained by his pharmacological management. Additional examinations, including computer tomography of the brain , diagnostic lumbar puncture and extensive blood testing, were negative for any abnormalities. A neurology consult revealed uncertain diagnosis. A PHT level was obtained at the recommendation of the poison control center. The patient's PHT level was 48 mcg/mL (therapeutic range: 10 to 20 mcg/mL). The patient had no history of seizures or records of prescribed PHT. Supportive care was continued, and eventually the patient was discharged with no further complaints 72 hours after MICU admission. To challenge the uniqueness of the use of PHT as cocaine adulterant a random blood screen of PHT levels was performed in 10 patients presenting with complaints related to cocaine use to an academic ED in New York City (7 were seen for chest pain, 1 for suicidal ideation, 1 for new-onset seizures, and 1 for headache associated with uncontrolled hypertension). One patient with chest pain had a PHT level of 4 mcg/mL.

The patient presenting with headache had a level of 5 mcg/mL. None of the patients screened had any history of seizure disorders or medical indications for the use of PHT. The patients with positive screening tests were unaware of cocaine adulteration.

DISCUSSION

The history of PHT as a cocaine adulterant is still unclear. The isolation of cocaine by Albert Niemann in 1859 facilitated storage and experimentation by scientists and entrepreneurs. Soon, widespread unrestricted access and increasing reports of fatalities among cocaine consumers became a matter of public health. In response, the United States government passed the Harrison Narcotics Act of 1914 banning nonprescription use of cocaine-containing products.[3] This act resulted in the end of the first American cocaine epidemic. In the 1970s, the introduction of crack ushered in the second epidemic of American cocaine use. Crack then became the preferred presentation of cocaine among young and low-income populations. In 2005, the United States (U.S.) government reported that retail-level prices for cocaine had increased and purity had decreased. Unsurprisingly, many substances have been listed as cocaine adulterants. This list includes local anesthetics (i.e., procaine, lidocaine, and tetracaine), other stimulants (i.e., amphetamine, caffeine, methylphenidate, and strychnine), lysergic acid diethylamide, phencyclidine, heroin, marijuana, and hashish. Similarly, quinine, talc (i.e., magnesium silicate), ascorbic acid, boric acid, chalk, laundry detergent, meat tenderizer, laxatives, plaster of Paris, cornstarch, and lactose are also known cocaine diluents.[4] Cocaine has been part of U.S. drug-use history for more than a century, whereas PHT was introduced as an effective drug for the treatment of epilepsy only after 1940.[5] The use of PHT as a cocaine adulterant was first documented in the 1990s.[1] Since then, no other reports have been published.

Assessment of Prevalence

In the U.S., information related to collection and analysis of drugs with legitimate medical use is maintained by the U,S. Drug Enforcement Administration, Office of Diversion Control. The National Forensic Laboratory Information System (NFLIS) monitors illicit drug use and trafficking, including the diversion of legally manufactured drugs into illegal markets, by systematically collecting results from drug analyses conducted by state and local forensic laboratories. These laboratories analyze controlled and non-controlled substances secured in law enforcement operations across the country. The NFLIS annual reports include information on specific substances and the characteristics of drug evidence, such as purity, quantity, and drug combinations. Since the inception of the NFLIS in September 1997, an estimated total of 1,660,216 drug reports were submitted to state and local forensic laboratories in the U.S. Regional laboratory analyses of confiscated cocaine showed variability in cocaine purity from 48% (2008) being the lowest and 75% (2006) the highest (mean value 60%). Many of the cocaine-related combinations included excipients used to dilute cocaine. These included non-controlled substances such as procaine, inositol, caffeine, boric acid, benzocaine, and lactose. PHT was not listed as a cocaine adulterant; it is unknown if it was not found or not tested for. The specific locations or the amount of drugs confiscated and analyzed were not specified. Another source for drug information and toxicity statistics is the National Poison Data System (NDPS), formerly performed by the Toxic Exposure Surveillance System (TESS) until 2004, in which no role of PHT as cocaine adulterant was found.

Interaction between PHT and Cocaine

When examining the mechanism of action of cocaine and PHT, it is similarly difficult to elucidate a common link between the 2 drugs that could result in an augmented high. Cocaine blocks the recovery of dopamine at the adrenergic neuronal junction, which leads to increased dopamine receptor activation at the neuronal synapse. In addition to dopamine, cocaine also inhibits norepinephrine and serotonin reuptake, which causes an influx of sodium and an efflux of potassium and results in an action potential at the synapse and subsequent nervous system stimulation .[6] Conversely, PHT can cause central nervous system depression. PHT restricts the repetitive firing of action potentials -- seen with sustained depolarization -- and slows voltage-activated sodium channel recovery in presynaptic neurons. These effects are responsible for its antiepileptic properties.[7] At toxic levels, oral PHT can induce central nervous system manifestations including nystagmus, ataxia, slurred speech, tremor, lethargy, confusion, and disorientation.[8] No cardiac toxicity develops when ingested orally. Conversely, cocaine toxicity includes central and peripheral nervous system manifestations, such as pallor, tremors, tachycardia, hypertension, increased core temperature, anxiety, paranoia, restlessness, hypervigilance, hallucinations, paranoid delusions, and convulsions. As a result of these opposing pharmacological traits, there is no physiological mechanism to explain an augmentation effect of the mechanism of action of cocaine by PHT.

Drug Metabolism

The metabolism of cocaine occurs mainly in the liver but also involves other pathways. More than 10 cocaine metabolites have been discovered; many retain some of the activity of the parent compound, thereby increasing its toxicity. Ecgonine methyl ester, one of the major metabolites, is formed via cholinesterases in the plasma. A reduction in levels or activity of plasma cholinesterase shifts the metabolism of cocaine toward other toxic metabolites. It has been suggested that PHT may act as an inducing agent for plasma cholinesterase[9] and may therefore promote cocaine clearance. The second major metabolite, benzoyl ecgonine, is formed through spontaneous or enzymatic

hydrolysis of cocaine and may be unaffected by the presence of PHT.[10] Aside from these 2 mechanisms, cocaine is mostly metabolized through the cytochrome P450 enzyme family, more specifically N-demethylation into norcocaine .[11]

Ironically P450 enzyme induction by PHT does not occur with acute exposure but certainly can be a factor in chronic use of cocaine adulterated with PHT or in patients taking PHT for therapeutic purposes, such as cocaine-induced seizures. Likewise, PHT relies predominantly on the P450 system for metabolism and uses many different subgroups of that system. Whereas PHT is a substrate of 2D6 and an inducer of 3A4 (12,13), cocaine is an inhibitor of 2D6 and a substrate of 3A4. It is possible that cocaine could potentially hinder the metabolism of PHT through the inhibition of 2D6, thus increasing the concentration of PHT, but PHT undergoes numerous transformations through the P450 system, which would minimize this effect. PHT could also potentially induce the metabolism of cocaine through the induction of the 3A4 subgroup. However, in general, no metabolic pathway explains the possibility of cocaine high augmentation by PHT. In fact, through examining these pathways, it appears that PHT may enhance the elimination of cocaine.

Clinical Use of PHT in cocaine patients

Metabolic pathways suggest that PHT enhances cocaine breakdown and excretion at an elevated rate. Consequently, it has been tested as a treatment option for cocaine abuse. In one double-blind, placebo-controlled study for the treatment of cocaine abuse, patients treated with PHT had lower rates of cocaine in urine specimens and longer cocaine-free periods. Observations were based on cocaine urinalysis, patients' self-reported use, overall function, reduced craving intensity, and subject retention.[14] Conversely, in a pilot study to determine the effects of PHT on cocaine self-administration in a human laboratory model, PHT altered neither the self-administration nor the effects of cocaine.[15] A systematic review of the use of anticonvulsants, including PHT, to treat cocaine dependence found no significant differences for any of the efficacy measures compared to placebo. In fact, placebo was superior to PHT for fewer side effects.[16] Interestingly, PHT has been proposed as an alternative for the treatment of cocaine dependence and cocaine-related seizures. However, a limited number of small clinical trials were insufficient evidence to support the clinical use of PHT with this purpose.[17,18]

CONCLUSION

The use of PHT as a cocaine adulterant is still a current practice. Due to the impurity of the street cocaine, clinicians should always consider the presence of adulterants in the differential diagnosis of atypical manifestations of a cocaine-related visit. Awareness of the local trends of cocaine adulteration might facilitate the medical management of these patients, thereby avoiding increased cost and improving resources utilization. On the other hand, we need to acknowledge that a limited number of small clinical trials are insufficient evidence to support the clinical use of PHT for the treatment of cocaine dependence.

Address for Correspondence: Carlos J Roldan, MD, Department of Emergency Medicine, University of Texas Health Science Center at Houston, Houston, Texas, 1133 John Freeman Blvd, JJL 4th Floor, Houston, Texas 77030 address. Email: carlos.j.roldan@uth.tmc.edu.

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

REFERENCES

1. Katz A, Hoffman R, Silverman R. Phenytoin toxicity from smoking crack cocaine adulterated with phenytoin. *Ann Emerg Med*. 1993;22:1485-1487.
2. Larocque A, Hoffman RS. Levamisole in cocaine: unexpected news from an old acquaintance. *Clin Toxicol (Phila)*. 2012;50(4):231-241.
3. Spillane JF. *Cocaine: From Medical Marvel to Modern Menace in the United States, 1884-1920*. Baltimore and London: The John Hopkins University Press. 2000;p94.
4. Burnett LB, Roldan CJ, Adler J. Cocaine Toxicity, in Medscape.com Edited 05/2012. Available at: http://emedicine.medscape.com/article/813959.
5. Glauser T, Ben-Menachem E, Bourgeois B, et al. ILAE Treatment Guidelines: Evidence-Based Analysis of Antiepileptic Drug Efficacy and Effectiveness as Initial Monotherapy for Epileptic Seizures and Syndromes. *Epilepsia*. 2006;47(7):1094–1120.
6. O'Brien CP, Hardman JG, Limbird LE, et al. *Goodman and Gillman's The Pharmacological Basis of Therapeutics*. 10th Edition. New York: McGraw-Hill Medical Publishing Division; 2001.
7. Yaari Y, Selzer ME, Pincus JH. Phenytoin: mechanisms of its anticonvulsant action. *Ann Neurol*. 1986;20(2):171-184.
8. Craig S. Phenytoin poisoning. *Neurocritical care*. 2005;3:161-170.
9. Puche E, Garcia M, Garcia de la Serrana H, et al. Probable pseudocholinesterase induction by valproic acid, carbamazepine and phenytoin leading to increased serum aspirin-esterase activity in epileptics. *Int J Clin Pharmacol Res*. 1989;9(5):309-311.
10. Kolbrich EA, Barnes AJ, Gorelick DA, et al. Major and minor metabolites of cocaine in human plasma following controlled subcutaneous cocaine administration. *J Anal Toxicol*. 2006;30(8):501–510.
11. Jatlow P. Cocaine: analysis, pharmacokinetics, and metabolic disposition. *Yale J Biol Med*. 1988;61:105-133.
12. Lexicomp Online. Hudson, OH; c1978-2012 [7/27/2012; cited 2012 July 30]. Available at: https://online.lexi.com/lco/action/home
13. Komatsu T, Yamazaki H, Asahi S, et al. Formation of a dihydroxy metabolite of phenytoin in human liver microsomes/cytosol: roles

of cytochromes P450 2C9, 2C19, and 3A4. *Drug Metab Dispos*. 2000;28(11):1361-1368.

14. Crosby RD, Pearson VL, Eller C, et al. Phenytoin in the treatment of cocaine abuse: a double-blind study. *Clin Pharmacol Ther*.1996;59(4):458-468.
15. Sofuoglu M, Pentel PR, Bliss RL, et al. Effects of phenytoin on cocaine self-administration in humans. *Drug Alcohol Depend*. 1999;53(3):273-275.
16. Minozzi S, Amato L, Davoli M, et al. Anticonvulsants for cocaine dependence. Cochrane Database Syst Rev. 2008; 2:CD006754.
17. De Lima MS, De Oliveira Soares BG, Reisser AA, et al. Pharmacological treatment of cocaine dependence: a systematic review. *Addiction*. 2002;97(8):931-949.
18. Hayase T, Yamamoto Y, Yamamoto K, et al. Comparison of effect of ethanol and anticonvulsants on cardiovascular drug-modified cocaine toxicity. *Pharmacol Biochem Behav*. 2000;67(1):151-159.

The Ethics of the Missing Straw

David Bell, MD, PhD
Eric Katz, MD
Anne Klokow, MD

Maricopa Medical Center, Department of Emergency Medicine, Phoenix, Arizona

Supervising Section Editor: Rick McPheeters, DO
Submission history: Submitted August 31, 2013; Revision received December 1, 2013; Accepted December 5, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.19452

This case report details the emergency department course of a 34 year-old female who presented with abdominal pain and vaginal bleeding after reportedly falling one week earlier. She was subsequently found to have a drinking straw within her uterus next to an eight week-old live intrauterine pregnancy on ultrasound. This case report and discussion reviews the literature on retained foreign bodies in pregnancy while addressing the added complications of an evasive patient and a difficult consultant with significant intra-specialty disagreement. [West J Emerg Med. 2014;15(2):131–133.]

INTRODUCTION

In many ways, the medicine we practice as emergency physicians (EPs) is straightforward. Part of the rewarding nature of our field is simply due to that simplicity—a patient comes in with an acute problem, we hopefully resolve the problem, and the patient leaves satisfied while we receive high Press-Ganey scores. And yet sometimes, we stumble across cases that challenge us both ethically and intellectually. This is one such case. A difficult patient, an unusual medical problem, and an obstinate consultant all come together to highlight several unique challenges that we commonly encounter in emergency medicine, but which we rarely consider at length.

CASE REPORT

The patient was a 34-year-old female with a chief complaint of abdominal pain. Her triage vitals were blood pressure 110/57; heart rate 93; respiratory rate 16; temperature 36.4°C. She stated she fell in her yard several days ago resulting in abdominal pain with some vaginal bleeding starting the morning of presentation. Upon further discussion during the physical exam, the patient admitted she missed her period and, in an effort to initiate it, stuck a drinking straw into her vagina. She inserted the whole straw inside and could still feel it poking her. She denied being pregnant and insisted she simply wanted to begin her period. Her pelvic exam was benign with a closed os, minimal blood in the vault, minimal right adnexal tenderness, and no signs of a foreign body or any lacerations. Basic labs revealed the patient was pregnant with a βHCG of 90,034. A subsequent ultrasound showed an intrauterine foreign body that appeared implanted within the myometrium with the width of a common straw and an indeterminate length. An approximately 8-week-old live intrauterine device (IUP) was noted next to the foreign body. Upon discussing these findings with the patient, she admitted she knew she was pregnant and was trying to perform an abortion with the straw.

An obstetrics (OB) consultation was placed. The initial OB consultant stated the patient was at high risk for septic shock and miscarriage. Recommendation, prior to the consultant seeing the patient, was to admit the patient for observation and antibiotics, as well as possible dilatation and curettage if she became unstable. However, shortly after requesting the consultation, a new OB attending took over the service. The new attending stated they did not believe the patient was in any danger of infection and thought it unlikely that anything had been inserted into the uterus dismissing the ultrasound finding simply as an "artifact." The new OB consultant's recommendation was to discharge the patient with outpatient follow-up. Feeling uncomfortable with simply discharging a patient who was potentially at high risk for infection, the EP performed a literature search which failed to provide guidance on the management of a patient with an intrauterine foreign body that was placed post-conception and/or non-sterilely. To make matters more confusing, the patient was an African immigrant and stated she did not want her husband to know of her condition or why she was in the hospital. She listed this as one reason why she would be unable to follow-up with an outside clinic. By this time, the husband arrived at the hospital and was becoming annoyed no one would tell him about his wife's condition. The husband's behavior was appropriate for the (unusual) situation and the patient denied sexual, physical

or emotional abuse at home. With the EP present, the patient eventually discussed the situation with her husband. Both the patient and her husband repeatedly requested removal of the foreign body due to pain, and both unequivocally stated they felt the patient's health was more important to them than the health of the fetus. The patient was reluctant to be discharged home. At the request of the patient and her husband, the EP contacted another hospital, and the OB on call there refused to accept the patient's transfer.

DISCUSSION

At this point, what should the EP do? This is a case that is fraught with challenges and ethical difficulty. The patient is evasive, the medicine is unusual, the consultant is obstructive, and there is potential for significant harm.

EPs always need to maintain their part of the physician-patient relationship; an EP has a duty to protect the patient from harm.[1] Aside from the significant medical question of infection, there are reasons to believe the patient will be at risk of harm if she is discharged. This is a patient who has gone to unusual lengths to abort her fetus. The EP has to consider the possibility of deeper psychological problems and other self-harming behavior. Similarly, the mechanism of attempted abortion, as well as the patient's strong desire not to let her husband know her reason for being in the hospital, strongly suggests some domestic unease. Furthermore, the patient is a foreign national who has questionable social support and will likely have less than optimal follow-up. The patient is obviously at a high risk for a bad outcome if she is discharged, and the EP should be reluctant to discharge the patient home with anything less than an optimal plan and concrete follow-up.

What makes this case more challenging than most is the medicine involved is not straightforward. Clearly, something appears to be in the uterus. Inserting foreign objects into the uterus, while not common, is certainly a known method of causing an abortion, although retaining foreign bodies is much rarer.[2-4] Such foreign bodies have been known to cause infection and sepsis.[5-7] Furthermore, as the numerous case reports of IUPs show, even sterile foreign bodies can lead to perforation and migration with their respective complications.[8-10] However, the exact risk of infection and perforation is unknown, as is the timeline in which they may occur. At best, one can say that the patient appears to be in danger of infection with a distinct possibility of septic shock. Nonetheless, the patient had normal vital signs, appeared non-toxic, and was not in a life-threatening condition.

It is at this point one turns to consultants for clarification and guidance, as the decisions for optimal medical care are clearly outside of the EPs expertise. Unfortunately, just when a consultant was needed most, intra-specialty disagreement complicated the case. The first consultant clearly thought this case was quite serious and needed immediate action. The second consultant thought exactly the opposite downplaying the patient's risks, questioning the patient's credibility, denying

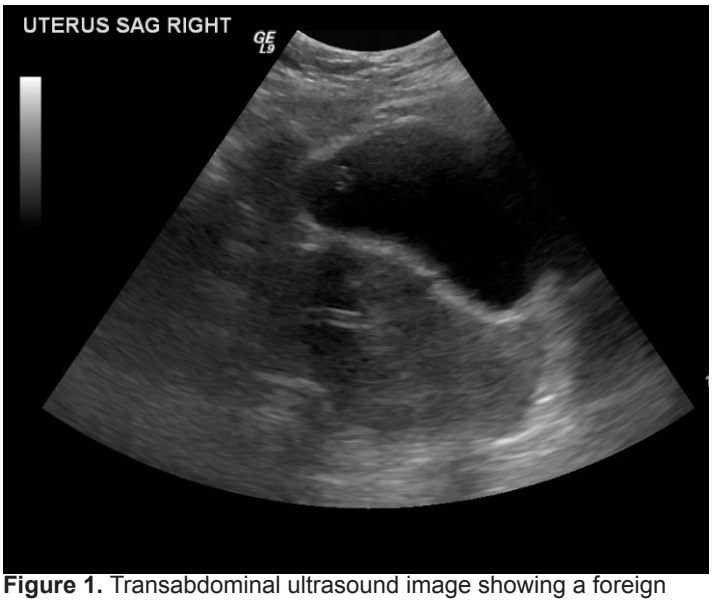


Figure 1. Transabdominal ultrasound image showing a foreign object within the uterus.

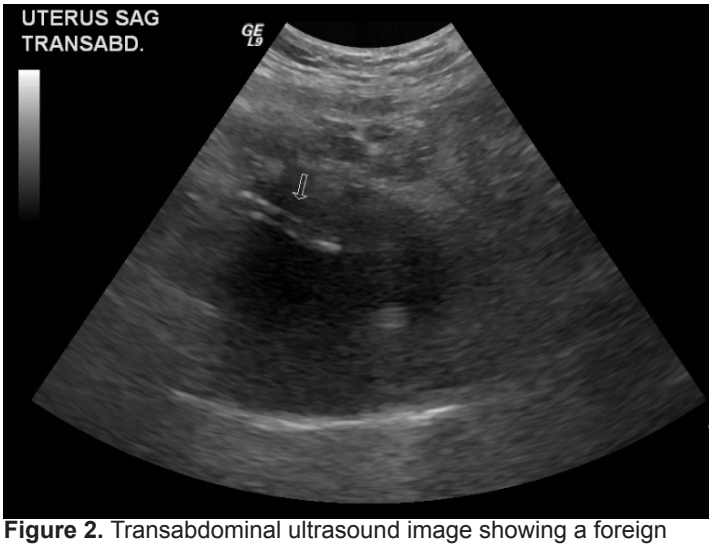


Figure 2. Transabdominal ultrasound image showing a foreign object within the uterus.

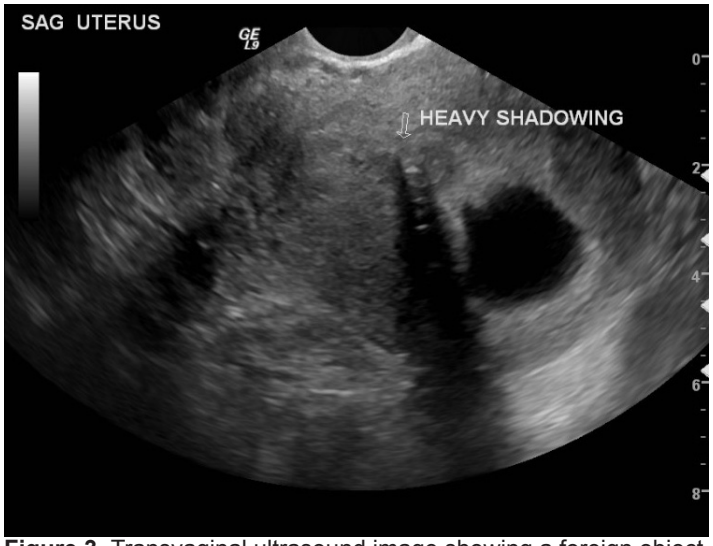


Figure 3. Transvaginal ultrasound image showing a foreign object within the uterus (arrow) next to a gestational sac.

objective evidence of the situation (the ultrasound of the straw), and making recommendations that appeared solely focused on discharging the patient.

Thankfully, situations such as this one are noteworthy for their infrequency, and the objectivity of one's consultants is rarely called into question. However, it is a situation that EPs ought to be prepared for. One of the unique aspects of emergency medicine is that we are more at the mercies of our consultants than any other hospital service. Our consultants have specialized medical knowledge, admit all of our patients, perform vital interventions, and provide almost all of our follow-up patient care. If an irreconcilable disagreement arises between the EP and a consultant, then from both a legal and a medical perspective, the EP must do, ultimately, what they believe is best for the patient; it is our primary professional responsibility to embrace patient welfare.[3,11] If the consultant adamantly refuses to admit or treat the patient, several options still remain. The EP can place the patient in an observation status in the emergency department and watch for signs of instability essentially using serial reevaluations to allow the patient's condition to indicate its progression. This also allows more time to arrange thorough out patient follow-up or to perhaps call for another consult when a more agreeable consultant is available. It would also be quite appropriate to contact the hospital's risk management team, ombudsman, or ethics representative, both for our own protection and for the patient's. Throughout this process, be certain to clearly and thoroughly document any concerns, as well as the steps taken to look after the patient's welfare and the reasoning for doing so. Avoid documentation of any disagreement with the consultant being sure to limit comments to the facts of the case, rather then focusing upon frustration or anger.

CONCLUSION

In this case, the EP followed many of the above recommendations. The OB attending refused to admit the patient, and the EP had unresolved and significant worries about discharging her home. At this point, the EP called the chairman of the OB department and, after much discussion among the EP, the chairman, and the OB attending, the OB service agreed to admit the patient for observation. The patient was observed on the OB floor for one day before signing out against medical advice. Several days later, she presented to an affiliated hospital with a surgical abdomen. Emergent laparoscopy revealed a common drinking-straw in her peritoneal cavity. The patient had a miscarriage during the postoperative period.

Address for Correspondence: Anne Klokow, MD. Maricopa Medical Center, Department of Emergency Medicine, 2601 E. Roosevelt, Phoenix, AZ 85008. Email: amklokow@gmail.com.

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

REFERENCES

1. Beauchamp TL, Childress JF. *Principles of biomedical ethics*. 5th ed. Oxford, England: Oxford University Press; 2001.
2. Osborne DF. Attempted abortion with retention of intrauterine foreign body. *Can Med Assoc J.* 1964; 90(7):494-495.
3. Weisner M. Fertility and induced abortion in poor Chilean women from the perspective of medical anthropology. *Enfoques Aten Prim*. 1988; 3(3):23-32.
4. Misago C, Fonseca W, Correia L, et al. Determinants of abortion among women admitted to hospitals in Forteleza, North Eastern Brazil. *International Journal of Epidemiology*. 1998; 27:833-839.
5. Simetka O, Prochazka M, Huseyn Zade S, Revilla J. Abortion induced by a foreign body with artificial perforation of the uterus, followed by sepsis, coma, necrosis of the uterus and hysterectomy. A case report. *Aust N Z J Obstet Gynaecol.* 2009; 49(3):337-338.
6. Okonofua FE, Onwudiegwu U, Odunsi OA. Illegal induced abortion: a study of 74 cases in Ile-Ife, Nigeria. *Trop Doc*. 1992; 22(2): 75-78.
7. Sood M, Juneja Y, Goyal U. Maternal mortality and morbidity associated with clandestine abortions. *J Indian Med Assoc*. 1995; 93(2):77-79.
8. Miranda L, Settembre A, Capasso P, et al. Laparoscopic removal of an intraperitoneal translocated intrauterine contraceptive device. *Eur J Contracept Reprod Health Care*. 2003; 8(2):122-125.
9. Mülayim B, Mülayim S, Celik NY. A lost intrauterine device. Guess where we found it and how it happened? *Eur J Contracept Reprod Health Care*. 2006; 11(1):47-49.
10. Mederos R, Humaran L, Minervini D. Surgical Removal of an intrauterine device perforating the sigmoid colon: a case report. *Int J Surg*. 2008;6(6).
11. Code of ethics for emergency physicians. A Compendium of ACEP Policy Statements on Ethical Issues. ACEP Website. Available at: http://www.acep.org/Clinical---Practice-Magagement/Code-of-Ethics-for-Emergency-Physicians/?_taxonomyid=117952. Last accessed November 15, 2013.

Delayed Presentation of Deep Sternal Wound Infection

Linda Joseph, MD
Rebecca K. Jeanmonod, MD
St. Luke's University Hospital and Health Network, Bethlehem, Pennsylvania

Supervising Section Editor: Rick McPheeters, DO
Submission history: Submitted August 21, 2013; Accepted November 12, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.11.19344

Deep sternal wound infections (DSWI) are infections of the sternum, mediastinum, or the muscle, fascia and soft tissue that overlie the sternum, typically occurring within a month of cardiac surgery. They are infrequent though severe complications of cardiac surgery. Diagnosis is made by the clinical presentation of fever, chest pain, or sternal instability in the setting of wound drainage, positive wound cultures, or chest radiographic findings. We describe the case of an elderly man presenting 6 months after cardiac surgery with DSWI. Due to the atypical nature of such a late presentation, definitive therapy was delayed. Given a severely ill patient with multiple risk factors for poor wound healing, the clinician must maintain a high index of suspicion for DSWI despite a delayed presentation. [West J Emerg Med. 2014;15(2):134–136.]

A 77-year-old male presented to the emergency department (ED) with 4 days of worsening substernal chest pain radiating to the left shoulder. The patient reported a growing mass at the superior aspect of his sternum, which was first noted several months earlier as "a small lump," but had been increasing in size over the preceding week. Review of systems was otherwise negative.

The patient's medical history was significant for aortic valve replacement (AVR) 6 months prior with an associated chronic pericardial effusion. He additionally had hypertension, peripheral vascular disease, non-insulin dependent diabetes mellitus, chronic foot ulcers, cirrhosis, chronic renal insufficiency, and chronic obstructive pulmonary disease (COPD) on steroid therapy.

On examination, the patient was afebrile, tachycardic at 112 with a blood pressure of 96/60 mmHg. His heart was irregularly irregular with no murmurs. He had a tender crepitant fluctuant soft tissue mass at the sternal notch, superior to a healed sternotomy scar, which extended into the anterior neck. Remainder of exam was unremarkable.

Laboratory findings were remarkable for a white blood cell count of 6.43×10^3/uL with 20% bands, chronic thrombocytopenia of 45×10^3/uL, and acute on chronic renal insufficiency.

Bedside ultrasound demonstrated areas of discrete fluid collection in the suprasternal mass. Bedside echocardiogram showed normal ejection fraction, normal function of the bioprosthetic aortic valve, no valvular vegetations, and a moderate pericardial effusion similar to past studies. A non-contrast computed tomography of the chest showed gas and fluid containing collections anterior to the manubrium (Figure).

Broad-spectrum antibiotics were started for sepsis, and cardiology and cardiothoracic surgery were consulted. They felt the fluid collection was unlikely infectious in etiology since the patient's AVR took place 6 months prior to presentation. The patient was admitted for sepsis with the medical team identifying a likely source as a toe ulcer with cellulitis.

Figure. A non-contrast computed tomography of the chest showing gas and fluid containing collections anterior to the manubrium.

During a 3-week hospital course, the patient was found to have methicillin sensitive *Staphylococcus aureus* bacteremia and remained on parenteral antibiotics. After failing to improve, the sternal mass was aspirated and grew out the same pathogen. He underwent operative sternal wound drainage, debridement, and wound VAC placement. DSWI was confirmed by sternal biopsy and wound culture. After a protracted hospital course, the patient was transported to a long-term care facility. Over the course of several weeks, the sternal wound healed without need for reconstructive surgery.

DISCUSSION

Although uncommon, sternal wound infections are potentially life-threatening complications of cardiac surgery. They are associated with prolonged hospitalizations, increased cost of care, significant morbidity and increased short- and long-term mortality.[1,2] Sternal wound infections are described as either superficial, involving only the skin and subcutaneous tissue, or deep, involving the underlying muscle, fascia, bone (sternum), and body spaces (mediastinum).[3,4] According to the Centers for Disease Control and Prevention (CDC), DSWI, (osteomyelitis and mediastinitus), are diagnosed by meeting one of the following criteria:

1. Positive cultures from mediastinal tissue or fluid obtained during an invasive procedure.
2. Evidence of mediastinitis seen during an invasive procedure or histopathologic examination.
3. At least 1 of the following signs or symptoms: fever (>38°C), chest pain, or sternal instability *and* at least 1 of the following:
    - Purulent discharge from mediastinal area.
    - Organisms cultured from blood or discharge from mediastinal area.
    - Mediastinal widening on imaging test.[5]

The reported incidence of deep sternal wound infections (DSWI) after sternotomy ranges from 0.22% - 4%[2-4,6-8] with mortality rates ranging from 9.7% - 23.5%.[2,3,7] Risnes et al[9] demonstrated that the risk of death following DSWI remains higher than in uninfected controls for up to 10 years.

Although most literature reports that the mean interval between initial cardiac surgery and diagnosis of DSWI is about 14 days, there have been cases of delayed presentations of DSWI. Sakamoto et al[7] conducted a retrospective analysis of 863 patients who underwent cardiac surgery and found the mean interval to be 23.6 +/- 16.0 days, with intervals ranging from 7 to 51 days. Bor et al[10] described the time from initial cardiac surgery to diagnosis of DSWI as ranging from 3 to 417 days after surgery, with a median of 7 days and 67% of patients presenting within 14 days.

There are several well-cited preoperative, operative, and postoperative risk factors for DSWI. Preoperative factors include advanced age, diabetes, male gender, COPD, obesity, hypertension, peripheral vascular disease, cigarette smoking, need for preoperative use of intra-aortic balloon pump, and preoperative use of inotropes.[3,7,11] These factors contribute to poor wound healing and wound dehiscence. They also reflect the general poor health and compromised immune system of the patient population undergoing sternotomy and cardiac surgery. Operative factors include emergent operations, coronary artery bypass grafting (CABG), bilateral internal thoracic artery use, use of inotropic agents, prolonged operation time (> 5-8hrs), blood transfusions, combined CABG and valve or aortic surgery. Postoperative factors include use of postoperative IABP, re-exploration, postoperative hyperglycemia, prolonged placement of drainage tubes and prolonged intensive care unit stays.[7]

DSWI should be suspected in the setting of known sternotomy with a clinical presentation of fever, chest pain, sternal instability, systemic toxicity, or wound drainage. As per CDC guidelines, diagnosis is eventually confirmed by these symptoms in the setting of a widened mediastinum on chest radiograph, presence of fluid drainage, or positive blood or wound cultures.[5] Given significant morbidity and mortality, patients with suspected DSWI presenting to the ED should be immediately started on broad-spectrum antibiotics with eventual narrowing of coverage based on wound culture. In the setting of a severally ill patient with signs and symptoms of DSWI, DSWI should not be excluded on the basis of time course alone. Most patients should be taken to the operating room urgently for wound exploration and debridement of nonviable or infected tissue.[12] Afterwards, based upon the severity of the patient's illness and operative findings, the sternum can be closed immediately or after a period of open wound management. Some patients will require reconstructive surgery with muscular flaps to achieve adequate wound closure and healing.[7,12]

CONCLUSION

DSWI typically presents within a few weeks, but can present remotely from cardiac surgery. In an ill- appearing patient with a history of compromised immune system, frequent skin infections, and poor wound healing, DSWI should remain high on the differential, even if cardiac surgery occurred several months prior to presentation. Antibiotics should be started as soon as possible and surgical consultation should be acquired for emergent operative wound exploration, drainage, debridement, and closure.

Address for Correspondence: Linda Joseph, MD, St. Luke's University Hospital 801 Ostrum St. Bethlehem, PA 18015, Email: Linda.Joseph@sluhn.org

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

REFERENCES

1. Toumpoulis IK, Anagnostopoulos CE, DeRose JJ. Impact of deep sternal wound infection on long term survival after coronary artery bypass grafting. *Chest*. 2005;127:464-471.
2. Filsoufi F, Castillo JG, Rahmanian PB, et al. Epidemiology of deep sternal wound infection in cardiac surgery. *J Cardiothorac Vasc Anesth*. 2009;23(4):488-494.
3. Omran AS, Karimi A, Ahmadi SH, et al. Superficial and deep sternal wound infection after more than 9000 coronary artery bypass graft (CABG): incidence, risk factors and mortality. *BMC Infection Diseases*. 2007;7:112.
4. Bryan CS, Yarbrough WM. Prevention of Deep Wound Infection after Coronary Artery Bypass Grafting: a review. *Tex Heart Inst J*. 2013; 40(2):125-139.
5. CDC/NHSN Surveillance Definition of Healthcare-Associated Infection and Criteria for Specific Types of Infections in the Acute Care Setting. Jan 2013. Available at: http://www.cdc.gov/nhsn/pdfs/pscmanual/17pscnosinfdef_current.pdf.
6. Kubota H, Miyata H, Motomura N, et al. Deep sternal wound infection after cardiac surgery. *J Cardiothorac Surg*. 2013;8:132.
7. Sakamoto H, Fukuda I, Oosaka M, et al. Risk factors and treatment of deep sternal wound infection after cardiac operation. *Ann Thorac Cardiovasc Surg*. 2003;9(4):226-232.
8. Ferris TG, Torchiana DF. Public release of clinical outcomes data-online CABG report cards. *N Engl J Med*. 2010;363(17):1593-1595.
9. Risnes I, Abdelnoor M, Almdahl SM, et al. Mediastinitis after coronary artery bypass grafting risk factors and long-term survival. *Ann Thorac Surg.* 2010;89:1502.
10. Bor DH, Rose RM, Modlin JF, et al. Mediastinitis after cardiovascular surgery. *Rev Infect Dis.* 1983;5:885.
11. Loop FD, Lytle BW, Cosgrove DM, et al. J. Maxwell Chamberlain memorial paper. Sternal wound complications after isolated coronary artery bypass grafting: early and late mortality, morbidity, and cost of care. *Ann Thorac Surg.* 1990;49:179.
12. El Oakley RM, Wright JE. Postoperative mediastinitis: classification and management. *Ann Thorac Surg*. 1996;61:1030.

Emergency Department Crowding and Loss of Medical Licensure: A New Risk of Patient Care in Hallways

Robert W. Derlet, MD[*]
Robert M. McNamara, MD[†]
Amin Antoine Kazzi, MD[‡]
John R. Richards, MD[*]

[*] University of California Davis Medical Center, Department of Emergency Medicine, Sacramento, California
[†] Temple University School of Medicine, Department of Emergency Medicine, Philadelphia, Pennsylvania
[‡] American University of Beirut, Department of Emergency Medicine, Beirut, Lebanon

Supervising Section Editor: Rick McPheeters, DO
Submission history: Submitted June 24, 2013; Revision received September 20, 2013; Accepted November 22,2013
Electronically published February 25, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.11.18645

We report the case of a 32-year-old male recently diagnosed with type 2 diabetes treated at an urban university emergency department (ED) crowded to 250% over capacity. His initial symptoms of shortness of breath and feeling ill for several days were evaluated with chest radiograph, electrocardiogram (EKG), and laboratory studies, which suggested mild diabetic ketoacidosis. His medical care in the ED was conducted in a crowded hallway. After correction of his metabolic abnormalities he felt improved and was discharged with arrangements made for outpatient follow-up. Two days later he returned in cardiac arrest, and resuscitation efforts failed. The autopsy was significant for multiple acute and chronic pulmonary emboli but no coronary artery disease. The hospital settled the case for $1 million and allocated major responsibility to the treating emergency physician (EP). As a result the state medical board named the EP in a disciplinary action, claiming negligence because the EKG had not been personally interpreted by that physician. A formal hearing was conducted with the EP's medical license placed in jeopardy. This case illustrates the risk to EPs who treat patients in crowded hallways, where it is difficult to provide the highest level of care. This case also demonstrates the failure of hospital administration to accept responsibility and provide resources to the ED to ensure patient safety. [West J Emerg Med. 2014;15(2):137–141.]

INTRODUCTION

Crowding in emergency departments (EDs) nationally and worldwide has impacted the quality of care.[1] Increases in patient mortality, medication errors, pain, length of hospital stay, and other deleterious effects have been documented.[2] When an ED is crowded, all licensed beds may be occupied, and overflow patients frequently are placed in hallways to receive care. In such circumstances, emergency physicians (EP) are placed in the difficult position of providing care to patients with suboptimal nursing support and lack of privacy, which precludes a full history and physical examination. Placing new patients back in the waiting room until a licensed ED bed becomes available poses a further risk, as there is no way to directly observe or monitor patients. Some hospital administrators insist that care in hallways be provided but fail to provide logistical support needed to accomplish this task.[3] Some ED staffing groups indirectly force physicians to see patients in unlicensed areas by emphasizing metrics such as patients seen per hour .[4] Regardless of the cause, patient care in ED hallways is fraught with delays and difficulties in initiating laboratory testing, providing medication, supervising intravenous (IV) lines, recording vital signs, monitoring cardiac activity, or responding to new patient symptoms.[5] The problem is further compounded when a physician has to simultaneously provide care to an excess number of patients in the hallway and in official ED beds, and often extra physicians are not available to share the burden. In addition to risk of poor patient outcome, physicians themselves are at risk. We describe a case of ED hallway care that resulted in the EP facing discipline by the state medical board.

CASE REPORT

A 32-year-old overweight male with recently diagnosed type 2 diabetes presented to an urban, university hospital ED

with a chief complaint of palpitations, shortness of breath, light-headedness, and "feeling ill." He had seen his primary care physician twice in the previous weeks for similar symptoms, and he had started an exercise program to address his new-onset diabetes. After an unsuccessful attempt to see his primary care physician again that day, the patient came to the ED for care. The triage nurse charted the patient's chief complaint as "chest pain with shortness of breath for one week increased with exertion" and recorded a heart rate at 140. The remaining vital signs at triage were a blood pressure of 128/71 mm/Hg, respirations of 28, and a temperature of 35 degrees C. Room air pulse oximetry was recorded at 95%. At the time of his arrival, the ED was over 250% of capacity (patients/beds), and the institution was on ambulance diversion. Since all 40 licensed ED beds were occupied, the patient was placed on a gurney in one of several narrow hallways within the ED. Twenty patients were already receiving hallway care when the patient presented to the ED, and another 40 were in the waiting room. The triage nurse immediately performed an electrocardiogram (EKG) per ED policy, which was immediately reviewed by an attending physician on duty at that time. This physician noted there was no ST-elevation myocardial infarction (STEMI) on the EKG and, at the request of a nurse, wrote orders for a basic lab panel. As it was the end of shift, this physician had no further involvement with the patient and did not mention his/her involvement to the next attending. On average, attending physicians in this ED screen as many as 25 patient EKGs for STEMI while providing direct supervision to an additional 30 patients during a 10-hour shift.

Four hours after triage, the patient was formally evaluated by an off-service PGY1 (post-graduate year one) resident. The resident was aware that an attending physician had already viewed the EKG and ordered labs. The resident charted the results, including the comments from the EKG, and performed a history and physical. The prior attending was off-shift, thus the resident presented the case to a new attending physician. At that time the ED remained crowded, with all its resources overwhelmed. The oncoming attending physician had immediately become overwhelmed with critically ill and injured patients and was repeatedly confined to the resuscitation room with medical or trauma codes. During the first 2 hours of the shift, this new attending physician performed over 10 initial EKG screenings. At 5 hours post-arrival, the resident was able to present the case to the new ED attending physician, who then examined the patient in the hallway. The history obtained by the resident noted the patient never actually had chest pain, and this was confirmed by the attending physician. The point-of-care glucose was 463 mg/dL, and the initial diagnostic impression was probable hyperglycemia with dehydration. Because the patient was in the hallway, there was no formal location to maintain his paper records, and the EKG was no longer available for review. This attending physician did know that, per ED policy, a patient with dyspnea and chest pain would have automatically received a triage EKG and assumed it had been reviewed by the prior attending physician. If the initial screening review of the EKG had been concerning, the patient would have been moved out of the hallway to a monitored licensed bed, or "doubled up" in the central treatment area of the ED. Six hours after presentation the patient's laboratory studies returned with results consistent with the initial impression of possible mild diabetic ketoacidosis (DKA) and dehydration. The blood glucose was 417 mg/dL, bicarbonate 19 mmol/L, and an anion gap of 15. Venous blood gas pH was 7.34. PCO2 was mildly decreased. Other labs were within normal limits. The patient was treated with 3 liters of IV normal saline, as well as 5 units IV and 5 units subcutaneous regular insulin. A chest radiograph was performed and was normal. Oxygen saturation was checked multiple times and ranged from 95 to 98% saturation on room air.

After therapy, the patient's glucose decreased to the 200 mg/dL range, and a repeat chemistry panel showed normal bicarbonate with no anion gap. The patient's heart rate ranged from 66 to 114 for much of the stay in the ED, and by time of discharge was normal. The dyspnea had improved. The patient passed an oral trial of fluids, felt improved, and wanted to return home. Because this was a new onset DKA, albeit mild, an informal discussion was held with the hospitalist about admission. The hospitalist, based on the quick resolution of symptoms and patient's access to his primary care physician, recommended outpatient management. The patient was then discharged home with the final diagnosis of hyperglycemia, mild DKA, dyspnea secondary to metabolic acidosis, and dehydration. A more aggressive regimen for his diabetes was prescribed, and home equipment for self-monitoring ordered. Close follow-up with his primary care doctor was specified. The patient was discharged 8 hours after arrival in the ED. Discharge vital signs were 154/86 mm/Hg, heart rate 94, respiratory rate 20, temperature 37 degrees C, and 98% room air saturation. He never occupied a licensed ED bed and was never on a cardiac monitor, as these were all in use.

Two days later, the patient developed severe shortness of breath; therefore, 911 was called and EMS activated. The patient had a cardiac arrest en route to the same hospital from which he was discharged two days earlier. Resuscitative efforts were unsuccessful, and the patient expired. An autopsy was performed and showed multiple acute and chronic pulmonary emboli as the primary cause of death. The initial EKG was retrospectively interpreted by a cardiology attending physician as showing "right axis deviation, multiple ST and T-wave changes suspicious for anterior ischemia." The autopsy did not show coronary artery disease or evidence of myocardial infarction.

INSTITUTION REVIEWS AND ACTIONS

This case was reviewed internally by the ED quality of care committee. They concluded there was "opportunity for

improvement," citing hospital and ED system problems, but not specific to an individual physician. Additional review occurred at the level of the hospital system-wide claims analysis committee. Similar conclusions were reached: severe crowding conditions, coupled with the inherent dangers of delivering care in an ED hallway, combined to cause the errors that occurred. Further, it was concluded that if this patient had received care in a bona fide ED bed, his evaluation and outcome would likely have been different. All patients with DKA, even mild, are expected to be monitored closely and have consistent nursing and physician care. The diagnosis of pulmonary embolism would be considered extremely unlikely in an active 32-year-old male with only tachycardia based on a pulmonary embolism severity index score of 62, which estimates a 30-day mortality from pulmonary embolus at 0-1.6% .[6] Included in the discussion was that the ED quality of care committee and ED chair had repeatedly complained to hospital administration for years about dangerous conditions for patients from crowding, and they had presented many cases of poor outcomes from hallway care. The hospital had failed to provide the resources requested, such as additional nursing staff, additional staffed and monitored space (anywhere in the hospital), and logistical support required for patient safety. During that period the hospital had unoccupied inpatient beds which were not staffed for financial reasons. Prior to and around the time of this case, hospital administration would routinely close one of the evaluation areas in the ED, citing nursing shortage.

The family eventually sued the institution for wrongful death. The institution settled for $1 million, without consulting any of the involved physicians. External consultants felt that sending home a patient with an alleged abnormal EKG, compounded by delays in cardiology interpretation, exposed the hospital to liability. A small administrative hospital committee without ED representation then apportioned blame. The ED attending physician was apportioned a substantial amount of the blame, which required a report to the state medical board. The hospital did not provide legal or other administrative representation for the initial medical board investigation interview, which resulted in referral to the State Attorney General's office. They then initiated action against the EP for negligence based on failure to personally review the EKG of a patient with a cardiac risk factors and chest pain and do a further cardiac workup. There was no mention of the fact that the autopsy showed normal coronary arteries without infarct, and that the patient had pulmonary emboli. The board's position was supported by an external physician reviewer with no data on ED crowding, and no interaction with the physician being investigated. This resulted in a hearing to revoke the physician's medical license. To avoid this outcome, the physician reluctantly agreed to a settlement that stipulated a published public reprimand of his ED care by the State Medical Board.

DISCUSSION

This case illustrates the risk of a poor outcome, medical malpractice, and potential loss of medical licensure when caring for patients in crowded ED hallways. Even though ED chairs had repeatedly warned senior hospital leadership about the unsafe conditions, the requested additional logistical support was not provided. No hospital administrator was named in the lawsuit, and no individual other than the treating physician had his or her career placed in jeopardy. Although hospital system problems were taken into account in the malpractice case, the state medical board's action targeted the treating physician as if he or she were working alone. Administrative indifference to ED problems with negative impact on EPs is not limited to this case. Recently a physician in Colorado attempted to activate a hospital-wide disaster response when the ED he was staffing became dangerously crowded. The administrator on duty did not agree, so the physician then contacted the hospital Chief Executive Officer (CEO). Nothing was done to improve immediate patient care and safety, but the communication with the CEO began a cascade that lead to the physician's firing without cause.[7]

What went wrong with this case? Many factors contributed, most which could have been corrected by the hospital well in advance of this patient's arrival to the ED: First of all, this patient should not have had such a long wait to receive treatment for his putative DKA. Conditions that result in prolonged "door to doctor" times should be corrected by providing appropriate resources for ED throughput. This includes the expedient transfer of admitted patients to inpatient beds. Delays in moving admitted patients out of the ED are referred to as "exit block." This has long been recognized as the major cause of crowding and long waits and is beyond the control of the ED.[8] Methods exist to offload patients out of the ED. Inpatient boarding in the ED has been long recognized as one of the prime causes of ED crowding.[9] Transferring admitted patients boarding in the ED to inpatient hallways has been shown to be safe and effective.[10] Patients actually prefer inpatient hallway boarding compared to remaining in ED hallways.[11, 12] Many institutions throughout the country have adapted this strategy.

The ED should never be allowed to reach 250% capacity. Delaying the transfer of admitted patients from the ED results in long waits for patients arriving at external and internal triage in need of emergency care. Long wait times for patients are also against United States Federal law: Emergency Medical Treatment and Active Labor Act (EMTALA) citations have been issued to hospitals by the U.S. Department of Health and Human Services (DHHS) because of long waiting times in the ED.[13] Interventions to reduce crowding have been published by the American College of Emergency Physicians (ACEP), American Academy of Emergency Medicine (AAEM), and others.[14, 15] Most of these measures require substantial cooperation and resources from hospital administration. Examples include additional flexible

treatment areas with adequate nurse staffing, enhanced resources for triage, additional hospitalists to admit patients, faster laboratory and radiology turnaround times, technicians to transport patients, and staffing to provide bedside registration.[16] Calling a hospital "internal disaster" is another option. As a full internal disaster is likely unpalatable to most administrators, a policy and procedure can be developed for use in limited or focused disasters applicable to the ED. This would involve calling in additional hospital nursing and physician staff, ancillary support, and opening up additional space to care for emergency patients.

Patients should not be routinely evaluated and treated in ED hallways where care is inferior.[17] Accurate monitoring is difficult to achieve in the hallway. Nursing care may be fragmented and inconsistent. Close coordination of care is increasingly difficult in an ED hallway. Intravenous lines run dry. Delays occur in delivery of medications.[18] Worsening patient conditions may not be recognized, and patients will suffer.[19] In some states, the law requires a 4:1 patient to nurse ratio in the ED to protect patients. The authors are aware these rules may be subverted during periods of ED crowding. Hospitals must develop strategies to avoid ED hallway care.

ED crowding leads to physician fatigue and errors.[20] Physicians have their attention split amongst so many patients that they cannot always focus effectively on the details of each patient's case. Missing details and subtle clues in complex patients can mean the difference between the correct and incorrect diagnosis. In the quest to meet external standards for early intervention in acute myocardial infarction many EDs task the already busy EP with screening large numbers of EKGs for STEMI. As in this case, the screening physician may not be the eventual treating physician. Studies have shown that simultaneously caring for multiple complex and critically ill patients results in increased medical errors.[21]

Finally, it should be recognized that the EP in this case had no control of the core issues resulting in hallway care, which potentially contributed to the misdiagnosis. With the advantage of a retrospective review, it was concluded that available clues in this case were missed and hallway care likely was a factor. The hospital administration did not step forward and take responsibility with the investigating authorities regarding its failure to provide appropriate safeguards during times of high patient demand for ED services. Instead, they allowed the EP to stand alone as a scapegoat for an issue that involved their neglect of serious systems problems in the ED. This case demonstrates that, in certain hospitals, EPs may be at risk for losing their medical licenses even though they are not at fault for the crowded conditions contributing to a poor outcome. Even if cleared of wrongdoing, this process can potentially damage a physician's career and reputation.

CONCLUSION

In conclusion, EPs should be aware of limitations and risks of providing care for patients in ED hallways. Hospital administrators should be informed that long waiting times, relentless crowding, delays in transferring admitted patients to inpatient areas, as well as ED hallway care, is unacceptable. ED leadership should demand that communication by EPs to hospital administration of unsafe conditions occur without fear of retaliation. Hospital resources must be urgently provided for real solutions to ensure patient safety.

Address for Correspondence: Robert W Derlet, MD, Department of Emergency Medicine, UC Davis Medical Center, 4150 V Street, PSSB 2100, Sacramento, CA 95817. Email: rwderlet@ucdavis.edu.

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

REFERENCES

1. Bernstein SL, Aronsky D, Duseji R, et al. The effect of emergency department crowding on clinically oriented outcomes. *Acad Emerg Med*. 2009;16:1-10.
2. Sprivulis PC, DaSilva JA, Jacobs IG, et al. The association between hospital overcrowding and mortality among patients admitted via Western Australian emergency departments. *Med J Aust*. 2006;184:208-112.
3. Derlet RW, Richards JR. Ten solutions for emergency department crowding. *West J Emerg Med*. 2008;9:24-27.
4. Silverman M. Am I really too slow? *Emergency Physicians Monthly*. November 30, 2009.
5. Derlet RW. Overcrowding in emergency departments: Effects on patients. *Cal J Emerg Med*. 2000;1:2-3.
6. Donze J, LeGal G, Fine MJ, et al. Prospective validation of the pulmonary embolism severity index: a clinical prognostic model for pulmonary embolism. *Thromb Haemost*. 2008;100:943-948.
7. Ream K. Physician's claim of breach of staffing contract and of EMTALA violation dismissed. *Common Sense*. 2012:19:7-8.
8. Martin M, Champion R, Kinsman L, et al. Mapping patient flow in a regional Australian emergency department: a model driven approach. *Int Emerg Nurs*. 2011;19(2):75-85.
9. White BA, Biddinger PD, Chang Y, et al. Boarding inpatients in the emergency department increases discharged patient length of stay. *J Emerg Med*. 2013;44:230-235.
10. Viccellio A, Santora C, Singer AJ, et al. The association between transfer of emergency department boarders to inpatient hallways and mortality: a 4-year experience. *Ann Emerg Med*. 2009;54:487-491.
11. Richards JR, Ozery G, Notash M, et al. Patients prefer boarding in inpatient hallways: correlation with the national emergency department overcrowding score. *Emerg Med Int*. 2011;2011:840459.
12. Walsh P, Cortez V, Bhakta H. Patients would prefer ward to emergency department boarding while awaiting an inpatient bed. *J*

Emerg Med. 2008;34(2):221-226

13. Ballard DW, Derlet RW, Rich BA, et al. EMTALA, two decades later: a descriptive review of fiscal year 2000 violations. *Am J Emerg Med*. 2006;24:197-205.
14. American College of Emergency Physicians. Emergency Department Crowding: High Impact solutions. *ACEP*. 2008.
15. Bernstein ST, Boggs R, Derlet RW, et al. Emergency Department crowding: past present and future directions. AAEM Policy Statement. *American Academy of Emergency Physicians*. July 10, 2006.
16. Pines JM, Pilgrim RL, Schneider SM, et al. Practical implications of implementing emergency department crowding interventions: summary of moderated panel. *Acad Emerg Med*. 2011;18:1278-1282.
17. Maa J. The Waits that matter. *New Engl J Med*. 2011;364:2279-2281.
18. Johnson KD, Winkelman C. The effect of emergency department crowding on patient outcomes: a literature review. *Adv Emerg Nurs J*. 2011;33(1):39-54
19. Derlet RW, Richards JR. Overcrowding in emergency departments: problems of the past return with a fury. *Emergency Medicine News*. 1998;20:2-3.
20. Yee P. Brain Cramp: The emergency physicians worst nightmare. *Ann Emerg Med*. 2002;39:329-330.
21. Calder LA, Forster A, Nelson M, et al. Adverse events among patients registered in high-acuity areas of the emergency department: a prospective cohort study. *CJEM*. 2010;12(5):421-430

Incarcerated Diaphragmatic Hernia with Bowel Perforation Presenting as a Tension Pneumothorax

Ryan P. Offman, DO*
Ryan M. Spencer, BA†

*Mercy Health Partners, Muskegon, Michigan
†Michigan State University College of Osteopathic Medicine, East Lansing, Michigan

Supervising Section Editor: Rick McPheeters, DO
Submission history: Submitted August 4, 2013; Revision recieved September 23, 2013; Accepted October 3, 2013
Electronically published February 19, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.10.19034

We present an interesting case of a patient with a previously known diaphragmatic hernia in which the colon became incarcerated, ischemic and finally perforated. She had no prior history of abdominal pain or vomiting, yet she presented with cardiovascular collapse. She was quickly diagnosed with a tension pneumothorax and treated accordingly. To our knowledge, this is the only case report of a tension pneumothorax associated with perforated bowel that was not in the setting of trauma or colonoscopy. [West J Emerg Med. 2014;15(2):142–144.]

INTRODUCTION

Tension pneumothorax and its associated symptoms of tachycardia, hypotension, and eventual cardiovascular collapse are caused by the condition in which the pressure within the pleural space exceeds that of atmospheric pressure. This arises as the result of a 'one-way valve' scenario where air and/or fluid is allowed to enter the pleural space but is prevented from escaping. Tension pneumothorax typically has been described as occurring in patients on ventilators in an ICU setting, patients who have undergone trauma or resuscitation attempts, and those with acute exacerbations of chronic obstructive lung disease.[1] Other causes of tension pneumothorax are much more rare. Here we describe a unique case of atraumatic tension pneumothorax in a 50-year-old woman in the presence of an empyema secondary to perforation of an incarcerated segment of large bowel through a diaphragmatic hernia.

CASE REPORT

A 50-year-old female with a past medical history of hypertension, type 2 diabetes, gastroesophageal reflux disease, paraesophageal gastric hernia, diaphragmatic hernia, and sickle cell trait presented to the emergency department (ED) with acute onset left-sided pleuritic chest pain and severe dyspnea. Relevant past surgical history includes Nissen fundoplication and appendectomy. Upon her arrival she was hemodynamically unstable with a heart rate of 162 beats per minute, blood pressure 75/62, and respiratory rate of 36 per min. Her oxygen saturation was 98% on room air.

The patient was seen immediately. Upon initial examination she was noted to be in severe respiratory distress and could not speak more than one or two words between breaths. While attempting to obtain an initial history from the patient her BP began to drop further and she became more tachycardic. Monitor revealed continued sinus tachycardia with no ectopy. Upon auscultation it was noted that breath sounds were completely absent in the left hemithorax. Bedside ultrasound was immediately available and showed an abnormal pleural line, lack of comet-tail artifacts, and absence of lung sliding motion. This was felt to be consistent with pneumothorax. Due to her worsening cardiovascular instability it was decided that there was not sufficient time to obtain a portable chest radiograph and emergent needle thoracostomy was performed. After applying betadine solution to the overlying skin, a 14-gauge intravenous catheter was inserted in the left second rib interspace in the mid-clavicular line. Immediately a large release of air was appreciated. The patient's BP increased from 63 systolic to 100 systolic and her heart rate began to decrease. Additionally, she was no longer exhibiting signs of respiratory distress as she was much less tachypneic and no longer using accessory muscles. The patient now demonstrated appreciable, however diminished, breath sounds on the left. Of note, this was the only intervention given to this patient at this time. At this point, the patient had not yet received any fluid resuscitation, vasoactive medications, nor antiarrhythmics.

At that point it was felt that the patient was stable enough to obtain a chest radiograph. The radiograph showed some residual pneumothorax but also showed what appeared to be bowel in the left hemithorax (Figure 1). Lung markings could

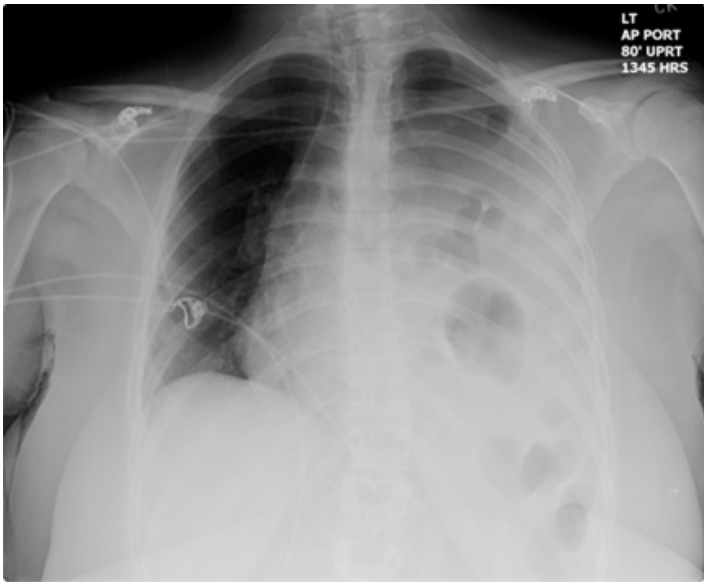


Figure 1. Portable chest radiograph obtained after needle thoracostomy, demonstrating near complete opacification of the left hemithorax with the presence of bowel loop.

not be visualized 3 centimeters from the apex, although the exact size could not be determined due to overlying bowel**.** The radiologist's report noted that the splenic flexure of the colon was herniated through the left hemidiaphragm. There was some aeration in the left upper lobe of the lung, the remainder of the left hemithorax was opacified. Cardiothoracic surgery was consulted, reviewed the chest radiograph and made the recommendation to replace the needle thoracostomy with a Heimlich valve to temporize the patient as we obtained an axial computed tomography (CT) . After placing the Heimlich valve, a CT of the chest was obtained and showed a left diaphragmatic hernia with an intrathoracic loop of colon along with a loculated hydropneumothorax suspicious for empyema (Figure 2). This was compared to a CT from 12 days prior when she presented to the ED with abdominal pain and vomiting. The previous scan showed no evidence of incarceration of the bowel loop or the presence of an empyema. She had been following the diaphragmatic hernia with a surgeon on an outpatient basis. On that visit, her symptoms resolved and was discharged. She returned 2 days prior to this presentation with posterior thoracic pain. At that time, chest radiograph was obtained and showed the known hernia but no acute process, specifically no pneumothorax. She was felt to have musculoskeletal pain and was once again discharged. However, the CT scan obtained on the current visit showed an increased amount of colon within the thorax and the colon appeared to be significantly more distended when compared to the prior CT. This was suspicious for incarceration of the colon.

The patient continued to remain hemodynamically stable while in the ED. Her surgeon that she was seeing regarding the diaphragmatic hernia was contacted and came to see her in the ED. Upon reviewing the radiograph and CT results, plans were made to take the patient to the operating room for an exploratory laparotomy. This revealed a 3.5 cm diaphragmatic defect approximately 3 cm lateral to the esophageal hiatus through which a 10 cm segment of transverse colon was incarcerated. The segment of incarcerated transverse colon appeared ischemic and there was a 2 mm perforation. General surgery could not reduce the hernia. Therefore, cardiothoracic was called in to perform a thoracotomy and radial incision of the diaphragm "because of the narrowness of the diaphragmatic hernia." Bowel, as well as omentum, was found to be "edematous and matted," and was reduced and passed into the abdomen. No frank pus or stool was observed in the thoracic or peritoneal cavities. The ischemic segment of bowel was resected to healthy colon both proximally and distally and a side-to-side anastomosis was created. A chest tube was inserted into the left posterior thoracic cavity and the diaphragmatic defect was repaired.

Cultures of serosanguineous secretion from the chest tube showed growth of *Escherichia coli* and *Enterococcus Avium*. She was placed on antibiotic therapy. She gradually regained bowel function and continued to improve quite well. She was discharged after a 9-day hospital stay in good condition.

DISCUSSION

Although we did not have the luxury to obtain a confirmatory radiograph prior to intervention, it is felt that the presenting absence of breath sounds cannot be attributed simply to the presence of bowel in the thoracic cavity.

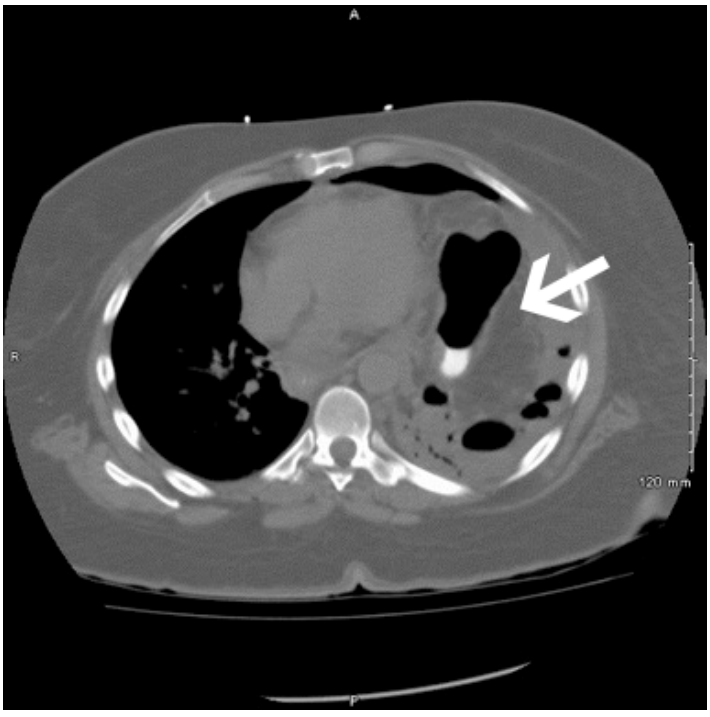


Figure 2. Axial computed tomography scan of the chest, demonstrating near complete atelectasis of the left lung along with a loculated hydropneumothorax with multiple air pockets, suggestive of empyema.

This patient presented with cardiogenic shock. This was proven through response to initial therapy and subsequent radiologic and laboratory testing. Workup revealed no other etiologies of acute cardiogenic shock and she certainly revealed tension physiology upon initial presentation which responded immediately to relief of pressure within the chest cavity. Moreover, had her presentation been solely due to displacement of bowel, she would have clinically worsened with the introduction of free air into her chest. Although we were surprised at the small volume of the pneumothorax after initial stabilization, we postulate that very little external air was needed to provide tension physiology with so much of her hemithorax occupied by incarcerated bowel. In addition, her bowel was found to be grossly ischemic ruling out the possibility that her pulmonary findings were due to a sudden worsening of the size of the hernia. Although radiology felt CT images to be consistent with empyema, surgical findings did not support this. However, CT appearance is notoriously inaccurate when characterizing pleural fluid.[2] Retrospectively, we are left with no other reason for her cardiovascular collapse other than tension pneumothorax from her ischemic, perforated bowel.

While there have been several cases reported in the literature of tension pneumothorax due colon perforation associated with colonoscopy, the patient described in this case did not undergo any recent endoscopic procedures.[3,4] Additionally, there have been case reports of tension pneumothorax in the setting of traumatic diaphragmatic hernia.[5,6] However, the patient described in this case did not have any history of either remote or recent traumatic injury.

Here we have reported an unusual case of tension pneumothorax in the setting of a chronic diaphragmatic hernia with perforation of incarcerated transverse colon and subsequent formation of an empyema. The cause of the patient's diaphragmatic defect is not completely apparent, although it is unlikely to be a congenital (Bochdalek or Morgagni) hernia as it was not noticed during prior surgical procedures or imaging studies in the distant past. As previously stated, it had been discovered prior to this described ED visit that the patient in this case had a diaphragmatic hernia with the presence of colon in the left hemithorax. However, imaging studies confirmed that the colon was not incarcerated and that surgical intervention was not immediately necessary at that time. She had been following the hernia with her surgeon on an outpatient basis with repeat imaging studies as needed. Even though the hernia had remained stable for quite some time, upon incarceration and perforation, she rapidly decompensated. This case illustrates that, while rare, formation of a tension pneumothorax is an important consideration when following patients with known diaphragmatic hernia. Moreover, their presentation is unlikely to be typical of incarcerated bowel.

Address for Correspondence: Ryan Offman, DO, Emergency Health Partners-Muskegon, 1500 E. Sherman Blvd, MI 49444
Email: dr.ryanoffman@gmail.com

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

REFERENCES

1. MacDuff A, Arnold A, Harvey J. Management of spontaneous pneumothorax: British Thoracic Society pleural disease guideline 2010. Thorax. 2010;65(Suppl 2):ii18-ii31.
2. Abramowitz Y,Simanovsky N, Goldstein MS, Hiller N. Pleural effusion: Characterization with CT attenuation values and CT appearance. Am J Roentgenol. 2009;192:618-23.
3. Ball CG, Kirkpatrick AW, Mackenzie S, et al. Tension pneumothorax secondary to a colonic perforation during diagnostic colonoscopy: report of a case. Surg Today. 2006;36:478-480.
4. Ignjatovic M, Jovic J. Tension pneumothorax, pneumoretroperitoneum, and subcutaneous emphysema after colonoscopic polypectomy: a case report and review of the literature. Lagenbecks Arch Surg. 2009;394:185-189.
5. Ramdass MJ, Kamal S, Paice A, et al. Traumatic diaphragmatic herniation presenting as a delayed tension faecopneumothorax. Emerg Med J. 2006;23:e54.
6. Kelly J, Condon ET, Kirwan WO, et al. Post-traumatic tension faecopneumothorax in a young male: case report. World J Emerg Surg. 2008;3:20.

Popliteal Artery Injury Associated with Blunt Trauma to the Knee without Fracture or Dislocation

Ahmet Imerci, MD*
Kemal Özaksar, MD†
Yusuf Gürbüz, MD†
Tahir Sadik Sügün, MD†
Umut Canbek, MD‡
Ahmet Savran, MD§

* Erzurum Palandoken State Hospital, Department of Orthopaedics and Traumatology, Erzurum, Turkey
† Hand Microsurgery Orthopaedic Traumatology (EMOT) Hospital, Izmir, Turkey
‡ Mugla Sitki Kocman University of Medicine, Department of Orthopaedics and Traumatology, Mugla, Turkey
§ Izmir Tepecik Education and Research Hospital, Department of Orthopaedics and Traumatology, Turkey

Supervising Section Editor: Rick McPheeters, DO
Submission history: Submitted May 20, 2013; Revision received November 26, 2013; Accepted December 5, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.18223

Popliteal artery injuries are frequently seen with fractures, dislocations, or penetrating injuries. Concern about arterial injury and early recognition of the possibility of arterial injury is crucial for the salvage of the extremity. This article provides an outline of the diagnostic challenges related to these rare vascular injuries and emphasizes the necessity for a high level of suspicion, even in the absence of a significant penetrating injury, knee dislocation, fracture, or high-velocity trauma mechanism. The importance of a detailed vascular examination of a blunt trauma patient is emphasized. [West J Emerg Med. 2014;15(2):145–148.]

INTRODUCTION

Blunt trauma to the lower extremity has been associated with a 28% to 46% rate of injury to the popliteal artery in the form of transection, occlusion, laceration, perforation, arteriovenous fistula, or intimal injury.[1-3] The popliteal artery, by virtue of its ligamentous fixation and anatomic relationships to the femur, tibial plateau, and knee joint apparatus, is uniquely susceptible to injury with blunt extremity trauma.[1,4] This arterial injury is frequently associated with knee dislocation following blunt trauma, and is seen with increasing frequency.[1]

Popliteal artery injury is mainly associated with high energy injury, including knee dislocation and complex tibial plateau fractures or supracondylar femur fractures.[5] Delay in its diagnosis is the leading cause of amputation in this limb-threatening injury. Failure to revascularize within 6–8 hours results in an unacceptably high amputation rate.[3]

This paper describes a rare case of blunt injury without dislocation or fracture of the knee associated with vascular injury and its delayed diagnosis resulting in amputation.

CASE REPORT

Having the first intervention done at another health center, a 38-year-old male patient engaged in mining, presented to our emergency department (ED) with an isolated crush injury of the left leg 18 hours after lateral and medial sides of his left knee was temporarily squeezed at a high speed between a tree and an operative machine. The patient's history revealed that he presented to the hospital 5 hours after the injury as the skin did not have a laceration. Investigation of the epicrisis reports and hospital records revealed that the patient underwent Doppler ultrasonography at the sixth hour. The Doppler examination revealed the presence of a probable thrombus

Figure 1. Ecchymosis, hemorrhagic bullae, and cyanosis posterior of the left knee and cruris.

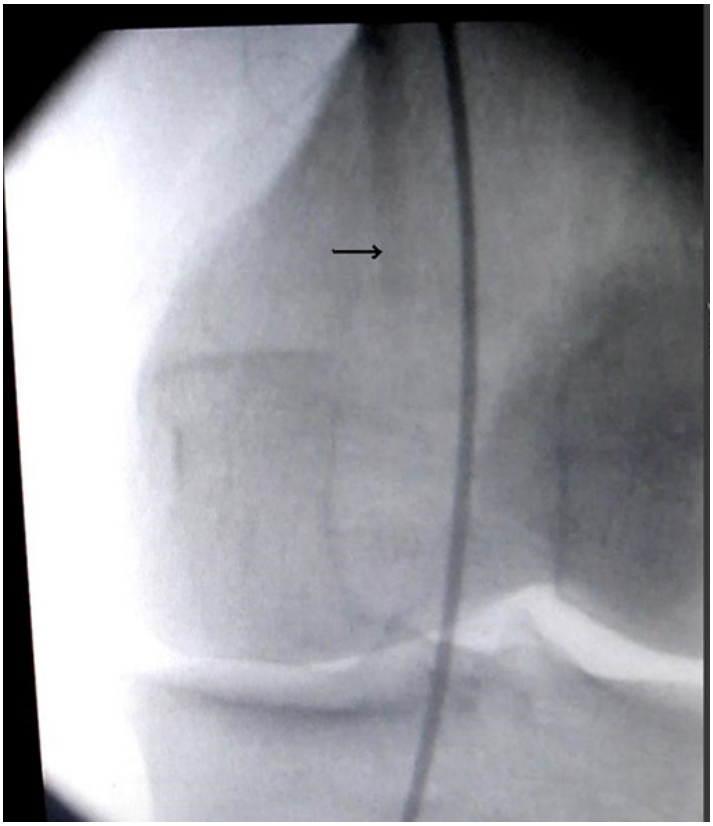


Figure 2. Angiography showing occlusion of the popliteal artery. Note the no extravasation of dye (black arrow).

Figure 3. Left above-knee amputations performed after blunt trauma of the knee.

in the popliteal area. An embolectomy was attempted with a Fogarty catheter, but was unsuccessful as the catheter did not pass through the distal area. The patient presented to our ED at the 18th hour of the injury. In the immediate physical examination of the patient, the left knee level showed signs of paleness, coldness, anesthesia, hemorrhagic bullae, and ecchymosis posterior to the knee (Figure 1). Tibialis posterior, dorsalis pedis, and popliteal arterial pulses were not felt. There was no motor function of the ankle and the toes. Successive tendon examination did not show any signs indicating a possible tendon injury. Radiographs of the leg did not show any bone pathology. The previously performed angiography revealed that the popliteal artery was totally occluded and there was no pathway to distal flow (Figure 2). The patient was transfused 2 units of blood for hematocrit <27%, and as his general condition deteriorated an above-knee amputation was undertaken.

Under spinal anesthesia, the patient was placed in the supine position in a sterile field. Knee tendon examinations did not reveal any pathological finding under anesthesia.

A fish-mouth incision was performed over the skin and the fascia. The bone was shortened 10 cm proximal to the femoral condyle. The popliteal artery and the vein were identified, tied, and cut. The proximal sciatic nerve was dissected, tied, and cut. The adductor magnus muscle was attached to the distal femur rudiment with ticron sutures. The amputation stump was sutured (Figure 3). Following surgery, when the amputation was investigated, 3 cm of intimal damage to the popliteal artery was detected and necrosis of the muscle, tendon, and neurovascular structures was present at this level. During the post-operational period, no complications developed in the wound and the patient was followed-up on for 2 years.

DISCUSSION

The most common cause of vascular injuries in the extremities is penetrating trauma. The second most common cause is blunt trauma, including traffic accidents, falls from a height, and crush injuries.[6] The mechanism of popliteal artery injury caused by blunt trauma involves vascular occlusion secondary to thrombi of the ruptured vascular intima.[1,5] Vascular injury can have devastating consequences in patients, as irreversible ischemia can occur in as short as 6 to 8 hours. Ischemia may bring about long-term morbidity or even amputation of the affected limb. Blunt popliteal artery injury has been reported to result in amputation rates of nearly 30-60%.[7,8] Depending on the degree of injury to the vessel, this diagnosis may be obvious or occult.[9]

Vascular injuries include infrequently occurring intimal tears with the majority becoming obvious in 48 to 72 hours from the time of the injury. Thus, serial clinical examination is justified for at least 48 hours in patients with intimal arterial lesions. In other words, a negative physical examination does reliably exclude vascular injury requiring surgery in patients

with blunt injury.[7] Physical examination can be combined with Doppler pressure measurements and the combination of an ankle brachial pressure index of >0.9 and a normal physical examination can reliably exclude vascular injury requiring surgery.[7,10,11]

Abou-Sayed and Berger concluded in their review that patients with hard signs of arterial insufficiency should either undergo emergency intervention or preoperative angiography, depending on the severity of ischemia.[4]

The success rate of arterial repair with direct anastomosis is lower in patients with complete ischemia due to a blunt trauma or crush injury of a long vessel segment compared to those with penetrating injuries.[12,13] In our case, we considered that symptoms were apparent when the arterial intima was ruptured and thrombosis or damaged wall presented with soft tissue swelling gradually. Crush injury with swollen soft tissues might obliterate the collateral ligament. We believe that all signs and symptoms increase the risk of amputation due to a crush injury.

As Boisrenault et al articulated, assessment of vascular lesions in bicruciate lesions of the knee, with or without dislocation, is initially based on clinical examination and pulse-taking. In case of ischemia, an emergency vascular operation is sought, which generally leads to surgical exploration preceded by in-theater arteriography.[14] The success rate in delayed cases is low. Rapid diagnosis and surgery decrease the ischemia period and thus, the amputation rate. In their studies, Subasi et al[6] stated that the existence of physical examination findings, such as bleeding from a penetrating wound, pulsatile hematoma, and the absence of distal pulses, are sufficient to establish a diagnosis of the injury. In the case of uncertain presence of distal pulses, especially in the case of blunt trauma, angiography is necessary.

Blunt knee injury producing an ischemic or pulseless extremity on presentation should alert the clinician immediately to a probable arterial injury. This should allow for prompt evaluation and repair, and further diagnostic tests are often not needed.[15] The patient with palpable distal pulses after blunt knee trauma presents more of a dilemma for the clinician in terms of evaluation and rapid diagnosis of an arterial injury. The overwhelming morbidity of limb amputation associated with a delay in diagnosis or missed injury mandates that a high degree of suspicion must be maintained and a comprehensive evaluation for injury must be done. Limb loss does occur, however, which is a certain disaster for the patient and a potential medico-legal problem for the physicians who decide to rely on physical examination alone.[14,15]

Remarkably, knee dislocations frequently spontaneously reduce before ED presentation, but still carry the same associated risk of arterial injury. Hence, physicians must aggressively search for occult knee dislocation with concomitant vascular injury in the case of a pulse-less lower extremity immediately after a minor or major lower-extremity trauma. Physical examination of the knee for ligamentous instability can alert the physician to the possibility of an occult knee dislocation.[3,11] Clinicians must remain vigilant for the possibility of a spontaneously reduced knee dislocation, and appraise the ligamentous stability of the knee and neurovascular function of the leg in patients with knee pain after trauma.[3,11-16,17] In the current case, the ED evaluation, as well as a ligamentous evaluation in the operating room in all directions while under anesthesia, suggested no evidence of instability. There was no evidence of dislocation or fracture on the radiograph.

Emergency physicians should discipline themselves about the risk of concomitant vascular injury associated with knee dislocation and its forthcoming consequences of belated diagnosis. To prevent devastating results, physicians should proceed by obtaining a detailed exam and any indicated imaging, and refer the patient to a more appropriate center as necessary.[7,18]

This rare case report aims to present the importance of initial evaluation and stresses the significance of a detailed vascular exam in blunt knee trauma. In conclusion, the physicians should bear in mind that arterial injuries do not solely result from penetrating injuries with fractures and dislocation but may also be caused by the blunt trauma of the extremities.

Address for Correspondence: Ahmet Imerci, MD. Department of Orthopaedics and Traumatology, Erzurum Palandöken State Hospital, 25000, Erzurum, Turkey. Email: ahmet_dr81@hotmail.com.

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

REFERENCES

1. Chapman JA. Popliteal artery damage in closed injuries of the knee. *J Bone Joint Surg Br*. 1985;67:420-423.
2. Drapanas T, Hewitt RL, Weichert RF HI, et al. Civilian vascular injuries: a critical appraisal of three decades of management. *Ann Surg*. 1970;172:351-360.
3. Steele HL, Singh A. Vascular Injury after Occult Knee Dislocation Presenting as Compartment Syndrome. *J Emerg Med*. 2009;4:1.
4. Abou-Sayed H, Berger DL. Blunt lower-extremity trauma and popliteal artery injuries. *Arch Surg*. 2002;137:585–589.
5. Kim JW, Sung CM, Cho SH, et al. Vascular injury associated with blunt trauma without dislocation of the knee. *Yonsei Med J*. 2010;51:790-792.
6. Subasi M, Cakir O, Kesemenli C, et al. Popliteal artery injuries associated with fractures and dislocations about the knee. *Acta*

Orthop Belg. 2001;67:259-266.

7. Nicandri GT, Dunbar RP, Wahl CJ. Are evidence-based protocols which identify vascular injury associated with knee dislocation underutilized? *Knee Surg Sports Traumatol Arthrosc*. 2010;18:1005-1012.
8. Harrell DJ, Spain DA, Bergamini TM, et al. Blunt popliteal artery trauma: a challenging injury. *Am Surg*. 1997;63:228-233.
9. Shearer D, Lomasney L, Pierce K. Dislocation of the knee: imaging findings. *J Spec Oper Med*. 2010;10:43-47.
10. Plummer JM, McFarlane ME, Jones KG, et al. Popliteal artery transection associated with blunt knee injury: controversies in management--a case report. *West Indian Med J*. 2005;54:82-84.
11. McDonough EB Jr, Wojtys EM. Multiligamentous injuries of the knee and associated vascular injuries. *Am J Sports Med*. 2009;37:156-159.
12. Bakia JM, Tordoir JH, van Heurn LW. Traumatic dissection and thrombosis of the popliteal artery in a child. *J Pediatr Surg*. 2012;47:1299-1301.
13. Hossny A. Blunt popliteal artery injury with complete lower limb ischemia: is routine use of temporary intraluminal arterial shunt justified? *J Vasc Surg*. 2004;40:61-66.
14. Boisrenoult P, Lustig S, Bonneviale P, et al. French Society of Orthopedic Surgery and Traumatology (SOFCOT). Vascular lesions associated with bicruciate and knee dislocation ligamentous injury. *Orthop Traumatol Surg Res*. 2009;95:621-626
15. Gable DR, Allen JW, Richardson JD. Blunt popliteal artery injury: is physical examination enough for evaluation? *J Trauma*. 1997;43: 541–545.
16. Laing AJ, Tansey C, Hussey AJ, et al. Occult knee dislocation: the importance of secondary survey. *Emerg Med J*. 2004;21:635-636.
17. Perron AD, Brady WJ, Sing RF. Orthopedic pitfalls in the ED: vascular injury associated with knee dislocation. *Am J Emerg Med*. 2001;19:583–588.
18. Nicandri GT, Chamberlain AM, Wahl CJ. Practical management of knee dislocations: a selective angiography protocol to detect limb-threatening vascular injuries. *Clin J Sport Med*. 2009;19:125-129.

Complete Ventricular Asystole in a Patient with Altered Mental Status

Stephen Zanoni, MD*
Gerald Platt, MD*
Shaun Carstairs, MD*
Mark Hernandez, MD†

* Naval Medical Center San Diego, Department of Emergency Medicine, San Diego, California
† Scripps Hospital Chula Vista, Department of Emergency Medicine, Chula Vista, California

Supervising Section Editor: Rick McPheeters, DO
Submission history: Submitted July 8, 2013; Revision received September 23, 2013; Accepted October 28, 2013
Electronically published February 25, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.10.18884

Patients who present with recurrent syncope are at risk for having underlying conduction disease, which may worsen if not promptly recognized and treated. We describe a patient who initially presented to a Mexican clinic with recurrent syncope and an electrocardiogram that showed complete heart block. After being transferred to our emergency department, he deteriorated into complete ventricular asystole with preserved atrial function and required placement of a transvenous cardiac pacemaker. [West J Emerg Med. 2014;15(2):149–151.]

INTRODUCTION

Syncope is a common chief complaint in the emergency department (ED), with underlying conduction disease as a rare but serious possible cause. Ventricular asystole with preserved atrial function is a rare presenting rhythm in the ED and is not commonly reported as a cause of syncope.[1] We present a case of a patient who presented with syncope that progressed to altered mental status due to complete ventricular asystole.

CASE REPORT

A 68-year-old male was transferred to our ED in Southern California via ambulance from the United States (U.S.)-Mexico border after being initially assessed in a Mexican medical clinic. Paramedics reported that he had presented to the clinic because of recurrent fainting episodes witnessed by his family in Mexico. According to the ambulance personnel, his electrocardiogram (ECG) performed at the clinic demonstrated third-degree atrioventricular block. The ECG that was performed in Mexico was not transported with the patient and was therefore not available for review.

While at the Mexican clinic, the patient reportedly decompensated and had an episode of altered mental status with what were described as "unstable" vital signs. The full details of what occurred were not available, but it was reported that the practitioners at the clinic initiated transcutaneous pacing and administered an unknown sedation medication and started the patient on a dopamine infusion. He was then transferred to the U.S.-Mexico border via ambulance. Just prior to arrival at our hospital, the patient's intravenous access was lost in the ambulance, and his transcutaneous pacing became ineffective. Upon arrival in the ED, he was lethargic and could not follow commands. He was cyanotic, had a weak but palpable pulse, shallow respirations, and a native heart rate of 30 beats/min with no capture by the pacemaker. We immediately increased the amperage of his transcutaneous pacer, which resulted in cardiac capture, and the patient developed a palpable pulse of 70 beats/min with improvement of his blood pressure to 114/53 mmHg. His core temperature was normal. The patient's neurological exam improved as he now had spontaneous movement of all 4 extremities. His initial oxygen saturation was 88%, which improved with supplemental oxygen administered via bag valve mask. As his saturations improved, he began to be able to follow commands. However, he became agitated and combative and endotracheal intubation with rapid sequence induction was performed.

Following intubation and mechanical ventilation, his oxygen saturation improved to 100%. An ECG obtained while the pacer was turned off (Figure) revealed ventricular asystole with preserved atrial function. Emergent cardiology consultation was obtained for transvenous pacer placement. Secondary assessment revealed no jugular venous distention, normal heart sounds without murmurs and clear lungs bilaterally. The abdomen was soft and non-distended. Extremities revealed resolving cyanosis without edema.

The patient's daughter was contacted and arrived after intubation and initial stabilization. She reported the patient

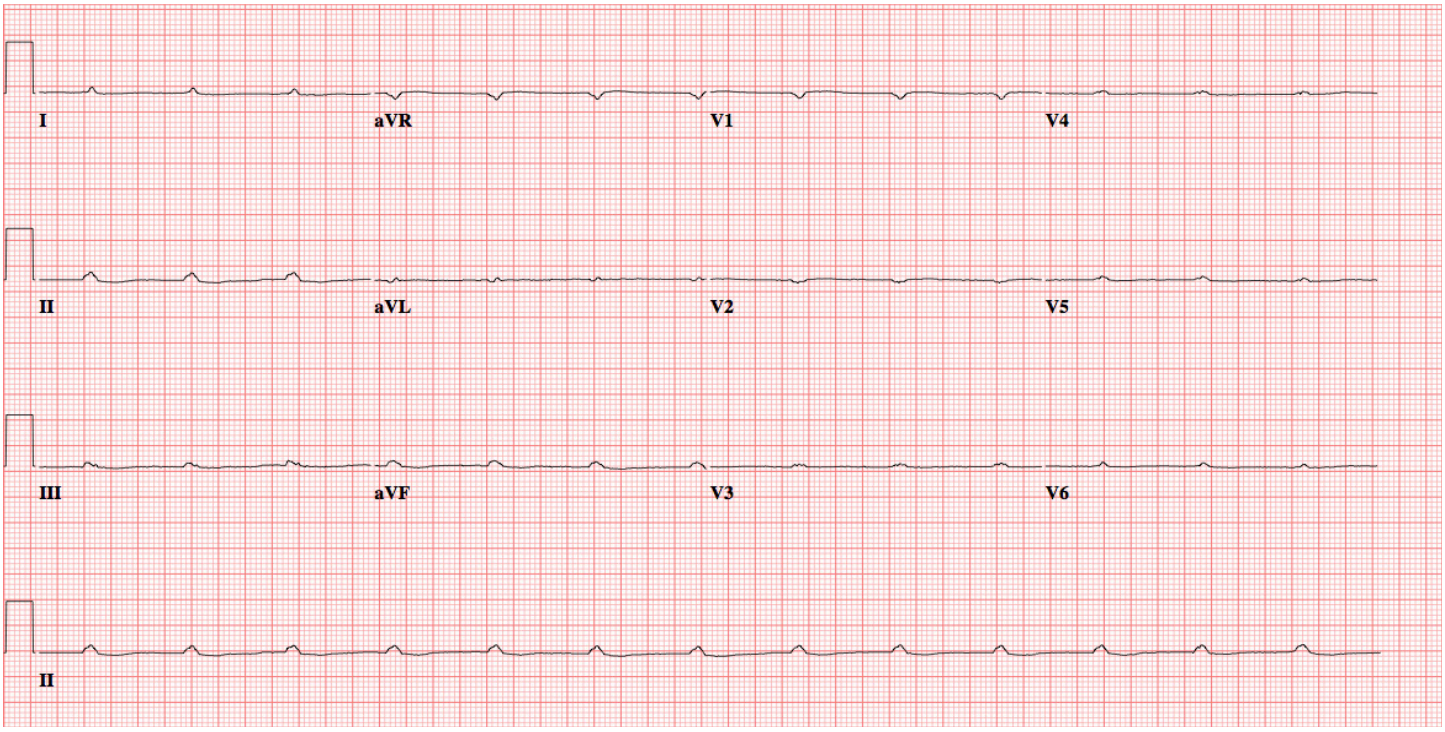


Figure. Electrocardiogram demonstrating complete ventricular asystole.

took atenolol for hypertension, smoked "heavily," and was recently started on bupropion for smoking cessation. He had no known prior cardiac history. The prior week, while vacationing in Mexico with family, he had increasing weakness, shortness of breath, and several syncope episodes. According to the daughter, he had no recent chest pain, nausea, vomiting, or focal weaknesses. There was no history of trauma. She stated he had no recent increases in his medication dosages, and he had forgotten to bring his atenolol with him to Mexico.

Arterial blood gas following intubation revealed a pH of 7.13, pCO2 of 45 mmHg, pO2 of 422 mmHg, and a lactate of 14.2mmol/L. Bedside glucose was 220 mg/dL. Cardiac enzymes revealed a troponin T of 0.31µg/L with a normal creatine kinase. Complete blood count was unremarkable. His comprehensive metabolic panel was significant for an anion gap of 24 with normal electrolyte levels. His potassium was normal at 4.0 mEq/L. His creatinine and blood urea nitrogen were 2.3 mg/dL and 28 mg/dL respectively. Urine drug screen and serum alcohol were negative. The cardiologist's official read of the ECG was complete ventricular asystole with preserved atrial function at a rate 100 beats per min. No p-wave enlargement was appreciated. Intervals, axis, and ST morphology could not be assessed due to the underlying rhythm.

A sodium bicarbonate infusion was started and the patient was taken to the interventional cardiology suite for transvenous pacer placement. Afterward, he was admitted to the intensive care unit. On hospital day 2, a transthoracic echocardiogram was significant for mild mitral regurgitation and mild left ventricular hypertrophy. He was successfully extubated on hospital day 3, had intact mental status, and was able to consent for placement of implantation of a permanent dual chamber pacemaker. He was discharged after successful completion of the procedure with close follow up. His cardiac enzymes trended downward from initial presentation, and he did not have a percutaneous coronary angiogram performed during his inpatient stay. He was diagnosed with complete atrioventricular block due to advanced conduction disease with no reversible etiology. Amiodarone or other anti-dysrhythmic medications were not administered during his stay.

DISCUSSION

Ventricular asystole with preserved atrial function is an extreme consequence of conduction disease, with a poor prognosis if not treated emergently. In the case described above, no reversible etiology was discovered and no medication or illicit substance was suspected to be the cause. The patient did have a prescription for atenolol, but he was not recently taking it, making beta-blocker poisoning unlikely as an etiology. Other previously reported causes include: digitalis therapy in the setting of hypokalemia,[2] Lyme disease,[3] increased vagal tone or vagal stimulation,[1,4,5] and blunt chest trauma resulting in acute tricuspid insufficiency.[6,7] In one case, the rhythm occurred in a 46-year-old male presenting with chest pain and no known coronary disease one minute following nitroglycerin therapy.[8]

In previous case reports, the rhythm either self-terminated or was readily stabilized by appropriate pacing therapies. Interestingly, manual external pacing at a rate of 52 beats per minute generated a blood pressure of 108/62 mmHg in a patient who experienced this rhythm during pulmonary artery catheter placement.[9] In another case report, the patient was administered 500 mg intravenous of aminophylline and reverted to sinus rhythm shortly after without other cardiac pacing modalities.[7]

In our patient, no reversible cause for his complete heart block was found. His electrolytes and Lyme studies were normal. Medication overdose was unlikely given his noncompliance. His echocardiogram was negative for major structural abnormality. However, our patient did not have a non-contrast head computed tomography or a PCA performed. These studies, in particular the PCA, may have aided in discovering a potential cause. Regardless of the underlying etiology, sustained ventricular asystole is a rhythm that requires immediate intervention with external cardiac pacing and subsequent placing of implantable cardiac pacemaker.

Address for Correspondence: LT Stephen Zanoni, MD, Naval Medical Center San Diego, Department of Emergency Medicine, 34800 Bob Wilson Dr., San Diego, CA 92134. Email: steve@thezanonis.com.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The views expressed are those of the author(s) and do not reflect the official policy of the Department of the Army, the Department of Defense or the United States Government.

REFERENCES

1. Yerg JE, Seals DR, Hagberg JM, et al. Syncope secondary to ventricular asystole in an endurance athlete. *Clin Cardiol.* 1986;9:220-222.
2. Leon-Sotomayor L, Myers WS, Hyatt KH, et al. Digitalis-Induced ventricular asystole treated by an intracardiac pacemaker. *Am J Cardiol.* 1962;10:298-301.
3. Weissman K, Jagminas L, Shapiro MJ. Frightening dreams and spells: a case of ventricular asystole from Lyme disease. *Eur J Emerg Med.* 1999;6:397-401.
4. Korantzopoulos P, Grekas G, Lampros S, et al. Markedly prolonged atrioventricular block with ventricular asystole during sleep. *South Med J.* 2009;102:870-874.
5. Schuurman PR. Ventricular asystole during vagal nerve stimulation. *Epilepsia.* 2009;50:967-968.
6. Wang NC. Immediate pacing for traumatic complete atrioventricular block and ventricular asystole. *Am J Med.* 2012;123:e3-4.
7. Khoury MY, Moukarbel GV, Obeid MY, et al. Effect of aminophylline on complete atrioventricular block with ventricular asystole following blunt chest trauma. *Injury.* 2001;32:335-338.
8. Younas F, Janjua M, Badshah A, et al. Transient complete heart block and isolated ventricular asystole with nitroglycerin. *J Cardiovasc Med.* 2012;13:533-535.
9. Chan L, Reid C, Taylor B. Effect of three emergency pacing modalities on cardiac output in cardiac arrest due to ventricular asystole. *Resuscitation.* 2002;52:117-119.

Urinary Metabolomic Analysis to Detect Changes After Intravenous, Non-ionic, Low Osmolar Iodinated Radiocontrast for Computerized Tomographic Imaging

Deborah B. Diercks, MD, MSc*
Kelly P. Owen, MD*
Vladimir Tolstikov, PhD*
Mark E. Sutter, MD*
Jeffrey A. Kline, MD†

* University of California Davis, Department of Emergency Medicine, Sacramento, California
† Carolinas Medical Center, Department of Emergency Medicine, Charlotte, North Carolina

Supervising Section Editor: Joel M. Schofer, MD
Submission history: Submitted November 24, 2012; Revision received April 24, 2013; Accepted November 12, 2013
Electronically published February 28, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.11.15343

Introduction: Contrast-induced nephropathy is a result of injury to the proximal tubules caused by oxidative stress and ischemia. Metabolomics is a novel technique that has been used to identify renal damage from drug toxicities. The objective of this study is to analyze the metabolic changes in the urine after dosing with intravenous (IV) contrast for computed tomograph (CT) of the chest

Methods: A convenience sample of patients undergoing a chest CT with IV contrast who had at least one of the following: age ≥50 years, diabetes, heart failure, chronic kidney disease, coronary artery disease, or diastolic blood pressure >90 mmHg -- were eligible for enrollment. Urine samples were collected prior to imaging and 4-6 hours post imaging. Samples underwent gas chromography/ mass spectrometry profiling. We measured peak metabolite values and log transformed data. Paired T tests were calculated. We used significance analysis of microarrays (SAM) to determine the most significant metabolites.

Results: The cohort comprised 14 patients with matched samples; 9 /14 (64.3) were males, and the median age was 61 years (IQR 50-68). A total of 158 metabolites were identified. Using SAM we identified 9 metabolites that were identified as significant using a delta of 1.6.

Conclusion: Changes in urinary metabolites are present soon after contrast administration. This change in urinary metabolites may be potential early identifiers of contrast-induced nephropathy and could identify patients at high-risk for developing this condition. [West J Emerg Med. 2014;15(2):152–157.]

INTRODUCTION

Contrast-induced nephropathy (CIN) is the third leading cause of acute renal failure in hospitalized patients. Although the exact mechanism for the development of CIN is unknown, data support that factors involved in the process may include direct toxicity to the renal tubular epithelium, oxidative stress, ischemic injury and tubular obstruction.[1] The increasing number of indications for the use of computed tomography (CT) with contrast in the emergency department (ED) has led to concerns that the incidence of contrast- induced nephropathy may increase. It has been reported that the use of contrast-enhanced CT has increased over 200% in the last decade. [2] Studies have reported that the risk for CIN is highest is patients with heart failure, diabetes, prior renal failure, hypertension, and increasing age.[3] Unfortunately, there are currently no methods to identify subjects at risk for CIN; therefore, these patients are only identified after they develop signs of acute kidney injury, which in many cases (such as

in chronic kidney disease patients) leads to the progression toward end stage renal disease.

Recent advances in the use of metabolomics have allowed the identification of specific metabolite patterns or profiles associated with acute kidney injury. Metabolic profiling refers to the measurement of a group of small molecule metabolites that reflect cellular responses to organ injury. In particular, using high performance liquid chromatography (HPLC) and gas chromatography (GC) coupled to mass spectrometry has allowed the quantification and identification of these metabolites.

We hypothesize that patients who are prone to CIN have a metabolic change that predisposes them to CIN soon after contrast administration; if this is the case, metabolomics is an ideal mechanism for the early identification of patients at risk of developing CIN as it allows the identification of the multiple markers of injury. However, the first step in proving this hypothesis is to determine that a metabolomic change occurs after intravenous contrast administration. We therefore completed a pilot study evaluating changes in urinary metabolites before and after intravenous contrast administration in a high-risk group of patients for developing contrast-induced nephropathy. The specific aim was to identify urine metabolites that change after IV contrast administration.

METHODS

This is a pilot study of prospectively identified patients undergoing a CT of the chest with intravenous contrast during their ED evaluation. The institutional review boards at all participating centers approved this study.

Study Setting and Selection of Participants

A convenience sample of patients was enrolled. To be eligible for the study, patients had to be >18 years old, undergoing CT angiography of the chest and have at least 1 of the following high-risk features for CIN: Diabetes,[4-5] coronary artery disease,[3] congestive heart failure,[4,6] chronic kidney disease (baseline creatinine >1.5mg/dl or GFR <60 ml/min/1.73 m^2.[7] Additionally, patients must have been given a physician assessment of >75% likelihood of hospital admission. All patients received between 110 and 130 mL of non-ionic radiocontrast (Omnipaque 350, GE Healthcare Inc., Princeton, NJ), delivered via automated timing injector into an arm vein.

We excluded patients from the study if they had an estimated GFR <15 mls/min/1.73m^2, a history of organ transplantation, were currently on immunosuppressive medications, were septic or on antibiotic therapy, had a history of, or were currently receiving dialysis of any type, or had an exposure to iodinated radiocontrast within 3 days prior to the study.

Data Collection and Processing

After a patient was identified as fulfilling inclusion and exclusion criteria, and informed consent was obtained, we collected data prospectively. This included race, demographics, dietary history, medical history, and physical examination. Additionally, electrocardiogram (ECG) findings as documented by the treating emergency physician were recorded. Medical history was confirmed through patient self-report and review of the medical record when available. Medications administered in the ED and before arrival were also recorded. The final ED diagnosis was recorded and based on the treating physician's impression. No laboratory tests were mandated as part of the trial study. Laboratory tests and chest radiography findings, as documented by a board-certified radiologist, were obtained from the medical record.

Urine samples were collected as a midstream sample or via a foley bag if one was already in place at the time of study enrollment. Urine samples were collected prior to imaging and 4-6 hours post imaging. Samples were divided into 2 ml aliquots and frozen at -80°C. We compared each patient's pre-CT urine peak metabolite levels to their own individual post-CT urine peak metabolite levels.

We followed all patients by chart review throughout their index stay to document in-hospital events. Serum creatinine levels were recorded at presentation, 24 and 48 hours.

GC-MS Analysis

Urine samples require no preparation prior to freezing. Neat urine samples were lyophilized without further pretreatment. To the dried samples, we added 20 µl of 40 mg/ml methoxylamine hydrochloride in pyridine, and samples were agitated at 30°C for 30 min. Subsequently, 180 µl of trimethylsilylating agent N-methyl-N-trimethylsilyltrifluoroacetamide (MSTFA) was added, and samples were agitated at 37°C for 30 min. GC–MS analysis was performed using a Agilent 6890 N gas chromatograph (Atlanta, GA, USA) interfaced to a time-of-flight (TOF) Pegasus III mass spectrometer (Leco, St. Joseph, MI, USA).[8-10] Automated injections were performed with a programmable robotic Gerstel MPS2 multipurpose sampler (Mülheim an der Ruhr, Germany). We performed initial peak detection and mass spectrum deconvolution with ChromaTOF software (version 2.25, Leco), and later exported samples to the netCDF format for further data evaluation with MZmine and XCMS. We identified samples with >50% certainty. If this degree of certainty was not met they were given the name (unknown) followed by a numeric number.

Statistical analysis

We presented continuous data as medians and interquartile range (IQR). The statistical analysis was performed on (natural) log-transformed peak concentration data to account for increases in the data variance that can occur. We performed paired T-test to determine differences in the metabolites with a p-value threshold of <0.05 mg. We used SAM, which assigns a score to each metabolite on the basis of change in metabolite expression relative to the standard

Table 1. Delta values and significant metabolites.

Delta	Significant metabolites	False discovery rate
0.07	80	0.086
1.3	22	0.045
1.6	11	0.016
2.0	4	0.004

Table 2. Demographics and patient characteristics.

Variable	N (%) or mean (% or SD)
Age (median, IQR)	61 (50,68)
Male	9 (64.3)
Caucasian race	7 (50.0)
Smoker	8 (57.1)
Heart failure	5 (35.7)
Diabetes	6 (42.9)
Hypertension	11 (78.6)
Coronary artery disease	5 (35.7)
Heart rate bpm (median, IQR)	92.5 (79,112)
Systolic blood pressure mm/Hg	140 (119,157)
Serum creatinine mg/dL (median, IQR)	0.94 (0.83,1.14)
Serum BUN mg/dL (median, IQR)	13.5 (9,22)
48 hour change in creatinine mg/dL (median, IQR)	0.02 (-0.04, 0.03)
Time since last meal hours (median, IQR)	11.2 (6.5, 19.2)
ED treatment	
Diuretics	3 (23.1)
Steroids	2 (15.3)
Final diagnosis	
Chest pain-not otherwise specified	6 (42.9)
Asthma/COPD	2 (15.4)
Heart failure	1 (7.7)
Other (pulmonary emboli, pneumonia, effusion)	5 (35.7)

BPM, beats per minute; *IQR,* interquartile range; *BUN,* blood urea nitrogen, *ED*, emergency department; *COPD*, chronic obstructive pulmonary disease

deviation of repeated measurements, to determine the most significant metabolites. For metabolites with scores greater than an adjustable threshold, SAM uses permutations of the repeated measurements to estimate the percentage of metabolites identified by chance, the false discovery rate (FDR). This adjustable threshold is determined by a tuning parameter (delta). The optimal delta was selected based on the target identification of 10 significant metabolites. (Table 1) We performed statistical analysis using MetaboAnalyst (Canada).[11]

RESULTS

The cohort comprised 14 patients with matched samples pre and post CT; 9 were males (64.3%) and the median age was 61 years (IQR 50-68). A majority of the patients had a history of hypertension 11 (78.6). There was no difference between the creatinine values at pre-CT compared to 24 or 48 hours. The most common final diagnosis was chest pain not otherwise specified. The median time from the last food intake in these patients was 11.2 (IQR 6.5, 19.2) (Table 2).

We identified a total of 158 metabolites. In the univariate analysis we identified 20 metabolites with significant difference between pre- and post-peak concentrations (Table 3).

Using SAM we identified 9 metabolites that were identified as significant using a delta of 0.6. The false discovery rate was 1.6%. All 9 covariates had a significant decrease in peak concentration after CT. Of the covariates, 2 were not identified as known metabolites. In addition, 3 were noted to be amino acids, 2 were heterocylcic compounds, and 1 was a breakdown product of carbohydrates.

DISCUSSION

Alteration in renal function has been reported to occur in 4-20% of patients undergoing CT angiograms of the chest and head. Contrast-induced nephropathy (defined as an increase of serum creatinine of 25% or higher within 2 days of receiving contrast or as a rise in plasma Cr of 0.5 mg/dL above baseline) is a leading cause of acute renal failure in this hospitalized patient population.[12] Identification in the alteration in renal function is delayed when using serum creatinine as a marker of injury. In this study we were able to detect changes in urinary metabolites within 6 hours after the administration of intravenous contrast.

This study provides the foundation to further evaluate the association of change in urinary metabolites and the development of CIN after CT imaging of the chest with contrast and identify specific metabolism pathways associated within the kidney after contrast administration. Intravenous contrast has been associated with renal toxicity. The mechanisms behind the development of CIN are complex. It has been suggested that the development of CIN is a result of the interplay of vasoconstriction, oxidative stress, and direct tubular toxicity leading to hypoxia of the outer medulla.[13,14-15] Numerous studies have reported on the effects of contrast media on various urinary enzymes and markers of glomerular and tubular function.[16-20] The complex pathophysiology behind the renal effects of intravenous contrast provides the opportunity to identify multiple markers of potential renal injury.[21]

After contrast dye administration there is a transient increase in renal blood flow followed by a decrease in renal blood flow due to vasoconstriction. This vasoconstriction

Table 3. Paired T test of log adjusted peak concentrations.

Peaks(mz/rt)*	p.value	-log10(p)
Furoylglycine	0.00123	2.90872
Unknown31&	0.00182	2.73956
Creatinine	0.00213	2.67232
Unknown28&	0.00548	2.26161
5-hydroxymethyl-2-furoic -acid	0.00586	2.23236
Levoglucosan	0.00599	2.2227
Cystine	0.01051	1.97832
Adenosine	0.01106	1.95643
Sucrose	0.01361	1.86613
Unknown34&	0.01609	1.79347
Glycerol-3-galactoside	0.01646	1.78365
Quinic acid	0.02317	1.63509
Palmitic acid	0.0237	1.62519
Taurine	0.02783	1.55552
Succinic acid	0.03123	1.50543
Idonic acid	0.04061	1.39138
hydroxypyridine	0.0421	1.37569
Deoxyribonic acid	0.04327	1.36383
Lactobionic acid	0.0449	1.34779
Glutamine	0.04787	1.31997

* mz/rt: specific mass-charge ratios (mz) /LC column retention times (RT)
&metabolites that were not identified were labelled "Unknown" followed by a numeric value

may be mediated by adenosine, Angiotensin II or calcium. In addition, there appears to be direct damage to the kidney as a result of morphological alterations including proximal tubularvacuolar transformation, interstitial edema and tubular degeneration.[22]

Studies have reported that ischemia from renal vasoconstriction causes cell sodium and chloride concentrations to rise, and cell potassium and phosphorus concentrations as well as cell dry weights to fall.[21] These changes are most pronounced in the proximal straight tubule (PST) cells. PST show deranged electrolyte homeostasis for a prolonged period after injury.[23] Amino acids such as glycine, alanine, and taurine have been shown to be protective to renal tubular cells when studied in vitro. In one study, the majority of amino acid derangements noted in the were PST cells were reversed except for low glycine contents in the cortex, whereas in the outer medulla aspartate, glycine and taurine contents were diminished.[24] These results indicate increasing manifestation of PST cell injury in the reflow period. Studies have shown that as PST are injured there is a depletion of intracellular pools of amino acids as a result of injury to Na+ pump function. In injury there is also an alteration of catabolism of alanine and glutamate formation.[25-26] We noted a decrease in glycine, n-(2-furanylcarbonyl)-in the urine, during our study. This raises the potential of amino acid supplementation as a potential nephrotoxicity protectant.

We also reported a decrease in 5-hydroxymethlfurfural (HMF) levels after CT of the chest. HMF is largely formed by breakdown of hexoses such as glucose and fructose. It is bioactivated through sulfonation of its acyclic hydroxyl functional group to 5-sulfooxymethylfurfural (SMF). It can also be metabolized in the kidneys to 5-hydroxy-methyl-2-furoic acid and other compounds such as glycine compounds in the kidney which are then secreted in the urine. The presence of HMF has been associated with nephrotoxicity in rats although never directly studied in humans.[27] HMF is produced by the oxidation of sugars through decarboxylation, oxidation, dehydration and reduction. The exact mechanism of decreased elimination in the urine is unknown.

In the kidney, adenosine locally activates adenosine A2 receptor and adjusts blood flow to meet demand. It is an intra-renal metabolite that accumulates in the kidney during renal ischemia. Elevated serum adenosine levels have been associated with a reduction in glomerular filtration rate (GFR). As GFR decreases there is an increase in sodium absorption further reducing GFR. In addition, adenosine is directly toxic to the renal cells. Our decline in urine adenosine may be due to the increase vasospasm and therefore renal ischemia associated with contrast administration.[28] We also noted a decrease in the urinary metabolites of creatinine and cystine. Both of these metabolites are associated with the breakdown products of amino acids and are reabsorbed in the proximal tubule. It is possible that the reduction in GFR as a result of contrast dye leads to increased reabsorption of these metabolites.

Prior studies have shown that the alteration of urinary concentration of metabolites that occurs as a result of toxic exposure is somewhat dependent on the toxic agent. For example, a study of nephrotoxicity as a result of $HgCl_2$ treatment resulted in a decreased concentration of citrate and 2-oxoglutarate while acetate, succinate, and lactate were increased.[29] These results are thought to be secondary to damage in the proximal tubule caused by this agent and the resulting uncoupling of oxidative phosphorylation. The injury to this area may result in alterations in loss of absorption capacity of amino acids. We also noted a decrease in urinary succinate levels in our study. However, unlike HgCl2-induced changes we also saw a reduction in alanine concentration. It has been suggested urinary changes in metabolites may be specific to different types of toxin-induced renal injury. It may be the overall pattern of the changes in urinary metabolites that can be used to identify early injury,rather than a single metabolite change.[29]

LIMITATIONS

This is a pilot trial that was underpowered to detect an association of specific metabolites with CIN. The aim of the study was to evaluate urinary metabolite changes before and

after intravenous contrast. Given our sample size for this pilot project, we were not powered to detect changes in creatinine. Also related to the small sample size, we were unable to adjust for underlying medical illnesses, time since last meal, or baseline renal function.

CONCLUSION

Diagnostic testing using CT is becoming routine in the ED. As the indications for this diagnostic modality increase, so does the potential increase in adverse events associated with this test procedure. CIN is a well-described complication of this diagnostic modality; however, current methods of identifying this disease are insensitive and require lengthy observation. In this study we have shown that there are changes in urinary metabolites within 6 hours of CT imaging. It is therefore possible that urinary metabolites may be potential novel markers of renal injury.

Address for Correspondence: Mark E. Sutter, MD. Department of Emergency Medicine, 2315 Stockton Blvd. Sacramento, CA 95817. Email: mark.sutter@ucdmc.ucdavis.edu.

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

REFERENCES

1. Messana JM, Cieslinski DA, Humes HD. Comparison of toxicity of radiocontrast agents to renal tubule cells in vitro. *Ren Fail.*1990;12(2):75-82.
2. Mitchell AM, Jones AE, Tumlin JA, et al. Incidence of contrast-induced nephropathy after contrast-enhanced computed tomography in the outpatient setting. *Clin J Am Soc Nephrol.* 2010;5(1):4-9.
3. Mitchell AM, Kline JA. Contrast nephropathy following computed tomography angiography of the chest for pulmonary embolism in the emergency department. *J Thromb Haemost.* 2007;5(1):50-54.
4. Mehran R, Nikolsky E. Contrast-induced nephropathy: definition, epidemiology, and patients at risk. *Kidney Int Suppl.* 2006;100:S11-15.
5. Bartholomew BA, Harjai KJ, Dukkipati S, et al. Impact of nephropathy after percutaneous coronary intervention and a method for risk stratification. *Am J Cardiol.* 2004;93(12):1515-1519.
6. Freeman RV, O'Donnell M, Share D, et al. Nephropathy requiring dialysis after percutaneous coronary intervention and the critical role of an adjusted contrast dose. *Am J Cardiol.* 2002;90(10):1068-1073
7. Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. *J Am Coll Cardiol.* 2004;44(7):1393-1399.
8. Weckwerth W, Loureiro ME, Wenzel K, et al. Differential metabolic networks unravel the effects of silent plant phenotypes. *Proc Natl Acad Sci U S A.* 2004;101(20):7809-7814.
9. Nikiforova VJ, Kopka J, Tolstikov V, et al. Systems rebalancing of metabolism in response to sulfur deprivation, as revealed by metabolome analysis of Arabidopsis plants. *Plant Physiol.* 2005;138(1):304-318.
10. Schad M, Mungur R, Fiehn O, et al. Metabolic profiling of laser microdissected vascular bundles of Arabidopsis thaliana. *Plant Methods.*2005;1(1):2.
11. Xia J, Psychogios N, Young N, et al. MetaboAnalyst: a web server for metabolomic data analysis and interpretation. *Nucleic Acids Res.* 2009;37(Web Server issue):W652-660.
12. McCullough PA, Wolyn R, Rocher LL, et al. Acute renal failure after coronary intervention: incidence, risk factors, and relationship to mortality. *Am J Med.* 1997;103(5):368-375.
13. Rauch D, Drescher P, Pereira FJ, et al. Comparison of iodinated contrast media-induced renal vasoconstriction in human, rabbit, dog, and pig arteries. *Invest Radiol.* 1997;32(6):315-319.
14. Liss P, Nygren A, Olsson U, et al. Effects of contrast media and mannitol on renal medullary blood flow and red cell aggregation in the rat kidney. *Kidney Int.* 1996;49(5):1268-1275.
15. Heyman SN, Brezis M, Epstein FH, et al. Early renal medullary hypoxic injury from radiocontrast and indomethacin. *Kidney Int.* 1991;40(4):632-642.
16. Parvez Z, Patel NB, Nelson JE. Urinary adenosine deaminase binding protein, a marker of contrast media induced acute renal damage. *Clin Chim Acta.* 1990;190(1-2):111-113.
17. Jakobsen JA. Renal effects of iodixanol in healthy volunteers and patients with severe renal failure. *Acta Radiol Suppl.* 1995;399:191-195.
18. Jakobsen JA, Berg KJ, Brodahl U, et al. Renal effects of nonionic contrast media after cardioangiography. *Acta Radiol.* 1994;35(2):191-196.
19. Jakobsen JA, Nossen JO, Jorgensen NP, et al. Renal tubular effects of diuretics and X-ray contrast media. A comparative study of equimolar doses in healthy volunteers. *Invest Radiol.* 1993;28(4):319-324.
20. Kaneko K, Ikebe A, Shimazaki S, et al. Differences in renal tubular toxicity of high- and low-osmolality contrast media. *Nephron.* 1989;51(4):579-580.
21. Tumlin J, Stacul F, Adam A, et al. Pathophysiology of contrast-induced nephropathy. *Am J Cardiol.* 2006;98(6A):14K-20K.
22. Thomsen HS, Dorph S, Larsen S, et al. Urine profiles and kidney histology after ionic and nonionic radiologic and magnetic resonance contrast media in rats with cisplatin nephropathy. *Acad Radiol.* 1995;2(8):675-682.
23. Wilson PD, Hartz PA. Mechanisms of cyclosporine A toxicity in defined cultures of renal tubule epithelia: a role for cysteine proteases. *Cell Biol Int Rep.* 1991;15(12):1243-1258.
24. Beck FX, Ohno A, Dorge A, et al. Ischemia-induced changes in cell element composition and osmolyte contents of outer medulla. *Kidney Int.* 1995;48(2):449-457.
25. Weinberg JM, Buchanan DN, Davis JA, et al. Metabolic aspects of protection by glycine against hypoxic injury to isolated proximal tubules. *J Am Soc Nephrol.* 1991;1(7):949-958.

26. Weinberg JM, Nissim I, Roeser NF, et al. Relationships between intracellular amino acid levels and protection against injury to isolated proximal tubules. *Am J Physiol.* 1991;260(3 Pt 2):F410-419.
27. Bakhiya N, Monien B, Frank H, et al. Renal organic anion transporters OAT1 and OAT3 mediate the cellular accumulation of 5-sulfooxymethylfurfural, a reactive, nephrotoxic metabolite of the Maillard product 5-hydroxymethylfurfural. *Biochem Pharmacol.* 2009;78(4):414-419.
28. Vallon V, Osswald H. Adenosine receptors and the kidney. *Handb Exp Pharmacol.* 2009;(193):443-470.
29. Kim KB, Um SY, Chung MW, et al. Toxicometabolomics approach to urinary biomarkers for mercuric chloride (HgCl)-induced nephrotoxicity using proton nuclear magnetic resonance ((1)H NMR) in rats. *Toxicol Appl Pharmacol.* 2010;249(2):114-126.

Hospital Factors Impact Variation in Emergency Department Length of Stay More Than Physician Factors

Scott P. Krall, MD*
Angela P. Cornelius, MD†
J. Bruce Addison, MD†

* Texas A&M University System Health Science Center College of Medicine, Department of Emergency Medicine, Corpus Christi, Texas
† Christus Spohn Hospital, Corpus Christi, Texas

Supervising Section Editor: Jeremy Hess, MD, MPH
Submission history: Submitted July 25, 2011; Revision received November 11, 2011; Accepted December 19, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.6860

Introduction: To analyze the correlation between the many different emergency department (ED) treatment metric intervals and determine if the metrics directly impacted by the physician correlate to the "door to room" interval in an ED (interval determined by ED bed availability). Our null hypothesis was that the cause of the variation in delay to receiving a room was multifactorial and does not correlate to any one metric interval.

Methods: We collected daily interval averages from the ED information system, Meditech©. Patient flow metrics were collected on a 24-hour basis. We analyzed the relationship between the time intervals that make up an ED visit and the "arrival to room" interval using simple correlation (Pearson Correlation coefficients). Summary statistics of industry standard metrics were also done by dividing the intervals into 2 groups, based on the average ED length of stay (LOS) from the National Hospital Ambulatory Medical Care Survey: 2008 Emergency Department Summary.

Results: Simple correlation analysis showed that the doctor-to-discharge time interval had no correlation to the interval of "door to room (waiting room time)", correlation coefficient (CC) (CC=0.000, p=0.96). "Room to doctor" had a low correlation to "door to room" CC=0.143, while "decision to admitted patients departing the ED time" had a moderate correlation of 0.29 ($p < 0.001$). "New arrivals" (daily patient census) had a strong correlation to longer "door to room" times, 0.657, $p<0.001$. The "door to discharge" times had a very strong correlation CC=0.804 ($p<0.001$), to the extended "door to room" time.

Conclusion: Physician-dependent intervals had minimal correlation to the variation in arrival to room time. The "door to room" interval was a significant component to the variation in "door to discharge" i.e. LOS. The hospital-influenced "admit decision to hospital bed" i.e. hospital inpatient capacity, interval had a correlation to delayed "door to room" time. The other major factor affecting department bed availability was the "total patients per day." The correlation to the increasing "door to room" time also reflects the effect of availability of ED resources (beds) on the patient evaluation time. The time that it took for a patient to receive a room appeared more dependent on the system resources, for example, beds in the ED, as well as in the hospital, than on the physician. [West J Emerg Med. 2014;15(2):158–164.]

INTRODUCTION

Emergency departments (ED) nationwide are encountering extended delays in evaluating patients.[2-5] The following attempts have been made to improve the ED patient evaluation process: additional ED beds, additional hospital beds, and improved patient through-put and discharges. Hoffenberg et al[6] evaluated

291 EDs, and assessed 386,837 patient visits within a 19-month period. A significant improvement with length of stay (LOS) was noted within the slowest EDs. By using "best demonstrated processes," the slowest EDs decreased their average LOS by only 29 minutes.[6] Kyriacou et al conducted a 5-year study using time intervals to analyze the ED patient care efficiency.[7] When an ED bed was immediately available, LOS was decreased by 36 minutes.[7] The most successful process changes addressed external factors to the ED.[8] These factors included increased flexibility of inpatient resources, float nurses who responded to acute care needs in the ED, and a transition team (mid-level provider along with registered nurse) who cared for inpatients boarded in the ED.[8] Other factors are an integrated admission service across affiliated hospitals/systems, an early alert system that notified key personnel before "critical bed" criteria were met, and a multi-disciplinary team to round in the ED and analyze resource needs.

With the increasing number of patient visits, decreasing numbers of EDs[9] and the diminishing availability of ED care as a resource, efficiency has become an important issue in providing emergency patient care and is driving hospital administration to encourage emergency physicians (EP) to improve ED metrics. The ED is a complex system. Understanding the contributions to the total time a patient spends in the department are keys to improving patient flow. The factors that affect ED flow include department size, the staffing of physicians, nursing, and the numerous ancillary services. EPs are one part of the equation in the evaluation process. They directly affect the evaluation interval by how long it takes the physician to assess the patient once the patient receives a room. Physicians determine the patient evaluation time based on the time to complete their directed evaluation, and discharge the patient. We used typical ED metric variation to try and quantify the effect of each interval of ED patient flow. The goal of the investigation was to analyze the relationship of ED metrics, to the time it took for a patient to receive an available ED treatment room ("door to room" time). We also wanted to evaluate the correlations between physician-controlled factors within the process and ED flow, to provide valuable insights into whether management efforts should focus at the level of the individual physician or larger hospital-based factors.

MATERIALS AND METHODS

Study Design

The design was to analyze the correlation between several different ED treatments intervals by collecting interval data of the average daily time for a 1-year period. With this information we used the intervals directly impacted by the EP and what effects they have on the "door to room" interval (bed availability) in an ED. Our null hypothesis was that the foundation of the delay in a patient being placed in an ED room for treatment correlates to multiple factors, not just any one interval in the process of ED evaluation.

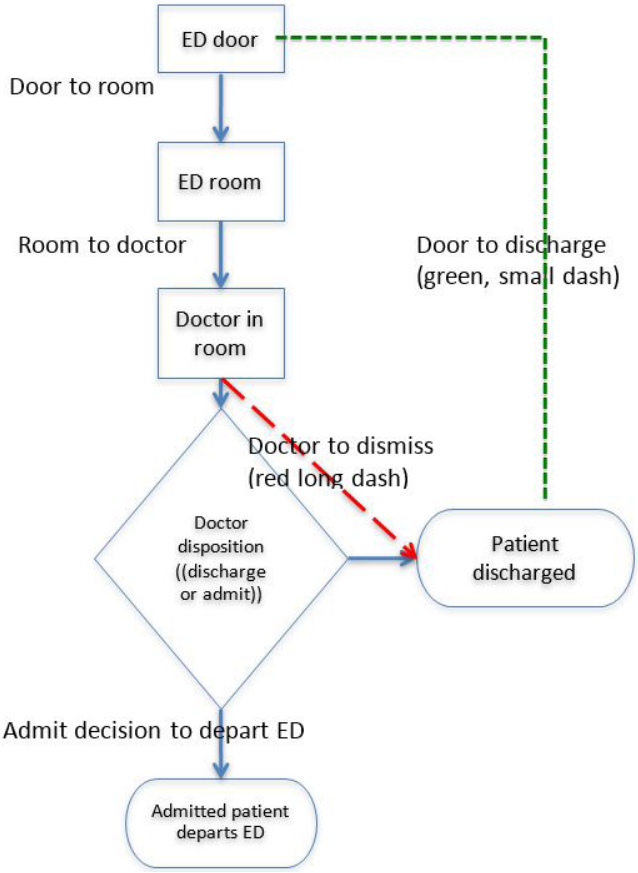


Figure 1. Emergency department (ED) intervals.

Study Setting and Population

We conducted this study at an urban county hospital facility. The hospital was a designated level 2 trauma center by the American College of Surgeons for a 12-county region, as well as having emergent cardiac catheterization capability. This facility saw 41,000 patients annually, with an admission rate of 18%. Twenty-five percent of those admissions were admitted to the intensive care units. The ED had 2 major resuscitation/trauma rooms, with an additional 15 rooms in the main department and 6 rooms for evaluation of lower acuity patients. The main part of the ED was essentially divided into 2 nursing patient care teams. The room assignments were staffed as the following: 2 registered nurses and a technician to a major resuscitation room and 6 acute care rooms. Physician staffing in the main part of the ED was an attending physician and two emergency medicine residents (of variable levels of training) for a 24-hour period. The radiology department, which was located directly behind the ED, was equipped with a CT, ultrasound, as well as MRI capabilities.

Study Process

The ED had a typical evaluation process. All walk-in patients checked in at the triage window, which was staffed by an ED technician. The patient signed in on a triage complaint form with their name, time of arrival, and chief complaint

Table 1. Emergency department (ED) intervals (determine the variables used for correlation analysis for linear relationships) address independent variable comments(8), as well as why intervals attributed to the physician(6), corresponding Meditech intervals and definitions:

ED interval	Meditech interval	Definitions
Door to room	Received to room	Time it takes for the patient to be placed back into a room i.e. bed availability for evaluation, waiting room time
Room to doctor (LIP)	Room to provider	Time it takes for a physician to see the patient in the room after the patient is placed in the room, interval directly impacted by physician getting into the room
ED length of stay for discharged patients	Received to dismiss	The time interval in minutes between arrival time and discharge time
Admit decision to depart ED time	Admit to dirty bed	The time interval in minutes between the decision to admit and the physical departure of the patient from the ED treatment area, ED boarding time
Doctor to discharge time	Provider to dismiss	The time interval in minutes between MD contact with the patient and doctor orders discharge time. Time determined by the physician directed evaluation.
Total patients per day	New arrivals	Total number of patients that signed up for triage that day.

Definitions from a consensus group created to address standardization of performance measures in emergency medicine.[11]

Table 2. Study emergency department (ED) intervals divided, based on median length of stay (LOS) of 154 minutes.

Interval: (median)	LOS ≤ 154 (SD)	LOS > 154 (SD)	Difference
Door to room (minutes)	38.4 (21)	88.8 (31.5)	50.4 (131%)
Room to doctor (LIP) (minutes)	28.2 (8)	33 (8)	4.8 (17%)
ED LOS for discharged patients (minutes)	132 (17.5)	199 (33)	67.0 (51%)
Decision to left ED time (minutes)	199 (99.7)	237 (141)	38.0 (19%)
Doctor to discharge time	69.6 (16)	82 (17)	12.4 (18%)
Total patients per day	114 (14)	131 (16)	17.0 (15%)

(time of arrival started the ED intervals). Then a registered nurse evaluated the patient. A vast majority of the patients received an evaluation using the Emergency Severity Index (ESI) index based on the stability of the patient as well as need for evaluation.[10] If their complaint had potential severity, and/or if a room was available, the patient would be immediately assigned to an ED room. If no beds were available, the triage nurse asked the patient to wait in the lobby until a room was available for further evaluation (arrival to room interval). A vast majority of the patients that arrived by ambulance were immediately taken to a room for evaluation (in that situation, time of nurses initial triage started the ED intervals). The EP (or physician extender) then assessed the patient to determine if further evaluation or consultation was necessary. The EP determined if the patient needed to be admitted to the hospital for further treatment, or discharged home with discharge instructions for care at home. If a patient was admitted to the hospital, the time that the order was given was used to determine the beginning of ED "boarding" (admit decision to hospital bed). Boarding occurred only after a physician (a hospitalist or patient physician) had accepted and agreed to treat and follow the patient during their hospital stay.

Method of Measurement

Average daily time interval data was collected from the ED tracking system, Meditech©. This was a dynamic tracking system that required the staff to time mark the following events: when the patient received a room (room), when the physician assessed the patient (doctor), physician order to admit patient to the hospital for further treatment (admit) and when the patient was discharged from the ED (dismiss). The intervals collected included classic ED measures as defined by Welch et al[11] (Table 1). We chose the intervals as they represented the separate event points entered in every visit, and represented the distinct steps in the flow of a patient through-put in an ED related to the physicians' care (Figure 1). The data collected were the average of the interval for each 24-hour day during a year covering the period from November 2008 to November 2009. April 2009 was used as a representative month for Figures 2, 3, and 4, which visually represented the pattern of relationships of ED intervals. April 2009 was chosen because of the very similar correlation coefficients to the results for the entire year.

Primary Data Analysis

We divided the intervals based on if the physician

Table 3. Summary "Door to Room" interval correlation coefficients to the following intervals

Interval:	Pearson Correlation	p-value
Room to doctor (LIP)	0.143 (low)	p=0.006
Emergency department (ED) length of stay for discharged patients	0.804 (Very strong)	p<0.001
Decision to left ED time	0.290 (Medium)	p<0.001
Doctor to discharge time	0.000 (None)	p=0.996
Total patients per day	0.657 (Strong)	p<0.001

had any direct impacts on the majority of the time in the interval. The time it took the physician to get into the room to evaluate the patient was directly controlled by the priority the physician placed on getting into the room to see the patient. The physician determines the duration of the "room to dismiss interval," based on the complaint dictating the duration of the patient evaluation. We analyzed the relationship of each interval using simple correlation (Pearson Correlation coefficients) to the "arrival to room" interval. We felt that variation in the arrival to room time was the primary determinant of extended evaluation times within the department and wanted to understand what intervals correlated best to extending the time the patients spent in the waiting room. We also divided data by intervals based on the average ED length of stay published by Centers for Disease Control on 2008 Emergency Department Statistics.[1] The 2008 average ED LOS was divided into the following two groups – less than average LOS and greater than average LOS. The information was presented to demonstrate the change in interval times based on the extended LOS (Table 2.) We performed statistical analysis using SPSS Version 16, Chicago Illinois. The Institutional Review Board approved this study as exempt.

RESULTS

We divided ED median interval times (Table 2) into 2 groups based on the CDC reports of national median LOS in 2008 (154 minutes),[1] to compare "good" days to days with delays in patient progress through the department. When the LOS extended beyond the CDC-reported median, the following intervals were increased: room to doctor 17%, doctor to discharge 18%, ED LOS for discharged patient 51%, and door to room 131% (Table 2). Delays with patients receiving a "room" within the ED had no effect on "room to doctor" intervals" (Table 3). "Door to room" interval correlation coefficients (CC) show a low CC of 0.143 for "room to doctor" interval. The "doctor to discharge" time interval demonstrates no correlation to the variation with the "door to room" (see Figure 2). Variation within the physician-directed components of the evaluation and acuity of the treatment did not have a correlation with the delay in time it took for a patient to receive an ED "room." "Admit decision time to depart ED time" had a moderate correlation to the "door to room" interval. The "door to room" component of the "door to discharge" interval had a strong correlation (see Figure 4) to delays in a patient receiving a room. Another major factor in department function was the "total patients per day," with a strong correlation of 0.657 to the variation in the "door to room" time interval.

DISCUSSION

Patient evaluation within the ED is part of a very complex system that involves both the ED and additional departments/

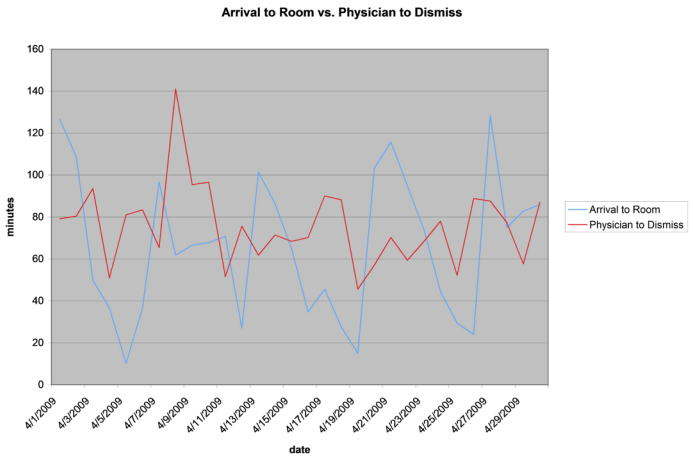


Figure 2. Door (arrival) to room versus doctor (physician) to discharge time (correlation coefficient 0.00, April correlation coefficient -0.065) reviewer d.

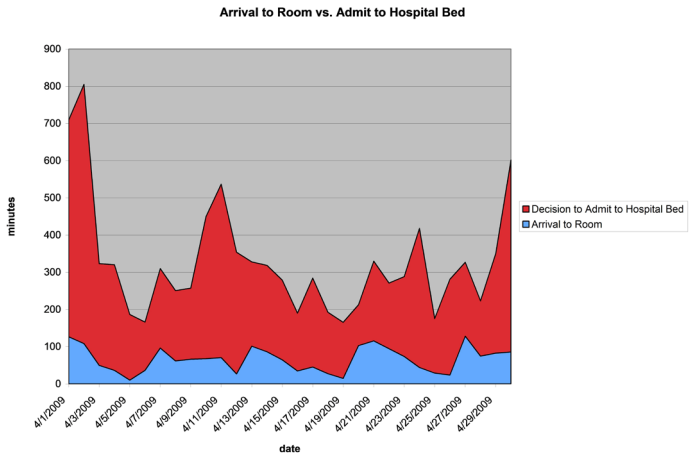


Figure 3. Door (arrival) to room versus decision to left emergency department time (correlation coefficient 0.29, April correlation coefficient 0.24).

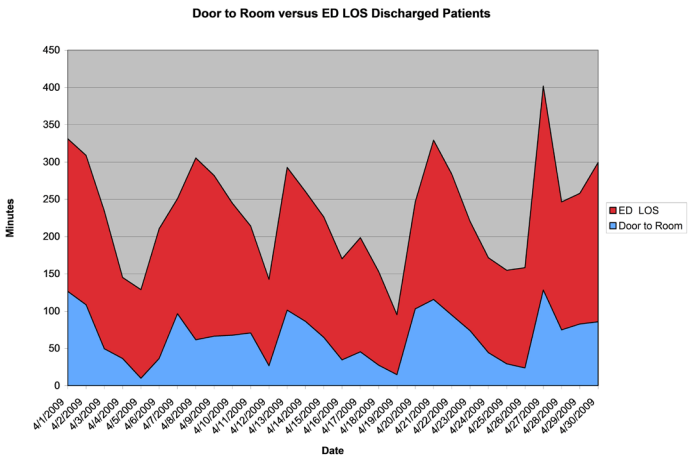


Figure 4. Door to room versus emergency department (ED) length of stay (LOS) for discharged patients (correlation coefficient 0.804, April correlation coefficient 0.73).

systems within the hospital. Literature review supports no impact on the LOS interval by improving ED process within 2/3 of hospitals with best practices.[6] This institution used several best practice methods that included triage orders. This best practice method alone has been shown to save 37 minutes of time that a patient occupies an ED bed.[12,13] We also evaluated lower acuity complaints in a separate area in the department.[14] Physicians controlled the evaluation time of the patient, but the actual evaluation was dictated by the complaint complexity and the critical nature of the complaint. The "physician to dismiss" had no correlation to the variation.

"Door to discharge" interval had a very significant correlation of 0.804 ($p<0.001$) to the variation in the "door to room" interval, which showed the component effects on the "door to room" interval on total LOS for the ED. Dividing the data based on the CDC median LOS from 2008 showed an average of 14.3 more patients per day seen, as the LOS in the ED increased beyond the 154 minutes. "Total patients per day" correlation with the "door to room" interval supported the effect of increasing the number of patients with delays in patients receiving a room. Indirectly, it supported the effect of availability of ED beds within the department and the impact it had on ED LOS. Ding et al noted that higher ED occupancy rates had a much greater effect on increasing "door to room" time or waiting room time.[15] Asaro et al showed that an increase from the 20th to the 80th percentile in ED arrivals increased the wait room ("door to room") time by 42 minutes and the LOS by 49 minutes.[16] There was no direct evaluation of the causes for the delay in receiving a room. The lack of room availability was an obvious factor that caused the delay, thus supporting the need for ED space to properly evaluate patients. The Kyriacou et al study showed that when an ED bed was immediately available, LOS was decreased by 36 minutes.[7] The time that it took for a patient to get into a room appeared more dependent on the ED system resources than on the physician.

The interval in ED LOS directly impacted by the hospital was the time it took for an admission to be transferred to an inpatient bed for care (boarding time). The Institute of Medicine identified boarding in the ED as one of the major issues in ED flow.[17] "Admit decision to depart ED" had a moderate correlation to the variation of the "door to room" interval. This variation demonstrated the effect that inpatient hospital bed availability independently has on ED LOS. McCarthy also identified ED crowding as delaying treatment and LOS.[18] Previous evaluation of hospital bed occupancy in the literature showed attempts to optimize inpatient hospital bed utilization, resulting in 85-100% hospital occupancy.[19] Bagust et al discussed the subsequent difficulty of transferring patients from the ED to inpatient beds.[19] Modeling of the dynamics for such a hospital system supports the occurrence of bed shortages and crisis at these occupancy levels.[19] An example includes the Toronto area: area hospitals closed 30%(2,890) of their acute care beds through 1997 resulting in minimum crowding. When 943 beds were closed between 1998 and 2000 and occupancy rates exceeded 90%, with a peak at 96% for acute care beds in the region, ED crowding became a frequent occurrence.[20] Vicellio et al has shown that placing admitted patients in inpatient hallways decreases those patients' LOS within the ED and available ED beds. By doing this, it also removed the additional workload from the ED staff. Thus, showing the effect of non-physician staffing, whether within the hospital or in the department, can have on ED efficiency.[21] The moderate correlation of "door to room" to "admit decision to left ED" demonstrates the large effect the inpatient hospital resource availability can have upon the ED patients' LOS. Rathlev et al showed that for every additional surgical elective admission, it prolonged the ED LOS by 0.21 minutes per ED patient, 2.2 minute for each additional ED admission, and 4.1 minutes for every increase in hospital occupancy.[22] Lucas et al concluded very similar correlations with the number of admissions as well as occupancies using 5 facilities varying from 30,000 census up to 99,280 census.[23] Continued financial drivers to maintain high occupancies will result in the need for the hospital care system to be more efficient when evaluating patients, transferring patients from the ED to a hospital bed, and discharging patients from the hospital.

Overall, EPs infrequently have direct responsibility for other personnel, staffing, and management. They can advocate, encourage and set the examples to increase patient flow within the ED. However they need the support and recognition of hospital administration to maintain the necessary workforce and available beds to accomplish that goal. Incentivizing the physician outside the "room to doctor" interval, specifically the arrival ("door") to physician interval, will only lead to individual physician frustration with the entire system given the lack of direct ability to affect patient movement through the system and complicated workforce system issues throughout the hospital. Managing the ED

is complex and requires significant partnering between the hospital staff, emergency staff, and the physicians to optimize patient care.

LIMITATIONS

The information was from a single institution and only applies to that institution. Every institution has variable methods of triage, as well as handling ambulances, registrations, and their own focuses on patient evaluation. This makes the actual intervals difficult to compare between institutions, but the evaluation of the entire system remains pertinent to understanding the overall ED process.

Another major consideration in interpreting the information was in the mechanism of tracking the status of the patient. The individual staff had to mark the status of the patients in the system, and we continue to use paper charts as a visual queue of patients waiting to be seen. Both can contribute to variation in the intervals being recorded, but the large sample size should minimize the potential for error.

Third, there was no direct evaluation for the specific causes of daily variation in intervals or in individual patients as they processed through the ED. The study used average daily interval data with correlation, which shows linear relationships. (It does not analyze for specific factors.) We did not analyze specific complaints, and surges, or specific causes of the delays.

We performed statistical analysis using simple correlation of the intervals, to demonstrate a linear relationship between the various intervals. The outside, confounding variables, radiographs, laboratory or acuity markers were not included in the data collected (in simple correlation, we could miss their contribution or be the cause of the correlation). This study looked at how each interval affected the time a patients waited for an ED room. While the above confounders exist as part of the intervals' daily variation in presentation complaint and evaluation, even with this daily variation, the simple correlation remained, demonstrating the significant component effect of time to receive a room in the patients' LOS.

CONCLUSION

Physician-dependent intervals had minimal correlation to the variation in the "door to room" time. The average "doctor to discharge" time required to evaluate the patient remains relatively consistent and does not correlate to variation in "door to room" times. The "door to room" interval was a significant component to the variation in "door to discharge," i.e. LOS, so delay in having a bed available in the ED correlates to extended ED "door to room." The hospital-influenced "admit decision to hospital bed," i.e. hospital inpatient capacity, interval had a correlation to delayed "door to room" time. The other major factor affecting department bed availability was the "total patients per day," and the correlation to the increasing "door to room" time also supports the effect availability of resources (beds) had on completing patient evaluation. The time that it took for a patient to receive a room appeared more dependent on the system resources, for example, beds in the ED as well as in the hospital, than on the physician.

Address for Correspondence: Scott P. Krall, MD. Department of Emergency Medicine, 2606 Hospital Blvd., Corpus Christi TX 78405. Email: Scott.krall@christushealth.org.

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

REFERENCES

1. National Hospital Ambulatory Medical Care Survey: 2008 Emergency Department, Summary Table; US department of health and Human Services, Division of Health Care Statistics.
2. Shute N, Marcus MB. Code Blue Crisis in the Emergency Department. Turning Away Patients. Long Delays. A Surefire Recipe for Disaster. *US New & World Report*. 2001;131(9) :54-61.
3. Richards JR, Navarro ML, Derlet RW. Survey of Directors of Emergency Departments in California on Overcrowding. *West J Emerg Med*. 2000;172(6);385-388.
4. Derlet R, Richards J, Kravitz R. Frequent Overcrowding in U.S. Emergency Departments. *Acad Emerg Med*. 2001;8(2):151-155.
5. Herring A, Wilper A, Himmelstein DU, et al. Increasing Length of Stay Among Adult Visits to U.S. Emergency Departments, 2001–2005. *Acad Emerg Med*. 2009;16 (7):609-616.
6. Hoffenberg S, Hill BH, Houry D. Does Sharing Process Differences Reduce Patient Length of Stay in The Emergency Department? *Acad Emerg Med*. 2001;8(5):578.
7. Kyriacou DN, Ricketts V, Dyne PL, et al. A 5-Year Time Study Analysis of Emergency Department Patient Care Efficiency. *Ann Emerg Med*. 1999;34(3):326-335.
8. Schneider S, Zwemer F, Doniger A, et al. Rochester, New York: A Decade of Emergency Department Overcrowding. *Acad Emerg Med*. 2001;8(11):1044-1050.
9. Lambe S, Washington DL, Fink A, et al. Trends in the Use and Capacity of California's Emergency Departments, 1990-1999. *Ann Emerg Med*. 2002;39(4):389-396.
10. Wuerz RC, Milne LW, Eitel DR, et al. Reliability and Validity of a New Five-Level Triage Instrument. *Acad Emerg Med*. 2000;7:236e42.
11. Welch S, Augustine J, Camargo CA Jr, et al. Emergency Department Performance Measures and Benchmarking Summit. *Acad Emerg Med*. 2006;13(10):1074-1080.
12. Russ S, Jones I, Aronsky D, et al. Placing Physician Orders at Triage: The Effect on Length of Stay. *Ann Emerg Med*. 2010;56(1):27-33.
13. Retezar R, Bessman E, Ding R, et al. The Effect of Triage Diagnostic

Standing Orders on Emergency Department Treatment Time. *Ann Emerg Med*. 2010;57(2):89-99.e2.

14. Considine J, Kropman M, Kelly E, et al. Effect of emergency department fast track on emergency department length of stay: a case-control study. *Emerg Med J*. 2008;25: 815-819.
15. Ding R, McCarthy ML, Desmond JS, et al. Characterizing Waiting Room Time, Treatment Time, and Boarding Time in the Emergency Department Using Quantile Regression. *Acad Emerg Med*. 2010;17(8):813-823.
16. Asaro PV, Lewis M, Boxerman SB. The Impact of Input and Output Factors on Emergency Department Throughput. *Acad Emerg Med*. 2007;14(3):235-242.
17. Institute of Medicine of the National Academics; The Future of Emergency Care in the United States Health System, Brief Report; June 2006.
18. McCarthy ML, Zeger SL, Ding R, et al. Crowding Delays Treatment and Lengthens Emergency Department Length of Stay, Even Among High-Acuity Patients. *Ann Emerg Med*. 2009;54(4):492-503.
19. Bagust A, Place M, Posnett JW. Dynamics of bed use in accommodating emergency admissions: stochastic simulation model. *BMJ*. 1999;319(7203):155-158.
20. Schull MJ, Szalai J, Schwartz B. Emergency Department Overcrowding Following Systematic Hospital Restructuring: Trends at Twenty Hospitals over Ten Years. *Acad Emerg Med*. 2001;8(11):1037-1043.
21. Vicellio A, Santora C, Singer AJ, et al. The Association Between Transfer of Emergency Department Boarders to Inpatient Hallways and Mortality: A 4-Year Experience. *Ann Emerg Med*. 2009;54(4):487-491.
22. Rathlev NK, Chessare J, Olshaker J, et al. Time Series Analysis of Variables Associated With Daily Mean Emergency Department Length of Stay. *Ann Emerg Med*. 2007;49(3): 265-271.
23. Lucas R, Farley H, Twanmoh J, et al. Emergency Department Patient Flow: The Influence of Hospital Census Variables on Emergency Department Length of Stay. *Acad Emerg Med*. 2009;16(7):597-602.

Impact of a Teaching Service on Emergency Department Throughput

Courtney M. Smalley, MD*
Gabrielle A. Jacquet, MD, MPH*
Margaret K. Sande, MD, MS*
Kennon Heard, MD†
Jeffrey Druck, MD†

* Denver Health Residency in Emergency Medicine, Denver Health & Hospital Authority, Denver, Colorado
† Department of Emergency Medicine, University of Colorado School of Medicine, Aurora, Colorado

Supervising Section Editor: Douglas S. Ander, MD
Submission history: Submitted March 12, 2013; Revision received July 26, 2013; Accepted August 28, 2013
Electronically published February 19, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.8.16316

Introduction: There are 161 emergency medicine residency programs in the United States, many of which have medical students rotating through the emergency department (ED). Medical students are typically supervised by senior residents or attendings while working a regular shift. Many believe that having students see and present patients prolongs length of stay (LOS), as care can be delayed. Our institution implemented a unique method of educating medical students while in the ED with the creation of a teaching service, whose primary goal is education in the setting of clinical care. The objective of this study was to explore the effect of the teaching service on efficiency by describing LOS and number of patients seen on shifts with and without a teaching service.

Methods: This was a retrospective chart review performed over a 12-month period of visits to an urban academic ED. We collected data on all patients placed in a room between 14:00 and 19:59, as these were the hours that the teaching shift worked in the department. We categorized shifts as 1) a teaching service with students (TWS); 2) a teaching service without students (TWOS); and 3) no teaching service (NTS). LOS and median number of patients seen on days with a teaching service, both with and without students (TWS and TWOS), was compared to LOS on days without a teaching service (NTS).

Results: The median LOS on shifts with a dedicated teaching service without students (TWOS) was 206 minutes, while the median LOS on shifts with a teaching service with students (TWS) was 220 minutes. In comparison, the median LOS on shifts when no teaching service was present (NTS) was 202.5 minutes. The median number of patients seen on shifts with the teaching service with students (TWS) was 44, identical to the number seen on shifts when the teaching service was present without students (TWOS). When the teaching service was absent (NTS), the median number of patients seen was 40.

Conclusion: A teaching service in the ED is a novel educational model for medical student and resident instruction that increases total ED patient throughput and has only a modest effect on increased median length of stay for patients. [West J Emerg Med. 2014;15(2):165–169.]

INTRODUCTION

Medical students often rotate through emergency departments (EDs) as part of their medical education. There are currently 161 Emergency Medicine residency programs in the United States, many of which have medical students rotating through the ED.[1] The ED is a somewhat unique environment for student education as the focus for supervising physicians is often on acuity of complaint, length of stay,

and prompt disposition.[2,3] In this typical educational model, medical students present cases to the supervising resident or attending who are working a ED typical shift. A common belief is that medical students significantly delay patient length of stay (LOS) as care can be prolonged in the setting of presenting and teaching.[4-7] In an era of ED crowding, LOS is a significant core measure for staff and administrators in providing efficient patient care.[8]

In 2007 our medical school instituted a required 2-week rotation for third-year medical students in the ED. At the same time, our emergency medicine (EM) residency hoped to improve the training of their residents in the disciplines of medical education and evidence-based practice. In light of this, a teaching service was created to satisfy the needs of both the medical school and the residency.

The teaching service is comprised of 1 attending physician, 2 third-year residents, and 4 to 6 third-year medical students. The teaching service is present in the ED seeing patients from 14:00-19:59 on Mondays, Tuesdays, Thursdays, and Fridays (excluding academic holidays). For 2 hours prior to the start of their clinical shift the teaching service meets for small group teaching. The first hour is resident-led didactics for the medical students. This provides residents with protected time to teach while also providing an opportunity to receive feedback on their teaching skills from an experienced attending. During the second hour the attending physician presents a topic from evidenced-based medicine or the "teaching how to teach" curriculum. One day of the week this 2-hour period is spent in simulation where residents lead cases with medical student involvement.[9] It is worth noting that the teaching service is not restricted to low-level acuity patients. Because there is a dedicated attending and 2 third-year residents, the teaching service picks up patients in the same manner as the other providers without restrictions on triage level.

Since its creation, the teaching service has expanded. During the 2009-2010 academic year, a total of 162 third-year medical students rotated on the required EM rotation, with approximately two-thirds rotating as part of the teaching service at University of Colorado Hospital, a large-volume tertiary referral hospital. This study focuses exclusively on patient data from the University of Colorado ED, as the teaching service at other sites is organized differently.

Our study investigates the LOS and number of ED patients seen by the teaching service with students (TWS) when compared to the teaching service without students (TWOS), and when compared to no teaching service (NTS).

METHODS

This was a descriptive analysis single site study to determine median LOS and number of patients seen in the ED on shifts with a teaching service and without a teaching service. Our local institutional review board approved the protocol for this study, and informed consent was waived as no identifying patient data were collected.

Study Setting

The investigative site was a 410-bed academic tertiary care urban teaching hospital with approximately 65,000 ED visits per year. The staffing model centers on resident education. Each 8-hour shift is staffed by 2 EM attending physicians supervising a second- and third-year EM resident. This team is independent of the teaching service. A varying number of interns and fourth-year medical students also work in the ED and staff patients with third-year residents, with attending oversight. Physician assistants also staff the ED and see lower acuity patients; we excluded from analysis patients seen by physician assistants.

Study Population

The study population consisted of patients seen in the ED from July 1, 2009 to June 30, 2010. We included all patients roomed in the ED between the hours of 14:00 and 19:59 as this is the time the teaching service actively sees patients in the ED. Any patient roomed during this time was included, even if their workup or disposition was determined later in the evening. Eligible patients were those seen in the ED with the following dispositions: (1) discharge from the ED, (2) discharge after medical screening exam, and (3) discharge to nursing home. We excluded patients if they were admitted or discharged to a psychiatric facility, as LOS would likely be skewed by the amount of time patients were awaiting their beds or awaiting placement by psychiatry at an outside facility. Patients were also excluded if they left without being seen. We determined LOS and number of patients seen for patients who were placed in a room between 14:00 and 19:59 and were ultimately discharged from ED. Attending, resident and medical student schedules were reviewed to determine which providers were part of the teaching service for each day of the study period, and were classified into 3 categories for analysis: (1) teaching service present (TWS); (2) teaching service present but without medical students (TWOS); and (3) no teaching service (NTS). The teaching service is present in the ED on Mondays, Tuesdays, Thursdays, and Fridays. Days designated as 'teaching service present but without medical students' include academic holidays when the students were excused and the final Friday of each -week rotation, when the students are administered their exam for the rotation and do not have clinical duties in the department. On these days, the 2 third-year residents work one-on-one with the attending and function as an extra independent physician team. Lastly, on weekends and conference days (Wednesdays, Saturdays, and Sundays), the department functions without a teaching service (NTS). The students work exclusively as part of the teaching service. They do not work with independently with core clinical faculty on any Wednesdays, weekends, or holidays. Of note, fourth-year medical students are integrated in normal work flow, and as such, were not studied as a group. As the fourth-year medical students are randomly distributed throughout the days, their effect was considered marginal.

Table 1.

14:00-19:59	Teaching service with students (TWS)	Teaching service without students (TWOS)	No teaching service (NTS)
Total patients seen (number of patients)	6880	2188	6333
Median length of stay (minutes)	220	206	202.5
Median # of Patients per Shift	44	44	40

Table 2. Comparison of teaching service groups versus no teaching service.

	No teaching service (NTS)	
	Length of stay	Number of patients seen
Teaching service with students (TWS)	p<0.001	p<0.001
Teaching service without students (TWOS)	p=0.3	p=0.007

Data Collection

We collected LOS in minutes for all study patients in a de-identified manner. Patients were categorized by teaching service status. As LOS was not expected to be normally distributed, we used median and interquartile range (IQR) to describe the data. As an exploratory analysis, we compared LOS between TWS and the remaining groups (TWOS, NTS). We believed that the LOS might differ between weekdays and weekends. We determined median LOS for the NTS group (the only option for weekends) in 2 ways: including weekends and excluding weekends. Finally, we compared median LOS and number of patients treated among the groups with the Wilcoxan Rank Sum Test using JMP 9.0 (SAS Institute, Cary NC). A p-value <0.05 was considered significant. We also performed a post-hoc analysis to determine if the median LOS and number of patients evaluated in 2 two teaching service groups differed from the no teaching service using Dunnet's test on the ranked LOS values. As this was an exploratory analysis, we did not do a formal sample size or power calculation.

RESULTS

There were a total of 63,000 visits to the ED over the 12-month study period. After applying set exclusion criteria, we included 15,401 patients in our analysis. All but 1 day of the 365 days of the study window had provider schedule information that enabled them to be correctly categorized.

The median (IQR) LOS for patients seen on shifts when TWS was present (n=6880) was 220 (146 to 320) minutes. The median LOS for patients seen on shifts when TWOS was present (n=2188) was 206 (140 to 300) minutes. The median LOS for NTS was 202.5 (37 to 292) minutes when weekend days were included (n=6333) and 216 (146 to 313) minutes when only weekdays were evaluated (2210). The median LOS for the TWS group was significantly different from the NTS group (p<0.001), but the medians for the TWOS group and the NTS group were not significantly different (p=0.3) (Tables 1 and 2).

The median (IQR) number of patients seen on shifts when TWS was present (n=159) was 44 (39 to 48). The median (IQR) number of patients seen on shifts when TWOS was present (n=49) was 44 (40 to 47). The median (IQR) number of patients seen on shifts when NTS was 40 (37 to 45) when weekend days were included (n=156) and 40.5 (37 to 48) when only weekdays were evaluated (n=52). Both teaching groups were significantly different from the NTS group (p<0.001 for the TWS group p=0.007 for the TWOS group) (Tables 1 and 2).

DISCUSSION

This study had 2 key findings. First, median LOS for patients treated during a shift with TWS was approximately 15 minutes longer than shifts with TWOS (an extra physician team) and shifts where there was no teaching service (normal ED staffing). We would advocate that this is a minimal increase given the value added in both student and resident education from the care of these patients. The presence of a teaching service was also associated with approximately 4 more patient evaluations per shift, and the number of patients seen did not decrease when third-year students were a part of the teaching service team.

A handful of studies have examined LOS in EDs in the setting of medical student education. Generally, previous studies have shown that students were associated with prolonged ED patient LOS.[4-7] A 2009 study by James et al quantified the effects of trainees on LOS when staffing with a preceptor and found that in their pediatric ED, LOS was 9% higher in patients seen by trainees.[4] Another study by Gerbeaux et al[5] corroborated these findings during a medical student strike and found that during the 4 days without medical students the LOS decreased by 24%. The James study correlates well with our own results of a 6.8% increase in LOS of the TWS service as compared to the TWOS. Both studies suggest a reduction in efficiency when students are added to care teams, as evidenced by increased LOS. However, these data were compiled in the setting of the

trainee staffing patients with the attending physician alongside the other resident providers. Other studies show no significant change in LOS under different models of teaching or were unable to quantify changes on LOS. Our study supports this finding; although there is no doubt that there was an effect on LOS, this effect was minimal. Of note, none of these studies examine fourth-year medical students, who may contribute more to patient care and LOS considering they have more clinical training.[10-12]

Limited research has been done examining the general flow of the ED in the setting of separating out trainee students. This is the first study to examine the LOS and number of patients seen during shifts with and without students on a dedicated teaching service. Our study differs from previous research in that medical student teaching can be done in a manner that does not tax the ED by significantly changing LOS or number of patients seen.

There are a few limitations of this study. First, we only evaluated LOS and number of patients seen during a specific time period (14:00 – 19:59), which impacts the generalizability of our findings to other times of day. The optimal study design to measure the effect of the teaching service on LOS would be to randomly assign the teaching service to shifts and compare LOS between shifts with and without the teaching service. However, in our setting this design is not feasible secondary to constraints of the student schedule. Given these inherent limitations, we were unable to evaluate the impact of teaching service on LOS during other times of day such as early day or night shifts. In addition, volume fluctuation on weekends and holidays when the teaching service was not present may also affect the generalizability of our results. It is also worth noting that this study looked at a teaching service with third-year medical students who are early in their clinical training; therefore, one may not be able to directly apply these findings to groups teaching medical students who are further along in their training.. We did not analyze results with regard to calendar time during the academic year, nor were we ableable to factor in any improvement in students' efficiency. Lastly, we were unable to correlate patient acuity levels to the different subgroups to determine if the TWS group saw lower acuity level patients and if this in turn impacted LOS or number of patients seen; however, this would be an interesting area of future study.

Finally, this model requires an increase in resident and attending coverage, which could increase overall cost for extra faculty. It is likely that a teaching service without additional staff may have a greater effect on LOS and decrease patient throughput. It is unclear how this study would fare at other institutions since our model requires additional staff to run the teaching service. We also did not examine fourth-year medical students, as at our institution. the third-and fourth-year medical students rotate in separate entities with the fourth years being considered the level of an "intern," and therefore staff their patients as such. During NTS days, there are no third-year medical students present in the department and therefore they are not dispersed among clinical faculty. The fourth-year medical students are always dispersed among clinical faculty and are not included in the dedicated teaching service model.

CONCLUSION

Further investigation of this teaching model is necessary to validate the effects of LOS and number of patients seen at other facilities. A prior study reveals the teaching service is well regarded,[12] but to date neither an assessment of student or resident skill has been done nor outcomes studied to evaluate the effect of a teaching service on patient care.

A teaching service in the ED is a novel educational model that provides dedicated teaching time to both students and residents amidst a busy urban academic ED. This study comprises the first evaluation of LOS and number of patients seen with the advent of a dedicated teaching service to instruct medical students. The effect of such a teaching service increased the number of patients seen during a shift and had a minimal effect on patient median length of stay.

Address for Correspondence: Courtney M. Smalley, MD. Denver Health & Hospital Authority, Department of Emergency Medicine, Campus Box B-215, 12401 E. 17th Avenue, Aurora, CO 80045. Email: courtney.smalley@ucdenver.edu.

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

REFERENCES

1. FREIDA Online. American Medical Association. Available at: https://freida.ama-assn.org/Freida/user/viewProgramSearch.do Accessed March 7, 2013.
2. Thomas HA, Beeson MS, Binder LS, et al. The 2005 Model of the Clinical Practice of Emergency Medicine: the 2007 update. Acad Emerg Med. 2008; 15:776–9.
3. The ACGME Outcome Project. ACGME core competencies. Available at: http://www.acgme.org/outcome/comp/compCPRL.asp. Accessed March 20, 2011.
4. James C, Harper M, Johnston P, et al. (2009), Effect of Trainees on Length of Stay in the Pediatric Emergency Department. Academic Emergency Medicine, 16: 859–865.
5. Gerbeaux P, Ledoray V, Liauthaud H. Medical student effect on emergency department length of stay. Ann Emerg Med. 2001; 37:275–278.
6. Lammers R, Roiger M, Rice L, et al. The effect of a new emergency medicine residency program on patient length of stay in a community hospital emergency department. Acad Emerg Med. 2003;

10:725–730.

7. Salazar A, Corbella X, Onaga H, et al. Impact of a resident strike on emergency department quality indicators at an urban teaching hospital. Acad Emerg Med. 2001; 8:804-808.
8. Burt C, McCaig L. Staffing, capacity, and ambulance diversion in emergency departments. Adv Data. 2006; 376:1–23.
9. Druck J, Houghland J, Anderson B. Teaching how to teach: a novel teaching rotation. Med Educ. 2011 May; 45(5):525-526.
10. Chan L, Kass L. Impact of medical student preceptorship on ED patient throughput time. Am J Emerg Med. 1999; 17:41–43.
11. French D, Zwemer F, Schneider S. The effects of the absence of emergency medicine residents in an academic emergency department. Acad Emerg Med. 2002; 9:1205–1210.
12. Denninghoff KR, Moye PK. Teaching students during an emergency walk-in clinic rotation does not delay care. Acad Med. 1998 Dec; 73(12):1311.

Emergency Department Length of Stay: Accuracy of Patient Estimates

Brendan T. Parker, MS
Catherine Marco, MD
University of Toledo Medical Center, Toledo, Ohio

Supervising Section Editor: Erik Barton, MD, MS, MBA
Submission History: Submitted January 5, 2013; Revision received June 25, 2013; Accepted September 16, 2013
Electronically published December 3, 2013
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.9.15816

Introduction: Managing a patient's expectations in the emergency department (ED) environment is challenging. Previous studies have identified several factors associated with ED patient satisfaction. Lengthy wait times have shown to be associated with dissatisfaction with ED care. Understanding that patients are inaccurate at their estimation of wait time, which could lead to lower satisfaction, provides administrators possible points of intervention to help improve accuracy of estimation and possibly satisfaction with the ED. This study was undertaken to examine the accuracy of patient estimates of time periods in an ED and identify factors associated with accuracy.

Method: In this prospective convenience sample survey at UTMC ED, we collected data between March and July 2012. Outcome measures included duration of each phase of ED care and patient estimates of these time periods.

Results: Among 309 participants, the majority underestimated the total length of stay (LOS) in the ED (median difference -7 minutes (IQR -29-12)). There was significant variability in ED LOS (median 155 minutes (IQR 75-240)). No significant associations were identified between accuracy of time estimates and gender, age, race, or insurance status. Participants with longer ED LOS demonstrated lower patient satisfaction scores ($p<0.001$).

Conclusion: Patients demonstrated inaccurate time estimates of ED treatment times, including total LOS. Patients with longer ED LOS had lower patient satisfaction scores. [West J Emerg Med. 2014;15(2):170–175.]

INTRODUCTION

Patient satisfaction with medical care is crucial to ensuring a healthy and productive physician-patient relationship and patient compliance with recommended therapies. Managing a patient's expectations in the emergency department (ED) environment is challenging.[1] With the ED tending to be the gateway to access care in the hospital, the perception of the hospital may be solely based on the care received in the ED. Higher satisfaction is believed to improve health outcomes, decrease litigation against the hospital, may influence the selection of ED for the next visit and the possibility of reimbursement.[2-5]

Wait times can have a huge influence, both positive and negative, on patient satisfaction.[6-9] Lengthy wait times have shown to be associated with dissatisfaction with ED care.[10-11] Wait times can be viewed two dimensionally: actual wait time (AWT) and the patient's perception of wait time (PWT). Understanding the relationship between these 2 dimensions is important because if they are inaccurate, it may be a source of unwarranted dissatisfaction. Understanding that patients are inaccurate at their estimation of wait time, which could lead to lower satisfaction, provides administrators possible points of intervention to help improve accuracy of estimation and possibly satisfaction with the ED.

This study was undertaken to establish the accuracy of the patients wait time in a university hospital ED and examine possible associations of accuracy with demographic factors. A second objective was to examine potential associations between length of stay (LOS) and satisfaction with medical care.

METHODS

Study Design

We undertook this prospective convenience sample survey to measure ED treatment and wait times, and patient estimates of these wait times. A trained research assistant verbally administered a patient survey, who then recorded the responses of each patient. The survey collected patient estimates of wait times for the following time points: arrival to triage, triage to treatment room, treatment room to nurse, treatment room to physician, lab sample collection to discharge, and total time. We defined lab tests as any test that included urine or blood, excluding radiology tests. Demographic information collected included age, gender, ethnicity, insurance status, education level, presence of a time piece, and satisfaction with the medical care they received that day based on a 5-point Likert-type scale. After the patient survey was completed, the research assistant collected data from the ED electronic medical records and recorded actual time points for each patient. This study received approval from the institutional review board.

Setting and Population

The study was undertaken at the University of Toledo Medical Center (UTMC) ED from March 2012 to July 2012. The hospital is a 320-bed Level 1 trauma center, urban, university hospital. The ED has an annual patient volume of 34,000 with a 24% admission rate. Convenience sample of all adult ED patients, age 18 and over, who were ED patients in the UTMC ED were eligible for the study. Patients who were in distress, unable to communicate or who chose not to participate were excluded from enrollment.

Data Analysis and Sample Size

The primary outcome of the study is the within-person difference between a patient's estimate of their wait time(s) and their actual wait time(s). This within-person difference was calculated as patient wait time (PWT) minus actual wait time (AWT). The raw difference between the patient's estimate of time and the actual time was then used to describe the patient's accuracy. Positive differences indicated overestimation and negative differences indicated underestimation. We summarized the differences with mean and interquartile ranges (25^{th} percentile and 75^{th} percentile), and the mean difference was tested for significance from zero using a 2-tailed paired t-test. With the study examining 4 time intervals, we used a 2-tailed p-value <0.01 to indicate statistical significance. Patient characteristics and satisfaction are described with frequency and percentage.

RESULTS

Of the 314 total respondents, data were collected on 309 patients. The 5 patients who elected not to be included in the study were for a variety of reasons not related to patient satisfaction, including the desire to leave because of their transportation and the patient determination of inability to accurately answer the questions due to the medications they had received during treatment. As described in Table 1, we gathered data across a spectrum of patients who visited the UTMC ED. Overall total LOS in the ED was defined as time of arrival to time of discharge as documented in the electronic medical records (EMR) and are reported in Table 2. In general, patients underestimated patient's total time in the ED. The median difference between patient's estimate and actual total LOS of -7 minutes (interquartile range [IQR] -29, 12) was statistically significant (p<0.001).

Results for the different time points are reported in Table 3. The number of patients analyzed for this varied depending on if they passed that checkpoint and if it was recorded properly, among other things. Looking at the different time points,

Table 1. Demographics and other characteristics of the emergency department visit, n=309.

	Freq (%)
Age (years)	
≤60	262 (85%)
>60	47 (15%)
Male	134 (43%)
Female	175 (57%)
Ethnicity	
African American	128 (41%)
Asian	4 (1%)
Caucasian	162 (52%)
Hispanic	12 (4%)
Other	3 (1%)
Insurance	
Self-pay	76 (25%)
Medicare or Medicaid	51 (17%)
Medicare and Medicaid	38 (12%)
Private insurance	97 (31%)
Multiple insurance	37 (12%)
Other	10 (3%)
Education	
Some HS	63 (20%)
HS diploma/GED	92 (30%)
Some college	103 (33%)
College degree	38 (12%)
Postgraduate degree	13 (4%)
Patient wore a watch	63 (20%)
Clock was in exam room	152 (49%)
Patient looked at cellphone	202 (65%)
Satisfaction with medical care	
Very satisfied	187 (61%)
Satisfied	79 (26%)
Neutral	25 (8%)
Dissatisfied	9 (3%)
Very dissatisfied	9 (3%)

HS, high school; *GED,* general educational development

Table 2. Overall total length of stay, n=307.

	Median	25th percentile	75th percentile	Signed-rank p-value
Patient estimate of total length of stay (LOS)	150.0	75.0	240.0	
Actual total LOS	155.0	97.0	224.0	
Difference between patient's estimate and actual total LOS*	-7.0	-29.0	12.0	< 0.001

Table 3. Time points.

	n	Patient estimate (median ±IQR)	Actual time (median ±IQR)	Difference: patient minus actual (median ±IQR)	Signed-rank test p-value on difference
1. Arrival to triage nurse	222	6 (3, 15)	9 (4, 16)	-1 (-4, 3)	0.05
2. Triage assessment to treatment room	182	10 (5, 15)	17 (11, 27)	-8 (-14, 1)	< 0.001
3. Treatment room to nurse	218	2 (1, 10)	0 (0, 4)	1 (0, 5)	< 0.001
4. Treatment room to doctor	218	15 (5, 30)	23 (11, 37)	-6 (-17, 4)	< 0.001
5. Labs to decision made about discharge	135	75 (45, 180)	99 (60, 186)	-9 (-40, 22)	0.03

IQR, interquartile range

Table 4. Subgroups.

	n	Patient estimate of LOS, min (median ±IQR)	Actual total LOS, min (median ±IQR)	Difference between total PWT and AWT (median ±IQR)	Mann Whitney Wilcoxon P-value
Gender					
Male	132	138 (83, 225)	148 (102, 214)	-7 (-26, 14)	0.5
Female	175	150 (75, 240)	157 (91, 233)	-8 (-31, 12)	
Age					
≤60 years	261	135 (75, 225)	152 (96, 220)	-8 (-29, 12)	0.72
>60 years	46	180 (90, 255)	175 (120, 248)	-5 (-16, 8)	
Ethnicity					
African American	128	120 (68, 218)	140 (91, 205)	-10 (-28, 15)	0.93
Caucasian	160	150 (90, 240)	160 (106, 225)	-7 (-30, 10)	
Other	19	180 (90, 255)	187 (117, 257)	0 (-25, 8)	
Insurance					
Self pay	75	120 (60,180)	130 (89,177)	-4 (-27,8)	
Medicaid/Medicare	89	165 (90,240)	188 (109, 247)	-11 (-32,16)	0.85
Private	96	143 (90,240)	156 (97,237)	-7 (-29, 6)	
Other	47	150 (90,210)	163 (103, 224)	-3 (-29, 12)	
Education					
Some high school	63	120 (60, 180)	131 (88, 197)	-10 (-30, 16)	
High school diploma	92	173 (90, 270)	172 (116, 240)	-9 (-26, 15)	0.63
Some college	102	120 (60, 225)	138 (77, 233)	-6 (-32, 8)	
College/Postgrad	50	173 (120, 210)	175 (123, 215)	-5 (-22, 12)	
Timepiece possession					
Presence	277	150 (90, 240)	156 (102, 226)	-6 (-27, 12)	0.14
None	30	120 (50, 180)	125 (68, 173)	-21 (-32, 2)	

LOS, length of stay; *PWT,* patient wait time; *AWT,* actual wait time; *IQR,* interquartile range

Table 5. Patient satisfaction.

	Very satisfied (median ±IQR)	Satisfied (median ±IQR)	Not satisfied (median ±IQR)	Kruskal Wallis p-value
n	186	78	43	
Patient estimate of total length of stay (LOS) (minutes)	120 (60, 210)	150 (90, 240)	195 (150, 270)	< 0.001
Actual total LOS (minutes)	141 (89, 210)	157 (102, 226)	206 (150, 257)	0.001
Difference between patient's estimate and actual total LOS*	-10 (-30, 6)	-3 (-24, 16)	-8 (-26, 19)	0.20

IQR, interquartile range

patients underestimated by 1 minute (IQR -4, 3) the time from arrival until triage nurse and significantly underestimated by 8 minutes (IQR -14, 1) triage assessment until transfer to the treatment room. Once in the treatment room, patients significantly overestimated by 1 minute (IQR 0, 5) the time until a nurse came to see the patient and significantly underestimated by 6 minutes (IQR -17, 4) the time until the patient saw the physician. If lab samples were drawn, patients underestimated by 9 minutes (IQR -40, 22) the time from when the samples were drawn until discharge. In general, we found that patients poorly estimated their wait times at 4 different time points during their visit to the UTMC ED.

Table 4 represents our data for different subgroups of our population. For gender, there was no statistically significant difference detected between male (-7 minutes (IQR -26, 14)) and female (-8 minutes (IQR -31, 12)) estimations of their LOS compared to their actual total LOS. For age, there was no statistically significant difference detected between patients under age 60 (-8 minutes (IQR -29, 12)) and those older than 60 (-5 minutes (IQR -16, 8)). There was also no statistically significant difference detected between different ethnicity's estimation and their actual total LOS. Those patients designating African American underestimated by 10 minutes (IQR -28, 15), Caucasians underestimated by 7 minutes (IQR -30, 10) and all other ethnicities collectively having a median difference of zero (IQR = -25, 8). For insurance status no statistically significant difference was detected between self pay (-4 minutes (IQR -27, 8)), Medicare/Medicaid (-11 minutes (IQR -32, 16)), private insurance (-7 minutes (IQR -29, 6)), and those categorized as "Other" (-3 minutes (IQR -29, 12)). Patients who carried more than one health insurance, military insurance or others were put into one category of "Other." For education, while we found a decrease in the difference between PWT and AWT with more education, there was no statistically significant difference found between patients with some high school (-10 minutes (IQR -30, 16)), high school diploma (-9 minutes (IQR -26, 15)), some college (-6 minutes (IQR -32, 8)) and college/postgraduate degree (-5 minutes (IQR -22, 12)). Although we found a large difference between having a timepiece (-6 minutes (IQR -27, 12)) versus not having one (-21 minutes (IQR -32, 2)) during the visit, our data found no statistically significant difference. We defined having a time piece as whether the patient had a watch, the presence of a clock in the room or whether the patient looked at their cell phone for any reason during their visit. In our study, we saw no statistically significant difference between accuracy of estimations and gender, age, ethnicity, insurance status, education or presence of a time piece with the patient.

Our study, as reported in Table 5, found correlations between LOS and patient satisfaction. In regards to total estimated LOS, we found a statistically significant difference between patients who described their experience as "very satisfied" (120 minutes (IQR 60, 120)), "satisfied" (150 minutes (IQR 90, 240)), and "not satisfied" (195 minutes (IQR 150, 270)). People who described their experience as "neutral," "dissatisfied," or "very dissatisfied" were grouped under "not satisfied." In regards to total actual LOS, we also found a significant difference with our 3 satisfaction categories. Although we found differences in estimation and actual total length of stay, the difference between estimated and actual total LOS had no statistical difference between the 3 categories. In the figure, we broke down percentage of patients in each patient satisfaction category and compared that to total length of time by hour. For any patients with actual total LOS over 7 hours, they were grouped into the 7-hour category. Trend lines were provided in the figure to show the general change for each satisfaction category. The top trend line shows a decrease in the percentage of "very satisfied" patients while the middle and bottom trend lines show an increase in the percentage of "satisfied" and "not satisfied" patients respectively. In general, our study showed that with an increase in time spent in the ED, there is a decrease in patient satisfaction. While we found no correlation between inaccuracy and satisfaction level, we did find longer ED LOS was associated with lower satisfaction.

DISCUSSION

Previous studies have demonstrated that ED patients are inaccurate in estimating their wait times in the ED.[10-12] Many factors, such as perceived severity of their case and

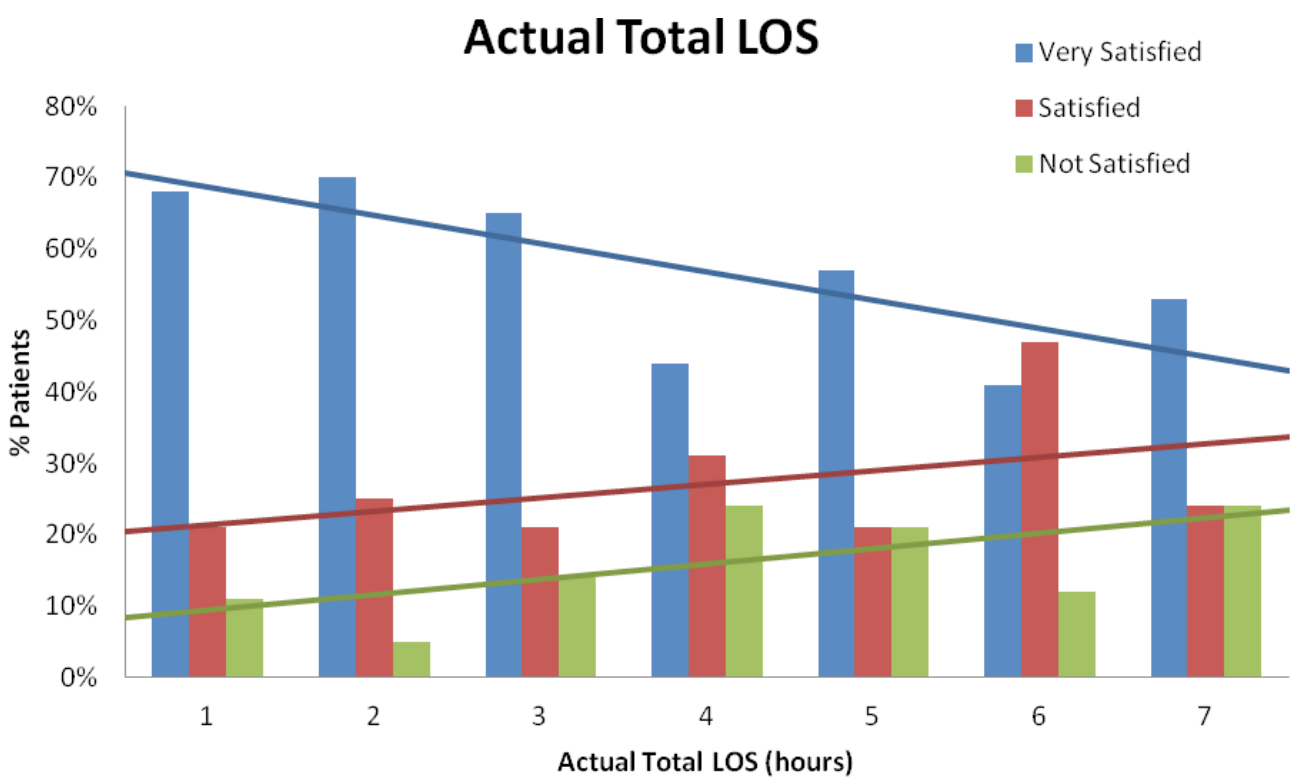


Figure. Patient satisfaction with actual total length of stay (LOS).

activity in the ED at the time of visit, can influence how a patient perceives time. The data we collected, which was surprising and different than the literature,[10,12] showed that patients collectively underestimated their total LOS. Our data also suggest a small interquartile range for the difference between the patient's estimation and actual total LOS. This suggests that while patients are significantly inaccurate at their estimation ($p<0.001$), they are remarkably closer than first hypothesized. With multiple time points having a significant difference between the perception and actual time, our study suggests that during the entire process of an ED visit, patients are relatively inaccurate at estimating the treatment time.

Understanding that ED patients have a variety of backgrounds in the ED, our study also examined subgroups that included gender, age, ethnicity, insurance status, education and presence of a time piece. Our initial intention was to look for any points of intervention that administration could use to help those whose perception was inaccurate improve, therefore possibly improving their satisfaction. Our study suggests that there are no differences in estimations and actual wait times based on these 6 subgroups. Further studies into patient's perception of urgency or pain may bring insight into reasons for inaccuracy.

Another aspect of our study that provided insight into wait times was the correlation between LOS and patient satisfaction. Patients who were very satisfied had shorter wait times, while those that were not satisfied had longer wait times. Decline in satisfaction is a major concern for the ED as it may lead to less patient compliance with recommended treatments, poor health outcomes, and increased litigation. In this study, patients who had decreased satisfaction commented on the lack of communication between the staff and patient. While each patient's expected stay varies depending on their complaint, future research could look at how to improve communication with staff to help improve patient satisfaction.

LIMITATIONS

This study was conducted at a single institution and results may not be generalizable to other locations. As a prospective convenience sample, the study may not be completely reflective of the population. The study relied on patient self reports and the accuracy depends on patient effort and veracity. As the study was verbally administered, some questions and statements could have varied between patients therefore our patients' responses and data could be altered. With verbal administration from a trained research assistant, patient satisfaction may have been slightly altered with the perception that the institution was giving the patient preferential treatment. Other factors related to patient satisfaction were not included in this study, such as patient expectations of treatment, crowding of the ED or politeness of staff. Satisfaction may have also been altered as the survey was sometimes administered right after painful medical injections or with the staff in the room performing different tasks.

CONCLUSION

Patients demonstrated inaccuracy in estimation of ED treatment times, including total LOS. We found no significant

associations between accuracy of estimations of treatment times and age, gender, ethnicity, insurance status, education or presence of a time piece. Patients with longer ED LOS had lower patient satisfaction scores.

Address for Correspondence: Brendan T. Parker, MS. Department of Emergency Medicine, The University of Toledo Medical Center, 3000 Arlilngton Ave. MS 1088, Dowling Hall 2455, Toledo, OH 43614. Email: brendan.parker@rockets.utoledo.edu.

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

REFERENCES

1. Lateef F. Patient expectations and the paradigm care shift of care in emergency medicine. *J Emerg Trauma Shock.* 2011;4(2):163-167.
2. Waitzkin H. Doctor-patient communication. Clinical implications of social scientific research. *JAMA.* 1984;252:2441-2446
3. Linn LS, Brook RH, Clark VA, et al: Physician and patient satisfaction as factors related to the organization of internal medicine group practices. *Med Care.* 1985;23:1171-1178
4. Mack JL, File KM, Horwitz JE, et al. The effect of urgency on patient satisfaction and future emergency department choice. *Health Care Manage Rev.* 1995;20:7-15
5. Freudenheim M. In a Shift, an H.M.O. Rewards Doctors for Quality Care. *The New York Times*. July 11, 2001
6. Cordell W, Olinger M, Kozak, et al. Does Anybody Know What Time It Is? Does Anybody Really Care? *Ann Emerg Med*. 1994;23(5):1032-1036.
7. Mowen JC, Licata J, McPhail J. Waiting in the Emergency Room: How to Improve Patient Satisfaction. *J Health Care Marketing* 1993;13(2):26-33.
8. Thompson, D, Yarnold P. Relating Patient Satisfaction to Waiting Time Perceptions and Expectations: The Disconfirmation Paradigm. *Acad Emerg Med.* 1995;2(12):1057-1062.
9. Tran P, Schutte W, Muelleman R, et al. Provision of Clinically Based Information Improves Patient's Perceived Length of Stay and Satisfaction with EP. *Am J Emerg Med*. 2002;20(6):506-509.
10. Waseem M, Ravi L, Radeos M, et al. Parental Perception of Wait Time and Its Influence on Parental Satisfaction in an Urban Pediatric Emergency Department: Are Patients Accurate in Determining Wait Time? *South Med J*. 2003;96(9):880-883.
11. Little N. Image of the Emergency Physician. *Emergency Medicine Risk Management: A Comprehensive Review*. 1991;11-16.
12. Thompson D, Yarnold P, Adams S, et al. How Accurate are Waiting Time Perceptions of Patients in the Emergency Department. *Ann Emerg Med.* 1996;28(6):652-656.
13. Tran T, Schutte W, Muelleman R, et al. Provision of Clinically Based Information Improves Patients' Perceived Length of Stay and Satisfaction with EP. *Am J Emerg Med.* 2002;20(6):506-509.

Impact of Decontamination Therapy on Ultrasound Visualization of Ingested Pills

Jason Bothwell, MD*
Carl Skinner, MD*
David Della-Giustina, MD†
Christopher Kang, MD*
Laura Cookman, MD*
Brooks Laselle, MD*

* Madigan Army Medical Center, Department of Emergency Medicine, Tacoma, Washington
† Yale School of Medicine, Department of Emergency Medicine, New Haven, Connecticut

Supervising Section Editor: Jeffrey R. Suchard, MD
Submission history: Submitted May 17, 2013; Revision received August 24, 2013; Accepted October 28, 2013
Electronically published February 26, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.10.18112

Introduction: Acute toxic ingestion is a common cause of morbidity and mortality. Emergency physicians (EP) caring for overdose (OD) patients are often required to make critical decisions with incomplete information. Point of care ultrasound (POCUS) may have a role in assisting EPs manage OD patients. We evaluated the impact of different liquid adjuncts used for gastric decontamination on examiners' ability to identify the presence of tablets using POCUS, and assessed examiners' ability to quantify the numbers of tablets in a simulated massive OD.

Methods: This prospective, blinded, pilot study was performed at an academic emergency department. Study participants were volunteer resident and staff EPs trained in POCUS. Five non-transparent, sealed bags were prepared with the following contents: 1 liter (L) of water, 1 L of water with 50 regular aspirin (ASA) tablets, 1 L of water with 50 enteric-coated aspirin tablets (ECA), 1 L of polyethylene glycol (PEG) with 50 ECA, and 1 L of activated charcoal (AC) with 50 ECA. After performing POCUS on each of the bags using a 10-5 MHz linear array transducer, participants completed a standardized questionnaire composed of the following questions: (1) Were pills present? YES/NO; (2) If tablets were identified, estimate the number (1-10, 11-25, >25). We used a single test on proportions using the binomial distribution to determine if the number of EPs who identified tablets differed from 50% chance. For those tablets identified in the different solutions, another test on proportions was used to determine whether the type of solution made a difference. Since 3 options were available, we used a probability of 33.3%.

Results: Thirty-seven EPs completed the study. All (37/37) EP's correctly identified the absence of tablets in the bag containing only water, and the presence of ECA in the bags containing water and PEG. For Part 2 of the study, most participants - 25/37 (67.5%) using water, 23/37 (62.1%) using PEG, and all 37 (100%) using AC - underestimated the number of ECA pills in solution by at least 50%.

Conclusion: There may be a potential role for POCUS in the evaluation of patients suspected of acute, massive ingested OD. EPs accurately identified the presence of ECA in water and PEG, but underestimated the number of tablets in all tested solutions. [West J Emerg Med. 2014;15(2):176–179.]

INTRODUCTION

Background

Acute toxic ingestion is a common cause of morbidity and mortality. In 2011 the Annual Report of the American Association of Poison Control Centers' National Poison Data System recorded more than 2.3 million accidental or intentional toxic exposures and 1,158 deaths.[1] Emergency physicians (EP) caring for overdose (OD) patients are

often required to make critical decisions with incomplete information regarding the substance(s), quantity, or time elapsed since ingestion. In cases of suspected overdose, patients may be obtunded and unable to provide essential information or may withhold details of the ingestion.

Importance

Due to this uncertainty, EPs often rely on diagnostic testing to supplement their clinical suspicion. This may involve laboratory analysis for serum drug concentrations or imaging for the identification of pills in the stomach. Both plain radiography and computed tomography have been described, but neither is reliable and both involve radiation exposure.[2-3] Furthermore, neither can be performed in real time at the bedside by the treating physician. A few case reports and one small prospective trial describe ultrasound for identification of pills in the stomach.[4-7] However, there are no published data on the ability of practitioners to visualize ingested tablets during decontamination therapy with activated charcoal or polyethylene glycol, both of which are commonly administered to overdose patients. This is of potential consequence in pre-hospital care situations, where some protocols initiate decontamination therapy prior to hospital arrival.

The massive OD presents a unique problem for EPs. Not only do patients have the potential to rapidly decompensate, but many medications often believed to be relatively benign can have devastating consequences. Some experts recommend more aggressive hemodialysis for suspected massive overdoses, as gastric decontamination parameters may be altered.[8] In these cases, any test that successfully identifies the massive OD could potentially alter management and outcome. As this time, there are no prospective studies evaluating EPs' ability to quantify pills in a suspected massive overdose.

Study Objectives

The primary objective of this feasibility study was to evaluate the impact of the presence of different liquid adjuncts used for gastric decontamination on examiners' ability to identify the qualitative presence of tablets using ultrasound. A secondary objective was to assess examiners' ability to quantify the numbers of tablets in a simulated massive overdose.

METHODS

Study Design and Selection of Participants

This prospective, blinded pilot study was approved by the institutional review board. Study participants were volunteer emergency medicine resident and staff physicians from an academic emergency department. Eligible participants previously completed a minimum 2-day ultrasound training course consisting of 16 hours of basic and advanced point of care ultrasound applications. An emergency medicine intern performed recruitment to avoid any appearance of coercion, and provided an information and "opt out" sheet. As there was no risk to participants, informed consent was waived. Prior to participation, all participants watched a training video demonstrating tablets under ideal sonographic conditions. The video consisted of a 6-second clip showing 50 enteric-coated aspirin in 1 liter of water. (Participants were not informed of which tablet or substance was represented in the video clip.

To compare the visibility of a simulated massive OD of tablets in 3 different media, 5 sealed bags were prepared with the following contents: 1 liter of water, 1 liter of water with 50 regular aspirin (ASA) tablets, 1 liter of water with 50 enteric-coated aspirin tablets (ECA), 1 liter of polyethylene glycol (PEG) with 50 ECA, and 1 liter of activated charcoal (AC) with 50 ECA.

The contents were placed in black, opaque plastic sealable bags, thus blinding the participants to the contents . Prior to sealing, bags were manually squeezed to eliminate air. Bags were placed in random order on a table in a linear arrangement. One at a time, participants performed ultrasounds on the bags using a Zonare One™ (Zonare Medical Systems, Mountain View CA) ultrasound machine with a 10-5 MHz linear array transducer. No restrictions were imposed on the order of bags scanned, the number of scans per bag or scanning time. The study took place in 3 separate sessions, 1 week apart, with random numbering for each bag prior to each session. No bag was used for longer than 1 hour, to minimize the effects of pills dissolving during the study.

Data Collection and Processing

After scanning, participants filled out a brief, standardized questionnaire responding to the following questions: (1) Were pills present? YES/NO; (2) If pills were identified, estimate the number (1-10, 11-25, >25). The primary investigator manually transferred data into an electronic spreadsheet Microsoft Excel 2007™ (Microsoft Corporation, Redmond, WA) at the conclusion of each session, and one of the associate investigators then independently the data.

Outcome Measures

The primary outcome was the ability to detect the qualitative presence of pills in water and in the different gastric decontamination media (PEG and AC). The secondary outcome was the ability of the participants to accurately estimate the number of pills in solution.

Primary Data Analysis

All data were analyzed using Microsoft Excel 2007™ (Microsoft Corporation, Redmond, WA). We used a single test on proportions using the binomial distribution to determine if the number of physicians who saw tablets differed from 50% chance. For those tablets that were identified in the different solutions, we used another test on proportions to determine whether the type of solution made a difference. Since there were 3 options from which to choose, we used a probability of 33.3% in the calculation.

Table 1. Participants' visualization of tablets in different solutions.

Solution	# Tablets	# Participants (out of 37) who saw tablets	# Correct	p-value	95% Confidence interval
Water	0	0	37/37 (100%)	< 0.0005	90.6-100%
Water	50 ASA	2	2/37 (5%)	< 0.0005	1.5-17.7%
Water	50 ECA	37	37/37 (100%)	< 0.0005	90.6-100%
Polyethylene glycol	50 ECA	37	37/37 (100%)	< 0.0005	90.6-100%
Activated charcoal	50 ECA	20	20/37 (54%)	0.116	38.4-69%

ASA, aspirin; *ECA,* enteric-coated aspirin

Table 2. Estimated number of tablets visualized in various solutions.

		Estimated # of tablets seen				
Solution	# Tablets	1 to 10	11 to 25	>25	Total correct	p-value
Water	0	0	0	0	37 (100%)	< 0.0005
Water	50 ASA	1	0	1	1 (3%)	0.444
Water	50 ECA	8	17	12	12 (32%)	0.138
Polyethylene glycol	50 ECA	9	14	14	14 (38%)	0.113
Activated charcoal	50 ECA	18	2	0	0 (0%)	< 0.0005

ASA, aspirin; *ECA,* enteric-coated aspirin

RESULTS

A total of 37 physicians completed the study, and no participant required more than 5 minutes. The number of physicians who visualized tablets in various solutions is shown in Table 1. For those physicians who saw (any) tablets in a solution, the number they estimated for that solution is shown in Table 2. For Part 1 of the study, all (37/37) study participants correctly identified:

(1) the presence of ECA in the bag containing water
(2) the presence of ECA in the bag containing PEG

All 37/37 (100%) participants were also accurate in determining the absence of ECA tablets in a water-only model where pills were non-existent, and 35/37 (95%) did not visualize ASA in water. For Part 2 of the study, most participants -- 25/37 (67.5%) using water, 23/37 (62.1%) using PEG, and all 37 (100%) using AC -- underestimated the number of ECA pills in solution by at least 50%.

DISCUSSION

This simple bag model suggests that point of care ultrasound is potentially useful for detecting the presence of tablets in a massive OD, but less useful for quantifying them. Participants were accurate in determining the absence of tablets in a water-only model and in accurately determining the presence of ECA in both water and in PEG. This suggests that ECA is readily visualized in these solutions with ultrasound by EPs.

AC appears to substantially interfere with the sonographic visualization of ECA, given that only 20/37 (54%) correctly identified the presence of ECA tablets in the bag containing AC. This was not significantly different from random chance (p=0.116). When tablets were visualized in AC, the estimated number was very low compared to the total number of pills present. If ultrasound is used clinically, it should be noted that any AC impairs accurate visualization. Participants were also generally unable to identify regular ASA in water (2/37, 5%), likely because the tablets had dissolved. In our own experience with this experiment using clear plastic bags, regular ASA dissolves within 60 seconds when placed in room-temperature water. This suggests that the use of ultrasonography to detect regular aspirin tablets may not be feasible. ECA, however, remains intact and visible with ultrasound in solution for well over an hour, with only minor flaking of the coating.

Regardless of the solution tested, the majority of participants substantially underestimated the number of tablets when they were present. Even though each tablet-containing bag held 50 tablets, only 12/37 (32%) of participants estimated more than 25 tablets in water, and 14/37 (38%) estimated more than 25 tablets in PEG. For the tablets in AC, none of the participants estimated greater than 25 tablets. Applying this to an actual patient-care situation, any tablets visualized on ultrasound in a human stomach would potentially be an underrepresented of the total number present, and any search for tablets in a suspected OD should be performed prior to the administration of AC. As always, the clinical presentation of the patient would factor into the interpretation of any ultrasound images. However, it appears that underestimation would be the most common error in quantifying the size of the ingestion.

This is the first controlled trial, to our knowledge, that examines the effects of gastric decontamination solutions on the accuracy of diagnostic ultrasound. It is also the first study to evaluate participants' ability to quantify tablets in solution. The use of ultrasound for detection of pills in the stomach was first described in the late 1980s,[5] but there have been few studies since then expanding on this early foundation of knowledge. Our study suggests the feasibility of ultrasound as a tool for identifying ECA tablets in certain solutions. Clarifying the role of point of care sonography in massive OD requires further investigation. This study, performed in artificial but sonographically ideal conditions, needs validation in human subjects. Other warranted investigations include the sonographic properties of capsules, sustained- release preparations, other enteric-coated medications, and whether sonographic properties vary between medications or due to the specific coatings applied to those medications.

The use of point of care ultrasound has increased substantially in recent years. As outlined in the most recent ultrasound guidelines from the American College of Emergency Physicians a number of applications are "evolving."[9] As EPs expand their familiarity with ultrasound technology, it is likely that the list of applications will expand as well, possibly to include gastric ultrasound for pill identification in cases of massive ingestion.

LIMITATIONS

Only 2 types of tablets were evaluated in this study: regular ASA and ECA. Accordingly, these results may not be applicable to other medications or formulations. We used 50 tablets to simulate a "massive overdose." This number can be debated and clearly varies based on the medication ingested. However we feel this number is a reasonable estimate of a fatal ingestion for over-the-counter medications that are readily available and commonly consumed during intentional ingestions. Finally, this in vitro study using bag models and non-physiologic solutions for a simulated massive OD cannot be extrapolated to human subjects. It is expected that visualizing pills in a plastic bag would be easier than doing so under normal clinical conditions.

CONCLUSION

There may be a potential role for point of care ultrasound in the evaluation of the acutely poisoned patient. We found that regular ASA in solution is difficult to identify with ultrasound, but that ECA is easily identified in water and in PEG. AC, however, appears to substantially impair visualization of ECA. We also discovered that EPs are likely to significantly underestimate the number of tablets present. Further studies will be required to evaluate and validate the potential role for this application in patients with acute ingestions.

Address for Correspondence: Jason Bothwell, MD, Department of Emergency Medicine, Madigan Army Medical Center, 3844 Long Lake Loop SE, Lacey, WA 98503. Email: jasonbothwell@yahoo.com.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The views expressed are those of the author(s) and do not reflect the official policy of the Department of the Army, the Department of Defense or the United States Government.

REFERENCES

1. Bronstein AC, Spyker DA, Cantilena LR, et al. 2011 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 28th Annual Report. *Clin Toxicol*. 2011;49(10):910-941.
2. Savitt DL, Hawkins HH, Roberts JR. The Radiopacity of Ingested Medications. *Ann Emerg Med*. 1987;16(3):331-339.
3. O'Brien RP, McGeehan PA, Helmeczi AW, et al. Detectability of Drug Tablets and Capsules by Plain Radiography. *Am J Emerg Med*. 1986;4(4):302-312.
4. Amitai Y, Silver B, Leikin J, et al. Visualization of Ingested Medications in the Stomach by Ultrasound. *Am J Emerg Med*. 1992;10(1):18-23.
5. Maublant JC, Hassine H, Sournac M, et al. Ultrasonic Visualization of Tablets in the Gastrointestinal Tract. *J Nuc Med*. 1988;29(1):129.
6. Schlager D. Ultrasound detection of foreign bodies and procedure guidance. *Emerg Med Clin North Am*. 1997;15(4):895-912.
7. Nordt SP, Campbell C, Medak A, et al. Ultrasound Visualization of Ingested Tablets: A Pilot Study. *Pharmacotherapy*. 2011;31(3):273-276.
8. Erickson T. The New Overdoses. Course lecture presented at: American College of Emergency Physicians, *Scientific Assembly,* October 2008; Chicago, IL.
9. American College of Emergency Physicians Policy Statement: Emergency Ultrasound Guidelines. October, 2008.

Impact of an Abbreviated Cardiac Enzyme Protocol to Aid Rapid Discharge of Patients with Cocaine-associated Chest Pain in the Clinical Decision Unit

Faheem W. Guirgis, MD*
Kelly Gray-Eurom, MD, MMM*
Teri L. Mayfield, MD*
David M. Imbt, MD*
Colleen J. Kalynych, MD*
Dale F. Kraemer, PhD†
Steven A. Godwin, MD*

* University of Florida, Department of Emergency Medicine, Jacksonville, Florida
† University of Florida College of Medicine, Jacksonville; Center for Health Equity and Quality Research

Supervising Section Editor: Amal Mattu, MD
Submission history: Submitted August 9, 2013; Revision received November 5, 2013; Accepted November 22, 2013
Electronically published February 25, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.11.19232

Introduction: In 2007 there were 64,000 visits to the emergency department (ED) for possible myocardial infarction (MI) related to cocaine use. Prior studies have demonstrated that low- to intermediate-risk patients with cocaine-associated chest pain can be safely discharged after 9-12 hours of observation. The goal of this study was to determine the safety of an 8-hour protocol for ruling out MI in patients who presented with cocaine-associated chest pain.

Methods: We conducted a retrospective review of patients treated with an 8-hour cocaine chest pain protocol between May 1, 2011 and November 30, 2012 who were sent to the clinical decision unit (CDU) for observation. The protocol included serial cardiac biomarker testing with Troponin-T, CK-MB (including delta CK-MB), and total CK at 0, 2, 4, and 8 hours after presentation with cardiac monitoring for the observation period. Patients were followed up for adverse cardiac events or death within 30 days of discharge.

Results: There were 111 admissions to the CDU for cocaine chest pain during the study period. One patient had a delta CK-MB of 1.6 ng/ml, but had negative Troponin-T at all time points. No patient had a positive Troponin-T or CK-MB at 0, 2, 4 or 8 hours, and there were no MIs or deaths within 30 days of discharge. Most patients were discharged home (103) and there were 8 inpatient admissions from the CDU. Of the admitted patients, 2 had additional stress tests that were negative, 1 had additional cardiac biomarkers that were negative, and all 8 patients were discharged home. The estimated risk of missing MI using our protocol is, with 99% confidence, less than 5.1% and with 95% confidence, less than 3.6% (99% CI, 0-5.1%; 95% CI, 0-3.6%).

Conclusion: Application of an abbreviated cardiac enzyme protocol resulted in the safe and rapid discharge of patients presenting to the ED with cocaine-associated chest pain. [West J Emerg Med. 2014;15(2):180–183.]

INTRODUCTION

In 2007 there were 2.1 million cocaine users leading to 64,000 emergency department (ED) visits to evaluate for possible myocardial infarction (MI).[1] Approximately 57% of these patients were admitted to the hospital at an annual cost of $83 million.[1] Cocaine use has significant cardiovascular complications, including myocardial infarction, arrhythmias, aortic dissection, hypertensive crises, cardiomyopathy, and endocarditis.

Cocaine is well absorbed through all body mucous membranes and can be administered through several routes. The

Table 1. Demographics of 101 patients treated in clinical decision unit for cocaine chest pain.

Descriptor	Rate (SD)
Patients (n)	101
Visits (n)	111
Patients with >1 visit (n)	8
Average age	41 (10.2)
Race	80% Black, 20% White
Documented cocaine use	88%
Sex	69% male, 31% female
Average systolic BP (mmHg)	133 (22.4)
Average diastolic BP (mmHg)	80 (15.7)
Average heart rate	93 (19.3)

BP, blood pressure

onset of action varies from 3 seconds to 5 minutes depending on the route of administration. Also dependent on the route of administration are peak effects and duration of action, which vary from 1 to 20 minutes and 5 to 90 minutes, respectively.[1]

A 24-fold increased risk of MI has been reported within 1 hour of cocaine use and two-thirds of MI events occur within 3 hours of cocaine ingestion.[2,3] However, Amin et al[4] reported an 18-hour median length of time between cocaine use and MI onset among 22 patients presenting after cocaine ingestion. Other studies reported a range extending from 1 minute up to 4 days.[3] The extended time frame may be secondary to metabolites that cause delayed or recurrent vasoconstriction.[5]

Generally accepted MI rates range from 0.7% to 6% of patients with cocaine-associated chest pain. These low rates of MI have prompted some hospitals to use observation units to reduce the number of admissions.[6,7] In a prospective trial of 302 low- to intermediate-risk patients who underwent observation there were no cardiac deaths noted. Only 2% of patients sustained a non-fatal MI, and only 1% of patients sent home had a cardiac complication.[8] Another retrospective review found that out of 187 patients observed in a chest pain unit, 87% were discharged and only 1% had a cardiac complication.[9] A separate long-term prospective study found that out of 219 patients there was a zero rate of myocardial infarction at 1 year.[10]

Prior studies have demonstrated that low- to intermediate-risk patients with cocaine-associated chest pain can be safely discharged after 9-12 hours of observation.[8] Additional literature suggests that in patients with undifferentiated chest pain, a rapid rule-out protocol using 2-hour delta CK-MB measurements is safe and effective.[11,12] Therefore, our institution developed an abbreviated 8-hour cocaine-associated chest pain protocol for monitoring patients in the clinical decision unit (CDU). The goal of this study was to determine if patients treated with the 8-hour protocol were safely discharged from the CDU.

METHODS

This was an institutional review board approved (University of Florida College of Medicine, Jacksonville) retrospective review of patients treated in a CDU for cocaine-associated chest pain under the 8-hour protocol between May 1, 2011 and November 30, 2012.. To qualify for treatment in the CDU, patients had to be considered low to intermediate risk and meet the following inclusion criteria: 1) history of cocaine use within 72 hours; 2) normal EKG or EKG showing no acute changes and no left bundle branch block; 3) first set of cardiac markers (CK, CK-MB, and Troponin-T) negative; and 4) Thrombolysis in myocardial infarction risk score (TIMI) 0-3. We excluded patients if any of the following were true: 1) prior history of coronary artery disease (CAD), stents, or coronary artery bypass graft (CABG); 2) prior history of valvular disease, cardiomyopathy, or rhythm disturbance; 3) systolic blood pressure >160 or diastolic blood pressure >100 at time of transfer; 4) prior history of diabetes mellitus; 5) other causes of life-threatening chest pain; 6) active chest pain at time of transfer; 7) less than 18 years of age.

For patients who met inclusion criteria, the 8-hour protocol encompassed serial cardiac biomarker testing with Troponin T, CK-MB, and total CK at 0, 2, 4 and 8 hours after presentation to the ED. The rapid rule out protocol used delta CK-MB at 0, 2, and 4 hours to evaluate for a rise in CK-MB of 1.6 ng/mL for each 2-hour interval.[11,12] A CK-MB >5 ng/ml with >5% fraction of the total CK was considered positive. Troponin-T was considered elevated if >0.04 ng/ml. If the patient's cardiac biomarkers became positive at any point, the patient was consulted to the cardiology service and admitted for further workup. If cardiac enzymes remained negative, there was no change in the EKG, and the patient was free of chest pain or arrhythmia, the patient was discharged following the 8-hour observation period without additional testing.

We identified potential patients for inclusion in this study by reviewing the CDU chest pain log for patients treated between May 1, 2011 and November 30, 2012. Data collected on identified patients included initial vital signs, EKG findings, chest radiographs, cardiac biomarkers (Troponin-T, CK-MB, total CK), drug testing (if available), time of cocaine ingestion, disposition, and repeat visits for chest pain, adverse cardiac events or death within 30 days of CDU admission.

To determine death within 30 days, we searched hospital records for patient visits greater than 30 days after discharge from the CDU. For those who had not visited the hospital greater than 30 days post initial visit, we used the Social Security Death Index and Florida Vital Statistics databases to determine if patients expired and if death was within 30 days of treatment with the protocol. If the patient did expire, the cause of death was recorded. We reviewed charts for primary and secondary outcomes; primary outcomes were positive cardiac biomarkers, adverse cardiac events, or death within 30 days of discharge. Secondary outcomes were discharge versus admission and specialist consultations. If admitted, cardiac enzymes, stress testing, procedures, and discharge summaries were reviewed and recorded.

We entered the data collected into REDCap (Research

Table 2. Outcomes for 101 patients treated in clinical decision unit for cocaine chest pain.

Outcome	Rate
Positive cardiac biomarkers	0
Positive ΔCK-MB	1 (1.6 ng/mL)
Adverse cardiac events or death <30 days	0
Admissions	8
Cardiology consultations	6
Positive additional cardiac biomarkers	0
Stress Testing/Interventions	2 Stress tests – negative
Abnormal chest radiograph	0
Electrocardiogram changes	0

Electronic Data Capture), hosted at the University of Florida and transferred to STATA12 (StataCorp LP, College Station, Texas). Frequency statistics were used to describe the study population and any chest-pain related care or death within 30 days of presenting to the ED. We calculated mean and standard deviations for numeric statistics. Exact 95% confidence intervals were provided by STATA 12's cii command.

RESULTS

There were 101 unique patients comprising 111 admissions to the CDU for cocaine-associated chest pain during the study period. Eight patients had more than 1 visit; 1 patient had 4 visits and 7 patients had 2 visits each. Patient demographics are in Table 1.

One patient had a delta CK-MB of 1.6ng/ml, but had negative Troponin-T at all time points. There were no patients with positive Troponin-T or CK-MB, notable EKG changes, or pertinent abnormal chest radiographs over the observation period (Table 2). Additionally, there were no myocardial infarctions and no deaths during the 30-day follow-up period. Most patients (103) were discharged home from the CDU, while 8 were admitted to the hospital. Of the 8 admitted patients, 2 had stress tests that were negative and 1 had additional cardiac biomarkers, which were also negative. All 8 patients were subsequently discharged home, and none were diagnosed with new cardiac disease or acute coronary syndrome upon discharge.

None of the patients included in this study were found to have any of the primary outcomes (MI or death within 30 days); therefore, we were unable to determine the true sensitivity of our 8-hour protocol for ruling out MI in patients with cocaine-associated chest pain. However, the estimated risk of missing a patient with positive cardiac biomarkers, adverse cardiac event, or death within 30 days, using our protocol is almost surely less than 5.1% and most likely less than 3.6% (exact 95% CI, 0-3.6%; exact 99% CI, 0-5.1%) based on a zero event rate.

DISCUSSION

Cocaine-associated chest pain is a frequently encountered complaint in the ED, especially in urban inner city hospitals. In this study, the use of an ED observation unit for the monitoring and treatment of patients with cocaine-associated chest pain showed that an 8-hour protocol using delta CK-MB and frequent Troponin-T assays appears to be safe in patients who have used cocaine within 72 hours of presentation.

In general, the incidence of MI in cocaine users presenting with chest pain is low, and cocaine use does not predispose patients to coronary artery disease after adjusting for other risk factors.[13,14] Because of this, low- to intermediate-risk patients with cocaine-associated chest pain can usually be observed in the ED without admission to the hospital. The use of ED observation units has allowed for the extended treatment of patients with cocaine-associated chest pain outside of the acute care area; however, bed space in observation units can be limited. Given the low risk of adverse cardiac events in this population it is reasonable to attempt to safely shorten the duration of observation for these patients.

Current evidence supports observation periods of 9 to 12 hours for low- to intermediate-risk cocaine-associated chest pain.[8] Ruling out cardiac events more rapidly should involve the use of additional imaging, provocative testing, or more frequent serum biomarkers for detecting myocardial damage. Hendel and colleagues reported on the use of single-photon emission computed tomography myocardial perfusion imaging (MPI) in 151 patients with cocaine chest pain and found that it effectively ruled out cardiac events and lowered admission rates.[15] Paraschin and colleagues applied cardiac CT angiography (CTA) in 24 cocaine users presenting with chest pain. They found that cardiac CTA was normal in 96% of patients, and positive in only 1 patient (4%) who had non-significant coronary stenosis.[16] The use of imaging studies and/or provocative testing can expedite patient disposition; however, the risks of contrast allergies, contrast-induced nephropathy, and radiation exposure are all important considerations.

Our approach used a "rapid rule-out" protocol to safely and expeditiously rule out adverse cardiac events in patients presenting with cocaine-associated chest pain.[11,12] This approach allowed for rapid disposition and discharge without radiation exposure from additional imaging or the resources required for provocative testing. In our cohort, only 2 patients required additional stress testing and 103 patients were discharged safely from the CDU after the 8-hour protocol. No patient experienced an adverse cardiac event, a positive provocative test, or positive additional cardiac biomarkers. Further, there were no deaths found in this cohort within 30 days of discharge. We believe we have demonstrated that the described 8-hour cocaine-associated chest pain protocol is safe and effective in this population.

LIMITATIONS

First, this retrospective study identified patients by reviewing the CDU chest pain logs. Although we performed

a judicious search, the possibility of missing a small number of patients cannot be excluded. Second, the lack of positive cardiac biomarkers, adverse cardiac events, or deaths in our cohort limits our ability to make a true estimate about the sensitivity of the 8-hour protocol. However, we believe that our data give a realistic representation of patients who frequent urban EDs with complaints of cocaine-associated chest pain. Our data are reflective of the literature, in that the low- to intermediate-risk group of patients presenting with cocaine-associated chest pain has an inherently low rate of MI. Given our low estimated risk of no more than 5.1%, we feel it is safe to use our protocol in this population for the rule out of MI.

CONCLUSION

Our study demonstrated that an 8-hour abbreviated protocol safely ruled out MI in patients presenting to the ED with cocaine-associated chest pain. This protocol has the advantages of shortening ED length of stay, limiting radiation exposure, and reducing provocative testing.

ACKNOWLEDGMENTS

The authors would like to thank Rita H. Duke for the many hours spent performing chart reviews and entering data. This manuscript would not have been possible without her tireless efforts. We would also like to thank Dr. Robert L. Wears MD, MS, PhD for his statistical expertise and helpful advice.

Address for Correspondence: Faheem W. Guirgis, MD, 655 W 8th St, Jacksonville, FL 32209. Email: faheem.guirgis@jax.ufl.edu

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

REFERENCES

1. Maraj S, Figueredo VM, Lynn Morris D. Cocaine and the heart. *Clin Cardiol*. 2010;33(5):264-269.
2. Hollander JE, Hoffman RS. Cocaine-induced myocardial infarction: An analysis and review of the literature. *J Emerg Med*. 1992;10(2):169-177.
3. Mittleman MA, Mintzer D, Maclure M, et al. Triggering of myocardial infarction by cocaine. *Circulation*. 1999;99(21):2737-2741.
4. Amin M, Gabelman G, Karpel J, et al. Acute myocardial infarction and chest pain syndromes after cocaine use. *Am J Cardiol*. 1990;66(20):1434-1437.
5. Brogan WC, 3rd, Lange RA, Kim AS, et al. Alleviation of cocaine-induced coronary vasoconstriction by nitroglycerin. *J Am Coll Cardiol*. 1991;18(2):581-586.
6. Feldman JA, Fish SS, Beshansky JR, et al. Acute cardiac ischemia in patients with cocaine-associated complaints: Results of a multicenter trial. *Ann Emerg Med*. 2000;36(5):469-476.
7. Weber JE, Chudnofsky CR, Boczar M, et al. Cocaine-associated chest pain: How common is myocardial infarction? *Acad Emerg Med*. 2000;7(8):873-877.
8. Weber JE, Shofer FS, Larkin GL, et al. Validation of a brief observation period for patients with cocaine-associated chest pain. *N Engl J Med*. 2003;348(6):510-517.
9. Kushman SO, Storrow AB, Liu T, et al. Cocaine-associated chest pain in a chest pain center. *Am J Cardiol*. 2000;85(3):394-396, A10.
10. Cunningham R, Walton MA, Weber JE, et al. One-year medical outcomes and emergency department recidivism after emergency department observation for cocaine-associated chest pain. *Ann Emerg Med*. 2009;53(3):310-320.
11. Fesmire FM, Percy RF, Bardoner JB, et al. Serial creatinine kinase (CK) MB testing during the emergency department evaluation of chest pain: Utility of a 2-hour deltaCK-MB of +1.6ng/ml. *Am Heart J*. 1998;136(2):237-244.
12. Fesmire FM. Delta CK-MB outperforms delta troponin I at 2 hours during the ED rule out of acute myocardial infarction. *Am J Emerg Med*. 2000;18(1):1-8.
13. Chang AM, Walsh KM, Shofer FS, et al. Relationship between cocaine use and coronary artery disease in patients with symptoms consistent with an acute coronary syndrome. *Acad Emerg Med*. 2011;18(1):1-9.
14. McCord J, Jneid H, Hollander JE, et al. Management of cocaine-associated chest pain and myocardial infarction: A scientific statement from the american heart association acute cardiac care committee of the council on clinical cardiology. *Circulation*. 2008;117(14):1897-1907.
15. Hendel RC, Ruthazer R, Chaparro S, et al. Cocaine-using patients with a normal or nondiagnostic electrocardiogram: Single-photon emission computed tomography myocardial perfusion imaging and outcome. *Clin Cardiol*. 2012;35(6):354-358.
16. Paraschin K, Guerra De Andrade A, Rodrigues Parga J. Assessment of myocardial infarction by CT angiography and cardiovascular MRI in patients with cocaine-associated chest pain: A pilot study. *Br J Radiol*. 2012;85(1015):e274-278.

Factors Important to Applicants to Osteopathic Versus Allopathic Emergency Medicine Residency Programs

Bruce Alfred St. Amour, DO St. Mary Mercy Center, Department of Emergency Medicine, Livonia, Michigan

Supervising Section Editor: Anita Eisenhart, DO
Submission history: Submitted May 13, 2013; Revision received October 8, 2013; Accepted October 28, 2013
Electronically published February 25, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.10.18164

Introduction: Our objective is to evaluate the factors important to osteopathic applicants when selecting an American College of Osteopathic Emergency Physicians accredited emergency medicine (EM) residency and to compare these results with previous allopathic EM studies.

Methods: We gave osteopathic applicants a survey during interview season to be filled out anonymously at the end of their interview day. This survey included 18 factors which the applicants were asked to rank between 1 ("not very important") to 4 ("very important"). We then compared results to prior results of the same survey.

Results: Forty applicants (67%) out of 60 completed the survey. From these individuals, we noticed differences in the top factors listed by the applicants when compared to allopathic interviewees, the most notable being the unimportance of geographic location of the program to osteopathic applicants as manifested by osteopathic student average score of 2.8 (standard deviation 0.75) verses allopathic student average of 3.6 (standard deviation 0.06).

Conclusion: Of the top 5 factors listed by the applicants, only 1 (AOA-approved residency) is an objective factor that the program has a role in controlling. The remainder are mainly subjective factors based on applicant's perceptions of the program. [West J Emerg Med. 2014;15(2):184–187.]

INTRODUCTION

Individuals graduating from medical school are increasingly choosing emergency medicine (EM) as the choice of residency.[1] Previously DeSantis and Marco concluded that the top 5 factors important to students when choosing a preferred residency program include friendliness, environment, interview experience, academics, and location,[2]. In a study by Laskey and Cydulka EM residency graduates were asked to look back and rate the importance of 18 items influencing residency choice.[3] They found that institutional reputation, hospital facilities, program director reputation, and spousal influence were most important in making this decision. Other studies have asked similar questions of students going through the allopathic (MD) match process.[4,5]

The osteopathic (DO) match process for EM is slightly more complicated. The DO match for 216 positions is administered approximately one month before the MD match for 1,556 positions. Osteopathic students are eligible for positions in the MD residency but the opposite is not true. If an osteopathic student matches in the DO match then they are automatically withdrawn from the MD match. Each year a number of DO students withdraw from the DO match in hopes of matching to a MD program,[6] but there is no guarantee of success. The factors affecting the DO students' choice of residency are not clear. Such knowledge may be helpful in the process of designing new and existing EM programs and would provide useful knowledge to program directors. To our knowledge, there have been no studies that explore this topic.

Therefore, the specific aim of this study is to determine factors important to applicants to an osteopathic EM residency when selecting a program. The secondary goal is to compare these factors to those of allopathic EM programs.

METHODS

This was a cross-sectional study identifying the key factors used when considering a specific osteopathic EM

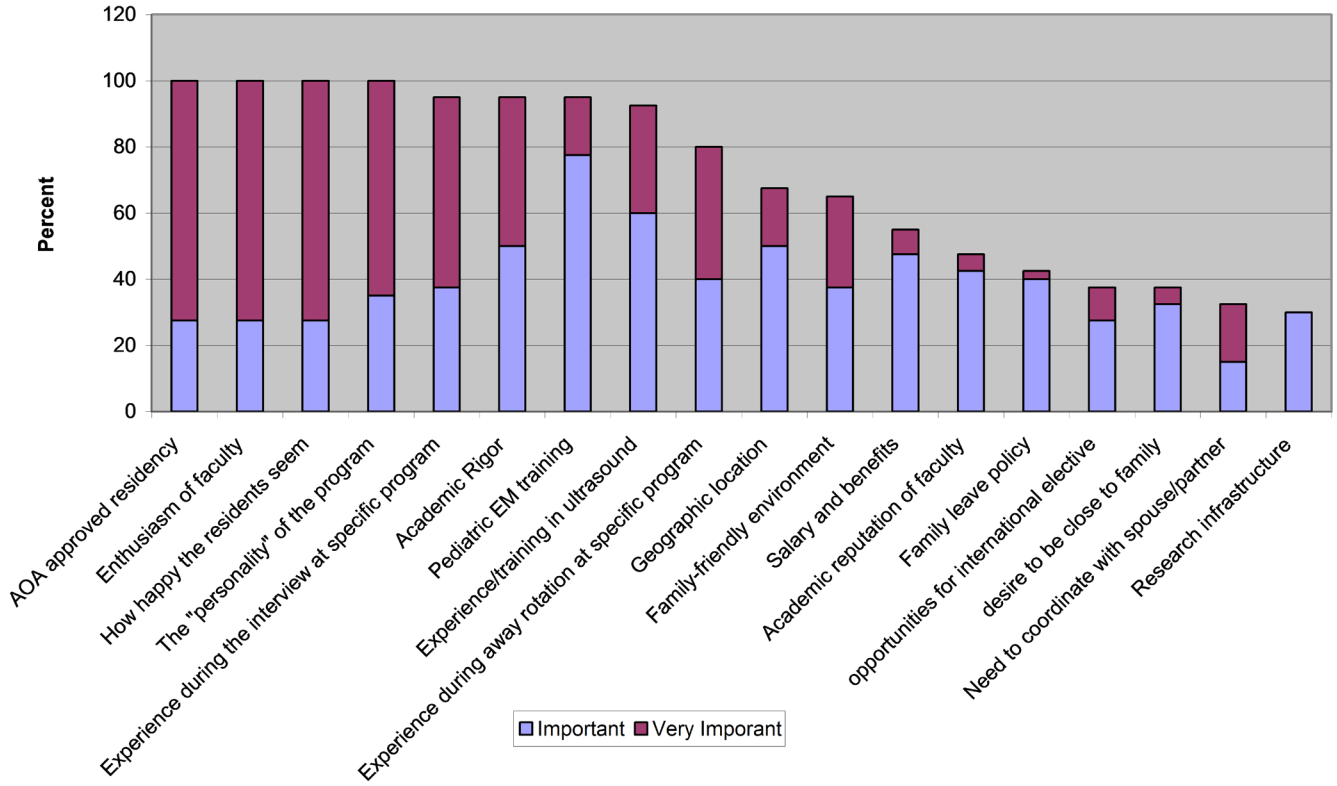


Figure 1. Percent important and very important.

residency program. After institutional review board approval, we administered the survey to all applicants who interviewed at the St. Mary Mercy Hospital EM Residency during the 2012-2013 residency selection season. Dr. Yarris graciously gave the survey tool to us for direct comparison. Participation in the survey was voluntary, and data were anonymous.

Study Setting and Population

This study was conducted at the graduate medical education offices at St Mary Mercy Hospital, where the residency applicants spend the majority of their interview day. The survey was distributed to all applicants on each interview date along with the rest of their interview day materials by the residency coordinator. An information sheet explaining the research intent of the survey, and explicitly stating that participation was voluntary, accompanied the survey. All applicants present on interview day were eligible to participate. The survey was administered on interview days, which typically fall on Wednesdays during the months of September through December.

The applicants had the opportunity to complete, or not complete, the survey in private, and then place it in a clearly labeled, locked box. The survey did not contain any identifying information, and the investigators were not able to determine which applicants chose to participate in the study, nor were they able to link individual responses with specific applicants. We did not analyze the data until after the selection committee formulated the rank list, and no member of the residency selection committee had access to the survey results until after the rank list was made.

Measurements

The survey instrument used a 4-point interval scale for responses (Appendix). The survey included questions to assess the relative importance of several factors that applicants may consider in selecting an EM residency. The remaining questions asked demographics information, including age, gender, and race.

Data Analysis

We collapsed Likert-scale responses to factors considered by applicants into “important” and “not important”, where the “important” factors were marked “somewhat important” or “very important.” We included all other responses in the “not important” group. All comparisons were assessed for the presence of confounding factors such as age and gender of subject were considered as potential confounders. We compared differences between demographic groups using a chi-square test for homogeneity of binomial proportions.

RESULTS

Of the 60 applicants who interviewed between October and December 2012, 40 completed the questionnaire (66.7%). Two respondents did not answer the demographic questions. Of the 38 reporting their age in our study, the mean was 28 years, with a range from 24 to 43. Sixteen of the respondents

(42%) were women. The majority of our participants were medical students, which is consistent with most new applicants to residency. Thirty-nine were fourth-year medical students and the other was a first-year resident. In the paper published by Yarris et al,[4] there was a close balance between individuals who were single and married (45% and 49% respectively). In our study, 14 (36.8%) were married or in a committed relationship while the remaining individuals stating they were single (63.2%). Sixty-seven percent of the respondents described themselves as, ethnically, white. We ranked the questions by the percent responding "Important" or "Very Important," and that ranking is shown in Figure 1.

DISCUSSION

As a specialty, the number of applications to EM residencies continues to increase, producing a more competitive environment for the applicants to navigate. To our knowledge, this is the first study to address these factors for osteopathic candidates. The program location for the Yarris[4] study is a 4-year residency (the majority of allopathic programs are 3 years in length). Our study is similar, in that all EM programs approved by American Osteopathic Association are 4-year programs. This allowed stronger direct comparisons between applicants.

One of the intentions of this study was to see what similarities and differences occur between DO and MD applicants. When compared with Yarris et al,[4] the top factors that applicants look for are program personality, faculty enthusiasm and how happy the residents appear. This is similar to our survey results. The major difference was location, ranking fourth on their results, whereas with our survey geographic location settled to the bottom 50% of factors important to applicants. The reason for this may be that the number of osteopathic EM programs is significantly less, and to acquire a slot in these programs, one must be flexible to move to the location that has selected them. Another reason may be that 82% of residencies are in areas prone to snow, and very few are in warm climates.

The importance of first impressions appears to hold true. When a program shows a kind, concordant and pleasing presentation towards their prospective candidates, they have an increased possibility of attaining the persons they would like to have for their program. Additionally, factors for applicants that are of lesser importance were research infrastructure and desire to be close to family. This is also similar to Yarris et al,[4] who demonstrated that salary and family leave policy are in the lower tier of importance for applicants. Comparisons between the osteopathic program and the allopathic program are included in Figure 2.

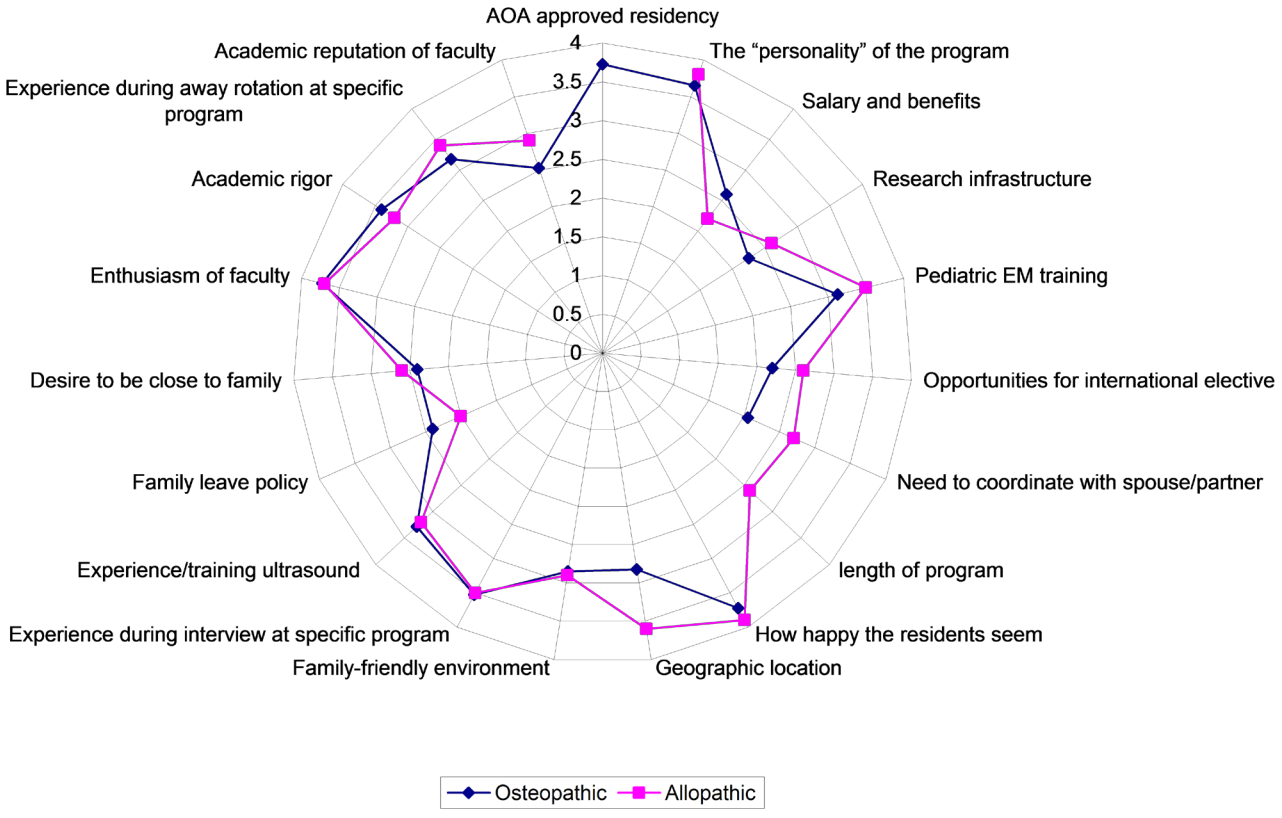


Figure 2. Allopathic versus osteopathic programs.

LIMITATIONS

The limitations in our study are similar to those of other surveys. It was limited to one smaller community hospital; however, the majority of osteopathic EM training is done in smaller community hospitals. Also, the number of interviewees at our program may not be similar to other programs throughout the country. It is also difficult to define what the "personality" of a program is, and some subjectivity is present with the individual applicants. Additionally, since we had a response rate of 66.7%, we were missing additional data from the other one-third of persons who did not respond.

CONCLUSION

Osteopathic EM residencies continue to become increasingly competitive. Information that can help both applicants and program directors with this search is valuable. In our study, the most important factors were happy residents, faculty enthusiasm and program personality, which is similar to allopathic programs.

Address for Correspondence: Bruce Alfred St. Amour, DO. 220 49th St. Des Moines, IA 50312. Email: bruce_st_amour@hotmail.com.

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

REFERENCES

1. Bland KI, Isaacs G. Contemporary trends in student selection of medical specialties: the potential impact on general surgery. *Arch Surg.* 2002;137:259–267.
2. DeSantis M, Marco CA. Emergency medicine residency selection: factors influencing Candidate Decisions. *Acad Emerg Med.* 2005; 12:559-561.
3. Laskey S, Cydulka RK. Applicant Cconsiderations Aassociated with Sselection of an emergency medicine residency program. *Acad Emerg Med.* 2009;16(4):355-359.
4. Yarris L, Delorio N, Lowe R. Factors applicants value when selecting an emergency medicine residency. *West J Emerg Med.* 2009;10(3):159-162.
5. National Resident Matching Program, Data Release and Research Committee: Results of the 2009 NRMP Applicant Survey by Preferred Specialty and Applicant Type. Available at: http://www.nrmp.org/data/ApplicantResultsBySpecialty2009.pdf.
6. National Matching Service, Outcome of all Applicants 2012 Available at: http://www.natmatch.com/cgibin/direct_filedisp.pl.

4,871 Emergency Airway Encounters by Air Medical Providers: A Report of the Air Transport Emergency Airway Management (NEAR VI: "A-TEAM") Project

A National Emergency Airway Registry Study

Calvin A. Brown III, MD*
Kelly Cox, MD†
Shelley Hurwitz, PhD‡
Ron M. Walls, MD*

* Brigham and Women's Hospital, Harvard Medical School, Department of Emergency Medicine, Boston, Massachusetts
† University of Illinois-Peoria, Department of Emergency Medicine, Peoria, Illinois
‡ Brigham and Women's Hospital, Harvard Medical School, Department of Medicine, Boston, Massachusetts

Supervising Section Editor: Christopher Kang, MD
Submission history: Submitted June 13, 2013; Revision received October 3, 2013; Accepted November 11, 2013
Electronically published February 25, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.11.18549

Introduction: Pre-hospital airway management is a key component of resuscitation although the benefit of pre-hospital intubation has been widely debated. We report a large series of pre-hospital emergency airway encounters performed by air-transport providers in a large, multi-state system.

Methods: We retrospectively reviewed electronic intubation flight records from an 89 rotorcraft air medical system from January 01, 2007, through December 31, 2009. We report patient characteristics, intubation methods, success rates, and rescue techniques with descriptive statistics. We report proportions with 95% confidence intervals and binary comparisons using chi square test with p-values <0.05 considered significant.

Results: 4,871 patients had active airway management, including 2,186 (44.9%) medical and 2,685 (55.1%) trauma cases. There were 4,390 (90.1%) adult and 256 (5.3%) pediatric (age ≤ 14) intubations; 225 (4.6%) did not have an age recorded. 4,703 (96.6%) had at least one intubation attempt. Intubation was successful on first attempt in 3,710 (78.9%) and was ultimately successful in 4,313 (91.7%). Intubation success was higher for medical than trauma patients (93.4% versus 90.3%, p=0.0001 JT test). 168 encounters were managed primarily with an extraglottic device (EGD). Cricothyrotomy was performed 35 times (0.7%) and was successful in 33. Patients were successfully oxygenated and ventilated with an endotracheal tube, EGD, or surgical airway in 4809 (98.7%) encounters. There were no reported deaths from a failed airway.

Conclusion: Airway management, predominantly using rapid sequence intubation protocols, is successful within this high-volume, multi-state air-transport system. [West J Emerg Med. 2014;15(2):188–193.]

INTRODUCTION

Out-of-hospital tracheal intubation is an accepted but controversial practice. Prior research on patient outcomes after pre-hospital intubation has yielded mixed results with several studies suggesting that, in select groups of trauma patients, it may increase mortality.[1-5] Training, protocols, oversight, and frequency of individual provider intubation all might influence success rates and clinical outcome, and previous reports may not represent success rates possible from highly skilled clinicians working in high volume systems.[6,7] Previous small studies of air transport intubations have found success rates from 66% to 97%, with higher success seen after the institution of rapid sequence intubation (RSI) protocols.[8-13] We report a large consecutive series of intubations performed within an 89 rotorcraft, multi-state transport system in order to characterize methods, success rates and rescue techniques

when intubation is performed by flight paramedics and nurses using standardized rapid-sequence intubation protocols.

METHODS

Study Design

This is a retrospective analysis of consecutive intubations performed by paramedics and flight nurses. Institutional review board approval was obtained at both Brigham and Women's Hospital and the University of Illinois –Peoria.

Setting

Intubations were performed by flight personnel within a large air medical company based in the central part of the United States. The company operates 89 206L Long Ranger helicopters flying from 85 bases. The system services more than 1,000 hospitals and records an average of 26,000 flights per year with a combination of inter-facility transfers (60.2%) and on-scene calls (39.8%). Most flight bases are rural, stand-alone structures and are not associated with urban or academic medical centers. Intubations were performed by paramedics and nurses with advanced airway management training. All providers underwent robust quarterly training sessions consisting of seminars on airway assessment, difficult airway management and rapid sequence intubation under the tutelage of regional medical directors. Lectures focused on difficult airway detection, RSI pharmacology and airway management in specific clinical scenarios, such as head injury and shock. Additionally, case-based skills sessions, utilizing METI ECS simulators of differing ages (neonatal, pediatric and adult), were mandatory with special attention placed on straight and curved blade direct laryngoscopic technique, augmentation maneuvers (optimal external laryngeal manipulation) and the use of rescue airways for difficult intubations. Each paramedic and flight nurse had to perform 4 intubations under the supervision of the regional medical director. Airway scenarios were pulled from actual flight logs during which particular airway challenges or hazards were encountered. These scenarios ranged from facial and airway trauma to airway obstruction to airway management in the morbidly obese. During the simulator sessions, providers were tested not only on intubation technique and tube placement but also on correct dosing, timing and application or RSI medications. Extraglottic device and cricothyrotomy (both percutaneous and surgical) techniques were also practiced every 3 months. Paramedics and flight nurses had an average of 12 years of prehospital experience and logged annually more than 25 airway procedures through a combination of field intubations and simulated airway scenarios. Two-thirds of these airway procedures are performed in simulation. Airway management was guided by standard intubation protocols, reviewed and approved annually by the senior medical director. Indications for airway management include failure of oxygenation and ventilation, inadequate airway protection, or airway deterioration during transport. Protocols outlined the indication for pretreatment agents (i.e. lidocaine and fentanyl for head injury with presumed elevated intracranial pressure), use of induction agents and neuromuscular blockers for rapid sequence intubation, and algorithms for crash and failed airway management. Induction agents included etomidate, midazolam and ketamine. Paralytics included succinylcholine, rocuronium and vecuronium. Standard drug doses were used for airway management and are listed in Table 1.

Table 1. Rapid sequence intubation drugs and intravenous doses.

Drug	Dose
Succinycholine	1.5 mg/Kg
Rocuronium	1.0 mg/Kg
Vecuronium	0.1 mg/Kg
Etomidate	0.3 mg/Kg
Ketamine	1.5 mg/Kg
Midazolam	0.3 mg/Kg
Fentanyl	1-3 µg/Kg
Lidocaine	1.0 mg/Kg
Atropine	0.02 mg/Kg

Selection of Participants

We included all adult and pediatric patients who underwent either an intubation attempt or placement of an extraglottic device by any system flight personnel over a 36-month period from 1/1/07 to 12/31/09 in our analysis. Patients intubated by flight crew personnel but not flown (ongoing cardiopulmonary arrests in the field who were taken by ground to the closest local hospital or patients who never ultimately left the original facility) were not included in the database and therefore not analyzed. No significant changes were made to protocols or training during this time.

Methods of Measurement

We categorized each intubated patient as either a medical or trauma intubation based on information provided to dispatchers or gathered on scene. A difficult airway assessment was done, whenever possible, by each operator prior to administration of medication and, if appropriate for neuromuscular blockers, rapid sequence intubation was performed. The operator evaluated mouth opening, cervical spine mobility, mandible and tongue size and location of the thyroid cartilage prior to airway intervention. This approach has been used successfully to predict difficult direct laryngoscopy in emergency department patients [14,15] In the absence of significant predicted difficulty, the intubator performed orotracheal rapid sequence intubation by direct laryngoscopy. An extraglottic device (combitube, laryngeal mask airway or King laryngeal tube) was used as the initial airway maneuver if the patient was deemed to be too difficult to safely receive neuromuscular blockade. Extraglottic devices placed as the first planned method of airway control

Figure 1. Coverage map for Air Evac EMS, Inc (2010).

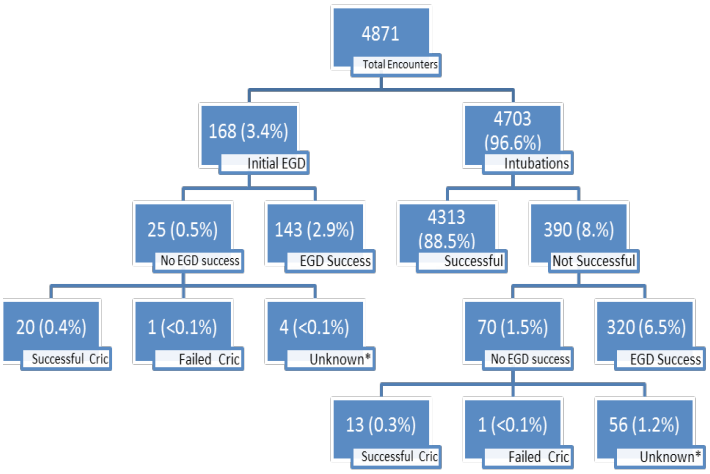


Figure 2. Breakdown of airway management encounters. *CRIC,* cricothyrotomy*; EGD,* extraglottic device

occurred with sedation only. In-flight cardiopulmonary arrests with a crash airway were managed with immediate direct laryngoscopy, without RSI meds, followed by extraglottic device placement if laryngoscopy failed. A failed airway was defined as either a failed intubation attempt in concert with an oxygen saturation below 90% despite maximal supplemental oxygen and assisted ventilations or 3 failed attempts by an experienced operator.[14] Failed airways were managed, per protocol, with either a rescue device (combitube, laryngeal mask airway or laryngeal tube), continued bag mask ventilation, or cricothyrotomy. Malleable stylets were used in the endotracheal tubes during intubation and all blunt trauma patients were placed in cervical collars on-scene. Helicopters were not equipped with video larygoscopes. Each patient transport generated a flight record as well as a standardized intubation report that was completed in real time by the operator. Whether an intubation took place is a demand function on the electronic flight record and cannot be bypassed. Recorded variables included the patient age, sex, estimated weight, flight classification, protocol and drugs used, initial airway management maneuver, number of intubation attempts, intubation success, and use of rescue devices. During the registry period, an intubation attempt was defined as insertion of any laryngoscope blade with passage of an endotracheal tube beyond the patient's lips. Confirmation of endotracheal tube placement was done by auscultation and colorimetric end-tidal CO_2 detection. Capnography was used during transport. Successful endotracheal intubation was defined by tube passage resulting in chest rise, and color change on a colorimetric end-tidal CO_2 detector. Vitals signs were noted to be "stable" or "unstable" both before and after intubation but specific values were not contained in the database. Intubation reports were monitored for completeness by regional medical directors with a reporting compliance of 100%.

Data Collection and Processing

After each flight, operators recorded flight and intubation data onto a structured data form. Data were reviewed for completeness and entered by the medical director and nurse educator into the company's main database (SQL database, Microsoft Access™, Redmond, WA). We then imported the data into a spreadsheet (Microsoft Excel™, Redmond, WA) for analysis. Structured queries were performed to retrieve data relevant to primary and secondary endpoints.

Primary Data Analysis

We present descriptive data with 95% confidence intervals (95% CI) where appropriate and binary comparisons using chi square test. Analysis was completed using SAS 9.13 (SAS Institute, Cary, NC).

RESULTS

During the review period, 4,916 intubations were recorded into the database. Forty-five intubations were not performed by prehospital personnel and were excluded, resulting in 4,871 intubations available for analysis, including 2,186 (44.9%) medical and 2,685 (55.1%) trauma cases. Given the total flight volume, approximately 6% of annual flights required crew members to perform advanced airway procedures during transport. There were 4,390 (90.1%) adult and 256 (5.3%) pediatric (age ≤ 14) intubations with a mean age of 46.1 years. 225 (4.6%) encounters did not have an age recorded. Of these, 3,155 patients (64.8%) were male and 1,716 (35.2%) were female.

Of 4,871 encounters, 4,703 (96.6%) underwent an orotracheal intubation attempt as the first airway maneuver (Figure 2). First attempt intubation success was 78.9% (n=3710, 95% CI 77.7, 80.0) and ultimate intubation success was 91.7% (n=4313, 95% CI 90.9, 92.5). Of successful intubations, 96.7% (n=4171) were intubated in 2 or fewer attempts and 99.9% (n=4308) in 3 attempts or less. Five

Table 2. Attempts for successful versus unsuccessful intubations.

Success	Attempts	Frequency	Cumulative frequency
No	1	22	22
No	2	143	165
No	3	181	346
No	4	44	390
Yes	1	3710	4100
Yes	2	461	4561
Yes	3	137	4698
Yes	4	5	4703

Table 3. Success medical versus trauma.

Success	Medical	Trauma	Cumulative frequency
No	139 (6.6%)	251 (9.67)	390
Yes	1986 (93.4%)	2345 (90.3%)	4313
Total	2107	2596	4703

patients received a fourth attempt. Overall, the intubation success rate was higher for medical patients than for trauma patients (93.4% versus 90.3%; p=.0001 JT test, Table 2, Table 3).

For unsuccessful intubations (n=390), 22 (5.6%) had 1 attempt, 143 (36.7%) had 2 attempts, 181 (46.4%) had 3 attempts, and 44 (11.3%) patients had 4 attempts before another management method was performed. Of these patients, 350 (90%) had an attempt at extraglottic device (EGD) placement as the first rescue maneuver which was successful in 320 cases. The King LT was successful 91.6% of the time (207/226 attempts), the combitube was successful 90.1% (100/111 attempts) and the laryngeal mask airway, although used rarely, was successful 100% of the time (13/13). Fourteen patients underwent cricothyrotomy, six as the first rescue method (all of which were successful) and eight after a failed attempt at EGD use, with one failure from the latter group. For the remaining 56 (14.4%) patients, no additional details were recorded although, per protocol, they would have been managed with prolonged bag and mask ventilation.

One hundred sixty-eight (3.4%) of all patients were initially managed with an extraglottic device without an antecedent attempt at endotracheal tube placement. In this group, successful oxygenation with an EGD occurred in 143 (85.1%) and was unsuccessful in 25 (14.9%). Rescue cricothyrotomy was attempted in 21 of the 25 (80%) patients for whom EGD management was unsuccessful, with only one failure. Four cases had no additional details available, although per protocol they would have been mask ventilated until transport was complete. Operators reported no deaths during transport due to a failed airway. Overall, successful oxygenation and ventilation (successful intubation, EGD use or surgical airway) was 98.7% (n=4809, 95%, CI 98.4, 99.0).

DISCUSSION

Air-medical teams often take care of the most severely compromised patients for whom early and decisive airway management may have a direct impact on patient outcome.[6,7] Tracheal intubation, however, is a complex procedure requiring extensive knowledge of airway anatomy, human physiology, pharmacology and various rescue strategies should intubation fail. Even in the simplest of scenarios, intubation represents a high-stakes situation for every patient. Reporting quality care and procedural competency is paramount in this select group of critical-care providers. In this system, which involves specialized training and a skills maintenance program, complemented by protocol-driven practice, airway management with tracheal intubation and selected use of extraglottic devices is performed with high levels of success. Our finding of a 91.7% intubation success rate and a 98.7% oxygenation and ventilation success rate confirms results from smaller studies and suggests these findings may be typical of other air-medical programs.[8-13,15] Additionally, this contributes to our understanding of air-transport airway management competency by evaluating intubation procedures and success, on a large scale, within a private, non-academic program and suggests that high-level airway management is likely ubiquitous with these specialized providers.

Surgical airways were performed at a similar rate and with similar success to prior published reports of pre-hospital cricothyrotomy.[16,17] Extraglottic devices were used early and often in our study population, with the King laryngeal tube used most frequently. All EGDs had high rates of placement and successful use suggesting these tools continue to be a helpful adjunct for pre-hospital airway management. The baseline skill set of our operators was high. This particular air transport company provides an extensive airway training program for new employees and maintains skills through quarterly training sessions, all with strict oversight by regional medical directors. It is difficult to gauge the exact impact of this educational regimen on intubation success, although augmenting real-life experience with extensive simulation is unlikely to make their performance worse. We do know that intubation volume (or lack thereof) is linked to performance. Early investigations of out-of-hospital intubation success by ground providers have shown varying results, but as many as 16% of pre-hospital intubations may be unsuccessful.[18] Minimal airway management requirements for paramedic certification and skill degradation because of infrequent field intubations have been blamed for these inconsistent results.[19,20] Procedural performance is associated with frequent repetition, and paramedics in high volume settings have shown higher rates of intubation success; however, most EMS systems cannot provide high volume procedural exposure.[21] One review from Pennsylvania found that endotracheal intubation took place in only 0.7 percent of all patient encounters in 2003. Two-thirds of providers performed 2 or fewer intubations and nearly 40% did not perform any.[19] Although a causal relationship cannot

be made given the study design, our findings suggest that this group of pre-hospital providers who receive elaborate initial training and ongoing assessments to maintain intubation skill, practicing within a protocol-driven system for uniformity, perform advanced airway management with a high degree of success. While we await equal performance from ground personnel, a trend has developed that shows specially trained air-transport providers have the skills required for safe and effective emergency airway management. Going forward, intubation trends seen in hospital settings should be investigated further in the pre-hospital arena. This includes the integration of video laryngoscopes and optical devices, the refinement of difficult airway protocols and development of pre-intubation checklists. Air transport personnel should continue to be on the vanguard of airway management research.

LIMITATIONS

Our study has some important limitations. First, these intubations were performed by highly skilled paramedics and flight nurses within a high volume air-transport company, predominantly during inter-facility transfers; therefore our results represent what operators with a similar skill set and exposure would likely achieve and may not be generalizable to all pre-hospital providers. Second, self-reported data have intrinsic limitations, and under-reporting of poorly performed intubations including those with multiple failed attempts and adverse events is possible. Close compliance monitoring by regional medical directors and mandatory reporting fields for intubation in the electronic flight record, and a reporting compliance of 100% limits the possibility of selective reporting. Additionally, the structured data form was designed for internal, administrative purposes and was not originally created with a developed research protocol. This has resulted in some non-standard definitions and variables in our data pool. Looking ahead, operational definitions can be clarified during a prospective collection phase. First, the definition used for tracheal intubation attempt required actual attempted insertion of an endotracheal tube, while the most widely accepted definition of intubation attempt simply requires insertion of the laryngoscope blade beyond the teeth. Therefore, our finding that 3.4% of all patients had an EGD device placed as the first airway maneuver may not be exact if some of those patients underwent direct laryngoscopy (without an attempt at tracheal tube placement) first. This may also result in an overestimation of the first attempt success rate found in our sample. However, even if every patient who had an EGD placed was categorized as a first attempt failure, the first attempt success rate remains high. Second, many data points that would have been helpful in refining our results were not collected. Vital signs surrounding intubation were noted to be "stable" or "not stable," although specific values were not recorded. In addition, it was not specified when, exactly, the intubation took place whether before lift off or in-flight. The latter group would pose unique challenges because of the physical constraints of a small rotorcraft. We are unable to make detailed statements about intubations that took place while in the air.

Drugs used during intubation were documented in the registry as classifications of medications instead of specific agents. In other words when RSI was performed, etomidate or midazolam could have been used in standard doses if the patient was hemodynamically stable. Either drug would have qualified as the "induction" agent for the electronic record; however, the specific drug was not documented. This makes intubation comparisons stratified by particular drugs impossible. Finally, destination tube confirmation was not recorded, and therefore an overestimation of intubation success within our sample is possible. [22-25] All tubes, however, were verified with auscultation, chest rise, and colorimetric ETC02 detection. In addition, waveform capnography was a recommended part of the post-intubation care during transport, making the chance of an unrecognized misplaced tube very low. We did not include patients intubated by Air Evac EMS personnel but not flown because of critical instability, ongoing CPR or death in the field included in our analysis since a transport flight record was not generated for these encounters. Certainly, this population had high morbidity and mortality and perhaps some may have died as a result of failed airway management. Not knowing or including this group in our analysis biases our findings towards better performance and may have left out serious airway complications that escaped our surveillance. Approximately 1% of our study population (60 patients) had no recorded successful airway maneuver. It is impossible to know how these encounters ended. Since there were no operator reports of patient death due to a failed airway, one interpretation is that these patients were successfully bag-and-mask ventilated until transport was complete. Another reasonable conclusion is that the lack of information on these encounters means we may be under-reporting deaths due to failed airway management.

This group of advanced-level providers underwent advanced simulation airway training multiple times per year as part of routine competency checks. Although simulation used to augment real-life experience is generally considered beneficial, the effect this extra training has on final performance is difficult to gauge.

Finally, our primary outcome was successful intubation rather than neurologic function or mortality. How these performance characteristics translate to patient survival and function requires further study.

CONCLUSION

In this high volume air transport agency, skilled pre-hospital providers, using rapid sequence intubation protocols, are successful with airway management techniques including tracheal intubation and ventilation with an extraglottic device. Surgical airways are rarely required but likely to be successful when performed. These data may be used to better define attainable performance and training standards for pre-hospital air transport providers responsible for emergency airway management.

Women's Hospital, Department of Emergency Medicine, 75 Francis Street, Boston, MA 02115. Email: cabrown@partners.org.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. Dr. Cox is the medical director for Air Evac EMS, Inc.

REFERENCES

1. Davis DP, Hoyt DB, Ochs M, et al. The effect of paramedic rapid sequence intubation on outcome in patients with severe traumatic brain injury. *J Trauma.* 2003;54(3):444-453.
2. Winchell RJ, Hoyt DB. Endotracheal intubation in the field improves survival in patients with severe head injury. Trauma Research and Education Foundation of San Diego. *Arch Surg.* 1997;132(6):592-597.
3. Sloane C, Vilke GM, Chan TC, et al. Rapid sequence intubation in the field versus hospital in trauma patients. *J Emerg Med.* 2000;19(3):259-264.
4. Murray JA, Demetriades D, Berne TV, et al. Prehospital intubation in patients with severe head injury. *J Trauma.* 2000;49(6):1065-1070.
5. Davis DP, Peay J, Sise MJ, et al. The impact of prehospital endotracheal intubation on outcome in moderate to severe traumatic brain injury. *J Trauma.* 2005;58(5):933-939.
6. Poste JC, Davis DP, Ochs M, et al. Air medical transport of severely head-injured patients undergoing paramedic rapid sequence intubation. *Air Med J.* 2004;23(4):36-40.
7. Davis DP, Stern J, Sise MJ, et al. A follow-up analysis of factors associated with head-injury mortality after paramedic rapid sequence intubation. *J Trauma.* 2005;59(2):486-490.
8. Lowe L, Sagehorn K, Madsen R. The effect of a rapid sequence induction protocol on intubation success rate in an air medical program. *Air Med J.* 1998;17(3):101-104.
9. Germann CA, Baumann MR, Kendall KM, et al. Performance of endotracheal intubation and rescue techniques by emergency services personnel in an air medical service. *Prehosp Emerg Care.* 2009;13(1):44-49.
10. Rose WD, Anderson LD, Edmond SA. Analysis of intubations. Before and after establishment of a rapid sequence intubation protocol for air medical use. *Air Med J.* 1994;13(11-12):475-478.
11. Sing RF, Rotondo MF, Zonies DH, et al. Rapid sequence induction for intubation by an aeromedical transport team: a critical analysis. *Am J Emerg Med.* 1998;16(6):598-602.
12. Murphy-Macabobby M, Marshall WJ, Schneider C, et al. Neuromuscular blockade in aeromedical airway management. *Ann Emerg Med.* 1992;21(6):664-668.
13. Ma OJ, Atchley RB, Hatley T, et al. Intubation success rates improve for an air medical program after implementing the use of neuromuscular blocking agents. *Am J Emerg Med.* 1998;16(2):125-127.
14. Walls RM, Murphy MF. Identification of the difficult and failed airway. In: Walls RM and Murphy MF (eds): *Manual of Emergency Airway Management*, 3rd edition. Philadelphia, Lippincott Williams and Wilkins, 2008;81-93.
15. Reed MJ, Dunn MJG, McKeown DW, et al. Can an airway assessment score predict difficulty at intubation in the emergency department? *Emerg Med J.* 2005;22(2):99-102.
16. Thomas S, Judge T, Lowell MJ, et al. Airway management success and hypoxemia rates in air and ground critical care transport: a prospective multicenter study. *Prehosp Emerg Care.* 2010;14(3):283.
17. McIntosh SE, Swanson ER, Barton ED. Cricothyrotomy in air medical transport. *J Trauma.* 2008;64(6):1543-1547.
18. Robinson KJ, Katz R, Jacobs LM. A 12-year experience with prehospital cricothyrotomies. *Air Med J.* 2001;20(6):27-30.
19. Wang HE, Kupas DF, Paris PM, et al. Preliminary experience with a prospective, multi-centered evaluation of out-of-hospital endotracheal intubation. *Resuscitation.* 2003;58(1):49-58.
20. Wang HE, Kupas DF, Hostler D, et al. Procedural experience with out-of-hospital endotracheal intubation. *Crit Care Med.* 2005;33(8):1718-1721.
21. Burton JH, Baumann MR, Maoz T, et al. Endotracheal intubation in a rural EMS state: procedure utilization and impact of skills maintenance guidelines. *Prehosp Emerg Care.* 2003;7(3):352-356.
22. Warner KJ, Carlbom D, Cooke CR, et al. Paramedic Training for Proficient Prehospital Endotracheal Intubation. *Prehosp Emerg Care.* 2010;14(1):103-108.
23. Jones JH, Murphy MP, Dickson RL, et al. Emergency physician-verified out-of-hospital intubation: miss rates by paramedics. *Acad Emerg Med.* 2004;11(6):707-709.
24. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. *Ann Emerg Med.* 2001;37(1):32-37.
25. Jemmett ME, Kendal KM, Fourre MW, et al. Unrecognized misplacement of endotracheal tubes in a mixed urban to rural emergency medical services setting. *Acad Emerg Med.* 2003;10(9):961-965.
26. Colwell CB, McVaney KE, Haukoos JS, et al. An evaluation of out-of-hospital advanced airway management in an urban setting. *Acad Emerg Med.* 2005;12(5):417-422.

Ambulatory Cardiac Monitoring for Discharged Emergency Department Patients with Possible Cardiac Arrhythmias

Donald Schreiber, MDCM*
Ayesha Sattar, DO*
Dorian Drigalla, MD†
Steven Higgins, MD‡

* Division of Emergency Medicine, Stanford University School of Medicine, Stanford, California
† Department of Emergency Medicine, Texas A&M College of Medicine, Temple,Texas
‡ Department of Cardiology, Scripps Memorial Hospital, La Jolla, California

Supervising Section Editor: Amal Mattu, MD
Submission history: Submitted July 12, 2013; Revision received October 30.,2013; Accepted November 11, 2013
Electronically published February 28, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.11.18973

Introduction: Many emergency department (ED) patients have symptoms that may be attributed to arrhythmias, necessitating outpatient ambulatory cardiac monitoring. Consensus is lacking on the optimal duration of monitoring. We describe the use of a novel device applied at ED discharge that provides continuous prolonged cardiac monitoring.

Methods: We enrolled discharged adult ED patients with symptoms of possible cardiac arrhythmia. A novel, single use continuous recording patch (Zio®Patch) was applied at ED discharge. Patients wore the device for up to 14 days or until they had symptoms to trigger an event. They then returned the device by mail for interpretation. Significant arrhythmias are defined as: ventricular tachycardia (VT) ≥4 beats, supraventricular tachycardia (SVT) ≥4 beats, atrial fibrillation, ≥3 second pause, 2nd degree Mobitz II, 3rd degree AV Block, or symptomatic bradycardia.

Results: There were 174 patients were enrolled and all mailed back their devices. The average age was 52.2 (± 21.0) years, and 55% were female. The most common indications for device placement were palpitations 44.8%, syncope 24.1% and dizziness 6.3%. Eighty-three patients (47.7%) had ≥1 arrhythmias and 17 (9.8%) were symptomatic at the time of their arrhythmia. Median time to first arrhythmia was 1.0 days (IQR 0.2-2.8) and median time to first symptomatic arrhythmia was 1.5 days (IQR 0.4-6.7). 93 (53.4%) of symptomatic patients did not have any arrhythmia during their triggered events. The overall diagnostic yield was 63.2%

Conclusion: The Zio®Patch cardiac monitoring device can efficiently characterize symptomatic patients without significant arrhythmia and has a higher diagnostic yield for arrhythmias than traditional 24-48 hour Holter monitoring. It allows for longer term monitoring up to 14 days. [West J Emerg Med. 2014;15(2):194–198.]

INTRODUCTION

Symptoms attributed to possible cardiac arrhythmias, such as syncope, palpitations or dizziness, are common presenting complaints to the emergency department (ED) and may account for 3-4% of all ED visits. ED management is driven by risk assessment and current guidelines.[1-5] Admission rates, however, remain high and inpatient management is expensive.[6] Outpatient management for ED patients has been complicated by logistical barriers to outpatient ambulatory cardiac monitoring such as the need for cardiology consultation, availability of cardiac monitoring devices and patient compliance.

Importance

The gold standard for diagnosing an arrhythmia as the etiology for the patient's symptoms is electrocardiogram (ECG) documentation of a rhythm disturbance at the time of symptoms.[1-5] The optimal device for ambulatory cardiac

monitoring is based on the frequency of symptoms. Typically, a 24-48 hour Holter monitor is used.[1-3,6,7] However, consensus is lacking in the literature on the optimal duration of cardiac monitoring. The American College of Emergency Physicians' clinical policy on syncope suggests that Holter monitoring beyond 24 hours is unlikely to increase detection of significant arrhythmias.[3] Other authors report that 24-hour Holter monitoring is insufficient, and ambulatory cardiac monitoring for 1-6 weeks may be required.[7-10]

Goals of this Investigation

In this study, we describe the use of a novel portable ambulatory cardiac monitoring device applied upon discharge from the ED that provides continuous monitoring for up to 14 days. Our goal was to determine the diagnostic yield of such a device and to determine the value of prolonged monitoring of low-risk discharged ED patients with possible cardiac arrhythmia.

METHODS

Study Design and Setting

We completed an observational study at 3 academic EDs in the United States between February 2011 and February 2012.

Selection of Participants

We enrolled a convenience sample of discharged adult ED patients >18 years of age with symptoms suggestive of possible cardiac arrhythmia who were deemed candidates for outpatient ambulatory cardiac monitoring. All diagnostic testing, final disposition and use of the device were at the discretion of the attending emergency physician. The ambulatory cardiac rhythm monitor was a single use long-term, continuous recording patch (Zio®Patch - iRhythm Technologies, Inc. San Francisco, CA) that was applied by trained ED personnel at discharge. The device is a FDA-approved diagnostic adhesive patch that is affixed to the left anterior chest wall. It continuously records the cardiac rhythm for up to 14 days.

Methods and Measurement

Incorporated into the Zio®Patch is an event marker button that patients are instructed to press when symptomatic. Activation of the event marker button is termed a triggered event, and for purposes of this study it is assumed that the triggered event represented symptoms of possible cardiac arrhythmia. The patient was also provided a diary for written entries of symptoms and times. The cardiac rhythm for 45 seconds before and 45 seconds after a triggered event or diary entry was marked for review. Subjects were instructed to wear the device up to 14 days and then simply mail the device in the supplied pre-addressed postage-paid envelope to the company's facility. They were also instructed to return to the ED for any recurrent or worsening symptoms. The complete set of continuous rhythm data and the file of triggered events (if any) were downloaded from the device and analyzed using a proprietary algorithm. An initial report was prepared and then sent electronically for cardiology review and is then returned to the ordering physician. The cardiologist reviewed the entire record including all triggered events and the patient diary and generated a summary report.

Outcomes

We defined significant arrhythmias as ventricular tachycardia (VT) ≥4 beats, paroxysmal atrial fibrillation (PAF), supraventricular tachycardia (SVT) ≥4 beats, ≥3 sec pause, 2nd degree Mobitz II or 3rd degree AV block, or symptomatic bradycardia. Serious arrhythmias were defined as: VT >120 for 30 seconds, Complete or 3rd degree heart block, symptomatic second degree heart block, type II, pause >6 seconds and symptomatic bradycardia <40 beats per minute for >30 seconds. If any serious arrhythmias were detected on the initial report, the ED or cardiologist was immediately notified; the patient was then contacted and asked to return, all in accordance with existing policies and procedures for reporting critical results. We also defined analyzable time as the percentage of the entire patient ECG data record that had sufficient fidelity to enable a rhythm analysis. Diagnostic yield was defined as the percentage of all patients who had a triggered event without any arrhythmia found or who had a significant symptomatic arrhythmia detected.

Analysis

We described descriptive statistics using parametric and non-parametric techniques where appropriate. All device data were maintained on the company's encrypted secure database. We de-identified the data and provided the results for analysis without any protected health information. The study was reviewed and approved by each institution's institutional review board.

RESULTS

Characteristics of Study Subjects

We enrolled a total of 174 patients; the average age was 52.2 (± 21.0) years and 55% were female. Palpitations (78, 44.8%), syncope (42, 24.1%) and dizziness (11, 6.3%) were the most common indications for ambulatory device placement. Other indications included the detection of specific arrhythmias, such as ventricular tachycardia (14, 8.0%), atrial fibrillation (4,2.3%), bradyarrhythmias (5,2.9%) or unspecified arrhythmias (20, 11.5%) All 174 patients (100%) returned their cardiac monitor for review.

Study Results

Eighty-three patients (47.7%) had ≥1 significant arrhythmias (excluding chronic atrial fibrillation), and 17 (9.8%) were symptomatic at the time of their arrhythmia. Nine patients (5.2%) had ≥2 arrhythmias. Significant arrhythmias

Table 1. Summary of arrhythmias.

	n	All n=174	Arrhythmia only n=85
Ventricular tachycardia (≥4 but <8 beats):	13	7.5%	15.3%
Ventricular tachycardia (≥8 beats):	1	0.6%	1.2%
Pause (>3 seconds):	4	2.3%	4.7%
AV block (2nd degree Mobitz II or 3rd degree):	2	1.1%	2.4%
Supraventricular tachycardia (≥4 but <8 beats):	19	10.9%	22.4%
Supraventricular tachycardia (≥8 beats):	48	27.6%	56.5%
All atrial fibrillation:	11	6.3%	12.9%
Chronic atrial fibrillation:	4	2.3%	4.7%
Paroxysmal atrial fibrillation:	7	4.0%	8.2%
Torsades/Ventricular fibrillation:	0	0.0%	0.0%

Table 2. Median time to first arrhythmia in days.

	Days
Atrial fibrillation*	0.4
Ventricular tachycardia	3.1
Supraventricular tachycardia	0.8
Pause	4.2
2nd or 3rd degree AV block	5.8

*Excludes chronic atrial fibrillation

are further detailed in Table 1. There were no significant gender differences identified. Median time to first arrhythmia was 1.0 days (interquartile range [IQR] 0.2-2.8) and median time to first symptomatic arrhythmia was 1.5 days (Mean 3.9, IQR 0.4-6.7). The mean times to first symptomatic event for each arrhythmia are outlined in Table 2. Seven (4.0%) patients required immediate physician notification for serious arrhythmias. There were no patient deaths.

In total, 93(53.4%) symptomatic patients did not have any arrhythmia during their triggered events. We calculated the diagnostic yield of 63.2% is calculated to be the number of triggered events without arrhythmias (n=93) and the number of significant symptomatic arrhythmias detected (n=17). Median device wear time was 6.9 days (IQR 5.8 -9.2), and the analyzable time was 98.6% of the total recorded data.

DISCUSSION

In this study we describe a novel ambulatory cardiac monitoring device that can easily be applied to patients upon discharge from the ED. It was well tolerated for prolonged monitoring and compliance was excellent. Single channel ECG data quality was also excellent with more than 98% of the total recording time analyzable.

The device had high diagnostic yield for low-risk patients discharged from the ED with primary complaints of palpitations, syncope or dizziness. We observed that the 24-48 duration of traditional Holter monitoring may be inadequate for identifying significant arrhythmias in these patients. For example, the median time to the first triggered arrhythmia in this study for ventricular tachycardia and sinus pauses was 3.1 and 4.2 days respectively, outside the window of traditional Holter monitoring.

The current primary criterion for establishing a cardiac arrhythmia as the cause of syncope, near syncope, palpitations or dizziness rests on the correlation of the arrhythmia with symptoms.[1-3,6,7] The clinical challenge in the ED is to identify and admit the high-risk patients and refer the low-risk patients for outpatient evaluation if appropriate.[1,6,7] The role of ambulatory cardiac monitoring for ED patients is guided by clinical suspicion for an arrhythmia, the anticipated frequency of recurrent symptoms and whether the patient is low risk and safe for discharge. In practical terms, the logistics of organizing adequate follow up within 14 days of the index ED visit is often problematic for a variety of administrative, financial and system-based reasons. This study demonstrated the ease and utility of initiating ambulatory cardiac monitoring with the Zio®Patch at the time of the index ED visit.

Several devices are currently available to assess cardiac rhythm disturbances in ambulatory patients. The traditional Holter monitor was first introduced to clinical practice in the 1940s.[7] The first device was a 75-pound backpack with a reel-reel tape recorder and large batteries. Current technology incorporates flash memory, weighs less than 200 grams and stores 24-48 hours of continuous ECG data but still requires electrodes and wires for patient use.[6,7] The major advantages of traditional Holter monitors are the ability to continuously record ECG data and the fact that transmission of data is not patient dependent. However, non-compliance with use of the device or maintaining a log of symptoms limits diagnostic utility.[1,7]

Event recorders do not record continuous ECG data but require patient activation at the time of symptom onset. These devices may be applied to the chest wall at the time of the

event and must be activated by the patient. A brief, typically 90-second, single lead ECG recording is captured and stored on the device. Because of limited data storage capability, these data must be transmitted to a central monitoring center for validation and analysis. Although these devices can be used for cardiac monitoring over longer periods of time, they are limited by the fact that patients must be able to activate the device following symptom onset.[7] This may be difficult to achieve, for example, if the patient had syncope or suffered an injury related to the event. Finally, these devices cannot be used to document asymptomatic arrhythmias.[2,7]

External continuous loop recorders (ELR) are attached to the patient by chest electrodes or a wristband. They continuously record the ECG recording but only save the data if activated by the patient. The continuous looping memory feature allows the device to store a fixed length of pre-activation and post-event ECG data.[7]

Mobile cardiac telemetry systems (MCOT) provide up to 30 days of real-time continuous cardiac monitoring without the need for patient activation or data transmission. The relative disadvantages of these devices are the higher costs, the need for electrodes and bulky recording devices, as well as the potential burden on the clinician who must be available to review the large amount of data.[1,2,6] Implantable loop recorders (ILRs) are surgically implanted subcutaneous devices that continuously record single-lead ECG signal through 2 electrodes. However they are very expensive and necessitate an invasive procedure.[1]

Traditionally, ambulatory cardiac monitoring is usually initiated with a 24-48 hour Holter monitor. Bass reported a diagnostic yield of 15% with 24-hour Holter monitoring that did not increase even if the device was applied for 72 hours.[8] In a prospective study evaluating ELR in syncope, symptom-rhythm correlation was found in 56% with an ELR worn for 1 month versus only 22% for 48 hour Holter monitoring.[7,9] In, studies on selected patients with palpitations comparing ELR and 48-hour Holter monitors, the diagnostic yield with Holter monitors was 35%-39%.[10] In our study, the overall diagnostic yield was 63.2%, which is considerably higher than traditional 48-hour Holter monitoring and points to the value of up to 14-day ambulatory cardiac monitoring.

The absence of an arrhythmia during syncope, palpitations or a triggered event does not by itself provide a definitive diagnosis but does allow the clinician to exclude an arrhythmia as a potential cause and is thus clinically useful. Over half our patients (53.4%) did not have an arrhythmia despite a triggered event. This allows the clinician to potentially exclude an arrhythmia as an etiology of the patient's symptoms and potentially avoid further cardiac evaluation.

LIMITATIONS

As an observational convenience study, enrollment was based on clinician discretion alone. Concomitant Holter monitoring could not be provided. There was no randomized comparison to Holter monitor or other traditional management approaches. Demographic data were limited to age and gender, and we did not analyze or compare other clinical characteristics. No long-term follow-up data was obtained. Attributing all triggered events to symptoms may be incorrect without formal patient review. Certain events such as syncope may have occurred during the monitoring period without arrhythmia but the patient may not have been able to activate the event recorder button. While this may have lowered the diagnostic yield, any important arrhythmia would have been detected during the continuous monitoring.

CONCLUSION

This study demonstrated the utility of the Zio®Patch, a novel ambulatory cardiac monitoring device that is applied at discharge for up to 14 days following an ED visit for syncope, palpitations or dizziness. This approach provided relatively prompt diagnoses at both ends of the clinical spectrum, including the documentation of normal sinus rhythm in patients with symptoms, as well as serious asymptomatic arrhythmias in others. Further outcome and economic studies are required to determine if this device will reduce hospital admission rates and improve the diagnostic efficiency for these patients.

Address for Correspondence: Donald Schreiber, MDCM, FRCPC, FACEP, Division of Emergency Medicine, Stanford University School of Medicine, 300 Pasteur Dr., Stanford, CA 94304. Email: donalds@stanford.edu.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. Technical support from iRhyhm Technologies, Inc., San Francisco, CA. iRhyhm Technologies, Inc had no role in the design of the study, the statistical analysis of the data, interpretation of the results or the preparation or review of the manuscript.

REFERENCES

1. Sheldon RS, Morillo CA, Krahn AD, et al. Standardized Approaches to the Investigation of Syncope: Canadian Cardiovascular Society Position Paper. *Canadian Journal of Cardiology.* 2011;27:246–253.
2. Moya A, Sutton R, Ammirati F, et al. Guidelines for the diagnosis and management of syncope (version 2009) *Eur Heart J*. 2009;30:2631–2671.
3. Huff SJ, Decker WW, Quinn JV, et al. Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department with Syncope. *Ann Emerg Med*. 2007;49:431-444.
4. Zimetbaum P, Josephson ME. Evaluation of Patients with

Palpitations. *New Engl J Med*. 1998;338(19):1369-1373.

5. Thavendiranathan P, Bagai A, Khoo C, et al. Does This Patient with Palpitations Have a Cardiac Arrhythmia? *JAMA*. 2009;302(19):2135-2143.
6. Brignole M, Hamdan MH. New Concepts in the Assessment of Syncope. *J Am Coll Cardiol*. 2012;59:1583-1591.
7. Zimetbaum P, Goldman A. Ambulatory Arrhythmia Monitoring: Choosing the right device. *Circulation*. 2010;122:1629-1636.
8. Bass EB, Curtiss EI, Arena VC, et al. The duration of Holter monitoring in patients with syncope. Is 24 hours enough? *Arch Intern Med.* 1990;150:1073-1078.
9. Kinlay S, Leitch JW, Neil A, et al. Cardiac event recorders yield more diagnoses and are more cost-effective than 48-hour Holter monitoring in patients with palpitations. A controlled clinical trial. *Ann Intern Med.* 1996;124:16-20.
10. Gula LJ, Krahn AD, Massel D, et al. External loop recorders: determinants of diagnostic yield in patients with syncope. *Am Heart J*. 2004;147:644-648.

Improving Bariatric Patient Transport and Care with Simulation

Brad D. Gable, MD, MS*†
Aimee K. Gardner, PhD‡
Dan H. Celik, MD*
Mary Colleen Bhalla, MD*§
Rami A. Ahmed, DO*§

* Summa Akron City Hospital, Department of Emergency Medicine, Akron, Ohio
† Ohio University Heritage College of Osteopathic Medicine, Athens, Ohio
‡ University of Texas Southwestern Medical Center, Dallas, Texas
§ Northeast Ohio Medical University, Department of Emergency Medicine, Rootstown, Ohio

Supervising Section Editor: Douglas Ander, MD
Submission history: Submitted July 2, 2013; Revision received November 15, 2013; Accepted December 3, 2013
Electronically published February 28, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.18855

Introduction: Obesity is prevalent in the United States. Obese patients have physiologic differences from non-obese individuals. Not only does transport and maintenance of these patients require use of specialized equipment, but it also requires a distinct skill set and knowledge base. To date, there is no literature investigating simulation as a model for educating pre-hospital providers in the care of bariatric patients. The purpose of this study was to determine if a 3-hour educational course with simulation could improve paramedics' knowledge and confidence of bariatric procedures and transport. This study also examined if prior experience with bariatric transport affected training outcomes.

Methods: Our study took place in August 2012 during paramedic training sessions. Paramedics completed a pre- and post-test that assessed confidence and knowledge and provided information on previous experience. They had a 30-minute didactic and participated in 2 20-minute hands-on skills portions that reviewed procedural issues in bariatric patients, including airway procedures, peripheral venous and intraosseous access, and cardiopulmonary resuscitation. Study participants took part in one of two simulated patient encounters. Paramedics were challenged with treating emergent traumatic and/or medical conditions, as well as extricating and transporting bariatric patients. Each group underwent a debriefing of the scenario immediately following their case. We measured confidence using a 5-point Likert-type response scale ranging from 1 (strongly disagree) to 5 (strongly agree) on a 7-item questionnaire. We assessed knowledge with 12 multiple choice questions. Paired-sample *t*-tests were used to compare pre- and post-simulation confidence and knowledge with a significance level of $p \leq 0.05$. We used analysis of covariance to examine the effect of previous experiences on pre-and post-educational activity confidence and knowledge with a significance level of $p \leq 0.05$. Proportions and 95% confidence intervals are presented as appropriate. We determined the magnitude of significant pre-post differences with Cohen's *d.* We assessed scale reliability using Cronbach's alpha and was found to be reliable with scores of 0.83 and 0.88 across pre- and post-test responses, respectively.

Results: Participants exhibited a significant increase in confidence in performing procedures ($p<0.01$) and knowledge of bariatric patient management ($p<0.001$) after the simulation. The current study also found an increase in knowledge of transport, vascular access/circulation and airway management ($p<0.001$). Participant background showed no effects on these changes.

Conclusion: This study suggests that simulation paired with a didactic is an effective method of education for paramedics caring for and transporting bariatric patients. The data show a significant increase in knowledge and confidence with a 3-hour training session, irrespective of previous training or experience with bariatric patients. This is the first study of its kind to apply simulation training for the pre-hospital care of bariatric patients. [West J Emerg Med. 2014;15(2):199–204.]

INTRODUCTION

Obesity is prevalent in the United States (U.S.). According to the National Center for Health Statistics, more than a third of adults in the U.S. are obese.[1] Obesity is associated with several chronic medical conditions, including hypertension, diabetes,[2] obstructive sleep apnea (OSA),[3,4] and obesity hypoventilation syndrome.[5,6] Obese patients have a greater likelihood of requiring medical intervention than those who are not overweight.[7] Due to the increased probability of needing medical support, pre-hospital providers need to be current and competent regarding bariatric treatment and transport needs.

Obese patients have physiologic differences from non-obese individuals. These include increased chest wall resistance, increased abdominal pressure, decreased lung capacity, increased airway resistance, increased subcutaneous tissue, and anatomical distortion.[8-10] As a result, assessment and treatment of these patients requires specialized knowledge and skill. In addition to the medical challenges posed by bariatric patients, there are also issues regarding transportation and handling of these patients.[11-13] Not only does transport and maintenance of these patients require use of specialized equipment, but it also requires a distinct skill set and knowledge base regarding specific maneuvers. Thus, adequate treatment of bariatric patients requires specific training regarding these issues.

To address the distinct needs of bariatric patients, pre-hospital providers must be trained to care for overweight and obese patients. Traditional training often involves didactic presentations and lectures. However, simulation may be an ideal adjunct to the traditional bariatric training approach. Simulation can provide a dynamic hands-on educational model for instructing first responders on the unique challenges of caring for bariatric patients. Simulation has demonstrated a 4 times greater retention of information versus traditional lecture-based education.[14] Simulation is also an ideal means to incorporate the learning theory of "deliberate practice," which is essential for the development of clinical expertise.[15] Thus, simulation may be a well-suited alternative or adjunct to traditional training approaches for this special population.

To date, there is no literature investigating simulation as a model for educating pre-hospital providers in the care of bariatric patients. Examining the use of new instructional methodologies within bariatric transport can help identify the efficacy of these approaches and expand understanding of how to best train pre-hospital providers. This study sought to determine if a 3-hour training session could improve paramedics' knowledge of bariatric transport and confidence to perform bariatric procedures. Additionally, we examined how participant experiences (number of bariatric patients transported in past year and previous training) impacted the efficacy of this training. The current study assessed paramedics with a pre- and post-training survey.

METHODS

Setting

This study was performed at a fire department in a rural Midwestern township. This publicly operated, combined fire and emergency medical services (EMS) department serves a suburban population of 40,373.[16] Sixty-nine full-time and 35 part-time paramedics respond to approximately 4,000 calls per year.

Study Participants

All fire department paramedics who were present on one of 3 scheduled continuing medical education sessions participated. We excluded paramedics who were not available for the entire training session due to clinical duty. Dates of the sessions were August 7th, 8th, and 9th, 2012.

Table 1. Design of 1-day 3-hour training session for paramedics transporting bariatric patients.

<table>
<tr><th></th><th>13:00-13:05</th><th>13:05-13:40</th><th>13:40-14:05</th><th>14:05-14:30</th><th>14:30-14:40</th><th>14:40-15:05</th><th>15:05-15:30</th><th>15:30-15:35</th><th>15:30-16:00</th></tr>
<tr><td>Group 1</td><td rowspan="4">Pre-test</td><td rowspan="4">Introduction and background</td><td colspan="2" rowspan="2">Scenario 1</td><td rowspan="4">Break and reset misc. procedures, medical case</td><td>Airway procedures</td><td>Misc. procedures, medical case</td><td rowspan="4">Post-test</td><td rowspan="4">Debriefing and summary</td></tr>
<tr><td>Group 2</td><td>Airway procedures</td><td></td></tr>
<tr><td>Group 3</td><td>Airway procedures</td><td>Misc. procedures, medical case</td><td colspan="2" rowspan="2">Scenario 2</td></tr>
<tr><td>Group 4</td><td>Misc. procedures, medical case</td><td>Airway procedures</td></tr>
</table>

Study Design

Study participants were given identical pre- and post-training surveys that focused on confidence in bariatric care, as well as knowledge of bariatric clinical issues. (See Measures below and Appendix) All surveys were de-identified but included a unique number to link pre- and post-questionnaires. Bariatric training consisted of a didactic portion, a skills portion, and a simulated patient encounter. (See Table 1 for curriculum outline). An EMS physician and a practicing paramedic who were not part of the research team reviewed the survey to ensure proper language appropriate for the study participants, to ensure there was no bias and to ensure the questions matched the demographics and experience of the study participants.

The didactic portion included a 30-minute lecture that defined obesity, discussed associated health risks, presented unique EMS care issues, reviewed transfer and mobility concerns, addressed scene and personal safety, and promoted sensitivity and professionalism with regards to bariatric patients.

The 2 20-minute hands-on skills portions reviewed procedural issues in bariatric patients, including airway procedures (Bag-valve-mask, intubation, laryngeal mask airways), peripheral venous and intraosseous access (Vidacare® EZ-IO; Shavano Park, TX), and cardiopulmonary resuscitation (Nasco Life/form® Fat Old Fred Manikin; Fort Atkinson, WI). These task trainers were chosen to demonstrate these skills, as they have the anatomic and physiologic changes present in bariatric patients. In addition, homemade task trainers were used to further practice these skills. Such task trainers included bariatric arms made of ballistics gel and fluid tubing for peripheral venous access, and ballistics gel added to IO mannequins to simulate the greater amount of subcutaneous tissue present in bariatric patients. During the peripheral access/intraosseous access and CPR station, learners were given a case of a bariatric patient needing resuscitation and were to perform venous access and cardiopulmonary resuscitation on the above task trainers.

Study participants took part in one of two simulated patient encounters. The encounters used a water-filled bariatric suit (Simulaids, Inc. Bariatric Rescue Suit; Saugerties, NY), which was placed over a mannequin (Rescue Randy; Saugerties NY) (Figure) and a tarp-style transportation device (Graham Megamover® 1500; Green Bay, WI). The mannequin with bariatric suit, when filled with water, weighed approximately 350 pounds. The first case involved an elderly, morbidly obese male taking dabigatran who fell down a flight of stairs and was unable to get up. The second case described a middle-aged male who used prescription narcotic pills and fell from the toilet and became hypoxic. Although paramedics were challenged with treating emergent traumatic and/or medical conditions, the focus of the simulated patient encounters was on extricating and transporting the bariatric patients. Each group underwent a debriefing of the scenario immediately following their case.

Figure. Mannequin in a water-filled bariatric suit on top of a tarp-style transportation device.

Following the completion of skills stations and the simulation scenario all paramedics were given a post-training survey. After all surveys were collected, the participants discussed the particular case they were presented with during their simulation scenario. The aim of this large-group discussion was to facilitate open dialogue of the difficulties presented during the scenarios and the resultant strategies used to overcome the obstacles. Lastly, the paramedics were given a brief didactic presentation to summarize the medical and transport issues presented by bariatric patients.

Our Institutional Review Board determined that the study was exempt from review due to our de-identifying the data set.

Measures

As part of the pre-survey we collected information on previous training and experience ,including number of years of experience, previous bariatric transport participation and the number of bariatric patients transported in the previous year. This information sought to examine if training effects differ among experience levels. For all measures, participants used a Likert-type response scale ranging from 1 (strongly disagree) to 5 (strongly agree).

The current study also assessed confidence in bariatric transport as part of the pre- and post-surveys with a 7-item questionnaire. Example items included "I feel confident that I can properly assess a bariatric patient" and "Can identify local resources to help in emergency management of bariatric patients" (Appendix). For each item subjects used the Likert-type response previously described. We assessed study participants' knowledge on the treatment and transportation of bariatric patients with 12 multiple-choice items. We developed the multiple choice questions based on the learning objectives for the educational session. These questions evaluated knowledge of bariatric airway management, vascular access/circulation, and transport (Appendix).

Table 2. Comparison of bariatric confidence and knowledge scores before and after simulation.

Variable	Pre-simulation mean (SD)	Post-simulation mean (SD)	Difference	Cohen's d	95% CI lower	95% CI upper	*p*-value
Confidence	3.67 (0.45)	4.34 (0.40)	0.67	0.55	-1.01	-0.23	<0.01
Bariatric knowledge	6.03 (1.63)	10.00 (0.93)	3.97	1.46	-1.15	-0.84	<0.001
Transport	1.94 (1.01)	3.31 (0.75)	1.37	1.35	-1.77	-1.06	<0.001
Access/Circulation	1.67 (0.79)	2.39 (0.69)	0.72	0.71	-0.94	-0.34	<0.001
Airway	1.89 (0.92)	3.58 (0.55)	1.69	1.51	-2.11	-1.34	<0.001
Other	0.53 (0.51)	0.72 (0.45)	0.19	0.34	-0.39	0	=0.05

Statistical Analysis

We performed all statistical analysis with SPSS (version 18.0; Chicago, IL). Paired-sample *t*-tests were used to compare pre- and post-simulation confidence and knowledge with a significance level of $p \leq 0.05$. We used analysis of covariance (ANCOVA) to examination the effect of previous experiences on pre-and post-educational activity confidence and knowledge with a significance level of $p \leq 0.05$. Proportions and 95% confidence intervals are presented as appropriate. Scale reliability was assessed using Cronbach's alpha. Additionally, we determined magnitude of significant pre-post differences with Cohen's *d*.

RESULTS

Pre- and post-training responses from 36 paramedics were available for evaluation. We excluded responses from 5 paramedics who did not complete either a pre-test or a post-test due to being called away from the training session to respond to an emergency call. Of the final sample, 22% (8/36) had participated in bariatric transport training previously (none of which included simulation) and 42% (15/36) had transported at least 5 bariatric patients in the past year. Paramedic experience ranged from 1 to 35 years, with an average of 19.5 years.

Participants exhibited a significant increase in confidence in performing procedures ($p<0.01$) assessed by comparing the overall mean confidence for the 7 statements on the pre-test to the post-test (Table 2). Participants also significantly increased their knowledge of bariatric patient management ($p<0.001$) assessed by comparing the mean number of answers correct on the pre-test to the post-test (Table 2). The data also show an increase in knowledge of transport, vascular access/circulation and airway management ($p<0.001$) (Table 2).

Finally, we examined the magnitude of these effects using Cohen's *d*. Conventionally, the size of an effect is determined to be small if greater than 0.20, medium if greater than 0.50, and large if greater than 0.80 (Cohen, 1992). As indicated in Table 2, changes in bariatric confidence and knowledge exhibited large effects. Participant background (number of bariatric patients transported in past year and previous training) showed no effects on these changes ($p=0.43$; $p=0.65$, respectively).

The bariatric transport knowledge scale exhibited good reliability, with a Cronbach's alpha at 0.83 and 0.88 across pre- and post-simulation responses, respectively.

DISCUSSION

The effectiveness of simulation-based training has been confirmed in a number of healthcare realms.[17-26] However, researchers have yet to investigate the efficacy of this approach within bariatric transport. One of the strengths of this study is its novelty, as this is the first study to examine the use of simulation in the training of pre-hospital personnel for treating bariatric patients.

These results reveal that a 30-minute didactic lecture combined with simulation training can have a valuable impact on paramedic bariatric transport and medical knowledge. Specifically, paramedics exhibited increases in understanding of the unique airway, vascular access, and transport needs of bariatric patients. Despite 22% of the trainees having had previous formal training in bariatric transport and over 40% having transported at least 5 patients in the last year, the improvement in knowledge and confidence among both previously trained/experienced providers and novices was similar. Although a large portion of paramedics had previously been educated on bariatric transport and treatment, having an opportunity to learn in a hands-on format may have allowed participants to gain a deeper understanding of bariatric patient issues. Simulation is a unique educational technique. Simulation in combination with traditional teaching methods may prove with future randomized studies to be superior for learning certain skills than traditional teaching alone.

Study participants also reported an increase in confidence to perform procedures on bariatric patients after the training. This enhanced confidence can manifest itself in a number of ways for paramedics. Paramedics who are more confident caring for bariatric patients may also be more likely to exhibit guidance to other paramedics not similarly trained, speak up to team members performing tasks incorrectly, and execute procedures with controlled emotions. This outcome, combined with the observed knowledge improvement, is especially noteworthy. These findings fall in line with previous simulation-based studies that indicate leadership

and teamwork training provided in simulated environments demonstrates both increased confidence to perform new procedures and decreased apprehension to try new techniques clinically.[27-30,] This is particularly important as many bariatric patients will require a multidisciplinary team effort to safely manage and transport them to definitive care.

The current study demonstrates favorable trainee outcomes after participating in a 30-minute lecture, a 50-minute simulation, a 25-minute case-based scenario, and a 25-minute task training skill station. Although the didactic portion of this training may have contributed to some of the knowledge acquisition, we believe that it was not the sole contributing factor. The initial didactic discussion was included to provide an overview of bariatric patient concerns, provide understanding of why the training was necessary, and to assure participant safety during the scenarios. Further, as trainees had varying degrees of experience previously caring for bariatric patients, the lecture ensured that all trainees would enter the simulation sessions with an adequate baseline knowledge level to participate in the simulation scenarios. For example, in the initial lecture, participants were told that they may not be able to use traditional cervical spine and long board immobilization and that alternative means may be necessary. During the simulation, trainees had to troubleshoot immobilization techniques to identify effective and ineffective strategies. This understanding, as well as its long-term retention, is unlikely to be demonstrated without interactive hands-on training provided in simulated environments.[14] According to Kirkpatrick[26] training should be evaluated at 4 levels: reactions, learning, behavior, and results. Whereas many simulation studies investigate solely trainee attitudes, our investigation went an additional step and examined if trainees learned the knowledge relevant to managing bariatric patients.

During the large-group discussion we sought the feedback of the learners. The most common critique was that the scenarios they encountered could have been more challenging. The extrication and transportation took place in a firehouse, which is a local storm and disaster shelter and contains widened staircases and handicap -accessible bathrooms. Future training sites could include more difficult extrication sites or modification of existing sites to simulate the inside of a home or apartment building.

Obese patients present unique challenges to healthcare providers. As this population continues to grow, the healthcare system can expect to encounter such patients with increasing frequency and must prepare accordingly. Knowing how to deliver appropriate care in a safe and effective manner, while maintaining the patient's dignity, is of the utmost importance.

LIMITATIONS

This small pilot study evaluated learners immediately before and after a simulation-based curriculum that incorporates a didactic session, skill stations, and simulated scenarios. Further investigation into the long-term retention of the knowledge learned from this education session is needed. Additionally, the results reflect the educational session as a whole, and further studies would be needed to determine the effect that simulation or the didactic session has on knowledge acquisition. Future studies should include an assessment of skills as well.

Additionally, the data were obtained from a single EMS service and may not be generalizable to all levels of pre-hospital providers. Future studies should examine if such learning is successfully transferred to the work environment and how such knowledge impacts patient outcomes.

CONCLUSION

The current study suggests that a simulation-based curriculum that incorporates a didactic session, skill stations, and simulated scenarios is an effective method of education for paramedics caring for and transporting bariatric patients. This study shows a significant increase in knowledge and confidence with a 3-hour training session, irrespective of previous training or experience with bariatric patients. This is the first study of its kind to apply simulation training for the pre-hospital care of bariatric patients.

Address for Correspondence: Mary Colleen Bhalla, Summa Akron City Hospital, Department of Emergency Medicine, 525 East Market Street, PO Box 2090, Akron, OH 44304.
Email: bhallam@summahealth.org.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. The authors alone are responsible for the content and writing of the paper. No funding or financial support was obtained for this research.

REFERENCES

1. Ogden CL, Carroll MD, Kit BK, et al. Prevalence of obesity in the United States, 2009–2010. *NCHS Data Brief*. 2012;(82):1-8.
2. National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—The evidence report. *Obes Res*. 1998;6(Suppl 2):51S–209S.
3. Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. 1993;328:1230–1235.
4. Duran J, Esnaola S, Rubio R, et al. Obstructive sleep apnea-hypopnea and related clinical features in a population-based sample of subjects aged 30 to 70 yr. *Am J Respir Crit Care Med*. 2001;163:685–689.
5. Leech JA, Onal E, Baer P, et al. Determinants of hypercapnia in occlusive sleep apnea syndrome. *Chest*. 1987;92:807–813.
6. Mokhlesi B, Tulaimat A, Faibussowitsch I, et al. Obesity hypoventilation syndrome: prevalence and predictors in patients with obstructive sleep apnea. *Sleep Breath*. 2007;11:117-124.

7. Cui R, Iso H, Toyoshima E, et al. Body mass index and mortality from cardiovascular disease among Japanese men and women: the JACC study. *Stroke*. 2005;36(7):1377-1382.
8. Rabec C, de Lucas RP, Veale D. Respiratory complications of obesity. *Arch Bronconeumol*. 2011;47:252–261.
9. BaHammam A. Acute ventilatory failure complicating obesity hypoventilation: update on a critical care syndrome. *Curr Opin Pulm Med*. 2010;16:543–551.
10. Sabbane M, Claret P, Lefebvre S, et al. Predicting peripheral venous access difficulty in the emergency department using body mass index and a clinical evaluation of venous accessibility. *J Emerg Med*. 2013;44(2):299-305.
11. Hignett S, Griffiths P. Manual handling risks in the bariatric (obese) patient pathway in acute sector, community and ambulance care and treatment. *Work*. 2009;33:175-180.
12. Grant P, Newcombe M. Emergency management of the morbidly obese. *Emerg Med Aus.* 2004;16:309-317.
13. Hignett S, Griffiths P. Risk Factors for moving and handling bariatric paitents. *Nursing Standard*. 2009;vol 24, #11:40-48.
14. Dale E. *Audio-Visual Methods in Teach*ing.3rd Ed. New York: Holt, Rinehart, & Winston; 1969:108
15. Ericsson K. Deliberate practice and the acquisition and maintenance of expert performance in medicine and related domains. *Acad Med*. 2004;79(10 Suppl):S70-81.
16. Unknown. (2010) *OH - Jackson Township*. [online] Available at: http://www.census.gov/popfinder/?fl=39:3915138094 [Accessed: November 16, 2012].
17. Seymour N, Gallagher A, Roman SA, et al. Virtual reality training improves operating room performance: results of a randomized, double-blinded study. *Ann Surg.* 2002;236: 458-464.
18. Sroka G, Feldman L, Vassiliou MC, et al. Fundamentals of laparoscopic surgery simulator training to proficiency improves laparoscopic performance in the operating room- a randomized control trial. *Am J Surg.* 2010;199:115-120.
19. Britt R, Novosel T, Britt LD, et al. The impact of central line simulation before the ICU experience. *Am J Surg*. 2009;197:533-536.
20. Barsuk J, McGaghie W, Cohen ER, et al. Use of Simulation-based mastery learning to improve the quality of central venous catheter placement in a medical intensive care unit. *J Hosp Med.* 2009;4:397-403.
21. Barsuk J, McGaghie W, Cohen ER, et al. Simulation-based mastery learning reduces complications during central venous catheter insertion in a medical intensive care unit. *Crit Care Med.* 2009;37:2697-2701.
22. Holcomb JB, Dumire RD, Crommett JW et al. Evaluation of trauma team performance using and advanced human patient simulator for resuscitation training. *J Trauma.* 2002;52:1078-1086.
23. Wayne DB, Didwania A, Feinglass J, et al. Simulation-based education improves the quality of care during cardiac arrest team responses at an academic teaching hospital: a case-control study. *Chest.* 2008;133:56-61.
24. Chopra V. Gesink B, de Jong J, et al. Does training on the anesthesia simulator lead to improvement in performance? *Br J Anaesth.* 1994;73:293-297.
25. Wayne D, Siddall V, Butter J, et al. A longitudinal study of internal medicine residents' retention of advanced cardiac life support skills. *Acad Med.* 2006;81:S9-S12.
26. Kirkpatrick DL. Evaluation of training. In: Craig RL., ed. *Training and Development Handbook: A Guide to Human Resource Development.* 2nd ed. New York: McGraw-Hill; 1976:1-26.
27. Capella J, Smith S. Teamwork training improves the clinical care of trauma patients. *J Surg Educ*. 2010;67(6):439-443.
28. Ten Eyck R, Tews M. Improved fourth-year medical student clinical decision-making performance as a resuscitation team leader after a simulation-based curriculum. *Simul Healthc*. 2010;5(3):139-145.
29. Evans L, Dodge K. Simulation training in central venous catheter insertion: improved performance in clinical practice. *Acad Med.* 2010;85(9):1462-1469.
30. Barsuk J, McGaghie W. Use of simulation-based mastery learning to improve the quality of central venous catheter placement in a medical intensive care unit. *J Hosp Med*. 2009;4(7):397-403.

Staff Perceptions of an On-site Clinical Pharmacist Program in an Academic Emergency Department after One Year

Zlatan Coralic, PharmD, BCPS*†
Hemal K. Kanzaria, MD, MS‡
Lisa Bero, PhD†
John Stein, MD, MAS*

* University of California San Francisco, Department of Emergency Medicine, San Francisco, California
† University of California San Francisco, Department of Clinical Pharmacy, San Francisco, California
‡ University of California Los Angeles, Department of Emergency Medicine, Los Angeles, California

Supervising Section Editor: David Slattery, MD
Submission history: Submitted April 29, 2013; Revision received November 18, 2013; Accepted November 22, 2013
Electronically published February 26, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.11.18069

Introduction: Emergency department clinical pharmacists (EPh) serve a relatively new clinical role in emergency medicine. New EPh may still face barriers prior to working in the emergency department (ED), including staff acceptance. We aimed to assess staff perceptions of a university hospital EPh program 1 year after implementation.

Methods: We sent an electronic survey consisting of 7 multiple-choice questions, 17 5-point Likert-scale questions, and 1 free-text comment section to ED providers and nurses. The qualitatively validated survey assessed staff's general perceptions of the EPh and their clinical work.

Results: We received responses from 14 attending physicians, 34 emergency medicine residents, 5 mid-level providers, and 51 nurses (80% response rate). Overall, the ED staff strongly supported the presence of an EPh. All of the respondents consulted the EPh at least once in their previous 5 ED shifts. Most respondents (81%) felt the EPh's availability for general consultation and aid during resuscitations served as the major contribution to medication and patient safety. The participants also expressed that they were more likely to consult a pharmacist when they were located in the ED, as opposed to having to call the main pharmacy.

Conclusion: The EPh model of practice at our institution provides valuable perceived benefit to ED providers. [West J Emerg Med. 2014;15(2):205–210.]

INTRODUCTION

Background

Emergency department clinical pharmacists (EPh) have had a presence in emergency medicine since the 1970s.[1] However, it is only within the last decade that evidence has begun to emerge supporting the clinical benefits of an EPh's bedside practice in the emergency department (ED).[2] The EPhs are doctors of pharmacy and are usually residency trained or have training in critical or ambulatory pharmacotherapy[3]. While the duties of an EPh vary institutionally, most EPhs provide multiple services including: 1) face-to-face consultations with emergency physicians, residents, nurses and patients, 2) active assistance with medication management in resuscitations, and 3) provision of distributive services (i.e., order processing and drug supply management). New EPhs may face barriers to practicing within the emergency medicine (EM) clinical team, including institutional financial support, clinical training, and staff's acceptance and unfamiliarity with clinical pharmacists.

Importance

The EPh serves a relatively new clinical role within EM

practice, one that is just starting to be recognized and accepted by the EM community and the national pharmacist societies.[4] The published literature to date supporting EPh practice has been positive showing cost avoidance[5,6] and reduction in adverse drug events.[7-9] However, EDs may be concerned if the addition of pharmacist in the ED may lead to staff's dissatisfaction and delays in patient care.[10]

Goals of this investigation

The primary goal of this investigation was to assess staff perceptions of an EPh program at a university hospital ED 1 year after implementation of the program.

METHODS

Setting and Description of the EPh Program

In October 2009, 2 full-time clinical pharmacists established an EPh program in our institution's ED with the intent of providing clinical and distributive pharmacy services. Prior to the EPh program, the level of service provided by central pharmacy to the ED included stocking of automated medication dispensing machines and remote order verification. One of the EPhs was a recent general pharmacy residency graduate, while the other had worked as an intensive care unit clinical pharmacist for 3 years. Neither pharmacist had previous experience in emergency medicine or had relevant relationships with the ED staff. Both of the EPhs were certified in basic life support, advanced cardiovascular life support, and pediatric advanced life support. The University of California San Francisco Medical Center Department of Pharmacy funded the EPh program . The annual funds necessary to support a benefited pharmacist 8-hour shift 7-days per week (1.4 full time equivalents) was approximately $ 270,000.

The ED has 30 patient rooms with an estimated 40,000 visits per year and is located in an academic setting with a 4-year EM residency training program. The two EPhs initially provided 8 hours per day coverage and were physically present in the ED 7 days per week. The initial hours of coverage were from 15:30 until midnight and were chosen to cover the period with the highest patient volume. After 4 months, the hours of coverage expanded to 12 hours per day (noon until midnight) after additional funding was obtained. The EPh program was implemented following the general guidelines published by the Agency for Healthcare Research and Quality,[3] and focused on improving medication safety and quality of care delivered through prospective and retrospective review of medication orders, EPh bedside response to all resuscitations, in-person medication-related consultations, and rapid preparation of all urgent medications (e.g., rapid sequence intubation medications, thrombolytics etc.).

Survey Design and Selection of Participants

The survey tool we used was originally developed from qualitative work and has been used previously to assess staff perceptions of EPhs at a different institution.[11] The survey participants identified themselves only by their role in the ED (e.g., nurse, resident) and the researchers remained blinded to the individual participant. All of the participants provided electronic consent prior to taking the survey. There were no incentives offered for completing the survey. The survey consisted of 7 multiple-choice questions, 17 5-point Likert-scale questions (1 - strongly agree, 5 - strongly disagree), and 1 free-text comment section. We further categorized the free-text comments into general praise, constructive feedback, and negative comments. The ED staff was identified using the department's roster of providers. The University of California San Francisco Committee on Human Research approved this study through expedited review.

Data Collection and Processing

In January 2011, 1 year and 3 months after the implementation of the EPh program, we sent the survey electronically via institutional e-mail to the ED staff using software available in the public domain (SurveyMonkey.com, LLC; Palo Alto, California). Participants were allowed a 2-week period to respond to the survey with 1 e-mail reminder sent after the first week. We excluded incomplete surveys (less than 20% answered questions) from the final analysis. The survey was advertised at the institution's residency teaching conference and nursing shift-change to encourage participation.

Primary data analysis

Survey results were exported and analyzed in Microsoft Excel (Microsoft Corporation, Redmond, WA). The results of the survey are presented using descriptive statistics.

RESULTS

Characteristics of Study Subjects

Of the 130 individuals contacted to participate in this study, we received survey responses from 104 (80% response rate) (Table 1). Two participants answered only the first page of the survey (questions 1-4), and we excluded their results from the final analysis. There were a total of 29 attending physicians, 36 residents, 57 nurses, and 8 mid-levels eligible to participate. Clinicians, including attending physicians, EM residents, nurse practitioners, and physician assistants, accounted for 51% of respondents, while nurses accounted for 49%. There were 49 (47.1%) participants who had greater than 6 years of experience in EM.

Main results

Respondents' general perceptions to EPhs are shown in Table 2. Overall, ED providers at our institution supported the presence of an EPh. All of the participants consulted the EPh at least once in their last 5 shifts. Most respondents felt the EPh's availability for general consultation and aid during resuscitations served as the major contribution to medication

Table 1. Demographics of participants surveyed regarding perceptions of emergency department (ED) clinical pharmacists (n=104).

	n (%)
Gender	
Female	67 (64.4)
Role in the ED?	
MD Attending	14 (13.5)
MD Resident	34 (32.7)
Nurse practitioner/Physician assistant	5 (4.8)
Registered nurse	51 (49.0)
Years of experience in emergency medicine?	
<1	13 (12.5)
2 to 5	42 (40.4)
6 to 10	25 (24.0)
>10	24 (23.1)
Years of experience in this ED?	
<1	22 (21.2)
2 to 5	46 (44.2)
6 to 10	17 (16.3)
>10	19 (18.3)

and patient safety. Participants felt that the EPh should review high risk (67.3%) and rarely used medications (60.6%). The nurses felt stronger (54.9%) about the EPh reviewing all medication orders when compared to providers (20.8%).

Survey respondents strongly agreed or agreed that EPh were useful in various clinical situations, including the selection of an appropriate antibiotic (89%), advice on other non-antibiotic medications (94%), drug choice in pregnancy (91%), consultations regarding drug interactions (100%) and toxicology-related cases (85%), assistance with procedural sedation (82%), and resuscitations (96%). The survey participants also felt that the EPh was useful in making medication decisions based on clinical efficacy (87%) and less so based on medication pricing (58%).

The responses to perceptions of the EPh's role in the ED are shown in Table 3. The survey participants strongly agreed that EPhs have a beneficial role in the care of EM patients. The participants also strongly agreed with consulting EPh when they were located in the ED, as opposed to having to call the main pharmacy, with a mean Likert score 1.03. Furthermore, the participants strongly agreed that the EPh was a valuable teaching resource for the ED staff with a mean Likert score of 1.11.

The free-text responses were provided by 62 (60%) participants, and were further categorized into general praise 51 (82.4%), constructive feedback 10 (16%) and 1 negative comment (1.6%), with unedited examples shown in the Appendix. The constructive feedback themes included: increasing hours of EPh coverage, having a dedicated ED pharmacist, encouraging a pharmacist teaching role with the residents, prospective interventions, medication in-services, and increasing training time for new EPhs.

DISCUSSION

This is the first study that evaluates the perceptions of ED staff towards EPh only 1 year after the start of an EPh program. In general, the respondents were very supportive of the EPh. One prior study evaluated a well-established EPh program and showed similar results.[11] Our findings contribute further, indicating that within a short time, the EPh program can be integrated into the EM model to provide support to physicians, nurses, and patients.

Study participants indicated frequent EPh consultation. While nursing staff more often consulted the EPh multiple times during their 5 most recent shifts, all participants consulted the EPh at least once in the same time period. This difference may be due to nurses encountering medication-related issues more frequently in the ED than physician providers (i.e. medication compatibilities, rate of administration, location of medications, high-risk medication double-checks). The difference might also reflect a nurses' initiative to verify the safety of ordered medications prior to administration, as medications in the ED are usually administered without safety mechanisms found in other areas of the hospital. For example, most floor patients receive patient-specific medications that have been verified, labeled, or packaged by pharmacists – adding a safety layer which usually does not exist in an ED environment. Furthermore, our EPh's workstation was adjacent to the resuscitation rooms in close proximity of the main nursing station, which could further explain this difference. Nevertheless, providers and nurses frequently consulted the EPh in person during the study period. The provider-nurse-pharmacist face-to-face interaction may be especially important during resuscitations when high-risk medications are administered at the point of care, usually from verbal orders, in a high-stress environment.

A recent controversial mandate proposed by The Joint Commission to reduce medication errors was the recommendation for prospective pharmacist review of all non-urgent medications administered in the ED. Although later revised and liberated in interpretation due to the EM community's concern about delay in therapy,[10] the mandate did identify an important issue of medication safety in EM. Our EPhs review medication orders prospectively and retrospectively; however, EPh triage their time based on patients' acuity, dedicating most time to prospective provider consultations (~50%), clinical participation in resuscitations and emergent clinical scenarios (~20%), and order review and administrative tasks (~30%). Our survey did not reflect any perceived delays in care other than 1 negative comment, and the majority of participants strongly agreed that the EPh presence improves quality of care in the ED. Further,

Table 2. Staff's general perceptions of the emergency department (ED) clinical pharmacist.

Survey Question	Overall n = 104 n (%)	Providers n = 53 n (%)	Nurses n = 51 n (%)
"How many times in your last 5 shifts in the ED during which an emergency pharmacist was on duty, have you consulted the emergency pharmacist?" (select one)			
Multiple times per shift	62 (59.6)	23 (43.4)	39 (76.5)
At least once per shift	29 (27.9)	21 (39.6)	8 (15.7)
A few times	13 (12.5)	9 (17.0)	4 (7.8)
Not at all	0 (0)	0 (0)	0 (0)
"Which of the following do you think is most important in maximizing the emergency pharmacist's contribution to medication safety? (select one)			
Attend codes/resuscitations	31 (29.8)	12 (22.6)	19 (37.3)
Order review	14 (13.5)	7 (13.2)	7 (13.7)
Being available for consult	53 (51.0)	29 (54.7)	24 (47.1)
Staff education	5 (4.8)	4 (7.5)	1 (2.0)
Patient education	1 (1.0)	1 (1.9)	0 (0)
"Which of the following types of orders should the emergency pharmacist check before they are administered? (select all that apply)"			
All orders	39 (37.5)	11 (20.8)	28 (54.9)
Urgent orders	29 (27.9)	18 (34.0)	11 (21.6)
Non-urgent orders	7 (6.7)	3 (5.7)	4 (7.8)
High risk medications	70 (67.3)	42 (79.2)	28 (54.9)
Rarely used medications	63 (60.6)	35 (66.0)	28 (54.9)

recent multicenter study of comparable EPh programs found a significant interception of medication errors via EPh consultative activities, and to a lesser extent, via order review.[8] In another study, the bedside presence of EPh was associated with decreased door-to-balloon time in ST-segment elevation myocardial infarction.[12] The potential of ED pharmacist to reduce door-to-medication administration times and impact on clinical patient outcomes need further study.

Realistically, it would be challenging to implement a prospective review of all medications ordered and administered in the ED due to frequent emergent situations and clinical scenarios in which delaying therapy would be unethical (i.e. pain control). It is our opinion that the most benefit from EPh's presence in the ED will be gained by proactive clinical support and triaged prospective review of medications based on clinical urgency and the likelihood of the medications causing harm.

The cost containment data associated with EPh presence in the ED has been limited to studies performed in individual institutions.[5-6] The average baseline salary of a hospital pharmacist at our institution ranges from $130,000 – $150,000 per year.[13] However, one prospective observational study showed that during a 4-month period, pharmacist interventions in the ED similar to those performed by our EPhs reduced cost by $192,923 via prevention of additional treatments, drug cost avoidance, and provision of drug consultations.[5] In another study, the investigators found a total cost avoidance of $1,029,776 in the same time frame via prevention of drug interactions/incompatibilities, therapeutic recommendations, and avoidance of adverse drug events and medication errors.[6] Furthermore, our model of EPh practice may represent a financially sound approach to incorporating a pharmacist into the ED, as the EPh fulfilled central pharmacy distributive needs (order processing and dispensing) while physically providing clinical support in the ED via interventions similar to those previously described.

Our survey indicated that only 58% of participants found the EPh useful in decisions based on medication pricing. This may reflect the general medication practice in the ED, where the cost of commonly prescribed medications is infrequently used for clinical decision-making.

One of the concerns voiced in our survey was the heavy reliance of nurses and residents on the EPh for medication-related support. Especially for residents who subsequently practice at sites where EPh are not available, it is possible that EPh may have a detrimental effect on their training. Our group adjusted the EPh practice by extending a bedside teaching role with the residents and the nurses, including periodic medication related in-services. Further, by popular demand from EM residents, a 4-week clinical pharmacy elective was created focusing on drug therapies relevant to EM. The elective has been filled with EM residents 10 out of 25 months since it became available and with pharmacy residents for the remainder of the time. The impact of these added interventions are currently being investigated.

Table 3. Staff's responses concerning the role of the emergency department clinical pharmacist (EPh).

Question	ED staff	Mean score	Agree or strongly agree			Neutral			Disagree or strongly disagree		
			number	% of total	95% CI	number	% of total	95% CI	number	% of total	95% CI
The EPh improves quality of care in ED.	Overall	1.08	103	99	[95, 100]	1	1	[0, 5]	0	0	[0, 3]
	Providers	1.11	52	98	[90 , 100]	1	2	[0, 10]	0	0	[0, 6]
	Nurses	1.04	51	100	[94, 100]	0	0	[0, 6]	0	0	[0, 6]
The EPh is an integral part of the team.	Overall	1.11	102	98	[93, 100]	2	2	[0, 7]	0	0	[0, 3]
	Providers	1.15	51	96	[87, 100]	2	4	[0, 13]	0	0	[0, 6]
	Nurses	1.06	51	100	[94, 100]	0	0	[0, 6]	0	0	[0, 6]
I make more use of pharmacists when they are located in the ED.	Overall	1.03	104	100	[97, 100]	0	0	[0, 3]	0	0	[0, 3]
	Providers	1.04	53	100	[95, 100]	0	0	[0, 5]	0	0	[0, 5]
	Nurses	1.02	51	100	[94, 100]	0	0	[0, 6]	0	0	[0, 6]
It is helpful when the EPh checks medication orders before they are carried out.	Overall	1.20	96	92	[85, 97]	6	6	[2, 12]	2	2	[0, 7]
	Providers	1.28	46	86	[77, 96]	4	10	[2, 19]	2	4	[0, 13]
	Nurses	1.12	50	98	[90, 100]	1	2	[0, 10]	0	0	[0, 6]
EPh during resuscitations enhances my ability to deliver safe quality care to my patients.	Overall	1.20	99	95	[89, 98]	5	5	[2, 11]	0	0	[0, 3]
	Providers	1.28	49	92	[82, 98]	4	8	[2 ,18]	0	0	[0, 6]
	Nurses	1.12	50	98	[90, 100]	1	2	[0, 10]	0	0	[0, 6]
The EPh is a valuable teaching resource.	Overall	1.11	102	98	[93, 100]	2	2	[0, 7]	0	0	[0, 3]
	Providers	1.17	51	96	[87,100]	2	4	[0, 13]	0	0	[0, 6]
	Nurses	1.04	51	100	[94, 100]	0	0	[0, 6]	0	0	[0, 6]
The EPh is valuable as a patient educator.	Overall	1.26	99	95	[89, 98]	5	5	[2, 11]	0	0	[0, 3]
	Providers	1.25	50	94	[84, 99]	3	6	[1, 16]	0	0	[0, 6]
	Nurses	1.27	49	96	[87, 100]	2	4	[0, 13]	0	0	[0, 6]

LIMITATIONS

The limitations of our study include that our EPh program was established at a single university teaching hospital and that the results may not be generalizable to other ED settings. Additionally, one EPh was a residency-trained pharmacist while the other had experience in critical care,and our results may not reflect pharmacists with different levels of training. The EPh was physically present in the ED up to `12 hours per day, and acceptance of programs with less presence and visibility might vary.

The ED staff's perceptions of existing pharmacy services prior to the implementation of the EPh were not collected. Since the EPh was a novel intervention, there is a possibility that participants may have over-estimated the benefit of the EPh due to the prior minimal pharmacy support.

We are limited by the survey design and response rate, especially from the attending physicians. The results may have been subject to recall bias, and it is also possible that there was significant non-response bias. However, even if those with highly negative attitudes were more likely to not respond, our results would not vary substantially. For example, we performed a sensitivity analysis showing that if all 26 eligible participants who did not respond put all of their responses in the "strongly disagree" category, the overall Likert-mean score would change to 2.08, or "agree" overall.

CONCLUSION

The EPh model of practice at our institution, based on a model proposed by Faribanks,[3] maximizes the useof clinical pharmacists in the ED and provides valuable perceived benefit to providers. Influence on patient outcomes and the highest-impact model of practice for EPh will need to be addressed with future studies.

Address for Correspondence: Zlatan Coralic, PharmD, BCPS. 505 Parnassus Avenue M39, San Francisco, CA 94143. Email: zlatan.coralic@ucsfmedctr.org.

Conflicts of Interest: By the *West*JEM article submission agreement, all authors are required to disclose all affiliations, funding sources and financial or management relationships that could be perceived as potential sources of bias. Dr. Hemal Kanzaria is supported by the Robert Wood Johnson Foundation Clinical Scholars program.

REFERENCES

1. Elenbaas RM, Waeckerle JF, McNabney WK. The clinical pharmacist in emergency medicine. *Am J Hosp Pharm*. 1977;34(8):843–846.
2. Rudis MI, Attwood RJ. Emergency medicine pharmacy practice. *J Pharm Pract*. 2011;24(2):135–145.
3. Clinical pharmacists in emergency medicine. Agency for Healthcare Research and Quality 2008. Available at: http://www.ahrq.gov/downloads/pub/advances2/vol4/advances-fairbanks_20.pdf. Last accessed December 14, 2012.
4. Eppert HD, Reznek AJ. ASHP Guidelines on Emergency Medicine Pharmacist Services. *Am J Health Syst Pharm*. 2011;68(23):e81–e95.
5. Ling JM, Mike LA, Rubin J, et al. Documentation of pharmacist interventions in the emergency department. *Am J Health Syst Pharm*. 2005;62:1793-1797
6. Lada P, Delgado G. Documentation of pharmacists' interventions in an emergency department and associated cost avoidance. *Am J Health Syst Pharm*. 2007; 64:63-68
7. Rothschild JM, Churchill W, Erickson A, et al. Medication errors recovered by emergency department pharmacists. *Ann Emerg Med*. 2010;55(6):513–521.
8. Patanwala AE, Sanders AB, Thomas MC, et al. A prospective, multicenter study of pharmacist activities resulting in medication error interception in the emergency department. *Ann Emerg Med*. 2012;59(5):369–373.
9. Cesarz JL, Steffenhagen AL, Svenson J, et al. Emergency department discharge prescription interventions by emergency medicine pharmacists. *Ann Emerg Med*. 2013;61(2):209–214.e1.
10. Medication reconciliation standard letter to the Joint Commission. American College of Emergency Physicians, 2006. Avaliable at http://www.acep.org/Clinical---Practice-Management/Medication-Reconciliation-Standard-Letter-to-Joint-Commission---May-2006/. Last accessed December 14, 2012.
11. Fairbanks RJ, Hildebrand JM, Kolstee KE, et al. Medical and nursing staff highly value clinical pharmacists in the emergency department. *Emerg Med J*. 2007;24(10):716–718.
12. Acquisto NM, Hays DP, Fairbanks RJT, et al. The Outcomes of Emergency Pharmacist Participation during Acute Myocardial Infarction. *J Emerg Med*. 2012;42(4):371–378.
13. University of California Title and Pay Plan, 2012. Available at: http://ucsfhr.ucsf.edu/index.php/staffing/tpp_detail?tc=9247. Last accessed December 14, 2012.

Depression, Suicidal Ideation, and Suicidal Attempt Presenting to the Emergency Department: Differences Between These Cohorts

Bharath Chakravarthy, MD, MPH
Wirachin Hoonpongsimanont, MD
Craig L. Anderson, PhD
Michael Habicht, MD
Tim Bruckner, PhD
Shahram Lotfipour, MD, MPH

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

Supervising Section Editor: Leslie Zun, MD, MBA
Submission history: Submitted July 30, 2012; Accepted November 22, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.11.13172

Introduction: The World Health Organization estimates that one million people die by suicide every year. Few studies have looked at factors associated with disposition in patients with chief complaints of depression, suicidal ideation (SI) and suicidal attempts (SA) who present to the emergency department (ED). Our objective was to assess individual determinants associated with ED disposition of patients in depressed patients presenting to the ED.

Methods: We conducted a retrospective study using the National Hospital Ambulatory Medical Care Survey from 2006 to 2008. We used logistic regression to identify factors associated with discharge, in SI, SA and depression patients. Independent variables included socio-demographic information, vital signs, mode of arrival, insurance status, place of residence and concomitant psychiatric diagnosis.

Results: Of the 93,030 subjects, 2,314 met the inclusion criteria (1,362 depression, 353 SI and 599 SA). Patients who arrived by ambulance were less likely to be discharged (odds ratio [OR] 0.63, 95% confidence interval [CI] 0.43-0.92). Hispanic patients and patients age 15 to 29 were likely to be discharged (OR 1.61, 95% CI 1.16-2.24 and OR 1.55, 95% CI 1.15-2.10 respectively). Insurance status and housing status were not significantly associated patient was being discharge from EDs.

Conclusion: The Hispanic population had higher discharge rates, but the reasons are yet to be explored. Patients with SA and SI are discharged less frequently than those with depression, regardless of insurance type or housing status. [West J Emerg Med. 2014;15(2):211–216.]

INTRODUCTION

According to the World Health Organization (WHO) approximately one million people die by suicide every year, with a global mortality rate of 16 per 100,000. In 2004 suicide was the 16th leading cause of death worldwide.[1] This statistic, however, does not include additional morbidity caused by failed attempts and injuries, as well as visits to the emergency department (ED) for suicidal thoughts and ideation.

In the United States (U.S.), suicide ranks as the 11th leading cause of death and accounts for more than 33,000 deaths annually.[2] In 2005 Doshi found that suicide and self-inflicted injury accounted for more than 400,000 ED visits per year from 1997-2001 (0.4% of all ED visits).[3] This high burden of disease has made suicide prevention one of the goals of Healthy People 2010,[4] and the U.S. Surgeon General has recognized the need for improved identification, diagnosis and treatment of suicidal ideation.[5]

Several studies describe specific risk factors for suicidal ideation and suicide attempt. These factors include prior attempts, feelings of worthlessness or hopelessness, and

recent stressors.[6-10] To date no study has looked at risk factors associated with discharge status for patients who visit the ED with complaint of depression, suicidal ideation or suicidal attempt.

Our goal was to identify characteristics of patients who are safe for discharge. The objective of this study was to identify determinants associated with discharge in ED patients presenting with suicidal attempt (SA), suicidal ideation (SI) and depression.

METHODS

Study Design

We retrospectively examined predictors of discharge from the ED among patients with any diagnosis of SA, SI or depression, using the National Hospital Ambulatory Medical Care Survey (NHAMCS) data for the years 2006 to 2008. Because the NHAMCS coding included codes for depression in very young children, which we believe represent confusion between respiratory depression and depression, we restricted the data to patients age 6 years and older.

The NHAMCS is a national survey completed by the Centers for Disease Control (CDC) that provides a sampling of ED and outpatient visits, excluding federal, military and VA hospitals. NHAMCS has been used in multiple studies to generate national estimates of ED visits.[3,11-13]

We included 3 categories of ED visits using information from 3 cause-of-injury codes, 3 Patient Reason-for-Visit (RFV) codes, and 3 International Classification of Disease (ICD) codes in the NHAMCS data:

1. Suicide attempts were defined as any patient with injury codes related to overt self-inflicted injury (E950.0-E958) or RFV codes for suicide attempt (5820.0) or intentional overdose (5820.1).
2. Suicidal ideation was defined as any patient with an ICD supplemental code (V62.84) for that condition, without SA.
3. Depression were defined with ICD diagnostic codes 296.2, 296.3, 296.5, 298.0, or 311; or RFV 1110.0, without SA or SI.

The study used only data from an existing national survey and did not require institutional review board approval.

Data Analysis

We analyzed the data using STATA version 12.0, StataCorp, College Station, TX. We used Stata survey procedures, which take into account both clustering by survey location and data weighting, to estimate the percent of U.S. ED visits with various patient characteristics.

We used logistic regression to identify factors associated with discharge in SI, SA and depression patients. All of the variables of interest were included in a single logistic model to provide mutually adjusted estimates of the odds ratio for discharge from the ED. The logistic regression also used Stata survey procedures to adjust for clustering and weighting.

Definitions

1. We classified age into 5 groups to allow us to define a variety of relationship between age and the outcomes studied without making assumptions about the shape of these relationships. We categorized race and ethnicity into non-Hispanic white, non-Hispanic black, Hispanic, and others (Asian, Native American, Pacific Islander and More than one race).
2. We classified outcome as died, admitted to an intensive care unit (ICU), admitted to a psychiatric unit, admitted to another unit, left before assessment and treatment was completed, or discharged from the ED. Transferred patients were included in the admissions to a psychiatric unit, as psychiatric care was the most common reason for transfer.
3. We classified mode of arrival by emergency medical services (EMS) or other.
4. We considered triage vital signs abnormal if patient has temperature above 100.4 F, pulse > 100 beats per minute, oxygen saturation less than 93% or systolic blood pressure less than 90 mmHg
5. We grouped expected source of payment into 3 categories: insurance, including private insurance, Medicare, and worker's compensation; Medicaid; and self-pay, no charge, and other payment (any other sources of payment not covered by the prior categories)
6. We considered 4 categories of other psychiatric diagnosis as independent variables: mood disorder, anxiety, psychotic disorder, and substance abuse, following the definitions of ICD-9. All the depressed patients by definition had a mood disorder.
7. We categorized housing types as private residence; other residence, which includes nursing homes and institutions; and homeless.

RESULTS

Patient characteristics are shown in Table 1. Of 93,030 charts reviewed, 2,314 met our search criteria: 599 charts reported suicidal attempt (SA), 353 reported suicidal ideation (SI) but not SA and 1,362 reported depression with neither SI or SA. Using the sample weights, these cases represent a national total of 1,800,000 cases of SA (95% CI 1,500,000-2,100,000), 1,100,000 cases of SI without SA (95% CI 950,000-1,300,000), and 3,200,000 cases of depression without SI or SA (95% CI 2,700,000-3,600,000). A slightly higher percentage of female than of male patients appeared in all 3 groups. Patients in age group 15 to 29 were the largest age group among patients attempting suicide; patients 30-49 was the largest age group among those with suicidal ideation or depression. The majority of patients were non-Hispanic white, either self-pay or with Medicaid, and presented at the triage with normal vital signs. Half of the total (50.0%) were discharged from or left the ED. Forty-nine point seven percent of patients were admitted to the hospital in different units (ICU 4.2%, psychiatry unit 39.4% and other medical unit 6.1%); 0.3 % of patients died.

Table 1. Percentage of patients with conditions of suicide attempt, ideation and depression by various demographic, outcome, financial categories and associated disorders (with 95% confidence interval).

Patient Characteristic	SA n=599	SI n=353	Depression n=1,362	Total N=2314
Male	44.4 (39.6-49.3)	49.1 (42.5-55.7)	43.3 (38.7-48.0)	44.7 (41.7-47.7)
Age (years)				
6 to 14	4.6 (2.6-7.8)	3.2 (1.7-6.0)	3.5 (2.3-5.3)	3.8 (2.7-5.2)
15 to 29	41.2 (35.4-47.2)	37.0 (30.1-44.5)	26.8 (23.1-30.8)	33.0 (30.1-36.0)
30 to 49	37.8 (32.5-43.4)	43.1 (36.3-50.2)	45.5 (41.2-49.9)	42.8 (40.0-45.6)
50 to 69	14.8 (11.2-19.3)	15.6 (10.7-22.2)	16.8 (14.0-20.0)	16.0 (14.1-18.0)
70 or more	1.6 (0.6-4.3)	1.1 (0.2-5.0)	7.4 (5.4-10.0)	4.5 (3.4-6.0)
Race/Ethnicity				
White non-Hispanic	66.0 (60.5-71.2)	66.3 (57.8-73.8	63.5 (58.5-68.2)	64.8 (60.9-68.4)
Black non-Hispanic	13.2 (9.3-18.4)	18.4 (13.2-25.1)	14.3 (11.4-17.8)	14.7 (12.1-17.8)
Hispanic	6.2 (4.3-8.8)	5.8 (3.6-9.1)	8.1 (6.1-10.6)	7.1 (5.7-8.8)
Other, multiple, and unknown	14.6 (10.2-20.4)	9.5 (5.5-15.8)	14.2 (10.7-18.6)	13.4 (10.7-16.7)
Outcome				
Died	0.8 (0.3-2.2)	0.0	0.2 (0.0-1.4)	0.3 (0.1-0.8)
Admit intensive care unit	11.1 (7.8-15.5)	0.6 (0.1-4.0)	1.4 (0.7-2.8)	4.2 (3.0-5.7)
Admit psych	42.8 (37.4-48.4)	66.3 (58.5-73.3)	27.7 (23.4-32.3)	39.4 (36.1-42.9)
Admit other	5.7 (3.5-9.1)	5.6 (3.4-9.2)	6.4 (4.7-8.8)	6.1 (4.7-7.8)
Left	0.9 (0.3-2.3)	1.0 (0.4-3.1)	3.1 (1.9-4.9)	2.0 (1.4-3.0)
Discharge	38.7 (33.8-43.9)	26.5 (20.1-34.0)	61.2 (56.5-65.8)	48.0 (44.4-51.5)
Arrive by emergency medical services	52.8 (47.2-58.4)	25.1 (19.1-32.3)	21.7 (18.0-26.0)	31.7 (28.6-35.1)
Abnormal vitals	31.8 (27.5-36.4)	40.0 (34.0-46.4)	27.8 (23.8-32.1)	31.2 (28.6-34.0)
Expected Payer				
Insurance	46.5 (41.0-52.0)	39.3 (32.1-47.0)	50.6 (46.0-55.2)	47.2 (43.9-50.7)
Medicaid	27.3 (22.8-32.3)	26.2 (20.2-33.1)	25.0 (21.2-29.3)	25.9 (22.9-29.1)
Self Pay/None/Other	26.2 (22.0-31.0)	34.5 (26.9-43.0)	24.4 (20.5-28.7)	26.9 (23.9-30.0)
Other psychiatric diagnosis				
Anxiety	2.9 (1.5-5.5)	8.3 (5.5-12.4)	11.8 (9.4-14.7	8.5 (7.0-10.3)
Mood disorder	37.5 (30.9-44.6)	53.6 (46.4-60.7)	100 (by definition)	72.8 (69.3-76.0)
Psychotic disorder	3.1 (1.8-5.1)	12.3 (8.5-17.5)	7.5 (5.7-10.0)	7.1 (5.7-8.9)
Substance related	17.0 (13.6-21.1)	19.4 14.3-25.7	17.9 (14.9-21.3)	17.9 (15.7-20.4)

Table 2. Mutually adjusted odds ratios for factors associated with discharge disposition (n=1866 cases with complete data for these variables). From logistic regression using sample weights and clustering.

Factors	Odds ratio	95% confidence interval
Depression	1	Reference
Suicidal attempt	0.44	0.31 – 0.61
Suicidal ideation	0.20	0.12 – 0.31
Anxiety	1.36	0.77 – 2.40
Psychotic disorder	0.54	0.30 – 0.97
Substance-related	1.16	0.74 – 1.81
Arrive by emergency medical services	0.64	0.45 – 0.90
Abnormal vital signs	0.78	0.55 – 1.12
Male	0.75	0.54 – 1.03
Age ≥ 14	2.07	0.78 – 5.46
Age 15-29	1.59	1.14 – 2.23
Age 30-49	1	Reference
Age 50-69	0.67	0.45 – 0.99
Age ≥ 70	0.88	0.43 – 1.81
Non-Hispanic white	1	Reference
Non-Hispanic black	0.75	0.50 – 1.13
Hispanic	1.47	0.94 -- 2.30
Other	1.13	0.73 – 1.76
Private residence	1	Reference
Other residence	1.03	0.58 – 1.84
Homeless	1.02	0.51 - 2.03
Private insurance, worker compensation, and Medicare	1	Reference
Medicaid	0.92	0.64 - 1.33
Self-pay	1.25	0.83 - 1.87

Patients with SA (OR=0.43, 95% CI 0.31-0.61) or SI (OR=0.20, 95% CI 0.12-0.31) were much less likely to be discharged from the ED than patients with depression alone (Table 2). Among patients with depression, SA, or SI, patients with psychotic disorders (OR=0.54, 95% CI 0.30-0.97) and patients who arrived by EMS (OR=0.64, 95% CI 0.45-0.90) were less likely to be discharged. The odds of discharge from the ED tended to decrease with increasing age; patients aged 15-29 had higher odds (OR 1.59, 95% CI 1.14-2.23) and patients aged 50-69 had lower odds (OR=0.67, 95% CI=0.45-0.99) than patients aged 30-49. Sex, race, ethnicity, vital signs, type of insurance, and housing type were not associated with discharge status in these patients.

DISCUSSION

SI and SA patients had lower odds of being discharged when compared to depressed patients. This is expected, as clinical decisions in the ED tend to be cautious regardless of a patient's level of suicidality.[7] ED visits related to suicide have increased in volume over the last 2 decades.[8] Nationally we are facing an increase in the number of patients visiting EDs for psychiatric reasons. [9] This will undoubtedly add a strain to our existing healthcare budget. As such, admitting all patients with self-harm may be unrealistic.[10] A study conducted in 2012 showed excess mortality rates in patients who arrived in the ED with suicidal chief complaints when compared to that of the general population.[27] It is essential to identify high-risk patients effectively. Special caution must be taken by emergency physicians (EP) to ensure prevention of suicide-related mortality.

A recent study showed that 60%of transition from ideation to plan and attempt occur within the first year after ideation onset.[19] This is an opportune time to intervene with the cohort of patients who are expressing SI. Unfortunately to this date, few studies have shown a reduction in emergency healthcare use in these patients[11, 20-25].

Gairin et al[10] found that patients who received psychosocial assessment prior to discharge were more readily identified as high risk, and Kapur et al[11] found that a psychosocial assessment halved the risk of repetition of self-harm. Psychosocial assessment completed by case managers is a realistic and cost-effective intervention that can be done in the ED.[11] ED visits for psychiatric chief complaints can be reduced if there are resources available for outpatient

psychiatric services.[12] Further research should investigate longitudinal healthcare index markers after discharge from the ED or inpatient setting to explore if there is a difference in mental health when patients are admitted or discharged for SI, SA and depression.

Insurance status was not a factor in determining odds of being discharged. Similarly, a 3-hospital study conducted in 2007 showed that insurance status was not related to hospitalization decisions regarding suicide-related visits.[13] Yet another study showed that the uninsured were found to be least likely to be admitted for all mental disorders except suicide.[28] This suggests that disposition decisions in the ED tend to be extra conservative regarding suicidal patients and favor admission, regardless of patients' insurance status. This coincides with the ED philosophy of safeguarding against life-threatening illnesses first. On the one hand, it is refreshing to see that EPs are making disposition decisions regardless of payor type. On the other hand, our changing healthcare system may add an even greater responsibility on EPs and their ability to safely discharge the proper cohort of patients with self-harm.

Notably, being Hispanic increased the odds of being discharged from the ED, even when compared to other minorities and adjusted for other factors such as age, payor status, housing status and previous psychiatric illnesses. Many plausible explanations for our study result exist. Disparity in mental healthcare among Hispanic could be one of the reasons. Few studies found Latinos and Hispanics received depression care less than whites.[14,15] Furthermore, many Spanish-speaking people in the U.S. reported they do not speak English well or at all.[16] Bilingual patients are evaluated differently when interviewed in English as opposed to Spanish and that Hispanics are more frequently under-treated.[17] It is possible that depression complaints might be mistaken for nervousness, tiredness or a physical ailment in Hispanics.[17] Many Hispanics rely on help from their extended family and traditional healers during a mental health crisis.[17] (A study in Colorado reported that Hispanic populations underreported suicidal ideation and this could be a reason for less admission in the Hispanic group.[18]) Therefore, EPs should still consider other factors, such as abnormal vital signs or underlying psychiatric disease, when considering admission in this population.

LIMITATIONS

The limitations of this study stem from 2 sources: the limitations of using a large national survey that provides probability samples such as NHAMCS and the limitations of using depression as a marker for SI in the ED.

While national surveys such as NHAMCS are valuable and can be helpful for large epidemiologic studies, one must recognize the problems inherent in such a system. First, to create the trends and conclusions in this paper we have made a few assumptions concerning the data: (a) the sample is representative of the target population (in our case patients presenting to the ED with SI or SA); and (b) the data are an accurate representation of the true values for each. However, as discussed in a recent Annals of Emergency Medicine Journal Club these assumptions have the potential to be incorrect.[19]

Other limitations of NHAMCS include errors in data collection and reporting, such as the cluster of 0-6 year-olds with "depression." In addition to general coding errors, some errors were created by the nature of our study. For example, coding may miss unusual or severe attempts where the cause of the acute illness was not determined immediately and coding could miss suicidal ideation with other presentation.

Our study was also limited in the use of depression as a marker for suicidality. Suicidal ideation is a new code included starting in 2006 in the NHAMCS data, but we found that this was likely underutilized as there were more SAs than SIs in the data, which is counter to our experience in the ED. The National Health Interview Survey asks about a variety of mental health disorders, but does not directly ask about suicidal thoughts or behaviors. The Behavior Risk Factor Surveillance System Questionnaire is an adult survey that is completed; however, it only includes information on suicidality from 2009-2010. This will be an excellent source for future studies.

CONCLUSION

Our results show that being Hispanic has higher tendency to be discharged from ED after patient was evaluated for depression, SI, SA. Although healthcare providers should be aware of disparity in access to healthcare, cultural difference and language barriers as explanations for this possible false protective factors in this Hispanic population. Patient with SA and SI were being treated with extra precautions, which resulted in lower discharge rate which reflects emergency medicine mentality. Insurance status was not associated with disposition from ED in these patient populations. With changes in healthcare systems, the providers will need to be cautious about patient disposition either admission or discharge patients for the EDs. Further study is needed to explore whether resource availability including follow up appointment plays any role in discharge decision.

Address for Correspondence: Wirachin Hoonpongsimanont, MD. Department of Emergency Medicine, 101 The City Drive Rte 128-01 Suite 710, Orange, CA 92868. Email: Wirachin@gmail.com.

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

REFERENCES

1. Mathers C, Fat DM, Boerma JT, et al. The global burden of disease*: 2004* update. *Geneva, Switzerland: World Health Organization*. 2008.
2. Centers for Disease Control and Prevention (CDC). Web-based Injury Statistics Query and Reporting System (WISQARS) [Online]. National Center for Injury Prevention and Control, CDC. Available at: www.cdc.gov/ncipc/wisqars/default.htm. Accessed July 20, 2010.
3. Doshi A, Boudreaux ED, Wang N, et al. National study of US emergency department visits for attempted suicide and self-inflicted injury, 1997-2001. *Ann Emerg Med.* 2005;46(4):369-375.
4. U.S. Department of Health and Human Services. Healthy People 2010. 2nd ed. With Understanding and Improving Health and Objectives for Improving Health. Washington, DC: U.S. Government Printing Office. 2000.
5. U.S. Public Health Service. The Surgeon General's Call to Action to Prevent Suicide. Washington, DC: U.S. Department of Health and Human Services, Office of the Surgeon General. 1999.
6. Crandall C, Fullerton-Gleason L, Aguero R, et al. Subsequent suicide mortality among emergency department patients seen for suicidal behavior. *Acad Emerg Med.* 2006;13(4):435-442.
7. Drew BL, Jones SL, Meldon SW, et al. Emergency department visits for suicidality in three hospitals. *Arch Psychiatr Nurs.* 2006;20(3):117-125.
8. Ting SA, Sullivan AF, Boudreaux ED, et al. Trends in US emergency department visits for attempted suicide and self-inflicted injury, 1993-2008. *General Hospital Psychiatry.* 2012 (article in press)
9. Chakravarthy B, Tenny M, Anderson CL, et al. Analysis of mental health substance abuse-related emergency department visits from 2002 to 2008. Subst Abus. 2013;34(3):292-297.
10. Gairin I, House A, Owens D. Attendance at the accident and emergency department in the year before suicide: retrospective study. *Br J Psychiatry.* 2003;183:28-33.
11. Kapur N, House A, May C, et al. Service provision and outcome for deliberate self-poisoning in adults - Results from a six centre descriptive study. *Soc Psychiatry Psychiatr Epidemiol.* 2003;38(7):390-395.
12. Bruckner TA, Yonsu K, Chakravarthy B, et al. Voluntary psychiatric emergencies in Los Angeles County after funding of California's Mental Health Services Act. *Psychiatr Serv.* 2012 Aug;63(8):808-814.
13. Goldberg JF, Ernst CL, Bird S. Predicting hospitalization versus discharge of suicidal patients presenting to a psychiatric emergency service. *Psychiatric Services*. 2007;58:561-565.
14. Lagomasino IT, Dwight-Johnson M, Miranda J et al. Disparities in Depression Treatment for Latinos and Sites of Care. Psychiatric Services. 2005; 56(12):1517-23
15. Alegría M, Chatterji P, Wells K, et al. Psychiatr Serv. 2008;59(11):1264-1272.
16. US Census. (2003) Summary Tables on Language Use and English Ability: 2000. Available at: www.census.gov/population/www/cen2000/briefs/phc- t20/index.html
17. APA Fact Sheet. Mental Health Disparities:Hispanic/Latinos. American Psychiatric Association, Office of Minority and National Affairs. 2010
18. Betz ME, Krzyzaniak SM, Hedegaard H, et al. Completed suicides in Colorado: differences between Hispanic and non-Hispanic whites. S*uicidal and life Threatening Behavior*. 2011;41(4):445-452.
19. Cooper RJ, Schriger DL. Annals of Emergency Medicine Journal Club. The NHAMCS database, chart review methods, and more on regression modeling answers to the May 2010 journal club questions. *Ann Emerg Med.* 2010;56(4):431-437.
20. Houry D. Suicidal patients in the emergency department: Who is at greatest risk? *Ann Emerg Med.* 2004;43(6):731-732.
21. Miller M, Azrael H, Hemenway D. The epidemiology of case fatality rates for suicide in the northeast. *Ann Emerg Med*. 2004;43(6):723-730.
22. Ting SA, Sullivan AF, Miller I, et al. Multicenter study of predictors of suicidal screening in emergency departments. *Acad Emerg Med*. 2012;19:139-143.
23. Larkin GL, Smith RP, Beautrais AL. Trends in US Emergency Department visits for suicide attempts, 1992-2001. *Crisis.* 2008;29(2):73-80.
24. Larkin GL, Claassen CA, Emond JA, et al. Trends in US Emergency Department visits for mental health conditions, 1992 to 2001. *Psychiatr Serv.* 2005;56(6):671-677.
25. Harman JS, Scholle SH, Edlund MJ. Emergency department visits ford depression in the United States. *Psychiatr Serv.* 2004;55(8):937-939.
26. Nock MK, Borges G, Bromet EJ, et al. Cross-national prevalence and risk factors for suicidal ideation, plans and attempts. *Br J Psychiatry.* 2008;192(2):98-105.
27. Carter GL, Clover K, Whyte IM, et al. Postcards from the EDge project: randomised controlled trial of an intervention using postcards to reduce repetition of hospital treated deliberate self poisoning. *Br Med J.* 2005;331(7520):805-+.
28. Carter GL, Clover K, Whyte IM, et al. Postcards from the EDge: 24-month outcomes of a randomised controlled trial for hospital-treated self-poisoning. *Br J Psychiatry.* 2007;191:548-553.
29. Cedereke M, Monti K, Ojehagen A. Telephone contact with patients in the year after a suicide attempt: does it affect treatment attendance and outcome? A randomised controlled study. *Eur Psychiat.* 2002;17(2):82-91.
30. Larkin GL, Beautrais AL. Emergency Departments Are Underutilized Sites for Suicide Prevention. *Crisis.* 2010;31(1):1-6.
31. Mann JJ, Apter A, Bertolote J, et al. Suicide prevention strategies - A systematic review. *JAMA-J Am Med Assoc.* 2005;294(16):2064-2074.
32. Repper J. A review of the literature on the prevention of suicide through interventions in Accident and Emergency Departments. *J Clin Nurs.* 1999;8(1):3-12.
33. Choi JW, Park S, Yi KK, et al. Suicide Mortality of Suicide Attempt Patients Discharged for Emergency Room, Nonsuicidal Psychiatric Patients Discharged from Emergency Room, Admitted Suicide Attempt Patients, and Admitted Nonsuicidal Psychiatric Patients. *Suicide and Life-Threatening Behavior.* 2012;19:42(3):235-243
34. Hazlett SB, McCarthy ML, Londner MS, et al. Epidemiology of Adult Psychiatric Visits to U.S. Emergency Departments. *Acad Emerg Med.* 2004;11(2):193-195

Prospective Analysis of Single Operator Sonographic Optic Nerve Sheath Diameter Measurement for Diagnosis of Elevated Intracranial Pressure

Erica Frumin, MD
Joelle Schlang, BS
Warren Wiechmann, MD, MBA
Stacy Hata, BS
Sasha Rosen, BS
Craig Anderson, PhD, MPH
Laura Pare, MD
Mark Rosen, MD
John Christian Fox, MD

University of California at Irvine School of Medicine, Irvine, California

Supervising Section Editor: Laleh Gharahbaghian, MD
Submission history: Submitted February 22, 2013; Revision received June 13, 2013; Accepted September 16, 2013
Electronically published Novmeber 27, 2011
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.9.16191

Introduction: The accurate diagnosis of elevated intracranial pressure (eICP) in the emergent setting is a critical determination that presents significant challenges. Several studies show correlation of sonographic optic nerve sheath diameter (ONSD) to eICP, while others show high inter-observer variability or marginal performance with less experienced sonographers. The objective of our study is to assess the ability of bedside ultrasound measurement of ONSD to identify the presence of eICP when performed by a single experienced sonographer. We hypothesize that ONSD measurement is sensitive and specific for detecting eICP and can be correlated with values obtained by external ventricular device (EVD).

Methods: This was a prospective blinded observational study conducted in a neurocritical care unit of a level 1 trauma center. ONSD measurement was performed on a convenience sample of 27 adult patients who required placement of an invasive intracranial monitor as part of their clinical care. One certified sonographer/physician performed all ultrasounds within 24 hours of placement of EVD. The sonographer was blinded to the ICP recorded by invasive monitor at the time of the scan. A mean ONSD value of ≥5.2 mm was taken as positive.

Results: The sonographer performed 27 ocular ultrasounds on individual patients. Six (22%) of these patients had eICP (EVD measurement of >20 mmHg). Spearman rank correlation coefficient of ONSD and ICP was 0.408 (p=0.03), demonstrating a moderate positive correlation. A ROC curve was created to determine the optimal cut off value to distinguish an eICP greater than 20 mmHg. The area under the receiver operator characteristic curve was 0.8712 (95% confidence interval [CI]=0.67 to 0.96). ONSD ≥5.2 mm was a good predictor of eICP (>20 mmHg) with a sensitivity of 83.3% (95% CI=35.9% to 99.6%) and specificity of 100% (95% CI=84.6% to 100%).

Conclusion: While the study suggests ONSD measurements performed by a single skilled operator may be both sensitive and specific for detecting eICP, confirmation in a much larger sample is needed. Ocular ultrasound may provide additional non-invasive means of assessing eICP . [West J Emerg Med. 2014;15(2):217–220.]

INTRODUCTION

Elevated intracranial pressure (eICP) is common and potentially fatal. Timely diagnosis of this condition has a positive impact on morbidity and mortality, but the accurate evaluation of eICP presents a challenging diagnostic dilemma.[1] The gold standard for diagnosis of eICP, external ventricular device (EVD), is highly invasive, unavailable in the initial assessment, and may be contraindicated in coagulopathic patients. Neuroimaging modalities, including computed tomography (CT) or magnetic resonance imaging (MRI), provide delayed diagnosis and are logistically difficult in agitated or unstable patients.[2] Moreover, clinical diagnosis of eICP by emergency physicians has shown poor performance when compared to CT or optic nerve sheath diameter (ONSD) measurement.[3]

Physicians have observed and described the response of ONSD to changes in ICP extensively.[4-6] The optic nerve is surrounded by subarachnoid cerebrospinal fluid, which is encased in the optic nerve sheath, an extension of the dura mater. As ICP increases, the subarachnoid space fills, resulting in an increased ONSD. The expansion is greatest at the retrobulbar terminating segment of the nerve.[7] The expansion of the sheath is immediate and predictable.[4,5,8] These characteristics suggest that ONSD is a sensitive and early marker for eICP and may provide particular utility for monitoring the changing status of critical patients.

The clinical application of this anatomical phenomenon shows promise for diagnosing eICP. Sonographic measurement of ONSD has been shown to be a useful predictor of eICP when compared to CT findings of eICP.[3,9] Several studies have shown that ONSD is a good predictor of eICP as measured by EVD, while others have shown poor inter-rater reliability or inadequate image acquisition by less experienced operators.[9-13]

The range of proposed cut-off values for detection of eICP varies from 4.8 to 5.86 mm.[11,13] The small dimensions of the ONSD, variation in sonographic technique, operator experience, and observation of artifacts all may contribute to the lack of consensus on an optimal cut-off value.

In this study a single, experienced sonographer performed all scans to eliminate the variable of multiple operators and determine whether a single operator using a standard technique can demonstrate whether ONSD can predict eICP. To the best of our knowledge, no studies thus far have examined the correlation between ONSD performed by a single experienced sonographer with direct ICP measurements from EVD.

METHODS

This was a blinded prospective observational study performed on a convenience sample of patients presenting with a variety of intracranial processes in the neurocritical care unit of an urban level 1 trauma center between September 16, 2008, and July 11, 2009. Subjects included adult patients with invasive intracranial monitors placed during weeks that the study physician was available to perform the scans. Consent was obtained from the patients or the appropriate surrogates and the local institutional review board at our hospital approved this study. We excluded patients with ocular trauma or a known history of ocular pathology.

Ocular ultrasounds were performed on a Sonosite M-turbo (SonoSite Inc., Bothell, WA) machine with a 5–10 MHz linear probe.[15] One physician with RDMS certification and 3 years of clinical experience with sonography, including over 250 ocular ultrasound exams, performed all scans on patients in the supine position. Scans were performed on a closed eyelid with a generous amount of conductive gel. A linear probe was used to obtain an axial cross-sectional image of the optic nerve entering the fundus. The ultrasonographer fanned the globe superiorly and inferiorly to help visualize margins of the nerve. An average of 3 to 4 ultrasounds was required to obtain an optimal image with clear margins of the optic sheath. ONSD was measured from outer wall to outer wall 3 mm posterior to the globe.

Artifacts were avoided by instructing alert patients to look directly ahead to ensure that the visual axis was centered with the optic nerve running perpendicular to the fundus. The lids of unconscious patients were opened to assess globe axis. Only images taken with the optic nerve centered and with clear optic sheath margins were accepted. Clear images of the ONSD could be obtained for all patients. The sonographer accepted one measurement on each eye. The mean binocular ONSD was the outcome selected for statistical analysis. All measurements were obtained within 24 hours of EVD placement. Research associates recorded a single ICP measurement at the time of the scan and the study physician/ultrasonographer was blinded to the reading.

Research associats kept data logs and input the data to Stata (version 12.0, Stata Corp, College Station, TX). We performed a Shapiro-Wilk test to evaluate the ONSD and ICP distributions for normality, and produced a scatter plot to examine the data. A non-parametric Spearman rank correlation coefficient with a two-tailed p-value was used to assess for an association between the ONSD and ICP measurements. We assessed optimal cut-off value to detect ICP >20 mmHg, the pressure at which aggressive treatment usually begins, with a receiver operator characteristic (ROC) curve. We computed 95% confidence intervals (CIs) for sensitivity and specificity of potential cut-off values using exact statistics.

RESULTS

The sonographer performed 27 ocular ultrasounds on individual patients, 18 males and 9 females, with an average age of 45.6 years (range 18-76). Ten patients had hemorrhagic strokes (37%), 9 had subarachnoid hemorrhage (33%), 6 suffered blunt head trauma with various intracranial bleeds (22%), one had obstructive hydrocephalus (4%) and one had neurocysticercosis (4%). EVD showed eICP >20 cm H_20 in

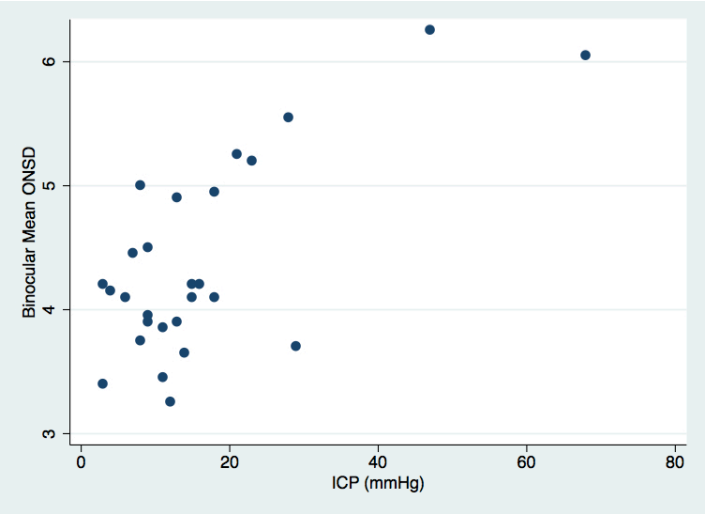


Figure 1. Scattergram of optic nerve sheath diameter (ONSD) versus intracranial pressure (ICP).

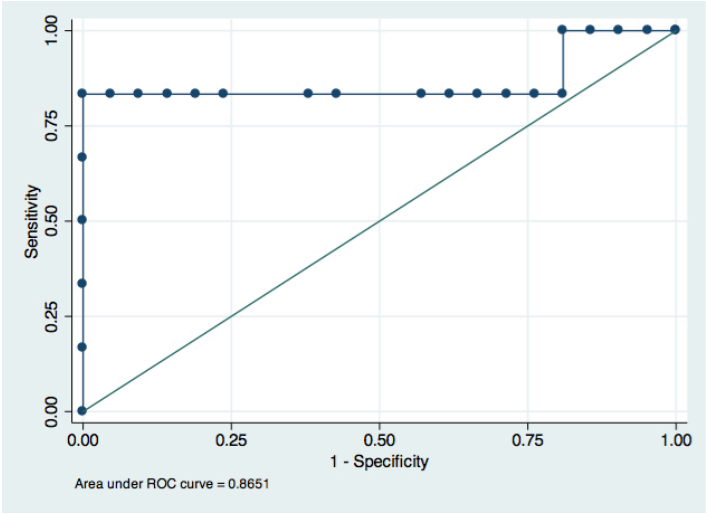


Figure 2. Receiver operator curve for optic nerve sheath diameter as a test for intracranial pressure >20 cm water.

6 (22%) of the study participants. Clear images of the optic nerve could be obtained for all patients.

Although the expansion of ONSD is predictable, the relationship is not purely linear and may be asymptotic.[4,5] The scatter plot produced by our data set supports this hypothesis (Figure 1). The Shapiro-Wilk test for normality demonstrated that neither ONSD (p=0.045) nor ICP (p=0.00002) had normal distributions. Given this finding, we assessed the relationship between ONSD and ICP using a non-parametric Spearman rank correlation coefficient, which produced a rho of 0.408 (p=0.03).

Because ONSD measurements were compared against invasive monitoring, a receiver operating characteristic (ROC) curve for eICP (>20 mmHg) was drawn to establish the optimal cut-off value to maximize sensitivity and specificity (Figure 2). The ROC curve demonstrated an area under the curve of 0.865 (95% CI=0.66 to 0.96). The ONSD cut-off value of ≥5.2 mm yielded the best test characteristics and accurately predicted eICP with a sensitivity of 83.3% (95% CI=35.9% to 99.6%) and specificity of 100% (95% CI=83.9% to 100%). The positive predictive value of ONSD ≥5.2 mm for eICP was 100% (95% CI=48% to 100%) and the negative predictive value of ONSD less than 5.2 mm was 95.5% (95% CI=77.2% to 99.9%).

DISCUSSION

This study assessed the ability of sonographic measurement of ONSD by a single operator to identify the presence of eICP in patients with a wide variety of traumatic, medical, and infectious pathologies. Prior studies have only examined patients with traumatic injuries or hemorrhagic strokes.

We demonstrated that sonographic measurement of ONSD is sensitive (83.3%) and specific (100%) for detecting eICP and that this measurement can be correlated with values obtained by EVD when performed by a single sonographer. The high positive predictive value (100%) of this test suggests that it can be used as an additional tool to evaluate for eICP. Given the inherent risk of false-positive results when non-expert sonographers measure ONSD, we see the utility of ONSD measurements in decision-making, such as expediting CTs and calling for emergency neurology consultations prior to CTs.

We determined an optimal cut-off value of 5.2 mm by using EVD as the basis for comparison. This value was in line with prior studies.[14] However, in the emergency department, where sensitivity is often more important than specificity, a lower ONSD threshold value may be more appropriate and would be a reasonable investigation for a follow-up study.

While the growing body of evidence suggests sonographic ONSD measurement has good predictive value for detecting eICP as measured by EVD, a few studies raise questions about the reproducibility of this exam.[10-14] Quality of image acquisition and sonographic interpretation are both factors that vary with the level of expertise. As of yet, the level of experience needed for competency in performing the ONSD scan is undefined. It is possible that some of the variability in optimal cut-off values are secondary to measurement of artifact or the result of variability in the way the scan and measurements are performed suggesting that this technique should be more adequately described.[11,13]

Sonographic measurement of ONSD would not replace placement of EVD; however, it has the potential to diagnose eICP early when other means are unavailable or contraindicated. The tool is simple, available, cost-effective, and incurs no-harm to the patient, providing an additional non-invasive means of assessing eICP.

LIMITATIONS

Our study is limited by design in that one sonographer

performed all observations with RDMS credentialing and extensive experience, limiting the generalizability of this study. However, this was a pre-hoc design element that was necessary to determine if the test was accurate in expert conditions prior to expanding to non-experts. Additionally, the threshold ONSD value of 5.2 mm reflects the characteristics of this cluster of 27 patients, as derived from an ROC curve. Although this may also limit the generalizability, this value was in line with previous studies. [14]

The study is also limited by its small size and wide 95% confidence interval (35.9% to 99.6%). The results should be validated in larger trials and at multiple centers. However, despite the small size, we believe that this sample represents an adequate spectrum of intracranial processes, including traumatic, hemorrhagic, obstructive, and infectious causes of eICP.

Additionally, we did not compare the performance of ONSD findings with the performance of common neuroimaging modalities (CT or MRI) for diagnosis of eICP, which, along with clinical picture, often serve as the basis for the decision to place invasive monitor. Future studies should consider making this comparison.

CONCLUSION

A non-invasive, inexpensive, rapid bedside assessment to detect eICP could aid in the early diagnosis of eICP, allowing for more rapid intervention. Measurement of ONSD has shown good performance for predicting eICP in this study as well as previous studies. As bedside ultrasound becomes widely available in emergency departments and critical care units, there is value in refining this technique and determining a universal cut-off to aid in this challenging diagnosis.

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

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

REFERENCES

1. Becker D, Miller J, Ward J. The outcome from severe head injury with early diagnosis and intensive management. *J Neurosurg.* 1977; 47:491-502.
2. Miller MT, Pasquale M, Kurek S. Initial head computed tomographic scan characteristics have a linear relationship with initial intracranial pressure after trauma. *J Trauma.* 2004; 56:967–972.
3. Tayal VS, Neulander M, Norton HJ, Foster T, Saunders T, Blaivas M. Emergency department sonographic measurement of optic nerve sheath diameter to detect findings of increased intracranial pressure in adult head injury patients. *Ann Emerg Med*. 2007; 49:508–514.
4. Hansen HC, Helmke K. Validation of the optic nerve sheath response to changing cerebrospinal fluid pressure: ultrasound findings during intrathecal infusion tests. *J Neurosurg.* 1997; 87:34–40.
5. Hansen HC, Lagre W, Krueger O, Helmke K. Dependence of the optic nerve sheath diameter on acutely applied subarachnoidal pressure –an experimental ultrasound study. *Acta Ophthalmologica*. 2011; 89:e528-532.
6. Galetta S, Byrne SF, Smith JL. Echographic correlation of optic nerve sheath size and cerebrospinal fluid pressure. *J Clin Neuroopthalmol*. 1989; 9:79-82.
7. Hansen HC, Helmke K. The subarachnoid space surrounding the optic nerves: An ultrasound study of the optic nerve sheath. *Surg Radiol Anat.* 1996; 18:323-328.
8. Liu D, Kahn M. Measurement and relationship of subarachnoid pressure of the optic nerve to intracranial pressures in fresh cadavers. *Am J Ophthalmol.* 1993; 116:548–556.
9. Soldatos T, Karakitsos D, Chatzimichail K, Papathanasiou M, Gouliamos A, Karabini A. Optic nerve sonography in the diagnostic evaluation of adult brain injury. *Critical Care.* 2008;12:R67.
10. Kimberly HH, Shah S, Marill K, Noble V. Correlation of optic nerve sheath diameter with direct measurement of intracranial pressure. *Acad Emerg Med*. 2008; 15:201-204.
11. Geeraerts T, Merceron S, Benhamou D, Vigué B, Duranteau J. Non-invasive assessment of intracranial pressure using ocular sonography in neurocritical care patients. *Intensive Care Med*. 2008; 34:2062-2067
12. Strumwasser A, Kwan RO, Yeung L, Miraflor E, Ereso A, Castro-Moure F, Patel A, Sadjadi J, Victorino GP. Sonographic optic nerve sheath diameter as an estimate of intracranial pressure in adult trauma. *J Surg Res.* 2011; 170: 265-271.
13. Rajajee V, Vanaman M, Fletcher JJ, Jacobs T. Optic nerve ultrasound for the detection of raised intracranial pressure. *Neurocrit Care*. 2011; 37:1059-1068.
14. Moretti R, Pizzi B, Cassini F and Vivaldi N. Reliability of optic nerve ultrasound for the evaluation of patients with spontaneous intracranial hemorrhage. *Neurocritical Care.* 2009; 11:406-410.
15. Blaivas M, Theodoro D, Sierzenski PR. Elevated intracranial pressure detected by bedside emergency ultrasonography of the optic nerve sheath. *Acad Emerg Med*. 2003; 10:376–381.

Visual Estimation of Bedside Echocardiographic Ejection Fraction by Emergency Physicians

Erden E. Ünlüer, MD*
Arif Karagöz, MD*
Haldun Akoğlu, MD†
Serdar Bayata, MD*

* Izmir Katip Çelebi University Atatürk Research and Training Hospital, Turkey
† Kartal Lütfi Kırdar Research and Training Hospital,Turkey

Supervising Section Editor: Laleh Gharahbaghian, MD, RDMS
Submission history: Submitted February 18, 2013; Revision received August 23, 2013; Accepted September 10, 2013
Electronically published January 6, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.9.16185

Introduction: The objective of this study was to determine whether bedside visual estimates of left ventricular systolic function (LVSF) by emergency physicians (EP) would agree with quantitative measurement of LVSF by the modified Simpson's method (MSM), as recommended by the American Society of Echocardiography.

Methods: After limited focused training, 2 trained EPs performed bedside echocardiography (BECH) procedures s between January and June 2012 to prospectively evaluate patients presenting to the emergency department (ED) with dyspnea. EPs categorized their visually estimated ejection fractions (VEF) as either low or normal. Formal echocardiography were ordered and performed by an experienced cardiologist using the MSM and accepted as the criterion standard. We compared BECH results for each EP using chi-squared testing and performed correlation analysis by Pearson correlation coefficient.

Results: Of the 146 enrolled patients with dyspnea, 13 were excluded and 133 were included in the study. Comparison of EPs vs. cardiologist's estimate of ejection fraction yielded a Pearson's correlation coefficient of 0.77 (R, $p<0.0001$) and 0.78 (R, $p<0.0001$). Calculated biserial correlations using point-biserial correlation and z-scores were 1 (rb, $p<0.0001$) for both EPs. The agreement between EPs and the cardiologist was 0.861 and 0.876, respectively. The sensitivity, specificity, positive predictive value, negative predictive value, and the positive and negative likelihood ratios for each physician were 98.7-98.7%, 86.2-87.9%, 0.902-0.914, 0.980-0.981, 7.153-8.175, 0.015-0.015, respectively.

Conclusion: EPs with a focused training in limited BECH can assess LVSF accurately in the ED by visual estimation. [West J Emerg Med. 2014;15(2):221–226.]

INTRODUCTION

Emergency physicians (EPs) are routinely called on to manage critically ill patients who may present with an indeterminate or changing hemodynamic status. Early in the patient's course, it may be difficult to firmly identify the underlying etiology. At this stage, successful management hinges less on an accurate diagnosis than on a timely determination of the prevailing hemodynamic process.[1] For this issue, the physical examination has been shown to be remarkably unreliable, whereas more invasive assessments of hemodynamics are effort-intensive, costly, and associated with significant morbidity and mortality.[2] Bedside echocardiography (BECH) offers a noninvasive evaluation method of cardiac function. Real-time assessment of the left ventricular ejection fraction (LVEF) offers a window into the causative or compensatory role that the left ventricle (LV) may play in the patient's disease process.[3] This information can direct initial resuscitation efforts, gauge the response to therapy, focus early diagnostic testing, and provide important prognostic data.[4]

To date, evaluation of goal-directed emergency department (ED) BECH has focused on the diagnosis of pericardial effusions, diastolic heart failure, and determination

of central venous pressure.[5-7] A study by Moore et al.[8] found 84% agreement between ED sonographers and cardiologists in determining LVEF in hypotensive patients. To date, most of the studies in the EDs are based on quantitative measurements of LVEF, such as single- or biplane Simpson ejection fraction, fractional shortening of left ventricular walls, or Mitral E point septal separation.[9] These formal measurements have some disadvantages, mainly because of time constraints in the EDs. Compared to formal echocardiographic methods for the evaluation of LVEF, visual estimation (eyeballing) can be done faster and is often easier to perform, even in studies with poor visual quality. Therefore, we aimed to compare visual ejection fraction (VEF) estimation on parasternal long axis view performed in ED with the modified Simpson's method (MSM, biplane method of discs) as recommended by the American Society of Echocardiography (ASE) performed by an experienced cardiologist.[10]

MATERIALS AND METHODS

This was a prospective, cross-sectional cohort study conducted from January to June 2012. The local Ethics Committee approved the study protocol. Written informed consent was obtained from each volunteer prior to his/her sonographic examination. All patients who were registered to ED with the complaint of dyspnea were screened for this study by 2 trained EPs at presentation before other diagnostic tests including echocardiography were performed. Treating physicians were not informed of the results of the BECH to prevent any potential morbidity from the use of a misinterpreted examination. Patients were ineligible if they were intubated, aged <18 years, had elevated cardiac biomarkers, were pregnant, had atrial fibrillation, had a known valvular pathology or surgery, or if ultrasonographic measurements could not be performed because of technical limitations. For sample size estimation, we selected primary outcome of "correlation between EPs and cardiologist.". We estimated to reach at least a correlation of 0.70 with a power of 0.99 and a Type I error of 0.05. Calculated sample size was 11 for a two-tailed correlation. We aimed to enroll at least 10 times this sample size and recruited 133 patients by the end of the study. Therefore, post-hoc power achieved at the end of the study was 1. Each EP had1 year of experience in their speciality and certified on focused abdominal sonography for trauma by Emergency Radiology Association in Turkey. Before the study, these investigators received additional ultrasound training in the area of limited echocardiography. This expanded training consisted of 3 hours of didactic session; a series revwew of normal and abnormal echocardiograms on unrolled 60 ED patients, performed in the presence of an experienced echocardiographer. Specific emphasis was placed on the technique for subjective estimation of LVEF.[11] The EPs categorized ejection fractions (EFs) as normal or low according to movement of the mitral valve during diastole and also kinetics of interventricular septum, apex, and posterior wall of the left ventricle during systole on parasternal long axis view. EF was diagnosed as low if anterior leaflet of mitral valve (ALMV) opening did not occur beyond the midcavitary plane, together with a global hypokinetic nature of the left ventricle (Figure 1). If ALMV movement occurred beyond the midlevel of the left ventricular cavity towards the interventricular septum together with good global kinetics of the left ventricle, the patient was diagnosed as having normal EF (Figure 2). We have used both the kinetics of the left ventricle and ALMV movement together to detect patients with segmental wall motion abnormalities. Of those granting consent, the EPs performed BECH in left lateral decubitus position using an M7R® model ultrasound machine with a 3.6-MHz microconvex transducer (Mindray Bio-medical Electronics Co., Shenzhen, China) and ultrasonographic views were recorded blindly by the EPs. This procedure took less than 2 minutes. An evaluation form was completed by each EP. EF is estimated qualitatively (Low/Normal) by EPs and quantitatively (%) by cardiologist. Formal echocardiography performed with M7R® model ultrasound machine was reported by experienced echocardiographers by MSM, which was ordered after the patient was stabilized within a maximum of 2 hours after the backperform of 2 EPs. Echocardiography results were reported blinded to our study.

Table 1. Past medical history features of the study population.

Feature	Subgroup	n	%
Sex	Male	59	44.4
	Female	74	55.6
History	Congestive heart failure	13	90.2
	Chronic obstructive pulmonary disease	28	78.9
	Diabetes mellitus	26	19.5
	Hypertension	37	27.8
	Cancer	1	0.8
	Chronic renal failure	14	10.5
	Cerebrovascular accident with sequel	8	6.0
Admitted for	Congestive heart failure	66	49.6
	Chronic obstructive pulmonary disease	43	32.3
	Pneumonia	20	15.0
	Cancer	3	2.3
	Chronic renal failure	1	0.8

STATISTICAL ANALYSES

Categorical data are reported as frequency percent and, when appropriate, range. Sensitivity, specificity, positive

Table 2. Comparison of visual ejection fraction (VEF) by both blinded emergency physicians and modified Simpson's method (MSM) by blinded cardiologist.

		Measured EF by cardiologist (MSM)					
		Low EF (MSM)		Normal EF (MSM)		Total	
		Frequency	Percent	Frequency	Percent	Frequency	Percent
Visual estimate of EF by emergency physician 1	Low EF (VEF)	74	55.6	8	6.0	82	61.7
	Normal EF (VEF)	1	0.8	50	37.6	51	38.3
	Total	75	56.4	58	43.6	133	100.0
Visual estimate of EF by emergency physician 2	Low EF (VEF)	74	55.6	7	5.3	81	60.9
	Normal EF (VEF)	1	0.8	51	38.3	52	39.1
	Total	75	56.4	58	43.6	133	100.0

EF, ejection fraction; *MSM,* modified Simpson's method

Table 3. Clinical utility values for detecting a low ejection fraction by both emergency physicians (EP) compared to a cardiologist.

	EP 1			EP 2		
	Value	%95 CI lower limit	%95 CI upper limit	Value	%95 CI lower limit	%95 CI upper limit
Sensitivity (%)	98.7	91.8	99.9	98.7	91.8	99.9
Specificity (%)	86.2	74.1	93.4	87.9	76.1	94.6
Positive predictive value	0.902	0.812	0.954	0.914	0.825	0.962
Negative predictive value	0.980	0.882	0.999	0.981	0.884	0.999
Positive likelihood ratio	7.153	3.757	13.619	8.175	4.079	16.383
Negative likelihood ratio	0.015	0.002	0.109	0.015	0.002	0.107

and negative predictive values, and positive and negative likelihood ratios were calculated for EPs using cardiologist reports as the criterion standard. These are reported with 95% confidence intervals (CI). We used Spearman's correlation coefficients with 95% CIs to express correlations between each EP's dichotomous estimate and dichotomized measured EF% (MFM) values by the cardiologist.

RESULTS

Patient enrollment occurred between January and June 2012. The 146 subjects enrolled in the study gave informed consent. We excluded 13 patients from the final statistical analysis, 6 because of poor image quality, 1 patient because of pregnancy, 2 because of previous thoracic surgery and 4 patients because of atrial fibrillation (Figure 3). Mean age of the study population was 69.76 years (SD: 11.74; 95% CI 67.75, 71.07); 59 (44.4%) were women and 74 (55.6%) were men. Past and present medical history features of the study population are shown in Table 1. All patients were examined by 2 EPs and by a cardiologist blinded to each other. VEF was reported as low or normal by EPs and EF was measured as a percentage by cardiologists. We dichotomized measured EF% (MFM) values as low and normal using a threshold of 55%, and compared this with the VEF of EPs with Spearman's correlation, which resulted with coefficients of 0.87 (95% CI, 0.82-0.90) and 0.88 (95%, 0.84-0.91). All values presented a significant relationship between the visual estimate of EF by EPs and MFM by the cardiologist. Both EPs estimated one patient out of 133 (0.8%) as having a normal EF, but in fact they had low EF according to the cardiologist. On the other hand, 8 (6%) and 7 (5.3%) patients with a normal EF were falsely estimated as having a low EF by each EP, respectively. Intra-class correlation coefficient of the 2 EPs was 0.952 (95% CI: 0.934, 0.966). There were 3 patients in whom the estimates of EPs were different, and in all of these patients measured EFs were above 45%. Comparison of VEF by EPs and measured EF by cardiologists using MSM is shown in Table 2.

EPs' visual estimate of EF was 98% sensitive and 86% specific for detecting a low EF in clinical settings. Clinical utility values for detecting a low EF by EPs compared to the cardiologist are shown in Table 3.

DISCUSSION

This study suggests that ED sonographers, with additional

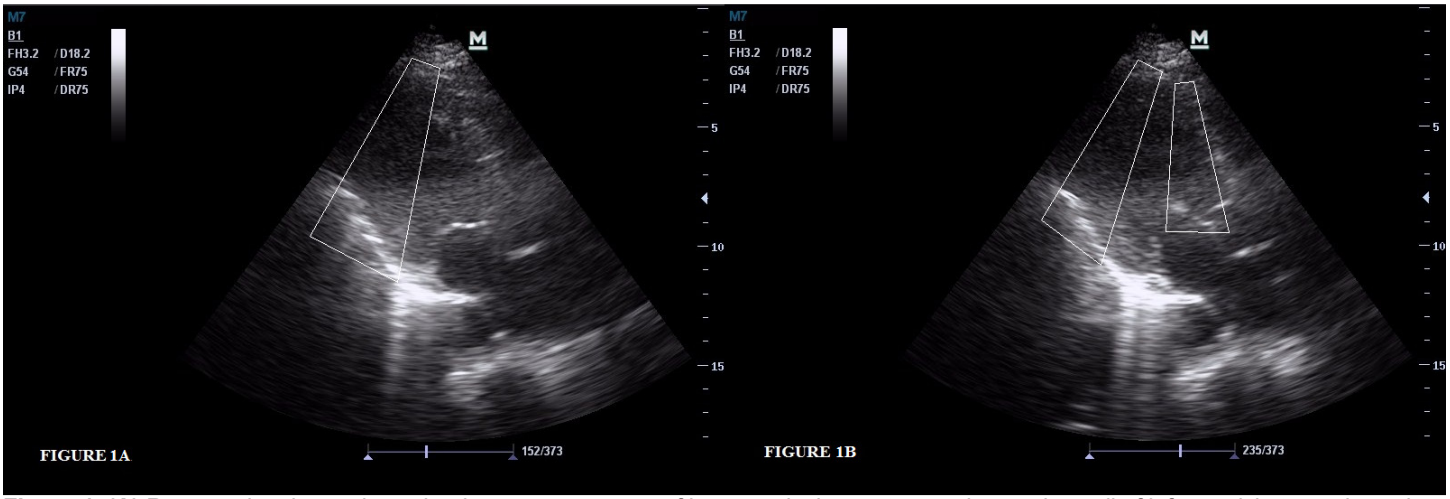


Figure 1. (A) Rectangular shape shows inadequate movement of interventricular septum and posterior wall of left ventricle towards each other during systole. **(B)** Small rectangular shape shows large distance between anterior leaflet of mitral valve and interventricular septum during diastole. Large rectangular shape shows interventricular septum and posterior wall of left ventricle distended from each other.

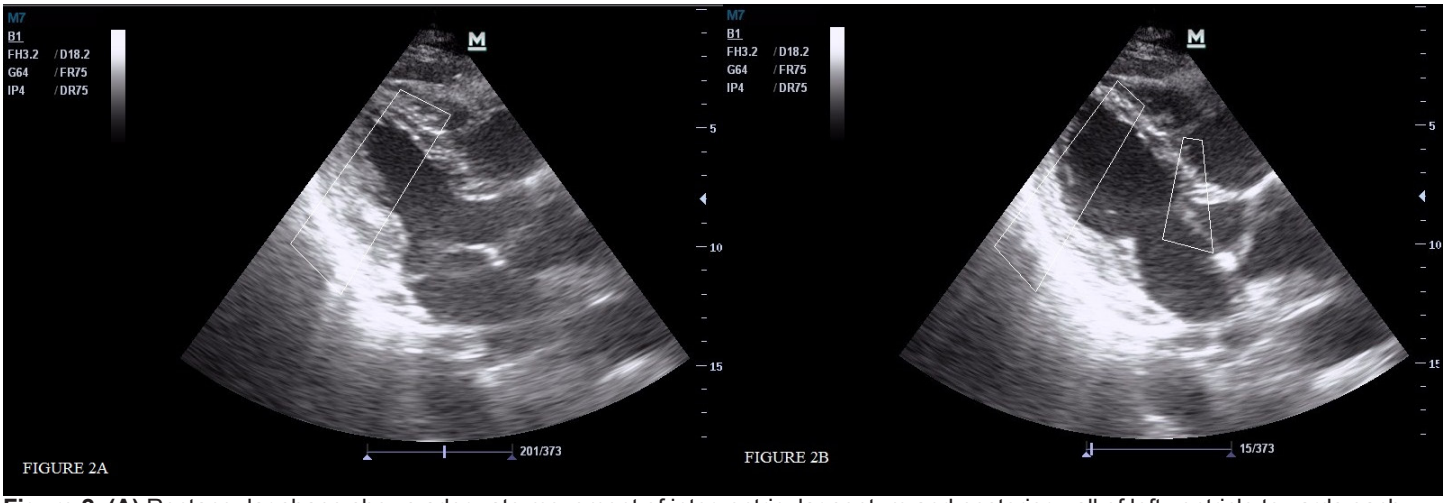


Figure 2. **(A)** Rectangular shape shows adequate movement of interventricular septum and posterior wall of left ventricle towards each other during systole. **(B)** Small rectangular shape shows close distance between anterior leaflet of mitral valve and interventricular septum during diastole. Large rectangular shape shows interventricular septum and posterior wall of left ventricle distended from each other.

focused echocardiography training, can perform limited echocardiography successfully for the purposes of obtaining left ventricular systolic function information. For this study, the highest agreement was found in the low EF group, and there were 8 patients with normal EF who were diagnosed as low EF by EPs. From a clinical perspective, our results indicate that the low EF diagnosed on ED ultrasound would seem to offer the greatest value in patients with dyspnea. If dyspnea is present, the physician may entertain possible diagnoses related to the heart. However, a normal LVEF in dyspnoeic patients may direct the physician towards a number of broader differential diagnoses and management strategies in clinical practice. Randazzo et al.[12] have shown that there was highest agreement between the EPs and formal echocardiography within the normal EF category, in contrast to our results. This discrepancy between results may be due to the patient selection of our study. In Randazzo's study, they included hypotensive patients with shock, and only 5.7% of 115 patients had dyspnea, but we excluded the patients with acute coronary syndromes and included the dyspneic patients in our study. Therefore, we have increased the number of patients with dyspnea. Also, in a study conducted by Weekes et al.[13], it was shown that visual estimation of LVEF compared to fractional shortening of LV yielded a correlation of 0.84, which is similar to our results. Besides these studies, multiple studies published in the cardiology literature agree that visual estimates of ejection fraction are, in some cases, superior to other more elaborate quantitative measures of LVEF.[14,15] Research has shown that a trained EP's estimate of LVEF and central venous pressure using bedside ultrasonography in stable and hypotensive ED patients agrees with those of the cardiologist, comprehensive echocardiograms, and invasive monitors.[12,16] Also, it has been previously shown by Gudmundsson et al. that eyeballing

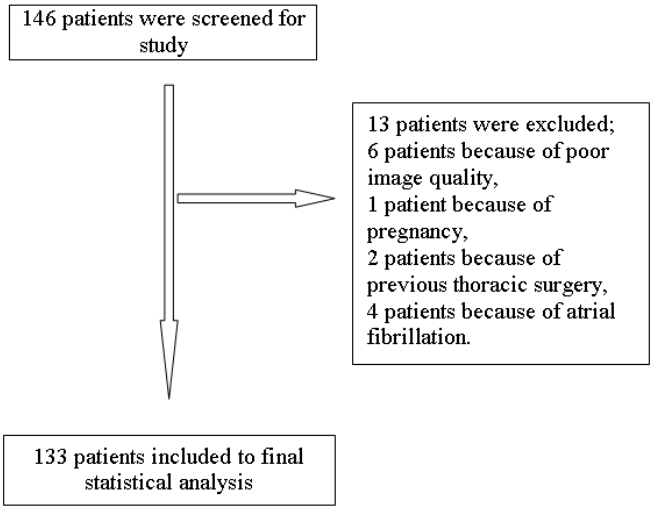


Figure 3. Study flow diagram.

ejection fraction correlated closely with all formal methods, such as Simpson's ejection fraction or fractional shortening.[11] This study was different from ours in that they have screened the patients within a week after an acute myocardial infarction or patients with stable angina and also a single cardiologist performed all examinations and conducted all procedures. We excluded the patients with acute coronary syndrome because the local wall motion abnormalities might have affected the results of visual estimation. Also the MSM and visual estimations for the EFs of the screened patients were done by different physicians to decrease the selection bias of our study group. Our study results are also in concordance with those shown by other cardiology study groups, indicating that visual estimation of LVEF can be used with a high level of accuracy.[15,17,18] Although these studies in cardiology literature showed that visual estimation of LVEF has reasonable agreement with the radionuclide EF in their study group, different EF measurement methods were performed by the same physicians. Because theserepetitive measurements might have been done to enhance their performance on the following measurements we thought that our method for this study reveals more accurate measuerement of visual estimation of LVEF. It was shown in a study by Akinboboye et al. that visual EF is an easy method to learn.[19] Furthermore, a systematic review has shown that the limits of agreement were similar for Simpson ejection fraction, wall motion scoring index, and visual estimation of EF.[20]

Our study was an attempt to correlate visual estimates of LVEF with echocardiographic methods accepted by AHA guidelines. We believe that this is the first study to report serial visual estimations of LVEF in ED patients with dyspnea. Our findings are useful because most EPs do not perform quantitative measurements of left heart systolic function due to time constraints in the ED. High positive and low negative likelihood ratios show that a low EF can be reliably ruled in or out using an EP's visual estimate. The serial visual assessments are quick and easily reproducible.

LIMITATIONS

Potential sources of error in our study included the possibility of patient sampling (convenience sampling). Our study had to be done with consecutive patient selection to decrease bias. Also, two EP investigators enrolled all the patients. To generalize our results, a study would need to be conducted with more than 2 investigators and increased patient enrollment.

CONCLUSION

Visual estimation of ejection fraction correlated significantly with cardiologist reports for the evaluation of left ventricular systolic function. Since it is readily and quickly performed, visual estimation could be used for ED management of patients, instead of the formal quantitative methods, which are more time consuming .

Address for Correspondence: Erden E Ünlüer, Department of Emergency Medicine, Izmir Katip Çelebi University Atatürk Research and Training Hospital. Email: erolerdenun@yahoo.com.

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

REFERENCES

1. Boldt J, Menges T, Wollbrück M, et al. Is continuous cardiac output measurement using thermodilution reliable in critically ill patients? *Crit Care Med.* 1994; 22:1913–8.
2. Baron BJ, Scalae TM. Acute blood loss. *Emerg Med Clin North Am.* 1996; 14:35–55.
3. Nishimura RA, Tajik AJ, Shub C, et al. Role of two-dimensional echocardiography in the prediction of in-hospital complications after acute myocardial infarction. *J Am Coll Cardiol.* 1984; 4:1080–7.
4. Kimura BJ, Bocchicchio M, Willis CL, et al. Screening cardiac ultrasonographic examination in patients with suspected cardiac disease in the emergency department. *Am Heart J.* 2001; 142:324–30.
5. Rubin M. Cardiac ultrasonography. Emerg Med Clin North Am. 1997; 15:745–62.
6. Unlüer EE, Bayata S, Postaci N, et al. Limited bedside echocardiography by emergency physicians for diagnosis of diastolic heart failure. *Emerg Med J.* 2012 Apr;29(4):280-3. Epub 2011 Mar 25.
7. Unlüer EE, Yavaşi O, Akoğlu Het al. Bedside assessment of central

venous pressure by sonographic measurement of right ventricular outflow-tract fractional shortening. *Eur J Emerg Med.* 2011 Dec 15. [Epub ahead of print]

8. Moore CL, Rose GA, Tayal VS, et al. Determination of left ventricular function by emergency physician echocardiography of hypotensive patients. *Acad Emerg Med.* 2002; 9:186–93.
9. Secko MA, Lazar JM, Salciccioli LA, et al. Can junior emergency physicians use E-point septal separation to accurately estimate left ventricular function in acutely dyspneic patients? *Acad Emerg Med.* 2011 ;18(11):1223-6.
10. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification. J Am Soc Echocardiogr. 2005; 18:1440-63.
11. Gudmundsson P, Rydberg E, Winter R, et al. Visually estimated left ventricular ejection fraction by echocardiography is closely correlated with formal quantitative methods. *Int J Cardiol.* 2005; 101(2):209-12.
12. Randazzo MR, Snoey ER, Levitt MA, et al. Accuracy of emergency physician assessment of left ventricular ejection fraction and central venous pressure using echocardiography. *Acad Emerg Med.* 2003; 10(9):973-77.
13. Weekes AJ, Tassone HM, Babcock A, et al. Comparison of serial qualitative and quantitative assessments of caval indeks and left ventricular systolic function during early fluid resuscitation of hypotansive emergency department patients. *Acad Emerg Med.* 2011; 18:912-21.
14. van Royen N, Jaffe CC, Krumholz HM, et al. Comparison and reproducibility of visual echocardiographic and quantitative radionuclide left ventricular ejection fractions. *Am J Cardiol.* 1996; 77:843-50.
15. Amico AF, Lichtenberg GS, Reisner SA, et al. Superiority of visual versus computerized echocardiographic estimation of radionuclide left ventricular ejection fraction. *Am Heart J.* 1989; 118:1259-65.
16. Moore CL, Rose GA, Tayal VS, et al. Determination of left ventricular function by emergency physician echocardiography of hypotansive patients. *Acad Emerg Med.* 2002; 9:186-93.
17. Jensen-Urstad K, Bouvier F, Höjer J, et al. Comparison of different echocardiographic methods with radionuclide imaging for measuring left ventricular ejection fraction during acute myocardial infarction treated by thrombolitic therapy. *Am J Cardiol.* 1998; 81:538-44.
18. van 't Hof AW, Schipper CW, Gerritse JG et al. Comparison of radionuclide angiography with three echocardiographic parameters of left ventricular function in patients after myocardial infarction. *Int J Card Imaging.* 1998; 14:413-8.
19. Akinbobeye O, Sumner J, Gopal A, King D, et al. Visual estimation of ejection fraction by two-dimensional echocardiography: the learning curve. *Clin Cardiol.* 1995; 18:726-9.
20. McGowan JH, Cleland JG. Reliability of reporting left ventricular systolic function by echocardiography: a systematic review of 3 methods. *Am Heart J.* 2003; 146:380-2.

Dysuria in the Emergency Department: Missed Diagnosis of *Chlamydia trachomatis*

Morgan D Wilbanks, MD*
James W Galbraith, MD†
William M Geisler, MD, MPH‡

* University of Alabama at Birmingham, School of Medicine, Birmingham, Alabama
† University of Alabama at Birmingham, Department of Emergency Medicine, Birmingham, Alabama
‡ University of Alabama at Birmingham, Department of Medicine, Birmingham, Alabama

Supervising Section Editor: Robert Derlet, MD
Submission history: Submitted July 15, 2013; Revision received November 26, 2013; Accepted December 5, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.18989

Introduction*:* The clinical presentation of genital *Chlamydia trachomatis* infection (chlamydia) in women is often indistinguishable from a urinary tract infection. While merited in the setting of dysuria, emergency department (ED) clinicians do not routinely test for chlamydia in women. The primary aim of our study was to evaluate the frequency of chlamydia testing among women presenting to the ED with dysuria.

Methods*:* We conducted a retrospective chart review of women 19-25 years of age presenting with dysuria to an urban ED and who had been coded with urinary tract infection (UTI) as their primary diagnosis (ICD-9 599.0) from October 2005 to March 2011. We excluded women who were pregnant, had underlying anatomical or neurological urinary system pathology, had continuation of symptoms from UTI or a sexually transmitted infection (STI) diagnosed elsewhere, or were already on antibiotics for a UTI or STI. We identified the rates of sexual history screening, pelvic examination and chlamydia assay testing and evaluated predictors using univariate and multivariate analyses.

Results: Of 280 women with dysuria and a UTI diagnosis, 17% were asked about their sexual history, with 94% reporting recent sexual activity. Pelvic examination was performed in 23%. We were unable to determine the overall chlamydia prevalence as only 20% of women in the cohort were tested. Among the 20% of women tested for chlamydia infection, 21% tested positive. Only 42% of chlamydia-positive women were prescribed treatment effective for chlamydia (azithromycin or doxycycline) at their visit; the remaining were prescribed UTI treatment not effective against chlamydia. Predictors of sexual history screening included vaginal bleeding (OR 5.4, 95% CI=1.5 to 19.6) and discharge (OR 2.8, 95% CI=1.1 to 6.9). Predictors of a pelvic examination being performed included having a complaint of vaginal discharge (OR 11.8, 95% CI=4.2 to 32.9), a sexual history performed (OR 2.5, 95% CI=1.1 to 5.8), abdominal pain (OR 2.2, 95% CI=1.1 to 4.4), or pelvic pain (OR 15.3, 95% CI=2.5 to 92.2); a complaint of urinary frequency was associated with a pelvic examination not being performed (OR 0.34, 95% CI=0.13 to 0.86).

Conclusion: Sexual histories, pelvic examinations, and chlamydia testing were not performed in the majority of women presenting with dysuria and diagnosed with UTI in the ED. The performance of a sexual history along with the availability of self-administered vaginal swab and first-void urine-based chlamydia tests may increase identification of chlamydia infection in women with dysuria. [West J Emerg Med. 2014;15(2):227–230.]

INTRODUCTION

Chlamydia trachomatis infection (chlamydia) is the most prevalent bacterial sexually transmitted infection (STI) in the United States, with over 1.4 million cases reported in 2011.[1] While the majority of cases are asymptomatic, chlamydia can present with dysuria (i.e., "acute urethral syndrome")

and resemble a urinary tract infection (UTI). This poses a diagnostic dilemma for providers as UTI and chlamydia have different clinical courses and treatments. Untreated chlamydia can cause serious complications, including pelvic inflammatory disease, infertility, and ectopic pregnancy. For women with a possible chlamydia-associated syndrome, ascertaining demographics and a sexual history can be used to stratify risk, as chlamydia rates are disproportionately higher in adolescents/young adults, African Americans, and those with new or multiple sexual partners.[1,2] Furthermore, high STI rates have been reported in women evaluated in an urban emergency department (ED) and diagnosed with UTI.[3]

The Centers for Disease Control and Prevention (CDC) recommends that sexually active women ≤25 years of age and older women at risk receive annual chlamydia screening. Approximately half of eligible women receive screening,[4] and symptomatic chlamydia infections remain under-recognized in ED settings.[3] Our study examined whether women presenting to a large urban ED with dysuria and diagnosed with a UTI had a sexual history performed, underwent pelvic examination, or received chlamydia testing, as well as which factors predicted these outcomes.

METHODS

Study Design

We retrospectively reviewed electronic medical records (EMRs) of female ED patients with a primary diagnosis of UTI by ICD-9 code and a chief complaint of dysuria. The study was approved by the University of Alabama at Birmingham Institutional Review Board.

Table 1. Patient characteristics of women presenting with dysuria

Variable	Study Sample (n=280)
Age, year (mean)	21.6
Race	n (%)
Black	197 (70)
White	68 (24)
Hispanic	9 (3.2)
Asian	2 (<1)
Unknown	4 (1.4)
Symptoms	
Dysuria	280 (100)
Abdominal pain	90 (32)
Urinary frequency	81 (29)
Back pain	58 (21)
Vaginal discharge	28 (10)
Vaginal bleeding	11 (3.9)
Vaginal odor	4 (1.4)
Genital ulcer	2 (<1)
Prior chlamydia (%)	23 (8.2)

Study Setting and Population

We evaluated females 19-25 years of age presenting to an urban ED (>75,000 annual adult visits) from October 2005 to March 2011. The upper age limit of 25 was chosen as this is the age cutoff for CDC-recommended annual chlamydia screening in sexually active women. We included females with primary ICD-9 code for UTI (ICD-9 599.0) and a chief complaint of dysuria through review of the ED physician's EMR documentation for the visit. No secondary diagnoses by ICD-9 were excluded. We excluded patients on the basis of pregnancy, current or recent treatment for UTI/STI, continuation of symptoms from UTI/STI diagnosed or treated elsewhere, or underlying anatomical or neurological urinary system pathology.

Study Protocol

We compiled records based on ICD-9 coding and inclusion and exclusion criteria. We reviewed the EMR for eligible subjects and collected the following data onto standardized collection forms: demographics, symptoms, sexual history, prior STI, results of pelvic examination and diagnostic testing, and treatment.

Data Analysis

We evaluated predictors of having a sexual history performed, receiving a pelvic examination, and receiving chlamydia testing initially by univariate analyses using chi-squared or Fisher's exact test. We then evaluated variables significant at the α=0.10 level in a multivariable logistic regression model. Analyses were performed using Stata (Stata Corp. Release 8.0, College Station, TX).

RESULTS

Characteristics of 280 eligible women evaluated are shown in Table 1. The majority were African American. In addition to dysuria, 29% reported urinary frequency. Abdominal pain was reported frequently (32%), while genital symptoms were reported less frequently, with complaints of vaginal discharge being present in 10%. Prior chlamydia was documented in 8%.

Only 47 (17%) women were asked questions pertaining to a sexual history, with 44 (94%) reporting recent activity. A sexual history was performed more often in women reporting vaginal bleeding (55% versus 15%, p=0.004), pelvic pain (45% versus 16%, p=0.023), or vaginal discharge (39% versus 14%, p=0.001), and less often in those reporting urinary frequency (10% versus 20%, p=0.048). However, a sexual history was not performed in the majority of women reporting vaginal discharge (60%), pelvic pain (54%), or vaginal bleeding (45%). Having a sexual history performed was not associated with

race or other symptoms. On multivariate analysis, having a sexual history performed remain predicted by the presence of vaginal bleeding symptoms (OR 5.4, 95% CI=1.5 to 19.6) or vaginal discharge (OR 2.8, 95% CI=1.1 to 6.9).

Pelvic examination was performed in 23%, more often in women with pelvic pain (82% versus 20%, p<0.001), vaginal discharge (79% versus 16%, p<0.001), vaginal bleeding (64% versus 21%, p=0.003), a sexual history documented (35% versus 12%, p<0.001), or abdominal pain (33% versus 17%, p=0.003), and less often in women with urinary frequency (9% versus 28%, p<0.001). This was not associated with race or other symptoms. On multivariate analysis, having a pelvic examination performed remained predicted by a complaint of vaginal discharge (OR 11.8, 95% CI=4.2 to 32.9) or having a sexual history performed (OR 2.5, 95% CI=1.1 to 5.8), pelvic pain (OR 15.3, 95% CI=2.5 to 92.2) or abdominal pain (OR 2.2, 95% CI = 1.1 to 4.4); a complaint of urinary frequency was associated with a pelvic examination less often being performed (OR 0.34, 95% CI=0.13 to 0.86).

Chlamydia testing was performed in 56 (20%) women (71% of women receiving a pelvic examination), more often in women with vaginal discharge (75% versus 14%, p<0.001), pelvic pain (72% versus 18%, p<0.001), having a sexual history performed (45% versus 15%, p<0.001), vaginal bleeding (45% versus 19%, p=0.047), or abdominal pain (32% versus 14%, p<0.001), and less often in women with urinary frequency (7% vs. 25%, p<0.001). Having chlamydia testing performed was not associated with race or other symptoms. On multivariate analysis, having chlamydia testing performed remain predicted by having a complaint of vaginal discharge (OR 11.3, 95% CI=4.2 to 30.1) or a sexual history performed (OR 3.4, 95% CI=1.5 to 7.9), pelvic pain (OR 8.4, 95% CI=1.6 to 44.1) or abdominal pain (OR 2.9, 95% CI=1.4 to 6.1); a complaint of urinary frequency was associated with chlamydia testing not being performed (OR 0.34, 95% CI=0.13 to 0.94).

Of 56 women tested for chlamydia, 12 (21%) tested positive. Only 7 of 23 (30%) women with prior chlamydia were tested, and 3 of these 7 (42%) tested positive. Of women with dysuria as their only symptom, 67% had a negative urinary nitrite; however, only 7% received chlamydia testing. Of 12 women with a positive chlamydia test, 83% had a negative urinary nitrite. Only 5 (42%) of chlamydia-positive women were prescribed CDC-recommended chlamydia treatment (azithromycin or doxycycline) at their visit; the remaining were prescribed UTI treatment not effective against chlamydia (trimethoprim-sulfamethoxazole or ciprofloxacin).

DISCUSSION

We found women 19-25 years of age presenting to an ED with dysuria and receiving a UTI diagnosis infrequently had sexual history, pelvic examination, or chlamydia testing performed (all ≤20%), confirming that chlamydia is an under-recognized etiology of dysuria seen in EDs. Although most chlamydia-infected women are asymptomatic, dysuria in these women (acute urethral syndrome) is well described.[5] We found that 21% of our study population receiving a chlamydia test were positive, supporting that this is a population for which testing is appropriate. However, since only 20% received chlamydia testing, there were likely missed opportunities for chlamydia diagnosis and treatment, leaving infected patients at risk for chlamydia complications. Most UTI treatment regimens are not effective for chlamydia.

We found that select genital symptoms were associated with having a sexual history performed and receiving a pelvic examination and chlamydia testing. Most genital symptoms are nonspecific for chlamydia and it is likely that the chlamydia positivity rate would have been high in women without genital symptoms. However, most women without genital symptoms did not receive testing. Though we do not suggest every woman presenting to an ED with dysuria undergo chlamydia testing, asking a few additional questions may allow a provider to stratify chlamydia risk and the need for testing. Females in this age range who have had recent unprotected intercourse, multiple sexual partners, or prior chlamydia may be at higher risk and are appropriate for chlamydia testing. Because the sensitivity of urine nitrite in diagnosing bacterial UTI varies widely in the literature,[6] a negative nitrite should not necessarily exclude UTI from the differential; rather, negative nitrites in the setting of positive leukocyte esterase may prompt a provider to also consider chlamydia testing in sexually-active women.

The availability of highly sensitive nucleic acid amplification tests (NAAT) for *C. trachomatis* that can be performed on a self-administered vaginal swab (SAVS) or first-void urine samples should expedite screening in the ED. In busy ED settings, the option of performing chlamydia testing on a SAVS or first-void urine rather than having to perform pelvic examinations (when not necessary for other gynecologic concerns) might be preferred by providers and patients. Because NAAT has a higher sensitivity on SAVS samples compared to first-void urine samples (i.e., detects more chlamydia infections in SAVS samples), the Centers for Disease Control and Prevention currently recommends SAVS as the optimal specimen type for chlamydia screening in women.[8] In addition, first-void urine samples are difficult to obtain in the ED due to the preference for mid-stream urine collection for UTI evaluation at the initiation of the visit. For this reason, a SAVS performed by the patient would be the best method of sample collection.

Since access to healthcare could prevent some women from receiving routine chlamydia screening by a primary care provider, an ED visit for urinary symptoms might be their only opportunity to have chlamydia diagnosed and treated.

LIMITATIONS

Our study took place in an urban ED that treats a large number of high-risk patients and our results may not be

applicable to other populations. The retrospective study design may have limited accuracy and completeness of data extracted from ED providers' documentation, and there could be bias in types of information recorded by providers. It is possible that some sexual histories obtained from ED providers were not recorded in the EMR. Follow-up data on women testing chlamydia-positive were not available and attempts to contact individuals in this study regarding future STI testing and results were not performed.

CONCLUSION

Women presenting to an ED with dysuria and diagnosed with a UTI did not routinely have a sexual history performed or undergo pelvic examination or chlamydia testing. When chlamydia testing was performed, the positivity rate was high. The majority of chlamydia-infected women received UTI treatment not effective for chlamydia. The performance of a sexual history along with the availability of self-administered vaginal swab and first-void urine-based chlamydia tests may increase identification of chlamydia infection in women with dysuria.

ACKNOWLEDGEMENTS

We thank Henry Wang, MD, and Joel Rodgers, MA, for their contributions.

Address for Correspondence: James W Galbraith, MD, University of Alabama at Birmingham, Department of Emergency Medicine, 619 19th Street South, JTN 266, Birmingham, AL 35249. Email: jgalbraith@uabmc.edu.

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

REFERENCES

1. Centers for Disease Control and Prevention. *Sexually Transmitted Disease Surveillance 2011*. Atlanta: U.S. Department of Health and Human Services ; 2012. Available at: http://www.cdc.gov/std/stats11/Surv2011.pdf. Accessed December 19, 2012.
2. Eggleston E, Rogers SM, Turner CF, et al. *Chlamydia trachomatis* infection among 15-35 year-olds in Baltimore, MD, USA. *Sex Transm Dis*. 2012;38(8):743-749.
3. Berg E, Benson DM, Haraszkiewcz P, et al. High prevalence of sexually transmitted diseases in women with urinary infections. *Acad Emerg Med*. 1996;3(11):1030-1034.
4. National Committee for Quality Assurance. The state of healthcare quality 2011. Washington (DC): National Committee for Quality Assurance; 2011. 77-78.
5. Stamm WE, Wagner KF, Amsel R, et al. Causes of the acute urethral syndrome in women. *New Engl J Med*. 1980;303(8):409-415
6. St John A, Boyd JC, Lowes AJ, et al. The use of urinary dipstick to exclude urinary tract infection. *Am J Clin Pathol*. 2006;126(3):428-436.
7. Yealy DM, Greene TJ, Hobbs GD. Underrecognition of cervical *Neisseria gonorrhoeae* and *Chlamydia trachomatis* infections in the emergency department. *Acad Emerg Med*. 1997;4(10):962-967.
8. Association of Public Health Laboratories and the Centers for Disease Control and Prevention. Laboratory Diagnostic Testing for *Chlamydia trachomatis* and *Neisseria gonorrhea*. Atlanta; 2009. Available at: http://www.aphl.org/aphlprograms/infectious/std/Documents/ID_2009Jan_CTGCLab-Guidelines-Meeting-Report.pdf. Accessed October 9, 2013.

Tackling the Global Challenge: Humanitarian Catastrophes

Kenneth V. Iserson, MD, MBA University of Arizona, Department of Emergency Medicine, Tuscon, Arizona

Supervising Section Editor: David T. Williams, MD
Submission history: Submitted October 24, 2013; Revision received November 24, 2013; Accepted December 5, 2013
Electronically published March 18, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.12.20125

"Humanitarian catastrophes," conflicts and calamities generating both widespread human suffering and destructive events, require a wide range of emergency resources. This paper answers a number of questions that humanitarian catastrophes generate: Why and how do the most-developed countries—those with the resources, capabilities, and willingness to help—intervene in specific types of disasters? What ethical and legal guidelines shape our interventions? How well do we achieve our goals? It then suggests a number of changes to improve humanitarian responses, including better NGO-government cooperation, increased research on the best disaster response methods, clarification of the criteria and roles for humanitarian (military) interventions, and development of post-2015 Millennium Development Goals with more accurate progress measures. [West J Emerg Med. 2014;15(2):231–240.]

INTRODUCTION

The phrase "humanitarian catastrophes" describes conflicts and calamities that generate both widespread human suffering and destructive events that require a wide range of emergency resources. These catastrophes, which occur with intimidating frequency, present both as acute crises and as chronic or cyclical disasters, each with different etiologies and responses. In both acute and chronic crises, external assistance generally originates from the most-developed countries—those with the resources, capabilities, and willingness to help. This paper describes what various acute and chronic crises entail and answers the following questions:

1. Why do we intervene in specific types of disasters?
2. How do we intervene?
3. What ethical guidelines shape our interventions?
4. What legal guidelines shape our interventions?
5. How well do we achieve our goals?

Acute Crises

Acute crises cause unstable or dangerous conditions suddenly and, for the most part, unexpectedly, which demand timely intervention to alleviate the situation.[1] These include cataclysmic natural events and potential pandemics, as well as human-caused horrors such as wars and genocide.

Natural Disasters

Typically, when we imagine a humanitarian catastrophe, our minds first picture the devastation that natural disasters produce. Thousands of people die and millions of lives are devastated annually as the result of hurricanes (typhoons), tsunamis, wildfires, tornados, earthquakes, floods, volcanic eruptions, landslides/avalanches, heat waves, and blizzards.

After natural disasters, personal emotion and media coverage play a large role in whether and how much assistance is provided. As social beings, the suddenness of natural disasters triggers profound feelings of empathy, which may prompt impulsive actions, such as monetary donations. Our response to natural disasters strongly relates to how extensive and for how long the media cover the story. The media play to people's skewed risk perception, reacting more to "death clusters" in sudden catastrophes than to scattered deaths from more routine causes, such as occur with cancer attributable to cigarette smoking (equivalent to three 747 jumbo jet crashes daily)[2] or traffic deaths (equivalent to a daily 100-passenger regional jet crash).[3,4,] Eventually, even after the most devastating natural disasters, media and donor fatigue set in and the response dwindles, as occurred after the 2010 earthquake in Haiti.

Current and Potential Pandemics

An epidemic exists when there are "more cases of disease than expected in a given area or among a specific group of people over a particular period of time."[5] An infectious disease becomes pandemic when it affects large numbers of people across a very wide area, such as on multiple continents.[6] (Endemic diseases with a stable prevalence are not pandemics.) Pandemics may stem from natural causes

or from the accidental or intentional release of biological warfare agents.[7,8]

Human immunodeficiency virus (HIV)/aquired immunodeficiency syndrome (AIDS) is the only current pandemic. However, unless they are recognized and stopped early, future pandemics will inevitably result from novel influenza strains, as they have over the past centuries. They may also stem from viral hemorrhagic fevers (e.g., Ebola, Lassa, Hanta, Bas-Congo Hemorrhagic Fever); antibiotic-resistant bacteria (e.g., methicillin-resistant *Staphylococcus aureus* [MRSA], enterococcus); extremely drug-resistant tuberculosis; other viral diseases (e.g., severe acute respiratory syndrome, 2012 SARS-like coronavirus); prion diseases (e.g., Creutzfeldt-Jakob Disease); or diseases caused by as-yet-unrecognized agents.

Self-preservation motivates interventions to prevent pandemics. These diseases pose the risk of worldwide devastation, affecting all populations regardless of their geography, economic status, or ethnicity. This awareness has resulted in greater international cooperation than any other disaster mitigation effort. Despite occasional failures, as when countries hide evidence of potentially pandemic diseases, the international community has repeatedly seen the benefits of such cooperation

War and Genocide

War is an organized, intentional and widespread armed conflict between political communities. A form of political violence, war is intended, as Prussian military general and theoretician Carl von Clausewitz wrote, "to compel our enemy to do our will."[9,10] This form of politically motivated violence has occurred in every society's history. Its characteristics include extreme aggression, social disruption, and usually high mortality.[11]

Genocide, often closely linked to war, is an attempt "to destroy, in whole or in part, a national, ethnical, racial or religious group."[12] Genocidal acts include "killing members of the group; [and] causing serious bodily or mental harm to members of the group."[12] They also include deliberately inflicting "conditions of life, calculated to bring about [a group's] physical destruction in whole or in part; imposing measures intended to prevent births within the group; [and] forcibly transferring children of the group to another group."[12]

Decisions to use military interventions or threats to intervene to stop or prevent wars or genocide would seem to depend primarily on treaty obligations. As with natural disasters, however, intervention may also depend on the slant and intensity with which media cover the violence and a potential intervening nation's emotional investment and perceived ethnic ties to those involved. More pragmatically, the chance of intervention occurring also may depend on national and international political gamesmanship, the economic hardship due to supplying troops, and whether appropriate types and numbers of troops are available.

CHRONIC CRISES

What are they?

Chronic or cyclical disasters occur over decades and generations with little change. For large portions of the world's populations, these conditions produce water and food insecurity; debilitating and life-threatening endemic diseases and the subset of neglected tropical diseases; and displacement from countries or homes, making them refugees or internally displaced persons (IDPs). We intervene in all these situations for similar reasons.

Water Insecurity

A water crisis occurs when the available potable, unpolluted water within a region is less than needed. About 5.6 billion people (2011), or 80% of the world's population live in areas with threats to water security.[13] There is still inadequate access to safe drinking water for about 884 million people and for sanitation and waste disposal for 2.5 billion people—mostly in poor and conflict prone countries.[14,15] Diarrhea due to contaminated water is "the biggest killer of children in sub-Saharan Africa—killing more than AIDS, tuberculosis (TB) and malaria combined."[16]

Food Insecurity

Food insecurity means that, over time, people have insufficient quantities of food on a consistent basis (food availability) and insufficient resources to obtain appropriate foods for a nutritious diet (food access), and use their food optimally with adequate water and sanitation (food use).[17,18] "Undernourished" or "chronic hunger" means "food intake that is insufficient to meet dietary energy requirements continuously.[19]

The United Nations Food and Agriculture Organization (FAO) estimates that 33 of 193 countries have critically inadequate food supplies. Worldwide, at the beginning of 2009, around 963 million people were undernourished or chronically hungry due to extreme poverty, while up to 2 billion people lacked food security intermittently due to varying degrees of poverty. Most undernourished people live in developing countries: about 65% live in India, China, Democratic Republic of Congo, Bangladesh, Indonesia, Pakistan, or Ethiopia. The highest proportion of undernourished people is in sub-Saharan Africa, where 1 in 3 people are considered chronically hungry.[20]

Debilitating/Life-Threatening Endemic Diseases

Endemic infectious diseases are those that remain prevalent in a particular locality, region, or population without the need for external inputs. The best-known examples are TB and malaria, both of which run rampant in the most-impoverished countries. Both diseases now receive worldwide attention, with extremely drug-resistant TB potentially becoming pandemic.

The world community has, for the most part, ignored many lesser known, "neglected tropical diseases" (NTDs),

because climate, vectors and reservoir hosts restrict them primarily to tropical regions.[21] Even though NTDs cause millions of years' worth of disability (Table 1), the world has largely ignored the World Health Organization (WHO)-recommended strategic interventions that could control, prevent and even eliminate various NTDs. These measures include preventive chemotherapy; intensified case management; vector control; provision of safe water, sanitation and hygiene; and veterinary public health. [21]

Refugees/Internally Displaced Persons

A refugee is a person who, "owing to a well-founded fear of being persecuted for reasons of race, religion, nationality, membership in a particular social group, or political opinion, is outside the country of his nationality and is unable to or, owing to such fear, is unwilling to avail himself of the protection of that country."[22] A 1967 United Nations (UN) Protocol expanded this definition to include persons who have fled war or other violence in their home country.[23]

Displaced persons generally refers to refugees who have left their home countries because of war rather than a fear of persecution, or have been forced to migrate within their home countries (internally displaced persons ([IDPs]). The Convention Governing the Specific Aspects of Refugee Problems in Africa also includes people who have left their countries of origin due to acts of external aggression, occupation, domination by foreign powers or serious disturbances of public order.[24]

Table 1. Global Neglected Tropical Disease disability-adjusted life years (DALYs),* 2004.[61]

Disease	DALY
Lymphatic filariasis	5,941,000
Leishmaniasis	1,974,000
Schistosomiasis	1,707,000
Human African trypanosomiasis	1,673,000
Trachoma	1,334,000
Hookword	1,092,000
Trichuriasis	1,012,000
Ascariasis	1,851,000
Dengue	370,000
Chagas Disease	430,000
Onchocerciasis	389,000
Leprosy	194,000

*DALYs represent the years of life lost (YLLs) plus the years lived with disability (YLDs). YLLs are the age-sex-country-time-specific estimates of mortality by cause, with death calculated using standardized rates at each age. YLDs are calculated as the prevalence of disabling sequelae, by age, sex, and cause.[62]

WHY DO WE INTERVENE?

Interventions in chronic crises are both altruistic and highly political, and they generally focus on the UN's 8 Millennium Development Goals (MDGs).[25] All 193 UN-member states and more than 23 international organizations have agreed to work toward achieving the MDGs by 2015. These goals are:

1. Eradicate extreme poverty and hunger.
2. Achieve universal primary education.
3. Promote gender equality and empower women.
4. Reduce child mortality rates.
5. Improve maternal health.
6. Combat HIV/AIDS, malaria and other diseases.
7. Ensure environmental sustainability.
8. Develop a global partnership for development.

Those MDGs directly related to what have been categorized as chronic humanitarian crises include #1, food insecurity; #6, endemic diseases—as well as to HIV/AIDS, the one current [acute] pandemic disease; and #7, which includes water insecurity.

The MDGs originated with the Organisation [sic] for Economic Co-operation and Development (OECD), a group of the most-developed democratic countries devoted to a free-market economy. Primarily tasked with stimulating economic progress and world trade, the OECD formulated a strategic development framework for the entire world based on resolutions of international conferences and summit meetings. Further refining the resolutions at international meetings, the UN General Assembly approved most of their recommendations as MDGs in 2000.[26]

While world pressure and an obligation to fulfill international promises have played a role in trying to meet the MDG objectives, the major impetus has been the availability of a large amount of money. Generally, non-governmental organizations (NGOs) are the "boots on the ground" that can make progress toward meeting the MDG objectives, and most NGOs follow the money.

The UN, for example, now provides funds through its various arms to advance these goals, as do individual governments. Not only has specific aid been supplied by various countries, but affected countries have also been able to make more internal funding available. With backing from the G8 finance ministers in 2005, most of the world's major financial giants (e.g., World Bank, International Monetary Fund, and African Development Bank) cancelled up to $55 billion in debt owed by "heavily indebted countries" so that they could put the money into programs aligned with the MDGs.

ACUTE AND CHRONIC CRISES INTERVENTION: HOW WE INTERVENE

Ideal Response

Crisis intervention should have the same goals in acute and chronic situations, although they will often differ in their speed, scope, and techniques. Responders, whether they be

from NGOs or governments, ideally assist local governments and agencies by providing short- and long-term planning for and an initial supply of water, sanitation, food and nutrition, agriculture, shelter, healthcare, government infrastructure, economy, and safety/security. While accomplishing this mission, responding agencies have a duty to ensure both quality, maintaining a minimum standard of service, and accountability, being answerable to those who receive the assistance and those who finance it.

Fund Solicitation

Soliciting funds constitutes an inevitable part of NGOs' response to both acute and chronic humanitarian catastrophes. These organizations often raise funds using pictures of starving babies and bloated corpses coupled with wildly overblown statistics. As Charny wrote, "Repeated use of these images has dulled the public to real suffering, while encouraging the public to view people as unable to solve their own problems."[27] The public now recognizes these appeals as sordid hype that results in donor fatigue, if not disgust. Unfortunately, it then becomes easier to ignore reports that accurately depict dire situations in subsequent disasters. While ethical guidelines exist for fundraising, even the most reputable organizations still use these unsavory methods, and few regulatory agencies spend much time policing the plethora of fraudulent disaster-related fundraising.[28,29]

Non-Monetary Resources

Many items provided to affected areas from both the private and public sectors are often inappropriate for the circumstances. Such items include outdated or unneeded medications, used clothing that local people will not wear, and unsuitable tools or shelters. Although they may have the best motivations, many relief workers lack even the most rudimentary knowledge about the country in which they are working, including the basic language, cultural, political, and technical skills required in that situation. In addition, they often have no logistical support, and thus must consume resources that the local population desperately needs.[30]

Potential Epidemics/Pandemics

According to WHO's Global Outbreak Alert and Response Network (GOARN), "Today, there is growing recognition that an outbreak anywhere can potentially represent an emergency of international public health concern."[31] Response to these threats is coordinated through GOARN and the U.S. Centers for Disease Control and Prevention's Global Disease Detection and Emergency Response (GDDER). GOARN combats the international spread of infectious outbreaks and helps build capacity to prepare for epidemics from known and emerging diseases through a collaboration of existing international institutions and networks.[32] GDDER uses non-traditional surveillance methods to provide early warning about international disease threats and provides experts for a rapid worldwide response.[33]

Military Force

Legally termed "humanitarian interventions," military force may include a variety of actions, often designated with these murky and overlapping terms: peacemaking (support mediation and military actions to achieve a cease-fire), peacekeeping (maintain, implement and monitor agreements to prevent further hostilities), and peace-building (help with final implementation of agreements and rebuilding the society). The lack of clarity in defining these roles may make all sides in the conflict mistrust these multinational forces.

In addition, these roles may be compromised, since "military culture assumes a certain independence and superiority of mission over that of longer established disaster management/humanitarian assistance efforts, [that may cause] displacement, marginalization, and increased tensions with local partners." [34]

WHAT ETHICAL AND LEGAL GUIDELINES SHAPE OUR INTERVENTIONS?

The underlying ethos of disaster/humanitarian relief activities is to do well, rather than simply to do good. In other words, because these situations are so complex and often involve so many organizations with different resources, expertise and agendas, beneficial motivations are insufficient. Instead, the aim is to achieve constructive, long-lasting solutions.

Ideally, there would be ethical and legal standards to guide various aspects of our response to humanitarian catastrophes, including when and why we should intervene and how to act when we do. Unfortunately, few unambiguous standards exist.

The most problematic area concerns the criteria for *when* and *why* we intervene, whether it is a civilian or military or an acute or chronic disaster. Disaster interventions optimally protect people and safeguard livestock, shelter and other essential property. Thus, the ideal criteria for intervention are needs driven: interventions should occur when an infusion of resources will prevent human suffering, or at least help suffering populations regain stability. Yet, experience shows that media coverage, political expediency, and non-governmental humanitarian agencies' (NGHA) pre-set agendas and available resources actually guide our interventions.

How we act once we decide to respond has more easily identifiable guidelines, although these, too, can be vague.

ETHICS OF INTERVENTION

Ethical codes help guide civilian organizations and their personnel when responding to humanitarian catastrophes. These codes include the *International Red Cross (ICRC)/Red Crescent Code*, for all types of disasters (Table 2); the Sphere Project's *Humanitarian Charter*, primarily for conflict-related

Table 2. Principles of conduct for the International Red Cross and Red Crescent movement and NGOs in disaster response.[63]

1. The humanitarian imperative comes first. This has been defined as "action [that] should be taken to prevent or alleviate human suffering arising out of disaster or conflict, and that nothing should override this principle."[64]
2. Aid is given regardless of the race, creed or nationality of the recipients and without adverse distinction of any kind. Aid priorities are calculated on the basis of need alone.
3. Aid will not be used to further a particular political or religious standpoint.
4. We shall endeavor not to act as instruments of government foreign policy.
5. We shall respect culture and custom.
6. We shall attempt to build disaster response on local capacities.
7. Ways shall be found to involve program beneficiaries in the management of relief aid.
8. Relief aid must strive to reduce future vulnerabilities to disaster as well as meeting basic needs.
9. We hold ourselves accountable to both those we seek to assist and those from whom we accept resources.
10. In our information, publicity and advertising activities, we shall recognize disaster victims as dignified human beings, not hopeless objects.

Table 3. Key provisions of the Humanitarian Charter (Sphere Project). It is the right of people affected by disaster or armed conflict to have:[64]

1. Life with dignity: right to have steps taken to preserve life where it is threatened, and a corresponding duty of others to take such steps.
2. Assistance to meet their basic needs. Those affected are entitled to protection and assistance. It is the state's primary role and responsibility to provide assistance when people's capacity to cope has been exceeded.
3. Governments must guarantee this right. International humanitarian law obliges states and other parties to agree to the provision of humanitarian and impartial assistance when the civilian population lacks essential supplies. Frequently, warring parties fail to respect the humanitarian purpose of interventions.
4. Humanitarian agencies can provide assistance where it is needed. Those with primary responsibility are not always able or willing to perform this role themselves.
5. Distinction must be made during armed conflict between combatants and non-combatants.
6. Principle of non-*refoulement* (that no refugee shall be sent to a country in which his/her life or freedom is threatened).

situations (Table 3); and the individual codes of many disaster-assistance organizations.

In acute disasters, the primary ethical guide is the Red Cross Code, which describes responders' ideal behavior after both natural and man-made disasters. As the Red Cross describes it, their *Code of Conduct* applies to the International Red Cross and Red Crescent Movement and to other NGOs that voluntarily adopt it. The *Code of Conduct* "seeks to guard our standards of behavior . . . [and] . . . seeks to maintain the high standards of independence, effectiveness and impact" in disaster response. During armed conflict, the ICRC believes that it "will be interpreted and applied in conformity with international humanitarian law."[35]

A wide range of secular and non-secular humanitarian agencies that deal with assistance during conflicts, including the ICRC, helped develop the Sphere Project's *Humanitarian Charter*. Its purpose is to serve as an "internationally recognized set of common principles and universal minimum standards in life-saving areas of humanitarian response."[36]

In addition, individuals who participate in these groups bring their own personal and professional values that, presumably, mesh the ICRC and Sphere codes and those of the organizations with which they work. The World Medical Association (WMA) provides additional guidance for healthcare workers, saying they should "ensure that the treatment of disaster survivors conforms to basic ethical tenets and is not influenced by other motivations." In addition, WMA reiterates a basic ethical triage principle, stating that they consider it ethical for the healthcare provider not to continue treating victims whom they cannot help, since they better can use the few available resources for many others in need.[1,37,38]

Ethics of Disaster Research

Research on all aspects of human catastrophes has languished, especially that dealing with the optimal criteria for and efficacy of different types of disaster response. In part, this has been due to inadequate resources. However, it also stems from researchers' queasiness about intruding in the midst of crisis—and from the responders' negative reactions to their efforts. However, the lack of evidence-based criteria for many disaster interventions raises ethical concerns about when, why and how we should respond to humanitarian catastrophes.

Ethical issues also pervade any disaster-related research project. Current thought, which is based on U.S. National Institute of Mental Health guidelines, is that such research must undergo the same rigorous ethical tests that guide all other research. This includes the assumption that those affected by a disaster retain the capacity to provide informed consent, although capacity assessment may need to be part of the protocol. They also imply that, while disaster-affected populations (and responders, if they are being studied) are not "vulnerable," researchers should still assess a study's risk-benefit and its effect on the subjects. As in other research, investigators should maintain confidentiality and privacy, subjects should receive study results, and redundant research should be minimized.[39]

LEGAL ASPECTS OF INTERVENTION

For most acute and chronic humanitarian crises, national

sovereignty and local laws govern the response and the responders. Nations must give their permission before other governments or supragovernmental agencies and NGOs can work within their borders. Their workers must abide by local rules and regulations.

Refugee Law

Refugee law determines which groups are eligible for special assistance through the UN or other governmental bodies. Related to, but distinct from international human rights law, refugee law comprises both the UN Convention and more limited instruments from various regional bodies.

Military Intervention

"Humanitarian intervention" involves a state or organization's threat or use of "military force against another state when the chief publicly declared aim of that military action is ending human rights violations."[40] Humanitarian interventions, whose objectives stem from Human Rights Law, involve protecting vulnerable populations (through safe havens) or delivering humanitarian aid.[34]

Human Rights Law. Human rights law derives from a series of advisory (non-binding) declarations and (binding) conventions. Most frequently cited is the so-called International Bill of Human Rights, which includes the UN's Universal Declaration of Human Rights (1948) and two conventions (treaties) passed by the General Assembly in 1966: the International Covenant on Economic, Social and Cultural Rights and the International Covenant on Civil and Political Rights. Both went into force when sufficient members signed and ratified it in 1976. Numerous advisory declarations from the UN and other international bodies, as well as global and regional conventions (treaties), make up the balance of International Human Rights Law.

The nature of and actions taken during governmental/supragovernmental (UN, NATO, etc.) interventions follow international humanitarian law or the law of armed conflict (Table 4), rules and principles that seek to save lives and alleviate suffering during armed conflict.[41] These include the Law of The Hague, once referred to as the law of war proper, the Geneva Conventions, and subsequent treaties and protocols (1864 to 2005).[42] Although not all were initially legally binding, they have, in many cases, evolved into enforceable "customary international law."

HOW WELL DO WE RESPOND TO HUMANITARIAN CRISES?

When considering how effective our responses and their results are to humanitarian crises, we can best judge them through our success in achieving the relevant MDGs or the general results of our interventions in most acute crises. Most of the MDGs' 21 targets and 60 official indicators have a deadline of 2015, with 1990 as the baseline year.[25] Subsequent sections will review the progress toward the MDGs related to food and water insecurity and to preventing and treating debilitating endemic and pandemic infectious diseases. The numbers in parenthesis are the official designators for that MDG target. The data are official government-supplied statistics supplemented by information from international agencies.[43]

ACUTE CRISES

After large *natural disasters*, civilian and governmental relief agencies generally provide excellent and rapid immediate relief. The many natural disasters each year bear that out. Yet, in cases where recurrence is likely, these agencies have had little effect on mitigating similar future devastation or damage or destruction through long-term planning.

Global pandemics have largely been prevented or, other than HIV, quickly contained when international cooperation has been functional. (China's denial of SARS allowed it to spread wider than it might have otherwise.[44]) WHO and the CDC have not only provided experts to help identify and contain potential outbreaks, but also trained and equipped many laboratories and clinicians around the world to assist in these efforts.

MDG INDICATORS (6.A AND 6.B): HAVE HALTED BY 2015 AND BEGUN TO REVERSE THE SPREAD OF HIV/AIDS, AND ACHIEVE, BY 2010, UNIVERSAL ACCESS TO TREATMENT FOR HIV/AIDS FOR ALL THOSE WHO NEED IT

Fewer people have becoming infected with HIV in recent years, with 2.7 million people being newly diagnosed in 2010, 21% lower than the 1997 peak.[45] Yet the international community did not meet the target of universal coverage of the 13.7 million infected individuals.[46] Access to treatment did increase in all regions so that, at the end of 2010, 6.5 million people were receiving antiretroviral therapy for HIV or AIDS in developing regions. Eastern Asia had the largest gain in coverage (38%), while sub-Saharan Africa, where more than 50% of eligible people do not receive antiretroviral therapy, had a 20% increase.[47]

The efforts to prevent or stop genocide have not been successful. This requires humanitarian (military) interventions, and the legal basis for these interventions remains sufficiently nebulous so that long delays and many deaths commonly occur before supragovernmental bodies take any action. As a result, the 1994 Rwanda genocide left 800,000 Tutsis dead, the Serbian genocide in Srebrenica resulted in about 8,000 Bosniak deaths, and genocide is ongoing in Darfur, Sudan.

The international community has had even less success in preventing **armed conflicts**. While overt global warfare has not occurred since World War II, a nearly constant succession of at least 44 civil and regional wars have resulted in approximately 6 million deaths (the most easily measurable statistic), and have caused large-

scale disabilities, social disruption, economic hardship, population dislocation, and long-term emotional damage. Current conflicts that cause at least 1,000 deaths/year are taking place in Columbia, Afghanistan, Somalia/Somaliland, Pakistan, Mexico, South Sudan/Sudan, Syria, and Iraq.[48] Other than Iraq, Sub-Saharan Africa countries account for all those with more than 20 battle deaths/100,000 population annually.[49]

CHRONIC CRISES

Most chronic humanitarian crises can be assessed using the relevant MDGs. Another assessment factor, not included in the MDGs, is the number of refugees and IDPs.

Refugees and IDPs have been increasing in number, despite a recent increase in repatriation. At the end of 2011, about 42.5 million people worldwide were refugees due to conflict or persecution. Developing countries that can ill afford it host 4 out of 5 of these refugees. In December 2011, the largest refugee populations under the UN's care were from Afghanistan (2.7 million) and Iraq (1.4 million). About 26.4 million people are IDPs and remain within their country's borders. [50]

MDG INDICATOR (7.C): HALVE, BY 2015, THE PROPORTION OF PEOPLE WITHOUT SUSTAINABLE ACCESS TO *SAFE DRINKING WATER* AND BASIC SANITATION

The world seems to have met this target. The proportion of people using an improved water source rose from 76% in 1990 to 89% in 2010, with more than 2 billion people gaining access to improved drinking water sources, such as piped supplies and protected wells. However, not only do rural areas disproportionately lack improved water sources (19%) compared to urban areas (4%), but also all figures may be overestimates, since they do not reflect the reliability and sustainability of a safe water source.[46] More troubling, the UN estimates that "by 2025, 1.8 billion people will live in countries or regions with absolute water scarcity."[51]

MDG INDICATOR (1.C): HALVE, BETWEEN 1990 AND 2015, THE PROPORTION OF PEOPLE WHO SUFFER FROM *HUNGER*

Falling short of the target, recent estimates show that about 850 million people, 15.5% of the world's population, live in hunger, even though incomes have increased. [46] Six million children die of hunger every year—17,000 every day.[52] A key marker for hunger is the prevalence of underweight children less than 5 years old. Slow progress, although insufficient to reach the global target by 2015, has nevertheless resulted in a decline in underweight children less than 5 years old in the developing world, from 29% in 1990 to 18% in 2010.[53] Yet progress has been spotty, with nearly one-third of children in Southern Asia being underweight in 2010. [46] The prevalence of child stunting, a key marker for hunger (undernourishment) defined as low height for age, fell from an estimated 44% in 1990 to 29% in 2010, which still leaves millions of children "at risk for diminished cognitive and physical development resulting from long-term under-nutrition."[53]

Table 4. Basic rules of international humanitarian law.[65]

1. Persons *hors de combat* and those who do not take a direct part in hostilities are entitled to respect for their lives and their moral and physical integrity. They shall in all circumstances be protected and treated humanely without any adverse distinction.
2. It is forbidden to kill or injure an enemy who surrenders or who is *hors de combat*.
3. The wounded and sick shall be collected and cared for by the party to the conflict which has them in its power. Protection also covers medical personnel, establishments, transports and equipment. The emblem of the Red Cross or the Red Crescent is the sign of such protection and must be respected.
4. Captured combatants and civilians under the authority of an adverse party are entitled to respect for their lives, dignity, personal rights and convictions. They shall be protected against all acts of violence and reprisals. They shall have the right to correspond with their families and to receive relief.
5. Everyone shall be entitled to benefit from fundamental judicial guarantees. No one shall be held responsible for an act he has not committed. No one shall be subjected to physical or mental torture, corporal punishment or cruel or degrading treatment.
6. Parties to a conflict and members of their armed forces do not have an unlimited choice of methods and means of warfare. It is prohibited to employ weapons or methods of warfare of a nature to cause unnecessary losses or excessive suffering.
7. Parties to a conflict shall at all times distinguish between the civilian population and combatants in order to spare civilian population and property. Neither the civilian population as such nor civilian persons shall be the object of attack. Attacks shall be directed solely against military objectives

MDG INDICATOR (6.C): HAVE HALTED BY 2015 AND BEGUN TO REVERSE THE INCIDENCE OF *MALARIA AND OTHER MAJOR DISEASES*

Reported **malaria** cases fell by 17% since 2000 and by more than 50% between 2000 and 2010 in 43 of the 99 countries with endemic malaria. [46] Yet the momentum in addressing this devastating disease is slowing, largely due to inadequate resources and increasing plasmodium resistance. Even at its 2011 peak of $1.9 billion, international funding was well short of the $5 to 6 billion required for universal access to malaria prevention and control measures. Simultaneously, *P. falciparum* resistance to the main treatment component, artemisinins, is increasing, and Anopheles mosquitoes have increasing resistance to the pyrethroids used to treat clothing and insecticide-treated mosquito nets (ITNs). Possibly more important in preventing insect-borne diseases other than malaria, ITNs were used by 39% of children in sub-Saharan Africa in 2010, compared to 2% in 2000.[54] However, there is no data on another MDG indicator,

the proportion of febrile children less than 5-years old who were treated with appropriate antimalarial drugs.

According to the UN's 2012 *Millennium Development Goals Report*, the world is on track to achieve the target of halting, and is beginning to reverse, the spread of **tuberculosis**. While the estimated 2015 global mortality from tuberculosis will have decreased 50% from that in 1990, more than one third of new cases still go unreported and are not treated in a WHO-recommended Directly Observed Therapy Short-Course (DOTS) program.[46] Of most concern to global health personnel, 84% of the estimated 290,000 cases of multi-drug resistant tuberculosis are not being diagnosed or treated appropriately.[55]

Little data exist on the "other diseases" addressed in the MDGs. However, progress is being made to prevent many of them. One of the greatest successes has been with **trypanosomiasis** or sleeping sickness, an epidemic in Africa that mobile teams arrested by systematically screening millions of at-risk people.[56] Another MDG Indicator (4.3) is to immunize 1-year-old children against **measles**, since that frequently lethal disease causes epidemics in many poor populations. According to the *Report*, "Through increased routine immunization coverage and large-scale immunization campaigns, sub-Saharan Africa has made the most progress, with an 85 per cent drop in measles deaths between 2000 and 2010. . . [Yet] recent complacency and declines in political and financial commitments to measles control have resulted in large measles outbreaks globally."[57]

Although they are MDG indicators, the UN has failed to report any progress either in providing **access to affordable essential drugs** in developing countries or in increasing the proportion of people with access to affordable essential drugs on a sustainable basis.[58]

CHANGES TO IMPROVE HUMANITARIAN RESPONSE

The data documenting the world's response to humanitarian disasters suggest that we can do better. Specifically, improvements should include:

- Improving NGO and government coalitions/cooperation pertaining to "goals" or "problems" and disaster responses.
- Educating governments and the public about the best response to disasters (i.e., provide money to legitimate groups, unless asked for specific personnel or material).
- Clarifying internationally accepted criteria for deciding when to initiate humanitarian (military) intervention, as well as the military forces' mission. This would avoid the inevitable charges of interventions being politically motivated, subjective or media-driven.[59]
- Evaluating humanitarian interventions based not only on short-term relief or military/security objectives, but also on long-term conflict resolution.
- Funding more disaster-relief research (best relief methods, short- and long-term outcomes).
- Encouraging fundraising methods that more accurately depict post-disaster situations.
- Spending relief funds within the affected areas.
- Structuring interventions to support the recipient government's own disaster relief efforts.
- Establishing MDGs for the post-2015 period (in process) that contain more accurate, uniform and obtainable targets. They should more clearly measure the effect and the effectiveness of relief agencies and humanitarian interventions than the existing MDG targets. They should also include criteria that emphasize country enablement, rather than "quick fixes" that use primarily outside assistance.[60]

CONCLUSIONS

1. Our responses to natural disasters and to preventing new pandemics have been timely and helpful. However, we have not consistently helped local entities institute plans to mitigate the effects of future events.

2. When human rights are violated on a massive scale as happens during wars and genocide, the international rules for "humanitarian (military) interventions" remain so unclear that most interventions occur only after countless lives are lost.

3. To have any meaningful effect, the international community must supply ongoing and sufficient funding to address the chronic crises of food and water insecurity and the prevention and treatment of endemic infectious diseases. Rather than a "quick fix," these problems represent chronic indolent crises that need constant attention.

4. To determine if and where progress actually occurs, new MDGs must incorporate measurements that are more accurate.

5. Research should expand to include when, why and how we respond to humanitarian catastrophes.

Address for Correspondence: Kenneth V. Iserson, MD, MBA. University of Arizona, Department of Emergency Medicine, Tucson, AZ 85724. Email: kvi@email.arizona.edu.

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

REFERENCES

1. World Medical Association. *WMA Statement on Medical Ethics in*

the Event of Disasters (2010). Available at: http://www.wma.net. Last accessed October 24, 2013.

2. Centers for Disease Control and Prevention (CDC). *Annual Deaths Attributable to Cigarette Smoking—United States, 2000–2004*. Available at: http://www.cdc.gov/tobacco/data_statistics/tables/health/attrdeaths/index.htm. Last accessed October 24, 2013.
3. Burns WJ, Slovic P. Risk perception and behaviors: anticipating and responding to crises. *Risk Anal.* 2012 Apr;32(4):579-82. doi: 10.1111/j.1539-6924.2012.01791.x.
4. National Center for Health Statistics. *Table 1103, Motor Vehicle Accidents—Number and Deaths: 1990–2009*. Available at: http://www.census.gov/compendia/statab/2012/tables/12s1103.pdf. Last accessed October 24, 2013.
5. CDC Reproductive Health: Glossary: Epidemic. Available at: http://www.cdc.gov/reproductivehealth/epiglossary/glossary.htm. Last accessed October 24, 2013.
6. Porta M (ed.). *Dictionary of Epidemiology,* 5th ed. Oxford: Oxford University Press, 2008:179.
7. Alibek K, Handelman S. *Biohazard: The Chilling True Story of the Largest Covert Biological Weapons Program in the World—Told from Inside by the Man Who Ran It*. New York: Random House, 1999 (Delta, 2000).
8. Broad WJ. Soviet defector says China had accident at a germ plant. *New York Times*. April 5, 1999.
9. Stanford Encyclopedia of Philosophy. *War*. Available at: http://plato.stanford.edu/entries/war/. Last accessed October 24, 2013.
10. von Clausewitz C (1832 original). Howard M, Paret P (trans. eds.). *On War (Vom Krieg),* Indexed ed. Princeton, New Jersey: Princeton University Press, 1984:75.
11. The Free Dictionary. *War*. http://www.thefreedictionary.com/War. Accessed October 24, 2013.
12. United Nations. Article 2 of the Convention on the Prevention and Punishment of the Crime of Genocide (adopted by UN in 1948).
13. Gilbert N. Balancing water supply and wildlife. *Nature News*. 29 September 2010.
14. WHO/UNICEF. *Progress in Drinking-water and Sanitation: Special Focus on Sanitation*. WHO/UNICEF Joint Monitoring Programme for Water Supply and Sanitation, 2008 July 17:25.
15. *The State of the World's Children. UNICEF Report 2012*. http://www.unicef.org/sowc/files/SOWC_2012-Main_Report_EN_21Dec2011.pdf. Accessed October 24, 2013.
16. Institute for Human Rights and Business website. Available at: http://www.ihrb.org/commentary/guest/water-security-a-different-perspective.html?gclid=CNm_3-3OxLICFcgtmAodhWoA8A. Last accessed October 24, 2013.
17. WHO. *Food Security*. World Health Organization. www.who.int/trade/glossary/story028/en/. Accessed October 24, 2013.
18. Food and Agriculture Organization of the United Nations (FAO) Agricultural and Development Economics Division (June 2006). *Food Security*. Available at: ftp://ftp.fao.org/es/ESA/policybriefs/pb_02.pdf. Last accessed October 24, 2013.
19. United Nations. *The State of Food Insecurity in the World—2012*. Rome: Food and Agriculture Organization of the United Nations, 2012:57.
20. The Secretary-General's High-Level Task Force on the Global Food Security Crisis. *Global Economic Turmoil Intensifies the Food Crisis. Immediate Action Is Required To Secure Adequate Long-Term Global Food Supply (2009)*. Available at: http://www.un.org/en/issues/food/taskforce/pdf/Global_Economic.pdf. Last accessed October 24, 2013.
21. World Health Organization. *Working to Overcome the Global Impact of Neglected Tropical Diseases. First WHO Report on Neglected Tropical Diseases.* Geneva: WHO, 2010. Available at: http://whqlibdoc.who.int/publications/2010/9789241564090_eng.pdf. Last accessed October 24, 2013
22. United Nations High Commission for Refugees (2012). *Text of "Convention Relating to the Status of Refugees."* Article 1.A.2, 1951. Available at: http://www.unhcr.org/3b66c2aa10.html. Last accessed October 24, 2013.
23. Goodwin-Gill GS. Convention relating to the Status of Refugees, Geneva 28 July 1951: Protocol relating to the Status of Refugees, New York, 31 January 1967. *United Nations Audiovisual Library of International Law*. Available at: http://untreaty.un.org/cod/avl/ha/prsr/prsr.html. Last accessed October 24, 2013.
24. Human Rights Education Associates. *Refugees and Displaced Persons*. Available at: http://www.hrea.org/index.php?doc_id=418. Last accessed October 24, 2013.
25. Millennium Development Goal Indicators: The Official Site for MDG Indicators. Available at: http://mdgs.un.org/unsd/mdg/Default.aspx. Last accessed October 24, 2013.
26. Devarajan S, Miller MJ, Swanson EV. Goals for development: history, prospects, and costs. *World Bank Policy Research Working Paper.* April 2002. Available at: http://elibrary.worldbank.org/content/workingpaper/10.1596/1813-9450-2819. Last accessed October 24, 2013.
27. Charny JR. Response to Lissner J. In: Jenson E. *Disaster Management Ethics: Disaster Management Training Programme*. Geneva: United Nations, INDP and DHA, 1997:52-53.
28. Association of Fundraising Professionals. *Code of Ethical Principles and Standards*. Available at: http://www.afpnet.org/Ethics/EnforcementDetail.cfm?ItemNumber=3261. Last accessed October 24, 2013.
29. Association of Fundraising Professionals. *International Statement of Ethical Principles in Fundraising* (December 7, 2006). Available at: http://www.afpnet.org/Ethics/IntlArticleDetail.cfm?ItemNumber=3681. Last accessed October 24, 2013.
30. Nuland SB. A report from a relief mission: after the deluge. *The New Republic.* 2005 April 11;232(4708):30-33.
31. WHO. Global Outbreak Alert and Response Network-GOARN www.who.int/entity/csr/outbreaknetwork/goarnenglish.pdf. Accessed October 24, 2013.
32. WHO. *Global Alert and Response*. http://www.who.int/csr/outbreaknetwork/en/. Accessed October 24, 2013.
33. CDC. *Global Disease Detection (GDD) Operations Center*. Available at: http://www.cdc.gov/globalhealth/GDDER/optcenter/. Last

accessed October 24, 2013.
34. Lederach JP. Military intervention in disaster relief: cooperative relationships and implications for long-term rehabilitation and development. In: Jenson E. *Disaster Management Ethics: Disaster Management Training Programme*. Geneva: United Nations, INDP and DHA, 1997:9-16.
35. International Federation of Red Cross and Red Crescent Societies and the International Committee of the Red Cross. *The Code of Conduct for the International Red Cross and Red Crescent Movement and Non- Governmental Organisations (NGOs) in Disaster Relief*. Available at: www.icrc.org/eng/assets/files/publications/icrc-002-1067.pdf. Last accessed 8 October 12.
36. Sphere Project website. Available at: http://www.sphereproject.org/about/. Last accessed October 24, 2013.
37. Iserson KV, Moskop JC. Triage in medicine—part I: concept, history, and types. *Ann Emerg Med*. 2007;49(3):275-81.
38. Moskop JC, Iserson KV. Triage in medicine—part II: underlying values and principles. *Ann Emerg Med.* 2007;49(3):282-7.
39. National Institute of Mental Health. *Ethical Issues to Consider in Developing, Evaluating, and Conducting Research Post Disaster* (2007). Available at: http://www.nimh.nih.gov/research-funding/grants/ethical-issues-to-consider-in-developing-evaluating-and-conducting-research-post-disaster.shtml. Last accessed October 24, 2013.
40. Marjanovic M. Is humanitarian war the exception? *Mises Daily.* April 4, 2011. Available at: http://mises.org/daily/5160. Last accessed October 24, 2013.
41. American Red Cross. *The International Red Cross and Red Crescent Movement and American Red Cross International Services Self-Study Guide.* Washington, DC: American Red Cross, 2007:8.
42. Pictet J. *Humanitarian Law and the Protection of War Victims*. Leyden: Sijthoff, 1975:16-17.
43. United Nations. *The Millennium Development Goals Report 2012.* New York: UN, 2012:66.
44. WHO targets SARS 'super spreaders' CNN. April 6, 2003. http://archive.is/sfqGg Accessed November 23, 2013.
45. United Nations. *The Millennium Development Goals Report 2012*. New York: UN, 2012:39.
46. United Nations. *The Millennium Development Goals Report 2012*. New York: UN, 2012:4-5.
47. United Nations. *The Millennium Development Goals Report 2012*. New York: UN, 2012:41.
48. Wikipedia. List of ongoing military conflicts. Available at: http://en.wikipedia.org/wiki/List_of_ongoing_military_conflicts. Last accessed October 24, 2013.
49. Gapminder.org (Accessed 14 October 2012).
50. United Nations. *The Millennium Development Goals Report 2012*. New York: UN, 2012:15.
51. *FAO (UN) Fact Sheet.* http://www.unwater.org/downloads/media/scarcity/i0142e07.pdf .Accessed October 24, 2013.
52. U.N. chief: Hunger kills 17,000 kids daily. *CNN.* November 17, 2009. (Accessed 2 May 2010).
53. United Nations. *The Millennium Development Goals Report 2012*. New York: UN, 2012:13.
54. United Nations. *The Millennium Development Goals Report 2012*. New York: UN, 2012:43.
55. United Nations. *The Millennium Development Goals Report 2012.* New York: UN, 2012:45.
56. WHO Media centre (2001). Fact sheet No. 259: *African trypanosomiasis or sleeping sickness*. Available at: http://www.who.int/mediacentre/factsheets/fs259/en/. Last accessed October 24, 2013.
57. United Nations. *The Millennium Development Goals Report 2012*. New York: UN, 2012:29.
58. United Nations. *The Millennium Development Goals Report 2012*. New York: UN, 2012.
59. Ferris E. Response to Lederach's Chapter.In: Jenson E. *Disaster Management Ethics: Disaster Management Training Programme*. Geneva: United Nations, INDP and DHA, 1997:17-18.
60. Poku N, Whitman J. The Millennium Development Goals and Development after 2015. *Third World Quarterly.* 2011;32(1):181-198.
61. World Health Organization. *The Global Burden of Disease: 2004 Update.* Geneva: WHO, 2008. Available at: http://www.who.int/healthinfo/global_burden_disease/2004_report_update/en/index.html. Last accessed October 24, 2013.
62. Murray CJL, Vos T, Lozano R, et al. Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990—2010: a systematic analysis for the Global Burden of Disease Study 2010. *The Lancet*. 2012;380(9859):2197-2223.
63. International Red Cross and Red Crescent Code. Available at: http://www.ifrc.org/en/publications-and-reports/code-of-conduct/. Last accessed October 24, 2013.
64. Sphere Project. *Humanitarian Charter and Minimum Standards in Humanitarian Response.* Bourton on Dunsmore, Rugby, United Kingdom: Practical Action Publishing, Schumacher Centre for Technology and Development, 2011:19-24.
65. International Committee of the Red Cross. Basic rules of the Geneva Conventions and their additional protocols. Available at: www.icrc.org/eng/assets/files/other/icrc_002_0365.pdf. Last accessed November 22, 2013.

Onset and Duration of Intravenous and Intraosseous Rocuronium in Swine

Michael Loughren, CRNA, PhD
Sarah Banks, CRNA, MS
Carleo Naluan, CRNA, MS
Paul Portenlanger, CRNA, MS
Arthur Wendorf, CRNA, MS
Don Johnson, RN, PhD

Department of Anesthesia & Operative Services, Madigan Army Medical Center, Tacoma, Washington

Supervising Section Editor: Michael Epter, DO
Submission history: Submitted October 30, 2012; Revision received January 30, 2013; Accepted September 4, 2013
Electronically published January 10, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.9.14484

Introduction: The intraosseous (IO) route has become a popular method to gain access to the peripheral circulation in emergency situations. Despite little supporting data, it is generally believed that IO absorption is immediate and equivalent to the intravenous (IV) route. It is important to determine if rocuronium can effectively be administered by the IO route. The aim of the study was to determine and compare the onset and duration of rocuronium when administered via the IO and IV routes in a normovolemic pig model.

Methods: We recorded electromyographic (EMG) data following tibial IO and peripheral IV administration of rocuronium (1.2 mg/kg) in 10 swine weighing between 56 and 71 Kg. We transformed data were transformed to percent of baseline, determined onset and recovery characteristics.

Results: The onset EMG-time profiles for IO and IV administration were very similar: tibial IO compared to IV administration did not statistically alter the onset of paralysis. The IO group took statistically longer than the IV group to return to 50 (p=0.042), 75 (p=0.034) and 95 (p=0.036) percent of baseline activity.

Conclusion: The duration of effect is statistically longer after IO administration but is more of an academic interest than a clinical concern. The results of this study suggest that rocuronium can effectively be administered via the IO route without the need for dose adjustments. [West J Emerg Med. 2014;15(2):241–245.]

INTRODUCTION

Intraosseous (IO) access was first contemplated in 1922 by Drinker while examining the marrow cavity of the sternum. The route became popular in the 1940s, and the sternal puncture kit for bone marrow infusions was a common component of emergency medical supplies during World War II.[1,2] Following World War II, the use of IO devices began to diminish and was all but abandoned with the rapid development of plastic catheters and routine venous cannulation. In the 1980s, the technique was re-introduced in response to the need for immediate vascular access during cardiopulmonary resuscitation (CPR). Currently, the technique is used throughout the United States and is recognized as an accepted alternative to intravenous (IV) access in pediatric emergencies and, increasingly, in neonatal and adult emergencies.[3-5] In addition, the ease, effectiveness, and safety of the technique have led to its use for prehospital and combat emergency care.[2,6-8] In current military operations, the use of IO access is being used from the medic at the point of injury to the emergency department at combat hospitals. Despite few data, it is generally believed that IO absorption is immediate and equivalent to the IV route. Pharmacokinetic evaluation of medications commonly used via

the IO route is necessary as dose adjustments may be needed to achieve the desired clinical effect. One such medication, rocuronium, a nondepolarizing neuromuscular blocker is used because of its rapid onset to facilitate endotracheal intubation in emergency situations where an IO catheter may be the only access to the vascular system. Information on the onset, duration, and optimal dose of IO rocuronium is beneficial to anesthesia and emergency medical providers.

METHODS

This study was a prospective, between subjects, experimental design. The aim of the study was to determine and compare the onset and duration of rocuronium when administered via the IO and IV routes in a normovolemic pig model. The study protocol was approved by the local Institutional Animal Care and Use Committee. The animals received care in compliance with the Animal Welfare Act and the Guide for the Use of Laboratory Animals.

We observed 10 Yorkshire-cross swine weighing between 56 and 71 Kg for 3 days to ensure a good state of health. They were fed a standard diet and restricted to nothing by mouth (NPO) after midnight the day of the experiment. On the day of the experiment, we randomly assigned them (5 per group) to either the IO or IV groups. We induced general anesthesia with an intramuscular injection of tiletamine and zolazepam (4-8 mg/kg) and then orally intubated the animals. Anesthesia was maintained with isoflurane (1%). We monitored physiologic variables throughout the experiment, and the temperature of the pig was maintained at greater than 36.0 degrees Celsius with the use of a forced air warmer as needed. We obtained electromyographic (EMG) data following a modified method previously described by Shi et al.[9] Specifically, an incision was made in the jugular furrow exposing branches of the vagus nerve, which innervate the sternomastoid muscle. We placed 2 subdermal needle electrodes (Medtronic USA, Jacksonville, FL) in the sternomastoid muscle for direct EMG recording. These electrodes were connected to a Nerve Integrity Monitor (NIM)-response 3.0 monitor (Medtronic USA, Jacksonville, FL). We used a Prass monopolar probe (Medtronic USA, Jacksonville, FL) in direct contact with a branch of the vagus nerve for stimulation of the sternomastoid muscle. The stimuli were 0.1 millisecound in duration, 5 mA in intensity (supramaximal) and repeated at 4 Hz. The NIM-response 3.0 monitor was set to run with a 50 millisecond time window with an amplitude scale set at 0.2 mV/division. Event capture was activated at a threshold of 0.02 mV. Once baseline EMG amplitudes were established, the investigators administered 1.2 mg/kg of rocuronium (APP Pharmaceuticals, Schaumburg, IL) in a single bolus either via a 20gauge IV catheter placed in an ear vein or a 15 gauge IO needle (Vidacare Corporation, San Antonio, TX) inserted in the proximal area of the tibia followed by a 10 mL normal saline flush. We confirmed IO placement prior to drug administration with aspiration of bone marrow and easy irrigation of 10 mL of normal saline. EMG amplitudes were measured at baseline and at 15 second intervals until termination of EMG activity. We then measured EMG activity every 5 minutes for the next 90 minutes or until there was a return to baseline values.

We transformed the initial data to percent of baseline and graphed as percent baseline versus time. Individual onset and recovery data were plotted using Excel (Microsoft, Redmond, WA). We used the square of the correlation coefficient (R^2) to guide the line of best fit. The line of best fit was used to determine the time from the end of injection to 90% reduction of baseline EMG activity ($Onset_{90}$), the time to maximum reduction ($Onset_{peak}$), and the maximum reduction of the neuromuscular response (peak effect).[10] The time from the end of injection to the return of 25%, 50%, 75% and 95% of baseline EMG activity was also determined from the line of best fit and used to characterize recovery of neuromuscular function.[11]

The results are expressed as mean ± standard deviation and range. We compared the $Onset_{90}$ and $Onset_{peak}$ of the IO and IV groups using a MANOVA. A repeated ANOVA was used to determine recovery of muscle paralysis with a with a post-hoc least significant difference test. An alpha of 0.05 was used for significance. We performed all analyses with SPSS version 18 (SPSS Inc, Chicago, Illinois).

RESULTS

The weights of the animals were not statistically different by group (p=0.47). Onset data for one subject in the IV group could not be collected because of technical reasons. Baseline EMG amplitudes ranged from 1.0 to 5.6 mV, and all animals achieved a 100 percent reduction in EMG activity.

Onset

EMG amplitudes in both groups declined rapidly after rocuronium administration. The time from the end of injection to 90% reduction of baseline EMG activity ($Onset_{90}$) and the time to maximum reduction ($Onset_{peak}$) were determined and used to characterize the onset of neuromuscular blockade.

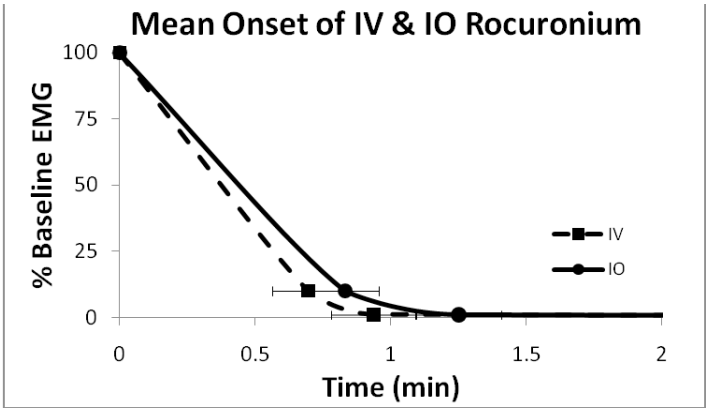


Figure 1. Time course to 90 and 100% reduction in baseline EMG following 1.2 mg/kg intravenous (IV) and intraosseous (IO) rocuronium (mean ± SEM).

Table 1. Onset of muscle paralysis following 1.2 mg/kg rocuronium (mean ± SD).

	Intravenous n=4	Intraosseous n=5	p-value
$Onset_{90}$ (sec)	42 ± 16	50 ± 17	0.240
$Onset_{peak}$ (sec)	56 ± 19	75 ± 21	0.105

Table 2. Recovery of muscle paralysis following 1.2 mg/kg rocuronium (mean ± SD).

	Intravenous n=5	Intraosseous n=5	p-value
25%	28 ± 6 minutes	37 ± 11 minutes	0.065
50%	34 ± 6 minutes	45 ± 10 minutes	0.042
75%	39 ± 6 minutes	50 ± 11 minutes	0.034
95%	43 ± 6 minutes	55 ± 12 minutes	0.036

The mean time to $Onset_{90}$ was 50 seconds in the IO group compared to 42 seconds in the IV group (p=0.24). The mean time to $Onset_{peak}$ was 75 seconds in the IO group compared to 56 seconds in the IV group (p=0.10). The results are summarized in Table 1. Figure 1 shows the initial time course of EMG recordings for IV and IO administration. Tibial IO administration did not statistically alter the onset of paralysis.

Recovery

Recovery from neuromuscular blockade was characterized by the time from injection to return of 25, 50, 75 and 95 percent of baseline EMG activity. Figure 2 illustrates the time course of mean EMG activity for the IV and IO groups. Both groups had a rapid and complete response from the rocuronium bolus and began to recover between 20 and 30 minutes following administration. The mean time for the IV group to recovery was faster than the IO group (p<0.05). The IO group took statistically longer than the IV group to return to 50, 75 and 95 percent of baseline activity. Table 2 presents a comparison of the mean times from injection to recovery of these baseline values.

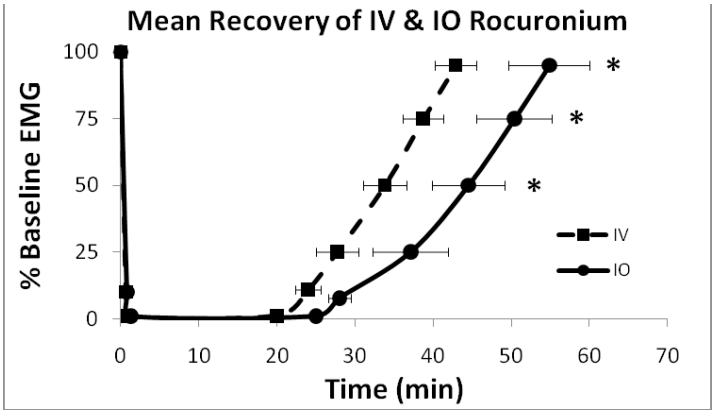


Figure 2. Time course to recovery of 25, 50, 75 and 95% of baseline EMG following 1.2 mg/kg intravenous (IV) and intraosseous (IO) rocuronium (mean ± SEM). *p<0.05

DISCUSSION

There are physiologic differences between the IO and IV routes that could theoretically lead to pharmacokinetics differences. It is generally thought that the most successful sites for IO infusion are long bones that have red marrow because of the richly vascular red marrow cavity. However, the red marrow of the long bones is gradually replaced by less vascular yellow marrow after age five. Yellow marrow contains approximately 95% fat cells and 5% nonfat cells, whereas red marrow contains 60% hematopoietic cells and 40% fat cells.[12] In 1990, Fiser recommended that tibial IO only be used in children because of this change of red to yellow marrow with age.[13,14] However, yellow marrow contains numerous venous sinusoids that can support modest IO infusion rates. In fact, the tibia and humerus are popular sites for IO access in adults, despite containing almost exclusively yellow marrow after 18 years of age.[12] In theory, a drug administered by the IO route could distribute to the bone marrow, creating an absorption phase. The bone marrow may act as a depot or reservoir that slowly releases drug into the circulation. The magnitude of this "depot effect" depends on the make-up of the bone marrow and the lipophilicity of the drug administered. Two basic phenomena or variables have potential to alter the pharmacokinetics of drugs delivered by the IO route: distribution to the bone marrow and blood flow to the bone. This study was designed to gain insight into the extent to which distribution into bone marrow affects the onset and duration of rocuronium when delivered via the IO route.

This study has two major findings. First, there was no statistical difference from the time of administration to complete neuromuscular blockade between the IO and IV administration of 1.2 mg/kg of rocuronium (p=0.10). Second, the recovery of neuromuscular function was significantly longer after IO administration (p=0.03). These results lend support to the notion that some portion of the dose immediately distributes to the marrow and is slowly absorbed over time. However, this portion does not appear to be sufficiently large as the time to onset of neuromuscular blockade was unaffected. Specifically, the fraction of the dose that immediately reached the central circulation was sufficiently large to produce neuromuscular blockade as quickly as the 1.2 mg/kg IV dose. These findings are similar to Prete and colleagues who compared the pharmacokinetics of IV, IO, and endotracheal atropine in macaques.[15] Although not significantly different (p=0.055), the mean plasma levels of atropine delivered by the IO route were noticeably higher than IV levels 5 minutes after administration (45 versus 20 nmol/L). This same trend was observed by Spivey and colleagues who compared IO and IV administration of diazepam in swine. Diazepam plasma levels were higher after IV for the first 5 minutes following administration but were

less than IO for the remainder of the 20-minute experiment.[16, 17] This trend has not been observed in all studies. For example, Orlowski compared plasma levels of lidocaine and calcium after IV and IO administration in dogs and found the plasma concentration versus time profiles were indistinguishable.[18] In the only human subject study that has compared the pharmacokinetics of drugs administered by the 2 routes, Von Hoff found there were no statistically significant differences between the maximum concentrations (Cmax) or the time to maximum concentration (Tmax) after IO (iliac crest) and IV administration of morphine sulfate.[19] Overall, the results support the bioequivalence of IO and IV administration of morphine sulfate in adults. However, the volume of distribution was significantly greater in the IO group, a finding that suggests that there may be some distribution in the marrow. This result is surprising because one would predict minimal distribution when administering a fairly hydrophilic molecule such as morphine into the iliac crest, a site that contains mostly red marrow.

Quaternary ammonium compounds such as rocuronium are relatively hydrophilic compounds. It was unexpected and of academic interest that rocuronium appeared to distribute to the bone marrow to an extent that would affect the recovery of a single bolus dose. However, despite being statistically significant, the increased duration of action would probably not have any clinical significance. The clinical scenarios in which rocuronium would be administered via an IO catheter are unlikely to need rapid recovery. Nevertheless, because a hydrophilic drug, such as rocuronium, shows signs of distribution to the bone marrow then other more lipophilic medications may have greater distribution to the marrow when administered via the IO route. The distribution could be significant enough to create an absorption phase, decreasing concentrations of the medication to such an extent that a dose adjustment is necessary to achieve the desired effect.

LIMITATIONS

The results may not be generalizable to humans; however, pigs are very similar in anatomy and physiology and should approximate results with humans. In fact, the tibia of a 70 Kg pig is shorter and may contain less marrow than a human of the same weight. This difference may underestimate any distribution or depot effect. Additionally, the results may not be generalizable to sternal IO administration. The sternum has a smaller marrow volume and is made up of red marrow compared to the adult tibia, which is made up of almost entirely yellow marrow. In theory, if a medication exhibits a distribution phase when given via the IO route, it should be minimized when administered via sternal IO.

CONCLUSION

In summary, IO and IV administrations of rocuronium (1.2 mg/kg) have similar onset characteristics. The duration of effect is statistically longer after IO administration but is more of an academic interest than a clinical concern. Rocuronium can effectively be used via the IO route without the need for dose adjustments.

Address for Correspondence: Michael J Loughren, CRNA, PhD, Department of Anesthesia & Operative Services, Madigan Army Medical Center, Tacoma, WA. Email: mike.loughren@gmail.com.

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

REFERENCES

1. Drinker CK, Drinker KR, Lund CC. The circulation in the mammalian bone marrow. *Am J Physiol.* 1922; 62:1-92.
2. Dubick MA, Holcomb JB. A review of intraosseous vascular access: current status and military application. *Mil Med.* 2000;165(7):552-559.
3. McCarthy G, Buss P. The calcaneum as a site for intraosseous infusion. *J Accid Emerg Med.* 1998;15(6):421.
4. McCarthy G, O'Donnell C, O'Brien M. Successful intraosseous infusion in the critically ill patient does not require a medullary cavity. *Resuscitation.* 2003;56(2):183-186.
5. Ramet J, Clybouw C, Benatar A, et al. Successful use of an intraosseous infusion in an 800 grams preterm infant. *Eur J Emerg Med.* 1998;5(3):327-328.
6. Glaeser PW, Hellmich TR, Szewczuga D,et al. Five-year experience in prehospital intraosseous infusions in children and adults. *Ann Emerg Med.*1993;22(7):1119-1124.
7. Glaeser PW, Losek JD, Nelson DB, et al. Pediatric intraosseous infusions: impact on vascular access time. *Am J Emerg Med.* 1988;6(4):330-332.
8. Glaeser PW, Losek JD. Intraosseous needles: new and improved. *Pediatr Emerg Care.* 1988;4(2):135-136.
9. Shi Y, Hou V, Tucker A, et al. Changes of extremity and laryngeal muscle electromyographic amplitudes after intravenous administration of vecuronium. *Laryngoscope.* 2008;118(12):2156-2160.
10. Hemmerling TM, Schurr C, Walter S, et al. A new method of monitoring the effect of muscle relaxants on laryngeal muscles using surface laryngeal electromyography. *Anesth Analg.* 2000;90(2):494-497.
11. Gerstner GE, Goldberg LJ. An analysis of mandibular movement trajectories and masticatory muscle EMG activity during drinking in the guinea pig. *Brain Res.* 1989;479(1):6-15.
12. Blebea JS, Houseni M, Torigian DA, et al. Structural and functional imaging of normal bone marrow and evaluation of its age-related changes. *Semin Nucl Med.* 2007;37(3):185-194.
13. Salassi-Scotter M, Fiser DH. Adoption of intraosseous infusion technique for prehospital pediatric emergency care. *Pediatr Emerg*

*Care.*1990;6(4):263-265.

14. Fiser DH. Intraosseous infusion. *N Engl J Med.* 1990;322(22):1579-1581.
15. Prete MR, Hannan CJ, Jr., Burkle FM, Jr. Plasma atropine concentrations via intravenous, endotracheal, and intraosseous administration. *Am J Emerg Med.* 1987;5(2):101-104.
16. Spivey WH, McNamara RM, MacKenzie RS, et al. A clinical comparison of lidocaine and bupivacaine. *Ann Emerg Med.* 1987;16(7):752-757.
17. Spivey WH, Unger HD, McNamara RM, et al. The effect of intraosseous sodium bicarbonate on bone in swine. *Ann Emerg Med.* 1987;16(7):773-776.
18. Orlowski JP, Porembka DT, Gallagher JM, et al. Comparison study of intraosseous, central intravenous, and peripheral intravenous infusions of emergency drugs. *Am J Dis Child.* 1990;144(1):112-117.
19. Von Hoff DD, Kuhn JG, Burris HA, 3rd, et al. Does intraosseous equal intravenous? A pharmacokinetic study. *Am J Emerg Med.* 2008;26(1):31-38.

The Effect of Compressor-administered Defibrillation on Peri-shock Pauses in a Simulated Cardiac Arrest Scenario

Joshua Glick, BS*
Erik Lehman, MS†
Thomas Terndrup, MD‡

* Penn State Hershey Medical Center, Penn State College of Medicine, Hershey, Pennsylvania
† Penn State Hershey College of Medicine, Department of Public Health Sciences, Hershey, Pennsylvania
‡ The Ohio State University, College of Medicine, Department of Emergency Medicine, Columbus, Ohio

Supervising Section Editor: Amal Mattu, MD
Submission history: Submitted April 15, 2013; Revision received July 4,2013; Accepted September 16, 2013
Electronically published January 6, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.9.18007

Introduction: Coordination of the tasks of performing chest compressions and defibrillation can lead to communication challenges that may prolong time spent off the chest. The purpose of this study was to determine whether defibrillation provided by the provider performing chest compressions led to a decrease in peri-shock pauses as compared to defibrillation administered by a second provider, in a simulated cardiac arrest scenario.

Methods: This was a randomized, controlled study measuring pauses in chest compressions for defibrillation in a simulated cardiac arrest model. We approached hospital providers with current CPR certification for participation between July, 2011 and October, 2011. Volunteers were randomized to control (facilitator-administered defibrillation) or experimental (compressor-administered defibrillation) groups. All participants completed one minute of chest compressions on a mannequin in a shockable rhythm prior to administration of defibrillation. We measured and compared pauses for defibrillation in both groups.

Results: Out of 200 total participants, we analyzed data from 197 defibrillations. Compressor-initiated defibrillation resulted in a significantly lower pre-shock hands-off time (0.57 s; 95% CI: 0.47-0.67) compared to facilitator-initiated defibrillation (1.49 s; 95% CI: 1.35-1.64). Furthermore, compressor-initiated defibrillation resulted in a significantly lower peri-shock hands-off time (2.77 s; 95% CI: 2.58-2.95) compared to facilitator-initiated defibrillation (4.25 s; 95% CI: 4.08-4.43).

Conclusion: Assigning the responsibility for shock delivery to the provider performing compressions encourages continuous compressions throughout the charging period and decreases total time spent off the chest. However, as this was a simulation-based study, clinical implementation is necessary to further evaluate these potential benefits. [West J Emerg Med. 2014;15(2):246–250.]

INTRODUCTION

During cardiac arrest, significant pauses occur during resuscitation, particularly during defibrillation and endotracheal intubation.[1-2] Results from several porcine and human studies suggest that these pauses are detrimental to survival.[3-5] Furthermore, a higher chest compression fraction (CCF) has been shown to correlate with increased chances of survival for out-of-hospital arrest patients in both shockable and non-shockable rhythms.[6-7] Moreover, longer pre-shock pauses in chest compressions have been associated with defibrillation failure[8], decrease in the likelihood of return of spontaneous circulation (ROSC)[9], and a decrease in survival to hospital discharge.[10] Associations between post-shock pauses and health outcomes have been mixed.

Current American Heart Association (AHA) and European Resuscitations Council (ERC) guidelines stress the importance of minimizing interruptions to chest compressions during cardiac arrest. Using manual over automatic defibrillation eliminates the need for lengthy computer analysis of pre-shock rhythm and minimizes the no-flow fraction.[11-12] However,

manual defibrillation has been associated with a higher frequency of inappropriate shocks and is rapidly falling out of clinical use over defibrillation with pads applied to the chest.[13] Moreover, both the AHA and ERC currently recommend continued chest compressions while the defibrillator is being charged, an action that only recently has been considered to be safe for healthcare providers.[14-16]

Fear of accidental shock to a provider during defibrillation efforts often leads to an increase in the duration of peri-shock pauses and no-flow fraction. Traditionally, during an in-hospital cardiac arrest, one provider is usually responsible for performing chest compressions and a second provider is responsible for attaching the pads, charging the defibrillator, and delivering the shock. We propose a modified protocol in which a single provider is responsible for performing chest compressions and delivering the defibrillation shock. A second provider would still be responsible for attaching the pads and charging the defibrillator. Modifying which provider is responsible for the administration of a shock may decrease the duration of peri-shock pauses because of provider certainty about safe shock delivery and lower the risk of accidental defibrillation. We hypothesized that combining the responsibilities of shock delivery and chest-compression performance may lower no-flow periods and ultimately improve the probability of successful resuscitation.

METHODS

Design

This was a prospective, randomized controlled study measuring peri-defibrillation pauses of trained healthcare providers in a simulated cardiac arrest scenario. The institutional review board approved the research study via exemption.

Setting and Population

Participants were recruited in the local emergency department (ED) and from the Colleges of Medicine and Nursing. Prior to enrollment, all participants had completed a certified basic life support course in the previous 2 years and provided verification of such. All participants were 18 years of age or older.

Study facilitators included 8 medical students who all received 2 hours of training regarding the use of simulation equipment and study protocols. All facilitators received a standardized set of verbal instructions to provide use during each assessment and performed several practice sessions to ensure that differences in facilitation and data collection were minimized. Instructions were also provided for extracting data from the recording software.

Protocol

During enrollment periods in the ED, we asked potential study participants (technicians, nurses, physicians) to participate if time permitted. Arrangements were made for staff members to participate at a later time if requested. We recruited all medical and nursing students by e-mail for participation. Interested students were scheduled for screening and enrollment at the Clinical Simulation Center. Participants were randomized to either a control or study group using a permuted block list.

After obtaining verbal consent, participants were then informed that a simulated patient represented by a nearby mannequin (Laerdal Resusci Anne® Simulator, #150-0001, Wappingers Falls, NY) was experiencing "cardiac arrest with a known shockable rhythm." The scenario information was provided in advance in order to remove the effects of variability in rhythm interpretation and focus on the measurement of differences in peri-shock pauses due exclusively to the defibrillation strategy. All subjects were asked to complete 1 minute of chest compressions on the simulation mannequin. Subjects were told when 30 and 45 seconds had elapsed and, at 50 seconds, that a defibrillator was being charged. After 1 minute of chest compressions, the facilitator notified the participant that a shock was ready.

All participants were currently trained in basic life support including a recent emphasis on reducing the duration of no-flow periods. Participants in the experimental group were instructed to clear by-standers, and after the facilitator confirmed that participants were clear, the participant administered defibrillation. These participants were instructed to continue chest compressions while clearing the patient. In the control group, the facilitator requested that compressions be continued during the charging period and then stopped in order to clear the patient prior to administering a shock. Defibrillation was performed by the facilitator immediately upon all participants being clear for safe defibrillation. All participants were instructed to resume compressions immediately after defibrillation. Each participant repeated the scenario 3 times, which allowed the experimental group to practice and become comfortable with the alternative technique. Accordingly, we used data from the third scenario for analysis.

Measurements

Following each simulated resuscitation, data regarding pre-shock and peri-shock times were measured and recorded for each participant (Laerdal PC SkillReporting System, #317000, Wappingers Falls, NY). We defined the pre-shock time period as the time from release of the last chest compression until administration of defibrillation. The peri-shock period was defined as the time from release of the last chest compression until the start of the first chest compression after defibrillation.

We collected and managed study data using Research Electronic Data Capture (REDCap) electronic data capture tools hosted at our facility. REDCap is a secure, web-based application designed to support data capture for research studies.

Statistical Methods

We computed descriptive statistics for all variables using means for continuous variables and percentages for categorical variables. After determining that the distributions of the outcome variables were approximately normal, we used a two-sample T-test to assess the statistical significance of the difference in mean pre-shock pause and in mean peri-shock pause between the control and study groups. All analyses were carried out using SAS Software version 9.2 (SAS Institute, Inc., Cary, NC).

RESULTS

During the period of July 2011 to October 2011, we recruited a total of 200 students and staff for participation in this study. Of these participants, complete and accurate data were available from 197 evaluations. Half of the participants (n=100) were enrolled in the control group and the remaining participants were enrolled in the study group (n=100). Demographic data for all study participants are shown in Table 1. The mean ages of the control and study groups were 29.4 ± 9.1 and 27.5 ± 7.0, respectively. The control group was 41% male, while the study group was 52% male. The majority of participants for both groups were medical students, but subjects also included nursing students, technicians, nurses, and physicians.

Table 2 highlights the differences in pre-shock and peri-shock pauses between the study group (compressor-administered defibrillation) and the control group (facilitator-administered defibrillation). The mean pre-shock pause time for the study group was significantly lower than the pause time of the control group (0.57 s versus 1.49 s, $p<0.001$). Furthermore, subject-initiated defibrillation resulted in a significantly lower peri-shock time compared to facilitator-initiated defibrillation (2.77 s versus 4.25 s, $p<0.001$).

Table 1. Demographics of participants in study measuring pauses in chest compressions for defibrillation.

	Compressor-initiated shock (n=100)	Facilitator-initiated Shock (n=100)
Mean age (Years)	27.5 ± 7.0	29.4 ± 9.1
Gender (Male)	52%	41%
Level of training	--	--
Medical student	56%	46%
Nursing student	2%	7%
EMT-B, EMT-P	9%	9%
Registered nurse	26%	28%
Physician assistant	0%	1%
MD (Resident)	7%	9%

DISCUSSION

We sought to determine if no-flow time would be reduced by combining the roles of chest compression performance and administration of defibrillation. In a simulation model, we found that the revised protocol resulted in a statistically significant reduction in peri-shock pauses, but the overall reduction in no-flow time was small.

Several studies have demonstrated that lengthy pauses in chest compressions for procedures such as defibrillation or endotracheal intubation can have a negative impact on the probability of a successful resuscitation. In a study using porcine models, Yu et al[5] demonstrated that pauses in chest compressions as brief as 10 seconds prior to defibrillation can lengthen the time required to obtain ROSC and ultimately decrease the probability of successful resuscitation. Using observational human data, Edelson et al demonstrated that successful defibrillation was associated with each 5-second decrease in pre-shock pauses.[8] In a second observational study, Sell et al[9] found that the likelihood of ROSC in human patients was associated with an optimal pre-shock pause of less than 3 seconds. These studies suggest that during cardiac arrest, longer periods without chest compressions prior to administering defibrillation can decrease the likelihood that resuscitation will be successful.

Our data demonstrated that combining the responsibilities of compression performance and defibrillation led to a significant decrease in pre-shock pauses. Given that prior research has shown that pauses greater than 3 seconds can have an impact on patient survival, the combination of roles used in our study may serve as one opportunity to decrease pre-shock pauses and possibly improve the likelihood of successful resuscitation.

Moreover, while automatic defibrillation requires a lengthy pre-shock interval for rhythm analysis and charging, the use of a manual defibrillator does not require the same pauses and allows compressions to be performed until just prior to administration of defibrillation.[11-12] The most recent guidelines of both the AHA and the ERC reflect these findings and recommend that chest compressions be performed while the defibrillator is being charged.[14-15] In a simulation study by Perkins et al[16], charging the defibrillator with concurrent

Table 2. Differences in mean pause time for defibrillation.

	Control group (n=99)	Study group (n=98)	Two-sample t-test (p-Value)
Pre-shock pause (s)	1.49 ± 0.72	0.57 ± 0.50	<0.001
Peri-shock pause (s)	4.25 ± 0.89	2.77 ± 0.92	<0.001

administration of chest compressions was perceived as safe by participants and led to decreases in pre-shock pauses. Furthermore, a recent study by Lloyd et al[17] found that providers performing chest compressions during defibrillation were exposed to minimal and safe levels of leakage current. This further strengthens the suggestion that performing chest compression up to and during defibrillation may be a safe procedure that can further minimize the no-flow fraction.

Our study results suggest that the combination of compression administration and defibrillation facilitates continuous compressions during the charging period, thereby decreasing the pre-shock interval. During a resuscitation, it is likely that a provider will continue to administer chest compressions during the charging period if the risk for accidental defibrillation is minimized. A provider who is in control of both compression and defibrillation may feel more comfortable providing compressions while the defibrillator is being charged, knowing that the risk of accidental shock delivery is minimized.

LIMITATIONS

Our study has 3 important limitations. First, variability in the research protocol among facilitators is possible. The facilitator was involved with the process of defibrillation and thus may have had an effect on the pause times for the control group. All facilitators were instructed to follow a scripted procedure for pausing compressions, clearing the patient, and administering defibrillation. The scripted procedure was rehearsed several times during the training process and was available during the data collection period. Nonetheless, any variability among the facilitators may have led to discrepancies in the collected pause times.

Second, while our data demonstrated a statistically significant difference in pause times, whether these findings are clinically significant remains untested. This study was conducted in a controlled environment and only 2 providers (participant and facilitator) were present during each evaluation. In an actual hospital setting, having multiple providers is common and may complicate the direct communication that was available during the simulation. However, the clinical utility of having the chest compressor push the defibrillation activation button may be much greater when multiple providers and noisy communications, may contribute to longer no-flow periods during defibrillation. Additionally, during our simulation the defibrillator was always within reach of both the participant and the facilitator, and the pads were appropriately placed on the mannequin. During a live resuscitation, the defibrillator and pads are not always immediately available to the provider. Finally, additional aspects of complex resuscitations, such as multiple intubation attempts and central line placement, can further increase the no-flow fraction and are not accounted for in our simulation.

To determine whether the differences in pause periods are truly significant, the adaptations in provider responsibilities used in this study need to be implemented in a clinical setting and data regarding both no-flow periods and patient survival should be collected. Alternatively, a secondary study could be performed with more than 2 providers in each simulation team, which would more accurately simulate the conditions of a live resuscitation. This secondary study could also include multiple rounds of compressions and defibrillations in a longer ACLS scenario to more accurately incorporate additional factors such as provider fatigue, group communication, and hands-off time for rhythm checks or intubation.

CONCLUSION

In a simulated cardiac arrest, assigning the responsibility for shock delivery to the provider performing compressions encourages continuous compressions throughout the charging period and significantly decreases total time spent off the chest. This modification may also decrease the risk of accidental shock and improve patient outcomes. However, as this was a simulation-based study, clinical implementation is necessary to further evaluate these potential benefits.

Address for Correspondence: Joshua Glick, BS, Address. Email: glickjosh@gmail.com.

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

REFERENCES

1. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. *JAMA*. 2005;293:305–310.
2. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *JAMA*. 2005;293:299–304.
3. Yu T, Weil MH, Tang W, et al. Adverse outcomes of interrupted precordial compression during automated defibrillation. *Circulation*. 2002;106:368–372.
4. Steen S, Liao Q, Pierre L, et al. The critical importance of minimal delay between chest compressions and subsequent defibrillation: a haemodynamic explanation. *Resuscitation*. 2003;58:249–258.
5. Walcott GP, Melnick SB, Walker RG, et al. Effect of timing and duration of a single chest compression pause on short-term survival following prolonged ventricular fibrillation. *Resuscitation*. 2009;80:458-462.
6. Vaillancourt C, Everson-Stewart S, Christenson J, et al. The impact of increased chest compression fraction on return of spontaneous circulation for out-of-hospital cardiac arrest patients not in ventricular

fibrillation. *Resuscitation*. 2011;82:1501-1507.

7. Christenson J, Andrusiek D, Everson-Stewart, S, et al. Chest compression fraction determines survival in patients with out-of-hospital ventricular fibrillation. *Circulation*. 2009;120:1241-247.
8. Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. *Resuscitation*. 2006;71:137-145.
9. Sell RE, Sarno R, Lawrence B, et al. Minimizing pre- and post-defibrillation pauses increases the likelihood of return of spontaneous circulation (ROSC). *Resuscitation*. 2010;81:822-825.
10. Cheskes S, Schmicker RH, Christenson J, et al. Perishock Pause: An Independent Predictor of Survival From Out-Of-Hospital Shockable Cardiac Arrest. *Circulation*. 2011;124:58-66.
11. Pytte M, Pedersen TE, Ottem J, et al. Comparison of hands-off time during CPR with manual and semi-automatic defibrillation in a manikin model. *Resuscitation*. 2007;73:131-136.
12. Berg RA, Hilwig RW, Kern KB, et al. Automated External Defibrillation for Prolonged Ventricular Fibrillation: Lethal Delays of Chest Compressions Before and After Countershocks. *Ann Emerg Med*. 2003;42:4:458-466.
13. Kramer-Johansen J, Edelson D, Abella BS, et al. Pauses in chest compression and inappropriate shocks: A comparison of manual and semi-automatic defibrillation attempts. *Resuscitation*. 2007;73:212-220.
14. 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2005;112:IV1–203.
15. Deakin CD, Nolan JP, Sunde K, et al. ERC Guidelines for Resuscitation 2010, Section 3: Electrical Therapies: Automated External Defibrillators, Defibrillation, Cardioversion and Pacing. *Resuscitation*. 2010;81:1293–1304.
16. Perkins GD, Davies RP, Soar J, et al. The impact of manual defibrillation technique on no-flow time during simulated cardiopulmonary resuscitation. *Resuscitation*. 2007;73:109-114.
17. Lloyd MS, Heeke B, Walter PF, et al. Rescuers in direct contact with patients during biphasic external hands-on defibrillation: An analysis of electrical current flow through defibrillation. *Circulation.* 2008;117:2510–2514.

Barriers and Disparities in Emergency Medical Services 911 Calls for Stroke Symptoms in the United States Adult Population: 2009 BRFSS Survey

Munseok Seo, DrPH*
Charles Begley, PhD*
James R. Langabeer, PhD*
Jami L. DelliFraine, PhD†

* Management, Policy and Community Health Division, University of Texas Health Science Center, Houston, Texas
† Virginia Commonwealth University, Department of Health Administration, Richmond, Virginia

Supervising Section Editor: Christopher Kahn, MD, MPH
Submission history: Submitted June 16, 2013; Revision received September 9,2013; Accepted September 9,2013
Electronically published January 6, 2014
Full text available through open access at http://escholarship.org/uc/uciem_westjem
DOI: 10.5811/westjem.2013.9.18584

Introduction: This study examines barriers and disparities in the intentions of American citizens, when dealing with stroke symptoms, to call 911. This study hypothesizes that low socioeconomic populations are less likely to call 911 in response to stroke recognition.

Methods: The study is a cross-sectional design analyzing data from the Centers for Disease Control's 2009 Behavioral Risk Factor Surveillance Survey, collected through a telephone-based survey from 18 states and the District of Columbia. The study identified the 5 most evident stroke-warning symptoms based on those given by the American Stroke Association. We conducted appropriate weighting procedures to account for the complex survey design.

Results: A total of 131,988 respondents answered the following question: "If you thought someone was having a heart attack or a stroke, what is the first thing you would do?" A majority of those who said they would call 911 were insured (85.1%), had good health (84.1%), had no stroke history (97.3%), had a primary care physician (PCP) (81.4%), and had no burden of medical costs (84.9%). Those less likely to call 911 were found in the following groups: 65 years or older, men, other race, unmarried, less than or equal to high school degree, less than $25,000 family income, uninsured, no PCP, burden of medical costs, fair/poor health, previous history of strokes, or interaction between burden of medical costs and less than $50,000 family income ($p<0.0001$ by X^2 tests). The only factors significantly associated with "would call 911" were age, sex, race/ethnicity, marital status, and previous history of strokes.

Conclusion: Barriers and disparities exist among subpopulations of different socioeconomic statuses. This study suggests that some potential stroke victims could have limited access to EMS services. Greater effort targeting certain populations is needed to motivate citizens to call 911. [West J Emerg Med. 2014;15(2):251–259.]

INTRODUCTION

In the United States (U.S.), acute stroke is the third leading cause of death and the single largest reason for disability.[1] It is a medical emergency that demands immediate emergency medical services (EMS) activation for both faster transport to definitive stroke facilities and faster initial medical treatment.[2-8] Health and medical experts and professionals have long recognized the necessity of calling 911 over other contacts. Research suggests that immediate EMS activation through 911 calls benefits patients at the stroke onset because such a call is a key factor in the "stroke chain of survival."[9] This chain sequences from recognition of stroke symptoms to calling

911, from EMS dispatch to hospital, from each of the initial medical contacts such as physician examination, computed tomography (CT), neurological evaluation, diagnosis to the decision of the appropriate therapy and administration of appropriate drugs or other interventions.[2-6,8,10,11] The American Stroke Association (ASA) categorizes the recommendation of calling 911 for stroke symptoms as "Class I," based on its usefulness and effectiveness.[9]

However, a considerably low proportion of patients experiencing the onset of a stroke actually call 911.[12,13] National registry data between 2005 and 2007 from 4 states (Georgia, Illinois, Massachusetts, and North Carolina) show that less than half (47.6%) of all stroke patients actually used EMS from the stroke's onset.[12] The data from the TLL Temple Foundation Stroke Project in rural East Texas show an even smaller proportion (38%).[13]

The question is, why do so many not call 911? One explanation might pertain to lack of knowledge or awareness of a stroke symptom.[14-16] Several studies, however, have found that knowledge and stroke symptom awareness are only partially associated with EMS 911calls.[2,17-19] This suggests that there may be factors deterring people from calling 911 at the onset of stroke symptoms.[2,13-17] This study also assumes disparities in calling 911 might exist among subpopulations with different socioeconomic statuses.

This study examines barriers and disparities in U.S. citizens' intentions to call 911 when they respond to stroke symptoms. This study, the first of its kind, hypothesizes that socio-economically vulnerable groups might be less likely to call 911, based on care-seeking behavior obstructing access to needed care.[20-23]

METHODS

Conceptual Model

This study's conceptual framework is the behavioral model of healthcare utilization developed by Anderson and Aday. Their model explains how healthcare use and outcomes are affected by socio-demographic, health system, and individual factors.[20,21] This model proposes 3 determinants of use: predisposing, enabling, and need. Predisposing factors (age, sex, race and ethnicity, marital status, and education) contribute to use. Enabling factors (family income, health insurance, burden of medical costs and having a primary care physician (PCP) are those that can either enhance or impede an individual's inclination to use EMS services. Need factors (health status and symptoms) reflect whether the illness is self-perceived or evaluated by providers.

Study Design

This is a cross-sectional 1-year study based on a complex survey design.[24] The survey was a monthly, state-based telephone survey carried out in 2009 and developed by the Centers for Disease Control and Prevention (CDC) Behavioral Risk Factor Surveillance System (BRFSS). The complex design reflects a disproportionate, stratified sampling based on random-digit dialing sampling from listed and unlisted numbers using computer-assisted telephone interviewing systems.[24]

Selection of Participants

Of all non-institutionalized adults (age 18 or older) in households (N=432,607) in all 50 states of the U.S. and the 4 U.S. territories in the 2009 survey, the study's participants (n=131,988 collected from 18 participating states and the District of Columbia) answered the following question: "If you thought someone was having a heart attack or a stroke, what is the first thing you would do?" They also answered questions about 5 stroke-warning symptoms. Respondents were asked about their knowledge of stroke symptoms, i.e., the 5 most evident stroke-warning symptoms according to the American Stroke Association (ASA).[25] The questions were as follows:

1. "Do you think sudden confusion or trouble speaking is a symptom of a stroke?"
2. "Do you think sudden numbness or weakness of face, arm, or leg, especially on one side is a symptom of stroke?"
3. "Do you think sudden trouble seeing in one or both eyes is a symptom of stroke?"
4. "Do you think sudden trouble walking, dizziness, or loss of balance is a symptom of stroke?"
5. "Do you think severe headache with no known cause is a symptom of stroke?"

Outcomes Measure

Answers to "If you thought someone was having a heart attack or a stroke, what is the first thing you would do?" included: "Take them to the hospital," "Tell them to call their doctor," "Call their spouse or a family member," and "Do something else." These were dichotomized as an outcome variable with "911 call" coded "1" and "no 911 call" coded "0."

Data

In the CDC's 2009 BRFSS dataset, 18 states and the District of Columbia (DC) participated in the optional stroke module, available only that particular year.[26-28] The states included Alabama, Arizona, Connecticut, Florida, Georgia, Idaho, Indiana, Kentucky, Louisiana, Minnesota, Mississippi, Missouri, Montana, North Carolina, South Carolina, Virginia, West Virginia, and Wisconsin. These states are distributed geographically and represent more than 31% of the weighted population of the entire U.S. per the BRFSS sample. The median response rate for the 18 states and D.C. was 54.9%, ranging from 40.0% to 65.9%.[29]

The 2009 BRFSS data contained as predisposing factors age, sex, race/ethnicity, marital status, and education level. It contained as enabling characteristics family income, burden of medical costs, having a PCP, and health insurance status. It contained as need factors general health condition, previous

Table 1. First response for a heart attack and a stroke.
Q: If you thought someone was (sic) having a heart attack or a stroke, what is the first thing you would do?

Answers	n	Raw %	Weighted %
Take them to the hospital	8,885	6.7%	6.0%
Tell them to call their doctor	1,050	0.8%	0.7%
Call 911	113,848	86.3%	87.9%
Call their spouse or a family member	1,092	0.8%	0.6%
Do something else	7,113	5.4%	4.9%
Total	131,988	100%	100%

Note: 2009 Behavioral Risk Factor Surveillance System data regarding the recognition of stroke symptoms and 911 call were collected from the following 18 states: Alabama, Arizona, Connecticut, Florida, Georgia, Idaho, Indiana, Kentucky, Louisiana, Minnesota, Mississippi, Missouri, Montana, North Carolina, South Carolina, Virginia, West Virginia, and Wisconsin.

Table 2. Proportion of the intent to call 911 per stroke symptom and the size of recognition per stroke sign in response to 911 call.

	Raw		Weighted % 911 call*
	n	%	
Q1: (Do you think) sudden confusion or trouble speaking (are symptoms of a stroke?)			
Yes	120,208	96.7	88.4
No	4,139	3.3	84.0
Q2: (Do you think) sudden numbness or weakness of face, arm, or leg, especially on one side (are symptoms of a stroke?)			
Yes	124,328	97.6	88.2
No	3,084	2.4	80.9
Q3: (Do you think) sudden trouble seeing in one or both eyes (is a symptom of a stroke?)			
Yes	94,124	89.0	88.3
No	11,683	11.0	84.9
Q4: (Do you think) sudden trouble walking, dizziness, or loss of balance (are symptoms of a stroke?)			
Yes	113,556	93.9	88.3
No	7,362	6.1	83.0
Q5: (Do you think) severe headache with no known cause (is a symptom of a stroke?)			
Yes	84,551	81.3	88.2
No	19,494	8.7	86.3
Whether any stroke sign among all five stroke symptoms was recognized			
Yes	127,946	96.9	88.1
No	4,042	3.1	81.1

*p<0.001 by chi-squared test.

Table 3. The intent to call 911 for multiple stroke symptoms.

Multiple stroke symptoms	n=131,988	Weighted %* 911 call	95%CI	
			Lower	Upper
None (Baseline)	3.1	81.1	77.9	84.2
Single	2.5	84.1	81.0	87.3
Two	5.6	85.1	82.6	87.6
Three	13.3	88.4	87.6	89.3
Four	24.9	87.6	86.9	88.3
Five	50.7	88.7	88.2	89.2
Total	100%			

*p<0.0001 by chi-squared test.

history of stroke, and the number of stroke symptoms. The data also included, of course, the participating states. These variables form the basis for the study's regression and variance analyses.

Analysis

This study employed several analytic strategies. The first was a bivariate statistical analysis to test relationships between all categorical variables using the chi-squared test. The second was a univariate logistic regression analysis to examine the size of the relationship of each factor with intent to call 911 without controlling for confounders. The third was a multivariate logistic regression to examine the factors significantly associated with intent to call 911 after controlling for confounders.

This analysis treated the answers "don't know" and "refused" as missing values. All explanatory variables were grouped as follows:[30] age group (18-44, 45-64, 65 or higher), sex (male or female), race and ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, or others: Asian, native Hawaiian or other Pacific Islander, and American Indian or Alaska native), marital status (married or unmarried status that includes divorced, widowed, separated, never married, and a member of an unmarried couples), and education (≤high school, ≤2 yrs college, 4 yr college or higher), family incomes (<$25,000; $25,000 to <$50,000; $50,000 to <$75,000; $75,000 or higher), financial burden of medical care (yes or no), health insurance (yes or no), general health condition (good or better versus fair or poor), previous history of stroke (yes or no), number of stroke symptoms (one to five). In the multivariate model, this study added the state variable to adjust for geographical variation by state. The study tested the interaction between financial burden of medical care and family income (≤ or ≥$50,000). The study also dummy coded (in long format) each of the five stroke-warning symptoms that were questioned separately in the 2009 BRFSS. This was done to compare how each symptom influenced intent to call 911.

Final weights were assigned to each respondent to account for differences in the probability of their selections, non-

Table 4. Proportion of the intent to call 911 per behavioral model factors.

	Call 911	95% CI Lower	95% CI Upper	Column %	p-value
Age					
18-44	88.1	87.4	88.9	47.7	***
45-64	88.5	88.1	89.0	34.6	
65+	85.9	85.4	86.4	17.7	
Sex					
Male	86.3	85.6	86.9	47.4	***
Female	89.4	88.9	89.8	52.6	
Race/ethnicity					
White non-Hispanic	88.1	87.8	88.5	74.0	
Black non-Hispanic	88.5	87.4	89.6	13.5	***
Hispanic	86.0	83.4	88.5	7.3	
Other non-Hispanic	85.4	83.3	87.5	5.2	
Marital Status					
Not married	86.8	86.0	87.5	37.0	***
Married	88.5	88.1	88.9	63.0	
Education					
≤ high school	86.6	85.9	87.2	38.3	***
≤ 2 year college	88.0	87.2	88.8	27.1	
≥ 4 year college	89.3	88.8	89.9	34.6	
Family Income					
<$25,000	86.0	85.1	86.9	24.9	
$25,000-<$50,000	87.2	86.4	88.0	26.2	***
$50,000-<$75,000	89.2	88.5	90.0	17.0	
$75,000+	89.9	89.2	90.6	32.0	
Health Insurance					
Uninsured	85.1	83.8	86.5	14.9	***
Insured	88.4	88.0	88.7	85.1	
Health Condition					
Fair/Poor	85.9	85.0	86.7	15.9	***
Good+	88.2	87.8	88.7	84.1	
Had Stroke					
No	88.0	87.6	88.4	97.3	***
Yes	83.9	82.3	85.4	2.7	
Have PCP					
No	85.6	84.4	86.8	18.6	***
Yes	88.4	88.0	88.8	81.4	
Med Cost Burden					
No	88.3	87.9	88.7	84.9	***
Yes	85.7	84.5	86.9	15.1	
Interaction					
Yes Burden, < $50,000	85.8	84.5	87.0	12.6	***
No Burden, < $50,000	86.9	86.2	87.6	38.4	
Yes burden, ≥ $50,000	87.4	84.1	90.8	2.7	
No burden, ≥ $50,000	89.8	89.3	90.3	46.3	
Total	87.9	87.5	88.2		

Odds ratio, 95% confidence interval (95% CI) and p-value were weighted estimates. P-value was calculated by chi-squared test to show the relationship between categorical variables.
*All p-values are based on Pearson chi-squared test.
***p-value<0.0001. *PCP*, primary care physician.

Continued →

coverage and non-response, and over-sampling of the age- and sex-specific or the race-, age-, and sex-specific population in each survey.[24,26,28] For all analyses, this study used Stata statistical software version 11.1 (Stata Corp, College Station, TX) and determined the statistical significance to be at 0.05.

RESULTS

Descriptive Statistics

The 2009 BRFSS surveyed a total of 131,988 respondents out of 73,684,464 adult citizens (from 18 states and the District of Columbia). Respondents answered this question: "If you thought someone was having a heart attack or a stroke, what is the first thing you would do?" A majority of them (87.9% [95% CI = 87.5 to 88.2]) chose "call 911" over "take them to the hospital," "tell them to call their doctor," "call their spouse or a family member," or "do something else" (Table 1).

As Table 2 shows, if respondents recognized the symptom of "sudden confusion or trouble speaking," 88.4% would call 911 (this was the symptom that generated the highest "call 911" response rate). For those who recognized not a single symptom, still 81.1% would call 911. And as Table 3 shows, if respondents recognized multiple symptoms, 88.7% would first call 911 (versus 81.1% for none). If they had to respond to any of the 5 stroke symptoms, 88.1% would first call 911.

As Table 4 shows, the majority of those who would call 911 were insured (85.1%), had good health (84.1%), no stroke history (97.3%), and a PCP (81.4%); and a financial burden of medical care (84.9%). The proportion of those who would call 911 was lower in the following subgroups: those 65 years and over, men, other race, unmarried, education of a high school diploma or less, family income of less than $25,000, uninsured, no PCP, financial burden of medical care, fair/poor health, history of stroke, and interaction between financial

Table 5. Findings from univariate logistic analysis.

	Odds ratio	95% CI		p-value
		Lower	Upper	
Age				
18-44	1.00			
45-64	1.03	0.95	1.12	0.503
65+	0.83	0.76	0.90	***
Sex				
Male	1.00			
Female	1.32	1.23	1.42	***
Race/ethnicity				
White non-Hispanic	1.00			
Black non-Hispanic	1.06	0.95	1.19	0.305
Hispanic	0.90	0.71	1.13	0.361
Other non-Hispanic	0.79	0.66	0.94	**
Marital Status				
Not married	1.00			
Married	1.18	1.09	1.28	***
Education				
≤ high school	1.00			
≤ 2 year college	1.11	1.01	1.22	*
≥ 4 year college	1.29	1.19	1.40	***
Family Income				
< $25,000	1.00			
$25,000-< $50,000	1.09	0.98	1.21	0.112
$50,000-< $75,000	1.31	1.17	1.46	***
$75,000+	1.40	1.25	1.56	***
Health Insurance				
Uninsured	1.00			
Insured	1.29	1.16	1.45	***
Health Condition				
Fair /Poor	1.00			
Good+	1.23	1.13	1.33	***
Had Stroke				
No	1.00			
Yes	0.71	0.63	0.80	***
Have PCP				
No	1.00			
Yes	1.26	1.14	1.40	***
Med cost Burden				
No	1.00			
Yes	0.80	0.72	0.90	***
Single	1.00			
Two	1.08	0.79	1.46	0.635
Three	1.44	1.12	1.85	**
Four	1.33	1.04	1.71	*
Five	1.48	1.16	1.89	**
Symptoms/Troubles				
Speaking	1			
Numbness/Face…	1.58	1.44	1.73	***
Seeing	0.34	0.32	0.37	***
Walking	0.64	0.60	0.69	***
Headache	0.16	0.15	0.17	***
Interaction				
Yes Burden, < $50,000	1			
No Burden, < $50,000	1.10	0.98	1.24	0.113
Yes burden, ≤ $50,000	1.16	0.84	1.60	0.383
No burden, ≥ $50,000	1.46	1.30	1.64	***

Odds ratio, 95% confidence interval (CI) and p-value were weighted estimates. ***p-value<0.0001. **p-value<0.01. *p-value<0.05. Interaction is between medical cost burden and level of family income ($50,000). *PCP*, primary care physician.

Continued →

burden of medical care and family income of less than $50,000 (p<0.0001 by X^2 tests).

The p-value of all factors was statistically significant in X^2 tests (Table 4) and the univariate model (Table 5). In the multivariate model; however, the only factors that were significant (p<0.05) were age, sex, race/ethnicity, marital status, and history of stroke. Groups that were particularly less likely to call 911 as a response to stroke symptoms were: respondents 65 years or older (OR=0.75, p<0.0001), other race (OR=0.77, p<0.05), history of stroke (OR=0.80, p<0.01), men (OR=0.74, p<0.0001) and unmarried (OR=0.89, p<0.05; Table 6).

DISCUSSION

Compared to previous BRFSS studies, the 2009 data revealed considerable improvement in the number of respondents for stroke-warning symptoms, states participating, response rate, and respondents recognizing all 5 symptoms.[26,28] The percentage of those that would call 911 increased by about 2%.[26,28]

Notably, the proportion of those who would call 911 (88.1%) seems exaggerated compared to earlier studies: in the 2004 Michigan BRFSS between 20.41% and 51.5.0% would call according to different stroke sign;[18] in a 2006 survey of upstate New York, between 33% and 72% would call in response to a specific stroke symptom; and in 2006-2007 Missoula County (Montana) survey, overall 74% for a baseline, 76% for a follow up, and between 41% and 51% for a specific stroke symptom. [16,31] These studies, however, are in line with studies using a single state or small-area population survey. In fact, this study's approximately 88% is quite consistent with previous results from multi-state BRFSS studies, e.g., 86% in 2001 (17 states and the U.S. Virgin

Table 6. Findings from multivariate logistic analysis

	Odds ratio	95% CI		p-value
		Lower	Upper	
Age				
18-44	1.00			
45-64	0.95	0.87	1.03	0.234
65+	0.79	0.71	0.87	***
Sex				
Male	1.00			
Female	1.36	1.25	1.47	***
Race/ethnicity				
White non-Hispanic	1.00			
Black non-Hispanic	1.10	0.95	1.27	0.192
Hispanic	0.85	0.66	1.09	0.193
Other non-Hispanic	0.77	0.62	0.94	*
Marital Status				
Not married	1.00			
Married	1.12	1.02	1.24	*
Education				
≤ high school	1.00			
≤ 2 year college	1.02	0.92	1.12	0.756
≥ 4 year college	1.06	0.96	1.18	0.241
Family Income				
< $25,000	1.00			
$25,000-< $50,000	0.97	0.85	1.10	0.614
$50,000-< $75,000	1.08	0.93	1.24	0.308
$75,000+	1.08	0.92	1.28	0.345
Health Insurance				
Uninsured	1.00			
Insured	1.12	0.96	1.31	0.156
Health Condition				
Fair /Poor	1.00			
Good+	1.03	0.93	1.14	0.543
Had Stroke				
No	1.00			
Yes	0.80	0.70	0.92	**
Have PCP				
No	1.00			
Yes	1.09	0.97	1.23	0.147
Med Cost Burden				
No	1.00			
Yes	0.91	0.79	1.04	0.174
Multi-Symptoms				
Single	1.00			
Two	0.96	0.67	1.36	0.799
Three	1.28	0.97	1.70	0.077
Four	1.17	0.89	1.54	0.253
Five	1.25	0.96	1.64	0.101
States (N=18+DC)				
Alabama	1.00			
Arizona	1.01	0.75	1.35	0.955
Connecticut	1.47	1.19	1.81	***
DC	1.01	0.82	1.25	0.926
Florida	1.22	1.01	1.48	*
Georgia	1.12	0.93	1.36	0.238
Idaho	0.69	0.58	0.83	***
Indiana	0.82	0.69	0.97	*
Kentucky	0.71	0.59	0.85	***
Louisiana	0.70	0.60	0.82	***
Minnesota	1.01	0.84	1.22	0.881
Mississippi	0.53	0.45	0.62	***
Missouri	0.84	0.69	1.03	0.092
Montana	0.62	0.52	0.74	***
North Carolina	0.97	0.81	1.16	0.721
South Carolina	0.90	0.75	1.07	0.242
Virginia	0.98	0.77	1.23	0.836
West Virginia	0.79	0.66	0.94	**
Wisconsin	1.07	0.86	1.33	0.554

Continued →

***p-value<0.0001. **p-value<0.01. *p-value<0.05. Odds ratio, 95% confidence interval (CI) and p-value were weighted estimates. Interaction effects of medical cost burden and family income on call 911 were not included because serious multicolinearity was detected. Symptoms/troubles were also not included because the variable format was not consistent with other variables' format.
PCP, primary care physician; *DC,* District of Columbia

Islands) and 86% in 2005 (13 states and DC) BRFSS.[26,28]

Furthermore, compared with estimates from the design-based population survey, the actual number of times 911 was called for stroke could be lower when patients are in a panic or unable to call on their own.[26,28] For example, the data from the Paul Coverdell National Acute Stroke Registry (PCNASR), CDC-funded national project, reported that only 48% of patients were transported by EMS from the scene of symptom onset of a stroke.[12] Similarly, the data from the Greater Cincinnati/Northern Kentucky Stroke Study (GCNKSS), a population-based epidemiology study of stroke, reported a rate of only 40.5% of EMS activation for emergency transport to the emergency department.[15] As mentioned above, the finding from the TLL Project, an acute stroke surveillance and intervention project, revealed a much lower rate, only 38% of

EMS 911 activation.[13] More seriously, the rate of 911 EMS calls by patients themselves was a great deal lower, 4.3%,[13] 6%,[32] and 7.1%.[33]

Nevertheless, this study could estimate, without selecting biased samples, the proportion of "would call 911" as intention of general population responding to recognition of stroke symptoms. That 88% would call 911can also be partially explained by behavioral theories, such as the theory of planned behavior, which suggests that individual's "intentions" are often quite remarkably different from their actual "behaviors." [34] While individuals might recognize stroke symptoms and have every intention of calling 911, once the real acute event occurs, they might behave quite differently. The embarrassment or unwanted attention generated from an ambulance arriving at a patient's house is just one reason why intent differs from actual behavior.[18]

Barriers and Disparities in 911 Calling

To overcome the low, real-world 911 EMS activation, experts should identify and eventually eliminate factors that discourage people from making the call and thereby benefit the chain of survival. Although several studies have done this in limited fashion, no large-scale study targeting the general population has been conducted on the comprehensive inclusion of factors that impede EMS 911 use.[2,13-18]

After controlling for other factors, this study found few significant factors associated with the intent to call 911 in response to recognizing stroke symptoms. However, in univariate or bivariate results from chi-squared tests, all factors showed significant association with intent to call 911.[2, 13-17] That is to say, in multivariate regression results no significant relationship was discovered between the intent to call 911 and any of the financial or enabling factors, such as family income, health insurance, having a PCP or a financial burden of medical care, although they are all independent factors influencing socio-economically or financially vulnerable populations.[18,21-23] Nevertheless, this study is consistent with earlier ones that found a significant association between intent to call 911 and the factors of age, sex, and race/ethnicity.[2,14,16]

Neither were need factors significant with the exception of "had a stroke." This is inconsistent with one study and consistent with another, i.e., when people were reminded of a previous bad experience with physicians or hospitals.[2,17] Future BRFSS surveys need to categorize "caller of 911," which is not included in the current survey, to further investigate the insignificant association of enabling factors with intent to call 911.

This study hypothesized that individually, socio-economically, or financially vulnerable populations might encounter more barriers to calling 911 for EMS services. It found, based on results from multivariate logistic regression, that the would-be disadvantaged populations least likely to call 911 for a stroke were: seniors (65 years or older); men; non-Hispanic others (not white nor black), i.e., Asians, Native Hawaiians or other Pacific Islanders, American Indians, Alaska natives; unmarried (divorced, widowed, separated, never married, or a member of unmarried couples); and people who had already had a stroke.

LIMITATIONS

This study has several important limitations. First, between these BRFSS survey data and actual data from hospitals considerable differences existed in magnitude of intention to call 911 in response to stroke recognition. However, inconsistencies between them might be ascribed to different characteristics of samples from design-based and clinical-based study design, or different regions.[2, 13-18] For example, Schroeder's study on the clinical basis found that older individuals would more likely call 911; however, people under 60, in Schroeder's study, accounted for only 30%, much lower than this study's 82%.[2] Incompatible with this study's findings, Schroeder's study found no significant association between EMS use and either race or sex; the proportion of female (55%) and white (71%) were similar to this study's (53% and 74%). Wein's study using other clinical patients also found no significant association between intent to call 911and any of the following factors: age, sex, race/ethnicity, education, health insurance, and living alone. The study did find that those employed were 19% more likely to call 911.[13] In contrast, other design-based survey studies similar to this one found significant associations between calling 911 and age as well as race/ethnicity.[14, 16]

Second, there are limitations related to the BRFSS survey itself, limitations found in other similar studies.[26,28,14,16,18] For example, the following could be limitations in our study as well: under-/over-reporting of real facts due to self-reporting, underrepresentation of some populations due to not having home phones or to the proliferation of cell phone use, and generalizability to all U.S. population due to limited number of participating states.

Third, this study's multivariate logistic model had to exclude some confounders so as to control for stroke severity scales, comorbidities, and other important clinical risk factors. These include as known risk factors diabetes, hypertension, cholesterol, and obesity[14]; as health behaviors, they include smoking, physical activity, and diet.[14,16] They also include type of stroke and stroke severity scale or stroke symptoms.[15] Despite these limitations, this study's adjusted point and interval estimates are correct in so far as reflecting the U.S. general population's intention to call 911 owing to the improved quality and response rate of 2009 BRFSS data along with controlling for geographical variation due to different characteristics of states, and multiple symptoms as proxy of stroke symptom severity or urgency.

CONCLUSION

The aim of this study was to examine which factors could

hinder people from calling 911 in an emergency of stroke onsets; however, the study offers no answer as to why people hesitate in calling 911. Such an answer may be in the purview of qualitative research.

To improve the chain of survival, experts recommend that people experiencing stroke symptoms should call 911. Indeed, time lost is brain lost. Nevertheless, a low proportion of people, less than a half (47.6%) actually call 911. If researchers cannot identify factors that discourage patients or people from calling 911 or those populations most vulnerable, then the benefits from EMS activation may be easily eroded. Researchers must identify those subpopulations that may be individually, socio-economically, or financially disadvantaged (e.g., the elderly, men, more minor groups of minorities, and those unmarried). Effective promotion that raises awareness of the importance of calling 911 should include hospital-based patient education programs or community-based education campaigns. These would do well to target Asian populations who were not educated in the U.S. Other promotion possibilities include multi-media campaigns or any other organized effort that concerns a chain of survival or management system for acute stroke care.[35]

Address for Correspondence: Jami L DelliFraine PhD, Management, Policy and Community Health Division, University of Texas Health Science Center, Houston, TX. Email: jami.l.dellifraine@uth.tmc.edu.

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

REFERENCES

1. Mayron R, Long RS, Ruiz E. The 911 emergency telephone number: impact on emergency medical systems access in a metropolitan area. *Am J Emerg Med*. 1984;2(6):491-493.
2. Schroeder EB, Rosamond WD, Morris DL, et al. Determinants of use of emergency medical services in a population with stroke symptoms: the Second Delay in Accessing Stroke Healthcare (DASH II) Study. *Stroke*. 2000;31:2591–2596.
3. Morris DL, Rosamond W, Madden K, et al. Pre-hospital and emergency department delays after acute stroke: the Genentech Stroke Presentation Survey. *Stroke*. 2000;31:2585–2590.
4. Curran C, Henry C, O'Connor KA, et al. Predictors of early arrival at the emergency department in acute ischaemic stroke. Ir J Med Sci. 2011;180(2):401-405. Epub 2011 Feb 6.
5. Williams LS, Bruno A, Rouch D, et al. Stroke patients' knowledge of stroke. Influence on time to presentation. *Stroke.* 1997;28:912–915.
6. Morris DL, Rosamond WD, Hinn AR, et al. Time delays in accessing stroke care in the emergency department. *Acad Emerg Med*. 1999;6:218 –223.
7. Lacy CR, Suh DC, Bueno M, et al. Delay in presentation and evaluation for acute stroke: Stroke Time Registry for Outcomes Knowledge and Epidemiology (S.T.R.O.K.E.). *Stroke.* 2001;32:63– 69.
8. Rossnagel K, Jungehulsing GJ, Nolte CH, et al. Out-of-hospital delays in patients with acute stroke. *Ann Emerg Med*. 2004;44:476–483.
9. Adams HP Jr, del Zoppo G, Alberts MJ, et al. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atheros. *Circulation.* 2007;115(20),e478-534.
10. California Acute Stroke Pilot Registry (CASPR) Investigators. Prioritizing interventions to improve rates of thrombolysis for ischemic stroke. *Neurology.* 2005;64(4):654–659.
11. Deng YZ, Reeves MJ, Jacobs BS, et al. IV tissue plasminogen activator use in acute stroke: experience from a statewide registry. *Neurology*. 2006;66:306–312.
12. George MG, Tong X, McGruder H, et al. Centers for Disease Control and Prevention (CDC). Paul Coverdell National Acute Stroke Registry Surveillance - four states, 2005-2007.*MMWR Surveill Summ*. 2009;58(7):1-23.
13. Wein TH, Staub L, Felberg R, et al. Activation of emergency medical services for acute stroke in a nonurban population: the T.L.L. Temple Foundation Stroke Project. *Stroke*. 2000;31:1925–1928.
14. DuBard CA, Garrett J, Gizlice Z. Effect of language on heart attack and stroke awareness among U.S. Hispanics. *Am J Prev Med*. 2006;30(3):189-196.
15. Kleindorfer D, Lindsell CJ, Moomaw CJ, et al. Which stroke symptoms prompt a 911 call? A population-based study. *Am J Emerg Med.* 2010;28(5):607-612.
16. Jurkowski JM, Maniccia DM, Dennison BA, et al. Awareness of necessity to call 911 for stroke symptoms, upstate New York. *Prev Chronic Dis*. 2008;5(2):A41.
17. Mosley I, Nicol M, Donnan G, et al. Stroke symptoms and the decision to call for an ambulance. *Stroke* 2007;38(2):361–366.
18. Fussman C, Rafferty AP, Lyon-Callo S, et al. Lack of association between stroke symptom knowledge and intent to call 911: a population-based survey. *Stroke*. 2010;41(7):1501-1507.
19. Ritter MA, Brach S, Rogalewski A, et al. Discrepancy between theoretical knowledge and real action in acute stroke: self-assessment as an important predictor of time to admission. *Neurol Res*. 2007;29(5):476-479.
20. Andersen R, Aday LA. Access to medical care in the U.S.: realized and potential. *Med Care*. 1978;16(7):533–546.
21. Aday LA. Economic and noneconomic barriers to the use of needed medical services.*Med Care*. 1975;13(6):447-456.
22. Shi L, Stevens GD. Vulnerability and unmet health care needs. The influence of multiple risk factors. *J Gen Intern Med*. 2005;20(2):148-154.
23. Shi L, Stevens GD, Lebrun LA, et al. Enhancing the measurement of

health disparities for vulnerable populations. *J Public Health Manag Pract*. 2008;14 Suppl:S45-52.

24. CDC BRFSS. 2009. Available at: http://www.cdc.gov/brfss/technical_infodata/surveydata/2009.htm Accessed December 24, 2010.
25. American Stroke Association. Learn to Recognize a Stroke. Available at:www.strokeassociation.org. Accessed December 28, 2010.
26. Centers for Disease Control and Prevention (CDC). Awareness of stroke warning signs--17 states and the U.S. Virgin Islands, 2001. *MMWR Morb Mortal Wkly Rep*. 2004;53(17):359-362.
27. Lutfiyya MN, Bardales R, Bales R, et al. Awareness of heart attack and stroke symptoms among Hispanic male adults living in the United States. *J Immigr Minor Health*. 2010;12(5):761-768.
28. Centers for Disease Control and Prevention (CDC).Awareness of stroke warning signs--13 states and the District of Columbia, 2005. *MMWR Morb Mortal Wkly Rep*. 2008;57(18):481-485.
29. CDC BRFSS 2009 Summary Data Quality Reports. Available at: http://www.cdc.gov/brfss/technical_infodata/quality.htm Accessed June 2010.
30. Begley CE, Behan P, Seo m. Who uses hospital emergency rooms?: evidence from Houston/Harris County Texas. *J Health Care Poor Underserved*. 2010;21(2):606-616.
31. Fogle CC, Oser CS, Troutman TP, et al. Public education strategies to increase awareness of stroke warning signs and the need to call 911. *J Public Health Manag Pract.* 2008;14(3):e17-22.
32. Rosamond W, Evenson K, Schroder E, et al. Calling emergency medical services for stroke: A study of 911 tapes. *Prehospital Emergency Care*. 2005;9:19–23.
33. Handschu R, Poppe R, Rauss J, et al. Emergency calls in acute stroke. *Stroke*. 2003;34:1005–1009.
34. Ajzen, I. The theory of planned behavior. *Org Behav Hum Decis Process*. 1991;50:179–211.
35. Tadros A, Crocco T, Davis SM, et al. Emergency medical services-based community stroke education: pilot results from a novel approach. *Stroke*. 2009;40(6):2134-2142.
36. Jurkowski JM, Maniccia DM, Spicer DA, et al. Impact of a multimedia campaign to increase intention to call 911 for stroke symptoms, upstate New York, 2006-2007. *Prev Chronic Dis*. 2010;7(2):A35.

Call for Papers

Gender-Specific Research in Emergency Medicine:
Investigate, Understand and Translate How Gender Affects Patient Outcomes

The 2014 *Academic Emergency Medicine* Consensus Conference, **Gender-Specific Research in Emergency Medicine** will be held on Wednesday, May 14, 2014, immediately preceding the SAEM Annual Meeting Dallas, TX. Original papers on the this topic, if accepted, will be published together with the conference proceedings in the December 2014 issue of *Academic Emergency Medicine.*

Gender-specific medicine is the "science of how normal human biology differs between men and women and how the manifestations, mechanisms and treatment of disease vary as a function of gender." While gender-specific medicine incorporates advances in reproductive health issues, the AEM consensus conference will focus on broad disease-specific EM issues that are relevant to both women and men. The key domains of the conference are cardiovascular/resuscitation, cerebrovascular, pain, trauma/injury/violence, diagnostic imaging, mental health and substance abuse.

Consensus Goal:
The goal of the 2014 AEM Consensus Conference is to stimulate EM researchers to methodically recognize, investigate and translate the impact of gender on their clinical research outcomes. The conference proposes to build a foundation upon which researchers can build interdisciplinary scholarship, networks of expertise, discussion forums, multicenter collaborations, evidence-based publications, and improved education. The overarching themes of the conference have been guided and informed by NIH research priorities on gender medicine and include study of the lifespan, sex/gender distinctions, health disparities/differences and diversity and interdisciplinary research.

Consensus Objectives:
1) Summarize and consolidate existing data and create a blueprint that furthers gender-specific research in the prevention, diagnosis and management of acute diseases

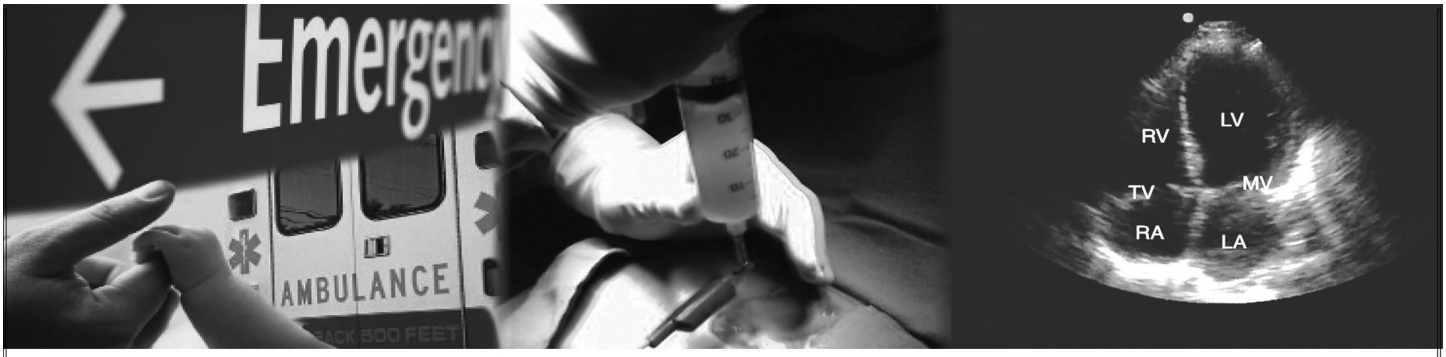
2) Discuss the conceptual models for designing studies and analysis that incorporate gender as an independent variable.

3) Build a multinational interdisciplinary consortium to study gender medicine for acute conditions.

Accepted manuscripts will describe relevant research concepts in gender-specific areas with priority placed on differential disease risk, vulnerability, progression and outcomes. They may include work in clinical/translational, health systems, policy or basic sciences research. Descriptions of specific research, projects, or collaborations may be used for illustrative purposes but should not comprise the core of the submission. Original contributions describing relevant research or concepts on these or similar topics will be considered, and original high-quality research may also be submitted alone or in conjunction with concept papers. Papers will be considered for publication in the December 2014 issue of *Academic Emergency Medicine* if received by Monday, March 11, 2014. All submissions will undergo peer review and publication cannot be guaranteed.

For queries, please contact Marna Rayl Greenberg, DO, MPH (Marna.Greenberg@lvh.com) or Basmah Safdar, MD (basmah.safdar@yale.edu) the 2014 Consensus Conference Co-Chairs.

Information and updates will be regularly posted in *Academic Emergency Medicine*, the SAEM Newsletter, and the journal and SAEM websites.



JOB ANNOUNCEMENT

INSTRUCTOR, ASSISTANT OR ASSOCIATE PROFESSOR POSITIONS DEPARTMENT OF EMERGENCY MEDICINE AMERICAN UNIVERSITY OF BEIRUT, FACULTY OF MEDICINE AND MEDICAL CENTER BEIRUT, LEBANON

The Department of Emergency Medicine is recruiting for full-time academic positions at the Instructor, Assistant or Associate Professor levels. Candidates must be experienced Emergency Medicine Physicians, graduates of nationally recognized Emergency Medicine residency training programs or board-certified or -eligible in Emergency Medicine by the American Board of Emergency Medicine or the American Board of Osteopathic Emergency Medicine, and must be fluent in English and, preferably, Arabic though the latter is not a requirement. Excellent opportunities exist for faculty development, research and teaching. The compensation is competitive and the positions offer excellent benefits.

Applicants should submit electronically: curriculum vitae, the names and addresses of four references, a summary of their accomplishments in the areas of clinical scholarly activities, teaching and research; and future plans. All requested documents should be forwarded to Dr. Eveline Hitti, Interim Chairperson of Department of Emergency Medicine, at the following e-mail address eh16@aub.edu.lb

Eveline Hitti, MD.
Assistant Professor of Clinical Emergency Medicine
Department of Emergency Medicine
American University of Beirut
P.O. Box 11-0236 - Riad El Solh 1107 2020
Beirut - Lebanon

AUB is an affirmative action institution and an equal opportunity employer

SCHOOL OF MEDICINE
UNIVERSITY of CALIFORNIA · IRVINE
Discover · Teach · Heal

Health Sciences Clinical Professor Series, Open Ranks
Department of Emergency Medicine

The University of California, Irvine School of Medicine, Department of Emergency Medicine anticipates openings in the HS Clinical Professor Series.

Requirements: The HS Clinical Series includes substantial patient care, medical student and resident teaching, and optional clinical research. Board preparation or certification in EM required. Fellowship or advanced degree, or both, strongly desired. The University of California, Irvine Medical Center is a 472-bed tertiary care hospital with all residencies. The ED is a progressive 35-bed Level I Trauma Center with 40,000 patients, in urban Orange County. Collegial relationships with all services. Excellent salary and benefits with incentive plan.

Salary and rank will be commensurate with qualifications and experience.

Application Procedure: Interested candidates should apply through UCI Irvine's RECRUIT system located at: https://recruit.ap.uci.edu/apply/.

Applicants should complete an online application profile and upload the following application materials electronically to be considered for the position:

1. Curriculum Vitae
2. Names and addresses of four references

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

SCHOOL OF MEDICINE
UNIVERSITY of CALIFORNIA · IRVINE
Discover · Teach · Heal

Post-Doctoral Scholar Fellow

The University of California, Irvine School of Medicine's Center for Trauma and Injury Prevention Research (CTIPR) is committed to the reduction of associated personal and societal burden of traumatic injury through conducting multidisciplinary research, translating research into policy and practice, serving as a regional and national resource, and working in close partnership with communities. The Center is based in the Department of Emergency Medicine and has strong working relationship with Trauma Surgery and the Trauma Registry. Recent projects include injury prevention, alcohol screening and brief intervention, and management mental health issues in the emergency department.

The Post-Doctoral Fellow will carry out injury research in close collaboration with mentors and colleagues in the Department of Emergency Medicine, Program in Public Health, and School of Social Ecology. The fellow will analyze data from current research projects and existing traffic injury data sets, develop skills as an independent researcher, and develop new projects.

Minimum qualifications:
Required doctoral degree in epidemiology, public health, or safety research, with a focus on injury, alcohol, or mental health research.

Other considerations:
1. Strong analytic skills and outstanding individual initiative.
2. Strong skills in data management and analysis, including experience using standard statistical packages.
3. Excellent scientific writing and spoken English skills.
4. Preference is given to applicants whose training and research interests align with the CTIPR.

Anticipated salary range:
http://www.som.uci.edu/academic-affairs/docs/postdoc.pdf

Applications are accepted until the position is filled.

Submit letter of interest, resume, research interests, and three references to:
Shahram Lotfipour, MD MPH (SHL@uci.edu, 714-456-2326) and Bharath Chakravarthy MD MPH (bchakrav@uci.edu, 714-456-6986).

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

The University of Utah
Division of Emergency Medicine

Academic-affiliated Community Physicians

The Division of Emergency Medicine at the University of Utah Health Sciences Center is recruiting for a number of physicians to staff University-affiliated community hospital emergency departments in western Wyoming and Utah regions. These hospitals are located in rural community sites that will also be used for training medical students, residents, and fellows. Direct access to the main University hospital will be available by EMR, telemedicine, and air medical transport. Opportunities for part-time work, off-site CME, and blended academic practices are also available.

The University of Utah is the primary medical teaching and research institution in the state. Candidates must be board certified/prepared and have an interest in education of residents and medical students. A competitive salary with an excellent benefits package is offered. The University of Utah is an EEO/AA employer and encourages applications from women and minorities.

Interested parties must apply online: http://utah.peopleadmin.com/postings/28004
or if you need more information, please contact:

Erik D. Barton, MD, MS, MBA
Division Chief
Division of Emergency Medicine
University of Utah School of Medicine
30 North 1900 East, RM 1C26
Salt Lake City, Utah 84132
(801) 581-2730
Fax: (801) 585-6699
erik.barton@hsc.utah.edu

UC Irvine Health
School of Medicine

The University of California, Irvine, School of Medicine, invites applicants for **Chair** of the **Department of Emergency Medicine.**

The University of California, Irvine, has a vibrant scientific community with outstanding collaborative opportunities. The Emergency Department is a 35-bed clinical unit with 7 resuscitation bays, caring for more than 47,000 patients per year. The ACS-verified Level I trauma center has 3600 activations per year, and the ACS-verified Burn Center cares for more than 300 patients. UC Irvine was recently verified officially as a Level II Pediatric Trauma Center. In addition, the department is designated as a Base Hospital, Joint Commission and Orange County Cardiac and Stroke Receiving Center. The department is the most capable in the area for disaster preparedness and response, and is one of only nine comprehensive emergency departments in California.

The department houses 19 full-time faculty (2 Ph.D.), four Clinical Instructor fellows, and a fully accredited PGY 1, 2, 3 EM residency (since 1988). The research effort is focused in the Center for Trauma and Injury Prevention Research and the Center for Disaster Medical Sciences. The Division of Emergency Ultrasound is internationally known, as UC Irvine was the first medical school to adopt a four-year integrated ultrasound curriculum. Other faculty in the department lead significant efforts in educational technology and simulation, and EMR implementation. The department publishes the Western Journal of Emergency Medicine: Integrating Emergency Care with Population Health, an open-access peer reviewed international journal.

Applicants for this position must have an M.D., or M.D./Ph.D. degrees, with Master's degree and/or subspecialty fellowship training desirable. Board certification in Emergency Medicine is required, as is an academic record sufficient for appointment in the Clinical X (Scholar) Line or In-Residence series at the full professor level. Candidates must have a strong record of scholarly activity and peer reviewed publications, including a research program with extramural funding. The candidates should also hold, or be eligible for, a medical license in the State of California. The successful candidate will be responsible for the effective management of all administrative and operational processes of the department, providing not only comprehensive, interactive clinical services, but also supporting the teaching, educational and research missions of the department, school and university. The candidate must have strong interpersonal skills, and be able to work cooperatively and congenially within a diverse academic and clinical environment. Candidates with leadership skills and vision for enhancing the clinical and academic components of a multi-disciplinary department are especially encouraged to submit applications to:

Applicants should complete an online application profile and upload their Curriculum vitae electronically to be considered for the position: https://recruit.ap.uci.edu/apply/JPF02153.

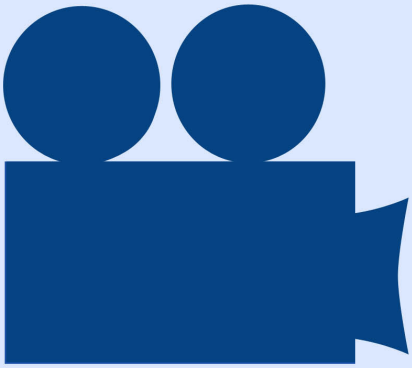
UCI is an equal opportunity employer committed to excellence through diversity and strongly encourages applications from all qualified applicants, including women and minorities. UCI is responsive to the needs of dual career couples, is dedicated to work-life balance through an array of family-friendly policies, and is the recipient of an NSF ADVANCE Award for gender equity.

REC

WestJEM CALL FOR VIDEO SUBMISSIONS

We are looking for recordings on:

- Ultrasound
- CT reconstruction
- MRIs
- Instructional/clinical techniques

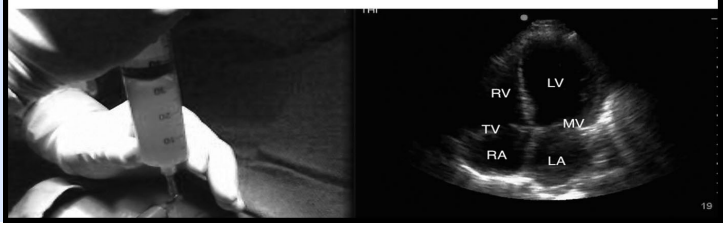


For inquiries & submission guidelines please contact: editor@WestJEM.org

As the scope of the *Western Journal of Emergency Medicine* expands, we are actively seeking section editors for the following areas of interest:

Disaster Medicine
Geriatrics
Resident Student Forum
Neuroscience
Trauma

*West*JEM is also seeking reviewers in all topics of emergency medicine. Please email your CV to Mark Langdorf at editor@westjem.org



“Being a part of a democratic group means that you get to make decisions affecting your practice daily.”

“It’s an amazing experience. I absolutely enjoy it and truly believe in CEP America’s Partnership model.”

—True McMahan, MD
ED Medical Director
Garden Grove Hospital

Find out why CEP America is different.
Hear True’s story by visiting
learn.cep.com/true-md

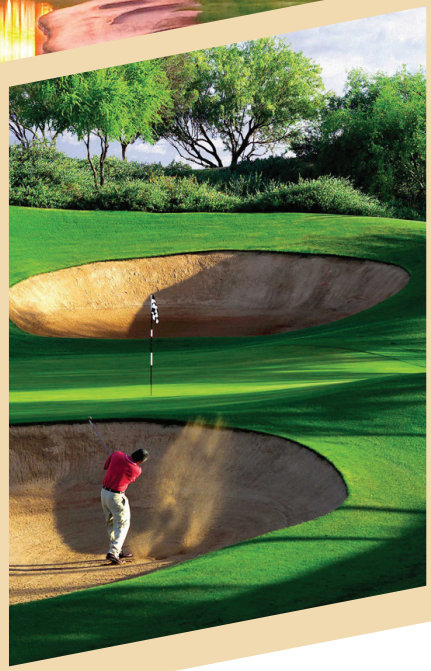
Your Life. Your Career. Your Partnership.

CEP America®

ACOEP Presents

THE EDGE '14

SPRING SEMINAR



SAVE THE DATE

***The Edge* - Spring Seminar 2014**
The Westin Kierland Resort & Spa
Scottsdale, Arizona

April 22 - 26, 2014

For More Information Visit:
www.acoep.org/edge